A text-book of the practice of medicine.

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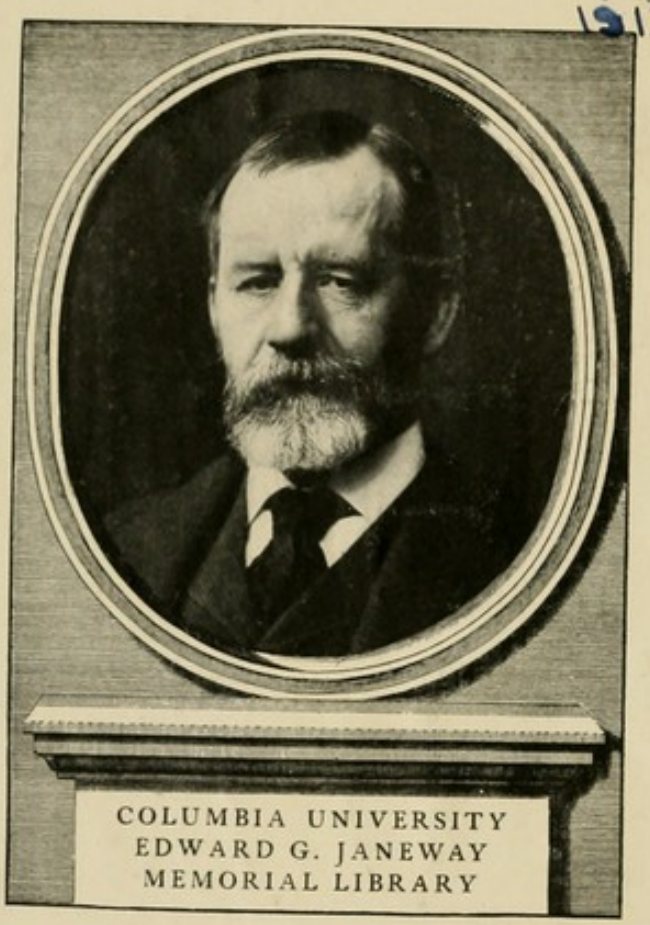
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A TEXT-BOOK

OF THE

PRACTICE OF MEDICINE

BY

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ILLUSTRATED

ELEVENTH EDITION, THOROUGHLY REVISED

PHILADELPHIA AND LONDON
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PREFACE TO THE ELEVENTH EDITION.

THE revision of this Edition, upon which constant serious consideration has been bestowed during the past two years so as to bring it abreast of our present knowledge of the diagnosis and treatment of disease, has it is hoped yielded substantial improvements. It is believed that by far the majority of modern methods of diagnosis of medical complaints have been presented with sufficient detail, while it has been found necessary to make mere mention of a few special tests, together with their references, owing to the exigencies of space. The subject of the treatment of individual diseases in text-books of medicine too often receives scantier attention than its intrinsic importance would seem to warrant. This branch of my theme has, therefore, received close scrutiny, and where demanded it has been enlarged.

Great care has also been taken to harmonize the Pathologic Sections with the present-day opinion of specialists in this department of medical science. As in former editions, so in the present one, the new matter introduced has been systematically classified.

While the work throughout has been carefully revised, among the more important additions may be mentioned: McPhedran's sign of peritonitis in typhoid fever, Burke's reflex sign in typhoid fever, Prendergast's test in typhoid fever, phlebotomy and transfusion in hemorrhage of typhoid fever, hot-air inhalations in diphtheria, Lee's sign in acute articular rheumatism, Iron's method of diagnosis of gonorrheal arthritis, Pastia's sign of scarlet fever, copper arsenite and copper sulphate in amebic dysentery, Erb's syphilitic spinal paralysis, Weil's test in syphilis, vegetable days in diabetes, sugar solution in diabetic acidosis, effect of atophan in gouty subjects, radium emanations in gout, salvarsan and sodium cacodylate in progressive pernicious anemia, benzol in leukemia, vaccine treatment of goiter, hexamethylenamine in acute bronchitis, artificial pneumothorax in hemoptysis, Schepelmann's sign in acute plastic pleurisy, oxygen in sero-fibrinous pleurisy, Stern's sign in tricuspid incompetency, Graham-Steell murmur in pulmonary incompetency, Karell milk-cure in valvular heart disease, electricity in arterial sclerosis, diastolic expiration in aneurysm, Boas' method of testing motor function of stomach, McCaskey's method of treating gastroptosis, Meiostagmin reaction in gastric cancer, Falk and Salomon's reaction in gastric cancer, larval superacidity, Boas' phenolphthalein

test for diagnosis of intestinal disease, Bastedo's test in appendicitis, chloride retention theory of renal dropsy, circumscribed serous spinal meningitis, progressive lenticular degeneration, dysbasia lordotica progressiva, myotonia atrophica, and the Towns-Lambert method of treating morphinism.

Of the new subjects which have been discussed, the following may be enumerated: Diseases of the parathyroid gland, auricular fibrillation, auricular flutter, extra systole, streptococcus tonsillitis, stenosis of the duodenum, Lane's kink of the ileum, status thymico-lymphaticus. In addition the following subjects have been rewritten: Antityphoid vaccination, diseases of the thymus gland, and pellagra.

I can only hope that the present Edition will maintain the position and standing which its predecessors have hitherto held. My indebtedness to Dr. H. Leon Jameson for kind aid cannot be sufficiently expressed. The Section on Nervous Diseases was kindly revised by Dr. Charles S. Potts, who by reason of his standing as a neurologist is eminently fitted for the task.

JAMES M. ANDERS.

1605 Walnut Street, Philadelphia,
September, 1913.

PREFACE.

THIS work is meant to introduce the student to the present state of our knowledge of the practice of medicine in general and of the diagnosis, differential diagnosis, and treatment of disease in particular. The historic development of the subjects treated has been either briefly given or intendedly omitted, since this scarcely falls within the scope of a practical treatise on medicine. Although the book as a whole is submitted to the critical judgment of a learned profession, it may be pardonable to emphasize, provisionally, a few features pertaining to the mode of treating the separate subjects, or the arrangement of the material under the latter—to indicate some of the more salient lineaments, so to speak, in the general design. Since in medical schools it is taught from a separate chair, the pathology (special) of the individual affections has almost invariably been taken up before the etiology; from this point the student will find the story of each affection a continuous one. The practitioner, however, must ever aim to associate the clinical symptoms with the morbid lesions.

Under special etiology the bacteriology has been prominently mentioned, since we owe to it the rapid progress that is being made in the study of the causation of disease.

The differential diagnosis has in many instances been tabulated—an ear-mark that I confidently believe will be found especially helpful. It may be stated that not less than fifty-six diagnostic tables are scattered throughout the work, and that by far the greater number of these are my own.

Such formulæ have been introduced into the text, and only such, as a more or less extended experience has shown to be possessed of real therapeutic importance. Whilst these, and all additional points relating to the treatment of the single affections, may serve as guides, particularly to the beginner, I fully appreciate how often the practising physician is

placed in a position in which he is compelled to form a therapeutic judgment for himself. Whenever the dosage is stated, the metric equivalent is placed in parentheses, the number of grams being stated in round numbers (℥j—4.0; ℥j—32.0) in order to render it of greater practical value. In all instances, however, in which this would involve an important difference in quantity the exact decimal figures are given. A considerable variation from the usual classification of diseases may be observed, but this is accounted for in the text wherever it occurs.

Preference has been given to the modern orthography and terminology, not only because it is more euphonious, but also because of its adoption by the standard lexicographers.

I have gleaned without stint from medical literature with a view to bringing the book up to date, and if I have failed to give full credit in every instance, my grateful acknowledgments are here due and are cheerfully made. The chief results of my personal experience and observation, extending over a period of two decades, and derived from both hospital and private practice, will also be found upon these pages.

I wish to thank Prof. W. C. Hollopeter, who has written some of the articles upon the diseases of children, as measles, chicken-pox, mumps, whooping-cough, and the acute diarrheas, and who has kindly aided in the preparation of those upon diphtheria and scarlatina.

My cordial thanks are due also to Dr. C. L. Furbush for kind aid in preparing some of the illustrations, to Doctors Robert N. Willson, Howard S. Anders, and Geo. W. Pfromm for valuable assistance while the work was passing through the press, and to Dr. A. M. Davis for preparing the index.

JAMES M. ANDERS.

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PART I.

INFECTIOUS DISEASES.

TYPHOID FEVER.

(*Enteric Fever; Abdominal Typhus; Ileo-typhoid; Nerven Fieber.*)

Definition.—An acute infectious disease of which the definitive cause is the specific bacillus of Eberth (*bacillus typhosus*). It is characterized, pathologically, by hyperplasia and sloughing of Peyer's patches and the solitary follicles of the intestines coupled with parenchymatous changes in the principal viscera, and clinically by its gradual onset, peculiar temperature-curve, swelling of the spleen, rose-colored spots, diarrhea, tympanites, sero-reaction, and a liability to certain complications (intestinal hemorrhage, perforation, etc.). The disease is a bacteremia, and typhoid infection is no longer an anatomic entity, and does not always produce typical typhoid fever.

History.—Although known beyond the reach of tradition, typhoid fever was clearly distinguished from typhus at a comparatively recent date. Louis of Paris in 1829 proposed the term *typhoide*, but it remained for Gerhard of Philadelphia to discriminate typhoid from typhus fever as the result of his own precise clinical observations. His account of the disease was ably corroborated by the writings of E. Hale and James Jackson, Sr. (1838, 1839). Later, Shattuck of Boston and Jenner of London made important contributions to the subject. Shattuck's experiments on typhus and typhoid fevers at the London Fever Hospital in England, and Alfred Stillé's studies of the former affection in Dublin and Naples, and of the latter in Paris, increased greatly our knowledge of these diseases. As a result of the labors of the above-mentioned American authors the true nature and identity of typhoid fever were appreciated in America at an earlier day than in either France or England.

Briefly, the decade from 1840 to 1850 witnessed the overthrow of erroneous notions concerning the similarity of typhoid and typhus fevers, on the one hand, and the establishment of their points of dissimilarity on the other.

Pathology.—The lesions produced by typhoid fever may conveniently be divided into two groups: (1) Primary lesions, due to the direct effect of the special bacillus upon the lymph-follicles of the intestines, the mesenteric and other lymph-glands, the gall-bladder, and the spleen. Typhoid septicemia without localizing lesions is also a recognized form of the disease. (2) Secondary lesions, due chiefly to the

indirect effect of the typhoid bacillus and secondary infection, for the occurrence of which the essential lesions furnish the golden opportunity.

(1) **The primary morbid changes** in the Peyer's patches and solitary glands of the intestines are divided, usually, into four stages:

(*a*) **The Stage of Infiltration.**—The lymph-follicles become engorged (hyperplasia), particularly Peyer's glands in the ileum and near to the valve, and, to a lesser extent, in the lower part of the jejunum. Frequently the solitary glands in the small intestines, the colon, and rarely the rectum, become similarly infiltrated. In mild cases a few Peyer's patches in the lower part of the ileum are alone the seat of infiltration and subsequent changes. The follicles are grayish-white in color, and may project from 3 to 5 mm. or more. Rarely the solitary glands, which vary in size from a mustard-seed to a large pea, also become prominent and show a bold attempt at pedunculation.

The *histologic changes* at first consist in a marked dilatation of the capillary blood-vessels, which later are more or less compressed (as a consequence of cell-infiltration), giving to the follicles their whitish, anemic appearance. The cellular elements partake of the nature of lymph-corpuscles. Some of these cells are larger and are epithelioid in character, with ten or more nuclei. The mucosa and muscularis adjacent to the glandular structures may be similarly infiltrated.

From the eighth to the tenth day the stage of infiltration terminates either in resolution (rare) or in necrosis and sloughing. The infiltrated cells may undergo granular or fatty degeneration, followed by absorption. This process—resolution—during its progress produces pitting of the swollen follicles. In consequence of these minute points of necrosis the plaques now present a characteristic reticulated appearance (plaques à surface réticulée). When resolution occurs accompanied by destruction of the follicles, small hemorrhages may take place into the glandular structure. These hemorrhages may occasion pigmentary deposits in the follicular depressions, giving rise to the so-called "shaven-beard" appearance.

(*b*) **Necrosis or Sloughing.**—In all save the milder grades of cell-infiltration the hyperplasia of the lymphatic tissue cannot subside before necrosis occurs. The latter process results partly from compression and choking of the blood-vessels by the cell-proliferation, and partly from the direct action of the typhoid bacillus, leading to so-called anemic necrosis. Thus, necrotic crusts (sloughs) are formed, which are gradually separated and cast off. While not all of the glands of Peyer which are the seat of cellular infiltration undergo subsequent necrosis, as a rule those situated in the lower portion of the ileum do, and show the process in its completest development. The depth to which the necrosis extends is quite variable. It may involve only the most superficial layers of the mucosa, or it may extend in depth till it reaches, or even perforates, the outer or serous coat; but usually this work of destruction does not dip below the submucosa or muscularis. The necrosed portions become detached—a process that proceeds from the periphery toward the center—leaving behind the typhoid ulcer. The stage of necrosis and sloughing begins between the eighth and tenth days, and ends on or about the twenty-first day.

(*c*) **Stage of Ulceration.**—The size and shape of the ulcers corre-

spond exactly to the necrosed areas in these respects. A single gland of Peyer generally presents several ulcers of irregular outline separated by strips of mucous membrane. Rarely, the entire plaque is implicated, in which case a large oval ulcer is the result, and at the lower end of the ileum the ulcers often coalesce until they almost encircle the bowel. The ulcers of the solitary glands assume a rounded form. The character of the floor of the ulcer will vary with the character of the intestinal coat which forms its base, though usually it is clean and smooth. The edges are usually irregular, engorged, soft, and frequently overhanging. In the lower segment of the ileum ulcers may be numerous, and in about 25 per cent. of the cases the typhoid ulcers are found in the large intestines—*i. e.*, in the cecum and colon.

Hemorrhage usually results from erosion of a vessel—an accident which is occasioned by the separation of the sloughs—but small bleedings may take place from the swollen, hyperemic edges of an ulcer. *Perforation* of the bowel occurs in a small percentage of cases (about 6 per cent.). J. A. Scott has pointed out two varieties: (*a*) Circular, pin-point in size, due to a perforative necrosis (common), and (*b*) a large aperture (as the result of extensive necrosis) ranging in size from the finger-tip to 3 cm. in diameter. The perforations may be multiple, though they are usually single. The small, deep ulcers are more apt to lead to complete perforation than larger ones, and the site of the orifice is usually in the course of the lower third of the ileum. The lesions of peritonitis invariably follow. Perforation of the large bowel is exceedingly rare. Exceptionally, the appendix is the seat of ulcer. Localized abscesses have been found under these circumstances. During the stages of necrosis and ulceration a catarrhal state of the mucosa of the intestines exists. The diarrhea which usually accompanies typhoid is ascribable, in part at least, to the catarrhal state of the large bowel.

(*d*) **Healing** follows promptly upon the formation of the ulcer. At first a granular tissue covers its floor. The mucous membrane is replaced, including the glandular elements and epithelial layer, and, as in the stage of necrosis and sloughing, so the healing process advances inward from the border of the ulcer. Indeed, it is this process that dislodges the necrotic crust. Occasionally, ulcers are seen extending in one direction while healing in another. The cicatrix formed by the healing of an ulcer presents a smooth and often pigmented surface.

The stages thus far described do not, strictly speaking, follow one another, since two or more may be illustrated at once by a group of ulcers occupying the intestine. When death occurs during a relapse fresh ulcers are observed by the side of those partially healed.

The Mesenteric Glands.—Changes in the mesenteric glands occur simultaneously with those in the intestines, and those situated opposite to the lower third of the ileum, the portion of the bowel showing the most extensive ulceration, are most profoundly involved. Hyperemia, and later swelling due to cell-infiltration, are among the earliest changes, and correspond with the lesions noted in the intestines (*vide supra*). The mesenteric glands exhibit great variations in size, ranging, as they do, from that of a pea to a hen's egg. Their color appearance is a grayish red. Resolution commonly occurs, but, if it does not, then necrosis of the central portion (due, most probably, to the same causes that

produce necrosis of the intestinal lymph-follicles) occurs. Le Conte[1] believes that perforation of the capsule of the glands, when it occurs, is due either to the presence of the staphylococcus or streptococcus or to thrombosis of the larger vessels of the mesentery outside of the glands. Still other glands become hyperemic and swollen (retroperitoneal, bronchial); but these usually tend toward resolution.

The Spleen.—With rare exceptions the spleen becomes enlarged in typhoid fever. At first hyperemic, the tissue then grows soft and granular, and at times is almost diffluent on section. Infarction is not a rare occurrence and may lead to suppuration. Keen has searched the literature and found only 9 cases of abscess. In rare instances, either spontaneously or as the result of injury, the spleen may rupture, and the records of 2000 post-mortems at the Munich Pathologic Institute furnish 5 cases. Perisplenitis rarely occurs (*vide* Spleno-typhoid, p. 45).

Gall-bladder.—The *gall-bladder* may show catarrhal inflammation, and rarely a croupous, diphtheritic, or ulcerative inflammation leading to perforation. Westcott has tabulated 30 cases of typhoid infection of the gall-bladder that resulted in perforation. Chiari's[2] and Flexner's[3] figures show that typhoid bacilli are found in the gall-bladder in more than 50 per cent. of the fatal cases. Chiarolanza[4] found that typhoid bacilli injected intravenously reached the gall-bladder in 17 out of 23 cases, entering through the capillaries of the mucosa and submucosa. (*Vide* Acute Infectious Cholecystitis.)

Mallory has shown that the typhoid bacillus produces a toxin which causes proliferation of the endothelial cells along the line of absorption from the intestines, both in the lymphatics and blood-vessels. These cells increase in size and number, and manifest phagocytic properties.

(2) **Secondary Lesions due Chiefly to the Continued Fever and to Secondary Infections.**—The lesions in other organs are of subsidiary importance, and are, for the most part, secondary in nature, though we cannot draw a sharp line of distinction between these and those that are primary. In the kidneys, pleura, pharynx, larynx and tonsils primary implantation of the typhoid bacillus may rarely occur (*vide infra*).

The *liver* early becomes hyperemic, and later is softer and paler than is natural. Handford has described necrotic areas, and Wagner minute lymphomata. Rarely infarction and abscess occur. Mesenteric abscess and perforative appendicitis may be followed by pylephlebitis. The microscope reveals parenchymatous and granular degeneration. The cells contain an abundance of fat, whilst their nuclei have lost, in great part, their outline.

The *kidneys*, like the liver, exhibit parenchymatous degeneration. They are somewhat pale-looking, are cloudy on section, and slightly swollen, and under the microscope granular and fatty degeneration of the epithelial cells of the convoluted tubules is observed. More rarely the lesions are those of *acute hemorrhagic nephritis*. Small areas of round-cell infiltration may develop late in the course of typhoid, and these may present an appearance similar to lymphomata or may undergo softening and suppuration, giving rise to miliary abscesses. The mucous membrane of the pelvis of the kidney is not infrequently the seat of a mild grade of catarrh, and, rarely, of diphtheritic inflammation.

[1] *Jour. Am. Med. Assoc.*, Oct. 22, 1904. [2] *Prag. med. Woch.*, 1903, No. 22.
[3] *Johns Hopkins Hosp. Reports*, vol. v. [4] *Ztschr. f. Hygiene u. Infectionskr.*, 1908, lxii., 1.

Typhoid cystitis is still more common, and the bladder may also be the seat of diphtheritic inflammation. Rarely orchitis is encountered. On making cultures from sections of the kidneys not a few observers have been able to demonstrate the presence of the specific bacillus of typhoid.

In the *lungs* are found morbid lesions in nearly all cases of typhoid fever, and belonging to the essential pathologic processes is bronchitis, due to a congested and catarrhal state of the bronchial mucous membrane. The lesions of lobular pneumonia present a complicating condition in many instances; those of lobar pneumonia also may be present, though less commonly. The so-called *hypostatic congestion* is often found, but is, I think, less frequent than is supposed by many authors. *Embolic infarctions*, having their origin in thrombi occupying the right side of the heart, are sometimes present. Gangrene may also occur.

Pleurisy is rarely met with. It is generally of the plastic variety, although empyema occurred in nearly 2 per cent. of the Munich cases. The initial lesion may be pleuritic. (*Vide* Pleuro-typhoid, p. 45.)

The *larynx* and the *pharynx* may manifest changes. Ulcers have been observed on the epiglottis and posterior wall of the larynx, and I have more than once seen them on the pharynx (*Pharingo-typhoid*). When situated in the larynx they may extend in depth till they reach the perichondrium, causing perichondritis, with or without edema of the larynx. Typhoid bacilli have been found in the ulcers (Eichhorst). Catarrhal, or even croupous, pharyngitis may occur, and a swelling of the follicles of the pharynx and base of the tongue is to be noticed in many cases. True aphthous changes, affecting the mouth and pharynx, may be present as a secondary event. The tonsils may present ulcers (*Tonsillo-typhoid.*) The *mucosa of the stomach* is sometimes congested, and may be the seat of typhoid ulcers, although this is rare.

Peritonitis is always found in fatal cases in which the bowel has been perforated. The condition is a general one, save in the rare instances mentioned below, and there is usually much fibrino-purulent effusion present. Diffuse peritonitis may be present without perforation, and results sometimes from a localization of the typhoid poison in the peritoneum, from rupture of suppurating mesenteric glands, and from direct extension of intestinal inflammation to the peritoneum.

The *heart* may be the seat of morbid changes. Acute endocarditis may be a very rare complication, while pericarditis occurs relatively more often—viz. in 14 of the Munich post-mortems before mentioned. Myocarditis is a common event, the cardiac muscle exhibiting parenchymatous and, less commonly, hyaline degeneration, and the latter change sometimes leads to sudden rupture of the muscular fibers, with a fatal result (myocardite ségmentaire). It is, however, a significant fact that in many instances, even of the severest type, the cell-fibers may show slight, if any, noticeable change. Out of 48 cases, 16 showed granular or fatty degeneration, and 3 a proliferative endarteritis in the small vessels (Dewevre).

The *arteries* have, in a number of instances, been found to be the seat of two forms of arteritis (Barié): (*a*) Acute obliterating arteritis and (*b*) partial arteritis. These conditions may affect the smaller ves-

sels, particularly those of the heart, but they occur most commonly in the arteries of the lower extremities. Thrombi are found in the right chambers of the heart and in the veins—most frequently in the femoral, and less often in the cerebral, sinuses. According to Flexner, thrombi may be caused by auto-agglutination of the red cells.

The *voluntary muscles* undergo parenchymatous and, occasionally, a hyaline change, though this is not a feature peculiar to typhoid fever. The latter form of degeneration does not affect the whole muscle, but only certain fibers, and, as a rule, the recti abdominis, the diaphragm, the adductors of the thigh, and the pectorals are the seats of the lesion. The parts affected are pale and possess a grayish, waxy lustre. Histologically, the process implies the transformation of the muscular fibers, and especially the cement substance, into a homogeneous, pliable mass. Regeneration of the fibers occurs during convalescence. Hemorrhages into, and rarely abscesses in, the intermuscular tissue occur.

The *nervous system* presents no gross lesions, if we except meningitis, the latter occurring as a complication; but it is exceedingly rare, having been present in only 11 of the 2000 Munich cases. In a few instances large cerebral hemorrhages have been met with, but these are apparently coincidental, while capillary hemorrhages into the cortex may be numerous. Meningeal hemorrhages may also occur. Slight edema of the cerebral cortex has been noted. The peripheral nerves are not infrequently the seat of parenchymatous change, with or without local neuritis, and the ganglia of the trunks of the vagi exhibit an inflammatory change which Levin believes is the cause of the laryngitis, pharyngitis, pharyngolysis, and arrhythmia sometimes observed.

The *blood* shows few important alterations. The red blood-corpuscles are relatively increased during the febrile period and markedly diminished during convalescence, but the great loss of water during the former period and a reabsorption during the latter will explain these interesting facts (Henry). Leukocytosis is absent (*vide infra*, p. 40).

Etiology.—Bacteriology.—The bacterium which is the specific cause of typhoid fever was discovered by Eberth, whose researches were later confirmed by the investigations of Gaffky and others.

General Characters.—It is a short, thick bacillus, about three times as long as it is broad, with rounded ends (Fig. 1). It is motile, due to the presence of cilia, and when stained, exhibits vacuolations that have been mistaken for spores. It is easily stained with all the anilin dyes.

Characteristic Growth.—Upon gelatin plates it develops in grayish translucent colonies with irregular borders and ridged surfaces. Upon agar the growth is not characteristic; upon the potato, especially if it has been rendered slightly acid, it forms a perfectly transparent growth that is only evident as a slight apparent increase of moisture upon the surface, and as offering a greater resistance to the point of the needle when scraped across it. It neither coagulates milk, liquefies gelatin, nor produces indol. The organism never forms spores. Moreover, the bacillus has no greater powers of resistance than the ordinary bacteria.

Experimental Typhoid.—Inoculated into lower animals, the bacillus frequently causes fatal results without producing the lesions characteristic of typhoid in human beings, although occasionally typical typhoid ulcers have been found. The susceptibility of lower animals, though nor-

mally slight, can be increased by preliminary injections of saprophytic bacteria, this result having been obtained by Alessi when he exposed animals to the gases produced by putrefying matters. It has been found that the ulcerative intestinal lesions produced by the inoculation of the bacilli or their toxins in large quantities into the blood of rabbits may also be caused by other bacteria, including the bacillus coli commune. Metchnikoff,[1] however, has administered foods contaminated with weak dilutions of bacillus-infected feces to chimpanzees; they contracted characteristic typhoid fever.

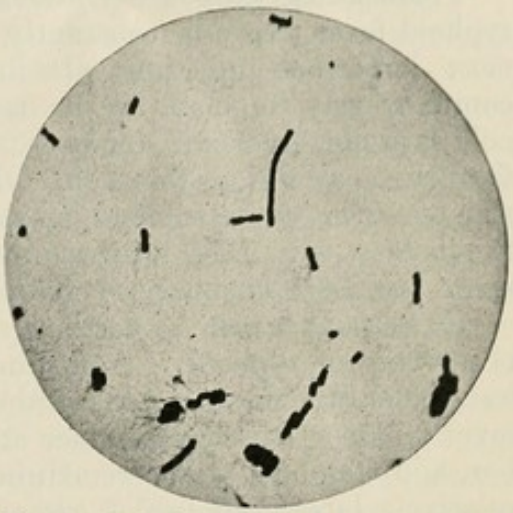


FIG. 1.—Typhoid bacilli with flagella; × 1000.

Usually, in making a *bacteriologic diagnosis* the typhoid bacillus is to be differentiated from those organisms that morphologically resemble it and present almost identical characteristics upon various culture-media, such as the bacillus of Shiga, paracolon bacilli, and the bacillus coli communis. These organisms can now be separated from the bacillus of Eberth, bacteriologists having pointed out the fundamental differences between these related forms. Smith and Tennant, in a study of the 1898 epidemic of typhoid in Belfast, failed to find typhoid bacilli in the water-supply, but were able to isolate varieties of the bacillus coli communis.

The real poison of typhoid fever is most probably a ferment (?) secreted by the bacillus—*typho-toxin*; and Brieger has extracted the latter agent, finding that it produces the fever, nervous symptoms, and the other manifestations characteristic of the affection.

Distribution in the Body.—The bacillus has been found in the intestinal tract, the lymph-glands, the contents of the intestine, the spleen, the liver, the gall-bladder, the rose-colored spots, the blood, and the bile. The bacillus typhosus is demonstrable in the stools (in about 50 per cent. of the cases), the urine (Wright and Semple), the sputa, the vomita, milk, and the sweat. The bacillus typhosus is most abundant in the duodenum and jejunum; it is practically constant in cultures made from the mucosa of the stomach (Jürgens). Less commonly it has been found in foci of suppuration and in exudations (pleural, endocardial).

The Bacilli Outside the Body.—The bacilli cannot maintain a permanent existence outside the human body. From time to time, however, the conditions indispensable to the growth and development of the typhoid germs prevail, and corresponding with such periods of time more or less extensive epidemic outbreaks of the disease may occur. It is known that the typhoid bacilli may retain their vitality for from seven to fourteen days in water, disappearing from the same on account of the presence of saprophytes; but an epidemic or an endemic of typhoid fever implies persistent contamination of the drinking-water. Multiplication of the bacilli may take place in water, in milk (very rapidly), and in the soil, where they preserve their vitality under favorable conditions (for eleven months—Robertson). Freezing does not kill them, as they may

[1] *Jour. Amer. Med. Assoc.*, April 16, 1910.

live in ice for several months (Prudden). They have been discovered in infected water, but they are thoroughly destroyed by boiling.

Predisposing Causes.—(*a*) **Geographic Location.**—In temperate zones typhoid fever prevails constantly to a greater or less extent, and is the most important infection. It has been shown in recent times to be comparatively common in the tropics (*e. g.*, India) as well as in many cold latitudes (Iceland, Norway). It exhibits an appalling prevalence and fatality in armies in the field. For example, in the Spanish-American war one-fifth of the soldiers in the national encampments suffered from the disease, with 1580 deaths; while in the South African war the British army lost 7991 men from typhoid fever and only 7582 died of wounds.

(*b*) **Seasons** exert a decided influence upon the frequency of the occurrence of typhoid. According to the statistics of Murchison, Bartlett, Osler, Hirsh, and others, the time of greatest liability to typhoid fever is during the late summer and the early autumn (August, September, and October). The remaining summer and autumn months yield a relatively larger number of cases than the winter and spring: again, in winter more cases are met with than in the spring, which furnishes fewest number of cases. After hot and dry summers typhoid fever is especially apt to be prevalent, and, according to Baumgarten, a relatively large amount of dust in the atmosphere may disseminate the typhoid germs. Epidemics, however, may occur at any season.

(*c*) **Condition of the "Ground Soil."**—Pettenkofer and his disciples contend that when the standing water in the soil reaches a high level fewer cases occur, and when it falls to a low level or below the mean height the cases become more numerous. This dictum, however, has not as yet been conclusively proven with reference to many localities. The poisoned foci, may be more effectively drained by the springs and streams, since the latter contain an increased quantity of solid matter when the ground-water is low. Epidemics of typhoid fever, however, occur repeatedly without regard to the condition of the ground-water.

(*d*) **Age.**—Typhoid fever may occur at any age. It is, however, especially frequent among young, robust individuals between the ages of fifteen and thirty years. Later in life it becomes progressively less frequent, though cases have occurred at or beyond the seventieth year. Young children are not exempt, and cases among them are of rather frequent occurrence, if we except those under one year of age. When contracted late in pregnancy typhoid may be congenital (Freund and Levy). The typhoid bacilli have been successfully cultivated from the fetus, and Mossé and Fraenkel[1] have confirmed the observation that the Widal test can be obtained from the placenta and blood of the fetus.

(*e*) **Sex** probably does not affect the degree of liability in typhoid.

(*f*) **Individual Predisposition.**—This may be *acquired* or *inherited*. An instance of *acquired* predisposition is to be noted in the great susceptibility which exists among persons who have recently moved from rural districts to cities. Thus, Louis found "that of 129 cases, 73 had not resided in Paris over ten months, and 102 not over twenty months." Defective ventilation, filth, overcrowding, and imperfect drainage increase susceptibility. There is evidence to show that the disease is on the increase in rural sections. Predisposition to typhoid may also be *inherited*. Most persons, however, enjoy natural immunity from the affection.

[1] *Journ. des Practiciens*, Jan. 28, 1899.

(*g*) **Intestinal Catarrh.**—Cases of influenza with catarrh of the gastro-intestinal tract may be followed by typhoid fever.

(*h*) **Nervous Influences.**—Great mental excitement and overwork.

Immunity.—The occurrence of typhoid fever confers an approximate, though not an absolute, immunity against subsequent attacks.

In this connection two questions present themselves for consideration:

(1) What are the methods of conveyance of the poison into the body?

Isolated cases and epidemics of typhoid fever are alike to be attributed to antecedent cases of the disease, and this fact presupposes that the bacillus of typhoid leaves the body of the sufferer. It does so in the stools, the urine, and occasionally in the vomitus and sputum. Park states that examinations, both in Europe and America, show that fully 2 per cent. of persons who have had typhoid fever are typhoid bacilli carriers. Hutchinson's[1] investigations show that 8.3 per cent. of typhoid fever patients are excreting the causal organisms when discharged from the hospital, while 6.3 per cent. of these do not continue to be infective for more than one month from that time. A few of them pass infected urine, but most, infected feces. During the declining and post-febrile stages the urine is probably the most dangerous excretion containing bacilli. Finally, one in every five hundred healthy adults who have never knowingly had typhoid fever is a carrier through contact with infection. It would seem that there are several periods of "effectiveness" and "ineffectiveness" (the latter coinciding with the early months of the year) of typhoid bacilli carriers. According to the Germans, the most common source of the bacillus is a patient or chronic carrier, the latter causing about 10 per cent. of the cases. Conradi claims that four times as many women as men become chronic carriers. Typhoid bacilluria may persist for a long time after apparent recovery. The dejecta and the urine, which are the principal sources of infection, may be conveyed to well persons by—

(*a*) **Infected Drinking Water.**—In most instances the poison is transmitted from those affected with the disease to those in health through the drinking-water supply, as shown by many epidemic outbreaks in which the mode of origin has been traced. Wells, storage reservoirs, springs, and rivers may become contaminated and cause an epidemic.

In the spring of 1885 a most deplorable epidemic occurred in Plymouth, Penna., a town of 8000 inhabitants. At first the nature of the affection was not recognized, and before it ceased to appear 1200 persons were affected, with 130 resulting deaths. This epidemic was investigated by Shakespeare and L. H. Taylor, and was found to have arisen from a single case of typhoid occurring in a house on a hill which sloped toward the water-supply of the town. This patient was ill during January, February, and March, while the ground was frozen and covered with snow, upon which the dejecta were thrown. On March 25th there was a considerable rainfall, followed by a sudden thaw, and the water ran at once through the various surface channels into a brook, which in turn emptied into the reservoir. On April 10th other cases of the disease appeared, and those citizens who obtained their water from other sources than the infected reservoir escaped. The recent outbreaks at Maidstone

[1] *Medical Chronicle*, Jan., 1912.

(1897) and at Butler, Pa. (1903), are equally convincing and instructive as regards the causative influence of a contaminated water-supply.

(*b*) **Infected milk** frequently conveys the poison. It may become polluted by water which has been used either to wash the cans or for diluting purposes, or the bacilli may be transferred to milk by the unclean hands of the milker. Numerous instructive epidemics, originating in infected milk, have been reported. E. B. Bigelow has reported a milk-borne epidemic which was traced to one male bacillus-carrier, and involving 204 cases. The occurrence of numerous cases among children suggests contaminated milk.

Solid forms of food (salads, celery, fruits) may be contaminated by infected water or dust or by the fingers of the nurse or the patient. During the late Spanish-American war the typhoid bacilli may have been conveyed from the latrines directly to the victims or to the kitchens and mess-tables by swarms of flies. Vaughan[1] confirms this view, and has also observed that "officers whose mess-tents were protected by means of screens suffered proportionately less from typhoid fever than did those whose tents were not so protected." He believes that fecal matter containing the typhoid germ may adhere to the fly, and be mechanically transported, and further suggests the possibility of the bacilli being carried in the digestive organs of the fly, and deposited with its excrement. H. W. Conn has shown that oysters while being fattened or freshened may become infected with water polluted by sewage, and Foote has shown that the typhoid bacillus will not only retain its vitality in the salt water in which the oysters are fed, but that it will live even longer in the oyster itself. Phillip Marvel has reported a small epidemic (comprising a total of 72 cases) due to infected oysters that occurred at Atlantic City during the months of August, September, and October of 1902. Newsholme[2] attributed one-third of a total of 56 cases of typhoid to the eating of raw shell-fish.

(*c*) **Contagion or Direct Transmission.**—This necessitates direct contact with the typhoid stools. It affords a ready explanation for contraction of the disease by internes and nurses who attend to the stools, the bed- and the body-linen of the patient, and by laundresses, who are affected with great relative frequency. Out of 1500 cases treated in the Johns Hopkins Hospital, 31 were contracted in this manner (Futcher).

(*d*) "**Ground-soil.**"—The typhoid poison which leaves the body must undergo development in the ground-soil before it is potent to cause the disease in others (Pettenkofer). The former great prevalence of typhoid in Munich was due to pollution of the soil (Childs).

(*e*) **Sewer-gas.**—The recent researches of Bergey and of Abbott show that sewer-gas, *per se*, cannot cause typhoid fever.

(*f*) **Sand-storms.**—Tooth states that sand-storms may contaminate articles of food with the bacillus.

(*g*) The hands of chronic carriers may be the medium of transference. Courmont[3] claims that dogs are typhoid bacilli carriers.

(2) **Through what channel or channels does the bacillus enter?**

(*a*) **In the vast majority of the cases the bacilli are swallowed.** In the stomach they meet with the acid gastric secretions, which often destroy them. The alkaline juices of the small intestine, however, furnish every condition necessary for their further growth and development. They

[1] *Phila. Med. Jour.*, June 9, 1900. [2] *Brit. Med. Jour.*, June 8, 1895.
[3] *Bulletin de l'Académie de Medecine*, Paris, June 28, 1910.

penetrate the mucosa and attack primarily the solitary follicles and Peyer's plaques. Next they invade the mesenteric glands, reaching the circulation, spleen, liver, and other organs a little later.

(*b*) The possibility that the bacilli may reach the blood-stream through the *respiratory organs* must be conceded; and hence the added possibility that they may set up initiatory lesions either in the tonsils, lungs, or pleura, passing thence into the circulation, must also be granted. Vaughan inclines to the opinion that the bacillus may be inhaled in the infected dust by troops on the march. Complete desiccation, however, soon destroys the typhoid germ. Primary localization of great severity may also occur in the kidneys and cerebrospinal meninges, giving rise to special clinical varieties (*vide infra*).

(*c*) **Typhoid Septicemia.**—By this is meant a general infection with the bacilli without localized lesions. The special mode of infection is not clear. Brion and Kayser[1] conclude from extensive bacteriologic and clinical studies that typhoid fever may start as a lymph-and-blood affection (possibly entering by way of the tonsils).

(*d*) Typhoid infection predisposes the system to secondary infections with various bacilli (streptococcus, staphylococcus, bacillus coli commune, pneumococcus). The portals of entrance for these micro-organisms are various (*e. g.*, respiratory tract, lymphatics).

Clinical History.—**I. Incubation.**—The average duration of the period of incubation, or the time between the introduction of the poison into the system and the appearance of the first active symptoms, ranges from ten days to three weeks; this interval may rarely be shorter, although oftener it is somewhat longer. During this period the patient may experience no deviation from health, but in most cases there are prodromal symptoms, such as languor, loss of appetite, nausea, headache, neuro-muscular pains in the back and limbs, a disinclination to exercise, etc. These symptoms last from a few days to a week or more.

II. General Symptomatology and Course.—On account of the peculiar temperature-curve in typhoid fever its course falls naturally into three periods—the stage of development; the acme or fastigium (corresponding to the height of the disease); and the stage of decline or defervescence. It is convenient to speak of the various weeks of the affection when referring to these stages. Thus, the first week represents the stage of development (*stadium incrementi*), the second and third weeks (in cases of average severity) the fastigium, while the fourth week in the typical form (the third week in mild cases) corresponds to the third stage (*stadium decrementi*) of the disease.

(*a*) **Stage of Development.**—The invasion, as a rule, is gradual, the symptoms being chilliness and feverishness, with increase in the severity of the prodromal symptoms. Typhoid fever rarely starts in with a distinct *rigor*. At or about this time nose-bleed may betray the nature of the disease. The symptoms just described are quickly followed by a prostration sufficiently well marked to compel most patients to take to their beds. From this latter event is usually dated the *onset* of the affection. It is safer, however, to regard the time of occurrence of the above-mentioned symptoms (elevation of temperature, with its attendant discomforts) as the time of onset, since many patients continue in their avocations for days after the first symptoms appear. The *onset* may be

[1] *Deutsches Archiv f. klin. Medicin*, last indexed, vol. xlv., p. 1832.

marked by symptoms resembling influenza (Bunce). In my experience cases in which general pains, including backache or slight pharyngitis, are seen at the onset are not rare. Again, invasion may be ushered in by various nervous symptoms (*e. g.*, convulsions, in children) or marked pulmonary features, especially those of severe bronchitis.

With the progress of the initial period the symptoms usually increase in severity; the *fever* rises day by day, terrace-like, till, at the end of four or five days, the second stage, or fastigium, is reached. Anorexia, thirst, and headache are often marked, the skin hot and dry to the feel, the tongue coated, the sleep disturbed, and constipation is generally present. The patient may complain of sensations of chilliness alternating with flushings of heat, and there is a slight cough. The *pulse* is somewhat quickened (from 90 to 110 per minute) and is full.

The *physical signs* are not prominent. The abdomen is often slightly distended and tender; the spleen is found to be swollen. The association of splenic enlargement and dry bronchitis point to this disease.

(*b*) **Fastigium,** or the **second stage,** commences on the fourth or fifth day, and lasts, in typical cases, about two weeks. During the first week of the fastigium (the second of the disease) the general symptoms become more marked. The *fever* remains high, the evening temperature usually reaching 103° or 104° F. (40.° C.), and exhibits the continued type. The *pulse* is accelerated, but not dicrotic. The headache disappears, and mental dulness and slowness are conspicuous, but there may be mild delirium, particularly at night. There is a dry *cough* and the physical signs indicate more or less extensive bronchitis. The *tongue* is coated and may become dry, the belly is somewhat swollen and tender, and diarrhea replaces constipation. The *spleen* is decidedly enlarged, and about the eighth day of the disease a number of roseate spots appear on the trunk. During the latter part of this week a grave or even fatal condition may be developed as a result of intense nervous or pulmonary symptoms, intestinal hemorrhage, or perforation.

During the second week of the fastigium (the third week of the disease) the marked general symptoms already noted persist in severe types of the affection. The *pulse* varies from 110 to 130, and is now often dicrotic, while the temperature may approach the remittent type. In addition this period furnishes most of the untoward *complications* (lobular pneumonia, hypostatic congestion of the lungs, intestinal hemorrhage, perforation, peritonitis), and in the absence of serious local complications grave general conditions may be presented. The duration of this stage varies with the severity of the type.

(*c*) **Stage of Decline or Defervescence.**—At the end of the second stage, and about the twenty-first day of the disease, in favorable cases the *fever* begins to decline, and with it the other general and local symptoms gradually disappear. This is followed by true convalescence. In protracted cases, however, the fourth week of the disease may present much the same clinical indications as the third, and these may even be intensified. Frequently an aggravated type of the *typhoid state* is now superadded, the symptoms being stupor, muttering delirium, subsultus tendinum, a rapid, feeble pulse, a dry, brown tongue, marked diarrhea, greatly swollen belly, and an involuntary discharge of feces and urine. Inflammatory complications may add to the perils of the condition.

In not a few cases the febrile period is prolonged into the fifth, and

rarely into the sixth or even the seventh week, and the fever observed when defervescence is retarded presents an irregular type. I have elsewhere reported a case in which it lasted not less than seven weeks.[1] About this time recrudescences and relapses may occur in typical cases. Different epidemics of typhoid fever, however, vary so greatly in their clinical characteristics as to make it impossible to include all cases in any outline of the course of the disease that might be attempted.

III. **Chief Clinical Features in Detail.**—(*a*) **Course of the Fever.**—During the stage of development (the first four or five days) the temperature usually rises in "step-ladder" fashion. The evening exacerbation is on each day from a degree and a half to two degrees higher than on the preceding, and the same is true of the morning remissions. A glance at the temperature-charts (Figs. 2 and 3) will show that the morning remissions touch a level from one-half to one degree lower than the preceding evening registers. This stage is rarely met.

When the fastigium is reached, the evening temperature may be 103°, 104°, or 105° F. (39.4°–40.5° C.), and is usually thus maintained, with the slight morning remissions. The tide-like character of fever-curve seen in the initial period is absent. Often, during the latter half of the fastigium (the third or fourth week of the disease) the morning fall of temperature becomes decidedly greater. According to my own observation, the height of the fastigium is reached a day or two after its onset or at the end of the first week of the affection. Marked morning remissions are a favorable indication. On the other hand, and contrary to the general rule, the morning temperature may be higher than the evening, forming a somewhat unfavorable symptom. Morning temperatures of 104° F. (40° C.) or over are indicative of a serious type. In many instances of mild grade the evening temperature at no time exceeds 103° (39.4° C.), but oscillates between 100$\frac{3}{5}$° and 102$\frac{3}{5}$° F. (38.1°–39.2° C.). In cases of average intensity the morning remissions touch 102°–102$\frac{3}{5}$° F. (39.2° C.), and the evening exacerbations reach 104–104$\frac{3}{5}$° F. (40.3° C.). When the temperature rises above 105° F. (40.5° C.) hyperpyrexia exists. Ampugnani made studies of hourly charts from 200 cases of typhoid fever, and found the maximum temperature to occur between three and six o'clock in the afternoon, and the minimum between four and eight o'clock in the morning. The duration of the fastigium exhibits a wide range and is dependent upon a variety of conditions—*e. g.* the degree of mildness or severity of the type, the presence or absence of complications, etc. In cases of a mild character it lasts from a few days to one week; in cases of average severity, from ten days to two weeks; in the severest forms, from two to four weeks.

In typical cases the end of the fastigium marks the beginning of the last stage (that of defervescence), and during this period the temperature falls by *lysis*. Measured by days, it declines by degrees, both the morning and evening temperatures being often one or two degrees lower than on the preceding day. Thus is formed a more or less regular step-like line of descent. To this general rule there are two notable exceptions: From the beginning of the period of defervescence the morning remissions may strike the normal point, while the evening ex-

[1] "A Case of Typhoid Fever; numerous Intestinal Hemorrhages, the Amount of Blood Lost being Seventy-eight and one-half Ounces; and Obstinate Vomiting, with Recovery," *International Clinics*, vol. i. 5th series, April, 1895, p. 29.

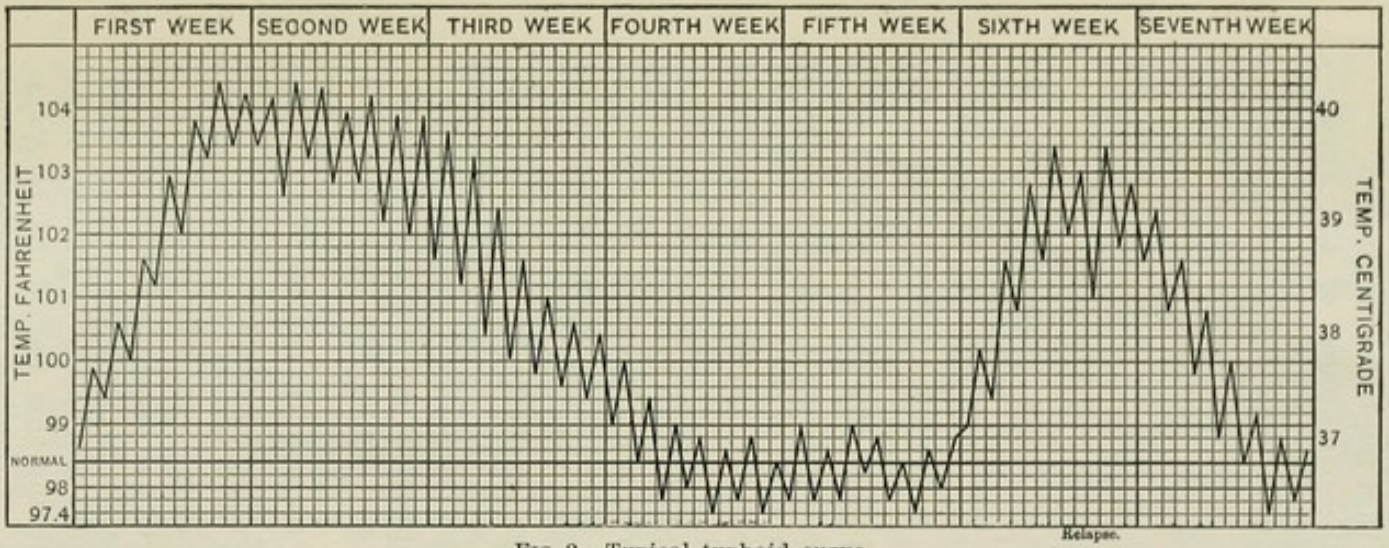


FIG. 2.—Typical typhoid curve.

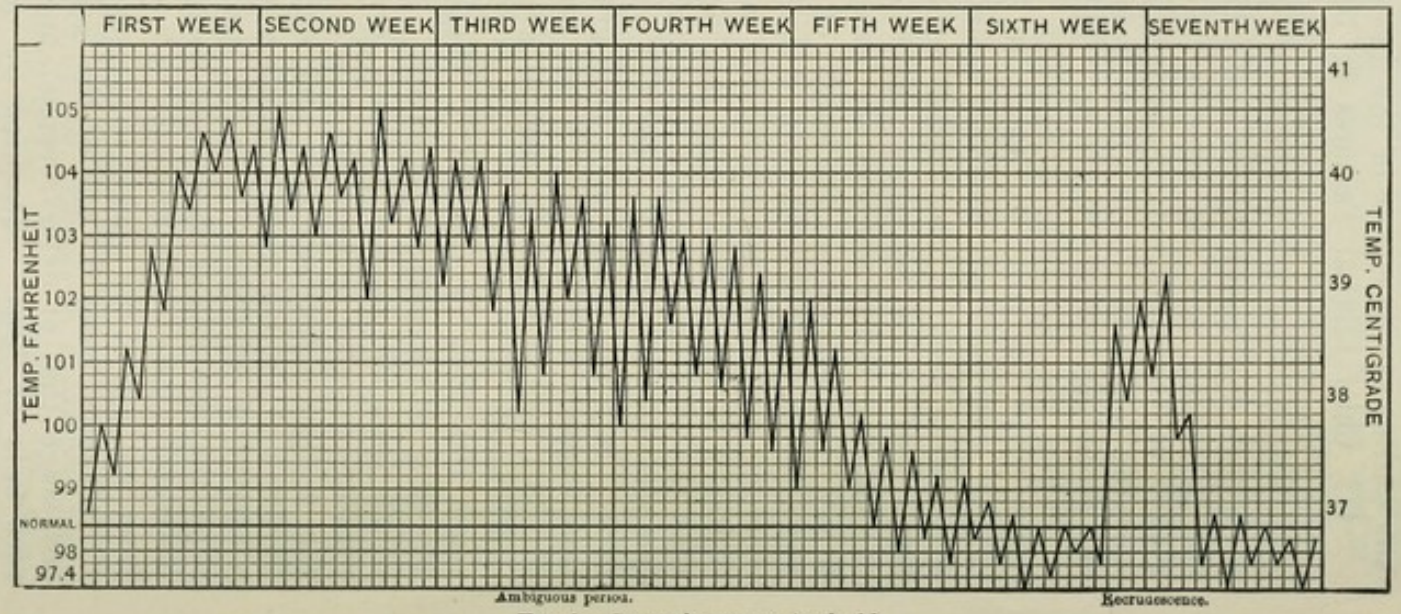


FIG. 3.—Curve in severe typhoid.

acerbations become less and less marked, until they also touch the normal. Under these circumstances the temperature-curve resembles somewhat that of the quotidian intermittents, and rarely the tertian fever-curve obtains. In comparatively rare instances the morning temperature shows a deeper remission on each successive day, while the evening temperature remains high for several days, when it also declines. This period lasts from one week to ten days—a longer time than the initial stage with its ascending type of fever.

In the severe and protracted forms of typhoid fever there occurs between the second stage (fastigium) and the third stage (defervescence) another, to which Wunderlich has given the name of the "ambiguous period." This lasts from a few days to a week or more, and is characterized by a striking diurnal range of temperature, with marked irregularities. It is probable that it is sometimes produced by an auto-intoxication.

Abnormal Course of the Fever.—The pyrexial peculiarities yet to be pointed out are less usual than the foregoing, although of sufficient frequency of occurrence to demand a brief description.

The first stage varies but little from the regular course described above. A sudden elevation of temperature, however, is seen in those cases that begin with a severe rigor, accompanied by pneumonic, catarrhal, and gastro-intestinal symptoms. Pepper and Stengel have reported seven cases with acute onset, and Moore, of Dublin, states that the whole course (since 1889) has become more typhus-like than formerly.

In the lightest forms the *fastigium* may be practically absent, defervescence setting in upon the first day of the fastigium. There is also a class of cases in which, throughout the greater part of their course, the fever is distinctly intermittent or remittent, and in which careful blood-examination fails to disclose the *plasmodium malariæ*. The same form of temperature-curve is seen in those rare instances of typhoid fever which occur in subjects previously infected with malaria. These two classes of cases run a favorable course as a rule.

Sudden deep temporary drops in the temperature may occur during the fastigium. (1) This may take place during the early part of the fastigium without obvious cause. (2) Intestinal hemorrhage almost invariably produces a sudden, and sometimes a great, fall of temperature. Osler has reported a case in which a drop of 10° F. (5.5° C.) followed melena. The blood does not appear in the evacuations of the patient for six to twelve hours or more after the temperature has begun to fall; and hence a critical decline of temperature during the latter part of the second and the third week of the disease suggests that hemorrhage has probably taken place. (3) The occurrence of *peritonitis* is marked by a sudden and considerable fall of temperature. (4) In the female, abortion or premature delivery occurring in the course of typhoid fever produces a decided lowering of the temperature. (5) Collapse of the circulation sometimes occurs with a notable remission of temperature—an ominous association of events, and one which I observed in two cases occurring in females in the Medico-Chirurgical Hospital. In one of these cases two such periods of collapse occurred, and in the other three, though both finally recovered under prompt and continuous stimulation. Occasionally *hyperpyrexia* is observed in typhoid

fever, and most frequently just before dissolution, when the thermometer may register 108° or even 109° F. (42.7° C). A fresh rise with marked irregularity of temperature may occur during the latter part of the fastigium or the period of decline, and is often dependent upon some local complication (late pneumonia, parotitis, etc.).

The stage of defervescence is sometimes much prolonged, though most frequently there is simply a slight evening elevation (99° to 100° F.—37.7° C.), the morning temperature being normal. The causes of retarded decline are, for the most part, obscure. I believe that many of them are ascribable to a mild grade of auto-intoxication, and in my hands a mild saline laxative has been the means of cutting them short in a number of instances. An examination should, however, be made for some localized inflammatory complication, though this is not always discernible, as in the case of suppuration in the mesenteric glands. Sluggish typhoid ulcers, which refuse to heal promptly, may act as a cause of the slow decline; they are often due to the post-typhoid anemia and exhaustion.

Post-typhoid Elevations of Temperature.—After both the evening and morning temperatures have become normal, fresh temporary elevations (102° or 103° F.—38.8° or 39.4° C.) frequently appear. They are, as a rule, unassociated with any other symptoms, and at the end of a few days the temperature falls rapidly to the normal. These are termed *recrudescences*, and are to be distinguished from true typhoid relapses. They are probably produced in various ways—by errors in diet, constipation, mental emotion, excitement. "There are cases in which the presence of the fever seems to be really a nervous phenomenon" (Osler). It is most common in children and in persons of a decidedly nervous temperament. Certain local sequelæ may cause post-typhoid fever, such as abscess and periostitis. Rarely during convalescence a sudden and marked elevation of temperature, accompanied or not by rigor, occurs, but it is usually of short duration and seldom is of serious import. I saw, with the attending physician, Dr. Modell, a case in which the temperature had been normal for six days, when rigors, followed by steep elevations of temperature, occurred several times and at intervals of thirty-six or forty-eight hours. These high temperatures were followed by a rapid decline to the normal and by sweating, leaving the patient profoundly exhausted. Subsequently the convalescence was slow, but uninterrupted.

Afebrile Typhoid.—As the term indicates, typhoid fever may run a course attended with all of the characteristic symptoms save only the fever. Cases of this kind are of great rarity.

(*b*) **Skin.**—The *eruption* is highly characteristic, and usually decides the diagnosis. It makes its appearance on or about the eighth day, and sometimes a little later. Occasionally it does not appear until the tenth or twelfth day of the disease. It consists of distinct, rose-colored, and slightly elevated papules, having a rounded or lenticular form and a diameter varying from one or two to three lines. The papules are almost invariably found upon the trunk, and especially upon the upper part of the abdomen and the lower part of the thorax, to which regions they may be wholly confined. They may, however, be absent from the usual seats and present elsewhere, so that the sides of the trunk, the

back, and the thighs should always be inspected. They disappear upon pressure, but reappear when the pressure is removed. These rose-colored spots last three or four days, and appear in successive crops, each one being made up, usually, of a few spots—a half-dozen to a dozen. Rarely the eruption is abundant on the trunk, even extending to the extremities and head; but there is no direct correspondence between the extent of the eruption and the severity of the cases. Occasionally the spots are entirely absent—a condition most frequently met with in children, and less often in elderly persons.

Other eruptions are often present, and their negative diagnostic value must be kept in remembrance. Minute pearly vesicles (*sudamina*) may appear. They are limited to the abdomen, the axilla, and to the inner surface of the thighs as a rule, and are in great measure due to profuse sweating.

A scarlet-colored *erythematous eruption* sometimes appears at a comparatively early period in typhoid fever. *Urticaria* and *purpura* are rarely seen. Out of 250 cases of typhoid fever among the soldiers in the Spanish-American war treated in the Medico-Chirurgical Hospital two manifested purpuric spots. *Extensive ecchymoses* may occur, but are rare, and merely symptomatic of the hemorrhagic diathesis. Cutaneous *boils* and *abscesses* due to secondary infection with the pyogenic cocci are a comparatively frequent and late development in the course of the disease. *Peliomata typhosa* in the form of little bluish subcuticular spots (the "tâches bleuâtres" of the French writers) may appear; but they are not related specially to typhoid fever, and in a recent case of my own were undoubtedly due to pediculi. *Gangrene*, chiefly of the lower extremities, has been noted in 214 cases (Keen), and is due to the diffusion of the bacilli and their toxic products, to an obliterating endarteritis, thrombosis, or embolism.

Profuse sweats form a conspicuous symptom in many epidemics of the disease, with or without accompanying fits of chilliness or rigors, and constitute the sudoral form of typhoid fever (Jaccoud). Some of these cases resemble ordinary intermittents. *Edema* of the skin may be observed and is due most frequently to anemia or cachexia, though sometimes to nephritis. A local form of edema affecting the leg is not uncommon, and for this form thrombosis of the femoral vein is chiefly responsible. A peculiar "musty" odor is exhaled from the skin in some instances of typhoid fever. The patient assumes the dorsal decubitus and is exposed, particularly in cases of prolonged duration, to the danger of the formation of *bedsores*. They are most prone to occur on the nates and the heels, and, once started, they are apt to spread till they attain to large dimensions, with extensive undermining of the skin. The condition is now serious. During and after the conclusion of convalescence the hair falls out, but, fortunately, it is invariably renewed. The *nails* sometimes become roughened and brittle, while transverse pale lines or ridges can usually be observed in them, marking the impairment of nutrition during the disease (*vide* Relapse). *Jaundice*, due to a variety of causes, is a rare symptom, and generally does not come on until the middle of, or until late in, the disease (Da Costa).

(*c*) **Digestive System.**—The symptoms referable to the gastro-intestinal canal, though not very striking in most cases, are of the utmost

importance and interest because of their direct connection with the pathognomonic lesions of typhoid. Beginning with the intestinal canal, and thence proceeding to the symptoms presented by the stomach, spleen, liver, throat, and mouth, will be a natural and convenient order.

At the onset of typhoid fever *constipation* is the general rule, and this may persist to the end of the illness, though more commonly a moderate diarrhea appears. Osler[1] in the Johns Hopkins Hospital, however, met an initial diarrhea in 322 out of 829 cases. During the second week of the affection the stools number, on the average, from two to four or more daily. It is only in comparatively rare instances that ten or more movements per diem occur, and the severity of the diarrhea depends largely upon the degree of catarrh, particularly of the large intestine. When, however, the ulcerative process is chiefly limited to the colon, it is an important factor in the production of the diarrhea. Indeed, in those instances—not altogether rare—in which there is urgent diarrhea of a *dysenteric character*, the ulcers are especially marked in the colon, with diphtheritic inflammation of the surrounding mucosa. Involuntary discharge of the feces may occur.

The *stools* present a characteristic yellow appearance, suggesting by their color and consistence a comparison with pea soup. They are usually either fluid or of the consistence of jelly, and are offensive and of an alkaline reaction. On standing they separate into two layers—an upper, liquid, cloudy layer, and a lower, thick yellow, sedimentary layer, in which, on macroscopic examination, remnants of food and grayish yellow fragments (necrotic crusts of Peyer's plaques) from a half to an inch in length may be detected. Microscopically, they have been found to contain undigested particles of food, epithelial débris, blood-corpuscles, crystals of triple phosphates in abundance, and innumerable bacteria. Laboratory experimentalists are able to demonstrate the presence of the typhoid bacillus in the dejecta. *Tympanites*, mainly affecting the colon, is a common though rarely a striking feature, and cases of a quite serious nature are observed in which the abdomen presents a concavity throughout the entire illness. The latter is less unfavorable, by far, as a symptom than excessive tympanites, which interferes with both the respiration and heart action. Tympanites is apt to be most marked in serious cases which have diarrhea as a prominent symptom, though the latter may not even be present. It is due to the generation of gas from decomposing food, and to the arrest of peristaltic movements in consequence of degeneration of the muscularis of the intestines. *Pain* is absent in the majority of cases, and when present is not intense, save in rare instances. Pressure upon the ileocecal region usually causes a *gurgling noise*, but, although this symptom is commonly present, it is not characteristic of the disease. There is generally also a slight degree of tenderness of the abdomen under pressure, most marked in the right iliac fossa, and hence, in all probability, due to the presence of ulcers in this region. Absence of tenderness, however, is not a safe indication of the absence of extensive ulceration. *Extreme sensitiveness* generally denotes peritonitis (often without perforation), although the symptom may be marked in constipation.

Intestinal hemorrhage occurs in from 4 to 7 per cent. of cases, its

[1] *Philada. Med. Journ.*, October 15, 1900.

frequency varying with different epidemics. The hemorrhages appear almost invariably during the latter part of the second and third week, being caused by the opening of blood-vessels during the necrotic or ulcerative process. The amount may be small, or it may be from 1 to 2 or 3 pints (0.5–1.5 liters), or even more. In one of my own cases the total amount of blood discharged from the bowel was nearly 5 pints (2.5 liters), and yet the patient recovered. The blood presents a dark hue, and that which is passed last may be tarry. Roman has examined the feces in 50 cases of typhoid fever with a view to the detection of occult bleeding. Blood was found in 14 cases, and of these, 7 were severe, 3 moderately severe, and 4 slight.

The significance of intestinal hemorrhage is always grave. On the other hand, recovery is possible even if the hemorrhage be copious and oft-repeated; and in general terms it may be said that death supervenes in from 30 to 40 per cent. of all cases. R. G. Curtin has recorded 60 cases, of which 46.6 per cent. died; he argues that cold applications to the skin and the necessary disturbance in giving a cold bath tend to produce melena. It occurred in more than the usual proportion of cases under my care during the Spanish-American War, probably owing to the fact that the men were conveyed from the various distant camps to the hospital. A fatal result may occur as the direct effect of a profuse hemorrhage. When death does not follow immediately, the signs of collapse and of anemia appear; yet intestinal hemorrhage sometimes exerts a favorable influence, stupor and delirium quickly giving place to consciousness. When typhoid fever occurs in the hemorrhagic diathesis, hemorrhage occurs from various outlets.

Perforation, which almost invariably produces fatal diffuse peritonitis, is the accident most to be dreaded. It does not bear a fixed relation to the severity of the affection. According to Fitz, who tabulated 4680 cases of typhoid fever, there is a mortality of 6.58 per cent. from perforation of the bowel. Scott's statistics, embracing 9713 cases from English, Canadian, and American hospitals, give a mortality of 3.6 per cent. from perforation. It is much more common in males than in females, and appears in a ratio of about 71 to 29. *Age* has a decided influence, the complication occurring oftenest between ten and forty years of age, while in children it is rare. It may occur at any time in the course of typhoid fever, but it is most common between the second and fourth weeks of the disease. In the cases analyzed by Fitz perforation was found in the ileum in 81.4 per cent., in the large intestine in 12.9 per cent., in the vermiform appendix in 2.5 per cent., and in the jejunum in 1.29 per cent. The accession of hypertension of the pulse is indicative of approaching perforation. The accident is usually announced by the sudden advent of acute *pain* in the abdomen, quickly followed by the symptoms of *collapse;* and the fact that diffuse peritonitis, following perforation, may develop insidiously must be recollected. The abdominal muscles become rigid, sensitive to touch, and later tympanites develops. Fluctuation can sometimes be elicited. On *percussion* splenic and hepatic dulness is often absent, but hepatic dulness is also wanting when the distended intestines lie in front of the liver. Shifting dulness in the right flank may be an early sign. The collapse of the circulatory system is evidenced by the pinched features,

hollow cheeks, vomiting, and the small, frequent pulse. A rising leukocytosis is a valuable diagnostic symptom. Crile found a rise in the blood-pressure in cases. Wilson has emphasized the importance of an early diagnosis and of immediate resort to operative intervention.

The instances that develop independently of actual perforation usually assume the local or circumscribed form of peritonitis; they are occasioned in various ways—*e. g.*, by direct extension of the inflammatory process from the intestinal ulcers, primary localization of the virus in the peritoneum, and rupture of the mesenteric glands. The condition presents corresponding areas of tenderness under gentle, and especially under prolonged, pressure, but it is difficult to diagnose intra-intestinal states. Generalized peritonitis may succeed the circumscribed variety. A. McPhedran[1] calls attention to a serous peritoneal effusion (ascites) and to the physical signs of exudate in the peritoneal cavity.

The *mesenteric lymph-glands* may soften or suppurate (*vide* Pathology), and, as before mentioned, may be the exciting cause of a recrudescence, or they may rupture and cause diffuse peritonitis.

The Spleen.—With few exceptions the spleen is enlarged in typhoid fever, the edge usually being palpable below the margin of the ribs, on or before the commencement of the fastigium. It generally goes on increasing in size till near the beginning of the third week, and lessens during the latter part of the third and fourth weeks. Swelling of the spleen is sometimes absent after a copious intestinal hemorrhage and in elderly typhoid subjects. When the tympanites is excessive, we can in most cases satisfy ourselves of its existence or non-existence by careful palpation. Suppurative infarcts or softening of the spleen may start a peritonitis. Rarely spontaneous *rupture* of the organ may occur, which is manifested by intense pain in the splenic region. Bryan reports a case and has collected 24 others from the literature.

The Liver.—A slight swelling of the *liver* can sometimes be detected. Among the least frequent complications is *jaundice* (*vide supra*); it may be due to cholangitis, to abscess, and to gall-stones. *Cholecystitis* caused by the typhoid bacillus (*vide* Pathology) may arise during the attack or long after complete recovery. In most cases the lesions are catarrhal, but they may be suppurative, in which event perforation followed by peritonitis may occur. *Calculous cholecystitis* is frequently caused by the typhoid bacilli, but may not manifest itself for a variable number of years. *Suppurative pylephlebitis*, secondary to perforative appendicitis, may be a complication. Multiple abscess may occur.

The Stomach.—The stomach presents no characteristic symptoms. Of the *anorexia* enough has been said, but during convalescence the appetite returns, becoming even voracious. *Nausea* and *vomiting* may occur during any stage of the disease, but are most common at the beginning. When they appear as late symptoms they are probably excited either by a typhoid ulcer or by peritonitis. Nausea is often traceable to other causes—*e. g.*, to errors in diet, or to the use of irritating medicaments, but vomiting also occurs from unknown and inevitable causes. It may become a grave symptom. Hiccough is a rare but serious symptom. Hematemesis has been observed, although practically unknown.

The Pharynx.—The pharynx frequently shows *catarrhal irritation*,

[1] *Cleveland Med. Jour.*, June, 1911.

and the patient may complain of dryness or a burning sensation in the throat. Actual *sore throat* may be present at the time of onset, associated with a diffuse *erythematous rash*, suggesting scarlatina.

The Tonsils.—There is a special form of typhoid—*tonsillo-typhoid* or *pharyngo-typhoid*—in which there appear upon the tonsils peculiar patchy elevations, whitish in color, which undergo subsequent ulceration. It is not improbable that these lesions result from the local action of the specific bacillus in an unusual situation. *Thrush*, affecting the mouth, throat, and even extending to the esophagus, not infrequently arises as a complication. The *tongue* is heavily coated, as a rule, with a yellowish-white fur; later it clears off near the edges and tip, while the center becomes dry or brown and sometimes fissured. The lips are also dry, sometimes fissured, and often covered with dry, black crusts (sordes). *Ulcerative stomatitis* may occur if the mouth be not kept clean. Under these circumstances secondary lesions evincing unpleasant and even serious symptoms may also arise in organs more or less remote from the mouth, and among these is *parotitis*, which is most probably caused by the staphylococcus or streptococcus reaching the parotid gland by way of Steno's duct. The condition is betrayed by such symptoms as pain, redness, and finally by fluctuation, with an elevation of the bodily temperature. It is a late-appearing development, and is usually unilateral, though it may be bilateral. *Suppurative otitis media*, a rarer complication, arises in a similar manner, the pathogenetic agents reaching the ear through the Eustachian tube.

(*d*) **Respiratory System.**—As pointed out in the section on Pathology, *bronchitis* is almost invariably present, but in the majority of instances the cough is slight. The condition is recognized by the existence of numerous sibilant râles. Very rarely is it a striking feature in the early stage of typhoid fever, and then, except this fact be remembered, room for error of diagnosis exists. Moreover, in cases that are improperly treated the bronchial secretions are apt to accumulate, and a well-marked bronchitis may be the result. It may be said, however, that, as a rule, bronchitis does not assume a severe type in cases which receive proper attention from the beginning, provided the patient be not unusually stupid or unconscious. When the nervous phenomena are pronounced, and the patient maintains the dorsal decubitus, bronchitis of a severe grade and affecting the smaller bronchi is almost inevitable. The occurrence of an intense generalized bronchitis is also favored by conditions such as corpulence, advanced age, and emphysema. These cases are apt to lead to lobular infiltration—*aspiration-pneumonia*.

Lobular pneumonia may take on a putrid nature and the consolidated area may become *gangrenous*. As a sequel, *pleurisy* with effusion or *empyema* may originate in consequence of the infiltrated lobules being contiguous to the pleura. If the lobules occupying the periphery of the lung become gangrenous, perforation of the pleura, leading to *pyopneumothorax*, may result. As pointed out by Gordinier and Lartigau,[1] in the majority of instances of typhoid pleurisies the aspirated fluid has been found to be purulent in character. Lobular pneumonia may be attended with *hurried breathing* or troublesome *cough*.

[1] *Amer. Journ. Med. Sci.*, January, 1901.

More commonly, the *local symptoms* are in abeyance, and this is especially true of the severer cases which are attended with profound nervous prostration and more or less unconsciousness. Sole reliance is to be placed upon the results of a *physical examination*, which should be repeated daily. Points or surfaces of dulness, near the bases of the lungs, are found on percussion. Fine moist râles, heard in every direction, and especially marked toward the bottom of the thorax, form a characteristic sign. A certain diagnosis of lobular pneumonia demands the combined presence of both the circumscribed dulness and moist râles.

Lobar pneumonia is a not uncommon complication. In a small percentage of cases it develops early, and is most probably the result of a special concentration of the poison in the lungs, giving rise to the so-called *pneumo-typhoid fever* (*vide infra*, Varieties). These cases are often mistaken for primary lobar pneumonia. Their onset may or may not be marked by a rigor, but it is usually more gradual than that of primary lobar pneumonia. At the end of the first week or thereabouts the pulmonary symptoms gradually abate, while those characteristic of typhoid (enlarged spleen, roseate spots, etc.) come to the fore. Wagner, Leichtenstein, and Aufrecht entertain grave doubts as to the existence of a pneumo-typhoid. I have, however, had under my care a case in the Medico-Chirurgical Hospital that was proven by the Widal reaction. Lobar pneumonia more often develops as a late complication—in the second or third week, or even during convalescence—but it is not attended by the usual phenomena (rigor, cough, rusty expectoration, intense chest-pain), and hence may be easily overlooked. The temperature may be either quite elevated or at times only moderately so. Lobar pneumonia, the complication, is principally due to the pneumococcus. The *diagnosis* is to be made from the physical signs, together with the peculiar temperature-curve, which may present marked irregularities. Pulmonary infarction and abscess of the lungs are occasional complications.

Hypostatic congestion of the lungs, due to enfeeblement of the cardio-pulmonary circulation, is a frequent concomitant, appearing in the third week of the disease. It is generally bilateral, and is promoted by the effects of gravitation. It is almost always associated with more or less edema of the lungs. The subjective symptoms, including fever, are usually negative, while the objective signs are those of partial or complete consolidation of the bases (defective resonance or dulness, broncho-vesicular breathing, with moist râles). *Miliary tuberculosis* rarely develops as either a complicating affection or, it may be, as a sequel. Of 249 autopsies in fatal cases of typhoid fever only four showed acute tuberculosis to have been associated.[1]

Laryngitis, indicated by hoarseness, is an occasional complication. The laryngeal ulcers may extend in depth to the perichondrium, and promote that grave condition, *perichondritis*, leading to necrosis of the cartilages with edema of the glottis and stenosis. A third form of laryngeal complication is that in which the muscles are deprived of their function because of paralysis (Gibb).

Epistaxis appears early in some cases and is a valuable diagnostic symptom. It may also occur during the fastigium, particularly toward

[1] "The Relation of Typhoid Fever to Acute Tuberculosis," *Amer. Jour. Med. Sciences*, May 4, 1904, by the writer.

its close, when it is of no diagnostic, but of grave prognostic, significance. In a case I saw with the late Dr. Snively it led to a fatal issue.

(*e*) **The circulatory system** presents no characteristic symptoms. The *heart-sounds* are but little affected, as a rule. In cases of asthenic type and in severe typical instances the first sound of the heart may grow quite feeble and ultimately resemble the second (embryocardia). Under these circumstances a soft systolic murmur may be faintly heard along the left border of the sternum. Among occasional *complications* presented by the heart is pericarditis, and still less frequent is endocarditis. Myocarditis is more common. The sudden development of circulatory collapse in the course of typhoid fever, as previously noted, may be due chiefly to myocardial inflammation; and there may be a brief though alarming derangement of the heart action, due to functional disturbances.

The *pulse* is accelerated, but not, as a general rule, in proportion to the height of the temperature until late in the affection. Its average rate is from 84 to 108. The temperature, moreover, may be of average height, while the pulse is normal or only slightly quickened throughout; and hence the increase in the pulse-rate cannot be due solely to the elevation of temperature. As before intimated, the extreme debility which comes on during the third week in severe cases may have, as one of its manifestations, a very rapid pulse, reaching to 160 or more (the so-called running pulse), and with or without marked irregularity. Slight *irregularity* is sometimes observed, either during the height or decline of the affection, but as a rule proves of no serious consequence. Marked temporary accelerations are often caused by undue exertion or mental excitement. The lowered arterial tension is shown by a dicrotism of the pulse—a non-characteristic symptom, however, since it is well marked in other acute infectious diseases, although less commonly. During convalescence the pulse often becomes abnormally slow (*brachycardia*). *Per contra*, less commonly, the pulse-rate is increased during convalescence. I have found the systolic arterial pressure during the fastigium to range from 110 to 125 mm. Hg. (Riva-Rocci instrument); it declines further late in the disease. The fall in the diastolic pressure is proportional. During convalescence the blood-pressure again rises, reaching the normal in from two to four weeks.

Venous thrombosis occurs in 1 per cent. of all cases (Murchison). Its most frequent seat is the left femoral, and the next most frequent the right femoral vein, and it is the immediate result of cardiac weakness, except perhaps in those rare instances that arise early in typhoid. In most cases there is, doubtless, more or less phlebitis, and the bacilli have been found in the thrombus. The condition may be bilateral. Coming on, as it usually does, during convalescence, it manifests itself by swelling and edema of the extremity affected. There are *pain* in the thighs and calves, and tenderness over the course of the vein, and often over the calf of the leg as well. It causes *fever* of a moderate grade and irregular type. In the course of from two to three weeks the swollen member may be reduced to its normal dimensions. This complication is usually not serious, but occasionally clotting extends into the pelvic veins, and into the vena cava, thence even into the right auricle, inducing fatal syncope, and sudden death has resulted from the detachment of emboli.

The thrombus may undergo suppuration, leading to systemic septic infection.

Thrombosis and *embolism* in the arteries, with renal, splenic, and pulmonary infarcts, may be encountered in typhoid fever.

The large or small arteries may become obliterated, either by embolism or thrombosis, in extremely rare instances, but whether the thrombosis under these circumstances is brought about by a peculiar condition of the blood which favors clotting, or by a localized arteritis, is not definitely known. If, as is usual, the femoral artery be involved, the blood-supply to the foot and leg is cut off and *gangrene* of those parts must follow. The condition may be bilateral. It may be detected



FIG. 4.—Chart illustrating the blood-changes in typhoid fever: upper curve, red corpuscles; middle curve, hemoglobin; lower curve, white corpuscles.

early, owing to the absence of a femoral pulse, before the signs of gangrene appear. The condition is highly dangerous.

The *blood* presents certain changes, some of which are valuable for diagnostic purposes. The red corpuscles may be relatively increased in number during the febrile period, owing to loss of water (*e. g.*, profuse sweats, diarrhea). There is, in most instances, little or no decrease in the number of red corpuscles till the end of the second week. They are markedly diminished, as a rule, during convalescence, the oligocythemia bearing a close relation to the severity of the disease.

There is a greater relative decrease in the amount of hemoglobin than in the number of red corpuscles. The number of white corpuscles remains at or a little below the health standard until late convalescence, when it sinks to a moderate degree—furnishing a count of about 2000 per c.mm. (*leukopenia*). This fact is an important aid in the differentiation of uncomplicated typhoid fever from leukocytotic affections. Leuko-

cytosis, however, occurs in typhoid, with hemorrhage and perforation, and especially in connection with "large abscesses, phlebitis, peritonitis, pneumonia, pleurisy, periostitis, cystitis, and cholecystitis" (Thayer). Transient leukocytosis occurs after cold baths. Naegeli[1] found an early neutrophilic leukocytosis of moderate degree which rapidly decreases. In the second stage neutrophiles and lymphocytes are still further decreased, the former at last disappearing, while the latter begin to increase again, and so continue until defervescence. During the decline of the fever the neutrophiles reach their minimum, the lymphocytes are greatly increased, and the eosinophile cells gradually return to their normal number. After the disappearance of the fever a lymphocytosis may occur. The blood-characters in typhoid are shown in the accompanying chart (Fig. 4).

(*f*) **Nervous System.**—The persistent *headache* that is almost always present is among the most prominent symptoms during the first week, but it diminishes steadily during the early part of the second, as a rule. It affects the temporal, occipital, and cervical regions, and when the onset is comparatively sudden, pain in the back is also a more or less conspicuous feature during the first few days of the illness. In a small class of cases, however, the effects of the typhoid bacilli or their toxins are manifested solely in the nervous system from the very onset. In such there are violent headaches, retraction of the head, rigidity, photophobia, and muscular twitchings (rarely convulsions)—all of which symptoms indicate *meningitis*. The diagnosis of meningitis as a complication must be made with extreme caution, since, no matter how complete the clinical picture may be, the post-mortem examination usually reveals a total absence of meningeal inflammation. It must be recollected, however, that the lesions may be wholly microscopic. *Vertigo* may accompany the headache, but it seldom outlasts the latter. Before delirium manifests itself wakefulness and restlessness at night are very annoying, and later the same symptoms may be observed associated with the delirium. In cases of moderate severity *mental dulness*, and even *actual hebetude*, are almost invariably present. Questions are apt to be answered inconsistently and in monosyllables.

Delirium is frequent in the severer cases. It is, however, not an uncommon event for those of moderate severity to be free from this symptom throughout the attack. It is, as a rule, most marked at night or at some time when the patient is left alone. His delusions may impel him to attempt to leave his bed, but more commonly there is *mild* or *noisy delirium*, with more or less restlessness. He may lie somnolent, soliloquizing in a loud whisper (muttering delirium), and this so-called *typhomania* may gradually give place to actual coma toward the close of the middle period of the disease. In not a few cases —mild or severe—*coma* is developed suddenly, and is often a mortal symptom. Still another unfavorable sign is a picking at the bed-clothes or a grasping at imaginary objects (carphologia).

The delirium may assume an hysteric type, the patient usually exhibiting the saddest emotions, and if he be an alcoholic he may be seized with *delirium tremens*. In a case of typhoid fever that I saw with Dr. S. W. Morton hysteric delirium developed during early convalescence, but did not last more than twenty-four or thirty-six hours.

[1] *Deutsche Archiv für klin. Med.*, Band lxvii., Hefte 3 u. 4.

The *motor nerves* also present notable disturbances in association with the sopor and the forms of delirium previously described. Slight twitchings of the muscles of the face and extremities are quite common, and when they affect the tendons of the wrist and fingers the term *subsultus tendinum* is applied. The lips, tongue (especially when protruded), lower jaw, and even the extremities, are often in a state of constant tremor. During this motor irritability the reflexes are increased, but when profound coma comes on they are either largely diminished or totally abolished. The toxins of the typhoid bacillus, acting poisonously upon the nervous centers, are undoubtedly the cause of the nervous symptoms in typhoid.

Nervous complications and sequelæ may arise. Chief among these is *paralysis*, which is most probably due to neuritis. The lesion may involve one, two, or more nerves, and in this way we may have either a paralysis of one limb or, more rarely, a true paraplegia. *Aphasia* may be a sequel, particularly in children. *Hemiplegia*, due to hemorrhage or a localized encephalitis, may occur either as a complication or sequence of the disease. Following typhoid fever, the patient may exhibit evidences of *mental enfeeblement*, and even *insanity* where a predisposition to this condition has existed; and insanity is relatively more common after this disease than after any others belonging to the same class. I have seen four instances, all of which recovered, while Osler has seen five, four of which ended similarly. It is in most cases, as pointed out by Wood, a confusional insanity, due to exhaustion and impairment of the nutrition of the nerve-centers, while in a smaller contingent it takes the form of a true melancholia. After the conclusion of typhoid, as well as during its course, *neuralgia* affecting the occipital and other cranial nerves is not infrequent. Great hyperesthesia of the skin and muscles is common during convalescence, attacking the lower extremities by preference (Strümpell). The so-called "typhoid spine" (Gibney) has also been observed, and consists in an acute inflammation of one or more vertebræ following typhoid. The chief symptoms are pain in the back and hips of a lancinating character. The point of origin appears to be the small of the back; thence the pains extend paroxysmally up and along the spine and to the abdomen. They subside gradually, leaving the back weak and painful on attempts at turning in bed, etc. Plantar and other skin-reflexes are increased, and the knee-jerks are preserved. G. E. de Schweinitz has described at length the *ocular complications* and *sequelæ* of typhoid fever. Affections of the conjunctiva and cornea and retinal hemorrhage are perhaps the most frequent, although optic neuritis and affections of the uveal tract also occur.

(*g*) **The Urinary System.**—*Urine.*—The urine is lessened in quantity and high-colored, with an increased specific gravity up to the arrival of the stage of decline. About this time, and rarely earlier, it grows light in color, larger in quantity than the normal, and the specific gravity is relatively diminished. Both urea and uric acid are increased during the earlier stages, and sometimes throughout the attack, while during convalescence both are diminished. On the other hand, the chlorids are diminished during the active stages of the disease and increased during its decline. A *febrile albuminuria* is quite common, and the sediment may show an excess of renal epithelium, a few blood-cells, and occasionally renal casts.

Acute nephritis may develop as a complication in the earlier or later course of the disease, and can be recognized to a certainty only by a thorough appreciation of the urinary phenomena. The urine is diminished in quantity, being often scanty, and there may be retention. It contains characteristic morphologic elements (albumin, casts, blood, and epithelium). The development of the *typhoid state* in this affection is rendered much more probable in the presence of this complication, and, moreover, uremic symptoms often put in an appearance at this juncture, and then the situation is really serious. Acute nephritis may arise at one or other of three different periods, and its significance varies with the time of onset: (*a*) at the beginning of the fever, when it often obscures the true nature of the malady. This is the *nephro-typhoid* of the German authors, and will be referred to hereafter (*vide infra*, Varieties); (*b*) in the early part of the fastigium or the second week of the disease. Coming on at this time—an event which I have observed in two instances—it is probably to be ascribed to the local effect of the toxin upon the renal tissues. Both of my own instances proved fatal, and in both an autopsy was refused. Wagner[1] has had 5 cases of recovery in succession, but the high mortality mentioned by Amat—10 deaths in 12 cases—is the more common experience. (*c*) Acute nephritis may arise as a sequel of typhoid, when there is almost invariably associated a decided edema. In this category of cases recovery is to be expected.

The *diazo-reaction* of Ehrlich is a valuable aid in diagnosis, but may be present also in acute phthisis, meningitis, measles, pneumonia, yellow fever, and other fevers. To obtain it two other solutions (*a* and *b*) are needed: We mix 1 part of solution (*a*), which consists of a 0.5 per cent. solution of sodium nitrite, with 50 parts of solution (*b*), which consists of 2 grams of sulfanilic acid, 150 c.c. of hydrochloric acid, and 1000 c.c. of distilled water. To this an equal volume of urine is added, and the contents of the test-tube are then thoroughly shaken. A layer of ammonium hydrate is now superimposed, and at the line of contact a ruby or pink ring develops. A more reliable change, however, is a rose-red (pink) hue of the foam. Says Cummins, "Upon employing a dilution of 1 : 150 other conditions are eliminated (except a small percentage of tuberculous cases)." It is present in about 70 per cent. of the cases. The reaction begins about the beginning of the second week, sometimes later, and lasts usually until defervescence is well advanced. A brownish ring is given by normal urine.

Diabetes mellitus is, in extremely rare instances, developed after typhoid. Hematuria has also been observed as a symptom of the hemorrhagic diathesis.

There is a post-typhoid, diphtheritic *pyelitis* in which the pelves and calices of the kidneys are the seat of membranous exudation, and later of erosion and ulceration. The urine generally contains blood and pus.

Simple *vesical catarrh* may rarely result from catheterization for retention. Typhoid cystitis, in which the bacilli are found in pure culture in the urine, is not rare (*vide* p. 53). It occurs principally in patients who are predisposed by local conditions.

Orchitis, *epididymitis*, *spermatocystitis*, *prostatitis*, and *ovaritis* are

[1] *Deutsch. Archiv für klin. Med.*, Bd. xxv. and xxxvii.

occasional sequels. Blumenfeld collected 69 cases of orchitis; it generally develops suddenly during convalescence.

(*h*) **The Joints.**—Typhoid, septic and rheumatic arthritis may occasionally arise as a complication. The first is usually *mon-articular* (particularly in the hip); the last two commonly *polyarticular*. Keen has collected "in all 84 cases involving the joints."

(*i*) **The Bones.**—*Periostitis*, due to injury and muscular strain and often leading to *necrosis*, is a not rare sequel of typhoid. The favorite seats are the tibia and ribs, though in a case of my own at the Philadelphia Hospital it affected the os calcis. Ebermaier found the bacillus typhosus in the pus from 2 cases of suppurative post-typhoid periostitis, although other bacilli (streptococci, staphylococci, pneumococci) are at times associated. *Osteomyelitis* may also occur. Keen has collected 216 cases in which the bones were attacked.

(*j*) **The Muscles.**—As in the case of the heart, so the voluntary muscles exhibit hyaline degeneration; also abscesses, in consequence of secondary infection or of infection with the typhoid bacillus itself. Typhoid abscesses likewise result from perforations of the gut.

Associated Acute Infectious Diseases.—**Malarial fever** may be combined with typhoid, though the relationship is not a vital one. In an analysis of 2122 cases of malaria typhoid fever was associated in 8.[1] Many instances of so-called typhoid-malarial fever, however, would be shown to be pure typhoid by a careful blood-examination.

Pseudo-membranous inflammation, as above intimated, may occur in the naso-pharynx, larynx, gall-bladder, and genitals. *Measles*, *scarlatina*, and *chicken-pox* have also been known to arise in the course of, or during convalescence from, typhoid fever.

Erysipelas is a rare secondary affection coming on either during the height of the affection or (more frequently) after its close. Typhus fever may be associated with typhoid, but this is rare.

Clinical Varieties of Typhoid Fever.—These are numerous, and may grow out of peculiarities manifested during the course of the affection, as may be observed not only in different epidemics, but also in the same epidemic. The groups of cases described here have reference particularly to the degree of severity of the type, which varies between the wide limits of extreme mildness on the one hand and extreme severity on the other. The course of the disease may also be modified by the occurrence of one or more of its manifold complications.

(1) **The Mild or Rudimentary Form (Typhus Lævissimus).**—Of this variety many cases occur, and especially among children. The spleen is almost always enlarged, the roseate spots are sometimes present, while the temperature is moderately elevated and often partakes of the same character as that of true typhoid. The fever, however, may pursue the remittent type. Complications presented by special organs are usually absent, but grave accidents (intestinal hemorrhage, perforation) are not impossible.

The *diagnosis* is always difficult, owing to the feeble development of the characteristic symptoms, and in the total absence of the latter is out of the question; but the recognition is assured if a casual connec-

[1] "The Complications of Malaria," *Journal of the American Medical Association*, vol. xxiv., p. 919, by the author.

tion between them and typical cases can be shown to exist, and if the Widal test gives a positive result.

(2) **The abortive form** has a sudden onset, and is often marked by fits of shivering. The characteristic features of the disease (enlargement of the spleen, abdominal symptoms, rose spots, etc.) appear earlier than in the usual type, and soon become quite well marked. The fastigium is short, and the temperature, from the seventh to the twelfth day of the illness, declines by a prompt lysis, with profuse sweating. With the rather rapid fall of temperature there is a no less rapid improvement in every other leading symptom. Convalescence is speedy.

(3) **The Ambulatory Form (Latent or Walking Typhoid).**—The patient continues to walk about, either experiencing but slight disturbance or being unwilling to take to his bed. Such cases do not come under the care of the physician in many instances. Others, on account of debility, anorexia, diarrhea, and other vague symptoms, finally consult their physician, who may discover the presence of all the characteristic features of the disease. A third contingent, belonging to this form, continue to move about, or even to follow their usual vocations, till seized suddenly with profuse intestinal hemorrhage or general diffuse peritonitis following perforation.

(4) **The afebrile** is an exceedingly rare form of the affection—in this country at least. Liebermeister, however, has met with a number of cases at Bâsle, the symptoms being lassitude, depression, headache, neuro-muscular pains, anorexia, slow pulse, furred tongue, constipation or diarrhea, with enlargement of the spleen and roseate spots. These cases are often confined to bed, and there are occasional attempts at evening exacerbations of temperature (100.5° F.—38° C.).

(5) **Severe or Grave Forms.**—These may be dependent either wholly or in great part upon the degree of virulence of the typhoid poison. A profound intoxication of the system, as shown by high temperature, violent nervous symptoms, and great prostration, is noted. The grave types may arise in the course of cases of average severity from the development of serious complications. Again, to serious forms belong those cases that begin with the characteristic symptoms of a localized inflammation—*e. g.* the *cerebro-spinal form*, in which the nervous symptoms greatly predominate at the onset; the *nephro-typhoid* (before alluded to), in which the preliminary symptoms are those of acute Bright's disease; the *pneumo-typhoid* (*vide supra*), which begins with the manifestations of a more or less frank pneumonia.

Pleuro-typhoid.—The cases begin as an acute pleurisy, and are followed, soon or late, by the diagnostic evidences of typhoid fever. Talamon[1] distinguishes these cases from simple pleurisy by the intensity and continuous course of the fever, by the general depression, headache, and vertigo, and by the sleeplessness. Eiselt[2] has described a special form under the name *spleno-typhoid*, in which the spleen is enormously enlarged without characteristic intestinal lesions. Perisplenitis with adhesions may be noted. The *sudoral form* and *tonsillo-typhoid* (before described) also belong to this category. The fever is often of remittent type.

[1] *La Médecine moderne*, Paris, 1891.
[2] *La Semaine médicale*, August 27, 1891.

Typhoid septicemia may present the grave symptoms of an extreme intoxication, often merging into the typhoid state. Visceral and cutaneous hemorrhages may be superadded. Cases of hemorrhagic typhoid fever have been reported by A. A. Eshner and T. H. Weisenberg[1] and others. They are probably due "to a condition of systemic intoxication and septicemia" (Nicholls and Learmouth). Many circumstances connected with the individual influence decidedly the general course of the affection, and these are based upon such factors as *age*, *habits*, etc.

(6) **Typhoid Fever in Children.**—The onset is rather more abrupt than in the adult, and certain prodromal symptoms are rarely present (epistaxis, chilliness). On the other hand, bronchial and nervous symptoms are often quite pronounced. Again, during the fastigium some of the usual typhoid features may be missing—*e. g.* diarrhea and tympanites—while the eruption may either be slight or absent. The disproportion between pulse-ratio and temperature is less marked than in adults (Butler). Intestinal hemorrhage is rare and perforation almost never occurs.

(7) **Typhoid Fever in the Aged.**—The course of the affection presents no regular type. The temperature is not as high as usual, but there is marked adynamia and serious danger from certain complications, such as pneumonia, nephritis, coma, cardiac exhaustion, and the like.

The diagnosis is difficult, owing to the prominence of the nervous and pulmonary symptoms on the one hand, and the frequent absence of the more characteristic symptoms of typhoid on the other.

Diagnosis.—Unless all the chief characteristic features be present with a clear history, it is a golden rule not to make a positive diagnosis. Obviously, then, the physician at the first visit, often about the close of the first week, cannot, in many cases, diagnosticate typhoid with absolute certainty. If the case have been a typical one, the history of the gradual development of the disease, marked by such symptoms as languor, anorexia, headache, dulness, slight chills, increasing fever, and sometimes nose-bleed, will be obtained, and justify a strong suspicion of typhoid. When, in addition, diarrhea and the objective symptoms, splenic enlargement, dry bronchitis, tympanites, gurgling, with tenderness in the ileo-cecal region, are present, the diagnosis of typhoid is made highly probable. After the lapse of a few days—the beginning of the second week—the roseate spots usually appear. The most certain method of making an early, positive diagnosis is by an examination of the blood for the bacillus typhosus. Mabee and Taft[1] have described the method of making blood-cultures from the ear, and found that in early cases of typhoid (*i. e.*, within the first week) an accurate diagnosis in from 90 to 100 per cent. is easily possible. Says Peabody,[2] while blood-culture in ox bile is the earliest indication, at a later stage, when the organism can no longer be isolated from the blood, the agglutination reaction (*vide infra*) is usually present. In obscure cases the occurrence of intestinal hemorrhage or a characteristic decline by lysis is helpful. To show a casual relation between an obscure case and one that is clearly typhoid leaves little to be desired. The diagnosis should include the particular stage of the disease. Briefly, the most trustworthy diagnostic features are the gradual onset, peculiar temperature-curve (made up of the "step-

[1] *Amer. Jour. Med. Sci.*, March, 1901.
[2] *Boston Med. and Surg. Jour.*, June 1, 1908.

ladder" stage of development, the continued type of the fastigium, and the decline by lysis), enlarged spleen, the rose-colored spots, cultural experiments, and the sero-reaction.

Serum-diagnosis.—The results of the investigations of Pfeiffer and, later, those of Grüber and Widal have given us a specific sero-reaction.

Johnston recommends the following technic: The blood is obtained upon a clean glass slide from a needle-prick of the ear or finger of the suspected case. It is allowed to dry, and is then carried to the laboratory. A loop of bouillon-culture of genuine typhoid bacilli is placed upon a clean cover-glass, and to this is added a large loopful of a watery solution of the dried blood-specimen. The cover-glass is inverted over the concavity of a hollow slide and sealed at the edges with melted vaselin. Under the microscope, with a high-power dry lens or with a one-twelfth oil-immersion lens, a rapid clumping of the bacilli in the hanging drop can be observed,[1] and their motions cease almost instantly.

Diagnostic Value.—There is a general consensus of scientific opinion as to the great clinical value of the Widal reaction. The large statistics of Kneass and Stengel, based on 2283 cases, coupled with more recent available figures, show the presence of the reaction in 95.2 per cent., and no reaction in non-typhoid cases in 98 per cent. A. C. Abbott[2] reports that, according to the records of Widal reactions in 4154 cases, the error does not exceed 2.8 per cent.

Of 230 cases examined, 219 gave a positive result (Anders and McFarland[3]). In 128 of these cases this result was obtained prior to the appearance of the rose spots, or before the eighth day; in 36 cases the first reaction occurred during the second week; in 45, between the seventeenth and twenty-first days of the disease; in 8, not until the twenty-fifth day, and in 2 cases as late as the twenty-eighth day.

Interfering Conditions.—In the first place, a previous attack of typhoid fever may produce a reaction. In 39 cases of pure typhoid tested at periods of from one to eighteen months after defervescence, 13 reacted positively (Cabot and Lowell). It may be possible for the scene to be dominated by some other morbid process (tuberculosis, etc.). Kraus[4] found that a complicating pneumonia caused the Widal reaction to disappear. On the other hand, the reaction may be present in Weil's disease and in meat poisoning when the *Bacillus enteritidis* (Gärtner) is present. Again, exceptional cases occur with no reaction throughout. Brill has reported 17 cases of this sort; in such cases, however, the examination must be repeated until after convalescence is completed.

Conradi[5] has discovered a new method of cultivating typhoid bacilli. A small amount of blood is obtained by lancing the ear; this is inoculated into a small sterile bottle of bile, to which a little peptone and glycerin has been added. After sixteen hours the bile is reinoculated onto lactose-litmus agar. A positive diagnosis can be made in thirty hours.

[1] *Medical News*, Nov. 14, 1896.
[2] *Phila. Med. Jour.*, Feb. 25, 1899.
[3] *Phila. Med. Jour.*, April 8, 1899.
[4] *Zeit. f. Heilk.*, Bd. xxi., H. 5.
[5] *Münch. med. Wochenschr.*, 1906, vol. liii, p. 1654.

Chantemesse[1] suggests an ophthalmic test, but the cutaneous reaction is a more simple test and "gave positive results in every case in which it was employed" (Deehan). C. B. Burke[2] has described a new reflex sign. Prendergast proposes a new typhoid fever test consisting in the injection with a fine hypodermic needle of a few drops of a suspension of dead typhoid bacilli of the strength of less than 5,000,000 per c.c. No reaction occurs in typhoid infection, but in the non-typhoid patient a well-marked area of redness around the injection develops within twenty-four hours.

The cases that begin with the well-defined local inflammatory lesions previously referred to (tonsillo-typhoid, pneumo-typhoid, pleuro-typhoid, nephro-typhoid) cannot be recognized at the outset. The same local inflammatory conditions may, independently of typhoid fever, be combined with a genuine typhoid state. In all instances of typhoid fever in which, at the time of onset, localization occurs, the degree of fever and prostration are apt to be out of proportion to the local symptoms, and the former are apt to continue after the subsidence of the latter. A careful observation of the symptoms after the first week will usually detect undoubted symptoms of typhoid. The Widal test decides these cases. Blood-cultures if made early will also set the diagnosis at rest. The bacilli may be obtained from the stools and urine.

Differential Diagnosis.—(1) **Typhus fever** (rarely met with) is to be differentiated by its appearance as an epidemic, by its sudden onset, by the deeper stupor, the besotted expression of the features, the injected conjunctivæ, the contracted pupils, the appearance on the fourth day of maculæ which are transformed into petechiæ; by the shorter course, the termination by crisis, and the absence of the Widal reaction.

(2) **Acute miliary tuberculosis** is to be differentiated from typhoid fever by the greater frequency of the pulse and respirations, the cough, and in some instances by the expectoration; by the diffuse cyanosis and the presence (sometimes) of choroidal tubercles. *Blood-examinations* may show leukocytosis, but the large mononuclears are not increased as in typhoid fever. There is an absence of the temperature-curve, the pulse, the characteristic eruption, and the Widal reaction and abdominal symptoms of typhoid. In doubtful cases lumbar puncture and blood-cultures should be undertaken, as tubercle or typhoid bacilli may be found.

(3) **Malarial fever** may assume the continued form of fever—*e. g.*, the æstivo-autumnal type, in which chills may be absent—and there are typhoids that affect both remittent and intermittent malarial fevers. Malaria can be differentiated from typhoid fever only by the detection of Laveran's hematozoa in the blood.

Should *typho-malarial fever* be suspected and the typhoid symptoms be unequivocal, the finding of the malarial organism would establish the diagnosis and differentiate the hybrid from pure typhoid.

(4) **Relapsing fever** is distinguished by its abrupt onset, with rigor, high fever, pain in the epigastrium; by its brief duration, termination by crisis, and the occurrence of a relapse at the end of a week; by the absence of the characteristic eruption and the sero-reaction of typhoid. The finding of the spirilla discriminates relapsing fever.

(5) **Meningitis.**—In striking contrast with the specific typhoid symp-

[1] *Progressive Medicine*, March, 1910, p. 186.
[2] *New York Med. Jour.*, Dec. 16, 1911.

toms, meningitis exhibits marked hyperesthesia, intolerance of light and sound, exaggerated reflexes, and often muscular rigidity before the stage of effusion; also restlessness, peevishness (unlike the dulness observed in typhoid patients), vomiting, and constipation (*vide* Acute Miliary Tuberculosis). The temperature maintains a lower level on the average, and is more irregular in type than in typhoid; the pulse is more irregular, and the nervous symptoms assume greater prominence in the earlier stages, particularly headache and delirium. On the other hand, true typhoid symptoms are wanting in meningitis.

(6) **Tuberculous meningitis** gives a characteristic previous or family history, occurs in young subjects, and the tendon and cutaneous reflexes exhibit wide variations as to intensity, within brief periods and throughout the whole attack. An examination with the ophthalmoscope may reveal choroidal tubercles. The Widal reaction is missing.

(7) **Catarrhal enteritis** in children, with prominent abdominal symptoms, may simulate typhoid fever very closely. In the former the symptoms are all gastro-intestinal, save perhaps the occurrence of slight febrile disturbance and certain nervous phenomena, while typhoid fever manifests a wider range of symptoms (some of which are peculiarly its own—notably the greater prostration, more marked fever, enlargement of the spleen, the sero-reaction, and the characteristic eruption). In young children the last-named symptom may be either wanting or atypical, in which case the existence of enlargement of the spleen coupled with other phenomena, particularly the Widal reaction, will suffice.

(8) **Salpingitis** on the right side may resemble typhoid. In the former there is usually a clear history either of antecedent vaginitis or of an abortion, and there exist special evidences of local peritonitis, but not the classic features of typhoid fever. A digital examination *per vaginam*, however, is necessary to the certitude of diagnosis in salpingitis.

The diagnosis between typhoid fever and *typhoid pneumonia*, *influenza*, *ulcerative endocarditis*, and *appendicitis* will be considered hereafter.

Prognosis.—As in all other acute infectious diseases, so in typhoid, the prognosis depends upon three main considerations:

(1) **The severity of the type of the infection**, which is indicated in great measure by the degree of fever. A temperature of 106° F. (41.1° C.) is a serious symptom, and, if maintained at this point for a few days, an almost certainly mortal one. I have not seen a single instance in which the temperature has touched 106° F. (41.1° C.) for two or three successive days that has recovered. If the temperature mounts to and keeps at 105° F. (40.5° C.) for more than three or four days, the prognosis should be made with due reserve. When the fastigium is much prolonged, even though the fever be not exceptional, the prognosis is usually grave; while, on the other hand, marked nocturnal remissions are of favorable omen. A sudden, deep fall, however, may imply danger (intestinal hemorrhage, collapse).

The researches of Isaac Ott have taught us that, while high temperature is an indication of danger in specific fevers, it is not always the cause of it. He regards high temperature as being only a part of an infectious process, and points out that the thermotaxic centers of the cortex may be so disordered as to alter the harmony between the heat-production and

heat-dissipation. Under these circumstances a specific fever of severe form may be associated with a slight elevation of temperature.

The power of resistance to the influence of high temperature is quite reliably indicated by the condition of the heart. So long as the pulse is regular and its rate does not exceed 110 or 120 beats per minute, the outlook is favorable. When, however, the pulse maintains an average rate of 130 or more—a condition with which there is usually associated some degree of cyanosis, pulmonary congestion, and edema—the outcome is to be regarded as doubtful. Collapse is apt to follow the occurrence of sudden complications (perforation, hemorrhage), but it may also arise causelessly. The absence of eosinophiles from the blood-picture is an unfavorable prognostic sign.

Serious types are also shown by certain nervous symptoms, such as wild delirium, stupor, and well-marked symptoms of motor irritation.

(2) **Circumstances of the Patient.**—Certain individual peculiarities render the prognosis highly unfavorable. It is *bad* in very *fat* persons. In such cases there is a great danger of sudden collapse, and this fact also holds to a less degree with reference to subjects of certain chronic diseases (Bright's disease, heart disease, gout, emphysema).

Age is an influential modifying factor. After puberty the gravity of the disease increases with increasing years. Indeed, it may be said that, as a rule, typhoid has an unfavorable prognosis in persons past forty years, for the reason that at this time of life there is an added liability to pulmonary complications and failure of cardiac reserve. In children (*vide* Clinical Varieties) the tendency to hemorrhage and peritonitis is reduced to a minimum, and the mortality is not over 1 per cent.

The **puerperal state** renders a typhoid patient liable to many accidents and peculiar complications, and independently of pregnancy the disease is more fatal among females than males. *Chronic alcoholism* is apt to be complicated with *delirium tremens*, often preceded by *pneumonia*. Such patients are also prone to heart-degeneration and exhaustion.

Environment affects the prognosis, poor sanitary arrangements and poor attention greatly diminishing, and the opposite conditions greatly augmenting, the chances for recovery. Improved methods of treatment in recent years have effected a decided lowering of the death-rate. Here it may be said that the average mortality rate of typhoid is from 8 to 10 per cent., as against 15 to 20 per cent. formerly. The death-rate was 2.3 per cent. lower among the inoculated South African soldiers than in the uninoculated. It must ever be remembered, however, that epidemics differ widely as to their mortality list—a fact which makes a precise statement regarding the question an impossibility.

(3) The third and last consideration is **the presence or absence of dangerous complications and accidents.** These have all been enumerated and their prognostic significance stated (*supra*). Merely to reiterate some of those that lend fresh peril to the typhoid patient, arranging them with some regard for the order of their relative gravity, may prove helpful to the student. They are—perforation with diffuse peritonitis, intestinal hemorrhage, lobar pneumonia, lobular pneumonia, sudden collapse (due to cardiac weakness), excessive tympanites (often with marked diarrhea), and hypostatic congestion of the lungs.

Relapses of Typhoid Fever.

A relapse is a repetition of all the characteristics of typhoid after the latter has run its course. As a rule, the return occurs from one week to ten days after the beginning of convalescence, though it may be either earlier or later; and occasionally a relapse develops before the temperature has become normal (*intercurrent relapse*). The cause of relapses is a reinvasion of the blood by the typhoid bacilli or their secretions from within the body, and the source of the bacilli is most probably the gall-bladder. The *pathologic lesions* differ in no essential way from those described as belonging to the primary attack, but the stages through which they pass are not quite so long.

In the *interval* between the primary attack and the relapse there may be present suspicious features, such as a slight enlargement of the spleen, a trivial evening rise of temperature, an unnatural apathy or dulness, and a more profound prostration than is usual. In the majority of instances, however, there are no premonitory symptoms. The *onset* is rather more sudden, and rigors are more common, than in primary typhoid. The temperature, however, rises in the characteristic "step-ladder" fashion, reaching the fastigium in two or three days, and the same relative abridgment of the fastigium and defervescence is observed. It follows that a relapse has a shorter duration than a primary attack, and, indeed, it rarely exceeds two or three weeks. The temperature may, however, touch a higher limit in the relapse than in the primary attack; but, with rare exceptions, when the primary typhoid is of average or even greater than average severity, the temperature in the relapse does not reach an equal height. The characteristic rash appears earlier —from the second to the fourth day—and is somewhat darker and coarser than that of the first attack. The spleen swells rapidly. The *intercurrent relapse* sets in while the temperature is declining; the fever again rises, and often ranges higher than in the primary attack.

Diagnosis.—Upon the points that are distinctive of a primary attack of typhoid fever rests the important diagnosis between a relapse and a recrudescence (*spurious relapse*). The latter is usually attributable either to errors in diet, to undue muscular exertion, or to great mental excitement; and, whilst it occurs during convalescence, it seldom lasts longer than one, two, or three days, and is not characterized by the diagnostic symptom-group of a relapse (peculiar temperature-curve, enlarged spleen, and specific eruption).

The **prognosis** of relapses depends very much upon the severity of the primary attack, those following severe attacks being relatively milder than those that follow the rudimentary, primary attacks.

The frequency of relapses differs widely in different epidemics. Hence the fact that the percentage of relapses as estimated by different authors ranges from 3 to 15 per cent. need excite no surprise. The relapse may repeat itself once, twice, or even thrice, and two relapses occur in about 1 per cent. of the cases. In a case which I[1] reported three successive and typical relapses occurred. The pale line or ridge which was mentioned (*vide* Clinical History) as noticeable in the nails after typhoid occurs similarly after each relapse, and in the afore-mentioned case of my own four distinct whitish, transverse ridges were noted. Da Costa has recorded five relapses in each of two cases.

[1] *Med. and Surg. Reporter*, vol. xlvii., p. 66.

Recurrences.—By this term is meant successive attacks separated by longer or shorter intervals after complete recovery from the primary attack. Typhoid fever usually bestows lasting immunity, but this is not an invariable rule. Eichhorst has studied 600 cases, and found that in 28 of the number (4.7 per cent.) a second attack occurred. Soldiers who are subjected to typhoid fever, commonly give a history of previous attacks (D. Parker). I have seen a number of typical recurrences of typhoid fever, in two persons, the intervals having been five and eight years respectively. Very rarely three separate attacks have occurred in the same individual, and a second is usually milder than the first attack.

Treatment.—(*a*) **Prophylaxis.**—The municipal authorities possess in thorough filtration a power that can be used to advantage. For example, in Vienna, by purification of the water-supply, the death-rate in typhoid fever was reduced from 12.5 per 10,000 to 1.1 per 10,000.

It has been well said that typhoid bacilli do not naturally inhabit water and milk, but man is their natural host, hence the primary source of the bacilli. Let us make sure that every typhoid bacillus is killed immediately on leaving every host and the disease is at an end (McCrae). The best means that can be employed during the attack, with a view to limiting the spread of typhoid, is *disinfection*, and the following description comprises its essential points:

Disinfection in typhoid may conveniently be divided into (*a*) that of the excreta (stools, urine, vomitus, and sputum); (*b*) of the bed and its coverings; (*c*) of the patient and the sick-room. While all of these subdivisions are of the greatest importance in the treatment of a case, *the disinfection of the excreta* (*a*) is perhaps most carelessly performed, and hence the importance of the statement that all stools and urine voided by the patient, as well as the vomitus and sputa, should be promptly treated as follows: The excreta should be received in a vessel that can be thoroughly disinfected inside and out with any of the several standard solutions, of which that of chlorinated lime (strength, 6 ounces per gallon) is the most effective and satisfactory.

It is my custom to order that one pint of the chlorinated lime solution be placed in the bed-pan (or other appropriate receptacle) *before* the discharges are received therein, and from one to two pints *after*. The whole is thoroughly mixed by stirring and shaking, care being taken that all solid masses are broken up. The vessel is then allowed to stand for three hours before it is emptied into the water-closet. Phenol is also efficient and cheap. The stool should be mixed with about twice its volume of a 1 : 10 to 20 phenol solution and allowed to stand for several hours.

Gwyn[1] has given the following results of his investigations into the question of typhoid bacilli in the urines of typhoid fever patients: They are present in from 20 to 30 per cent. of the cases, and may be exceedingly numerous. The organisms may persist for months or years. For the disinfection of the urine in the bladder, urotropin is serviceable when administered by the mouth. Under no circumstances, however, should its administration permit the disinfection of the voided urine to be neglected. As an irrigation, Gwyn recommends mercuric-

[1] *Philada. Med. Jour.*, Jan. 12, 1901.

chlorid solutions (1 : 100,000 to 1 : 50,000). To disinfect the urine, "the best solutions are, phenol (carbolic acid) 1 to 20, in an amount equal to that of the urine, or bichlorid of mercury 1 to 1000 in an amount one-fifteenth that of the fluid to be sterilized. These mixtures with the urine should stand at least two hours." [1]

(*b*) It should be an invariable rule to change the bed- and body-linen daily, and as often as soiled. The mattress should be protected by a rubber cover, and this, together with the soiled linen and blankets, should be received in a sheet that has previously been dipped in a 5 per cent. solution of carbolic acid. The rubber sheets are to be washed with the carbolic-acid solution, but all other bed-clothes must be boiled for half an hour. When the patient leaves the sick-room the mattresses are to be fumigated and aired daily for a week, and the rubber covers and bedsteads washed with a solution of mercuric chlorid (1 : 1000).

(*c*) After every stool the patient should be cleansed with a compress of cloth or cotton wet with a solution of mercuric chlorid (1 : 2000) or of carbolic acid (1 : 40). The bed-pan and hopper are to be similarly treated, and the cloths used immmediately burned. Fitz recommends that the feeding utensils be cleansed in boiling water after using.

Since it is well known that many epidemics are directly traceable to the drinking-supply of water and milk, it is necessary that all water and milk used by the patient and other members of the household be boiled for half an hour before being ingested; and if an epidemic be prevailing, the community at large should join in this precaution. In view of the significant rôle played by the bacilli-carriers, convalescents must be regarded as dangerous for a long time after apparent recovery. The patient should report to the physician for examination of the excreta until it is satisfactorily shown that no more typhoid bacilli are being passed.

Isolation of Patients.—It is advisable to isolate typhoid cases as far as possible—*e. g.*, in hospitals, to keep them in special wards; in private families, in special apartments. There is incontestable proof that typhoid fever is feebly contagious.[2] At the Johns Hopkins Hospital 1.81 per cent. of all cases are of hospital origin (Cole).

Prophylactic Inoculations.—Excellent results have followed the *preventive inoculation* of healthy persons with typhoid virus. The striking results of antityphoid vaccination are shown by the Medical Corps of the United States Army, *e. g.*, the Maneuver Division at San Antonio, Tex., in 1911: Mean strength, 12,801; cases of typhoid fever, 1; deaths from typhoid fever, 0.

The procedure of administration is as follows: "The initial dose is 500,000,000, and two successive doses of 1,000,000,000 are given at ten-day intervals. The antibodies develop in from five to ten days later, and are bacteriolysins, opsonins, and agglutinins, the last in large amounts. The last two measure the antibodies present, since their quantitative relations are quite constant. The increase in agglutinins is quite evident in four to five days; the fall begins in six weeks and the normal is reached in fifteen months. The injection is given in the deltoid region, the skin being previously washed with tricresol solution and soap. The needle

[1] *Jour. of Amer. Med. Assoc.*, April 6, 1912, p. 1015.
[2] *Philada. Hosp. Report*, 1891, vol. i., p. 149, by the writer.

puncture is closed with the compound solution of tricresol (cresol, 500; linseed oil, 350; potassium hydroxid, 80; water, to 1000). Tincture of iodin may be substituted for the latter purpose, but the compound cresol solution for use in camps is the ideal method" (R. W. Wilcox).[1] Irwin and Houston successfully treated a case of persistent typhoid bacilluria by means of a vaccine prepared from typhoid bacilli.

(*b*) **Treatment of the Attack.**—(1) **The general conduct of the case,** including skilful nursing, is of paramount importance to the typhoid patient. He should be put to bed as soon as the indications point to this disease, and kept there continuously in the recumbent posture till the end of the attack. The sick-room should have a sunny exposure if possible; should be cool and well ventilated, though free from strong currents; and perfect cleanliness both of the room and of the utensils employed in the management of the case should be attempted. The bed should be provided with a woven-wire mattress, upon which should be placed one of hair. A rubber cloth is spread beneath the sheet, and the latter kept smooth in order to lessen the danger from bed-sores. A seriously ill patient should lie on an air-cushion or, better still, a water-bed, and to avoid bed-sores he should be instructed to turn gently to either side from time to time. His back, hips, and heels should be bathed frequently with a mixture of alum and salt in dilute alcohol. The use of the bed-pan and urinal is an absolute necessity. When a good nurse cannot be had, the attending physician must note *in writing* the directions regarding the disinfection of the excreta, bed-linen, and utensils, as well as regarding the exhibition of the food, medicine, etc. The mouth and throat should be kept clean, since by so doing we obviate unpleasant and even dangerous complications (aphthous ulcer, thrush, parotitis, lobular pneumonia, etc.). If they arise, the nurse or attendant should wash the mouth and tongue several times daily with a solution of boric acid (3 per cent.), and the throat may be sprayed at equal intervals with a similar solution. A frequent moistening of the tongue and mouth, and particularly the lips, with glycerin and water (equal parts) gives great comfort when they are dry and parched.

(2) An **appropriate liquid diet** should be employed, and the best article of food is milk, which it is well to dilute with plain water (or lime-water), since aërated waters are objectionable in that they sometimes increase the meteorism. The daily quantity should not be less than three pints, and it is important that the stools be examined, since, if the milk be not thoroughly transformed, curds or (on microscopic examination) numerous fat-globules will be seen, in which case a smaller amount should be given. If curds or fat are still seen, the milk should be peptonized. Experience teaches that milk is often better taken and better borne when a little brandy, coffee, or tea is added to it. When milk cannot be taken or digested in sufficient amount, either whey, sour milk, or buttermilk may be tried; and if these be distasteful, we may replace them (wholly or in part) by meat-juices or broths of various sorts, together with one of the standard infant's foods made with milk or water. Albumin-water, prepared by straining egg-white through a cloth and adding an equal part of water, has given much satisfaction in my hands. It may be made pleasant to the taste by

[1] *Amer. Pract.*, March, 1912.

flavoring with vanilla or lemon, and with meat-juice and broths will often support a patient during the most trying period of the attack.

High calorie feeding is advised by Shattuck, Robertson, Shaffer and Coleman, and others, *e. g.*, a dietary made up as follows: a quart of milk, a pint of cream, six ounces of milk-sugar, eggs, toast, butter, cereals, potato, apple-sauce, and the like. There are typhoid subjects who cannot (on account of vomiting, etc.) take *per oram* sufficient nourishment to support life. In such cases we may supplement the usual method of feeding by rectal alimentation, when from 3 to 4 ounces (96.0–128.0) of peptonized milk, ½ ounce (16.0) of meat-juice, and a little egg-white may be combined, and employed at intervals of four hours. In early convalescence the patient may take milk-toast, well-cooked plain rice, entire eggs (diluted), or thin custards. Solid food should not be allowed till the temperature has been at the normal grade for one week at least. In cases in which the fastigium tends to become prolonged with increasing prostration, and those presenting the fever of exhaustion, the administration of soft food (eggs, finely scraped meat, well-cooked rice, plain) is often followed by improvement.

Pure cold water has a positive value as a diuretic in this disease. Cushing and Clarke[1] used large quantities of water internally (a gallon or more in twenty-four hours), administering it in small quantities at frequent, definite intervals. The toxic symptoms and mortality were lessened. The internal use of water stimulates renal activity by raising the blood-pressure.

(3) **Stimulants** are useful in about 50 per cent. of the cases. When the first heart-sound becomes weak or the vascular tone diminished, alcohol should be used regardless of the temperature. In severe types whisky is the best form; in milder ones some good wine, such as port, sherry, or madeira. It is well to begin with a moderate daily quantity, and then increase, if necessary, until the indication is fulfilled. If the patient so desires, we may use brandy instead of whisky, and it is usually toward the close of the second or during the third week of the disease that the indications for the use of alcohol arise. It is not only the best spur for a flagging heart, but is of equal value in combating unfavorable nervous symptoms due to the typhoid septicemia; and the time for commencing its use may be indicated first by the latter symptoms (*e. g.* delirium, coma, tremor). The quantity to be administered must be regulated by its effects, since it may act injuriously, and even aggravate the symptoms, though this is seldom the case. Threatened collapse may be met by full doses of alcohol (½ ounce—16.0—every hour), combined with strychnin (gr. $\frac{1}{15}$—0.004—every three hours), exhibited subcutaneously till the depression has been counteracted. Effective doses of diffusible stimulants, as champagne, are useful during periods of sudden circulatory depression. The cardiac stimulants mentioned above may be further supported by the use of digitalis and sulphuric ether. Stengel has recommended hypodermic injections of 1 to 2 grains (0.0648–0.1296) of camphor dissolved in 15 minims (1.0) of sterilized olive oil as a cardiac stimulant in typhoid fever.

(4) **Hydrotherapy.**—There is at the present day general agreement among medical authors that the best mode of treating typhoid fever is

[1] *Amer. Jour. Med. Sci.*, February, 1905.

by means of the *cold bath*, which was originally introduced by Currie, of London (more than a century ago), and reintroduced and successfully practised by Brand, of Stettin. The benefits offered to the patient by this method are so great and varied that it becomes the duty of every physician who treats typhoid fever to be prepared to employ it. The beneficial influences of the baths are as follows: (1) They absorb the body-heat directly, thus reducing the temperature and overcoming the ill effects of high fever, this action becoming more marked after a day or two of the treatment; (2) They improve the nervous symptoms, diminishing mental dulness, delirium, stupor, muscular tremors and twitchings, and inducing sleep; (3) They strengthen the heart, thus obviating the danger of sudden circulatory collapse and the consequences of increasing cardiac weakness (hypostatic congestion of the lungs, venous thrombosis, etc.); (4) They stimulate the respirations, whereby the inspirations are deepened and the tendency to pulmonary complications greatly lessened, especially severe bronchitis, lobular pneumonia, etc.; (5) The renal function is invigorated, and as a result the elimination of typhotoxins by the kidneys is increased (Roque and Weil); (6) On account of the cleanliness of the skin which they ensure, bed-sores rarely occur; (7) They may shorten the stay in the hospital or sick-room, but not the stay in bed, except, perhaps, in the lighter types.

Unquestionably, the good effects of this method receive striking confirmation from statistical reports which have been prepared by Brand, Osler, Jürgensen, and others abroad, and by Baruch, Wilson, and others at home. According to the warmest European advocates of the method, the mortality is less than 0.5 per cent. The results among American clinicians give an average mortality of 7.3 per cent. During five years 408 cases have been treated by the bath-method in the Royal Victoria Hospital, Montreal, with a mortality of 4.4 per cent.

The tub is to be brought to the bedside of the patient, and in hospital practice both bed and tub should be screened while the bath is in progress. After removing the night-dress and placing a large napkin around the loins, the patient should be lowered into the bath by a sheet held at each corner by an attendant (and, if seriously ill, with the least possible disturbance), and there carefully supported and held while in the bath. If sleeping, the patient must be awakened and the bath delayed for ten or fifteen minutes. Young subjects and adults in light cases of the disease may be handled properly by two persons, but I do not approve of allowing the patient to step from the bed into the bath, however light the case. While in the bath the skin-surface, particularly that of the back and limbs, is constantly rubbed by the attendants, in order to stimulate the peripheral circulation and as far as possible to avert chilliness and discomfort. The head of the patient rests upon a rubber air-cushion. At first he should be kept in the bath five to eight minutes; later, ten or fifteen minutes, according to the severity of the case. The head and face are bathed at once from a basin, and a cold compress is applied to the forehead, and, if prominent nervous symptoms be present, often associated with high temperature, water at 70° F. (21.1° C.) or lower should be poured from an elevation of about six inches upon the head and nape of the neck several times during the bath. The ears must be stopped with cotton when douching is practiced.

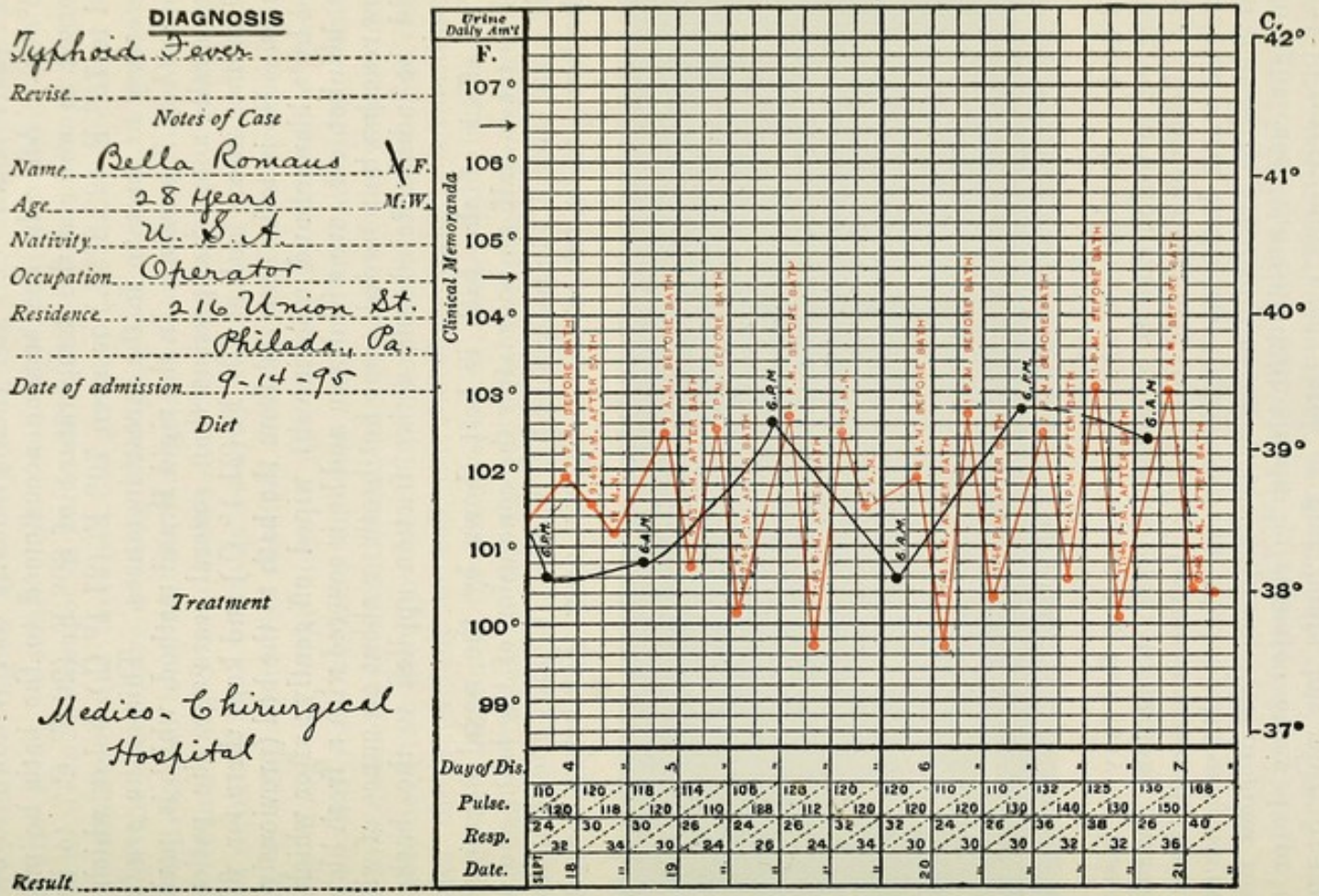


FIG. 5.—Chart illustrating the influence of cold baths in the treatment of typhoid fever.

If while in the water the patient complains bitterly of the cold or is very restless, a stimulant may be administered—f℥j (32.0) of whiskey, diluted—and if this fails he must be lifted into bed and further stimulated. If he be very young, highly sensitive, or elderly, it is best to place him at the commencement in water of a temperature of 85° or 90° F. (29.4°–32.2° C.), and then gradually cool it down to 80° F. (26.6° C.). After he has become accustomed to the bath he may be immersed in water at the temperature of 80° F. (26.6° C.), to be reduced to 75° F. (23.8° C.) or even 70° F. (21.1° C.), below which it is unnecessary to go save in the rarest instances. This is the *gradually cooled bath* of Ziemssen. In the rigid Brand method, which is, perhaps, less generally adopted at present than several years ago, the patient is lifted at once into a bath at 70° F. (21.1° C.) and kept there for fifteen minutes. He is to be removed from the bath to the bed (previously protected by a blanket and mackintosh), wiped off gently; after which the sheet, blanket, etc., are withdrawn and he is covered with a fresh blanket. If now reaction be retarded, some hot broth or about an ounce of whisky should be administered and active friction applied to the back and extremities.

The effect of the bath is best shown by the rectal temperature, which is taken half an hour after the conclusion of the bath, and again a half hour later if the patient be not asleep. Usually the temperature will be found to be two or three degrees lower than before the plunge

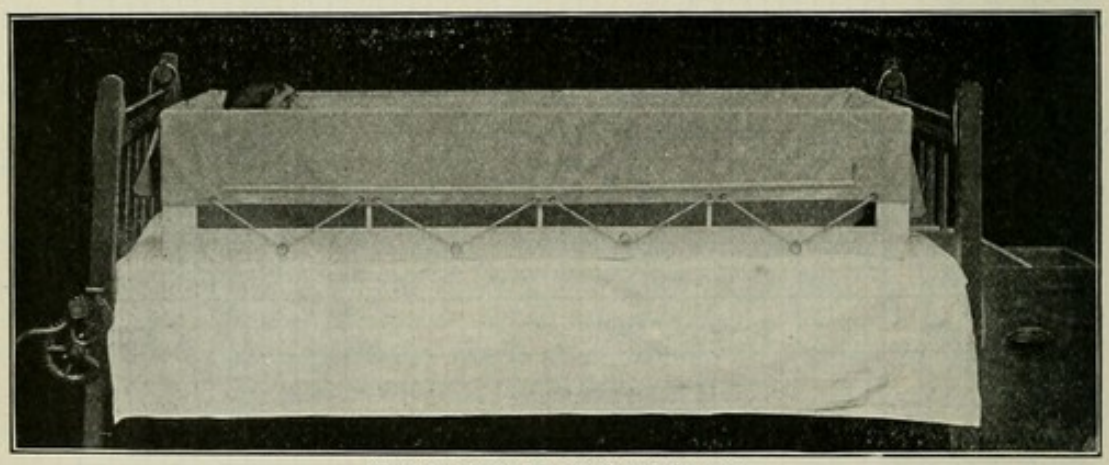


FIG. 6.—Portable bath-tub in use.

(see Fig. 5). In obstinate and severe cases the fall may be less than one degree, in which case it is advisable either to prolong the bath to twenty minutes or to reduce still further the temperature of the water. Protracted warm baths are highly recommended by Reisse and others when cold baths are badly borne or are unproductive of good results.

In light cases the cold bath should be repeated every six or eight hours; in severe ones, every three or four hours, but more frequently than once in three hours is not advisable, even in the worst cases. Sufficient water to immerse the patient to the neck (about 30 gallons—114 liters) should be used. During the night the patient should be allowed to sleep for six or eight hours if he can do so.

There are a number of convenient and satisfactory portable tubs in the market, but that devised by Dr. C. L. Furbush of Philadelphia

possesses certain leading advantages (Figs. 6, 7). The frame is made of light wood, and when folded is 4 inches (10.156 cm.) in depth, 14 inches (35.546 cm.) in width, and 5 feet 10 inches (1.778 m.) in length, so that it can be placed in a closet or beneath a bed. Less than two minutes are required to prepare the bath, which the patient receives while lying in bed. When in use the ends are fastened by brass pins hung on small chains, and these hold the frame in a fixed position. The tub proper is made of double-faced sheeting, reinforced in the middle, so as to resist the greatest amount of pressure. The sides of the sheet have a casing through which is passed a wooden rod 4 feet 4 inches long (1.320 meters), and outside of this a margin of $1\frac{1}{2}$ inches (3.808 cm.) is left for the brass eyelets, through which passes a rubber cord which is covered with woven cotton. This cord, which is attached to the sheet, is held to the frame by special brass fittings along the lower sides of the latter. By the use of the cord and wooden rods we have an even tension on both sides, combined with ample resistance to withstand the pressure of the water. An adjustable head-rest fits into the end of the frame. The wooden rod also enables the attendant to roll up the sheet quickly after the bath. Through the bottom of the sheet a 1-inch rubber tube is fitted with a stopper, and by means of this the tub can be emptied sooner than by a siphon. The frame is covered with ivory-enamel paint, and can be cleansed easily. The entire weight of the outfit is 25 pounds (11.33 kgms.).

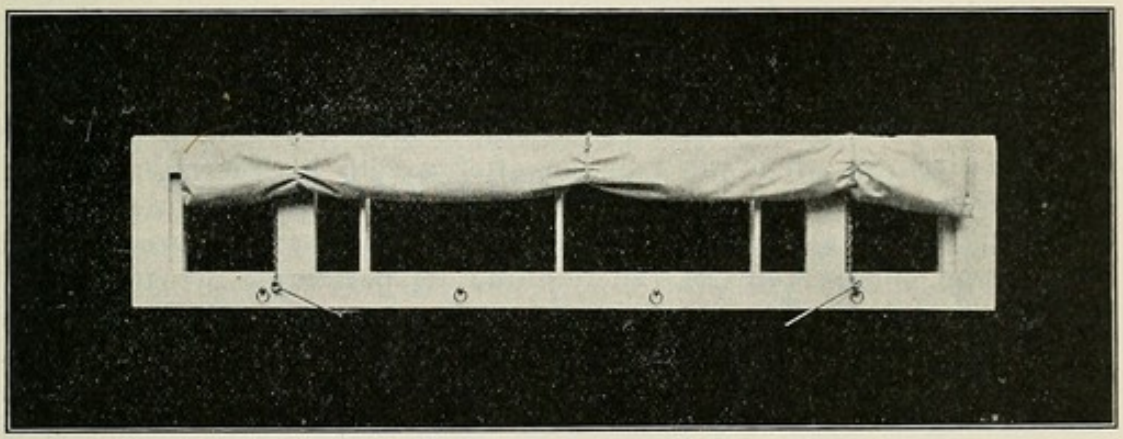


FIG. 7.—Portable bath-tub, folded.

Brand recommends that the baths be commenced when the temperature in the rectum reaches 102.2° F. (39° C.). The height of the temperature, *per se*, is not to be invariably regarded as an absolute indication for the employment of the cold bath, since the facts must be recollected that the essential effect is a stimulation of the nerve-centers which preside over the organic functions (respiration, circulation). Baruch insists that the object of cold baths with friction is to sustain the nervous system and circulation rather than for temperature reduction.

The *contraindications* to the use of baths are—(1) *Intestinal hemorrhage*, which is in itself attended with danger and requires absolute quiet for a time (four days), when the baths may be resumed if there is no recurrence. (2) *Peritonitis*, the occurrence of which always excites

suspicion of perforation. Here, again, rest and all that the term implies must be procured. (3) *Extreme Cardiac Weakness.*—The excitement in the necessary handling of the patient connected with the bath might prove fatal, as I have witnessed in one instance. This condition is sometimes met with in cases that come under observation at a late period, and in cases arising in aged and enfeebled subjects. (4) Cases that have progressed to an advanced stage (the third week of the disease) should not be immersed. Dangerous and even fatal collapse has been observed to follow cold baths under these circumstances.

Substitutes for the Cold Bath.—The prejudice which exists against the cold-bath treatment—at least in America—sometimes proves insurmountable. Again, there are many physicians who do not avail themselves of the means at command for carrying out hydrotherapy. In consequence of these facts substitutes for the tub baths are quite commonly in vogue. Among them, cold sponging of the body of the patient is often resorted to, though it secures for him trivial advantages as compared with those of the baths. If this method be employed, the water should be of the temperature of the air of the room or ward. The limbs should be sponged and dried in succession, and then the trunk. Whenever the temperature reaches 102.5° F. (39.1° C.) this measure is to be instituted, each sponging being continued until the desired effect has been produced (a reduction of the temperature of 1½° to 2° F.—1° C.), unless the patient gives signs of uneasiness, when it must be cut short. It may be repeated as often as required. To the water used equal parts of vinegar or spirits should be added. The efficacy of the cool sponging is enhanced by the simultaneous application of the ice-cap, either constantly or intermittently.

If this method fails, as it often does in severe types, the cold pack may form a satisfactory substitute; and I have found it of great use with children, in whom the reaction after a cold bath is often delayed or imperfect. The patient is placed upon a cot previously prepared by spreading over it a blanket, which is in turn covered with a sheet doubled and wrung out of water of the required temperature—70° to 80° F. (21.1°–26.6° C.). The sheet and blanket are now wrapped about the patient evenly, and he is left in the pack for a period varying from a half to one hour. Free diaphoresis generally ensues, and this aids in maintaining the fall of temperature. The effect, in most instances, is to reduce the body-heat two degrees or more, and the treatment may be repeated at intervals of three or four hours if needful. The wet sheet alone may surround the patient, and be sprinkled at short intervals with a watering-pot containing water at a temperature of 70° F. (21.1° C.). In desperate cases ice-water enemata may be tried. If carefully administered they accomplish a reduction of the temperature by two or more degrees. Leiter's coils may be applied to the head, chest, or abdomen.

Guaiacol has been used for its antipyretic effect by H. G. McCormick and others, from 10 to 30 minims (0.666–2.0) being applied to the skin surface. I have seen its use followed by rigors, hyperpyrexia, etc., but McCormick uses sufficient only to lower the temperature to 100° F. (37.7° C.), and has thus avoided all ill effects.

(5) **Internal Antipyretics.**—The most reliable of this group of medic-

aments (phenacetin, acetanilid, and antipyrin) are open to the serious objection that they depress cardiac power. Since heart-enfeeblement, which may develop either gradually or suddenly, is recognized by present-day clinicians as a common danger-signal of the disease, the time has come when the employment of coal-tar products should be discontinued.

(6) **Intestinal Antiseptics.**—Unquestionably these neither destroy the bacilli nor counteract the ill effects of their toxins, since both become active after they pass beyond the intestinal mucosa; but they are indicated in cases in which tympanites is a prominent manifestation. Some of the toxic substances occupying the intestines in this disease result from the acquired virulence of usually harmless organisms, and the amount of decomposable material is increased owing to defective hepatic and gastric secretions. Salol is broken in the intestinal canal into carbolic and salicylic acids, and has proved capable of controlling meteorism as nothing else has done in my hands. The dose is 2 to 3 grains (0.1296–0.1944) every three hours, preferably administered in capsule. With it I usually combine quinin in doses of 1 to 2 grains (0.0648–0.1296) each. Henry speaks in favor of thymol, and guaiacol carbonate has many advocates. Wilcox [1] urges that chlorin is capable of disinfecting the intestinal tract. Acetozone in daily doses of 15 to 20 grains is both an efficient and harmless intestinal antiseptic; it should be dissolved in a liter of water, flavored with some one of the volatile oils, and taken in divided portions. Systematic lavage of the intestinal tract is advisable in excessive tympanites. In cases in which pronounced meteorism occurs the use of hydrochloric acid in small doses after each feeding is serviceable, since the secretion of this agent, which normally inhibits putrefactive changes, is lessened.[2] Mild purgation with calomel, especially in the earlier stages, is useful. Carbolic acid, iodin, sulphocarbolate of zinc, and other antiseptic agents have their advocates.

Turpentine fulfils a leading indication. When the tongue is dry and brown, the abdomen distended, the general prostration marked, and often muttering delirium present—symptoms of the typhoid state—the use of this agent, together with alcoholics, constitutes the best mode of treatment. Of the rectified oil of turpentine, ♏ v–x (0.333–0.666) may be administered every third hour until relief is afforded.

(7) **Curative Inoculations with Cultures and Serum.**—In 1897 Bokenham [3] prepared an antityphoid serum from the horse. Chantemesse [4] has treated 1000 cases (using his own serum), with a death-rate of 4.3 per cent., while of 5121 patients who received routine treatment during the same period, 17 per cent. died. Hughes and Carter treated a number of cases with blood-serum derived from convalescent cases, but apart from a decided lowering of temperature the general course of the disease was not perceptibly modified. H. Forssman treated 20 cases with the typhoid serum of Kraus, and in those in which it was used early in the first week, the disease showed a mildness, which otherwise occurred only as an exception during the epidemic. M. W. Richardson,[5]

[1] *Med. News*, February 11, 1899.
[2] See *Therap. Gaz.*, April 15, 1900, by the writer.
[3] *Transactions London Pathological Soc.*, vol. xlix, p. 373.
[4] *Hyg. gen. et appliq.*, 1907, p. 577.
[5] *Boston Medical and Surgical Journal*, Oct. 3, 1907.

from a study of specific therapy in 130 cases of typhoid fever with Vaughan's non-toxic residue of the typhoid bacillus, concludes that it prevents relapses if inoculations are continued into convalescence. The value of vaccines for the following purposes must be conceded: "(1) As a means of prophylaxis; (2) in suitable cases when continued during convalescence, to prevent relapses; (3) to combat local infections with the typhoid bacillus, as, for example, bone suppurations which arise in the period of convalescence; and (4) for the removal of the typhoid bacilli from the feces and urine in the case of typhoid carriers." [1]

Chantemesse, Walters and Eaton, Sadler, and others report marked reduction of the mortality-rate of this disease under vaccine therapy. All observers, however, agree that very severe cases show no improvement. The dosage has varied greatly with different clinicians, *e.g.*, Sadler found an initial dose of 1,000,000 to 2,000,000 killed bacilli injected subcutaneously most beneficial, followed by a smaller dose every four or five days. "On the other hand, Walters and Eaton used doses of 100,000,000 to 500,000,000 bacilli." [2] W. Broughton-Alcock states that the living sensibilized bacilli should be preferred.

(8) **Treatment of Individual Symptoms and Complications.**—*Headache.*—Early in typhoid the headache demands relief. Absolute rest and cold to the head frequently suffice. Depressant analgesics are to be avoided so far as possible, although it sometimes becomes necessary to resort to them. At such times those least objectionable are to be selected. I have found that a mixture containing sodium bromid (gr. x to xv—0.6480 to 0.9720) and the deodorized tincture of opium (♏iij to v—0.1998 to 0.3330) in each dose, given at intervals of three or four hours, exercises a striking palliative influence. In occasional instances the above mixture fails, and then phenacetin (gr. ij to iij—0.1296 to 0.1944) may be substituted for the opium in the same combination.

Insomnia.—The cold baths or other measures calculated to relieve the headache often procure for the patient refreshing sleep. It is important not to allow him to go too long without sleep, since this tends to the development of a pronounced "typhoid state" and its concomitants. When the agents recommended for the headache fail, I employ morphin hypodermically in small doses (gr. $\frac{1}{16}$ to $\frac{1}{8}$—0.004 to 0.008) during the evening hours with excellent results, withdrawing the remedy so soon as decided amelioration of this symptom has taken place. Codein, sulfonal, and, more recently, veronal, trional, and chloralamid, have proved useful.

Chloral is certain in its action, but I have abandoned its use, since it apparently produced circulatory collapse in two instances.

Delirium.—Since the introduction of the Brand method delirium rarely calls for special medication. I have observed, in common with others, particularly during the advanced stages, that in cases in which the circulation was feeble and in which typhomania was a prominent feature, the administration of stimulants with a free hand completely dispelled the nervous phenomena. If alcohol fails, ether (♏x—0.666—at a dose) may be given hypodermically, and repeated in one or two hours if necessary. To combine with the arterial some nervous stimu-

[1] *Journal of the American Medical Association*, Dec. 10, 1910, by the writer.
[2] Editorial, *Jour. Amer. Med. Assoc.*, June 29, 1912, p. 2035.

lant (musk, valerian) will be found serviceable, particularly in cases in which the delirium assumes an hysteric type. Of special value are the bromids, hyoscin hydrobromate, the persistent use of ice to the head, and the agents suggested for the headache and insomnia.

Vomiting is rarely troublesome. Its chief cause is the irritation of the gastric mucosa, from improper diet or medication. After the removal of the cause, the use of ice in small pieces by swallowing affords relief. If vomiting occur during the period of development, minute doses of calomel, combined with sodium bicarbonate, may be prescribed with good effect. If it occur during the fastigium, the amount of milk taken should be reduced by one-half, peptonized, and then diluted, preferably with lime-water. If the patient experience a strong aversion to milk, it must be suspended temporarily and albumin water and broths substituted. Dry champagne may be administered simultaneously. Excessive irritability of the stomach calls for rest of the organ for a period of twenty-four hours, the patient being meanwhile supported by rectal alimentation and subcutaneous medication.

Diarrhea more than any other single symptom claims special attention. Two to four movements daily do not constitute diarrhea and do not demand treatment. It may be caused by overfeeding or by improper food —as shown by the stools, as a rule—in which case regulation of the diet is curative. It is often due to ulcerated and catarrhal lesions of the intestines, and particularly the large. Unquestionably, intestinal antiseptics which possess the property of insolubility are most valuable. Astringents may be combined with the latter or given separately. The subjoined formulæ have yielded good results in my own hands:

℞. Betanaphtol, ʒj (4.0);
Bismuth. subgallat., ʒij (8.0).
M. et ft. capsulæ No. xxiv.
Sig. One every two or three hours.

Or,

℞. Plumbi acetat., gr. xxiv (1.555);
Phenylis salicylat., ʒss (2.0).
M. et ft. capsulæ No. xii.
Sig. One every three or four hours, as required.

Large doses of bismuth (gr. xxx—2.0) every third hour are useful.

Late in typhoid fever, when the ulcers are fully developed, opium is of service; it tends to arrest the peristaltic action which keeps up the diarrhea and favors the spread of the inflammation to the peritoneum. When distention is increased by the use of opium it is to be omitted. I have recently observed brilliant results from the use of rectal injections of an astringent solution (tannic acid 1–2 per cent.), alternated with an antiseptic solution (salicylic acid 1–2 per cent.), each given once daily.

Constipation is to be relieved by simple enemata of soapsuds every second day. Calomel may be used in the early stage of dynamic cases. Its employment may be followed by symptoms of a milder type than are ordinarily encountered. If constipation exists during the third week, accompanied by an oscillating temperature-curve, saline laxatives in small but repeated doses may cut short the attack.

Tympanites.—This is sometimes a most distressing symptom, and treatment should be commenced early. As a remedy for tympanites turpentine is excellent and richly deserves a trial, but it does not, as some claim, influence the general course of the disease. When employed for this symptom alone I prefer to apply it in the form of stupes over the abdomen, although when, as is frequently the case, the gases occupy chiefly the large bowel, turpentine enemata should be given. Irrigation of the colon with the normal saline infusion has recently proved most effective in relieving excessive tympanites. Eserine, gr. $\frac{1}{100}$, every third hour, administered hypodermically, sometimes proves efficient.

The meteorism is often increased by the milk taken, and a change of food to meat-juices and albumin-water may be tried.

Hemorrhages.—The bowel-movements, if the hemorrhage has been copious, must be allowed to pass into the draw-sheet. The ice-bag (suspended if possible) should be applied to the right iliac region, and ice freely given by the mouth. Morphin, to control peristalsis, must be given, and, by preference, hypodermically. It may be supplemented by full doses of the acetate of lead. Cases in which slight oozing appears from time to time require turpentine. Adrenalin chlorid is serviceable, and Curtin[1] derived advantage from the use of suprarenal extract in cases in which there was general hemorrhage. The amount of food should be greatly restricted, and in serious bleedings abstinence from food for from twelve hours to three or four days is to be observed. When feedings are resumed, a teaspoonful or two of cold milk (repeated every two hours) may be given during the first twelve hours, then gradually increased in amount. For severe hemorrhages, *saline infusion*, either by the method of intravenous injection or by hypodermoclysis or enteroclysis, is to be strongly advised. The saline solution not only raises the blood-pressure in the vessels, but it also has a hemostatic action. The proper strength is 8 : 1000, and from 10 c.c. (3 fluidrams) to one-half liter may be employed if the collapse is marked, and repeated several times in the course of a day. Rectal injections may be somewhat larger. McCrae[2] advises calcium lactate in doses of 20 grains a day; it may be given subcutaneously, if rapid action is desired, in a 1 per cent. solution. Calcium salts are indicated where the coagulation time is slow. In case the blood gives a poor agglutination reaction, phlebotomy to the extent of about 10 ounces (according to the physical condition of the patient) may be tried. On the other hand, with the patient *in extremis*, transfusion may prove effective.

Peritonitis.—Operation for perforation offers some hope of cure, and with the progress of convalescence the chances of recovery from this accident improve. Deaver regards the acute development of pain and generalized abdominal rigidity and tenderness as an urgent indication for immediate celiotomy. Keen's statistics show that between twelve and twenty-four hours after perforation is the most favorable time for operation, this period giving 30 per cent. of recoveries. Le Conte[3] holds that it should be immediately undertaken. Rarely, appendicitis supervenes in typhoid fever. It demands prompt removal of the appendix.

[1] *Proceedings Phila. Co. Med. Soc.*, Nov., 1902.
[2] *Jour. Amer. Med. Assoc.*, Sept. 19, 1908.
[3] *Jour. Amer. Med. Assoc.*, Nov. 8, 1902.

Lobar Pneumonia.—Its treatment, when a complication, will be considered hereafter (*vide* Secondary Pneumonia). Pneumo-typhoid requires the same measures, until the true typhoid symptoms arise, as primary adynamic pneumonia (*vide* p. 126).

The *hypostatic congestion* of the lungs is to be met by heart-stimulants and by frequently changing the position of the patient.

Bronchitis.—No special measures are necessary when the bronchitis is confined to the larger tubes, as in typical cases, while, if diffuse, its management is like that of *broncho-pneumonia* (*vide* p. 559).

Laryngitis.—For this condition counter-irritation should be tried; and if this fails, a small blister may be applied below the angle of the jaw on either side. For edema of the larynx scarification and the inhalation of steam are useful measures. Then, should suffocation become imminent, tracheotomy should be performed without delay. Operation "gives a mortality of only 55.5 per cent." (Keen).

Bed-sores.—The preventive measures have already been considered, but the smallest bed-sore demands active treatment. It is to be kept clean and dusted with a powder composed of equal parts of boric acid, calomel, and bismuth; if sluggish, with a powder made up of aristol and iodoform.

Thrombosis of the femoral vein is best treated by elevating the part and keeping it at perfect rest. An ointment composed of equal parts of unguentum ichthyol (12 per cent.), lanolin, and unguentum belladonna, may be applied along the course of the affected vessel thrice daily.

(9) **Management of Convalescence.**—Some of the points connected with this subject have already been discussed (*diet, time for getting up*, etc.). I may add that should a recrudescence occur the patient should be kept at rest in the recumbent posture and a return made to the liquid forms of food. Often a moderate laxative serves a good purpose, particularly if an indiscretion in diet have been committed. The ulcers may not be healed, though the temperature may have been normal for a week or ten days; hence the patient should not be allowed to stir about for a period of two weeks after the temperature has become normal. At first his movements should be slow; he may soon, however, be allowed to exercise gently in the open air during seasons of favorable weather. Mental excitement is to be avoided. Occasionally during convalescence the diarrhea persists, being due to colonic ulceration, and is best treated by restricting the diet to milk and other light forms of albuminous food. The patient must be confined to bed. Medicinal treatment by the oxid of zinc internally and the use of astringent and antiseptic rectal injections, as before indicated, usually proves successful. Constipation is best relieved by simple enemata. Most patients require tonics. We should begin with a vegetable salt of iron in combination with a simple bitter (*e. g.*, infusion of gentian), and later an inorganic salt of iron, with quinin and strychnin, may be used. Relapses are to be treated as primary attacks.

Paratyphoid Fevers.

This term is applied to a group of affections that closely simulate typhoid fever clinically, but are due to different microbic cause.

Pathology.—The anatomic changes are simply those of septicemia with splenic swelling and occasionally non-specific ulcers in the intestine. H. G. Wells and L. O. Scott[1] have summarized the pathologic findings of 5 cases of paratyphoid and concluded that its pathology is different from that of ordinary typhoid. In 3 cases reported the ulcers resemble those of dysentery rather than those of typhoid; there are slight, if any, changes in Peyer's patches or the solitary follicles. The mesenteric glands show alterations, and focal necroses have been noted in the liver.

Etiology.—The disease is not a unit, but is probably caused by several members of the colon family. These organisms possess properties intermediate between the bacillus typhosus and the bacillus coli communis. Longcope[2] and others have found the paracolon bacillus; Ruxton, the paratyphoid bacillus, which is closely related to the bacillus typhosus. The paratyphoid bacillus is regarded as ubiquitous in certain parts of Germany. The *predisposing factors* and *sources of infection* are about the same as for true typhoid fever. Sacquépée and Bellot traced an epidemic comprising 19 cases to a cook (paratyphoid carrier). Minertz[3] claims paratyphoid to be an entirely different disease from typhoid, his experience confirming the fact that infection is derived from meat, especially pork, and not from human sources. Bainbridge[4] holds that meat-poisoning and paratyphoid fever are distinct diseases.

Symptoms and Course.—Typical cases usually manifest features that should arouse suspicion of their true nature. Brill has contrasted the diagnostic features of true typhoid fever and these allied conditions.

The incubation-period is somewhat briefer and the onset more abrupt than that of true typhoid. After three or four days of malaise the temperature rapidly rises to 104° F. (40° C.) or over, replacing the characteristic step-ladder curve. Mental dulness and apathy develop earlier and are marked. The initial headache is more intense, and constipation is common, although diarrhea is also observed. Brion's figures show diarrhea in 18 per cent. of the cases and melena in 5 per cent. The spleen is enlarged and rose-colored spots may appear, but the Widal reaction is absent. The duration of paratyphoid fever may be short, and the temperature decline by rapid lysis or crisis, or it may be long. Convalescence is also less protracted. J. H. Pratt refers to the frequency of complications (4 per cent.); they differ but little, either as to incidence or character, from those of typhoid fever. Relapses occur.

Diagnosis.—A bacteriologic diagnosis is essential. Cultures of paratyphoid bacilli can be obtained from the feces, urine, rose-spots, although preferably from the blood of the veins. The various subtypes of paratyphoid bacillus may be distinguished from one another by agglutination (Bielyaeff). It is necessary to test simultaneously the action of serum upon the typhoid bacillus and upon the paratyphoid bacillus obtained from the patient. Swan[5] suggests that if a blood-culture is

[1] *The Journal of Infectious Diseases*, I., No. 1, Jan., 1904.
[2] *Amer. Jour. Med. Sci.*, Aug., 1902.
[3] *Medizinische Klinik*, Berlin, Sept. 25, 1910.
[4] *Lancet*, London, March 30, 1912.
[5] *Amer. Jour. Med. Sci.*, May, 1906.

sterile, or if it is impossible to make such a culture, the patient should be catheterized under aseptic conditions and his urine examined bacteriologically. The paracolon bacillus may thus be obtained. Lessieur and Fischer state that the diagnosis should be based on an examination of the blood, as the agglutination test is frequently misleading.

Prognosis.—The course is usually favorable, although a few fatal cases have been reported.

The **treatment** does not differ from that of true typhoid fever.

TYPHUS FEVER.

(*Ship-fever, Camp-fever, Jail-fever, etc.*)

Definition.—An acute contagious disease of unknown specific etiology. It is characterized frequently by an abrupt invasion, and is marked by rigor, high fever, early nervous symptoms of great prominence, a maculo-petechial eruption appearing between the third and fifth days, and a termination by crisis.

Historic Note.—In 1759 the name *typhus*, which is at present universally employed, was given to it by Sauvages. In pre-sanitary times it prevailed in epidemic and endemic forms, particularly in Ireland and Russia, and its devastations among the armies were more destructive of human life than even shot and shell.

In 1812 typhus fever first appeared in America in the New England States. Its ravages did not cease until every Eastern State had been visited by the plague, when it totally disappeared. In 1836 it reappeared in Philadelphia, and with deadly effect. It has been shown by Anderson and Goldberger[1] that typhus is identical with a disease described by Brill, and so far from being extinct in the United States, as has been supposed, it has been endemic in Brooklyn, Chicago, New York, Philadelphia, and Atlanta in this country during the last two decades.

Pathology.—After death the eruption continues to be visible. Rigor mortis is often delayed.

Certain organs may present pathologic appearances, but they are not constant and are the result of the secondary infection which the typhus invites. The serous membranes are the seat of ecchymoses. There is hyperplasia of the lymph-follicles, but no subsequent ulceration. The muscles are dark and often show hyaline and granular changes; the *heart-muscle* is especially apt to undergo a granular degeneration. The *spleen* is considerably enlarged, soft (even diffluent at times), and of a dark (frequently bluish) red color. The *liver* is somewhat swollen and may be softened; the *kidneys* may manifest the changes belonging to nephritis or mere congestion. In the *lungs* are found a variety of lesions peculiar to different complicating conditions (bronchitis, lobular pneumonia, lobar pneumonia, pulmonary congestion, edema), and occasionally *pleurisy* (serofibrinous or purulent) may be present. *Nervous lesions* are conspicuous by their absence. Commonly there is *cerebral congestion*. *Meningitis*, however, is rare. The *blood-changes* are marked, the color being dark, the fluidity much increased, while the coagulability is greatly diminished.

[1] "Public Health Report," Feb. 2, 1912, p. 149.

Etiology.—The **direct cause** or special micro-organism connected with the typhus contagion has not, as yet, been isolated, notwithstanding the fact that the morphologic and biologic studies of the blood obtained by Brannan and Cheesman from the finger-tips of six patients during the mild epidemic of typhus in 1893 showed the presence of a bacillus that proved pathogenic for rabbits, guinea-pigs, and white mice.[1] Lewaschew[2] has also detected in the blood of typhus patients a distinctive micro-organism.

It is a known fact, nevertheless, that when typhus arises in a locality in which it was previously unknown it is dependent upon a transference of the typhus virus from without, and does not arise spontaneously. The different *modes of conveyance of this poison* are imperfectly known; it may leave the body in the expired air, in the epithelial scales thrown off, and in other excretory or secretory products. The poison is transmitted by *contagion* from the patient to others who approach him; and it may be transferred by means of *fomites* (wearing apparel, articles of furniture, etc.). Littlejohn[3] reports an epidemic, and states that only those in intimate contact with the sick appear to become infected. Wilder's experimental studies and investigations show that three insects are probable conveyors of the infection. These are the flea, the bedbug, and the body louse, especially the latter.

Predisposing Causes.—The influence of *insanitary surroundings* upon the spread of this affection is positive and vital. Among special conditions may be mentioned filth, poverty, famine, and overcrowding, and hence it may be inferred that typhus is a disease of the lower classes. It prevails in jails and camps. Among additional etiologic influences are overwork, intemperance, and depressing emotions.

Age.—The young and middle aged furnish a preponderant proportion of cases, owing to the fact that they are more liable to exposure to the virus than during other periods of life. *Sex* has no influence, and the *season* plays only a minor part. Epidemics, however, occur oftenest in winter, since the homes of the underfed pauper population are illy ventilated, and hence favor concentration of the specific poison. It almost invariably prevails in an epidemic form.

Clinical History.—**Incubation.**—This lasts from nine to twelve days. There may be prodromal symptoms during the concluding days of this period, such as anorexia, general malaise, etc., but in most instances *invasion* is sudden.

Pre-eruptive Stage.—The *early* symptoms are either a series of chills or one severe rigor, accompanied by vertigo, tinnitus, headache, muscular pains, profound prostration, and fever. The *temperature* quickly ascends to a high level, reaching 104° or 105° F. (40° or 40.5° C.) as early as the second or third day. The fever is continuous in type, and in severe cases a serious systemic condition may often be developed. The *pulse* is accelerated proportionately to the temperature and is of good volume. Bronchitis may be present, the appetite is lost, and the thirst is excessive, while a thick, yellowish-white coating covers the tongue. *Vomiting* occurs, and may be a prominent symptom. The *urine* is often scanty, its specific gravity is increased, and it may contain a trace of albumin. The cheeks are flushed and the eyes are injected.

[1] *Annual of the Universal Medical Sciences*, 1893, p. 60, section H.
[2] *Ibid.*, p. 61, section H.
[3] *Public Health*, Sept., 1899.

Nervous symptoms appear early—often at the very onset—and are quite pronounced. At first there may be either mild or active delirium, but soon there is stupor or even coma, and the face takes on a dull, stupid, besotted appearance. The spleen is generally enlarged.

Eruptive Stage.—Between the third and fifth days of the invasion the *characteristic eruption* appears *without an accompanying decline in the temperature.* The *rash* comes out first upon the trunk (chest and abdomen), extending thence over the rest of the body, but, strangely enough, often sparing the face. The crimson-red maculæ are changed in two or three days to a darker hue (petechiæ), and when coalescence occurs we have the spotted effect that has caused the name of *spotted fever* to be given to it. This name is also given to cerebro-spinal meningitis, in which the eruption, however, does not appear at any given time and is extremely inconstant. Not all of the maculæ are converted, but some may remain as rose-spots, particularly in mild forms of typhus, and these disappear when pressed upon, while the petechiæ do not. The skin-surface between the spots is sometimes diffusely hyperemic, and the eruption is usually rather abundant, though in well-authenticated cases it has been scanty or even wholly missing. The skin may also present darker and lighter blotches, producing a mottled appearance. In the stage of eruption the symptoms become aggravated in typical and severe cases. The temperature continues high, often reaching 106° F. (41.1° C.) or even higher, with slight nocturnal remissions. The pulse becomes quite rapid (120–140 or more), feeble, sometimes dichrotic, and irregular, and the respirations increase markedly in frequency. At this time severe bronchitis, leading to *broncho-pneumonia*, is apt to occur as a complication. The *tongue* is brown, fissured, tremulous, and occasionally black and rolled up, without power to protrude from the mouth. Sordes form on the teeth and lips. The *urine* is scanty, high-colored, and often albuminous.

The **nervous disturbance** is intense, and may take the form of typhomania, leading to complete coma or maniacal delirium. The patient often lies with eyes open, staring into space, yet unconscious (*coma-vigil*). The motor nerves show derangement (tremors, subsultus tendinum), and carphologia (picking at the bed-clothes) is a common symptom. The decubitus is dorsal; the flushed cheeks become dusky, the face expressionless, and the pupils often contracted. The prostration reaches an extreme degree, and absolute exhaustion often terminates life.

As a rule, in favorable cases the end of the febrile period comes by *crisis* between the fourteenth and seventeenth days of the disease. Immediately preceding the crisis there is generally a sudden rise of the temperature (*perturbatio critica*), and the decline may be interrupted by slight exacerbations. The occurrence of the crisis is marked by rapid improvement in the symptoms. The mind becomes clear (sometimes following a profound sleep), the eruption fades quickly, the facial phenomena disappear in inverse order of their appearance.

Leading Symptoms and Complications.—Course of the Fever.—Although the temperature rises rapidly on the first day of the illness—the highest grade is usually reached as late as the fifth or sixth day. Hyperpyrexia usually heralds a fatal termination, the temperature mounting to 108°,

109° F. (42.7° C.), or higher, though in light cases the acme may not exceed 103° F. (39.4° C.). The temperature pursues the continued type and ends by crisis. Occasionally the fever declines by rapid lysis. About the beginning of the second week the patient emits a disagreeable odor that is regarded as characteristic by some writers.

The **lungs** frequently present complications (*vide* Pathology), among which are *bronchitis*, *broncho-pneumonia*, and *hypostatic congestion*. Broncho-pneumonia is dangerous, its development often preceding a fatal termination, and it may lead to pulmonary gangrene and empyema. Sero-fibrinous pleurisy and lobar pneumonia also occur as complications, and to recognize the latter the physical signs must be appreciated, the rational symptoms being in abeyance.

The **heart** in typhus continues to grow progressively weaker until, in many cases, a fatal issue is reached. This is manifested by the change in the character of the first sound. A systolic *murmur* (probably of hemic origin) may be audible at the apex.

The **nervous phenomena** have been sufficiently detailed. *Meningitis* has been met with, but is rare as a complication. Arnold[1] noted optic neuritis in 8 out of 14 cases. *Hemorrhagic nephritis* rarely supervenes. During the febrile period the uric acid and urea increase in quantity, while the chlorids decrease.

The **digestive tract** rarely presents distressing symptoms and complications. *Hematemesis* is most common, and *cancrum oris* has been noted occasionally. Cases in which the mouth does not receive proper care are apt to develop *parotitis*, which often passes on to suppuration, and *septic processes*, causing abscesses in different parts of the body (joints, subcutaneous tissue), may arise as complicating events.

Among the **sequelæ**, *neuritis*, followed by *paralyses*, deserves first place, and *gangrene of the extremities* (toes, fingers) has been observed.

The **general course** and **duration** of typhus are variable. There is a *mild type* whose course is run in from seven to ten days, and in such the crisis occurs soon after the appearance of the eruption. In this type the development of serious symptoms or grave complications is the exception. A *malignant type*, however, also occurs (*typhus siderans*), and this often proves fatal before the time for the appearance of the rash.

Some epidemics are characterized by the relative frequency of light forms, and others by the severer types of the disease.

Diagnosis.—On the known presence of an epidemic, the special causative factors (unhygienic surroundings, exposure to the poison) and the course and characteristic symptoms, the diagnosis of typhus fever can be made. Of special value is the eruption—its time of appearance (third to fifth day), mode of distribution, and petechial character. The recognition of lighter types, on the one hand, and malignant, on the other, is not possible from the symptoms alone, but here a knowledge of the existence of an epidemic in the vicinity is often helpful.

Differential Diagnosis.—*Cerebro-spinal meningitis* may be distinguished by a more intense headache, by retraction of the head, hyperesthesia, intolerance of sounds, photophobia, palsies of the eye-muscles (strabismus), a tendency to convulsions, and by both the absence of the typhus eruption and the expressionless countenance. Quincke's lumbar puncture may be practised.

[1] *Wiener Klinische Wochenschr.*, Aug. 17, 1911.

Uremia is excluded by the previous history, the vomiting, headache, convulsions, coma, and by the absence of the high temperature and petechial eruption of typhus. Characteristic urinary phenomena are associated in uremia, and rarely *acute hemorrhagic nephritis.*

The eruption of *malignant measles* may bear a close resemblance to that of typhus; the rash in typhus, however, appears first upon the trunk, that of measles, upon the face. Koplik's spots do not appear in typhus. Points connected with the epidemicity of measles, as the occurrence of mild and typical cases, must be taken into account. *Typhoid fever* is readily differentiated from typhus (*vide* p. 48). Typhus fever must also be distinguished from *Rocky Mountain Spotted Fever* (*vide* p. 327).

Relapses are among the rarest of clinical events, and one attack, as a rule, bestows immunity for life.

Prognosis.—To arrive at a correct prognosis it is necessary to consider (1) the degree of severity of the particular type from which the patient is suffering, (2) the number and character of the complicating conditions present, and (3) circumstances connected with the individual, among which his food-supply and sanitary surroundings are to be recollected. Improved sanitation has reduced both the incidence and mortality-rate, which is now between 10 and 20 per cent.

Treatment.—This embraces, in the main, the same principles that were found to govern the treatment of typhoid fever.

Prophylaxis demands *thorough disinfection* and *absolute isolation.* A special hospital for contagious diseases is always to be preferred to the best accommodations obtainable in private families. When, however, patients cannot be transferred to special hospital wards the sick-room must be kept clean, well ventilated, and at a temperature ranging from 60° to 65° F. (15.5° to 18.3° C.). No one other than the doctor and nurse should be allowed to occupy or even enter the room. The thorough disinfection already described under Typhoid Fever must be enforced, and the importance of supplying fresh air to typhus patients, as emphasized long since by Alonzo Clark, has been abundantly shown by the great reduction of the mortality-rate among those treated in tents as compared with that in the hospital wards.

The **general management,** including the use of stimulants, in this disease does not differ from that advised in typhoid fever. Fresh water should be given freely at regular intervals. *Hydrotherapy* constitutes the best means at our command for controlling the temperature and the nervous symptoms. In addition, the use of antiseptic agents and tonic measures are to be recommended. The fact that typhus is a self-limiting affection gives those measures that are intended to combat exhaustion, and especially heart weakness, first rank in the treatment of this affection. Strychnin (gr. $\frac{1}{40}$—0.0015) and camphor in sterilized oil (gr. ij—0.13); one or both may be given hypodermically every third hour if there be failure of the circulation.

DYSENTERY.

Definition.—An infectious inflammatory disease of the large intestine, characterized anatomically by ulceration of the intestinal mucosa, and clinically by frequent mucous and bloody discharges, tenesmus, fever and prostration becoming profound. It is a truly epidemic disease, yet it also occurs constantly in endemic form, and particularly is this true of temperate climates.

Varieties.—Etiologically considered, two varieties are recognized: (1) bacillary and (2) amebic. Under bacillary dysentery a description of the sporadic form (catarrhal dysentery) will be given.

Historic Note.—Few diseases have been longer known than dysentery, of which we have a description by Hippocrates. Galen localized the chief seat of the affection in the colon, and in 1626, Sennertus defined its sporadic and epidemic character and some of its leading clinical features. To Morgagni belongs the credit of having made the first postmortem anatomic study of the disease. Further and more accurate pathologic contributions were made in the earlier part of the present century by Cruveilhier and Rokitansky, and, more recently still, the whole subject of the morbid anatomy of this disease has been carefully investigated by Virchow, whose results have settled most of the questions connected with the subject. In the United States dysentery has prevailed epidemically upward of a century, the time of greatest prevalence in different districts having been about the middle part of the present century (1847–55). Woodward has given us the only complete record of the various outbreaks in this country, and an account of the ravages of dysentery in both armies during the War of the Rebellion is given in his *Report*, which records 259,071 cases of acute and 28,451 of chronic dysentery. The disease is far less frequent than formerly, owing to the advance made in recent times in sanitary science.

Etiology.—A few general considerations, having reference to the causation of the different forms in common, may be adduced here.

Among **predisposing factors,** *season* heads the list, dysentery being most common in the summer and autumn; great and sudden changes of temperature are more potent than equal changes in humidity. *Climate* has a marked effect, and high temperature must be regarded as a powerful agency, since the disease is much more prevalent in warm than in cold climates, though it is met with in epidemic form as far north as Norway. *Malarial districts* suffer more than non-malarial. *Unhygienic conditions*, as shown by the local epidemic outbreaks in armies, jails, barracks, institutions, etc., predispose to the affection.

Among factors connected with the individual are: (*a*) *Catarrhal* conditions of the intestinal tract, particularly if this be caused by unripe fruit or other unwholesome forms of food; (*b*) *Age*. Although no age enjoys immunity against dysentery, most cases are met with in adults under thirty-five years. *Sex* and *race* are probably without appreciable influence.

(1) Bacillary Dysentery.

(Acute Dysentery.)

This term is appropriately applied to the usual acute epidemic form of the disease. I shall describe here two clinical types: (*a*) catarrhal

dysentery and (*b*) diphtheritic. It is probable, but not proved, that all of the cases of bacillary dysentery are due to a common micro-organism —the Shiga bacillus (*Bacillus dysenteriæ*).

The classification of catarrhal dysentery, therefore, still rests upon its clinical and pathologic manifestations, although many, if not all, of the cases as shown by the observations of Vedder and Duval[1] are etiologically identical with epidemic tropical dysentery. Flexner's statistical studies indicate that the Bacillus dysenteriæ (especially the so-called "Flexner-Harris" type), can be isolated from the intestinal discharges, and the intestinal mucosa of "a large percentage of children suffering from the diarrheal diseases prevailing along the Atlantic sea-board of the United States during the summer months."[2]

(*a*) Catarrhal Dysentery.

(*Sporadic Dysentery.*)

Pathology.—The solitary follicles are affected chiefly, and are the seat of hyperplasia, followed by necrosis, with the formation of small ulcers. This is common in children. There may be a purulent inflammation of the entire mucosa, with more or less erosion of the surface, and superficial ulceration exists. In both forms the lesions are mainly confined to the large intestine, though the ileum is sometimes implicated.

Special Etiology.—The catarrhal form of the disease is the one most commonly met in the United States, and is to be classed with acute dysentery; it may accompany some of the acute infections (scarlatina, malaria, typhoid fever, tuberculosis), and is seen in institutions.

Clinical History.—There may be *prodromes*, lasting one or two days, which take the form of a mild gastro-intestinal disorder (anorexia, slight pains in the abdomen, followed by diarrhea).

The *characteristic symptoms* are mild colicky pains in the abdomen, followed by discharges from the bowel, which at first number from three to six daily. Soon they become frequent and are accompanied by straining and tenesmus, and now their number ranges from ten to no less than one hundred or more per day. Indeed, the desire to go to stool may be almost constant, and the rectum is the seat of intense burning sensations during and after each evacuation of the bowel. The character of the discharges varies with the different periods of the affection. During the first thirty-six or forty-eight hours they are feculent (sometimes scybalous masses), rather copious, and intermingled with some mucus and blood. For the next four or five days the stools are scanty, measuring from 2 drams (8.0) to ½ ounce (16.0), and are made up of a sero-mucous fluid or of a muco-purulent material with blood. The chief constituents of the stools are mucus, blood, and pus, any one of which may preponderate.

Microscopic examination of the usually glairy stools shows red blood-corpuscles, numerous leukocytes, generally large, oval or round epithelioid cells containing fat-globules, vacuoles, and bacteria (especially those connected with putrefaction).

A few shreds (portions of necrosed mucous membrane) may appear

[1] *Jour. Exper. Med.*, Feb. 5, 1902.

[2] *Studies from the Rockefeller Institute for Medical Research.* Reprints, vol. ii., 1904, p. 134.

from time to time in the dejecta. At the close of the first week, and a little later, the discharges become less frequent and the amount of mucus and blood diminishes. The stools are now of a greasy brown or dark-green appearance, fecal matter reappearing in them, and soon they are again fully formed.

Other Symptoms Referable to the Alimentary Tract.—The *tongue* has a greasy coating—moist at first, dry later—and at last may become red and glazed. Anorexia is present, with excessive thirst, and vomiting may rarely occur. There will usually be tenderness over the line of the colon, but there is an absence of tympanites.

The *general symptoms* are well marked only in the severer types. The patient is debilitated, sometimes even collapsed, as shown by the small, frequent pulse, cool skin-surface, the rapid wasting, and weak, hoarse voice. The temperature is not much elevated, though it may touch 103° or 104° F. (39° or 40° C.), and the curve is an irregularly remittent one.

Diagnosis.—This can easily be made upon the intestinal features and from the character of the stools—frequent, small, slimy (or bloody) discharges, accompanied by distressing tenesmus.

Differential Diagnosis.—Symptoms simulating dysentery may appear in the course of certain rectal affections, such as *strangulated hemorrhoids*, *syphilis*, and *epithelioma*. In these conditions there is a different history and the symptoms of proctitis are less acute, while a physical examination of the rectum will settle the diagnosis in doubtful cases.

Prognosis.—The *duration* of mild cases is from eight to ten days, and in severe types from three to four weeks. The *prognosis* varies according to the type of the affection; but commonly this is not aggravated and recovery is to be expected. Occasionally, however, the disease is threatening to life. Serious nervous symptoms (delirium followed by coma) may develop and cause a fatal termination. When death occurs it is usually due to exhaustion, and is seen particularly in persons previously enfeebled by disease or in the very young and the aged. Complications influencing the prognosis are exceptional. This variety probably does not occur in extensive epidemics; but it prevails in tropical and subtropical countries, and also throughout Europe and North America.

(*b*) Diphtheritic Dysentery.

(*Acute Tropical Dysentery.*)

Definition.—An intestinal inflammation (usually colonic), accompanied by a croupous, or true, diphtheritic exudation. It is epidemic in Japan, but prevails wherever large numbers of persons are closely associated, as in armies, asylums for the insane, ships, and the like.

Pathology.—In mild grades a grayish-yellow, croupous exudate appears upon the inflamed mucosa, with a necrosis of the epithelial layer that is often limited to the top surface of the folds of the colon. In other instances the diphtheritic infiltration involves all the layers of the bowel, which now becomes greatly enlarged, its mucous membrane being converted into a yellowish-brown, thick, elastic mass, sometimes extending along the entire length of the large intestine. The changes may be confined to the circumscribed areas (flexures of the colon and

rectum), and thick sloughs may be cast off, leaving behind ulcers of corresponding size and depth. The morbid changes in some cases are principally ulcerative in character, simulating those described under Catarrhal Dysentery (*vide* p. 73). Indeed, the pathologic unity of the various forms of bacillary dysentery would appear to be almost established.

Bacteriology.—The distinctive pathogenic agent is the *Bacillus dysenteriæ* discovered by Shiga[1] during his investigations into Japanese dysentery. Flexner found the same organism. Duval, Harris, and Flexner have described different races of the Bacillus dysenteriæ, showing that decisive criteria of difference are observable, which separate this organism from the Bacillus typhosus. The Bacillus dysenteriæ is not normally found in the intestines. The Shiga bacillus, however, "is inactive to blood-serum from typhoid cases, but reacts with serum from dysenteric cases to which bacillus typhosus does not respond" (Flexner). It may be that a number of bacilli which closely resemble one another, yet different, are capable of causing epidemics of true dysentery. Pfuhl[2] found dysentery bacilli in the intestines of soldiers returned from China one year after the initial attack; this persistence may have a bearing on the geographic distribution of bacillary dysentery and its spread in the United States since the Spanish-American war.

Mode of Conveyance.—Messrs. Ryder, Richards, Peabody, Canavan, and Southard studied an institutional epidemic in which the first case was probably an introduced probable carrier; they believe that the epidemic was due to flies, and that occasional cases of dysentery depend mainly on contact-infection with the products of intramural carriers.

Clinical History.—The affection usually has an *acute onset*, and one characterized by an appearance simultaneously of severe local and general symptoms. There may be an initial *chill*, and there is *fever*, which rises rapidly, together with a marked and early appearing prostration and delirium. The fever-curve is of the irregularly remittent type and its range is somewhat higher than in the catarrhal form of the disease. The pulse is greatly accelerated and tends to become erratic both as to rhythm and volume. Active delirium is common and may alternate with or merge into coma. Severe abdominal *pains* are complained of, and the discharges may be numerous, containing shreds and large sloughs, or even tubular pieces, of false membrane. When these elements are present in the stools, the latter are of a dark-brown color, emitting a fetid odor, and generally containing more or less blood and mucus. The dejecta are more hemorrhagic, as a rule, than in the simple, catarrhal variety. *Tenesmus* may be intense. There is an absence of polynuclear leukocytosis in this disease.

The **physical signs** are often prominent. The belly in most instances is greatly distended, and on pressure very tender—signs due to the fact that the lesions are situated chiefly in the large bowel.

The **diagnosis** rests upon the intestinal symptoms and the character of the dejections, associated with a grave general condition suddenly developed. As accessory factors to the recognition of this variety are the finding of the false membrane in the dejecta, and the appearance of the cases in an epidemic form. An absolute diagnosis demands either the isolation of dysentery bacilli from the dejecta (which, however, are

[1] *Centralbl. f. Bakt. u. Parasitenk.*, 1898, xxiv., Nos. 22–24.
[2] *Münch. med. Wochen.*, Feb. 11, 1902.

rarely present in mild cases and during the first days of the disease) or the agglutination reaction of the blood-serum, and this serves to differentiate bacillary dysentery from allied maladies, including typhoid fever.

Complications.—These are both numerous and varied, and include perforation of the gut followed by peritonitis, either localized or generalized (according to its seat); also pleurisy, endocarditis, pericarditis, parotitis, "anasarca, phlebitis, and nephritis" (Rumford). Hepatic abscess is never observed (Shiga).

The **prognosis** is almost wholly unfavorable. The principal element of danger is the profound toxemia, which rapidly leads to fatal asthenia in cases in which the stools consist of a blackish fluid with a horribly fetid odor and of bits of gangrenous masses (Duncan). Shiga states that the toxemia is most marked in cases in which the lesions are located high up in the intestine, and that the disease is most fatal in winter. The numerous complications also exercise a lethal tendency. Occasionally recovery follows, though more frequently the disease takes on a chronic course.

Secondary Diphtheritic Dysentery.

Here the lesions are similar in kind, but less intense, as a rule, than those of the primary form. This variety is met with as a terminal condition in not a few acute and chronic diseases; it often occurs in pneumonia (Bristowe), and less commonly in typhoid fever. Among chronic affections, upon which this condition may become engrafted, are nephritis, organic disease of the heart, and pulmonary tuberculosis.

No characteristic *symptoms* attend upon its invasion. There may be slight diarrhea—two or four liquid stools daily—but it is not often accompanied by tormina and tenesmus, and the discharges rarely contain any noticeable amount of blood, mucus, or shreds of pseudo-membrane. Secondary diphtheritic dysentery often induces fatal asthenia.

Sequelæ of Bacillary Dysentery.—In all forms a relapse is likely to occur, each attack increasing the liability of the patient to subsequent ones. Moreover, in persons who have recovered from acute dysentery we often observe a disordered digestion and irritability of the bowels. Rarely, chronic nephritis follows dysentery. The most interesting sequel, however, is paralysis, which occurs mainly in the form of paraplegia (S. Weir Mitchell). Stricture of the bowel is rare.

Treatment.—Prophylaxis.—This embraces isolation and a thorough disinfection of the discharges, which contain the specific germ of the disease, as soon as passed. The drinking-water during the epidemic prevalence of dysentery should be thoroughly boiled, and healthy persons should avoid cathartics, the use of improper food, or such as stimulates intestinal peristalsis, while an unhygienic environment (overcrowding, etc.) is to be corrected as far as possible. Shiga recommends that the dead bacillus emulsion (heated at 60° C. for thirty minutes) and a specific immune serum be injected simultaneously. One injection produces active immunity and the author tested the method on about 10,000 men in the district of Japan "where epidemic dysentery prevails most seriously, and was able to diminish the mortality in the district from 20 to 30 per cent. to about zero." All sufferers from dysentery must be kept in bed, and should occupy a well-aired apartment.

The **diet** should consist of milk, whey, and light animal broths during the period of active intestinal symptoms. The blandest articles only

are either acceptable to the stomach or allowable in the diphtheritic variety, as Mellin's food (especially for children), egg-white, and zoolak, in small portions. During convalescence a return to the usual dietary is gradually to be made. All food should be given lukewarm.

Alcoholic Stimulants.—With the development of asthenia and cardiac failure stimulants must be employed, as in other acute infectious diseases. Diphtheritic dysentery calls from the very outset for free stimulation. The diffusible stimulants (*e. g.*, champagne) are often invaluable. Strychnin and digitalis (hypodermatically) may be required.

Medicinal Treatment.—If scybalous masses be passing, a dose of castor oil should be administered. It is well to convert dysentery into diarrhea. Measures to deplete the mucosa of the intestine and at the same time inhibit undue peristalsis are most effective, as magnesium sulphate. Dram doses may be given every hour or two, until the stools contain fecal matter and no more blood or mucus. In the later stages purgatives are attended with baneful effect.

Ipecacuanha has long been, and still is, regarded as possessing a specific influence in cases of dysentery. Its administration is usually preceded by a dose of opium (laudanum or morphin) which is given when the stomach has been empty for a few hours. Most authors recommend that large doses—gr. xx to ʒj (1.29 to 4.0)—should be administered; but it is probable that a small dose—gr. $\frac{1}{6}$ to $\frac{1}{4}$ (0.010 to 0.016) every half hour—is quite as effective; and in children the smaller doses are to be preferred and will be found to be quite efficacious. Other remedies should also be employed, and among these opium is particularly beneficial in combination with ipecacuanha or in the form of Dover's powder, which contains both agencies. Three chief symptomatic indications are met by the opium—pain, restlessness, and undue peristalsis—and to obtain the best effects from the opiate it should be administered in the form of morphin hypodermically. In cases in which tenesmus is an unusually distressing feature an opium suppository (gr. ij—0.1296) or laudanum (♏xxx—2.0, by enema) exercises a beneficial effect. Bismuth in full doses is useful (ʒss–j—2.0–4.0 every two hours), and I have frequently found the combined use of Dover powder, bismuth subnitrate, and salol of signal service. Cunningham, Stengel, and others have reported curative effects from the employment of sulphur; and Richmann prescribes the following powder:

℞. Sulphur sublimat., gr. xviij (1.20);
Pulv. Doveri, gr. v (0.33).
M. ft. chart. No. i.
S. To be taken every fourth hour.

Antiseptic substances by the mouth for the purpose of disinfecting the intestinal canal and favoring the healing of the ulcerated surfaces after the removal of the necrotic pseudo-membrane, such as benzonaphtol (gr. xl–lx—2.592–3.788—in the twenty-four hours in divided doses), salol, opium, and silver nitrate are among the remedies of choice. The naphtol preparations being insoluble should be given in capsule and the silver nitrate in pill form one hour after food. Iodoform in a pill or capsule in doses of $\frac{1}{2}$ to 3 grains (.032 to .194 gm.) has been much lauded. Bose and Vedel employed in 4 cases intravenous injections of sodium chlorid, 7:1000 being the maximum strength.

The injections should be made early, and repeated, so that they will develop sustained general reaction and a modification of the general condition which can lead to recovery. Care should be taken as to the quantity used and the rapidity with which it is injected (½ to 3 ounces each minute should not be exceeded). Kendall advises dextrose infusions (25 per cent.) in normal saline solution; this tends to restore the normal dextrose.

Antiseptic irrigation of the bowel would be, if properly carried out, a curative measure, since by this means we may destroy the distinct micro-organisms. Unfortunately, the bowel is frequently so irritable as to seriously interfere with this mode of medication. Preliminary to their use we may also employ cocain in the form of a suppository, or a small quantity of a solution of cocain (4 per cent.), or a laudanum enema (ℳxxx—2.0, in starch-water), after which a large injection may be tolerated if administered slowly and the flow be interrupted at intervals. Among the best agents are silver nitrate (gr. ss–j—0.032–0.064—ad ℥j—32.0), tannic acid (1 to 2 per cent.), salicylic acid (1 to 2 per cent.), and mercuric chlorid (1 : 6000). I have for a number of years been in the habit of employing these astringent and antiseptic solutions alternately, administering each once daily. Kuzmitzky,[1] MacDonald, and others have obtained good results with rectal injections of a tepid solution of potassium permanganate (1 : 4000) twice daily. The temperature of the water should, at first, range from 100° to 110° F. (37.7° to 43.3° C.), and subsequently this may be reduced. The patient during the administration of the enemata should assume the dorsal position, with the hips well elevated, and he should be turned from side to side during the injections. The existence of great irritability of the bowel may be met by using two catheters side by side, one of them serving as an outflow. Kruse[2] has produced a serum and has treated 100 cases with 8 deaths. Shiga has also discovered a serum of which he injects one dose of 10 c.c. in mild cases. In cases of medium severity, a second dose of 10 c.c. is injected after from six to ten hours, while in severe cases, a daily dose of 20 c.c. is repeated for two or three days. The mortality of dysentery under the use of this serum is reduced to less than one-half from that obtained from medical treatment.

Local means, in the form of hot fomentations, light poultices, and turpentine stupes, often afford much comfort. The various complications must be met by appropriate treatment, as under other circumstances.

Chronic Dysentery.

This form of the disease almost always succeeds an acute attack. A. Bassler thinks that "chronic dysentery due to the *Bacillus coli communis*" seems warranted.

Pathology.—In most instances the large intestine is still the seat of ulceration. Some of the ulcers show no signs of healing; in others this process is going on; while in still others it is completed and puckered cicatrices are presented. The ulcers are deeply pigmented, as is the unulcerated mucosa, which often presents a slate-gray or blackish color. The submucous and muscular coats are hypertrophied, as a rule, with occasional narrowing of the lumen of the bowel, and cystic degeneration of the intestinal glands is sometimes observed. In a small percentage

[1] *Woenno. Med. Jour.*, Nov., 1902. [2] *Deutsch. Med. Woch.*, Jan. 1 and 15, 1903.

of the cases ulceration does not occur, the mucosa presenting an uneven, puckered aspect, due to deposits of fibrous tissue.

Symptoms and Diagnosis.—Many of the most characteristic features of the acute form are either but feebly expressed or altogether wanting. This is particularly true of the tormina and tenesmus. Certain elements found in the stools of the acute type (blood, shreds of pseudo-membrane, and tissue) are also rarely present. True *dysenteric symptoms*, however, may arise during acute exacerbations, with or without pain or tenesmus; then from three or four to a dozen or more fluid *dejections* are passed daily. The latter are often frothy (when starchy articles of food are taken), composed chiefly of fecal matter and undigested particles of food and mucus; and in severe forms blood and pus may be constantly present in the discharges. In many cases the stools are semifluid (pultaceous), and rarely they contain scybala; or the rather frequent liquid or semifluid discharges may alternate with constipation. The lesions are then apt to be situated in the lowest portion of the large intestine. The *character* of the discharges is much influenced by the sort of food taken; thus, when a mixed dietary is partaken of, they are thin, more frequent, and contain more undigested masses of food. *Gaseous distention* of the intestines is often an annoying symptom.

The **physical signs** are negative, save only tenderness over the colon.

Associated symptoms referable to other organs are not without value in the diagnosis. The gastric digestion is poor, the appetite generally impaired (though variable), and the *tongue* is clean, red, and glazed, presenting the appearance of raw beef. There are progressive *emaciation* and *asthenia*, which eventually reach an extreme degree. The skin-surface becomes dry, harsh, and cool, the facies grim, the pulse exceedingly feeble, the mental faculties greatly weakened in the advanced stage; and, as in the acute form so in the chronic, death is usually due to asthenia—with this difference, that in the latter the end is reached more slowly. Peritonitis in consequence of perforation is rare.

Differential Diagnosis.—The disease is to be discriminated from *chronic diarrhea*. In chronic dysentery there is the history of an antecedent acute attack, with the appearance from time to time of exacerbating periods when mucus, pus, and often blood are contained in the discharges. The latter are, at the same time, more frequent and apt to be accompanied by more or less abdominal pain and tenesmus, and the presence of these features would serve to eliminate chronic diarrhea. From *tuberculous ulceration* of the intestines it is distinguished by the absence of any history of tuberculosis, family or personal, and of tuberculous new growths in other portions of the body, particularly the lungs.

The **complications** are the same as in acute dysentery, if we except the greater liability, due to the great and protracted weakness of the patient, to certain serious intervening diseases (chronic nephritis, tuberculosis, pneumonia). Ulceration of the cornea has been noted.

The **duration** is long, the disease lasting for months or even years.

Treatment.—This should be directed mainly to the local condition, and should consist in methodic irrigation of the bowel with a view to promoting the healing of the ulcers. Formerly it was sought to accomplish the latter indication by the use of certain remedies internally, as silver nitrate, balsam of copaiba, bismuth subnitrate, etc., but the only preparation which I have found useful is the zinc oxid (gr. v–x—0.324–

0.648) three times daily. The latter preparation is decidedly palliative, sometimes even curative.

Intestinal irrigation is to be tried, and various disinfectants and astringent remedies should be alternated as advocated in the acute form. Among individual remedies the silver nitrate (gr. ss–ij—0.032–0.129—ad ℥j—32.0) every second day is doubtless the best. On intervening days antiseptic remedies may be used in solution, such as mercuric chlorid (1 : 6000) or salicylic acid (1 to 2 per cent.); and of other useful agents I may mention tannic acid, alum, acetate of lead, and creolin.

Prior to the use of any of the above-mentioned enemata the bowels should be well flushed with a large injection of tepid water, so as to remove the fecal and other irritating materials. The same details are to be observed in carrying out this mode of treatment as in the acute forms of dysentery. Gallay[1] has related the curative effects of large enemata of a solution of crystallized silver nitrate in distilled water, a scruple to a quart (1.296 per liter), to which 20 or 30 drops of laudanum have been added. Amelioration follows the third or fourth washing, but a course of sixty is recommended to secure permanent relief. The lower part of the rectum should be examined with the speculum, and appropriate topical applications made if ulcers in this situation be discovered. It has been suggested that topical therapy can be facilitated in chronic cases by the production of an artificial anus, in the left iliac region, or an appendicostomy, but the value of the method is still doubtful.

The *dietetic* treatment in chronic dysentery is of the utmost importance, and light forms of proteids are to be selected, to the exclusion of vegetable substances. Milk is excellent when it can be taken. It is well to examine the stools, and if on microscopic examination curds or numerous fat-globules appear, the amount of milk should be reduced or skim-milk substituted. Egg-white, meat-broths or beef-juice, whey, and even light, nutritious solids may be allowed. The patient should wear flannels next the skin, and, while open-air exercise is useful, it should be moderate. During inclement weather the patient should remain in-doors. I have known change of climate, with proper regulation of the mode of living, to be productive of rather brilliant results. Tonics and alcoholic stimulants are sometimes required to assist the appetite, digestion, and systemic strength, and among the most efficacious tonic remedies are iron, strychnin, mineral acids, and arsenic.

CHOLERA (EPIDEMIC).

(*Asiatic Cholera; Cholera Algida, etc.*)

Definition.—Cholera is an acute, infectious, epidemic disease, due to the spirillum of Koch (vibrio choleræ Asiaticæ); and its characteristic symptoms are copious watery dejections, painful cramps, collapse, and suppression of the excretions. In some localities it is endemic.

Historic Note.—During the Middle Ages cholera made deplorable ravages, chiefly along the belts of the Ganges, and has probably been endemic in India for centuries. Only during the present century,

[1] "Radical Cure for Chronic Dysentery of Recurrent Type," *British Med. Journal*, No. 1779, p. 276.

however, has the disease been widely known in Europe and America, and when it has appeared it has always been in the epidemic form. The march of epidemics has been from east to west, along the lines of commerce and travel by land or sea, sometimes spreading over the entire globe. Space forbids an account of the progress of the various cholera outbreaks in Europe and America. It may be stated that there have been no distinct epidemic visitations in America since 1873. In India, Mecca, Java, China, and in the Philippine Islands numerous cases appeared during the winter, spring, and summer of 1902.

Pathology.—The body is much emaciated, the features sharp and drawn, and the skin of the dependent parts presents a mottled appearance. A post-mortem rise of temperature often occurs. The tissues are dry, owing to the draining of the liquids of the body, and hence putrefaction is delayed. Rigor mortis comes on directly after death, is persistent, and the muscles often contract so as to cause the body to assume various uncommon positions.

The Visceral Lesions.—The chief of these are confined to the intestinal canal, and depend largely upon the period of the disease at which death occurs. In the early stage the serosa of the small bowel is congested, presenting a roseate hue. The muscularis is relaxed. The mucosa is the seat of catarrh, being deeply injected, swollen, at times edematous, and often coated in the early stage with more or less tough mucus. Shortly the coils of intestine are filled with an almost transparent or slightly turbid liquid ("rice-water"), and occasionally a small amount of clotted blood is seen. The solitary follicles and Peyer's patches are swollen, and, in rare instances, become ulcerated. Denudation of the epithelial lining—most probably a post-mortem change—is the rule, and ecchymotic spots are visible in the intestinal mucosa. If the patient has died late in the disease (stage of reaction), patches of false membrane may be found anywhere along the intestinal canal, although chiefly in the large bowel; and this secondary croupous-diphtheritic process may attack other mucous surfaces (bile-ducts, vagina).

The *stomach* shows changes similar to those found in the intestines. At first the mucosa is congested; then, as the result of transudation, it becomes filled with "rice-water" material. At last the organ is empty and collapsed. The *esophagus* also exhibits analogous lesions.

The *spleen* is small, as a rule, though if death occur late it may show some degree of enlargement with softening.

The *liver* presents marked passive hyperemia and cloudy swelling, with minute spots of beginning fatty change. Desquamation of the epithelium of the cystic mucosa may occur and block the bile-ducts.

The *kidneys* show important lesions, being enlarged from passive congestion, especially the cortex, and the capsule being somewhat adherent. They exhibit cloudy swelling and decided coagulation-necrosis. Desquamation of the epithelium in the uriniferous tubules is extensive. Microscopically, the histologic changes are those of acute nephritis in the cases in which death takes place in the advanced stage. The *bladder-changes* differ in no way from those of other mucous membranes. Its mucosa is congested, ecchymotic, and may show diphtheritic deposit. The ureters and the pelves of the kidneys may present identical appearances.

The Circulatory System.—The pericardium is dry, the parietal layer being covered with an adhesive secretion, while the visceral layer is the

seat of ecchymosis. The *heart* is dry and anemic looking. The left ventricle is contracted, while the right is often distended with blood and soft clots. Outside of the heart the veins, including the cerebral sinuses, contain most of the blood. The blood is thick and its color dark, resembling "the juice of huckleberries"; its specific gravity, albumin, and corpuscles are all increased, while its coagulability is decreased.

Respiratory Organs.—The larynx, trachea, and bronchi are hyperemic, and at first covered with tenacious mucus; later they may present ecchymoses and diphtheritic processes.

When death occurs before the stage of reaction the lungs are bloodless, collapsed, and the mouth of the pulmonary artery may be distended. If life is prolonged until the third stage, the lungs may show congestion and edema or pulmonary infarction. The post-mortem may now also exhibit the lesions of broncho- or lobar pneumonia.

The **brain** and its membranes may be the seat of hyperemia, except when death takes place at a late period, and then the brain-substance may be more or less bloodless and edematous.

Etiology.—The causes are (*a*) *specific* and (*b*) *predisposing*.

(*a*) The **specific cause** is the spirillum of Koch, which is found in the intestinal canal of persons ill of cholera. Recent investigations into the bacteriology of the affection show that almost uniformly the cholera vibrio is associated with certain bacteria, most commonly the bacillus coli communis. True cholera is a *nitrite*-poisoning, the result of the growth of the specific spirillum. Koch's organism is not found in any other disease. Its form is that of a slightly curved rod, and its length about half that of the tubercle bacillus, but it is thicker and sometimes has the form of the letter S (Fig. 8). The cholera vibrio is motile, its motility being due to a single flagellum attached to one end. It has been grown successfully on media of various sorts (*e. g.* nutrient gelatine, forming colorless colonies and liquefying the gelatine) and equally successfully inoculated upon inferior animals.

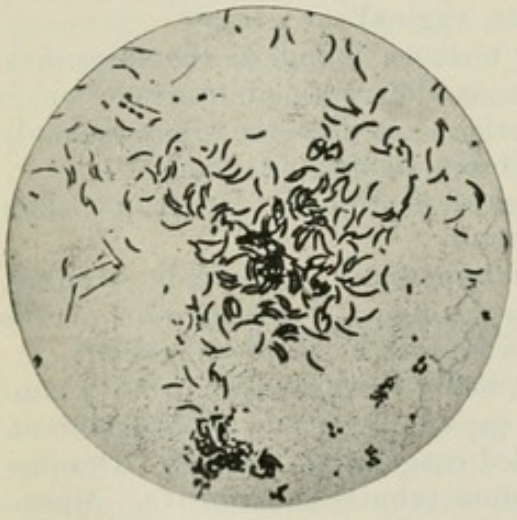


FIG. 8.—Comma bacilli (from the mouth); × 1000 (Günther).

The organism is found in a variety of positions—in the intestine, the dejecta (even quite early), and in great profusion in the pathognomonic "rice-water" stools. Kemp in his review has shown that the spirillum is often absent from the evacuations, and that in these cases the bacterium coli is usually present and sometimes streptococci. He believes, however, that the apparent absence is due to faulty technique. It may be seen in the stools and vomitus (rare) of well persons during epidemics, displaying virulent properties.

Outside the body they preserve their vitality in river or well water or upon the surface of moist linen for several weeks. C. Fränkel studied them in flowing water, and in other epidemic outbreaks they have been found in the water used for drinking purposes.

(*b*) **Predisposing Causes.**—(1) **Locality.**—Near to the sea-coast cholera is more common than in the inland districts or towns, and the frequency

of occurrence lessens with increasing altitude, this fact possibly being due to a gradual decrease in soil humidity and porosity.

(2) **Atmospheric Temperature.**—The spirillum of cholera can only flourish in a warm climate; hence the disease is *endemic* in certain tropical and subtropical climates; and hence also its *epidemic prevalence* is confined to temperate latitudes.

(3) **Seasons.**—For obvious reasons it is more common in the warm than in the cold months, most epidemics, both in Europe and America, having occurred toward the close of summer and in the early autumn. Winter frosts usually arrest an epidemic.

(4) **Age,** as a rule, has no decided effect. Old people, however, are very prone to the affection. **Sex** is without perceptible influence.

(5) **Debilitating Causes.**—Whenever the private conditions correspond to rigid scientific requirements during epidemic outbreaks cholera becomes less prevalent and also less virulent. On the other hand, defective municipal sanitation, disregard of proper hygienic rules, intemperance, overcrowding, etc., all predispose markedly to the disease.

(6) Mere attacks of **intestinal disorder** due to improper diet, cold, etc., are potent to disseminate the disease.

Modes of Infection.—The spirilla leave the body with the stools, but the most frequent bearer of cholera-poison is the drinking-water. Naturally, the individual susceptibility varies greatly (many persons being even insusceptible), and yet the degree of contamination of the drinking-water and the virulence of epidemics are almost strictly proportionate. As an illustration, Vienna had enjoyed exemption from cholera for nineteen years—a fact attributed to the excellent quality of the drinking-water and to hygienic improvements. In the same city the mortality-rate in the more recent epidemics has been small (7 per 1000) for a like reason. On the other hand, in 1872 there occurred in a single commune (Hamburg), which had a polluted water-supply (the Elbe) and no filtration plant, 17,862 cases, with the enormous death-rate of 42.3 per cent. Koch holds that man, not noticeably diseased, is the real bearer and reproducer of the cholera vibrios.

The *choleraic poison* may be conveyed with the water used for washing, cooking, and other purposes to other fluids imbibed by man (beer, milk, tea), and also to food-stuffs taken by him (lettuce, cresses, and other raw vegetables, fruits, meats, bread, butter). The organisms live and maintain their virulence on these articles of food from four to seven days at least. The infection may reach the esophagus with the water used for washing the mouth or teeth, or that used for washing the utensils, dishes, food-receptacles, etc. Again, the hands, commonly those of laundresses and nurses, may become soiled in the careless handling of bed-linen or garments worn by cholera patients or the stools, and convey the poison to the mouth or lips, to be carried into the stomach along with the drink or food. Healthy bacilli-carriers have been found in ships arriving from Mediterranean ports. Flies may transfer the infectious element to food-articles (Simmonds, MacKaig, and others).

Cholera is not contagious from mere contact with those ill of the disease. It is not acquired by inhalation (Shakespeare), and, since desiccation rapidly kills the organism, there is little probability that the latter is airborne. Nor is there any clinical evidence to show that the poison may

enter the system through the skin surface. Probably the germs are *swallowed*, and the acid gastric juice may then destroy them if the size of the dose of the poison is not too large, or they may pass into the intestinal canal and there manifest pathogenic powers. After the spirillum reaches the intestine, whether or not an attack is the result depends both upon the size of the poisonous dose and upon the personal degree of immunity.

Opposed to the drinking-water theory of this disease is that of Pettenkofer, which contends that the spirilla found in the serous evacuations of cholera patients must enter an appropriate soil and there undergo further development before becoming pathogenic. While soils possessing a certain degree of moisture and perviousness and contaminated with organic matter favor the growth and multiplication of the specific organism, these telluric conditions are not essential, as is shown by the virulence of the stools when swallowed in ample quantity. Pettenkofer and Rubino[1] claim that the fully developed poison rises from the subsoil into the lower atmospheric strata as a miasm.

Immunity is not conferred by a previous attack of cholera. Pfeiffer and Marx have proved the existence in the blood-serum of human beings of bactericidal bodies (not a true antitoxin) that cause rapid destruction of the cholera bacilli. To these anti-bodies is ascribed the "Pfeiffer serum reaction," by means of which the vibrios are differentiated from other micro-organisms. Pfeiffer and Marx have also shown that the virus of cholera can be effectively preserved by a 0.5 per cent. solution of carbolic acid, and that it in no way impairs its immunizing properties.

Clinical History.—The **incubation period** varies from a few hours to five days (average two to three days). During this prodromal period the patient is either quite well or (during the latter portion) exhibits certain *local* symptoms. These are occasionally nausea, a feeling of distress in the abdomen, increased peristalsis which may be visible or palpable, slight pain and tenderness, and either a mild or a decided diarrhea. The discharges are feculent, colored, and semifluid, or, more rarely, quite fluid, and may be quite copious. These symptoms may all be present, though oftener a few, and rarely a single one, is noted; moreover, they are not distinctive unless seen during an epidemic and unless the patients have been exposed to the poison. *Prostration* may be marked and there may be slight muscular cramps. The so-called *premonitory diarrhea* may terminate in recovery at the end of from one to three days, or be followed by an attack of cholera. This has three stages.

(1) **Stage of Serous Diarrhea.**—The *dejecta* are generally painless, very frequent, odorless, copious, and fluid or watery, and usually present the characteristic "rice-water" appearance. Rarely they are distinctly colored with bile, and in severe cases with blood, and rarely also are they frothy. Suspended in them are numerous small, whitish, mucous flakes; their reaction is neutral or alkaline, and they contain a small percentage of solid constituents made up largely of albumin and sodium chlorid. The microscope brings to view epithelium, mucus, triple phosphates, and numberless micro-organisms, of which latter the only ones characteristic are the comma-spirilla of Koch. In *cholera sicca* these serous evacuations are absent. Death comes quickly, and post-mortem examinations show the intestine to be filled with "rice-water" material, which is probably retained because of speedy paralysis of the musculature.

[1] *Sajous's Annual*, 1899, vol. ii., p. 214.

Gastric symptoms appear early. Vomiting soon becomes frequent, and at first the vomitus may be bilious; later it is characteristically serous and excessive in amount. *Thirst* is almost intolerable, anorexia is complete, and the tongue often has a thick coating, which early becomes dry. Gastro-intestinal *pain* is not severe, but a feeling of pressure or burning in the abdomen is experienced, and occasionally there are griping pains with tenesmus. The *physical signs* are few. The belly is usually flat and flaccid, though it may be scaphoid and hard, and in some cases palpation detects fluctuation.

Painful cramps in the *muscles* form an early characteristic symptom. They affect the voluntary muscles of the legs, calves, and feet, more rarely the arms and hands also. Their duration is momentary, but they recur at intervals, and are due to the local action of the toxins.

Owing to the withdrawal of fluid from the lymphatics and blood-vessels the tissues become dry and shrivelled and the blood much thicker. This condition of the blood obviously increases the labor of the heart, which beats rapidly, and there may be at first a distressing palpitation; but soon the heart grows more and more feeble and venous stasis ensues. The *pulse* is at first rapid, soft, and small; it may then be lost at the wrist. The cardiac impulse and heart-sounds may at last disappear.

The *facies* and *general appearance* also indicate loss of fluid. The cutaneous surfaces of the face and extremities grow cool: there is rapid general emaciation, which may become most pronounced, and the skin is wrinkled. The complexion assumes a livid or blue-gray tint, while the lips become quite dark. The extremities are cyanotic (the finger-tips in particular), the orbits are deeply sunken, the cheeks hollow, the features intensely pinched, the voice husky and feeble, and there is utter prostration. The *surface-temperature* drops below the normal, even to 96° or 95° F. (35.5°–35° C.), while, *per contra*, the internal or rectal temperature rises to 102° F. (38.8° C.) or over. The *mind* may remain clear until the close, but oftener the patient is apathetic, and in grave cases this condition may deepen into stupor or even actual coma. The *reflexes* are greatly diminished. S. Rogers[1] found a variable degree of leukocytosis, and the large mononuclear cells were usually increased—an important diagnostic sign.

The *urine* becomes very scanty and is highly concentrated, the standing specimen depositing a heavy sediment. On analysis albumin and casts (chiefly granular) are found. In the serious forms the kidneys fail to eliminate the urea, and there is finally complete anuria.

(2) **Stage of Algidity or Collapse.**—The symptoms which characterize this grave condition are the same as those noted under the latter part of the first stage, only intensified. *Asthenia* is extreme; the *pulse* is missing and the heart beats faintly; the *voice* is lost; *respirations* are perceptibly shallow; lividity is intense; the surface ice-cold; and there is usually *stupor* or even *coma*. The excessive *serous discharges* have given place to mere dribblings from the now relaxed anus. During this stage, which may last a few or many hours, the faint glimmerings of the vital spark are often extinguished.

(3) **Stage of Reaction.**—This sets in promptly, and the case may pursue a favorable course, with return to accustomed health by the end of a week or ten days. The first urine passed is usually albuminous and contains tube-casts. *Relapses* into the stage of collapse may occur

[1] *Brit. Med. Jour.*, July 12, 1902.

and be repeated; in many instances, however, this stage is both protracted and dangerous. It is aptly termed *cholera typhoid*, since a genuine typhoid state develops. The *skin* may present so-called choleraic eruptions (macular, roseolar erythema). Recovery may now take place, or a great diversity of local secondary inflammation may supervene.

Acute nephritis may arise in this stage and lead either slowly or directly to uremic poisoning, as shown by the projection upon the scene of grave nervous phenomena—headache, vomiting, delirium or coma, and convulsions. A fatal result may be looked for.

Complications.—In this place are to be enumerated the conditions due to secondary infection, including (commonly) septic and pyemic processes. Diphtheritic inflammations affecting mucous surfaces, but especially the throat, colon, and the external genitals, are among the more common. Bronchitis, pneumonia, and pleurisy may arise, and erysipelas and parotitis are not rare. During *convalescence* digestive disorders may show themselves, and indiscretions in diet may precipitate a relapse.

Clinical Types.—(*a*) **"Premonitory Diarrhea."**—This type has been outlined with sufficient fulness in the foregoing discussion.

(*b*) **"Cholerine,"** in which the symptoms are mild, resembling those of cholera nostras. Many of the symptoms characteristic of true cholera are also present, particularly the *cramps* and *prostration*, cold extremities, and scanty albuminous urine. The stools, however, are not typical of the disease, but are feculent in character, as in ordinary cholera morbus. The *duration* is from seven to ten days, subject to relapses.

(*c*) The more typical forms—both moderate and severe—have been described under the Clinical History (*supra*).

(*d*) **The Foudroyant or Asphyxic Form.**—This may kill instantly; more frequently the patient lives for a few hours, with or without vomiting and purging. *Cholera sicca* should be classed with this type. The virulence of the cholera-poison explains the intensity of the symptoms.

Differential Diagnosis.—This is difficult in the absence of an epidemic unless bacteriologic and microscopic tests be made, and yet these alone differentiate a sporadic case. The disease most commonly mistaken for cholera (especially cholerine) is *cholera morbus*, and the following points pertaining to the latter disease will eliminate it: 1. No connection with a previous case, but a frequent history of dietetic imprudence. 2. Absence of "rice-water" stools, which remain turbid with feces or covered with bile or blood. 3. Presence of colicky pains, but absence of painful tonic cramps of legs and feet. 4. Absence of cyanosis and collapse, as a rule, and of urinary suppression. 5. No cholera spirilla in the stools.

Arsenic-poisoning and other forms of *gastro-enteritis* must be discriminated by the history, the character of the stools, the absence of violent muscle-cramps and of the effects of great loss of fluid (cyanosis, shrunken body, profound collapse). Chemical tests are not to be neglected.

Prognosis.—This is dependent mainly on the type. Thus "cholerine" is very rarely fatal. It is impossible to state the average mortality, since it varies with each epidemic, but it has been found to range from 20 to 80 per cent. Many sufferers perish during the latter part of the first day or during the algid period; still more during the stage of reaction, the dangers of the latter period being as follows: asthenia,

cholera, nephritis with uremia, and the various complications (*vide supra*). The greater the difference between the surface temperature and that of the rectum, the more unfavorable the prognosis. The personal circumstances which render an attack grave are old age, alcoholism, previous ill-health, and debility. On the other hand, the death-rate may readily be lowered by prompt and judicious treatment.

Treatment.—Prophylaxis.—It has been owing in great measure to the efficient quarantine system of the United States that cholera has not gained a foothold on our shores since 1873.

Individual Prophylaxis.—In the first place, those nursing the sick can prevent the spread of cholera by prompt and thorough disinfection. The dejecta may be disinfected by pouring upon and mixing with them an equal part of a 5 per cent. solution of carbolic acid or an equal volume of a freshly prepared solution of chlorid of lime. The discharges thus treated must be covered and allowed to stand from fifteen minutes to half an hour, and then emptied into a pit in the earth containing quicklime, with which they should also be covered. It is of the utmost importance to guard against a pollution of the water-supply by these pits. Soiled clothing, linen, and the like should be promptly disinfected, and bedding had better be burned; none but the attendants should be permitted to enter the sick-room. The dishes used should be disinfected immediately after use or before leaving the sick-chamber. After handling the patient or anything that he has soiled the attendants should first disinfect and then carefully wash their hands. After vomiting and after an evacuation of the bowels the mouth and the parts around the anus should be wiped with a cloth wet with a solution (1 : 2000) of mercuric chlorid. The internal use of sulphuric acid is an important prophylactic. If convalescence supervene, the patient should be kept isolated for a week and the stools disinfected during that time. For the treatment of carriers an occasional course of calomel, with gallic acid in small doses during the intervals, are measures to be recommended.

Persons exposed should use boiled milk and water only. Certain forms of food must be avoided, especially salads and unripe fruits; also alcoholic stimulants. All uncooked food may be pernicious. Such persons should lead regular lives, avoiding fatigue, excesses, etc., and intestinal disturbance must be met speedily by the use of antiseptics, opiates, and astringents. In India, Haffkine[1] has used a protective virus with encouraging results. Thus, "of 1735 persons not inoculated in a certain section, 174 took the disease and 113 died, whereas of 500 inoculated but 21 were affected and 19 died." He has made, altogether, 70,000 injections in 40,000 patients without a single accident, and claims that the results have been entirely favorable. Klein concludes against Haffkine's anticholera inoculations, which, however, produce a temporary active immunity. Pfeiffer and Kolle's method, the injection of dead cholera vibrios, is to be preferred. Immunity as the result of vaccination is to be advised in countries where cholera is endemic and from time to time epidemic—*e. g.*, India. Kraus has obtained a specific toxin and antitoxin, but their practical value remains to be determined.

Treatment of the Attack.—(*a*) **Premonitory Diarrhea.**—In the instances which are not preceded by premonitory diarrhea opportunity to prevent the attacks does not present itself. To dispel the organisms from

[1] *Münch. med. Woch.*, Jan. 29, 1895.

the intestinal canal, castor oil and especially a course of calomel have been used. In this stage a double indication is presented—"to restrain the development of the bacilli in the intestine and to neutralize the cholera-poison." To meet this Cantani proposes tannic acid by irrigation (enteroclysis). He injects into the intestine ½ to 2½ quarts (liters) of water, or infusion of chamomile containing ʒiss to ʒv (6.0 to 20.0) of tannic acid, gtt. xx to xxx (1.20) of laudanum, and ʒv–xij (20.0–50.0) of gum arabic at a temperature of 80° F. Injections should be repeated four times a day, and in grave cases after each evacuation.[1] For the same purpose acetate of lead and opium, or large doses of bismuth, with or without Dover's powder, have been much employed.

(*b*) **Stage of Serous Diarrhea.**—The chief indication is to restore to the blood the watery elements withdrawn by the diarrhea. Not a moment is to be wasted. Opium, and preferably the salts of morphin, may be administered hypodermically (gr. ¼ to ⅓—0.0162–0.0216), if the evacuations prove too exhausting. Cantani advocates the injection of an artificial serum (*hypodermoclysis*) containing 1 dram (4.0) of sodium

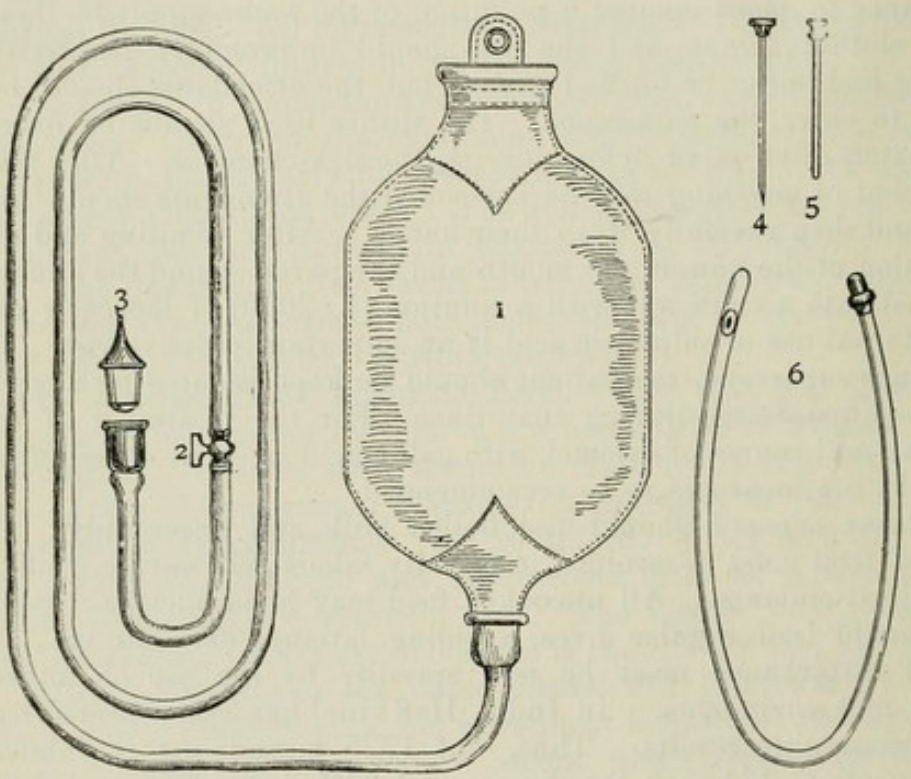


FIG. 9.—1, fountain syringe; 2, cock; 3, attachment for cannula; 4, needle; 5, cannula; 6, soft-rubber rectal tube, with two lateral openings, one a half inch from the end (not visible), the other two inches from the end. The latter is to be introduced by a combined rotatory and pushing motion to the depth of ten inches in enteroclysis, and the fluid then allowed to enter the colon slowly.

chlorid and gr. xlvj (3.0) of sodium carbonate per quart (liter) of sterilized water warmed up to 104° F. (40° C.) into the subcutaneous connective tissue. This solution may be introduced through the cannula of an ordinary aspirator, the fluid flowing by gentle pressure. Shakespeare recommends for hypodermoclysis a fountain syringe with a long flexible tube furnished with a cock; with another shorter tube, one end attached to the cock, the other having a needle-pointed cannula, a little longer, stronger, and with a somewhat wider caliber than the ordinary hypodermic needle (Fig. 9). The tube and cannula are first perfectly filled with a fluid, and then the cannula is inserted well in between the skin

[1] *Annual of the Universal Medical Sciences*, 1893.

and deep fascia of the flanks, buttocks, or interscapular region. The fluid should be made to flow slowly, allowing fifteen to twenty minutes for the introduction of 1 quart. This is preferred to intravenous injection, in which the liquid is diffused slowly.

The *vomiting* is to be relieved by bits of ice, small amounts of brandy and water at brief intervals, cocain, or by *lavage*. In this stage remedies by the mouth should be avoided, since they aggravate the gastric disturbance. Thébaud has treated 8 cases of cholera in Indo-China with a 3 : 1000 solution of sodium bicarbonate, to drink freely, up to 3 quarts a day. Heat should be applied externally with a view to assisting the peripheral circulation, and thus obviating collapse. Warm baths have been recommended for this purpose. Stimulants must be used to fulfil the same indications. They are of superior value even to the above-mentioned measures, and are to be given hypodermically, and either brandy, ammonia, or strychnin may be employed in large doses.

(*c*) **Stage of Algidity.**—If this develop, the case is desperate. The treatment of the preceding stage is to be persevered with, and enteroclysis and hypodermoclysis, hypodermic stimulation, and the external application of heat (*e. g.*, warm baths, to which from 200 to 300 grams of mustard have been added) are especially indicated. Additionally, intravenous injections of fluids have been strongly urged by informed observers. For this purpose the following standard of saline fluid may be chosen : sodium bicarbonate 1 part, sodium chlorid 6 parts, boiled water 1000 parts. The temperature of the fluid when injected varies from 100$\frac{2}{5}$° to 104° F. (38° to 40° C.), more frequently the latter (Shakespeare). The quantity demanded may be 1 or 2 quarts (liters), and the injection may need to be repeated in from one to three or four hours. Strychnin, hypodermatically, should also be used.

(*d*) **Stage of Reaction.**—During this stage the tannic acid may be replaced by a solution of salt in water (10 or 15 per cent.) for enteroclysis (Cantani), and it may be well to continue hypodermoclysis in some instances. Further than this, the treatment is essentially symptomatic. Food of the blandest sort and in small quantities must be allowed at frequent intervals if we would avoid enteritis and other unfavorable complications. Tonic remedies should be given cautiously, and rest and careful nursing insisted upon. Complications must be met in accordance with general principles.

Reference should be made here to the antitoxin and the vaccine that are being used by the Japanese in treating this disease. Schurupow's serum has given good results on therapeutic trial.

YELLOW FEVER.

(*Febris flava; Gelfieber, Ger.*)

Definition.—Yellow fever is an acute, highly infectious (but non-contagious) endemic and epidemic disease. It is characterized by a sharp period of invasion, followed by a period of remission, and the latter in turn by a relapse and certain symptoms peculiar to the affection (black vomit, jaundice, suppression of urine).

Historic Note.—Yellow fever is endemic only within certain geographic limits, where it also prevails epidemically when the conditions are favorable. According to general belief, it first appeared in 1647 in the

Barbadoes (West Indies). Subsequently, it was conveyed along the channels of commerce until it became widely disseminated, and chiefly in seaport towns. In 1699 an English vessel carrying slaves transported the disease to Mexico from the Atlantic coast of Africa. Guitéras classified the areas of infection thus: (1) The *focal zone*, in which the disease is never absent, including Havana, Vera Cruz, Rio, and other Spanish-American ports. (2) *Perifocal zones*, or regions of periodic epidemics, including the ports of the tropical Atlantic coast in America and Africa. (3) The *zone of accidental epidemics*, between the parallels of 45° N. and 35° S. latitude. Yellow fever was brought to the United States (Boston) in 1693, and since then has invaded in epidemic form numerous sea-coast cities, being carried thence to a number of inland towns. For example, in 1853 the disease prevailed throughout the Southern States, and since then six epidemic outbreaks (1867, 1873, 1878, 1897, 1898, 1899), though of lesser severity, raged in the same section. The disease has been conveyed to seaports in Great Britain and France, but has never been carried inland in those countries. The belief that the disease never originates outside of certain territorial limits was advanced for the first time by the College of Physicians of Philadelphia (1797).

Pathology.—The *skin* is jaundiced, and often ecchymotic spots are observed, but the internal viscera show no characteristic lesions in cases of average intensity. In severe forms congestion, hemorrhage, degeneration, and necrosis are the changes noted.

After death the *liver* is anemic, as a rule, but in the early stages of the disease it is markedly hyperemic. Its color varies, ranging from pale yellow to an orange hue, and punctiform extravasations cause mottling of the surface. Its size varies little from the normal. Parenchymatous degeneration of the hepatic tissue is common, though in places it may be entirely normal. The liver cells are swollen, containing fat and granular matter, with indistinctness or absence of nuclei.

The *gastro-intestinal mucosa* is the seat of acute catarrh (in severe types) and numerous minute hemorrhages, similar spots of extravasation being found on the various serous membranes of the body (meninges, pericardium, pleura, etc.). Hemorrhagic infarctions may be found in the various internal viscera. The black-vomit material is found in the stomach, and less frequently also in the smaller intestines.

The *spleen* is dark and friable, but is not enlarged. The *kidneys* show the lesions of diffuse nephritis, the microscope revealing cloudy swelling of the epithelium of the tubules, with fatty degeneration and tube-casts. The *heart-muscle* looks pale, and may be the seat of granular and fatty degeneration. The *brain* and its *meninges* are hyperemic, and degenerative changes occur in the sympathetic ganglia (Schmidt).

The *blood* is dark, and many of the red corpuscles, having disorganized, set free hemoglobin, as in malaria. Fatty degeneration of the walls of the small blood-vessels and the capillaries have been noted, and these, by allowing filtration of blood-serum, produce concentration of the blood. General glandular enlargement is often found.

Etiology.—Bacteriology.—At present writing, nothing is known of the micro-organism that causes this disease. H. Seidelin, however, has observed in the red blood-cells certain ring-like and ameboid forms which he believes have an etiologic relation to the disease. The infective character of the complaint is shown by the fact that it can be produced by

the inoculation of a susceptible person with the blood of a patient suffering from the disease.

Mode of Transmission.—The work of the Yellow Fever Commission of the U. S. Army (Drs. Reed, Carroll, Lazear, Agramonte) having thoroughly overthrown the claims of Sanarelli, that the bacillus icteroides is the specific cause of yellow fever, his bacillus is now regarded as a secondary invader. In 1881 C. J. Finley[1] pointed out that the disease is transmitted through the agency of the mosquito. It, however, remained for the commission mentioned above to furnish incontestable experimental proof that yellow fever is a mosquito-borne affection. They have shown that the *stegomyia fasciata* is probably the only carrier of the infecting agent. Twelve days after biting a yellow-fever subject the bite of the mosquito will infect a non-immune person. The insect is capable of infecting man for a period of several weeks. There is some evidence that the mosquito, once infective, is capable of transmitting the parasite for the balance of its life. The mosquitoes, however, are not infected by biting the dead bodies of yellow-fever patients, it being only during the first few days of the disease that the patient's blood is infective for the mosquito, and only the female mosquito bites. The clothing, vomitus, urine, and feces are non-infectious.

The stegomyia fasciata has been found as far north as Charlestown, S. C., and southward to the Rio de la Plata, and is extremely prevalent in Cuba. The larvæ only develop in comparatively clean water, and seldom breed far outside a city's limits. Yellow fever is thus a domiciliary infection. They bite principally late in the afternoon, and are not capable of long flights unless assisted by winds. The stegomyia only travels when it gets into a car, box, or drawer instead of a house; it "will not voluntarily leave a house, much less cross a street" (White).

Among *predisposing causes*, *season* heads the list. The disease prevails chiefly in summer, being completely arrested by one, or at most two, severe frosts. *Age* and *race* have some degree of influence, children being more liable than adults, males than females, and whites than blacks. The poison is not transferred by *fomites*. The march of an epidemic may be interrupted or even completely arrested by apparently trivial agencies—*e. g.*, watercourses, rows or clumps of shrubbery. One attack usually bestows permanent *immunity*, and natives of an infected district are far less liable to the disease than newcomers. Two attacks, however, have been reported (Boseman, Libby).

Clinical History.—**Incubation Stage.**—This varies, ranging from two to five or more days. During the incubation symptoms may appear, such as languor, headache, anorexia, but are not common.

Invasion Stage.—The *onset* is abrupt, an initial *chill* usually occurring, but it is very seldom severe or prolonged, a reactionary fever following promptly and the *temperature* rising to 103°, 104°, or even 105° F. (40.5° C.). The temperature is apt to be highest at the beginning, and then declines by lysis with slight evening exacerbations and morning remissions. Hyperpyrexia occasionally occurs on the first day of the illness. The chill and fever are accompanied by headache and pains in the loins and legs, often of great severity, and a little later, restlessness,

[1] *Annales de la Biol. Academie*, vol. xviii., pp. 147–161.

mental confusion, and a delirium that is sometimes violent in character may develop. In the majority of instances, however, the mind remains clear. The *pulse* is accelerated, but not in proportion to the height of the temperature; it is full and strong at the start, and is observed to fall while the temperature remains the same or even rises. The *face* is flushed, with slight icteroid addition. The early manifestation of jaundice is the most characteristic feature of the facies (Guiteras). The eyes are suffused and intolerant of light. The *gums* may be swollen and spongy; later on a red line is seen at their margins and they readily ooze blood. The *tongue* may or may not be coated, and nausea and vomiting may occur, the latter being one of the most characteristic symptoms of the disease. Associated with these symptoms there are epigastric oppression and burning sensations, with decided tenderness. The *vomitus* may be blood-streaked or contain chocolate-colored particles, and occasionally unaltered blood is vomited. Constipation is usually present, the stools showing a deficiency of bile. The *urine* is diminished in amount, dark-colored, and often contains a slight amount of albumin: this *early transient albuminuria* is a very characteristic symptom. The *initial stage* may last from six or eight hours to two or three days, or even longer, and is longer in the milder forms. With the termination of this stage there is a marked remission of the fever and other symptoms, the pulse becoming remarkably slow.

Stage of Remission.—From this moment convalescence may begin and proceed to full recovery without interruption, the happy event being often marked by critical discharges. In most instances, however, the patient presents certain symptoms and signs of ill-health during the stage of calm (more or less prostration, epigastric distress with tenderness, mental dulness or even stupor, and a yellowish tint of skin and urine), which lasts from a few to twenty-four hours, when another serious stage supervenes.

Stage of Secondary Fever or Collapse.—The patient becomes extremely weak, presenting the signs of profound *collapse*. The surface of the body is cool (extremities often positively cold), the skin in nearly all instances assuming a yellow or bronzed tinge. It is rarely absent during life, but always present after death. The *pulse* is rapid and compressible, and soon vomiting becomes distressing. *Hemorrhage* into the stomach occurs, the blood being acted upon by the gastric secretions, and producing the material which is expelled as the characteristic "*black vomit.*" Occasionally unaltered blood may be vomited; the stools also may be tarry. In the worst cases hemorrhages from other mucous surfaces are common (epistaxis, hematuria, metrorrhagia, etc.), and cutaneous hemorrhages also now occur. In this stage the *tongue* becomes dry, brown, or even black; less frequently it is smooth, red, and fissured.

In most cases the *urine* is deficient, containing albumin and casts (with careful centrifugation), and in rare instances there is complete anuria. The latter may precede the development of grave *nervous symptoms*, as convulsions, or even coma, which may be uremic.

In some instances the *temperature* rises during this period (secondary fever), and in favorable cases terminates by lysis, or it may assume the typhoid form and result fatally, and a decided slowing of the pulse may occur, as low as twenty-four beats even. In all cases that pursue a favorable course convalescence is slow and gradual, and it may be interrupted by certain complications (*e. g.*, abscesses). The *duration* of the

entire attack (composed of three stages) is variable, though as a rule it covers about one week.

Clinical Varieties.—Many different varieties have been described, each characterized by one or more prominent features, but none seem more justifiable than Finlay's[1] classification, in which he distinguishes three forms: (1) the *acclimation fever*, or *non-albuminuric yellow fever;* (2) the *plain albuminuric yellow fever;* (3) the *melano-albuminuric yellow fever*, characterized by the presence of blood or "black vomit" in the stomach or intestines. Relapses occur, but are rare.

Diagnosis.—The symptoms that justify a diagnosis in the initial stage, provided an epidemic be prevailing, are the sudden onset, severe nephralgia, cephalalgia, peculiar facies and pulse (a fall in the pulse-rate while the fever remains high or rising—Faget's sign), nausea, and vomiting of bile. In the early stage intense capillary congestion of the surface of the body is diagnostic and indicative of a severe type. In the third stage the co-existence of jaundice, the black vomit, and suppression of urine, with evidences of collapse, make the diagnosis easy. The mild or rudimentary form offers the greatest difficulty, since the clinical picture comprises only slight fever which, at the end of a day, is followed by speedy convalescence.

Serum-diagnosis.—Woodson and P. E. and J. J. Archinard have applied the Widal reaction (agglutination-test) in 100 cases, and claim that the serum-diagnosis of yellow fever is practicable and may be used on the second day. A dilution of 1 : 40 is advised.

Differential Diagnosis.—*Pernicious malarial fever* (estivo-autumnal) has not the early, deep jaundice, the slow pulse, the peculiar temperature-curve, the intense capillary congestion of the surface of the body, the black vomit, the early albuminuria, and the clear mind—all symptoms that mark yellow fever. On the other hand the crescentic or small ring-shaped forms of the plasmodium are pathognomonic of pernicious malarial fever, as is the effect of quinin upon the disease. Kemp has made a microscopic, spectroscopic, and chemical study of the black vomit of yellow and malarial fevers, and found that the pigment was derived from the blood, which had been acted upon by the gastric juices. The vomitus in malarial fever, however, contains in addition considerable quantities of bile-pigment and bile-salts, which are wanting in that of yellow fever. Further, in the latter, the vomited matter is much more highly acid. The diagnostic features of *dengue*, which has been confounded with *febris flava*, have been contrasted with those of the latter disease on p. 140.

Prognosis.—Different epidemics show widely different death-rates, and the most potent factor is the particular type of the disease in individual epidemics. Some outbreaks have been characterized by the lighter forms, and in such the death-rate has been low (1 per cent.). In other epidemics the type has been so virulent (with high temperature) as to make the mortality list high—even to 100 per cent. In general, mild epidemics give a mortality of 5 to 10 per cent., and severe ones of 30 to 50 per cent. The death-rate is lower in private than hospital practice.

Among the gravest symptoms are *intense capillary congestion*, coming on during the first stage, *suppression of urine*, *intense jaundice*, and *uremic toxemia*. The black vomit is not as fatal a sign as the symptoms previously mentioned.

It has been noted that a larger number of men, proportionately, than

[1] *Edinburgh Medical Journal*, Edinburgh.

women and children succumb to the disease, and that it is less fatal among negroes than among whites.

Treatment.—The measures that are employed in yellow fever may be considered under three main heads: (1) Prophylaxis; (2) general management; and (3) medicinal measures.

(1) **Prophylaxis.**—Reed claims that the present quarantine laws against yellow fever are needless and the detention system absurd. The effective way to prevent carrying of the fever poison is the destruction of the *Stegomyia fasciata*—on vessels at sea as well as in infected houses and districts on land. Well persons must be protected against the bites of the *Stegomyia* by careful screening. It is a twilight mosquito, resting in the middle of the day, hence non-immunes may visit infected localities between 9 A. M. and 3 P. M. with impunity. It is unnecessary to disinfect articles of clothing, bedding, or merchandise supposedly contaminated by contact with those ill of the disease. W. C. Gorgas [1] has shown that in Havana, since attention has been directed entirely to the mosquito, the minimum annual death-rate from yellow fever has been reached. The patient must be isolated and carefully screened.

"When a non-immune is going to be exposed to yellow fever it is better to be inoculated, so that he can be put to bed and treated from the beginning, than to take it accidentally" (Gorgas). To immunize a patient a single mosquito should be employed for each inoculation.

(2) **General Management.**—The sufferer from yellow fever must be put to bed at once, and an abundance of fresh air (without exposure to strong drafts) must be supplied. The medicaments and the nourishment are to be administered through a tube or spout-cup, so as to obviate raising the patient's head. Body- and bed-linen should be kept scrupulously clean, and the patient must not be allowed to leave his bed on any account. The *diet* should be of the lightest sort and entirely liquid, beginning with peptonized milk, koumiss, or light broths.

(3) **Medicinal Measures.**—At the outset it is well to gently stimulate the various excretory organs, and mild laxative diaphoretics and diuretics answer this purpose. Hydrotherapy may be employed to maintain the nervous tonicity and reduce the temperature, but when the spontaneous fall of temperature sets in it must be discontinued. The neuralgic pains, which attack principally the head, loins, and nerve-trunk, are to be relieved by morphin given hypodermically; and for the same symptom Bemiss highly recommends quinin by the rectum (gr. xx—1.296). Intestinal antiseptics may also be used throughout the attack.

During the stage of *remission* the powers of the system are to be fully maintained by a suitable dietary and by tonics and stimulants if required.

In the last stage, supportive measures must not be forgotten. *Rectal nutrient enemata* should be employed if marked gastric irritability prohibits feeding by the mouth. *Stimulants* are demanded, and these should also be administered per rectum if not retained by the stomach, or they may in some measure be administered hypodermically. The stomach is, as a rule, tolerant of iced champagne.

If irritability of the stomach be present, ice and hydrocyanic acid may be tried. Sodium bicarbonate (gr. x to xx—0.648 to 1.296) in Vichy, Apollinaris, or Seltzer water is a most useful remedy, and Stern-

[1] *Phila. Med. Jour.*, Jan. 4, 1902.

berg has used it in combination with mercuric chlorid with success in the following formula:

℞. Sodii bicarb., ʒiv (16.0);
Hydrarg. bichlorid., gr. ss. (0.032);
Aquæ puræ, Oj (480).—M.
Sig. For a severe case two teaspoonfuls every hour, day and night; for a mild case, every hour by day and every two hours by night; administer always ice-cold.

Perhaps the chief indication for the use of sodium bicarbonate is the extreme acidity of the various secretions, especially the gastric and renal. Sternberg contends that by fulfilling this indication we prevent in great measure the occurrence of acute nephritis and suppression of the urine. Hemorrhages and other symptoms must be treated by the usual means. During convalescence tonics are to be administered, and the customary diet can gradually be resumed.

Serum-therapeutics.—Prof. Sanarelli records favorable results from the use of his antitoxic serum. Morcour[1] points out that we need to try the serum only in grave cases, since mild cases recover with simpler methods and careful nursing. Wasdin, however, used Sanarelli's serum in 3 cases and noted no advantage over other treatment. Matienzo,[2] after a series of experiments on guinea-pigs and human beings with American serum, concludes: Intravenous and subcutaneous injections produce general reaction; no effect is produced upon the disease. The reaction obtained in convalescence proves that the antitoxin does not produce the cure.

CEREBRO-SPINAL MENINGITIS.

(*Spotted Fever; Cerebro-spinal Fever.*)

Definition.—An infectious disease, caused by the diplococcus intracellularis meningitidis (Weichselbaum). It is characterized anatomically by inflammation of the meninges of the brain and spinal cord, and clinically by an irregular course, a moderate febrile movement with somewhat characteristic and profound nervous symptoms (excruciating headache, pain in the back and upper part of the spine, contraction of the muscles of the nucha, hyperesthesia, delirium, and ofttimes coma). The disease may occur sporadically or in epidemics, or may even assume pandemic proportions.

Historic Note.—Cerebro-spinal meningitis was first recognized and described as late as the beginning of the last century (1805) by Viesseux of Geneva. During the next decade numerous limited epidemics were observed both in Europe and the United States, and subsequently recurring epidemic and pandemic visitations were noted, though

[1] *Proceedings Third Pan-American Medical Congress*, Feb. 4, 1901.
[2] *Med. News*, Jan. 13, 1900.

at longer and variable intervals of time. In nearly all the large cities in this country it has become endemic, and in Philadelphia since 1863.

Pathology.—The cases that prove speedily fatal do not present gross characteristic changes, but by the aid of the microscope leukocytes are discovered immediately around the cerebral vessels, and round cells in the cortex of the brain. In some cases the characteristic evidences of encephalitis are already noticeable. On the other hand, the cases in which death occurs after the disease has been fully developed show the lesions of suppurative inflammation of the meninges of the brain. The arteries, veins, and sinuses are much engorged; the ventricles are distended with liquid, but the pia mater is principally affected, its vessels being greatly enlarged, and a more or less copious sero-fibrinous or sero-purulent exudate occurring into the meshes of its network. The longer the duration of the case the more purulent is the exudation. The ventricles of the brain are filled with a similar exudation, and red blood-globules may be present at an advanced stage. The color of the exudate is at first almost clear (being composed of serum); it then changes to a milky turbidity, to a pale yellow, and, lastly, takes on a greenish-yellow color ("leek-green"). The subarachnoid space may be occupied by a uniform layer composed of fibrin and pus.

The brain-matter is congested, and sometimes softened in spots, and on section the gray matter may present punctate extravasations. When resolution occurs recovery may be complete, but frequently the pia mater remains thickened. The exudation may follow the auditory and optic nerves along their lymph-sheaths, and pus has been found in the internal ear as well as in the chambers of the eye.

The membranes of the spinal cord manifest lesions identical with those of the brain. They are vascular engorgements, followed by sero-fibrinous, and later still by sero-purulent, exudation beneath the arachnoid. The changes are more marked on the posterior than the anterior surface of the cord, and the exudate increases in amount in passing from above downward, in severe cases sometimes assuming the form of a sheath which completely surrounds the cord throughout its entire length. The pia mater is congested, and may be thickened, shaggy, and in places adherent to the cord, of which the gray matter may be the seat of serous infiltration, and rarely of softening. Barker describes certain changes that occur in the nerve-cells and the ventral horns of the nucleus dorsalis (Clarkii) of the spinal cord in epidemic cerebro-spinal meningitis.

The lungs may exhibit the changes peculiar to bronchitis or pneumonia. In the heart endocarditis may be noted, though rarely, and both the pleura and the pericardium may show inflammatory lesions and contain a serous or sero-purulent exudation. Hemorrhages into the serous membranes and into the skin may take place. The spleen may be enlarged, the increase in size and the degree of fever being proportional, and the liver is hyperemic. The kidneys are congested, and bacterial forms have been found associated in the latter with the lesions of acute nephritis and hemorrhage—conditions of which they were probably the cause.

Etiology.—**Bacteriology.**—The diplococcus meningitidis is the specific cause of epidemic cerebro-spinal meningitis. The special organism

can be isolated from the spinal fluid, the meninges of the brain and cord, the blood, the joint-lesions, and the nasal mucus.

The meningococcus, like the gonococcus, occupies a position within the polynuclear leukocytes, but never appears within the nucleus (Park), and like the latter is biscuit-shaped. The bacterium takes the usual stains. It develops upon agar-agar and upon Loeffler's blood-serum, manifesting characteristics of growth that simulate those of the pneumococcus. Councilman, Carl Fränkel, Boston, and others, by refined methods, have, however, been able to differentiate these organisms. Welch suggests that the meningococcus and the pneumococcus are possibly varieties of the same bacterium, while Netter regards the meningococcus as a degenerate form of the pneumococcus. Among the associated microbes are the pneumococcus, streptococcus pyogenes, staphylococcus aureus, bacillus coli communis, and the tubercle bacillus, and any one of the latter is capable of causing sporadic cerebro-spinal meningitis.

Predisposing Causes.—(1) **Age.**—Most cases occur in children and young adults, though no age enjoys perfect immunity. Of 94 cases occurring in children up to 15 years of age, 56 were under five years (Claytor).

(2) **Climate.**—The disease is unknown in tropical climates, but has occurred in all parts of the temperate zone, and is most prevalent in the more northerly portions of the latter.

(3) **Season** is not an important factor, though the disease prevails largely in winter and spring.

(4) **Unhygienic Influences.**—The disease often appears in ill-ventilated and overcrowded habitations—among the poorer classes, among soldiers crowded together in barracks, and among prisoners. Prolonged marching, and excessive physical or mental exertion, may heighten susceptibility. In certain epidemics the disease has raged exclusively in villages.

Modes of Conveyance.—Precisely how the contagion is transferred from an infected person to a healthy one is not known, but the disease is probably contagious. Hare[1] has recorded two cases in which the infection seemed to be transferred directly from the first to the second. The poison may be conveyed by *fomites* in cases that furnish intensely virulent poison. As to the manner in which the virus gains entrance to the system, our knowledge is imperfect, although Hunt[2] states that cerebro-spinal meningitis seems to be an inhalation disease. It is certain that this germ may enter the meninges (*a*) by blood metastasis; (*b*) by direct extension of an adjacent inflammatory process (*e. g.*, mastoiditis). Elser and Hontoon[3] believe that the disease may be spread by meningococcus carriers.

Clinical History.—The period of **incubation** is brief, though unknown. The *prodromal symptoms* are variable in different epidemics. Invasion may be sudden, a patient in vigorous health often being stricken down as though by a blow. In some rapidly fatal cases there is a short prodromal period, during which the patient complains of lassitude, headache, rachialgia, muscle- and joint-pains, and sometimes nausea and vomiting. In ordinary forms the prodromes may last from a few hours to a week or more, and the patient's complaint may be limited to cervical and occipital pains lasting a day or two; then, without any initial chill,

[1] *New York Med. Jour.*, Feb. 10, 1906. [2] *Boston Med. and Surg. Jour.*, Nov. 1, 1906.
[3] *Journal of Medical Research*, 1909, p. 397.

the *invasion-period* supervenes. In milder and sporadic cases the symptoms consist chiefly of languor and debility, headache, pain in the back and limbs, vertigo, vomiting, and sometimes diarrhea.

Most cases begin *abruptly*, between noon and midnight. The most distinctive and violent features are chill (often severe), *fever* of a moderate grade, a full and somewhat *accelerated pulse*, *raging headache*, and *vomiting*. In children the ushering-in symptom may be a *convulsion*. These phenomena are followed by pain in the back and cervical portion of the spine—an early and characteristic symptom. Attempts at flexion or rotation of the head increase the pain in the neck and movements of the body augment the spinal pains. Later, the muscles in the cervical region contract, at the same time becoming rigid, and produce the condition of opisthotonos. The patient may be unable to swallow.

The *temperature* is but moderately elevated. In a certain percentage of the cases it rapidly rises to 104° or 105° F. (40.5° C.), but soon falls to 102° or 103° F. (38.8° or 39.4° C.), at which level it is maintained with irregular undulations until defervescence, which takes place by lysis. In fatal cases death is preceded by a sudden great elevation of temperature to 108° and even 110° F. (43.3° C.). In the very young the thermometric range is lower than in adults.

The *pulse* is but slightly accelerated, if at all, in the early stages of the disease. Later, in twenty-four to thirty-six hours, it may in severe cases leap to 120 or even 140, its chief characteristic being the variability in its rate. In the early stage it is of good volume and tension; later, it may be soft and compressible, and in serious cases it becomes small and feeble. Polynuclear leukocytosis, moderate or severe, is constant.

The *respirations*, as a rule, increase in frequency and are sometimes quite irregular; but marked dyspnea, with slowing of the respirations, may be observed during the advanced stage, being due to pressure exerted by the exudation upon the respiratory center. Cheyne-Stokes breathing and sighing respirations may be present.

Nervous Symptoms.—The *headache* is racking and often persistent, though it is subject to remissions; it is intensified by light and sounds. There is vertigo in nearly all instances. The *pain* referred to the spine may be general or limited to either the lumbar or cervical region (rarely the dorsal), and the general myalgic pains are often intense, especially in the extremities and the abdominal region. With the cephalalgia and abdominal pain may be associated *vomiting*. *Hyperesthesia* is a prominent symptom, the gentlest touch being extremely painful; and *anesthesia* may follow. Any voluntary muscular movements, however, excite pain. In some cases *delirium* appears early, and in others rather late, while in the worst types death often occurs before delirium develops. On the other hand, in a small percentage of cases, this symptom is absent throughout the entire course, and always its character and intensity exhibit a remarkable variety. It may be mild or it may take the form merely of incoherent answers to questions. Active delirium, however, is common and is accompanied by hallucinations, during which the patient shouts loudly, and, unless restrained, gets out of bed. This form of delirium occurs in paroxysms that are most apt to appear at night, and in the female it is sometimes hilarious or hysteric. An erotic tendency, with priapism or seminal emissions, has been observed in males. The "maudlin"

delirium of the drunkard is sometimes seen, but sooner or later somnolence appears and may deepen quickly into coma, perhaps temporary, though more often it continues until recovery or death. Vomiting is common, usually late in the disease; it is doubtless of cerebral origin.

Symptoms of motor irritation are common, twitching of single muscles or groups often being seen, and occasionally muscular tremors. Muscular contraction is an almost constant feature. After a few days a tonic spasm of the muscles of the extremities sets in, bending the arms upon the chest, the forearm upon the arm, and the thumb upon the palm; the thigh is also flexed on the abdomen and the leg on the thigh. The opisthotonos may be followed by trismus, which can be considered a mortal symptom. Convulsions do not occur in adults, but are common in children; occasionally there is paralysis (facial hemiplegia).

Organs of Special Sense.—*Photophobia* is a prominent symptom, and the condition of the *pupils* is very variable. They may be dilated or contracted (more frequently the former) or remain normal; and in the majority of cases they are unequal in size and react poorly to light.

Strabismus is frequent, usually temporary, though it may recur during the attack. Rarely it is permanent. *Conjunctivitis* of moderate intensity and *keratitis* may occur, the former being common. Burville-Holmes[1] invites attention to anesthesia of the cornea and conjunctiva, which occurs in about one-half of the cases. *Ptosis* is almost always present. Intense purulent *irido-choroiditis* sometimes occurs; either temporary or permanent *blindness* and, rarely, *nystagmus* are noted. Among optical sequelæ are cataract and atrophy of the eyeball.

Deafness is common, there being an early intolerence of sound and a marked tinnitus aurium. Late suppurative inflammation of the middle ear, followed by rupture of the tympanum and *otorrhea*, may occur. The internal ear may be similarly involved, with uncertain *gait*.

Cutaneous symptoms appear, some of which possess considerable diagnostic worth. *Pallor* and *lividity* of the skin and visible mucous membranes often characterize the period of invasion, and shortly after the onset *herpes facialis* appears in more than half the cases. This symptom is significant for diagnosis. The separate lesions are extensive, and often coalescence of two or more is witnessed. Herpes facialis belongs in a peculiar sense to cerebrospinal meningitis; herpes labialis to malaria, and less frequently to pneumonia and meningitis. A *petechial eruption* is common, in the early epidemics, and more frequently in America than in Europe. To this symptom the disease owes the name, long since given to it, of "spotted fever." It may, however, be absent, and when present it is sometimes limited to a small superficial area, though more frequently it is diffuse. At first the eruption may be bright red (erythematous), later becoming darker, or it may be distinctly petechial from the start; purpuric spots of considerable size and sometimes large ecchymoses may appear, but these are most common in the more malignant types. Other forms of eruption are also seen (sudamina, urticaria, ecthyma, erythema, erysipelas, etc.), but are devoid of diagnostic value. *Gangrene* of the skin is occasionally noticed, and in some cases bed-sores are liable to arise; but there is no fixed time for the skin-lesions of cerebro-spinal fever to appear, and their duration is exceedingly variable.

[1] *Jour. Amer. Med. Assoc.*, 1908, l, 280.

Of **gastro-intestinal symptoms** *vomiting* is the most common. It usually lasts only for a brief period at the onset, though it may recur later at longer or shorter intervals, and is of nervous origin. The *appetite* may be good, but in many cases it is soon lost, the *tongue*, in a large proportion of the instances, being only slightly coated. In cases assuming the adynamic or *typhoid type* the tongue is apt to become dry and of a brown color, with the formation of sordes. Under these circumstances the abdomen is tympanitic and the bowels relaxed, and diarrhea may be urgent, resisting all efforts aimed at its relief. Retraction of the belly is common, and *constipation* instead of diarrhea is the general rule; the spleen may often be felt a little distance below the costal margin.

Renal symptoms are not prominent, though the *amount* of urine passed is often above the normal despite the febrile movement. It may be below, though rarely, while in still other cases it is about normal; and *retention* on the one hand and *incontinence* on the other have been observed. *Albuminuria* is sometimes met with, and rarely glycosuria.

Arthritis is not uncommon, particularly in the severer cases.

Kernig's Sign.—In 1884 Kernig first pointed out the impossibility of obtaining complete extension of the leg on the thigh when the patient is *sitting* and the thigh is flexed at a right angle to the trunk. The sign is produced by irritation of the meninges of the lower portion of the spinal cord and of the nerve-roots that constitute the cauda equina, Roglet thinks that one cause for this sign is intraventricular pressure.[1] Under this irritation, increased by the stretching effect of the sitting posture, the tonicity of the flexor muscles of the leg is increased, and as a consequence complete extension of the leg becomes impossible. The contracture disappears when the patient assumes the dorsal decubitus. If the patient cannot be propped up in bed, the thigh may be flexed upon the abdomen, when, if meningitis be present, complete extension of the leg will be prevented by contraction of the flexor muscles. Head's[2] statistics, embracing 156 cases, show that Kernig's sign is present in 84 per cent. of the cases of meningitis. It is not confined to cerebro-spinal meningitis, but is present in all meningeal affections. The time of its appearance is variable; hence, in order to be certain that the sign is not present, it should be looked for repeatedly. Again, the time of its disappearance varies; it may disappear during the preagonal period. The value of the sign is real, but its absence does not justify the exclusion of meningitis, while it may be present in other diseases (typhoid, tetanus). Herrick[3] points out that from its persistence into convalescence it may be utilized to make a retrospective diagnosis.

Macewen's sign (*vide* Tuberculous meningitis, p. 253), a hollow note on percussing over the inferior frontal or parietal bone, is an indication of fluid in the ventricle, but is not always present.

Brudzinski's Sign.—On attempting to bend the neck flexure movements in the ankle, knee, and hip-joints occur (identical reflex). Another, though less constant, sign is produced by passive flexion of one leg, which causes the fellow limb to draw up, and so remain (contralateral reflex).

Complications.—Many of these have already been mentioned in the portrayal of the symptoms—*e. g.*, destructive inflammations of the eye

[1] P. Roglet, *Gaz. heb. de Méd. et de Chir.*, July 15, 1900.
[2] *St. Paul Med. Jour.*, Sept., 1900. [3] *Amer. Jour. Med. Sci.*, July, 1899.

and ear and the paralyses of the cranial nerves. The purulent inflammations of the serous sacs which were referred to in discussing the pathology (pleurisy and pericarditis) are among associated conditions, and secondary bronchitis is common. *Pneumonia* (lobar and lobular) is a frequent complication. Endocardial murmurs are common, but pericardial friction is less so. *Hemorrhagic nephritis* is a rare complication.

Special and Atypical Forms.—(1) **Mild or Rudimentary.**—In this type the characteristic signs are either undeveloped or wanting, and the diagnosis is possible only during the prevalence of epidemics, which furnish typical cases. The most constant and significant symptoms are severe headache, languor, vertigo, nausea, and occasionally vomiting. Fever and contraction of cervical muscles are absent, as a rule. The duration is brief, rarely exceeding three or four days.

(2) **The Abortive Form.**—Here the initial symptoms are severe, but after two or three days they rapidly subside, leaving the patient convalescent. The disease is cut short by the acquisition of immunity, and not as the result of medical interference.

(3) **Intermittent Form.**—In this variety the symptoms, however intense, remit or almost wholly intermit every day or second day; these remissions are followed by a decided exacerbation or recurrence of the distressing features of the disease. Intermissions often occur at an advanced stage. There is not observed the strict periodicity that is seen in malaria. Neither is the malarial plasmodium found in the blood.

(4) **Typhoid Form.**—In certain cases the special features are characteristic of the "typhoid state," with protracted course.

(5) **Fulminant or Apoplectic Form.**—The symptoms characterizing this most malignant type of the affection are rather inconstant. There may be severe chill, loss of consciousness, followed by deep coma and death, the whole course occupying the space of a few hours only. I saw two such cases in the same family: the first, a girl of five years, was stricken at 2 P. M. and died at 9 P. M.; the other, a boy of seven years, was taken ill on the following day about the same hour, and died at 10 P. M. Other instances pursue a somewhat slower course, though manifesting the most striking malignancy. These begin with intense chills, violent headache, vomiting, early stupor, great prostration, contraction of muscles of the neck, moderate fever, and a feeble, progressively slowing pulse until it sometimes reaches 50 or even 40 beats per minute. The eruption, when it appears, takes the form of purpura. This form is most apt to be met with early in an epidemic, and with few exceptions proves fatal.

(6) Schlesinger[1] states that epidemic cerebrospinal meningitis affects a senile type in elderly subjects, with little tendency to fever, or opisthotonos.

Diagnosis.—The most important symptoms for diagnosis are the abrupt onset; intense pains (cervico-occipital and lumbar); prostration; vomiting; vertigo; somnolence, alternating with local or general tonic or clonic convulsions; delirium (often sportive in type); tonic contraction of the muscles of the neck, extending to the back; marked hyperesthesia; a slow, followed by a more rapid, though variable, pulse; irregular temperature-curve; and certain eruptions (petechial, herpetic).

Lumbar Puncture.—The value of Quincke's lumbar puncture as a means of diagnosis is absolute. It alone can render the diagnosis certain in many cases, and is a harmless measure, if rigid asepsis be

[1] *Jour. Amer. Med. Assoc.*, October 16, 1909.

observed. The patient is placed upon the right side, with the left knee well drawn up; a fine needle, three inches in length, and carefully guarded by the index finger of the operator, is introduced between the third and fourth lumbar vertebræ "one-half inch to the right of the median line" (Mallory and Wright), and directed slightly inward and upward. The forefinger of the disengaged hand must be used as a guide, and the site should be anesthetized by the application of a local freezing-mixture. The needle should enter the canal at a depth of two or three centimeters in children and four to six centimeters in the adult. If the fluid does not flow, the dura has probably not been penetrated, and *no form of suction* upon the needle should be attempted; the fluid should be allowed to fall drop by drop into a sterile test-tube held aslant. From five to ten cubic centimeters of the usually cloudy exudate should be withdrawn and subjected to a chemic, microscopic, and bacteriologic examination. Sugar, which is found normally, is absent from the cerebro-spinal fluid. Lorgo[1] insists that lumbar puncture must be repeated if the result of the procedure is at first negative. The fluid is said to be clear in tuberculous meningitis. If the presence of the diplococcus intracellularis in the nasal secretion can be shown, lumbar puncture is unnecessary. The precipitin reaction permits one to make a diagnosis, and sometimes with perfectly clear cerebro-spinal fluid (Vincent and Bellot[2]).

Differential Diagnosis.—(1) *Tuberculous Meningitis.*—In this affection there is usually a tuberculous history—either personal or family—with prodromes extending over many days (occasional vomiting, unnatural peevishness, constipation), unlike the sudden onset of meningitis. The retraction of the abdomen is greater, while the arching of the neck, the general myalgic pains, and the hyperesthesia are less; the herpetic and petechial eruptions are rare in tuberculous and common in cerebro-spinal meningitis. Cheyne-Stokes breathing and the well-marked changes of pulse belong peculiarly to the tuberculous form. By the aid of the ophthalmoscope choroidal tubercles may sometimes be detected.

Hand[3] urges lumbar puncture, and found polymorphonuclear leukocytes in excess wherever tubercle bacilli were absent.

(2) *Pneumonia.*—This affection may be complicated with a meningitis that affects chiefly the cerebral cortex. Hence, while there will be motor spasm (more or less localized) and tremors, there will also be less retraction of the head and less myalgic pain than in cerebro-spinal meningitis. Again, pneumonia precedes the development of the meningeal symptoms.

(3) *Typhoid Fever.*—The cerebral type of this affection may simulate closely meningitis. In both may be observed fever, delirium, somnolence, retraction of the neck, spasm, tremor, and profound prostration. The mode of onset, however, is different, being slower in typhoid and unaccompanied by vomiting, muscular spasm, or hyperesthesia. In typhoid there is also the characteristic mental dulness; the fever is higher, with a typical fever-curve; the roseate eruption and sero-reaction are characteristic, and there is greater enlargement of the spleen.

Sequelæ.—The leading sequelæ are permanent blindness (due to optic neuritis with atrophy) and deafness, which sometimes terminates in

[1] *Polyclinico*, March, 1901; Saunders' *Year-Book*, 1902.
[2] *Bulletin Académie de Médecine*, vol. lxi, p. 326.
[3] *Phila. Med. Jour.*, Aug. 30, 1902.

deaf-mutism; and in many cases headache outlasts the disease for months. Chronic hydrocephalus and mental enfeeblement are not rare sequels (Ziemssen). Various local paralyses are observed, probably due to certain peripheral lesions (neuritis and perineuritis).

Immunity.—Permanent immunity is rarely conferred by the occurrence of cerebro-spinal meningitis, *relapses* being common, and *second* (recurrent) attacks having been occasionally observed.

Duration and Prognosis.—In very mild forms the duration is from one to four or five days. The most malignant type runs an even shorter course, when, as is the rule, it terminates fatally. If recovery ensues, it is after a long, serious, and protean illness. The *abortive form* is necessarily of brief duration. In the ordinary type convalescence usually sets in at the end of one or two weeks, but a slow convalescence, hindered by numerous complications and sequelæ, is the rule.

Apart from the *fulminant form*, which nearly always proves fatal, the severity of the infection may be appreciated by noting the degree of fever and the intensity of the nervous symptoms, especially the vomiting, coma, headache, opisthotonos, character of the respirations, etc. *Complications* may likewise affect the prognosis, pneumonia, and suppurative inflammations of the pleura or pericardium, rendering it particularly grave. In *children* under two years the disease is very fatal, this period giving the highest mortality-rate; between two and five and after thirty years it is a more serious disease than during young adult life. The death-rate of cerebro-spinal fever varies greatly in different epidemics, ranging form 25 per cent. in the mildest to 80 per cent. in the severest.

Prophylaxis.—Disinfection of the nasopharynx, the expectoration, conjunctival secretions, and the urine is recommended with a view to destroying the specific poison. Meningococcus carriers must be discovered and treated. Isolation is to be carried out. Persons in any manner exposed and suffering from diseased conditions of the respiratory apparatus or pharynx should receive prompt and active treatment. Sophian and Black claim that the injection of dead meningococci confers considerable immunity.

General Management.—The sick-room must be quiet and somewhat dark. All excitement is to be avoided; the patient must not be allowed to leave his bed until convalescence is firmly established.

The *diet* should be composed of nutritious liquids, such as milk and animal broths, etc., and as soon as convalescence begins the dietary should be increased by the addition of semisolid substances (rice, eggs, milk-toast, etc.), and, finally, the more easily digestible solids. The period of convalescence may be much abridged by systematic feeding.

Medicinal Treatment.—Individual cases are to be treated according to the special indications presented. I regard it as extremely improbable that any case of this affection has been benefited by venesection.

Among medicinal agents narcotics are the most useful. Morphin hypodermically affords relief from intense headache, myalgic pains, muscular contraction, and other nervous symptoms in some cases. If the respirations be irregular, atropin may be combined with the opiate; and if the heart threatens to fail, strychnin may be administered. Should morphin fail, the bromids and chloral (the latter in small doses) are to be employed. In young children we must rely upon the bromids rather than the opiates.

Flexner and Jobling[1] present a report on 393 patients treated with Flexner's curative serum. Of these, 295, or 75 per cent., recovered and 98, or 25 per cent., died. The serum is injected directly into the subarachnoid space after the withdrawal of an equal amount of cerebro-spinal fluid by means of lumbar puncture. The injections should be repeated daily for three or four days. When the Macewen percussion-note, however slight, is obtained, Koplik proceeds to puncture. Cantas[2] advocates the injecting of the serum into the lateral ventricle. Wasserman[3] reports 102 cases treated with antimeningococcus serum; it had a curative effect when injected early (dose, 5 to 10 c.c., repeated two or three times a day). McKenzie and Martin have introduced an autogenous serum; they withdraw blood-serum of a patient suffering from meningitis and inject it into the spinal canal of the same or another meningitis patient. Such a serum is an actively bactericidal fluid.

For the tonic contraction of the muscles and violent cerebral symptoms, cannabis indica should be tried. Convulsions call for hot baths (105° F.) or ether inhalations. Mercury has been, and still is, advocated (mercuric chlorid, gr. $\frac{1}{24}$—0.002, every four hours to an adult; calomel, gr. $\frac{1}{12}$—$\frac{1}{16}$—0.005–0.004, every four hours to children). Belladonna and ergot have been employed in the early stages to diminish the congestion of the cerebro-spinal capillaries.

Stimulants are required if signs of heart-exhaustion appear. They may be freely exhibited in accordance with the customary rules.

After effusion of the exudate has taken place, the narcotics are to be replaced by agents that promote absorption, as potassium iodid.

The **local means** are also important. When tub-baths are not available, cold should be used locally, since it is both of value and very grateful to the patient. An ice-bag is to be put on the head, and, if possible, long ice-bags placed along the spine. In rare cases of sthenic type we may employ small blisters at the nape of the neck or over the mastoids; they are useful during the stage of effusion. In the usual form of the disease it is better to apply the thermocautery lightly over the mastoid region. A small amount of blood may be withdrawn by means of leeches or by a few wet cups placed behind the ears. Quincke's lumbar puncture and laminectomy with free drainage have been practised, and lumbar puncture should, if necessary, be repeated, but only in case benefit follows first puncture. The principal effect is the relief of the pressure upon the central nervous system. In cases in which lumbar puncture only brought 2 to 20 c.c. of fluid, Cantas[4] obtained 40 to 120 c.c. from the lateral ventricle.

Convalescence is prolonged, and requires to be diligently and judiciously treated. We must rely upon the generally accepted tonics—iron, cod-liver oil, arsenic, and strychnin; the potassium iodid and the mercury also being continued for their influence in promoting the absorption of the exudate. Special attention is, however, to be paid to the hygienic management of this period. An abundance of fresh air, sunshine, and easily assimilable food must be furnished at all hazards, and electricity and massage, judiciously employed, will hasten recovery.

[1] *Jour. Amer. Med. Assoc.*, July 25, 1908.
[2] *Bulletin de l'Academie de Medecine*, Paris, January 30, lxxvi., No. 5.
[3] *Deutsche medizinische Wochenschrift*, Sept. 26, 1907.
[4] *Loc. cit.*

LOBAR PNEUMONIA.

(*Croupous or Fibrinous Pneumonia; Pneumonitis; Lung Fever.*)

Definition.—An acute infectious disease caused by the *Micrococcus lanceolatus*, which produces a specific inflammation of the parenchyma of the lung and marked constitutional disturbances—chill, extreme prostration, and fever which terminates by crisis. There are different forms of lobar pneumonia, as primary lobar pneumonia, secondary lobar pneumonia, and pneumonia with the formation of new connective tissue.

Pathology.—Usually the lesions are confined to the whole of one lobe; less frequently to the whole of one lung, and rarely to parts of both lungs. From Jürgensen's analysis of 6666 cases the following statement, showing the different situations of the lesions and their relative frequency, was taken: Right lung, about 54 per cent.; left lung, about 38 per cent.; and both lungs, about 8 per cent. In the right lung the lower lobe was involved in 22 per cent., the upper in 12 per cent., the middle in nearly 2 per cent., and the whole lung in about 9 per cent. In the left lung the lower lobe was involved in about 23 per cent., the upper in about 7 per cent., and the whole lung in about 8 per cent. The disease involves whole segments of the lungs, and these may embrace more than one lobe.

The lesions of pneumonia are divisible into three stages: (*a*) Stage of congestion or engorgement; (*b*) Red hepatization (consolidation); and (*c*) Gray hepatization.

(*a*) **Stage of Engorgement.**—The part or parts implicated are of a dark-red color, and firmer to the feel, but less resilient and crepitant, than normal. The cut section drips a blood-stained serum, and dark blood exudes from the distended capillaries. The air-cells do not collapse, though they are not solid, since excised pieces float; but the weight of the lung-tissue is much increased. Collapsed portions may be observed which may readily be insufflated from the bronchus, and areas of extravasation may occasionally be noted near the pulmonary pleura.

On *microscopic examination* the alveolar epithelium is seen to be swollen, the capillaries greatly distended, and the air-cells containing alveolar epithelial cells, red corpuscles, and a few leukocytes. Similar elements occupy the small bronchi.

(*b*) **Red Hepatization.**—The affected tissue is solid, airless, and firm, resembling, as the term indicates, liver-tissue. It is reddish brown (mahogany) in color, presenting a dry, mottled appearance, and when, as is usual, an entire lobe is involved, it is more voluminous than normal and its surface is often furrowed by the impress of the ribs. Being airless, the affected portion does not crepitate, and its weight and specific gravity are increased. It cannot be inflated; is extremely friable, and its lacerated surface presents a finely granular aspect, this latter appearance being due to the minute plugs of inflammatory matter (fibrin) which fill the air-spaces. The air-passages and small bronchi are distended with similar material, and granular masses can be removed from the air-cells of a cut or lacerated surface by carefully scraping the latter. If death

takes place during this stage, the ante-mortem, dry, inflammatory exudate soon softens, and may flow from the cut section as a grumous, viscid fluid; the consolidated tissue sinks rapidly in water. The pulmonary pleura is covered with a fine sheet of fibrin, and in cases complicated by marked pleurisy the fibrinous, inflammatory exudate forms a thick coating upon the pleural membrane, and the sac may contain liquid effusion.

Microscopic examination shows the air-spaces filled with clotted fibrin, in whose meshes are held red blood-corpuscles, pus-cells, and changed alveolar epithelium. The interlobular connective tissue may be infiltrated with leukocytes and fibrillated fibrin, but the blood-vessels in the walls of the alveoli remain pervious. The pneumococci (micrococci lanceolati), less frequently also streptococci and staphylococci, are detectable.

(*c*) **Gray Hepatization.**—In this stage the fibrinous exudation becomes decolorized, the surface at first resembling granite in color, and later appearing uniformly gray. Associated with this change, and following it, there is fatty and granular degeneration of the inflammatory exudate, in consequence of which the latter becomes moist and soft. The exudate loses its granular character, while at the same time the friability of the lung-tissue is further increased, and from the surface of the cut section there flows usually a grayish-white or yellowish-white purulent liquid. Not less than one-half of the fatal cases die in the early part of this stage. The pleura is usually covered with a fine fibrinous exudation.

Microscopic examination shows the air-cells stuffed with leukocytes, while the other histologic elements (fibrin, red blood-cells) have disappeared; and with the full development of gray hepatization resolution usually commences. The exudate is now softened into a liquid material with disintegration of cellular elements, and is absorbed by the lymphatics. *Resolution* usually corresponds in time with the occurrence of the crisis, though it may begin later. Pratt[1] found larger phagocytic cells in all stages of the disease; it is likely that they play an important part in resolution. Among unfavorable terminations are—

(1) **Purulent Infiltration.**—Here the lung-tissue becomes very soft, friable, and is bathed in purulent material; and microscopic observation shows the pus-cells densely infiltrating the interalveolar tissue and filling the air-spaces as well. Necrosis of the lung-texture may occur, producing abscess.

(2) **Abscess.**—This is due to subsequent infection by streptococci, hence is a complicating lesion. The abscesses vary in size within wide limits, most frequently being situated near the base of the lung. In most instances the abscess-cavity has a fistulous connection with a bronchus, but occasionally the abscesses become encapsulated in fibrous tissue, their contents undergoing first caseous, and then calcareous degeneration. When multiple, they sometimes coalesce, forming large abscesses.

(3) **Gangrene** may rarely follow, but is due to a specific cause.

(4) **Induration.**—A. Fränkel states that in a few instances (about 1 per cent.) pneumonia ends in induration, and is found upon section to be smooth and its tissue resistant (*vide* Chronic Interstitial Pneumonia).

(5) Pneumonia, particularly of the apex, may terminate in phthisis. Tubercular infection commonly occurs in unresolved pneumonias.

Changes in other Viscera.—The *heart* often appears pale and is flabby,

[1] W. H. Welsch's *Festschrift*, p. 265.

but upon microscopic examination the muscular cell-fibers of the organ are not found to be degenerated, except in rare, protracted cases. The cardiac chambers, particularly the right, are distended with firm, tough clots, which are usually removable *en masse* from the great vessels in the form of arboreal casts. The blood tends to coagulate, owing to the fact that its fibrinous elements are vastly increased, although Dochez found the coagulation time to be generally prolonged. Flexner found that coagulation was favored by auto-agglutination of the red cells.

Pericarditis occurs in about 5 per cent. of the cases, and is relatively more frequent in right-sided or double pneumonia. *Endocarditis* is more common, especially the ulcerative form—in 11 out of 100 autopsies (Osler). With malignant endocarditis the lesions of meningitis are often combined, but as a separate complication the latter is rare.

The *spleen* is congested, moderately enlarged, and softened, and the *liver* is likewise hyperemic and somewhat swollen. In the *kidneys* are found the lesions of parenchymatous inflammation, and with remarkable frequency also those of chronic interstitial inflammation. A catarrhal state of the gastro-intestinal mucosa (often with jaundice) is common; and a frequent complicating change is croupous inflammation of the colon.

When the infection is caused by the Friedländer's bacillus the diseased portions of the lung are increased in volume, and multiple foci may be formed throughout one lobe (Kokawa). The cut section is characterized by a slippery sensation to touch owing to the presence of a large amount of mucus, especially in the early stages. Swelling, proliferation, desquamation, and necrosis of the epithelium is observed. The fibrino-hemorrhagic exudate is not great, the large, emigrated leukocytes, and the epithelial cells forming the principal constituents of the exudate in the later stages. The bacilli are taken up by the epithelial cells and leukocytes, which swell up and develop vacuoles. Other infections may be caused by the pneumobacillus—pleuritis, endocarditis, pericarditis, abscesses, otitis media, and osteomyelitis.

Etiology.—Bacteriology.—The generally accepted specific cause of pneumonia is the *Micrococcus lanceolatus* of Fränkel. It is a lance-shaped (slightly elliptic) coccus, united in pairs, when typical has the shape of two cartridges placed end to end, is surrounded by a pale capsule, and is present occasionally in the nose, Eustachian tubes, and larynx of healthy individuals. Netter found it in 20 per cent. of the specimens of buccal secretion taken from well persons, and "it is the migration of these ever-present germs into the pulmonary alveoli which causes pneumonia" (Wells). It is present in about 90 per cent. of all instances of pneumonia, and in persons who have had the disease it is detectable for many months or even years. It is generally present in pure culture, but may be associated with pyogenic organisms. It is probable that Friedländer's bacillus (discovered in 1883) and other micro-organisms (Eberth's bacillus, streptococcus of erysipelas, bacillus pestis) may also have the power to cause the disease; and Wassermann [1] suggests that specific forms of pneumonia may coexist in the same individual, as, for example, lobar pneumonia and influenzal pneumonia. The organism grows upon all the culture-media except potato, between the temperatures of 24° and 42° C. (McFarland). The *diplococcus pneumoniæ* (Fig. 10) can be readily demonstrated in the sputum by

[1] *Deutsch. med. Woch.*, Leipzig, Nov. 23, 1893.

treating a fixed cover-slip preparation with glacial acetic acid which is allowed to drain off and is replaced (without washing in water) by anilin oil-gentian violet solution; this is to be poured off and renewed two or three times.

The Pneumococcus in Other Diseases.—It has been found in pure culture in pleuritis (including empyema), pericarditis, meningitis, peritonitis, endocarditis, synovitis, bronchopneumonia (principally in adults), acute abscess, and other conditions.

The mode of infection is by inhalation, although there may be other portals of entry. The first effects of the germ are local—in the lung, though it may reach more distant organs. To the widespread distribution of the pneumococcus is due, in part, the septicemic process sometimes observed. The toxins of the *micrococcus lanceolatus* also become diffused throughout the system, producing a general disturbance (*toxemia*). *Secondary infection* with other specific organisms (streptococci, staphylococci, colon bacillus) commonly occurs in the various organs of the body.

Predisposing Causes.—(1) **Endemic Influence.**—That endemics of pneumonia, often of serious type, may occur in solitary buildings (barracks,

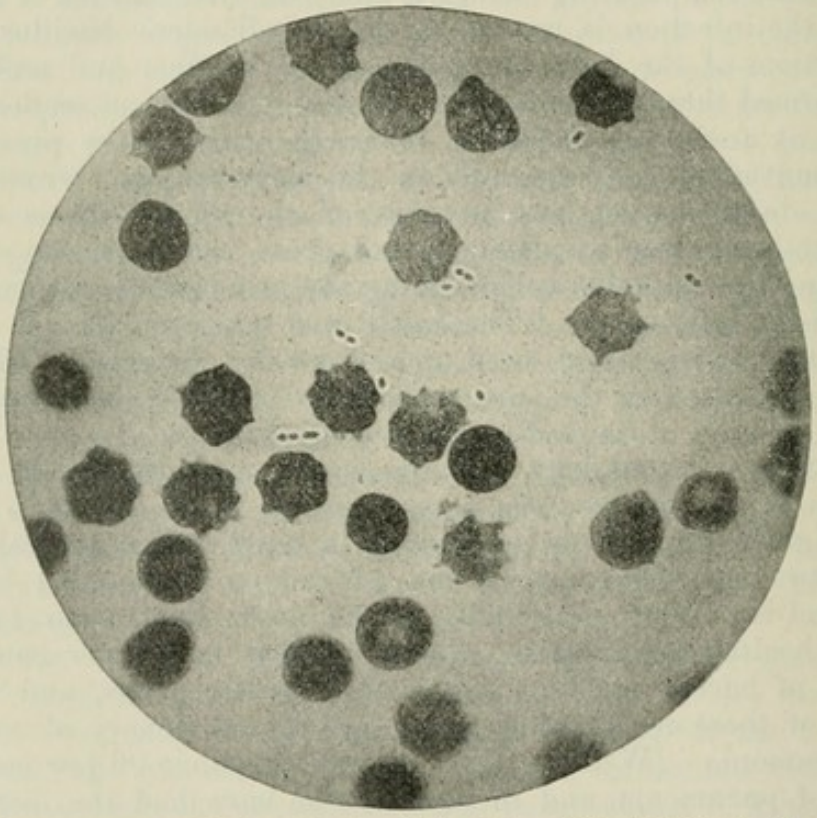


FIG. 10.—Diplococcus pneumoniæ, from the heart's blood of a rabbit; × 1000 (Fränkel and Pfeiffer).

tenement-houses, institutions, etc.) cannot be successfully denied, and we may attribute these outbreaks to defects in the local sanitary conditions.

(2) **Epidemic Influence.**—From time to time pneumonia prevails epidemically. Epidemics are caused by an increased virulence of the organism. Pneumonia may also originate in the endemic form in tenement-houses and institutions, and increase in its scope until it assumes an epidemic character. House epidemics occur, and in the winter of 1894 I saw, with Dr. W. K. Mattern, of Philadelphia, 3 cases develop in rapid succession in one family. A Sister of Charity, after nursing

two of the patients faithfully, also died of the disease. It is possible that the house-epidemic form may spread by *contagion*. An instructive epidemic is reported by W. B. Rodman, who states that 118 cases of pneumonia, with 25 deaths, occurred in a prison population of 735. B. Robinson insists upon his view that pneumonia is contagious.

(3) **Geographic Distribution.**—Pneumonia may be said to be an almost universally distributed affection. Climate, *per se*, does not exercise a notable influence. Delafield, however, points out the fact that in the United States the disease is of more frequent occurrence in the South than in the North.

(4) **Season.**—Of 5905 cases collected by Seitz in Munich, 36.8 per cent. occurred in the spring, 32 per cent. in winter, 15.7 per cent. in autumn, and 15.3 per cent. in the summer. In London most cases appear between the end of March and the end of June (Herringhan). My own analysis of the monthly mortality list covering the decade from 1894 to 1903 inclusive, for Philadelphia, gave the following numerical order: January, 4,210; February, 3,717; March, 3,496; April, 3,039; December, 2,860; May, 2,238; November, 1,936; October, 1,269; June, 1,165; July, 913; September, 826; August, 800.[1] The period of greatest frequency will be sometimes found to correspond in time with the period of the greatest vicissitudes of temperature and humidity. Exposure to cold is incapable, *per se*, of giving rise to pneumonia.

(5) "**Catching cold**" is often followed by pneumonia, but frequently there is no such history. The so-called "cold" is a predisposing cause, rendering the respiratory passages more than ordinarily susceptible to pneumonic infection. Such facts as these also explain why pneumonia occurs with undue frequency in persons following certain occupations.

(6) **Traumatism.**—Following injuries and contusions, especially of the chest, which lower the vital power and resistance of the tissues.

(7) **Age.**—Lobar pneumonia is common at all periods of life, but before two years of age it is comparatively infrequent. Between the ages of twenty and forty susceptibility is increased, and again after the sixtieth year of life it augments rapidly. McDonald[2] reports a case of antenatal pneumonia.

(8) **Sex.**—Males are more commonly attacked than females, the discrepancy in the relative number of cases being greatest from the twentieth to the fiftieth years of age, and being due to the more frequent abuse of alcohol by men.

(9) **Race.**—The negro, American Indian, and the Esquimaux are more susceptible to pneumonia than the white race.

(10) **Unhygienic Surroundings.**—The disease is more frequent among the lower than the higher classes—a fact due to the improved hygienic environment of the latter.

(11) **Circumstances Connected with Individuals.**—The alcoholic is especially prone to this disease, any or all habits that tend to depress the nervous system acting as predisposing causes. The increasing incidence of pneumonia is probably due in a measure to the recognized increase in frequency of the various forms of degeneration of the viscera, particularly of the heart and kidneys. Certain chronic diseases, therefore (chronic Bright's disease, organic heart-affections, carcinoma, diabetes,

[1] "Meteorologic Conditions in the Causation of Lobar Pneumonia," *Amer. Med.*, Sept. 1, 1904.

[2] *British Med. Jour.*, Nov. 11, 1911.

etc.), exert an influence. Emigrants would seem to be more susceptible than persons who have become acclimated.

(12) **Prior Attacks.**—One attack undoubtedly leaves the system more susceptible to the disease, so that repeated attacks—ten or more—may occur in the same individual.

(13) There has been noted a marked increase in the number of cases of lobar pneumonia during the past two decades, due to the prevalence of influenza. Wells has shown by statistical facts that the incidence of the disease has steadily increased during the last century.

Immunity.—The results of the investigations of Behring and Kitasato with the blood-serum of animals which had been immunized against tetanus and diphtheria led Drs. G. and F. Klemperer to experiment upon the lower animals with Fränkel's diplococcus. They found that the rabbit could be rendered immune by intravenous or subcutaneous injections of large amounts of the fluid bouillon-cultures or of the glycerin-extract. From 10 to 20 c.c. of serum taken from a non-receptive animal were injected into the veins of an animal that was suffering from typical pneumonia (artificially produced), whereupon the symptoms subsided rapidly and the animal entered upon a speedy recovery. The same serum, used in a similar manner upon healthy receptive animals, rendered them non-receptive.

Clinical History.—*Prodromes* are rare, and when present consist of a slight general indisposition, lasting a day or more. Rarely, there is cough, thoracic oppression, and slight chest-pains (simple bronchitis), that may or may not be connected with the pneumonic process. Here invasion may be marked by sudden, great thoracic oppression or by a gradual development of the local and general symptoms without rigor.

Usually the *invasion* is *very abrupt*, and marked by a severe *rigor*, which has a duration of from half an hour to an hour, during which period the patient feels most uncomfortable, and is, indeed, very ill. The initial chill may occur at any hour of the day or night, the *fever* rising immediately and rapidly, and the temperature often mounting to 104° F. (40° C.) or even higher in the course of a few hours. The *skin* becomes harsh and dry, the face flushed, and the cheek on the side affected often shows a circumscribed deep-red spot. *Prostration* is pronounced, and headache and other nervous disturbances (restlessness, delirium) accompany and follow the ushering-in symptoms.

The *thoracic symptoms* follow closely upon the termination of the chill. Inspiration, particularly if deep, causes a stabbing *pain* in the affected side; the respirations are hurried, somewhat jerking and shallow (panting), while the pain persists, and later *dyspnea* may become marked, with accelerated breathing. *Cough* sets in early, and is dry and painful during the first day or even longer, and may be attended with expectoration, which generally presents a characteristic *rusty* or *blood-stained appearance*. The *physical signs* rarely appear before the end of the first day, and sometimes as late as the third (central pneumonia); in the latter form the local symptoms, as cough, dyspnea, and sometimes pain, are either wanting or feebly expressed during the first three or four days, and the clinical picture is composed of the general features only.

Anorexia is usually complete; *thirst* is excessive, and commonly there is *vomiting* at the onset, the bowels being generally constipated, though diarrhea may not infrequently be present. The patient in most instances

lies upon the affected side until the pain has in great part subsided, and then he is apt to assume the dorsal position, exposing to full view an *anxious countenance*, with a characteristic *flush* upon the cheek, while the alæ nasi are seen to dilate forcibly during inspiration. Very frequently *herpes* on the lips or nose appears about this time, and forms a valuable diagnostic symptom. The nocturnal remissions are slight, the temperature being of the continued type, and the fever continues high—104° to 105° F. (40.5° C.)—for from five to ten days, and generally terminates by crisis. The pulse is somewhat quickened, but the *pulse-respiration* ratio is not maintained. The other general features last until the crisis occurs, or even increase in severity, but do not outlast this period; many of the local symptoms, however, and particularly pain, are greatly improved before the crisis is reached.

As will be seen hereafter, the general course of pneumonia is modified by a variety of interfering conditions that have relation to complications, individual circumstances, severity of the type, etc. In the instances in which the crisis is reached convalescence is rapidly established. The crisis may be accompanied by special symptoms, as copious sweating or diarrhea.

Leading Symptoms in Detail.—Local or Respiratory Symptoms.—Increased frequency of the respirations is a characteristic symptom, the rate varying from 40 to 60 per minute in adults, and in children from 60 to 90 or more. It is panting in character, particularly when pneumonia occurs in old subjects, and both inspiration and expiration are brief, though sometimes separated by a rather long pause. Expiration is usually accompanied by an audible "grunt," indicating great oppression, and while actual dyspnea is a frequent symptom, it may be absent or as the case progresses may become either increased or greatly diminished according to the severity of the type.

The *chief causes* of the rapid and labored breathing are the involvement of a large portion of the lung, associated general bronchitis, pericarditis or extensive pleurisy, cardiac failure, collateral congestion with edema, fever, and the intense pain in the side.

The *pulse-respiration ratio* is disturbed, the relation now being 1 to 2, or even 1 to 1.5, instead of 1 to 4, as in health (see Fig. 11).

Pain in the affected side is in most cases developed within a few hours after the initial chill, and after lasting two or three days gradually disappears. It is stabbing in character, and usually referred to the region immediately below the nipple or to the axilla, and rarely to other points (abdomen, flank—the so-called *abdominal symptom*). In most instances it is not severe until greatly intensified by the cough, which always aggravates this symptom, as does deep inspiration. The pain is due to implication of the pleura covering the inflamed lung, and may be entirely absent, especially in the aged and those showing marked toxemia.

The *cough*, like the chest-pain and respiration, is somewhat characteristic, being frequent, short, dry, and voluntarily repressed, because it is attended with increased suffering. Yet there are cases that run their entire course without cough—*e. g.*, in the aged and in drunkards.

The Sputum.—At first mucoid and frothy, it soon becomes of a characteristic *rusty* color. It consists of a frothy, fluid mucus containing an abundance of small viscid masses of a yellowish- or reddish-brown

color, from admixture of blood. The chief peculiarity of the sputum in fully developed cases is its *viscidity* and *tenacity*, often adhering to the receptacle even though the latter be inverted; owing to its adhesive quality it is ejected from the mouth with considerable difficulty by the patient. About the time of the crisis the sputum usually becomes more abundant, distinctly purulent, and its expulsion easy, but rarely it may be absent after the crisis. In severe types of the disease it may, at the outset, consist largely of pure blood, and in adynamic forms it is often thinner and darker in color (*prune-juice*). There are cases in which there is an abundance of muco-purulent expectoration when extensive associated bronchitis occurs, and, on the other hand, instances are met with in which nothing is expectorated save a little light-colored mucus. In old persons or in those previously enfeebled there may be no expectoration whatsoever. The amount is therefore exceedingly variable.

Under the microscope the sputum is seen to contain red blood-corpuscles, alveolar epithelium, the *Micrococcus lanceolatus* (usually with other micro-organisms), pus-corpuscles, and small fibrinous casts.

General Features.—The Fever.—As I have already stated, the fever rises rapidly during the initial chill, so that in eight to twelve hours the temperature reaches 104° or 105° F. (40.5° C.). It then remains high until the crisis, pursuing the continued type, with nocturnal remissions amounting to a degree or over, while the daily fluctuations correspond with the normal, except that they are now somewhat exaggerated. In children the rigor is almost always replaced by convulsions. The temperature has a lower average range in persons previously debilitated, in old people, and in drunkards, than in healthy adults and children. During the febrile period there may be observed a pronounced fall of temperature—pseudo-crisis—but the temperature again rises to its former height. This may occur quite early, though more often it precedes the true crisis by a day or two; and rarely it may take place repeatedly, and the temperature-curve bear a strong resemblance to the remittent or even the intermittent type, regardless of any malarial infection. The temperature may be unusually high, 106° F. (41.1° C.) or even 107° F. (41.6° C.), these striking elevations sometimes preceding the crisis (*perturbatio critica*), and hyperpyrexia is often the signal of approaching dissolution. It is especially characteristic of pneumonia, however, that the fever terminates by *crisis;* hence a mere glance at the temperature-chart may serve to complete the diagnosis in doubtful cases (see page 113). The crisis may occur anywhere from the end of the third to the fourteenth day, but in the majority of instances it is on the seventh or the ninth day. The temperature usually falls during the night, and the drop is accompanied by copious perspiration, so that by the following morning the thermometer is found to register at the normal, or more often a subnormal, point (96°–95° F.—35° C.).

The *duration* of the period of decline is usually from eight to twelve hours. It may be much shorter, but more commonly it is longer, or by lysis. The latter mode of termination is often due to some complication. A gradual fall of the temperature in this disease is more common at present than formerly. After the crisis the temperature may remain subnormal, or there may occur a slight postcritical rise; the respiration and pulse-rate quickly return to the normal.

Circulatory Symptoms.—The average pulse rate in typical cases is

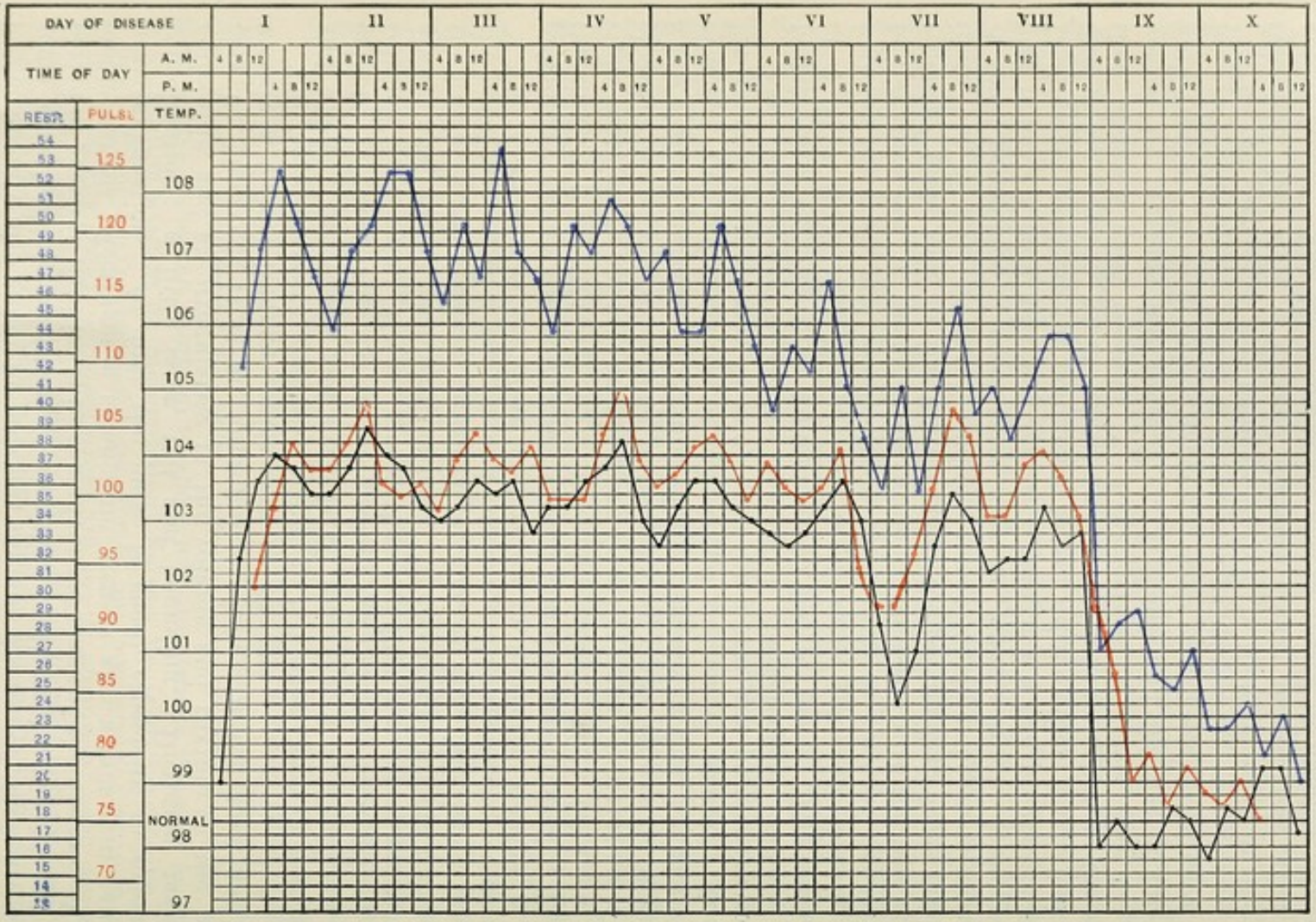


FIG. 11.—Chart of a case of lobar pneumonia with favorable course. A. T., aged thirty-two years; lower right lobe affected. Black, temperature; red, pulse; blue, respirations.

about 100 to 108 per minute, and when it exceeds 120 there is just cause for alarm. The rate may be increased either suddenly or gradually, but in any event augmented frequently implies danger. Cardiac failure is generally due to the effect of the pneumotoxin upon the heart, although less commonly also either to previous organic disease of the heart or to some complicating condition (pericarditis, collateral edema), and the period of greatest liability is in the advanced stage of the disease. Vasomotor paresis affecting the splanchnic area is also a factor in causing heart-exhaustion. At first the pulse is small, especially in extensive consolidation; a little later, full and bounding. Dicrotism may be noticeable, and an irregularity in the volume and rhythm of the pulse may be observed; it is an unpropitious sign. In the aged and the weakly a feeble, frequent pulse may be present.

The *blood-pressure* generally begins to fall after three or four days, and when it progresses and exceeds 25 mm. Hg. it is significant and calls for increased stimulation. A prompt fall indicates approaching dissolution, as a rule. Brem[1] states that the first sign of exhaustion is always a fall of the peripheral tension. MacKenzie points out that when the line of blood-pressure, measured in mm. of Hg., falls below that of the pulse-rate, there is danger, and vasomotor stimulants are required.

The *heart-sounds* are clear, and owing to increased tension in the pulmonary vessels the pulmonary second sound is accentuated. This is the state of things throughout in favorable cases. With failure of the right ventricle (a not rare event) there arise the signs of dilatation of this chamber (extension of cardiac dulness to the right, epigastric impulse, a low systolic murmur, shortening of the diastole, or fetal heart-sounds, cyanosis, and indistinctness of the second pulmonary sound). A soft, low-pitched murmur may be audible in the mitral and pulmonary zones.

The *blood-appearances* are somewhat characteristic. There is a leukocytosis varying from 10,000 to 40,000 or more. The reseaches of Lache[2] show that leukocytosis is of some value in determining between the crisis and pseudo-crisis, continuing in spite of the fall of temperature, etc., in pseudo-crisis, while it disappears with the true crisis. Stienon[3] finds that in the febrile stage the polynuclear forms predominate, but as soon as these diminish the eosinophiles begin to increase. A small percentage of myelocytes may be found. Slight leukocytosis may indicate a mild infection, but, as a rule, it is a bad prognostic sign. Leukopenia occurs in the malignant cases; on the other hand, leukocytosis of high degree, while indicating a severe infection, "it, at the same time, shows a good reaction."[4] Leukocytosis, however, may be prevented by previous infections (*e. g.*, typhoid) and the use of internal antipyretics. Ludwig Jehle[5] reports 6 cases of the agglutination of the pneumococcus by the serum of pneumonia patients.

The red corpuscles and hemoglobin remain little changed during the fastigium, but show a marked decrease almost immediately after the actual crisis.[6] The blood-plates are also increased in number (Hayem). Da Costa has collected 9 cases of phlegmasia alba dolens in pneumonia.

[1] *Johns Hopkins Hosp. Bull.*, 1905, xvi., 321.
[2] *Berliner klin. Woch.*, 1893, Nos. 36 and 37.
[3] *La Presse méd.*, 13, 1895.
[4] E. Becker, *Deutsch. med. Woch.*, Aug. 30, 1900.
[5] *Wein. klin. Woch.*, 1903, No. 32.
[6] Sadler, *Fortschritte der Medicin*, 1892; Leichtenstein, *Ueber der Hämoglobin-gehalt des Blutes*, etc., Leipzig, 1892.

Cerebral Symptoms.—*Headache* sets in early and may be a prominent and persistent feature. In many cases, and particularly in children, the disease is ushered in by convulsions, this symptom occurring more often in the apical than in the basilar form of pneumonia. *Delirium* may come on during the acme of the disease (rarely, it may start as an acute mania), and may assume a maniacal form, but oftener in my experience consciousness has been retained. In the drunkard *delirium tremens* usually develops, and may anticipate the symptoms referable to the lungs; and I fully agree with Osler in stating that it should be an invariable rule, if fever be present, to examine the lungs in delirium tremens. These cases may often be appropriately termed "*walking pneumonia*," since they go about until excitement gives way to a coma that deepens into death. In *adynamic forms* a low, muttering delirium and coma are frequent.

In the so-called *cerebral* pneumonia the nervous phenomena are quite pronounced, and simulate closely cases of cortical meningitis. It is often associated with excessively high fever, except in the aged, when the cerebral symptoms are also well marked, but the fever is moderate. Apical pneumonias are apt to assume the cerebral type, but in my experience this dictum is correct as relating to children only. Double pneumonias are commonly characterized by severe cerebral symptoms.

The Cutaneous Symptoms.—As stated before, herpes is common and its diagnostic importance is considerable. *Naso-labial herpes* is but little less frequent in this disease than in malaria, being present in about one-third of the cases. It usually comes out from the second to the fifth day of the disease, and rarely may appear upon the cheek, lobe of the ear, the genitals, forearm, or upon the mucosa of the tongue. Sweats are not common except at the time of the crisis, when they may be copious. The deep-red circumscribed spot upon one cheek (*mahogany flush*), usually on the side of the affected lung, has already been mentioned. Urticaria has been observed, though rarely.

Digestive System.—The mouth is dry, the tongue has a coating of a yellowish-white color, becoming dry and brown in cases representing a low form, and anorexia and thirst are present. *Vomiting* is not uncommon at the outset, and may be repeated, while constipation is the general rule and diarrhea the frequent exception. Sears and Larrabee[1] in an elaborate analysis of 949 cases found that pain below the costal margin was frequently present, and in several cases appendicitis, especially when the pain was associated with muscle spasm. Splenic enlargement of slight degree can usually be detected on palpation.

Urinary Symptoms.—The urine is febrile, diminished in amount, and high-colored, the urea and uric acid being greatly in excess. On the other hand, the chlorids are, according to the older authors, either diminished in amount or absent during the febrile stage, presumably for the reason that they pass into the inflamed lung-tissue. They are not, however, constantly absent, and sometimes they are not even lessened, in pneumonia; moreover, their disappearance is not peculiar to this disease. The above-mentioned facts justify two important inferences: (1) The absence of chlorids is a symptom of little diagnostic value; and

[1] *The Med. and Surg. Reports of Boston City Hospital*, Twelfth Series, Dec. 1, 1901.

(2) their reappearance in the urine toward the close of pneumonia is of small prognostic worth. Slight (febrile) albuminuria is common.

Physical Signs.—Stage of Congestion.—The density of the lung is increased, but the involved tissue is not consolidated and the pleura is not yet covered with fibrin.

Inspection.—The movements of the affected side (especially if the base be involved) are defective, the degree of expansion being much diminished. In double pneumonia the costal type of breathing, combined with a vigorous play of the abdominal muscles, is observed.

Palpation.—There is a slight increase in the tactile fremitus over the congested area, and defective expansion is noted.

Percussion.—The note may be normal, though more often it is briefer, higher-pitched, or even distinctly tympanitic.

Auscultation.—The breath-sounds are weak, and sometimes become broncho-vesicular upon deep inspiration, while over the unaffected lung-tissue they are exaggerated. If, as often happens, inflammatory products due to associated bronchitis occupy the small bronchi, subcrepitant râles may be audible. The crepitant râle, however, is rarely heard until the close of the first stage.

Stage of Consolidation.—*Inspection.*—There is little or no expansive motion of the chest over the affected area, while upon the unaffected side it is increased. The volume of the thorax on the diseased side is increased, as shown by mensuration, but the intercostal depressions are not effaced.

Palpation renders clearly perceptible the defect or absence of expansion. Vocal fremitus is usually much increased, though in exceptional instances it is diminished or absent—a circumstance which can, as a rule, though not invariably, be attributed to an associated pleurisy with more or less effusion. Frequently a friction-rub is felt before complete consolidation is established.

Percussion.—Varying degrees of dulness are obtained in this stage, and before the lung-tissue becomes thoroughly solidified the note may have a tympanitic quality. After complete consolidation there is usually marked or absolute dulness posteriorly, unchanged by full inspiration, while the note may be more or less tympanitic anteriorly, where the vibrations are more apt to reach the air in the larger bronchi. A sense of resistance is offered to the plexímeter-finger, but not to the same degree as in the case of a pleurisy with effusion. When the latter condition is associated and in massive pneumonia the percussion-note will be flat. Deadness is less marked in old people in whose ribs senile changes have taken place, which render them more resonant, or in cases in which the consolidated areas occupy the central portions of the lung. Above the solidified part Skodaic resonance is usually obtainable.

Auscultation.—Bronchial or tubular breathing is heard, as a rule, over the solidified lung, but it may be absent in consequence of the plugging of the large bronchi with exudate (so-called *massive pneumonia*). Bronchophony is usually obtainable over the portion of the lung affected, though this may also be absent, and for the same reason as in the case of the bronchial breathing: it sometimes takes the form of egophony. Subcrepitant râles, due to associated bronchitis, are sometimes heard with unusual distinctness (owing to the consolidation), and the crepitant râle at the end of inspiration is best heard at the beginning of consolidation, when the pleura receives its coat of fibrin

and while the lung is yet capable of sufficient movement to produce fine pleural friction. A distinct friction-rub may also be heard occasionally.

Stage of Gray Hepatization.—With beginning resolution the solid contents of the air-cells liquefy and are removed, so that air now re-enters the air-cells and permits a consequent increase in the movement of the lung.

Inspection.—The normal expansile movement gradually returns.

Palpation.—Tactile fremitus progressively diminishes.

Percussion.—The dull or tympanitic quality of the note is gradually lost, though the fact must be emphasized that the abnormalities in the note vanish more slowly than the other abnormal physical signs. Some degree of deadness often remains long after apparent recovery.

Auscultation.—With increased movement of the lung there may be a reappearance of the crepitant râle, due to interplay of the pleural surfaces, and the softened exudate in the air-cells gives rise to subcrepitant râles, heard both on inspiration and expiration (*râle redux*), with coarser râles over the bronchi. Bronchial breathing gradually gives place to broncho-vesicular, and the latter in turn to normal, breathing.

The Pneumococcus Septicemia.—The pneumococcus infection may cause severe toxic features and even speedy death without any, or with but little, involvement of the lung-texture. The general *invasion symptoms*, such as the chill, high fever, and nervous symptoms which always predominate, however, are present and persist until death ends all. Death is preceded by signs of cardiac failure, by vasomotor paresis, or, more rarely, by coma. In some of these cases localization of the morbid process may occur in organs other than the lungs, as the cerebral meninges, the endocardium, pericardium, and the pleura. An assured diagnosis in these atypical forms of the pneumococcus infection can be arrived at by a bacteriologic examination of the exudate obtained by aspiration. The pneumococcus can also be demonstrated in blood-cultures, provided that they are made with large quantities of blood.

Complications.—Many of these are due to the primary infection.

Pleurisy is, of necessity, associated in all instances in which the consolidation reaches the pleura. In most cases the presence of the diplococci has been demonstrated. Cases are met with in which the truly pneumonic symptoms are overshadowed by the intensity of the pleuritis and to these the term *pleuropneumonia* has been applied. There is often a copious effusion which is exceedingly rich in fibrin—a circumstance which distinguishes it from other forms of acute pleurisy. There may be the ordinary grade of pleurisy on the side of the pneumonia, and a severe grade on the opposite side which is apt to be purulent. Indeed, empyema has of late been shown to be a frequent complication of pneumonia, and it also occurs as a sequel. A condition that affects its incidence is involvement of the lower right lobe. Lambert and Daly[1] report 5 cases of empyema developing as a complication in lobar pneumonia, which showed a sudden rise of leukocytosis to nearly or more than double the count of the previous day. Its development is accompanied by replacement of ordinary dulness by flatness with great resistance, and by the disappearance of râles and breath-sounds, normal and abnormal. Other characteristic features of empyema are present, but if doubt surround the diagnosis, the needle should be introduced.

[1] *St. Paul Med. Jour.*, Dec., 1902.

There is a prompt rise of fever, the temperature leaping to 103° or 104° F. (39° or 40° C.) quickly, after which it is decidedly remittent in type, but there are no hectic chills. Fistulous connection with a bronchus, however, and the establishment of *empyema necessitatis* are common events, and may be preceded by diurnal chills, sweats, etc.

The occurrence of *septic phenomena* is a certain indication of secondary infection by streptococci. The pus is rarely absorbed and frequently becomes encysted. I saw one instance in which the effusion measured 8 liters, while ordinarily the amount ranges from 2 to 5 liters. Removal of the effusion by aspiration is promptly followed by the disappearance of the fever, but reaccumulation generally occurs.

Finally, if defervescence in pneumonia takes place by lysis or if an irregular fever persists, a residual purulent or sero-fibrinous effusion may be considered as the likely cause. This latter complication is attended by a paroxysmal cough which is excited by movement, and is not usually accompanied by expectoration.

Acute general bronchitis may pre-exist or arise as a complication, and often proves formidable, intensifying the fever and increasing the dyspnea, the tendency to heart-failure, and the cyanosis. The expectoration is freer than in uncomplicated pneumonia, and over the bronchi moist râles intermingled with sibilant and sonorous râles are audible.

Pericarditis.—This is an important and serious complicating affection. According to Chathard, it is oftener synchronous with involvement of the right than of the left lung, hence arises as often by a metastatic process as by direct extension. It was present in 4.66 per cent. of 665 cases and occurred most frequently in young adults. Although generally of the plastic variety, it is not infrequently sero-fibrinous, and rarely the effusion is purulent. The *diagnosis* can be made here as under other conditions (pericardial friction rub, etc.), but the complication is often insidious. The occurrence of increased dyspnea, with or without precordial pain, should serve as a signal and lead to a physical examination.

Endocarditis.—This is somewhat more frequent than pericarditis. Preble [1] has well said that endocarditis should always be suspected in a case of pneumonia, which is followed by an irregular temperature not sufficiently accounted for by some other complication, such as empyema. Out of 209 cases of malignant endocarditis collected by Osler, 54 cases occurred in pneumonia. Endocarditis complicates pneumonia in 1 per cent. of all cases and in 5 per cent. of the fatal cases (Preble). It is generally of the malignant type and may attack any valve (the aortic leaflets, however, being most commonly affected). There are no reliable symptomatic indications of this condition. The physical signs must be faithfully and systematically noted. Frequently murmurs are absent; and, on the other hand, the presence of a murmur alone is by no means diagnostic of the complication. Bradycardia is not uncommon, but oftener the pulse is rapid and feeble. The development of septic manifestations, especially irregular fever, chills, and sweats, renders the case highly suspicious, and when in addition there arises distinct evidence of embolic processes the diagnosis becomes highly probable. If, now, the symptoms of meningitis supervene, little doubt remains as to the character of the complications, since meningitis and endocarditis are often combined in pneumonia.

[1] *Amer. Jour. Med. Sci.*, Nov., 1904.

Netter, Weichselbaum, and Bignami have shown that acute endocarditis may be caused directly by the diplococcus of pneumonia.[1]

Chronic Endocarditis.—Pneumonia arising in the course of chronic endocarditis is apt to be attended by cardiac failure, with ensuing venous stasis. The murmurs of chronic valvulitis often disappear with the development of pneumonia.

Cardiac clots (ante-mortem) may form, but are rare. They result from weakness of the ventricular wall, especially in the right heart; and are most apt to arise, therefore, in cases in which the preagonal period is much prolonged. Venous thrombosis is rarely seen, and embolism of the larger arteries is a rare complication, the lesions and determining factors being similar to those in typhoid fever. The cerebral embolism, causing aphasia and even hemiplegia, has been observed, but seldom.

Pneumococcus meningitis may be a complication; it differs only in the bacteriologic findings from meningococcus meningitis. Pneumococcus meningitis may also occur independently of lung involvement.

The symptoms are not clearly defined; particularly is this true when it develops during the invasion-period and the basilar meninges are not involved. The presence of intense and persistent headache, rigidity of the nucha, wild delirium, followed by stupor, deepening into profound coma, affords a basis for a probable diagnosis. Its frequent association in the purulent form with ulcerative endocarditis has been pointed out above. The cerebrospinal fluid contains no sugar in this condition.

Peripheral neuritis is among the rare complications of this disease.

Parotitis is also sometimes seen, and may cause a fatal termination of the case. I have seen two instances, however, in which this was a complication, and both ended in recovery. It is thought to be associated usually with endocarditis, but not so in my cases.

Arthritis.—A pneumococcic arthritis occurs, but it is rare. The joint is occasionally primarily involved, showing the importance of toxemia (Herrick). It is most prone to develop after the crisis, and is associated with meningitis and endocarditis. The exudate is generally a thick creamy pus, less commonly sero-fibrinous. The changes may be either slight in the acute forms, or extensive and destructive of cartilage and bone, particularly in the more chronic cases. A recognition of the condition demands exploratory aspiration and bacteriologic examination. The mortality-rate is 65 per cent. *Rheumatism* and *otitis media* may be rarely met also, particularly in children.

Gastro-intestinal Complications.—*Croupous gastritis* may rarely intervene. Fussell[2] calls attention to complicating acute dilatation of the stomach; it causes vomiting, sudden epigastric distention becoming general, and collapse, which is an urgent symptom. *Croupous colitis* is a frequent concomitant, sometimes grave, giving rise to tympanites and diarrhea.

Peritonitis occurs, but with great rarity.

Jaundice may be observed; it is more frequent in serious than in mild forms of the disease. It is rarely intense, and is most probably an obstructive (hepatogenous) jaundice. N. V. Pétrov has reported 13 cases complicated with icterus, and in all observed local lesions (mainly catar-

[1] *Practitioner*, London, Aug., 1894.
[2] *Amer. Jour. of the Med. Sci.*, Dec., 1911.

rhal) of the duodenum and the biliary canals. A catarrhal or suppurative cholecystitis may rarely complicate lobar pneumonia.[1]

Acute nephritis is a complication, and its recognition is dependent upon the discovery of albumin and casts in the urine. In 20,107 cases of lobar pneumonia, acute nephritis occurred in 263, or 1.3 per cent. (Norris).

Clinical Varieties and Anomalous Types.—(1) **Typhoid Pneumonia.**—This relates to an adynamic, serious type of the disease with *typhoid symptoms*, and not to typhoid fever. It is often secondary to low fevers, to septicemia, diabetes, and chronic nephritis, and is also the variety met with in drunkards and in persons previously enfeebled. The onset is somewhat gradual. The physical signs may be ill defined, but the general features are always striking and characteristic. Prostration is extreme; there are delirium and often stupor; the temperature may or may not be high; while the respirations and pulse are almost always frequent. The skin is dry, and may show a dusky tint or slight jaundice. The tongue is dry, often brown, and vomiting is common; the sputa may be rusty or decidedly hemorrhagic. Splenic enlargement is often clearly perceptible. When recovery ensues convalescence is tedious. Some of the cases belong in the category of atypical pneumonias.

(2) **Epidemic Pneumonia.**—This is often of malignant type. The symptoms exhibit noticeable variations, according to the special etiology and to different epidemics. The pneumonias of *epidemic influenza* are complicated with or preceded by general bronchitis. The heart-power often becomes exhausted early, and then follow congestion and edema of the lungs. The physical signs are often slight.

The so-called *serous pneumonia* often complicates influenza; it is ascribed to streptococcus infection (*streptococcus pneumonia*). Septic phenomena often arise, such as irregular fever and sweats. The physical signs, for a time indefinite, when fully developed resemble those of bronchopneumonia. There may be a tendency to migration from one to the other lung. There may be a late-appearing rusty expectoration, and in some cases the sputa are mucopurulent throughout. The course is often protracted, and the fever may terminate by lysis. In two of my cases the pneumococcus was detected in the sputum in increased numbers. It is said that in mixed infection the micrococcus lanceolatus is abundantly present. In so-called "*larval pneumonia*" the general symptoms are mild and the local signs ill defined. The epidemic outbreaks that occur in institutions, tenement-houses, jails, etc., belong to this variety.

(3) **Latent Pneumonia.**—To this class belong *central pneumonias*. The sputum is to be stained and examined microscopically, when the pneumococcus will be found. The sputum is gummous and rusty, as a rule. When pneumonia arises in the course of emphysema the dilated air-cells are not filled with the exudate; hence dulness is less marked, and true tubular breathing may be absent. Before the crisis occurs consolidation usually advances to the periphery.

(4) **Migratory Pneumonia.**—By this is meant an extension of the specific inflammation to other parts of the lungs. Such extension may prevent the occurrence of the usual crisis, and often occasions an exacerbation of the general pneumonic features.

[1] "Cholecystitis as a Complication of Lobar Pneumonia, with a Report of Three Cases, and Remarks on Icterus in Pneumonia," by the writer.

(5) **Bilious Pneumonia** (" *Malarial Pneumonia* ").—In pneumonia occurring in malarial subjects the initial chill is prolonged and the fever paroxysmal or remittent. Jaundice and vomiting are common.

(6) In **children,** the first symptom is often a convulsion. Cerebral symptoms (delirium, stupor, coma) may appear early. The upper lobes of the lungs are frequently involved. Unless the objective indications be examined for, the disease is frequently overlooked. The characteristic sputum is rarely seen in juvenile pneumonia. Crozer Griffith reports 8 cases in which the pain suggested appendicitis.

(7) In **old persons** the initial chill is often absent or replaced by moments of chilliness. There may be nausea and vomiting. Prostration is profound; there is fever, but it does not range high and is irregular. Nervous phenomena, sometimes prominent, are not uncommon, but the local symptoms (cough, expectoration, and pain) are mild or wholly absent. The physical signs are defective owing to impairment of the respiratory movements; dulness on percussion (with a tympanitic quality), tubular breathing, and a few subcrepitant râles may, however, be noted. This affection is a most deceptive one in old people, the cases generally ending fatally after an illness of an apparently mild degree of intensity.

(8) **Abortive pneumonias** last no longer than twenty-four or forty-eight hours. The general features are rigor, high fever, and defervescence by crisis with profuse sweating. The sputum is rarely characteristic, and the physical signs variable; typical tubular breathing is rare, while râles and pleural involvement are common. Bechtold[1] has frequently observed this form affect all the members of a family.

(9) **Terminal Pneumonia.**—Many instances of pneumonia are discovered in the post-mortem room. These arise in advanced cases of chronic pulmonary tuberculosis, organic heart diseases, chronic Bright's disease, diabetes, and the like, and manifest no clinical symptoms other than slight elevation of temperature, an increase in the respirations, and lung consolidation. A fatal termination is the rule in terminal pneumonia.

(10) **Ether-pneumonia.**—Opinions are divided as to the frequency of occurrence of pneumonia after ether-narcosis. The aggregate number of cases from all sources (57,842) gives a percentage of 0.07. My own statistics, embracing 12,842 cases, give a percentage of 0.23.[2]

The principal causes are—(*a*) Season. According to my investigations, over 80 per cent. of the cases occur during the winter and spring months. The patient is sometimes carried from a heated operating-theatre through a cold corridor to a room or ward with a lower temperature. (*b*) "Catching cold," or exposure as may obtain during protracted operations. (*c*) Bronchitis, coryza, and the like present at the time of anesthesia. (*d*) Dried secretions or incrustations of foreign matter that are loosened by the ether and drawn into the lungs. (*e*) Abdominal operations give the highest percentage of cases, due, as my studies show, to the more protracted etherization. Mikulicz has shown that ether-pneumonia following these operations is caused by embolism. (*f*) Graves[3] believes that most cases are caused by the lighting up, or aggravating of, pre-existing foci in the lungs.

The *clinical features* are aptly compared with those of secondary pneumonia (*vide* p. 130). The *diagnosis* rests principally upon the typical

[1] *Münch. med. Woch.*, No. 44, 1905. [2] "Ether-pneumonia," *Univ. Med. Mag.*, Aug., 1898.
[3] *Boston Med. and Surg. Jour.*, Sept. 29, 1910.

physical signs. Owing to the extreme latency of the condition, and the danger that the symptoms may be regarded as being septic in nature, I would emphasize the importance of a physical examination of the thorax upon the sudden accession of *fever*, particularly if associated with thoracic *pain*, however slight, following an operation.

Relapses.—These are rare, and are usually rudimentary. *Recurrences*, however, are ordinary (*vide* Predisposing Causes, p. 110).

Course and Duration.—In most instances, the crisis occurs on the seventh or ninth day, and resolution is completed about one week later, making the total duration from twelve days to two or three weeks. Convalesence, however, may be delayed by complications or sequelæ, and fatal cases are apt to terminate on the seventh, eighth, or tenth day.

Sequelæ.—(*a*) **Delayed Resolution.**—The process of resolution may

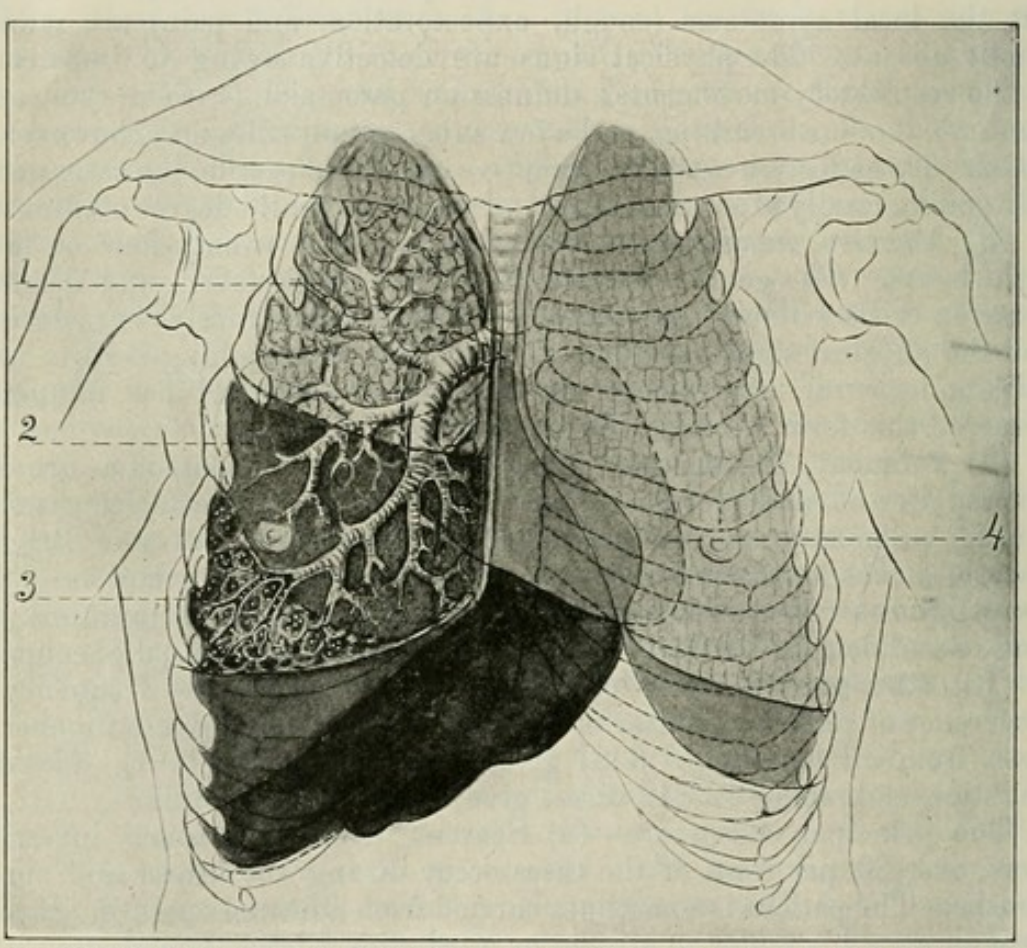


FIG. 12.—Lobar pneumonia: 1, unaffected area (upper lobe); 2, consolidated area (middle lobe); 3, resolving area (lower lobe); 4, heart in normal position.

not begin until the fourth, sixth, or even tenth week. Usually defervescence by crisis has taken place long before the physical signs indicate resolution; the fever may, however, fall by lysis. When resolution occurs it may lead to complete restoration of the anatomic entirety of the lung-tissue. Delayed resolution is often confused with certain sequelæ, especially empyema. Rarely proliferation of the interstitial connective tissue arises in postponed resolution, producing (*b*) **chronic interstitial pneumonia.** (*c*) **Abscess**; (*d*) **gangrene**; and (*e*) **tubercular phthisis** are also sequelæ.

Diagnosis.—The diagnosis is determined by special local and general symptoms, together with the physical signs. Of these, the abrupt onset with rigor, the course of the fever with termination by crisis, the

abnormal pulse-respiration ratio, the cough, the rusty expectoration, expiratory "grunt," leukocytosis, and the signs of lobar consolidation, are the most characteristic. Repeated physical examinations of the chest will often detect consolidation, in the absence of the accustomed symptoms. Again, when in the course of certain chronic affections (cancer, Bright's disease, diabetes, and organic affections of the heart) fever is developed, physical exploration of the thorax is imperatively demanded.

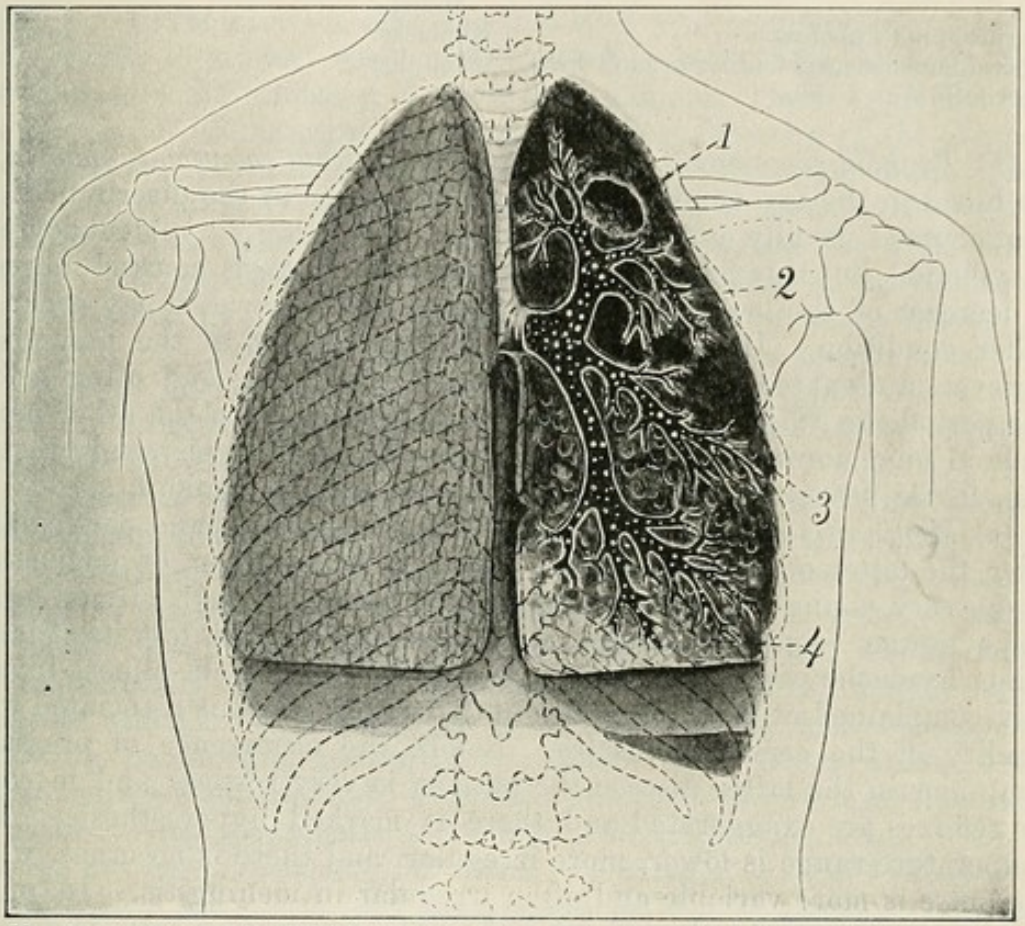


FIG. 13.—Acute pneumonic phthisis, posterior view: 1, cavity; 2 and 3, consolidation; 4, infiltration; the white spots indicate râles.

Differential Diagnosis.—This relates to (*a*) acute pneumonic phthisis, (*b*) pneumo-typhoid, (*c*) meningitis, (*d*) broncho-pneumonia, (*e*) acute pleurisy with effusion.

(*a*) PRIMARY LOBAR PNEUMONIA.	ACUTE PNEUMONIC PHTHISIS.
There may have been prior attacks.	Inherited predisposition or previous tuberculous disease.
Sudden, with severe rigor and rapid rise of temperature.	Generally more gradual—repeated fits of chilliness (rarely severe rigor), often following exposure or "cold."
Fever of continued type, terminating by crisis.	Fever of remittent type, often becoming intermittent, without crisis.
No drenching sweats, except at time of crisis.	Drenching sweats present and oft repeated.
Herpes common.	Absent.
Not much emaciation.	Rapid emaciation.
Pulse-respiration ratio much disturbed.	Less so.
Sputum rusty-colored, viscid, and sticky; contains pneumococcus.	Sputum may be blood-tinged; is more purulent and copious, and contains numerous bacilli and yellow elastic tissue.
Leukocytosis present.	Relative lymphocytosis.
Duration of febrile stage shorter.	Duration longer.

(*a*) Primary Lobar Pneumonia.	Acute Pneumonic Phthisis.
Physical signs, as a rule, first referable to base of lung.	First referable to apex.
Usually limited to one lobe or the lower segment of one lung.	Usually extension from apex to base.
Signs of consolidation, followed by resolution.	Signs of consolidation, followed by cavity-formation, with large gurgling râles at apex.
Apex of opposite lung not involved.	Apex of opposite side generally invaded.
Prognosis not hopeless.	Hopeless.
Tuberculous disease of other organs does not follow as a rule.	Often does.

(*b*) *Typhoid pneumonia* must be diagnosed from *pneumo-typhoid*, and the blood in the two conditions may be of service in the discrimination. Leukocytosis usually exists in pneumonia, and there is hypoleukocytosis in typhoid; but this fact is only of value when there is marked increase or decrease of the leukocytes, since figures about normal may occur in either condition. In pneumo-typhoid, after the end of the first week, however, undoubted symptoms of typhoid fever arise, and often before this period the Widal test will clear the diagnosis. On the other hand, typhoid pneumonia is characterized especially by great physical prostration, feeble heart-action, and other symptoms of the typhoid state.

(*c*) *Meningitis* is sometimes mistaken for pneumonia, and particularly when the latter occurs in children. The initial symptom of pneumonia in the very young is often a convulsion; whereas, though in meningitis this symptom is not uncommon, it is more apt to manifest itself later. When headache occurs in pneumonia it is frontal. It is almost invariably complained of in meningitis, but is occipital, and is associated with rigidity of the cervical muscles. Before the occurrence of pressure-symptoms in the latter disease the patient is very restless and morose; his reflexes are exaggerated and there is marked hyperesthesia. The temperature-range is lower, more irregular, and there is no crisis, while the pulse is more variable and often irregular in meningitis. In pneumonia with latent local symptoms the pulse-respiration ratio is greatly altered and the type of respiration peculiar (*vide ante*). The important rule, to examine for the physical signs in doubtful cases, must not be neglected, and if the subject be young the apex region in particular.

The differential diagnosis between pneumonia and broncho-pneumonia and pleurisy with effusion will be found on pages 558 and 592.

Prognosis.—The mortality from pneumonia in hospitals averages about 25 per cent. It is less in private practice—about 15 per cent. The death-rate, however, is greatly modified by the type of the individual epidemic; hence a precise statement as to the percentage of fatal cases cannot be ventured. Wells collected 223,730 cases, which gave a mortality of 18.1 per cent.

The elements that enter into a correct prognosis are in the main identical with those in other acute infectious diseases, and concern (1) the severity of the type of infection, (2) the presence or absence of complications, and (3) circumstances peculiar to the individual.

(1) **Severity of the Type of Infection.**—In sthenic cases this is shown by (*a*) the temperature-range, (*b*) the degree of heart-power, (*c*) the intensity of the nervous symptoms, and to some extent by (*d*) the size of

the area of lung-induration. It is a matter of common observation that the absence of leukocytosis is indicative of a grave type. In case the diplococcus be found in the blood, the prognosis is by most writers considered grave, and it is to be recollected that with the improved technique of the present day this organism is readily isolated. (*a*) *The Temperature-range.*—A continued high temperature, as, for example, 105° F. (40.5° C.), on two or three consecutive days without material remissions, is ominous. (*b*) *The Degree of Heart-power.*—A steadily rising pulse-rate after the fifth day indicates real danger, since it points indisputably to gradual cardiac failure. The same thing is shown by a diminution in the intensity of the second pulmonary sound; it indicates the giving out of the right ventricle. (*c*) *The Intensity of the Nervous Symptoms.*—Active delirium is not favorable at any stage, and is particularly unfavorable if it develop early. When it assumes the form of delirium tremens the case has usually passed beyond the hope of recovery. (*d*) *The Size of the Area of Lung-induration.*—I have observed that extension of the consolidation at an advanced stage belongs to serious types. The same may be said of double basic pneumonias.

Typhoid pneumonia, being of low type, gives an unfavorable prognosis, notwithstanding an absence of high temperature and of extensive inflammation of the lung-texture.

(2) **Presence or Absence of Complications.**—Cases in which there is involvement of a single lobe or two lobes, if it occur on the right side and without complications, generally terminate in recovery. In nearly one-half of the instances complications occur, and these greatly increase the death-rate. Among the most common is *pleurisy*, which, unless accompanied by considerable effusion, does not add fresh danger; when pleurisy attacks the non-affected side, however, it does. *Empyema*, following pneumonia, generally terminates in recovery unless septic phenomena are superadded. Extensive *bronchitis* is a most perilous complication in my judgment. *Pericarditis* decreases the chances for recovery, but by no means to the same extent as ulcerative *endocarditis*. *Cardiac clots* may form, but usually the patient is already moribund. *Abscess of the lung* and *gangrene* form highly unfavorable complications. *Congestion* and *edema* of the uninvaded portions of the lungs render the outlook bad, and these, together with cyanosis, are apt to be dependent upon failure of the right heart. *Acute meningitis* is exceedingly grave. Fenwick, as the result of an analysis of 10,000 cases, found that the quantity of albumin in the urine is of considerable prognostic value. *Gastro-intestinal* complications occurring at the outset are unpropitious.

(3) **Circumstances Connected with the Individual.**—Of these *age* heads the list, and after the twentieth year the mortality increases progressively until the seventh decade, when it rises more abruptly. It has been claimed that nine-tenths of the deaths after the seventy-fifth year are from lobar pneumonia. Under the twentieth year, according to the analysis of 708 cases at St. Thomas's Hospital by Hadden, H. W. G. Mackenzie, and W. W. Ord, the mortality is 3.7 per cent.

Sex has little influence. Napier's figures indicate that pneumonia is a more deadly disease in men than in women. The *alcoholic* rarely escapes death, and *adiposity* is an unfavorable condition.

Modes of Death.—Death is due to: (1) overwork or overdistention of the right ventricle; (2) from mechanical interference with respiration (rare); (3) pneumococcus infection of other organs, as the meninges, pleura, pericardium, endocardium; (4) pneumococcus toxemia and septicemia as shown by the typhoid state, *progressive heart weakness*, tympanites, and diarrhea; (5) vasomotor paresis is often the cause of death.

Treatment.—General Management.—The patient should be isolated in a well-aired apartment. Fresh air constantly breathed improves the appetite, lessens cough, diminishes the temperature, pulse-rate, and respiration-rate; in short, a less marked toxemia is observed than in patients treated by the more usual method.[1] Spolverini[2] points out that the pneumococcus in the sputum may remain virulent from fifty-five to one hundred and forty days, hence it is important to sterilize pneumonic sputum. An antiseptic mouth-wash should be advised. In severe forms the constant presence of a physician is required. The patient must be kept at *perfect rest*, and not allowed to leave his bed for at least one week after the occurrence of the crisis. The beneficial effects of rest, in the fullest sense of the term, are not appreciated to the extent they deserve. Perhaps the principal object is to support the powers of life until the crisis is passed.

The **diet** should be light, chiefly liquid, but of the most nutritious sort. *Milk* should constitute the chief article of diet; meat-broths or meat-juices, egg-white, and light farinaceous substances may also be allowed. The food, particularly the milk, is to be administered at stated brief intervals and in definite quantities. When resolution is delayed stronger forms of nourishment (scraped meat, etc.) may be given. After the crisis a gradual return may be made to the usual forms of solid foods. Page[3] and others advocate abstinence from practically all nourishment *except water*. Alcohol has considerable food-value; it lessens waste and improves the appetite and digestion. To accomplish this object, it should be given in small amounts, two or three ounces daily.

The *medicinal treatment* is that of a *toxemia*, although the patient himself is the main factor.

The use of calomel in fractional doses or one of the saline laxatives in the early stage is an important procedure. Subsequently the liver and bowels must be kept acting freely, so as to eliminate waste products and toxins, and to obviate "absorption of fermentative products from the alimentary canal" (Thornton). The action of the kidneys is best maintained by the regular use of water and that of the skin by sponge-baths. Stockton advises stimulation of the eliminative organs—skin, liver, kidneys—in senile pneumonia.

Cardiac stimulants are often indicated. It is well to begin their use as soon as the slightest tendency to cardiac failure is shown. When the pulse becomes more accelerated and feeble, the first sound of the heart less distinct, and the second pulmonic sound loses its accentuated character, or marked nervous symptoms or adynamia appear, then alcoholics must be used. At first they are to be employed in moderate doses (½ ounce—16.0—of whisky or brandy every three hours), to be increased

[1] *Medical Record*, 1906, No. i, p. i, by the writer.
[2] *Centralb. f. allg. Path. u. pathol. Anat.*, July 18, 1900.
[3] *Medical Record*, Dec. 23, 1905.

if the effect be favorable proportionate to the urgency of the indication. In the pneumonia of drunkards its early use is to be recommended.

If the alcoholic stimulants fail, other cardiants must be administered simultaneously. Of these, strychnin has been the most serviceable in my own hands, its mode of administration following the same rules as have been mentioned for alcohol—at first in moderate-sized doses, to be increased as occasion demands. Should urgent need of stimulation arise, strychnin should be exhibited hypodermically. It is my custom in desperate cases to use subcutaneously as much as gr. $\frac{1}{15}$ (0.0043) every two or three hours. As soon as the condition of the heart denotes restoration of cardiac power the size of the dose is to be reduced. In no other disease do strychnin and alcohol possess greater potency for good than in pneumonia; they lessen the depressing effect of the poison. For sudden heart-failure ether, administered hypodermically, is also very efficacious. In severe forms of pneumonia digitalis is invaluable during the advanced stages; it may be given in doses ranging from 5 to 15 minims (0.333–0.666) of the tincture every third hour. In cases in which extreme cardiac weakness with depression of respiratory forces supervenes the drug is to be administered hypodermically in the same dosage. Strychnin may be combined with the digitalis. The effect upon the pulse and heart-sounds should be the criterion of sufficiency. Löwenstein states that both camphor and caffein are far superior to digitalis. S. West[1] sees most benefit from caffein citrate with nux vomica. Recent experience enables me to speak strongly in favor of atropin administered subcutaneously in the threatened collapse that sometimes attends the crisis. Nitroglycerin is especially indicated when the renal secretion is scanty and the urine contains more than the usual trace of albumin. Ammonium, adrenalin chlorid, and camphor (gr. 1 to 2—0.027–0.054) in sterile oil, hypodermatically, are also excellent stimulants to the feeble heart of pneumonia.

Peripheral stimulants, such as cold or heat, either locally or generally, may be useful after the blood-pressure falls. It is claimed by some that central stimulation to an over-acting heart may hasten exhaustion rather than prevent the same.

Saline injections are valuable in overcoming a falling blood-pressure with increasing toxemia, for the purpose of filling the vessels. The hypodermic method of administration is preferable. From 1 to 2 pints (strength 0.7) may be injected, and allowed to flow under the skin from a rubber bag; and this may be repeated at intervals of eight hours if necessary. For an acute exacerbation of a chronic nephritis in the course of pneumonia, venesection with saline infusion is worthy of trial.

Respiratory Stimulants.—Beginning cyanosis is the signal for the use of respiratory stimulants, of which the best are strychnin and atropin, and they should be given hypodermically. *Oxygen*, if given freely, often serves to tide over periods of marked cyanosis. The gas should be inhaled directly from the cylinder until relief is afforded, when it may be allowed to escape near the patient's nose, so as to become mixed with air. Stoker[2] advises the continuous use of oxygen from the moment the disease declares itself.

[1] *British Medical Journal*, March 11, 1908.
[2] *Medical Press and Circular*, 1908, lxxxvi., 90.

Hydrotherapy.—In meeting high temperature, marked nervous symptoms, dyspnea, cardiac weakness, etc., hydrotherapy offers many superior advantages. When the temperature is high, ice-bags over the chest and abdomen are useful. Tub-baths have been for the most part omitted from consideration in the treatment of lobar pneumonia, rest being of the greatest importance. Cool sponging, combined with the ice-cap or the wet pack, serve as a substitute for the full baths (*vide* Local Measures *infra*).

Abortive Method of Treatment.—Petresco found large doses of digitalis (ʒj–ij—8.0 of the digitalis leaves in an infusion daily) administered at the onset to jugulate the disease. His experience covered 1192 cases, and showed a mortality range of 1.22 to 2.66 per cent. This plan of treatment aims to meet the chief pathogenic indication of pneumonia by passing through the lung-tissue an adequate proportion of leukocytes, and thus re-establishing the cardiopulmonary circulation.

Venesection.—This is a good measure in sthenic cases (which are not uncommon in rural districts), the temperature falling, the pain, the dyspnea, and the nervous symptoms being relieved and the pulse softened. The bleeding, however, must be free and rapid. Later in the course of pneumonia venesection may be resorted to if cyanosis and the signs of collateral pulmonary edema—due to a failing heart—arise, but at this period bleedings rarely yield good results, except in vigorous subjects.

Antiseptic Method.—This method is based upon etiologic indications. The best antiseptics are carbolic acid (♏j—0.066, every four hours), thymol (gr. ij–iij—0.129–0.194, every four hours), mercuric chlorid (gr. $\frac{1}{100}$—0.0006, every four hours). Creasote carbonate has been warmly advocated by Wilcox and others, and it merits a careful trial. It is claimed that this remedy aborts a large percentage of cases and mitigates almost all the rest; it tends to nullify bacterial activity, thus inducing crisis. Citric acid, given to diminish the coagulability of the blood, has proven to be a valuable remedy.

Antipneumococcus Serum and Serum obtained from Convalescents.—Washbourn, Pane, Fanoni, and others have reported favorable results from the use of antipneumococcus serum. On the other hand, many clinicians who have employed the serum are skeptical as to its therapeutic efficiency. It seems to possess considerable protective power, as shown by the Klemperer brothers (*vide* p. 110). I have collected 535 cases treated by serum therapy—61 cases by diphtheritic and 474 by antipneumococcic serum—with 85 deaths, a mortality of 18.3 per cent.[1] The results obtained by different observers are variable and fail to carry conviction. The use of killed cultivations, or "vaccines," are found to be useful (Latham and others). Robertson and Illman used bacterins in 20 cases with beneficient results. Raw states that, while not a specific remedy, they ought always to be used in cases of a virulent type. The estimation of the opsonic index is unnecessary.

Treatment of Special Symptoms.—The initial **pain**, when it is of an acute, agonizing character, is relieved by the hypodermic use of morphin. This counteracts the shock produced by the invasion-period, but it is to be omitted if the bronchi contain secretory products, since morphin dries these and favors their accumulation rather than their removal. Rarely is it necessary to continue this remedy after the second day.

Fever.—The fever of pneumonia is a temporary affair, and instead

[1] *Jour. Amer. Med. Assoc.*, Dec. 10, 1904, p. 1777.

of being hurtful may prove beneficial, since it furthers tissue-metabolism, and this aids in the destruction of the specific poison of the disease. While it is true that internal antipyretics possess the power to reduce temperature, their use is attended with danger from their action as cardiac depressants; if it be true, as before stated, that pneumonia usually kills through the heart, it follows that cardiac power must primarily be conserved. I have abandoned their use. (See Hydrotherapy, p. 128.)

In cases in which venesection is indicated the tinctures of veratrum viride and of aconite have been much vaunted as substitutes. The tincture of veratrum viride produces a good effect upon the congestion in the early stage, since it relaxes the arterial walls, and thus bleeds the patient into his own vessels, and "allows the return of the blood to the circulation when the stage of consolidation is reached" (H. C. Wood). It should be discontinued after the second day of the illness. The tincture of aconite, owing to its depressing influence upon the heart, should not be employed. The alkalies (*e. g.*, sodium carbonate) are employed to neutralize the acid produced by the causative bacteria. The salts of the organic acids will serve the same purpose (Brown) as potassium citrate.

The **nervous symptoms** are successfully met, as a rule, by hydrotherapy (including the ice-cap), by the arterial stimulants, and by the use of morphin, as before recommended.

Cough during the early stage is controlled by the morphin needed to combat the pain. In the more advanced stages, if there be present numerous moist râles and a scanty expectoration, stimulant expectorants (ammonium muriate, terebene) may be employed with happy effect; but ordinarily they do harm rather than good. Pilocarpin may aid resolution when this is delayed (Reiss); the heart must be guarded.

Complications.—The management of the complications does not differ from that which is appropriate when they occur as independent affections, though all depressing measures must be positively omitted. I would add that in pleuro-pneumonia aspiration is not well borne. Suppurative arthritis should be treated by incision and drainage. Among measures to prevent *ether-pneumonia*, I would urge an appropriate toilet (a thorough cleansing followed by the topical use of an efficient antiseptic solution) of the nasopharynx and mouth as a routine practice. Pneumonia occurring in malarial subjects demands the use of quinin.

Local Measures.—When in doubt as to whether venesection should be employed or not, it must be remembered that early local bloodletting (cupping, leeching) is followed by relief from pain and dyspnea, but that these measures should be reserved only for robust persons. Counter-irritation by means of sinapisms is useful at the onset, and if pains be severe, strapping the side affected gives much comfort. The cotton jacket has certain advantages in maintaining the free, local action of the skin. The *topical use of cold* in the form of ice-bags has been practised extensively by Lees of England and Mays of America with brilliant success. Other hydriatic measures, suggested by Baruch, as cold compresses, wrung out of water at a temperature of 60° F. (15.5° C.), and applied to the anterior and posterior portions of the chest (the edges overlapping in the axilla), give similarly good results. They should be made of several thicknesses of muslin, held together by basting, covered by flannel, and firmly secured by safety-pins. A reapplication every half hour or hour is advised.

Secondary Pneumonia.

Pathology.—The lesions are identical in character with those of primary lobar pneumonia, but the areas involved have not always the same regular distribution. Congestion surrounding the hepatized lung-tissue is not infrequently extensive. We see, post-mortem, a tendency to commingling with small areas of lobular pneumonia. Both the streptococcus and the micrococcus lanceolatus are frequently found on microscopic examination.

Etiology.—Most instances are secondary to the acute infectious diseases, and it is probable that the specific causes of certain of the latter (Eberth's bacillus, Pfeiffer's bacillus, etc.) have the power to excite the morbid changes of acute lobar pneumonia. *Colon-pneumonia*, due to the bacillus coli, is the result of hematogenous infection either from the intestinal or from the urogenital tract. In the majority of instances, however, in which this disease develops in the course of the acute infectious diseases the latter are to be regarded as merely furnishing an opportunity for infection by the micrococcus lanceolatus.

Symptoms.—The rational symptoms are often absent. Close observation may, however, detect more or less dyspnea, cough, and increased fever, and rarely the attack is heralded by a rigor, followed by fever, the pneumonic type of breathing, pain, cough, and the characteristic expectoration.

The physical signs, when carefully observed, usually serve to enlighten the physician as to the nature of the affection. Hence it is a natural corollary that repeated physical examination is demanded in all cases in which there is danger of intervening lobar pneumonia.

Diagnosis.—This rests chiefly upon the physical signs, which are the same as in primary lobar pneumonia. Obviously, when the local subjective symptoms and the characteristic sputa are present a correct diagnosis is easily made. The fact must be emphasized that bronchopneumonia arises in the course of infectious diseases far more frequently than does lobar pneumonia.

Prognosis.—The occurrence of lobar pneumonia as an intercurrent affection adds greatly to the gravity of the primary disease. It is especially dangerous when it appears as a sequel during convalescence from acute infectious diseases.

The treatment is similar to that of primary lobar pneumonia, though less satisfactory.

INFLUENZA.

(*La Grippe: Epidemic Catarrhal Fever.*)

Definition.—Influenza is an acute contagious disease, probably caused by the bacillus of Pfeiffer. Its chief symptoms are due to catarrh of the respiratory and digestive tracts, together with profound muscular and nervous prostration, and grave complications (especially pneumonia) often present themselves. The disease may be endemic, though oftener it prevails in an epidemic or pandemic form.

Historic Note.—Every quarter of the globe has been the scene of visitations of epidemic influenza. More rapidly than any other disease belonging to the same class does it traverse a region of country. As a rule, influenza develops into epidemic proportions in the East, whence it spreads with unparalleled rapidity in a westerly direction. The first epidemic of the disease in the United States appeared in 1647, and was subsequently described; and, though it has since then frequently prevailed, the outbreaks have not observed any regular periodicity. The last true pandemic of the affection originated in Bokhara in May, 1889, reached St. Petersburg in the following October, Paris in November, and London in turn early in December. In America the cases began to appear about the middle of December, and rapidly multiplied into an explosive epidemic, which reached its maximum in January, 1890. Influenza reappeared in epidemic form, though less extensively, during April and a part of May, 1891, and again in a briefer and lighter form in the winter of 1891–92. During the winter of 1892–93 only a few sporadic cases occurred. Subsequently, it prevailed in an epidemic form annually for seven successive years in limited sections of this country. During the early part of 1901, and again in the winter of 1902–03, pandemic visitations of the disease occurred in the United States.

Pathology.—There are no special anatomic lesions that characterize the disease. In the rare instances in which death occurs in uncomplicated cases the catarrhal changes of the respiratory and gastro-intestinal mucosa disappear after death. In the abdominal type of the affection there may be enlargement of the glands of Peyer and of the solitary follicles. Among the fatal complications are pneumonia (either lobular or lobar), serofibrinous pleurisy, empyema, purulent pericarditis, nephritis, and rarely cerebrospinal meningitis and acute hemorrhagic encephalitis. All of these, however, may rarely be of influenzal origin.

Etiology.—**Bacteriology.**—Early in the year 1892 Pfeiffer discovered the *influenza bacillus*. It is of about the same breadth as the bacillus of mouth-septicemia, and only one-half the length of the latter. When stained by the aid of gentle heat with Ziehl's carbol-fuchsin, one part; water, nine parts, or Loeffler's methylene-blue, it may be observed as a small dumb-bell, having knobbed ends connected by a rod-like shaft. These bacilli are obtained from the sputum and nasal secretions. Pfeiffer has shown that they may penetrate the peribronchial tissue and pass out to the pleura. They have also been found in the blood and other tissues. This bacillus can be cultivated only in media containing hemoglobin, and when inoculated into rabbits it causes symptoms resembling those of influenza. While the bacillus of Pfeiffer is generally supposed to be the sole pathogenic agent, this has not as yet been proved. The organism probably causes an intoxication and not an infection. Influenza is a common *secondary infection*, especially in childhood, and may occur in measles, diphtheria, scarlet fever, and other infections.

Modes of Conveyance.—A specific germ that is propagated with the unusual rapidity that marks the bacillus of Pfeiffer must be air-borne.

Pepper suggests that the micro-organism may be almost universally distributed, and that under certain extraordinary atmospheric or telluric conditions it acquires a degree of virulence that renders all subject to its attack. Influenza is communicable by *contagion*, and evidence is abundant to show that it may be transferred by *fomites*. In some epidemics the disease travels slowly, and follows principally the lines of ordinary human and commercial intercourse.

Manner of Invasion.—The contagion probably enters the system with the inspired air through the respiratory tract. Some contend that the infection atrium is the alimentary canal, while others believe that the primary point of infection may be the conjunctiva.

Predisposing Causes.—All persons are liable to the contagion. *Age* has slight influence, the period of greatest susceptibility being from the twentieth to the thirtieth year. The very young are least susceptible, and during an epidemic are apt to be affected last, while old persons are frequent sufferers. Subjects whose vitality is lowered by neuropathic heredity or chronic maladies are among the first to suffer during an epidemic.

Immunity.—A primary attack of influenza does not bestow immunity, since *relapses* are very common, in 10 per cent. of the cases (Turney). Many persons, too, suffer from the disease with the reappearance of fresh epidemics, so that two, three, four, or even more attacks may be observed in the same individual (*recurrences*). Recent investigations have shown that a decided antagonism exists between influenza and malaria.[1] Epidemic influenza increases susceptibility to pneumonia and probably also to typhoid fever and appendicitis.

Clinical History.—General Symptomatology and Course.—The *incubation period* is quite brief, rarely exceeding two or three days. The *onset* is generally sudden, with either a severe rigor or repeated slight shiverings, accompanied by a rapid elevation of temperature which may touch 104° or 105° F. (40.5° C.), intense headache, distressing myalgic pains, and great prostration. The primary fever, however, varies greatly in severity, as does also the character of the symptoms—both local and general. Profound prostration characterizes the vast majority of instances during the invasion period. Depression of spirits, restlessness, insomnia (more rarely undue somnolence), and frequently delirium are among the prominent nervous phenomena.

Rare Modes of Invasion.—The infection may set in (*a*) by vertigo, (*b*) by apoplectic features, (*c*) by bilious vomiting, (*d*) by an abrupt and profound prostration. Nose-bleed sometimes occurs.

The most striking symptom is *pain*, which in many cases is referable chiefly to the forehead, temples, occiput, eyeballs, and root of the nose. General neuro-muscular pains are often present. The principal seat of the pain is commonly the lumbar spine (rachialgia). I have frequently noted cutaneous hyperesthesia. The pains may take the form of neuralgia of individual nerves or of pleurodynic stitches, or there are

[1] "A Statistical Study of Influenza; its Potency to Lessen the Receptivity of the Body for Malaria, as well as to Increase the Receptivity for Pneumonia and, probably, Typhoid Fever," by the writer.—*Philadelphia Hospital Report*, 1895, vol. iv.

localized areas of burning, boring muscular pain. The *temperature* may, as before intimated, mount quite high at the beginning, and if so it usually remits during the first night. It subsequently pursues a comparatively low range. The temperature-curve is markedly irregular, and often terminates by an apparent crisis. The *pulse* is small, feeble, running, irregular, and even intermittent, and I have sometimes observed it to be unusually slow. The depressing effects of the poison upon the heart often reach a dangerous degree. No leukocytosis is present as a rule. An occasional mild leukocytosis (10,000–15,000), however, is noted in uncomplicated cases. In many cases *dyspnea* is a rather conspicuous symptom, occurring independently of pulmonary complications. The same is true of *cyanosis*. Sweating may be troublesome.

Clinical Types.—Different types have been described based on the differences in the local manifestations and the varying degrees of toxemia. Influenza is remarkably protean in its features, and the enumerated types quickly and frequently merge into one another. (*a*) *Respiratory Type.*—Local catarrhal symptoms usually develop in the course of one or two days. They are, as a rule, evidenced first by a suffusion of the conjunctivæ, with excessive lacrymation, frequent sneezing, and slight pharyngitis. A little later, in most instances, hoarseness and cough come on, the latter being hard, racking, paroxysmal in character, and resembling whooping-cough. The cough and other local symptoms are due to an intense, dry laryngo-tracheal irritation. In most instances the expectoration is scanty, and in these the physical signs are very generally negative. In a smaller proportion of the cases there is considerable expectoration, and the physical signs of ordinary bronchitis are manifested. (*b*) *Gastro-intestinal Type.*—The catarrhal symptoms may center in the digestive system, most frequently in children. In such, vomiting comes on early and is apt to be repeated at longer or shorter intervals. There is diarrhœa, more or less urgent, with sharp abdominal pain, as a rule. (*c*) The *cardiac* group of symptoms that occasionally supervenes comprises heart-failure and distress, with a rapid, feeble pulse (a toxic form). (*c*) The *nervous* or *typhoid* (toxic) *type* presents a continued fever, with the signs of the typhoid state. Two classes of nervous symptoms are seen—"comatose and delirious" (Bury). Patients may be seized with intense headache, or an epileptic or apoplectic fit, or there may be local paralysis or hemiplegia. Muscular rigidity, especially of the neck, is far from uncommon. (*e*) The *rheumatoid type* manifests itself by violent pains in the muscles all over the body. There is no visible change in either the joints or the nerve-trunks. (*f*) Huchard[1] calls attention to *apyretic forms*, in which there may be marked pulmonary congestion or actual lobar pneumonia without fever, without expectoration, and often without cough. (*g*) There are ambulatory forms which are important because they tend to spread the affection. (*h*) Franke describes a chronic form assuming the guise of catarrhal affections of the respiratory and gastro-intestinal passages. The raspberry tongue is characteristic of chronic influenza.

Leading Features and Complications.—(1) **Pulmonary.**—Severe bronchitis, particularly affecting the capillary tubes and leading to bronchopneumonia, is a common and very serious complication. As a

[1] *Bull. Acad. de Méd.*, Feb. 17, 1900.

secondary result we are apt to observe the development of collateral pulmonary edema, with its usual fatal termination; and while this complication is prone to develop in the so-called thoracic type of influenza, it is by no means limited to this class of cases. I have observed bronchopneumonia in cases in which the physical signs of bronchitis were not presented prior to its onset. It may originate apparently in the profound prostration of the nervous system—a condition which also annuls in great part the phagocytic action of the leukocytes. As a rule, both broncho- and croupous pneumonia may be definitely traced to exposure.

The nature of the condition is variable, and may at times be ascribed to *congestive collapse* and other conditions, rather than to the ordinary type of bronchopneumonia. *Congestion associated with edema* of the lungs occurs as a complication of influenza. Enlargement of the bronchial glands may also be noted, and the recognition of this condition may be aided by percussion over the upper four dorsal vertebræ, where dulness will be obtained (*vide* Streptococcus-pneumonia, p. 120).

Lobar pneumonia is also a frequent and very fatal complication. It may arise early and in rare instances insidiously, but it is much more apt to manifest itself after influenza has about exhausted its force upon the vital organs or during the early part of convalescence. The symptoms of invasion—severe chill, high temperature, followed by the usual physical signs—are *sudden in their onset* and lead rapidly to an extremely serious condition. When lobar pneumonia develops early in the course of influenza (a rare event), its symptoms are modified, the preliminary chill and pain in the side being often absent, and more frequently still the characteristic crepitant râle. Subcrepitant râles, however, are audible, and the dyspnea is out of proportion to the area of lung-tissue involved. Most of these features may also be observed in connection with the pneumonia that appears during convalescence. Marked leukocytosis is present as a rule. (See Apyretic Varieties, p. 133.)

Plastic pleurisy is commonly an associated condition, especially in cases of lobular or lobar pneumonia. Other forms of pleurisy also occur, though less frequently (sero-fibrinous and empyema). *Gangrene* and *abscess* of the lungs may arise as terminal complications.

Cardiac Complications.—Heart-failure often manifests itself, and may prove fatal, though rarely. Purulent pericarditis is a rare complication, and is often secondary to pleurisy or pneumonia, while attacks of angina, which usually interchange with simple weak heart (often associated with arrhythmia), have been noted in certain epidemics (Curtin and Watson).

Gastro-intestinal System.—There may be severe gastro-enteritis (particularly in children), with frequent vomiting and purging and abdominal pains, and, more rarely, hemorrhages occur from the stomach and bowel (*vide Gastro-intestinal Type*). Catarrhal jaundice may appear. Appendicular inflammation may be induced by influenza.

Nervous System.—The most frequent symptom is perineuritis, which probably causes much of the patient's sufferings. A soporose or even comatose condition may be observed. Delirium of a most active form sometimes appears, and particularly when certain other complications have arisen (pneumonia, pericarditis). Cerebro-spinal meningitis occasionally occurs. I have observed symptoms identical with those of meningitis appearing suddenly, and in the course of a day or two disappear-

ing just as suddenly. In addition to these symptoms, we should have the existence of suppuration elsewhere in the body (otitis, purulent pericarditis) or of pneumonia. Davis holds that in a large percentage (78 per cent.) of patients dying of influenzal meningitis, broncho-pneumonia occurs. A positive diagnosis demands the finding of the specific organism by means of lumbar puncture. Jundell found influenza bacilli in about 10 per cent. of 200 cases in which a symptomatic diagnosis had been made. Cerebral abscesses have also been noted (Bristowe). Kerr has reported disseminated lesions of the central nervous system following influenza. The severer nervous features and complications are mostly observed in the typhoid type of the disease.

Genito-urinary Tract.—Renal congestion, and even acute nephritis, may appear as a complication. A case of cystitis with hematuria has also been reported (Comby and Le Gendre).

The **diagnosis** of influenza except in ill-defined, sporadic cases rarely presents difficulty. Usually, the march of the epidemic, the abrupt onset, with alternating flashes of heat and chilliness, the brevity of the febrile stage, headache, sore eye-balls, rachialgia, and a prostration out of proportion to the catarrhal manifestations, form a conclusive assemblage of symptoms. In all cases, more particularly the obscure forms, the sputa, if there be any, should be studied microscopically. The bacillus of Pfeiffer may be conveniently stained with a solution of fuchsin-rubin (gr. 0.01 in 100.0 aqua destillata). Franke invites attention to the band-like redness of the half-arches as a diagnostic criterion.

(*a*) *Climatic catarrhal affections* are sometimes hard to discriminate from sporadic cases of influenza. The former are usually attributed to sudden and great vicissitudes of temperature or exposure to strong drafts of air, while the latter come on independently of such agencies. Again, in influenza the general features (nervous symptoms and debility) outweigh the local (catarrhal manifestations). Leichtenstern speaks of pseudo-influenza, or catarrhal fever (*influenza nostras*), believing that it bears the same relation to true influenza as does cholera nostras to Asiatic cholera. Its cause is unknown.

(*b*) *Typhoid fever* in its early stages is often simulated by influenza with intestinal symptoms. Influenza, however, gives the history of the prevalence of an epidemic, begins suddenly, does not show the typical temperature-curve of typhoid, may present splenic enlargement—but not to the same extent as typhoid—has no characteristic eruption, and does not give the characteristic sero-reaction. Again, the Pfeiffer bacillus may be discovered in the nasal and bronchial secretions in influenza.

(*c*) *Pneumonia* has quite frequently been mistaken for influenza, and especially when the thoracic symptoms in the latter have been unusually distinct. As already stated, lobar pneumonia may early complicate influenza in rare instances; but pneumonia is generally unilateral, while the lung-involvement in influenza is generally bilateral. In the former the physical signs indicative of consolidation are present; in the latter (unassociated with pneumonia) those suggestive of congestive edema (impaired resonance, stationary subcrepitant râles). The general features also present dissimilarities. Thus the nervous depression and the myalgic and neuralgic pains are more marked in influenza, while the pulse-respiration ratio is less disturbed than in pneumonia.

(*d*) *Cerebro-spinal meningitis* may manifest features that are almost identical with those characteristic of influenza. Thus during certain epidemics "grippe" patients may be stricken as by a blow; they suffer from intense headache—occipital and frontal—rachialgia, fever prostration, delirium, and stiffness of the muscles, with slight retraction of the head. There may be convulsions and vomiting at the outset. Here the history with reference to the character of the prevailing epidemic and the attendant circumstances must be carefully considered, but an absolute diagnosis is sometimes impossible unless a laboratory investigation of the discharges or lumbar puncture be made.

(*e*) Small-pox, in the pre-emptive stage, may be confounded with influenza, but the latter is soon diagnosticated by quick response to therapy and sweating, relieving the symptoms in twenty-four to thirty-six hours, whereas small-pox is resistant to all treatment, the appearance of the rash only bringing amelioration of the symptoms.

Sequelæ.—Among the sequelæ are phthisis, chronic bronchitis, abscess and gangrene of the lungs (the latter two being rare), tachycardia, and angina pectoris. Chronic gastro-intestinal catarrh, chronic nephritis, and less frequently cystitis, may also be mentioned. Latent forms of tuberculosis and chronic nephritis are often kindled into active and progressive affections by intercurrent influenza.

Among *nervous* sequelæ, which are both numerous and important, are to be noted especially insomnia, neuralgia, migraine, melancholia, with tendency to self-murder, meningitis, acute ascending myelitis, peripheral neuritis, and perineuritis. The organs of special sense manifest a great variety of sequelæ, such as otitis media, otitis interna, mastoid abscess, conjunctivitis, keratitis, iritis, irido-choroiditis, acute glaucoma, etc.

Prognosis.—The prognosis is, on the whole, good. Almost all fatalities are due to complications, especially *pneumonia*, and, less frequently, pulmonary congestion and edema, pleurisy, pericarditis, and cerebro-spinal meningitis. The comatose type is often fatal.

The *circumstances connected with the individual case* often affect the outcome. Thus influenza runs a more severe course, and hence offers a correspondingly more serious prognosis, in those enfeebled on account of previous chronic disease (phthisis, valvular disease of the heart, emphysema, nephritis) and in the young and the old than at other periods of life. During severe epidemics of influenza the mortality-list in most chronic diseases is considerably augmented. Though epidemics vary as regards the mortality, the general average death-rate is a little under 1 per cent. In some epidemics it may reach 2 per cent., while in others it may be less than $\frac{1}{2}$ of 1 per cent.

Duration.—The *duration* of the attack is brief, though subject to variations. In mild forms it is from two to four days, in the severe from seven to ten days; but complications and previous infirmities may prolong the attack. The *duration of particular epidemics* rarely exceeds from four to six weeks. Convalescence is usually protracted.

Treatment.—**Prophylaxis.**—Drugs which have been counselled for their preventive effect (quinin, salicin) are devoid of value. Those who are at either extreme of life or who are enfeebled by chronic organic disease, should be most carefully protected by proper wearing apparel, and should not be carelessly exposed to unfavorable weather conditions.

The inmates of hospitals and prisons have been known to escape the disease. **Isolation** should, therefore, be carried out in hospitals, and, whenever practicable, in private families, especially when the disease appears in households in which there are young children and aged persons. E. W. White has reported an epidemic of influenza that was successfully aborted by strict isolation of the patients. **Disinfection** of the catarrhal discharges, particularly the bronchial, which, as a rule, abound in the bacilli of Pfeiffer, is necessary. I must also insist upon disinfection of the naso-pharynx and mouth cavity.

Treatment of the Attack.—The cases may be grouped under three heads:

(*a*) **Mild or Rudimentary Form.**—The cases belonging to this type require careful hygienic management. However light the attack, the patient should remain in-doors and, if prostrated, in bed for a period of two or three days. The *diet* should be light and nutritious (milk, eggs, rice, gruels, fresh vegetables, stewed fruit), and cooling drinks are to be preferred to hot ones, among the former lemonade or cold oatmeal-water with lemon, and effervescent mineral waters (Apollinaris, lithia, Seltzer), being the best. The bowels should be moved regularly, avoiding, however, active purgation. The use of light wines is not objectionable if desired by the patient. In all cases of influenza, even of the mildest grade, I prescribe moderate doses of quinin (gr. iv—0.2592, three or four times daily), and if there be much headache combined with it, Dover's powder and monobromate of camphor (of the first two, gr. iij—0.194, each, and of the last gr. j—0.0648, in capsule), the dose to be repeated at intervals of three or four hours. To overcome the languor and debility I have found nothing so successful as strychnin.

(*b*) **Cases of Medium Severity.**—*General Management.*—This class of influenza patients betake themselves to bed, and should be kept there till convalescence is well advanced. During the febrile period the *diet* must be light, liquid, yet nutritious, and the food should be given every two or three hours. Although the patient has no desire for food, he should be urged to take it regularly. Moderate stimulation is also useful.

The *medicinal treatment* is, for the most part, simple and symptomatic. Calomel in moderate doses (gr. j every third or fourth hour) should be a remedy of choice for a day at least. An efficient diaphoretic, given within six or eight hours from the time of onset, may abort the attack. The neuralgia and myalgia may be relieved by the use of quinin, Dover's powder, and ergot; but if the pain be intense, morphin administered subcutaneously may be required. The temperature is somewhat reduced by these remedies, and especially by the quinin and Dover's powder, the latter acting as a diaphoretic. In addition, I am in the habit of ordering cool sponge-baths at intervals of two or three hours if the temperature be about 102° F. (38.8° C.). If not controlled in this manner, we may combine with quinin some antiseptic, such as salicylic acid or salol. I have sometimes found it necessary to add to the foregoing small doses of phenacetin (gr. ij—0.129). *Sleeplessness* may demand hypnotics, such as sulfonal, chloralamid, opium, and trional. It is necessary to utter a warning against the free use of coal-tar products, since they induce heart-failure.

The local catarrhal conditions (coryza, laryngo-bronchial irritation,

true bronchitis, etc.) must be treated according to the special indications presented in individual cases. For the coryza inunctions of animal fats over the forehead and bridge of the nose are useful. A flannel cap may be worn if agreeable to the patient. Steam inhalations through the nares and mouth often act beneficially, both upon the coryza and laryngo-bronchial irritation. For the latter common condition the following formula will be found serviceable:

℞. Codeinæ sulph., gr. iv (0.259);
Ammon. chloridi, ʒv (20.0);
Syr. prun. virgin., fʒij (60.0);
Spts. junip. comp., q. s. ad fʒiv (120.0).—M.
Sig. One teaspoonful every two or three hours.

If this prescription fail to mitigate the cough, we may resort to morphin hypodermically in small doses. The bronchitis may sometimes be controlled by the use of sodium benzoate, ʒij (gm. viij) in aq. menth. pip., ʒiv (gm. 120), of which a tablespoonful may be taken every two or three hours. In the later stages, particularly if bronchitis be associated with free secretions, the oil of eucalyptus (♏iij to v—0.199 to 0.333), in capsule, every four hours, has in my experience proved useful. To obviate pulmonary complications I have found strychnin (gr. $\frac{1}{30}$—0.0021), combined with the extract of gentian (gr. j—0.063), useful. Chest-pains may be relieved by the use of turpentine stupes and sinapisms.

(*c*) **Severe Forms.**—The *general management* is similar to that recommended in cases of medium severity, excepting that freer stimulation is usually demanded. The medicinal treatment must also be more active than in the previous form, and often is heroic. Especially must quinin be given and continued, since it not only serves to reduce the temperature somewhat, but also to sustain the vital forces, to control the nervous symptoms, and lessen the tendency to inflammatory complications. Flexner[1] recommends the daily injection for three or four days by means of lumbar puncture, of the immune serum of the influenza bacillus in complicating meningitis. Should there be sudden cardiac failure, it must be promptly met by cardiac stimulants (strychnin, camphor, ether, digitalis) given hypodermically. In addition to alcoholic stimulants, the aromatic spirits of ammonia is usually borne well, and should be administered. The various inflammatory complications must be treated as under other circumstances.

The Convalescence.—The greatest injury to patients at this period comes from going out too early. Usually the temperature is subnormal for several days—a circumstance due to the weakness of the patient—and so long as this condition obtains the patient is highly susceptible to a chill. Hence it is a good rule not to allow exposure to the external atmosphere until the temperature has been normal for several days. The diet should now be more liberal, and tonics, such as gentian, iron, and quinin, may be administered and continued until complete restoration of the patient's health has taken place. In every way possible exposure to reinfection during the period of convalescence is to be avoided. The sequelæ must be treated according to general rules.

[1] *Jour. Amer. Med. Assoc.*, 1911, lvii., 16.

DENGUE.

(*Break-bone Fever.*)

Definition.—An acute infectious disease occurring epidemically in tropical and subtropical countries. Its chief symptoms are—a double febrile paroxysm (separated by an interval), arthritic and muscular pains, and a skin-eruption in about one-half the cases.

Historic Note.—The disease was prevalent in Java as early as 1779, in India in 1824, and later in the West Indies, Spain, and in some of the southern American States. Mild epidemics have visited Philadelphia, New York, and Boston (during warm weather), but, as a rule, it has not traversed regions beyond 32° N. latitude.

Its *pathology* has not been studied, death being the rarest of events.

Etiology.—McLaughlin, of Texas, has isolated from the blood and cultivated a micrococcus. H. Graham[1] has discovered an ameboid form resembling the *plasmodium malariæ*, but having a longer life-cycle.

Predisposing Factors.—Its prevalence is favored by the summer season, and to a slight extent by faulty hygienic conditions. On the other hand, age, race, sex, and social status are all without effect, most persons being susceptible, a fact that accounts for its marvellously rapid diffusion. As a rule, susceptibility is exhausted by one attack. The epidemics spread along lines of travel by land and sea. Graham's experiments in Beirut indicate that dengue is not contagious, but *culex fatigans* may carry the infection from one person to another. Altitude is said to exercise an inhibitory influence.

Clinical History.—There is a period of *incubation* that lasts from one to four days and exhibits no prodromes.

Invasion then is *abrupt*, with a slight chill; fever follows, the temperature reaching its maximum—103° to 106° F. (39.4° to 41° C.) or over—at the end of the first or on the second day, and is accompanied by headache and by muscular and arthritic pains. The patient's sufferings are intense, the *pains* being described as "breaking"—a peculiarity to which the disease owes the popular name of "*break-bone fever.*" The painful *joints* are neither swollen nor tender, as a rule. Jones reports an epidemic in which severe pain was absent. The *respiration* and *pulse* are much quickened; there is anorexia and slight nausea. Febrile albuminuria is rare, delirium and mental torpor also; but *prostration* may become marked, and an *erythematous eruption* (*initial rash*) commonly appears. DeBrun[2] noted the symptoms during the epidemic at Beirut (1892), and states that the eruption is roseolar, morbilliform, scarlatinous, or papular. He distinguished three groups of cases: 1. With high fever and marked associated symptoms, and with eruption. 2. Fever absent, the symptoms mild, with eruption. 3. The eruption the only symptom. The eruption may appear early, but has no fixed time, is evanescent in mild cases, and is never constant in character. It is attended with burning and itching, and DeBrun noted desquamation. *Hemorrhages* from the various organs (nose, gums, stomach, bowels, lungs, kidneys, etc.) may occur, and reach even a dangerous extent. The *lymphatic glands* are often swollen; the mucosæ of the nose and throat are hyperemic; the eyes are congested and the face flushed. The disease is characterized by well-marked leukopenia.

[1] *Medical Record*, Feb. 8, 1902.

[2] *Rev. de Méd.*, No. 6, 1894.

The *initial fever* lasts three or four days, and ends with a deep remission accompanied by sweating. All the symptoms now vanish save a slight soreness and stiffness, but after two or three days the characteristic symptoms (including a roseolar eruption) reappear. This terminal eruption is rubeolar, commencing on the palms and backs of the hands, and extending upward. It is circular, dusky red, and sometimes slightly elevated. It extends quickly to other parts, being best seen on the back, chest, upper arms, and thighs. The spots disappear on pressure, and never or rarely become petechial (Manson). The *second* febrile paroxysm is usually milder and shorter than the first.

The **duration** of the disease is from seven to ten days, the attack being followed by a slow convalescence, which may be interrupted by a relapse. The slowness of the recovery is due to persistence of the pains, mental depression, and marked physical prostration.

Complications.—Meningitis has been rarely noted. Convulsions sometimes occur in children, and severe catarrhal inflammations of certain mucosæ (bronchial, gastric) may develop. Insomnia is common. Hyperpyrexia and pericarditis occur, though exceptionally.

Diagnosis.—The diagnosis of the epidemic form of the disease is an easy one after observation of the first few cases, but it is difficult to discriminate sporadic cases from *rheumatism*. The course of the fever, however, differs in the two diseases, while the eruption belongs to the former alone. *Influenza* may resemble dengue. Influenza occurs in the cold season, and herpes is usually the only eruption; the joints are rarely involved; there is no recurrence of the fever, and serious complications are more frequent. The discovery of the bacillus of influenza is decisive, and the existence of an epidemic of either condition suggests the true nature of the disease. *Scarlet fever* has an erythematous eruption, but the fever is continuous, angina is present, and the arthritic symptoms are wanting. As a rule, dengue prevails only in tropical and subtropical countries.

Yellow fever has been mistaken for dengue, and the two affections may prevail together, as in the Galveston epidemic of 1897. The differential diagnosis is difficult, as there are points of similarity—time of appearance, geographic distribution, and the character of the febrile paroxysm. To show contrast, however, I have arranged the following table:

Dengue.	Yellow Fever.
Affects all races.	Foreigners more especially.
Facies characteristic; face flushed.	Mucous membranes injected.
Irregular rise of fever, followed by remission, then a second moderate rise. Duration 5 to 9 days.	The temperature rises regularly. Duration of fever 72 hours.
The pulse keeps pace with the fever.	Pulse falls while the fever is rising.
Eruption frequent (terminal rubeola).	Eruption quite rare.
Vomiting rare.	Vomiting frequent.
Urine never contains albumin (?).	Urine early albuminous.
Jaundice absent.	Jaundice present and early appearing.
Hemorrhages from mucous outlets, generally slight, and black vomit rare.	Hemorrhages common and severe. Black vomit an alarming symptom.
Muscular and joint pains present.	Absent.
Prognosis favorable.	Often fatal.
Serum-diagnosis valueless. There is a well-marked leukopenia.	Serum-diagnosis present in 66 per cent. of cases; no leukopenia.
Second attacks common.	No second attacks.

The **prognosis** is, with rare exceptions, favorable, dangers arising only in the serious forms, particularly those showing hemorrhages.

Treatment.—Indications: (*a*) to harbor the patient's strength, and (*b*) to meet certain leading symptoms. The first is to be met by enjoining rest in bed, by a generous diet, and by the use of stimulants and tonics during convalescence. The fever may demand treatment, and and when this is high, hydrotherapy is indicated. For the intolerable pains morphin is to be administered hypodermically. Efforts to destroy the *culex fatigans* should be instituted. Isolation should be practised.

THE PLAGUE.

(*Bubonic Plague; Black Death.*)

Definition.—A specific contagious disease, occurring chiefly in unsanitary surroundings and characterized by high fever and cutaneous symptoms (petechiæ, etc.). Its course is severe and rapid, and it occurs in epidemics.

Historic Summary.—An Oriental disease, the plague, has long been endemic in certain portions of India. Most European countries have in the past been visited by epidemics of the malady, and among the most famous was the truly pandemic prevalence of "black death" in Europe during the fourteenth century. Another virulent outbreak occurred in London in 1665, destroying more than 70,000 persons. In May, 1894, a severe epidemic prevailed in Canton and Hong-Kong, to which cities it had been imported from Northern India. In September, 1896, the plague appeared in Bombay and the Bombay Presidency. Since then the plague has shown periods of decrease followed by others of decided increase, and the total plague-statistics for the Bombay Presidency from September, 1896, to January 13, 1899, are 214,197 cases and 169,240 deaths. In the autumn of 1899, 2 cases were brought to the New York harbor, and on March 6, 1900, it appeared in the Chinese quarters of San Francisco, and 31 cases were officially reported between that date and February 13, 1901. It has reached several European ports—Oporto, Hamburg, Glasgow, London. W. J. Simpson[1] has given a graphic account of the history and distribution of the plague.

Etiology.—**Bacteriology.**—During the epidemic at Hong-Kong, Kitasato and Yersin, working independently (1894), discovered the special organism of the plague (*bacillus pestis bubonicæ*). It stains deeply at the ends, giving the appearance of a pair of micrococci, but is really a short rod-bacillus with rounded ends. Pure cultures can be made, and when animals (mice, rats, guinea-pigs, rabbits) are inoculated with these the symptoms of the disease are produced.

Predisposing Causes.—These are (*a*) unhygienic conditions, and (*b*) seasons. Broca states that epidemics of pneumonic plague occur in winter, and those of bubonic form in summer.

Inside the body the bacillus has been found in the lungs (plague-pneumonia—where it is often combined with the pneumococcus and staphylococcus), in the enlarged glands, in the pus from the buboes and the blood. *Outside* the body, among infected materials are dust from sputum, plague-infected flies, fleas, the excreta, food, and soil.

[1] "*A Treatise on the Plague*," 1905.

Modes of Transmission and Entrance into the Body.—According to Kitasato, the bacillus enters either through the digestive (rare) or respiratory tract or the skin (*e. g.*, abrasions of the feet). The point of infection is usually a gland or group of glands (Flexner) causing the primary bubo. The bubonic pest is spread by two principal factors—the rat and man (Simond). In most outbreaks of human plague rats had the disease both before and during the epidemic (Clemow). The rat is the carrier from house to house, although man is the chief factor in the spread of the disease, the agent of transport for long distances. Flies, fleas, ants, and other insects may act as carriers from rat to man. The rat flea carries the contagion from one rat to another. Nuttall's studies indicate that transmission of the poison by stinging insects is extremely rare. Certain animals besides rats (mice, tarabagans, dogs, cats, rabbits, pigs, horses) may become infected and transmit the disease to healthy animals. McCoy and others have found the plague bacillus, pathogenic for rats and guinea-pigs, in the ground squirrel. Yersin established the contagion of plague by keeping inoculated rats and healthy mice in the same place (Payne, in Allbutt's *System*). The disease is commonly transmitted by foci of the infection (houses, ships), by fomites, and possibly by plague-infected food and immune carriers.

Clinical History.—Varieties.—The classification is based on the particular system of the body principally invaded as follows: (*a*) Bubonic (glandular); (*b*) septicemic (circulatory); (*c*) pneumonic. Formerly two distinct forms, (1) pestis minor, or larval plague, and (2) pestis major, or the severe epidemic form, were recognized.

Incubation.—This lasts from two to five or, rarely, eight days. In malignant epidemics it may be but three or four hours. Prodromata may be observed for from twelve to twenty-four hours; they are intense headache, vertigo, and an unsteady gait. The physiognomy is stupid.

(*a*) **Bubonic Type.**—This type corresponds to the so-called pestis minor (see *ante*), often a forerunner of severe epidemics. It is characterized by swelling of the lymphatics, lasting about a fortnight, with slight general disturbance, as a rule. The bubonic, however, may merge into the septicemic or pneumonic forms. Such symptoms as halting speech, staggering gait, great prostration, a peculiar physiognomy, and more or less lymphatic involvement are common to all varieties.

(*b*) **Septicemic Type.**—Invasion may be abrupt; less commonly it is preceded by the prodromes mentioned above; and rarely, bilious vomiting or hematemesis are the ushering-in symptoms. A prolonged rigor or repeated shiverings occur. The temperature does not rise to a high level (100° F.), owing to profound prostration, and the pulse becomes rapid and thread-like, although variable in force and character. Delirium or coma tends to supervene. Debility may now be extreme, and the patient may die in the initial period. More commonly this threatened collapse is survived, and then (second to the fifth day) the most characteristic feature almost always appears—secondary buboes or inflammation of the lymph-glands, most commonly the inguinal, but also the axillary and cervical. The latter enlarge and are painful. Resolution may occur, or they may remain unchanged, particularly in fatal cases. Suppuration may also occur, and rarely gangrene, forming the so-called carbuncle.[1] Petechiæ and the hemorrhagic diathesis, as shown by bleedings from the

[1] Saunders' *Year-Book*, 1902, p. 378.

lungs, stomach, and intestines, arise in the worst forms. In this variety blood obtained by puncture of spleen, liver, and other organs shows the microbe in pure culture.

(*c*) **Pneumonic Type.**—Pneumonic plague, where primary localization of the disease in lungs occurs, commences with a rigor, malaise, headache, nausea, vomiting, and pains in the limbs. Fever, varying in range from 102° to 105° F., hurried breathing with oppression, cough, and blood-tinged sputum, soon appear. The physical signs, especially the stethoscopic, may be those of bronchopneumonia. The local symptoms grow worse, cyanosis, delirium, and later coma supervene, while the heart's action fails and death occurs on the third, fourth, or fifth day of the illness. In cases which recover or become more protracted, buboes may appear, and rarely these develop early in plague pneumonia.

(*d*) An **intestinal type**, with marked **hematemesis**, bloody diarrhea, and abdominal pains, also occurs.

(*e*) **Abortive Type** (*Pestis Ambulans*).—Certain epidemics are distinguishable by the larger proportion of mild cases (Manson). The patient may be so little inconvenienced as to be able to be about throughout the illness.

Plague pneumonia may also be secondary to, or symptomatic of, other types, the microbe having reached the lung metastatically, or possibly has been inhaled into the lungs. This form likewise simulates lobular pneumonia in its clinical features, and a pure growth of the plague bacillus can be obtained on making cultures from the sputum.

Sequelæ.—Paralyses of various kinds, myocardial weakness, and recurring suppuration of buboes are the principal sequels of the disease.

Relapses rarely occur, and are dangerous.

Diagnosis.—The diagnosis can be made with ease and certainty when the disease occurs in endemic centers, but when it occurs elsewhere its recognition offers some difficulty. The bubonic type is easily recognized, as a rule. On the other hand, to differentiate between primary plague pneumonia and ordinary lobar or bronchopneumonia is puzzling. A certain diagnosis rests upon bacteriologic evidence alone.

Prognosis and Mortality.—The death-rate is high, ranging from 40 per cent. (rare) to 80 or even 90 per cent. Among favorable indications is suppuration of the buboes. On the other hand, a rapid disappearance of a group of swollen glands is a bad augury. Additional unfavorable indications are plague-pneumonia, intense toxic features, with cardiac dilatation, purpuric spots ("tokens"), and hemorrhages.

Treatment.—Prophylaxis.—The precautions to be taken by the individual relate to the abandoning of all unsanitary habits, the isolation of the sick, and the avoidance of prolonged contact with infected patients or dwellings. Personal cleanliness and freedom from abrasion of the lower extremities are important prophylactic measures (White). It would seem that doctors and even nurses and attendants in well-ordered and properly ventilated hospitals rarely take the plague.

The prophylaxis of the public embraces—(*a*) Isolation of the sick and thorough disinfection of the sick-room, the bed and bed-linen, the vomitus, and the stools. Kitasato advocates steaming the bed at 212° F. (100° C.) for one hour, or exposure for a few hours to sunlight, and the burning of all infected articles. "After recovery the patient is to be kept in isolation for at least one month." Cases of *pestis ambulans* must be found and treated on account of their bearing on the spread of

the graver types. The infected houses are to be thoroughly disinfected, and a pure water-supply procured. (*b*) Protective inoculation or treatment by "vaccination" of healthy persons seems efficient. Haffkine[1] states that at Hubli the difference in mortality of those inoculated and of those uninoculated averaged from 80 to 90 per cent. The dose was 2.5 c.c. The experiments of Strong and Teague indicate that prophylactic inoculation does not protect against pneumonic infection in man. Calmette recommends Yersin's antiplague serum for prophylactic purposes in preference to Haffkine's vaccine. Strong advocates the injection of attenuated living cultures of *Bacillus pestis* as a method of immunization. Buchanan[2] advocates the keeping of cats to destroy the root of the trouble—the rats.

Treatment of the Attacks.—The diet should be liquid and nourishing, while free stimulation is demanded from the onset. *Medicines* are used to combat symptoms as they arise. Delirium and pain are to be met by morphin or hyoscin, and high temperature by hydrotherapy.

Local Treatment.—Cantlie does not believe in local measures before suppuration occurs. On the other hand, Nesfield notes that early incision into a plague gland produces an immediate improvement in the patient's condition.[3]

Serum-therapy.—Anti-plague serum exercises a specific action (Yersin). Of 26 cases treated, 2 died—a mortality of 7.6 per cent. Calmette[4] states that serum injection provokes rapid destruction of the bacilli by phagocytosis. As a curative dose, 20 c.c. must be injected intravenously, and repeated in twenty-four hours if there be fever still. Choksys concludes that in the Yersin-Roux antiplague serum we possess an efficacious remedy, especially if used during the first few or even twenty-four hours, serious complications being averted.

ERYSIPELAS.

(*St. Anthony's Fire.*)

Definition.—A specific, acute contagious disease, characterized by a special inflammation of the skin and subcutaneous tissues, with a tendency to spread, high fever, moderate prostration, a disposition to mixed infection, and an average duration of fourteen days. It usually occurs as an endemic disease, though also in epidemic form.

Pathology.—Erysipelas is a specific inflammation involving the skin, subcutaneous and less commonly the mucous surfaces. When inflammation extends to the subcutaneous connective tissue there follows, as a rule, suppuration. The specific cocci are found in the superficial lymph-vessels and spaces of the affected skin. Beyond the border of the inflamed region they occupy chiefly the lymph-vessels, where they are finally overpowered by the phagocytic leukocytes. Microscopic examination reveals the changes of simple inflammation. Pericarditis, endocarditis (rarely malignant endocarditis), pleuritis, and nephritis may be noted.

Etiology.—Bacteriology.—The specific cause of the disease is the *streptococcus erysipelatis* of Fehleisen, which is identical with the ordinary pus-producing streptococcus. Petruschky has shown that

[1] *Proc. Roy. Soc.*, vol. lxv., No. 418.
[2] *British Med. Jour.*, May 30, 1908.
[3] *The Lancet*, London, Nov. 4, 1911.
[4] *Lancet*, 1454, Nov. 17, 1900.

streptococci derived from non-erysipelatous morbid processes in man were capable of producing a typical erysipelas. The streptococcus of erysipelas is a saprophytic organism ; it assumes the form of a serpent or chain, is small, and thrives on all kinds of culture-media. Its favorite situations are the lymph-vessels of the skin and the cutaneous connective tissue. It is especially abundant near to the advancing border of the erysipelatous area, but is rarely found in the blood-vessels, and in blood-serum it is caused to disappear by the action of the phagocytes ; yet in exceptional cases intra-uterine infection has occurred. G. E. Pfahler[1] found a diplococcus in 8 cases. Erysipelatous inflammation can also be produced experimentally by the staphylococcus.

Predisposing Causes.—(1) **Season.**—In a paper on "Seasonal Influences in Erysipelas, with Statistics,"[2] I have shown, as the result of an analysis of 2010 cases collected from different sources that the various seasons of the year exercise a potent influence upon the frequency of this affection. Thus month by month the cases increase, in slightly varying ratio, from August to April, the latter month giving the greatest number, and then there is a rapid decrease from April to August, when we find the smallest number. Again, one-half of all the cases occur during the months of February, March, April, and May, and 15.9 per cent. during the month of April alone. It was found that a low barometer and mean relative humidity invariably correspond with the annual period in which the greatest number of cases occur, and that the highest percentage of relative humidity corresponds with the months affording the fewest cases.

(2) **Age.**—From the notes of 1894 cases I found that in 25.8 per cent. the age of the patient was between twenty and thirty years. After fifty years the cases decrease rapidly, and more than 15 per cent. occur before the age of twenty. The great liability of newly-born infants is well known.

(3) **Sex.**—This factor was noted in 1767 cases, and a marked preponderance of the male over the female sex was noted (about 3 to 2).

(4) **Previous Attacks.**—Of 450 cases, there had been previous attacks in 39 (8.6 per cent.), in one instance four, and in another seven, while second and third recurrences were not uncommon.

(5) **Family predisposition** exercises a slight though decided influence.

(6) **Certain Antecedent Affections.**—Dr. M. Booth Miller examined the history of 301 cases, and found that acute coryza preceded the attack in 13 instances. Slight lesions of the Schneiderian mucous membrane may be assumed to exist in such instances. That certain chronic diseases (chronic Bright's, phthisis, organic heart disease, chronic alcoholism, cirrhosis of the liver) augment a receptivity to the complaint has also been brought to light by my researches.

(7) **Slight Injuries, Abrasions, etc.**—Erysipelas will not develop on a surface which does not present a break, but with this present may do so though the latter be so trivial as to escape observation. Slight abrasions and fissures, either in the mucous membrane of the nose or in the skin of the face or ear, as well as all forms of slight injuries, are liable to furnish a highway for the organism. Yet in 643 out of the 2010 cases mentioned above, previous lesions were noted in but 13. Women who have been recently delivered and persons subjected to surgical operations are peculiarly liable, and any deep-seated focus of irritation (necrotic bone, chronic abscess, appendicitis) may give rise to erysipelas.

[1] *Phila. Med. Jour.*, January 13, 1900. [2] *Proc. of the Amer. Climatolog. Assoc.*, 1893.

(8) **Antihygienic Surroundings.**—These doubtless predispose to the affection, as has been shown by the prevalence of erysipelas in hospitals and institutions in which the sanitary arrangements were markedly faulty.

Modes of Conveyance of the Contagion.—The latter may be air-borne for short distances at least. It has been collected from the air of rooms and wards occupied by erysipelas patients. It may be transferred for a longer or shorter distance by fomites, by instruments, unclean hands, etc. The infecting microbe is inoculated through small and even invisible lesions of the skin about the nose and mouth (spontaneous or facial erysipelas). It is possible for intravascular infection to occur.

Clinical History.—I shall discuss only *idiopathic* erysipelas, the traumatic variety falling within the domain of surgical treatises.

Incubation.—This is somewhat varied, though it ranges usually from seven to fourteen days. The *prodromal symptoms* are, for the most part, general in character, consisting in headache, restlessness, cough and sore throat, anorexia, and slight or moderate pyrexia. These endure for a very variable period—from a few hours to several days.

Invasion Stage.—The symptoms are (1) local and (2) general.

(1) At first the affected part feels hot, tense, painful, and is tender to the touch. A circumscribed area becomes red, swollen, firm, and shining, and simultaneously the subjective symptoms (pain, heat, etc.) become aggravated. The *point of election* is usually on the nose, but it may be on the ear, the face, or elsewhere about the head. The inflamed, swollen zone spreads, chiefly in the direction of one or the other side of the head. Separating the diseased from the unaffected skin there is a sharp line of demarcation—an elevated brawny ridge; this ridge presents a "zigzag irregularity of outline, like the burned edges of a sheet of paper" (Warren). While the inflammation is advancing there may be noted, beyond the border of the latter, little red streaks and spots that grow in area till at last they become confluent. Any natural prominence or fold in the integument may prevent extension of the inflammation (*e. g.*, nasolabial folds). In cases of *average severity* the face is much swollen, the eyes closed on account of tumefaction of the eyelids, the ears greatly enlarged (better marked on one side than the other), the scalp swollen and tender, and the facial lineaments often changed beyond recognition. Tenderness to pressure is a constant feature. In a minority of the cases the inflammatory process extends from the head to the arms, to the trunk, and even to the lower extremities (*erysipelas migrans*), and in such instances the face may be healed while the disease is yet extending. When the disease is arrested the peripheral ridge ceases to extend and grows pale.

The epidermal layer may become elevated over circumscribed areas, giving rise to larger or smaller vesicles or bullæ (*erysipelas vesiculosum*). Suppuration may attack these large vesicles, whereupon they fill with pus (*erysipelas pustulosum*). From intense infiltration the part or parts may become gangrenous—*erysipelas gangrænosum*. Enlargement of the cerebral lymph-glands is common. Desquamation follows erysipelas, and the complexion is more delicate than before the attack.

(2) **General Symptoms.**—With the *onset* of the attack the patient is seized with repeated fits of chilliness; less commonly, a severe rigor occurs. Immediately, and more rapidly than before, the *temperature*

rises to a height of 104° or 105° F. (40°–40.5° C.) on the evening of the first day. As a rule, the temperature reaches its maximum (105° to 107° F.—40.5° to 41.6° C.) on the third evening. Marked nocturnal remissions of temperature (2° to 5° F.—1.1° to 2.7° C.) after a few days of continued fever are the rule. At the end of a week the temperature declines rapidly to normal, *i. e.*, by crisis. Sometimes, however, the course of the fever is prolonged and defervescence may be less critical (lysis). In erysipelas migrans a long and decidedly irregular temperature-curve is presented, and the same remark applies when complications are present. Czyhlarz[1] reports 29 afebrile erysipelas cases, all in women. The *pulse* is frequent, of good volume, and soft. I have been able to confirm the observations of Da Costa, Strümpell, and others, that the cutaneous inflammation in erysipelas may advance to a slight extent even after the temperature has returned to the normal grade.

The *tongue* is furred, the anorexia intensified, and nausea and vomiting occur. The *bowels* are usually constipated, though I have observed instances in which marked diarrhea developed at a late stage. The inflammation may extend to the mucous membrane of the throat and larynx, causing swelling and edema of the parts. It may also involve the serous membranes, though rarely. The *nervous symptoms* are intense headache and restlessness, with some mental aberration at night. Actual nocturnal delirium appears in the severer forms, and in drunkards delirium tremens may suddenly develop. The *urine* presents the usual febrile characters. Commonly it contains a little albumin, and rarely acute nephritis occurs as a complication. Urobilinuria, the expression of an acute parenchymatous hepatitis, was present in 9 cases reported by Hildebrandt. A polymorphonuclear leukocytosis, parallel with the severity of the infection, occurs in erysipelas. The blood, however, must come from the warmed finger (Chantemesse and Ray[2]).

There is a direct correspondence between the intensity of the local and constitutional disturbances in this disease. Often in severe forms (such as are apt to arise in old, much enfeebled, or intemperate persons) of facial erysipelas the *typhoid* (adynamic) *condition* is developed.

Complications and Varieties.—An analysis of 1674 cases of erysipelas with particular reference to complications gave an interesting series of results. Some are given here in the order of frequency of occurrence: Abscess, 105; rheumatism, 20; delirium tremens, 10; lobar pneumonia, active delirium, phlebitis, pleurisy, each 7; acute nephritis, 6; synovitis and diarrhea, each 5; tonsillitis, 3; catarrhal pneumonia, otitis media, edema of the larynx, acute bronchitis, each 2.[3] Some of these conditions are septic in nature and due to the primary infection.

The fact that acute articular rheumatism is a relatively frequent complication of erysipelas is worthy of special notice, for the reason that the attention of the profession has not hitherto been called to it. The symptoms of rheumatism usually come on several days after the onset of erysipelas. In a few instances pneumonia appeared early, being due most probably to special localizations of the specific streptococcus. To such

[1] *Berliner klinische Wochenschrift*, Sept. 11, 1911.

[2] *Presse méd.* July 1, 1899; *Saunders' Year Book* for 1901.

[3] "The Complicating Conditions, Associated Diseases, and Mortality-rate in Erysipelas," by the author: *The Int. Med. Mag.* for Oct., 1893.

cases the term "pneumo-erysipelas" may be appropriately applied. The cases—2 in number—in which acute nephritis developed during the first few days of the attack should in like manner be termed "nephro-erysipelas." Meningitis was present in a single instance only.

Three other forms—namely, *phlegmonous* or cellulo-cutaneous, *relapsing* erysipelas, and *erysipelas neonatorum*—should be mentioned. The first exhibits an inflammation of the subcutaneous tissue, which tends to suppurate. Relapsing erysipelas constitutes the chronic form of the disease, recurring at intervals, and usually in the same locality. It is commonly due to some deep-seated focus of suppuration. Erysipelas neonatorum is the result of infection of the stump of the umbilical cord. From the navel the inflammation spreads to the thighs and genitals. As a rule, there is fever, followed in a few days by fatal collapse.

Sequelæ.—The hair often falls, but it is usually replaced by a fresh crop. Otitis media and chronic nephritis may date from an attack of erysipelas. *Per contra*, erysipelas is reputed to be curative of certain affections (eczema, lupus, carcinoma, sarcoma).

Out of 476 cases collected by me relapses occurred in 54 (11.3 per cent.), and in 1 of these instances 5 relapses occurred; in 2 others, 4.[1]

The **diagnosis** is made with ease after the eruption has fully developed, and its appearance, seat, and behavior, particularly the manner of extension of the brawny, ridge-like edge (best marked on the forehead), are the features that distinguish it from every other disease. A bacteriologic diagnosis is often possible, the streptococcus being found in the pus and secretions from the naso-pharynx.

Differential Diagnosis.—*Erythema* produces superficial redness, but is not attended with heat, swelling, or fever. *Urticaria* assumes the form of pale-red circular wheals, which cause marked itching and appear in successive crops, often disappearing in the course of a few hours. *Acute eczema* of the face, when intense, may resemble erysipelas; but it lacks the peculiar border and mode of progression so characteristic of the latter disease. Again, eczema produces troublesome itching, and the swelling is less than in erysipelas. *Chronic erythematous eczema* is met with later in life, is without fever, without any considerable swelling or pain, and excites intense itching. *Eczema nodosum* is characterized by its nodosities near the joints.

Course and Duration.—In my own experience, based upon 1880 cases,[2] the average duration (including the prodromal stage and period of convalescence) in persons under forty years of age is fourteen days. The course of the disease is much lengthened by complications, the pre-existence of chronic affections, and by age (after the fiftieth year).

The **prognosis** is favorable, and it is rare for erysipelas to assume a malignant type. Perhaps the chief dangers lie in certain complications, especially extensive suppuration, pneumonia, acute nephritis, delirium tremens, etc. Acute articular rheumatism is comparatively harmless; but *previous debility*, especially if dependent upon chronic diseases, as syphilis, chronic rheumatism, gout, tuberculosis, organic disease of the heart, and the like, increases the percentage of deaths considerably. Again, age has a positive influence upon the mortality, which it augments moderately after the forty-fifth year, and most decidedly after the sixtieth

[1] *Journal of the American Medical Association*, July 22, 1893, by the writer.

[2] "Points in the Etiology and Clinical History of Erysipelas," by the writer: *Journal of the Am. Med. Assoc.*, July 22, 1893.

year. Of 2663 deaths due to erysipelas (United States Census Report), the death-rate per 100,000 inhabitants was as follows: under 5 years, 31.34; 5 to 15 years, 0.81; 15 to 45 years, 2.80; 45 to 65 years, 8.88; 65 and over, 38.55 (Wm. L. Rodman). Death is due to exhaustion.

The *mortality-rate* is low, as shown by the results of my own collective investigations into the subject. I found the general average death-rate to be 5.6 per cent., while in cases from private practice it was 4 per cent. In persons over seventy years it was 46 per cent. The traumatic cases gave a mortality of 14.5 per cent.

Treatment.—The treatment of erysipelas falls naturally into four subdivisions: (1) *Dietetic;* (2) *Constitutional;* (3) *Local;* (4) *Prophylactic.*

(1) **Dietetic.**—Proper attention to the diet is of the first importance. It must be generous and composed of highly nutritious articles, and if the temperature be high, only liquid forms of nourishment should be administered in definite quantities and at stated, brief intervals. Rectal alimentation should be resorted to if the stomach rejects a suitable dietary. Lack of attention to the patient's diet during the primary attack tends to increase the frequency of relapse. In persons over fifty years of age, and in those in whom the vital processes have been lowered on account of previous chronic diseases, correct alimenation is of paramount importance, often abridging the course of the affection.

(2) **Constitutional Treatment.**—When, despite an appropriate diet, the pulse becomes very rapid and feeble, the heart's first sound indistinct, and the tongue dry, indications for the use of stimulants exist. Alcohol may be given with a comparatively free hand, 12 to 16 ounces (360.0–480.0) of whisky daily in divided portions. Strychnin gives prompt results, and digitalis may be used in severe cases. In marked gastric irritability champagne is to be preferred. The eliminative organs, especially the kidneys, are to be stimulated, so as to rid the economy of the bacillary toxins.

The tincture of the chlorid of iron was first extensively used in this disease by English authorities, and was formerly regarded by most clinicians as a truly specific remedy. In 74 cases of erysipelas which were treated by this remedy alone, the average quantity being 1 dram (4.0) daily in divided doses, in the Pennsylvania Hospital by Drs. Lewis, DaCosta, Longstreth, Meigs, and others, the death-rate was 4 per cent.[1] Other preparations of iron, however, are equally efficacious. Quinin is a valuable remedy in erysipelas, and during the past twelve years I have employed it in not less than 30 cases, confining its use to instances in which the temperature touched 103° F. (39.4° C.), and, with a single exception, in uncomplicated cases (22 in number) the nocturnal remissions were decidedly greater. In every instance iron in some form was administered simultaneously. Numerous *antiseptic* remedies have been recommended.

Antistreptococcus Serum.—André, Robinson, Cox, Anderson, and others have reported instances of its successful use. The serum is injected subcutaneously; its influence endures over several days, but it is important that the injections are repeated at forty-eight-hour intervals. Marmorek's serum (care being taken that it is not too old) is to be preferred. G. H. Sherman reports uniformly good results from the use of stock vaccines (streptococcic), especially when used early in the course of the disease. The dose is 20,000,000 for the adult, and where local im-

[1] "The Treatment of Erysipelas," by the writer, *Therapeutic Gazette*, July 16, 1894.

provement with the reduction of temperature does not occur, this should be repeated at the end of twenty-four hours.

Certain *symptoms* demand internal medication. When the fever is high, its reduction is best accomplished by means of cold spongings combined with the ice-cap, or cold or gradually cooled baths. Guaiacol applied externally has recently been employed for the same purpose.

For *marked nervous phenomena*, such as pain, sleeplessness, and active delirium, hyoscin hydrobromate (gr. $\frac{1}{100}$—0.0006) has been tried hypodermically at the Medico-Chirurgical, Pennsylvania, and Philadelphia hospitals, and has given promise of being a valuable remedy. It should not be employed when the heart-power is deficient. For the same indication we may utilize the following: Sodium bromid, gr. v (0.324) every two hours, or gr. xx–xxx (1.296–1.944) at night; morphin, gr. $\frac{1}{8}$ (0.008), and chloral, gr. x (0.648), in combination every half-hour for three doses; potassium bromid, gr. x (0.646), and tincture of cannabis indica, ♏x (0.666), in combination, and morphin, gr. $\frac{1}{6}$ (0.0108), hypodermically.

The treatment of the various *complications* must be conducted in accordance with general principles applicable to each.

3. **Local measures** have always held a prominent place in the treatment of erysipelas. In my paper previously cited those most frequently used were elm (37 cases); lead-water and laudanum (20 cases); carbolic acid (1 : 40), injected subcutaneously (18 cases); zinc oxid (14 cases); mercuric chlorid solution (14 cases); ichthyol ointment with lanolin (8 cases), etc. P. Ph. Smolitcheff,[1]

℞. Tr. iodi, 25.0 grams;
Ol. camphor,
Ichthyoli, *āā* 12.5 grams.—M.
Sig. For external use. Shake before applying.

Many of these preparations were prescribed for their effect in excluding the air—a leading indication. This I am in the habit of meeting by the use of carbolized vaselin or cool carbolized oil. Ichthyolcollodion (strength 10 to 50 per cent.), painted over the erysipelatous area and also over the surrounding healthy skin for 2 or 3 cm. has been advocated.

Tucker[2] recommends the application of a saturated solution of magnesium sulphate in water. This is applied in facial cases on a mask consisting of from fifteen to twenty thicknesses of ordinary gauze, of sufficient size to extend beyond the area involved, with a small opening to permit breathing, but none for the eyes. After thorough saturation with the solution, the mask is applied and covered with oiled silk or wax paper; it is wetted often enough to assure a moist dressing—usually every second hour. The dressing should not be removed oftener than once in twelve hours to permit an inspection of the parts.

A knowledge of the microbic nature of erysipelas has led to the local application of numerous antiseptic remedies. Mention has been made of the method of injecting carbolic acid. Here the aim is to check the spread of the inflammatory process by inserting the needle at numerous points just beyond the inflamed border. The method (introduced by Heuter) has been much practised by Henry at the Philadelphia Hospital, and is especially applicable in erysipelas migrans. In the statistics before given a solution of mercuric chlorid (1 : 4000) was used locally in 14

[1] *Medical News*, Nov. 14, 1903. [2] *Therapeutic Gazette*, June 15, 1908.

instances, to which I can add the results of 12 others at the Medico-Chirurgical Hospital and in private practice. In a few cases it was injected beneath the skin, as in the case of the carbolic acid. More recently it has been recommended to scarify the affected part and follow with the application of a solution of mercuric chlorid. In view of the fact that the streptococcus is found chiefly in the more superficial channels of the corium, it follows that it may be attacked directly by the mercuric chlorid solution when the latter is used after scarification; and this method of treatment is at once most promising and rational. G. L. Curtis[1] advises sodium sulphate, which acts by depriving the germs of oxygen, as a local application. MacLennan advocates a saturated solution of picric acid as a local remedy.

(4) **Prophylaxis** embraces isolation and care of the skin of the whole body. Bathing with a boric-acid wash (3 per cent.), at intervals of several hours, so as to disinfect the desquamating epidermis, removes a source of danger. It is probable that relapses are sometimes due to autoinfection. Frequent change of the body-linen is to be advised and removal to another room during convalescence may prevent a relapse. Admission of erysipelatous patients to hospitals should be refused, except such institutions be provided with an isolation building.

DIPHTHERIA.

(*Diphtheritis; Angina Maligna; Croup.*)

Definition.—An acute, contagious disease caused by the Klebs-Löffler bacillus, and characterized, anatomically, by a croupous-diphtheritic faucitis, less commonly rhinitis and laryngitis. Clinically, it is characterized by irregular fever, prostration, and albuminuria; also by the secondary development of toxemia, and often cardiac failure. It is commonly followed by peculiar paralyses. In large municipalities it behaves endemically, and from time to time epidemically. The disease, however, is less prevalent than formerly.

Pseudo-diphtheria.—There are forms of inflammation occurring most frequently in the pharynx and adjacent air-passages (and also in many other parts of the body) that are attended with the formation of a pseudo-membrane, and are not caused by the Klebs-Löffler bacillus. These cases have been studied exhaustively by Prudden and others, who have usually found the streptococcus. The latter, however, has been found in the inflamed mucous surfaces met with in erysipelas, scarlatina, and measles. Vincent's angina is a form of pseudodiphtheria.

Pathology.—The **true diphtheritic inflammation** has for its chief pathologic peculiarity the production of a fibrinous exudate. When the inflammation is superficial and of a mild grade, a croupous membrane is produced which can be easily removed from the mucosa, which it covers. In the severer types of the affection, however, the fibrinous membrane infiltrates all the layers of the mucosa, which undergoes necrosis more or less nearly complete. In the severest forms the submucous layer may also become necrotic. It is to be borne in mind that the production of the fibrinous exudate in diphtheria is always preceded by coagulation-

[1] *Med. Record*, April 20, 1901.

necrosis of the epithelium. The membrane-formation is accompanied by changes in the underlying tissue which represent a combination of degeneration and exudation (Councilman, Mallory, and Pearce). The mucous membrane surrounding the exudate is hyperemic, more or less edematous, and the seat of muco-purulent secretions.

The Pseudo-membrane.—Its composition comprises fibrin, pus, disintegrated leukocytes, flakes of necrosed epithelium, bacilli, and sometimes red blood-corpuscles. The fibrin has two main sources: (*a*) "The fibrinogen of the inflammatory matter," which transudes through the capillary walls; and (*b*) Disintegrated, migratory leukocytes, which form branching fibrillæ. Weigert holds that the inflammatory exudation is coagulated by a ferment derived from the disintegrated leukocytes.

The Klebs-Löffler bacilli are found in the meshes of the fibrillæ, in the granular fibrin, and on the adjacent mucous membrane; they are never found growing in living tissue, but always in necrotic tissue. Frequently other micro-organisms are associated (streptococci, staphylococci, etc.). The membrane presents a grayish-white color; it is thick, firm, and adherent, so that its removal entire cannot be effected without great difficulty, and without, as a rule, injury to the surface, as shown by bleeding, etc. The character of the pseudo-membrane is affected by the nature of the underlying structure; thus in the pharynx it is firmer and less easily separable than in the larynx and trachea, where a distinct basement-membrane is found (Flexner). As the membrane becomes older its color is apt to grow darker, becoming yellow or even dark brown. It sometimes becomes gangrenous, and softens or disintegrates, with the production of a very offensive brownish, semiliquid excretion. The advancing edge of the false membrane is usually thin. On the other hand, when the process has become arrested the edge is apt to look raised or wrinkled, and later it may be distinctly curled up.

The membrane may extend downward into the ramifications of the bronchi. In such cases there is apt to be a lobular pneumonia, but the lung may be invaded by the bacillus without any clinical indications. Lung-infection, due to the streptococci and (less commonly) the pneumococci, is common. A generalized bronchitis extending to the smaller bronchi is common from the irritation of aspirated substances. In rare cases the membrane has spread into the esophagus and even into the stomach.

After separation of a croupous membrane repair consists merely in a restoration of the epithelial layer—a process which is initiated by the fragments of epithelium that remain along the edges of the diseased area, and proceeds centrally. On the other hand, in true diphtheria, with necrosis (more or less complete) of the mucosa, sloughing occurs, and the missing structures are replaced by cicatricial tissues.

The Heart.—The muscular structure and the nervous mechanism suffer most. The histologic changes may be of the parenchymatous variety, but only in mild instances; whereas in severer cases fatty degeneration is conspicuous. In still other cases the chief pathologic characteristic is an interstitial myocarditis, and rarely the lesions of pericarditis and endocarditis have been noted. The heart is by no means always involved.

The **spleen** is commonly enlarged, though not to an excessive degree. The **blood** is dark, its coagulability is greatly diminished, and Canon and Frosch have in a few cases found the bacilli in the blood of those dying

of diphtheria. The red corpuscles are somewhat decreased in number during the course of the disease, while the white corpuscles are increased. Bouchut and Dulinsay consider the grade of leukocytosis of prognostic value, and claim that it varies directly with the severity. Grawitz has determined in numerous cases a higher specific gravity of the blood during diphtheria. The **lymphatic glands** of the neck become swollen, as a rule, and are often greatly enlarged, but they show little tendency to suppurate. In pronouncedly septic cases in which a mixed infection is found by culture a good deal of tumefaction of the neck occurs, this sometimes even obliterating the normal contour from jaw to clavicle.

The Kidneys.—The kidneys show degenerative changes, the usual *kidney-lesion* being a hyperemic swelling with edema of the interstitial tissues, and often hemorrhagic spots in the cortex. Sometimes there is a marked glomerulo-nephritis, and rarely a diffuse granular degeneration of the epithelium. Minute areas of necrosis have been observed in the internal organs, in which fibrin has been found deposited (Oertel). Welch and Flexner have produced, by artificial inoculation upon guinea-pigs, kittens, and rabbits, foci of cell-death in the lymph-glands throughout the body, in the spleen, liver, lungs, heart, and intestinal mucosa. When the dose is small and the animal lives several weeks, paralysis may develop.

The **nerves**, in cases of paralysis, have shown parenchymatous and interstitial inflammatory lesions. In paralysis of throat-muscles (*i. e.*, those near the locality of the pseudo-membranous inflammation) the latter show also round-cell infiltration and fatty degeneration of the fibers. The nerve-fibers of the central nervous system may also show fatty degenerative changes. In fatal cases lesions have been found to engage either the meninges, the cerebro-spinal substance, or the nerves.

Etiology.—True diphtheria is caused by the Klebs-Löffler bacillus, and all cases of supposed diphtheria in which the bacillus is absent are to be regarded as non-diphtheritic. The etiologic is, therefore, quite different from the pathologic significance of this term. Recent researches have removed all doubt as to the specific nature of the Klebs-Löffler bacillus.

Bacteriology.—The bacillus diphtheriæ nearly equals in length that of the bacillus tuberculosis, and is twice the diameter of the latter. It has rounded extremities, which are also frequently bulbous, giving it the appearance of a dumb-bell. At times one end only is clubbed, or, more rarely, one or both ends appear pointed. The bacilli are immobile, do not form spores, and stain readily, the best agent being alkaline methyl-blue. Their manner of taking the stain is important. The bacilli show alternating segments of darker and lighter stained areas, and often minute dots showing a most intense and deep staining. They grow on most culture-media, but for clinical purposes Löffler's blood-serum is important (3 parts blood-serum and 1 part neutral or slightly alkaline nutritive bouillon, containing 1 per cent. of glucose). Inoculated on this, they outgrow all other organisms that may be present, and within eight hours or less show numerous spots, one-half to one millimeter in diameter, which have a dull surface and a dense white or somewhat yellowish color. There are usually present also smaller points which have different appearances and which are colonies of other organisms. The former are the colonies of the bacillus diphtheriæ, and from these microscopic preparations and (by further cultivation) pure cultures can be obtained. The bacilli are semi-anaërobic, and thrive at the temper-

ature of the human body; a temperature of 122°–136.5° F. (50°–58° C.) causes their destruction in ten minutes.

Pseudo-diphtheria Bacillus or Bacillus Xerosis.—From many cases, often showing no lesions, an organism may be obtained that is identical in appearance, manner of culture, growth, etc. with the bacillus diphtheriæ, but inoculation with it causes no lesions. The works of Abbott, Roux, Yersin, and others seem to show that this is an attenuated form of the true bacillus, and varying grades of pathogenicity may be found between the two. The distinction from the pathogenic bacillus can only be made by determining the lack of infection after inoculation.

Site of Infection.—In the human family the seat of election of the bacillus diphtheriæ is usually the faucial mucosa, and less frequently other mucous surfaces and abraded skin. The bacilli do not penetrate the mucosa, and hence do not find their way into the lymphatic or circulatory system, but remain at or very near the site of the local changes.

The Toxins.—Toxins are absorbed from the diseased spots by the lymphatics and blood-vessels, and produce the general phenomena in uncomplicated cases. They have been isolated from artificial cultivations of the microbe, and when inoculated the chief ptomain of the Klebs-Löffler bacillus so modifies the solids and liquids of the body as to render the subject immune (Behring). Another, however, if employed in like manner, produces dangerous and even fatal symptoms (convulsions, paralysis, etc.).

It is certain that the bacillus can maintain an existence for months outside of the body, though its usual habitat is unknown unless it be the organic constituents of the superficial soil. The virulence of its products is modified by many individual conditions, and chief among these is a healthy and intact condition of the mucous membranes, which greatly reduces the susceptibility to the disease.

Associated Microbes.—With the Klebs-Löffler bacillus are frequently found other microbes, especially streptococci and staphylococci. These pass beyond the site of local infection, reaching the internal viscera and other structures, and, as will be seen hereafter, give rise to the serious septic element of the disease. W. Bloch and P. Sommerfield,[1] in studies on the pathogenicity of the Löffler bacillus, have verified the accepted statement with reference to the germ, their article being a good exposition of the present status of the bacteriology of diphtheria. From a study of 436 cases, the authors state that the Löffler bacillus was never found in culture, but always associated with other bacteria, among which streptococci played the greatest part. The two doctrines concerning the relation of streptococci to septic diphtheria are given, the one being that the streptococci increase the virulence of the diphtheria bacillus and cause sepsis by gaining access to the circulation; the other is that the diphtheria toxin, by its effect on the organism, prepares the way for an invasion by streptococci. The pneumococcus may be found.

Modes of Infection.—When the bacillus leaves the body of the sick it is contained in particles or shreds of the diphtheritic membrane or in the expired air. Infection may then occur (*a*) *By direct contact* with the shreds of membrane thrown off—*e. g.*, when the latter are ejected by coughing and lodge upon the conjunctivæ or faucial mucosa of bystanders. The deadly poison is sometimes transferred to the physician and

[1] *Arch. f. Kinder.*, Bd. li., Heft 2.

attendants, with resulting infection, from the sucking of tracheotomy tubes. (*b*) *By inhaling the air* surrounding the patient (contagion). Infection by contagion, however, does not extend beyond a radius of a few feet from the patient. (*c*) A very leading matter of conveyance of the bacillus from the sick to the healthy is by *fomites*. The contagion adheres tenaciously to a great variety of objects (toys, clothing, library books, letters, slates and drinking-cups in the public schools, etc.), and in this way the germs of diphtheria have been transferred over great distances and have given rise to the disease long after. The latter fact renders it difficult to trace certain cases to previous ones, to which they invariably owe their origin. (*d*) *Sewer gas, per se*, is to be regarded as non-pathogenic, or at least so far as this affection is concerned (Laws). It may, however, become a carrier of diphtheritic poison. (*e*) *Domestic animals* may be occasional carriers, especially cats. (*f*) The disease is kept alive in a community largely by virulent organisms in immune persons ("healthy carriers"—13.3 per cent.—Sobernheim). Rarely, "latent carriers," who conceal the bacillus for a time before they show the clinical evidences of diphtheria, may convey the disease.

Our knowledge as to how the infection occurs is incomplete. We know definitely the usual point of local infection in man, and also that a catarrhal mucosa or an open lesion of a mucous surface invites infection. It is not certain, however, that even a slight lesion of the mucous surface is essential to infection. Some writers claim still that the Klebs-Löffler bacillus may enter the blood through the respiratory system and give rise to primary constitutional symptoms, the local manifestations in the throat being secondary.

Predisposing Factors.—(1) *Age.*—This is the most important factor, diphtheria being, in the main, a disease of childhood. Most cases occur between the second and seventh years, while the receptivity diminishes rapidly after the tenth year. Instances have, however, been observed up to the fiftieth or even the sixtieth year. During the first year of life also it is rare. (2) *Sex.*—This is without appreciable influence. (3) *Season.*—Cases are more numerous in winter and spring than at other seasons. (4) *Climate.*—Diphtheria is met with less frequently in tropical than in temperate and cold climates. Humidity favors the propagation of the diphtheria germ, and hence damp cellars also promote the spread of the disease. (5) *Unhygienic Conditions.*—Unfavorable sanitary surroundings tend to lower vitality, thus increasing the susceptibility to the specific virus. Most epidemic outbreaks have held more or less intimate relationship with decomposing organic matter, defective drainage and sewage, cesspools, etc., though it is to be especially remembered that the disease often prevails in sparsely-settled rural districts.

Immunity.—A single attack does not confer perfect immunity. Second and third attacks not infrequently occur in the same individual.

Symptoms.—**Incubation.**—The *duration* of this period is from two to seven or ten days, and in a small percentage of the cases it may be longer. In virulent epidemics and when the disease is produced experimentally the incubation-stage is short—from twelve hours to two or three days. The *prodromal indications* of diphtheria are not strikingly characteristic. They may either be acute in character or very mild; but usually the child will complain of feeling weary and indisposed to

play, of sensations of chilliness, and of pain in the head, back, and limbs. In young children the onset of diphtheria may be marked by *convulsions*. There is nothing in this early stage of the disease to distinguish it from simple pharyngitis or tonsillitis. There may be some fever, not very high—an elevation of one or two degrees at most. The urine contains a small amount of albumin. R. Koch found diphtheria bacilli in the urine of 2 out of 26 diphtheria patients. The child often complains of discomfort in swallowing, and on examination the fauces will be found to be reddened, and in a short time the exudate will be found on the tonsils or soft palate. This is the usual type of **simple tonsillar diphtheria.**

Pharyngeal Diphtheria.—The symptoms are usually *slower of development* than in tonsillitis. The child is sluggish, looks heavy-eyed, languid, and pale for several days. The *fever* may not rise above 101° or 102° F. (38.8° C.). On examining the throat, however, it is found to be swollen and red, and if *lividity* is more pronounced than the swelling, it suggests the true nature of the disease. The *membrane* begins on the tonsils in the form of small patches of yellow exudate, resembling the thick, cheesy plugs of inspissated dead epithelium and secretion which issue from the mouths of the follicles of the tonsils during the course of acute or chronic tonsillitis. Quite early this exudate is easily removable. The membrane spreads from the tonsils to the soft palate and half arches within a few days, and it may also appear on the pharyngeal wall. During this stage the throat may become much swollen and the tonsils greatly enlarged, frequently meeting in the median line. The *glands* immediately beneath the angle of the lower jaw on one or usually both sides become hard, painful, and slightly enlarged; the swelling of these glands is not great in mild forms, although their presence, in association with the foregoing symptoms, is an almost infallible indication of the disease. The child, as a rule, shows grave constitutional symptoms for a few days and albuminuria is present. Acetonuria is common in the severer forms of the disease. The *temperature* is not characteristic, as a rule not being high, and the pulse is rapid and weak, being out of proportion to the general indications of the disease. The blood-pressure is below normal in about one-third of the cases (Rolleston), and the degrees of depression bear a direct relation to the severity of the infection. In mild cases the symptoms abate by the end of the first week, and the pseudo-membrane separates, leaving a red, inflamed surface behind. The child is prostrated for a number of weeks, and in about 20 per cent. of the cases neuritis, with its accompanying paralysis, occurs. Simple leukocytosis is present in diphtheria, although this symptom may be absent in mild cases.

Variations in Manifestation.—Diphtheria may exhibit variations as regards the seat of attack and the severity of the poisoning. In some epidemics the Klebs-Löffler bacillus seems to be more active, or more virulent, than in others. The severity of the attack does not seem to depend on the amount of the pseudo-membrane, but rather, according to Rotch, upon three factors: (1) the virulence of the bacteria; (2) the local resistance; and (3) the general resistance. The mucous membrane of any part of the body (lips, tongue, conjunctivæ, vulva, or glans penis) may be the seat of the membranous growth.

Malignant Diphtheria.—The symptoms are severe from the commencement. There are one or at most two days of slight illness, and

then alarming symptoms manifest themselves, *cardiac failure* possibly setting in without a specially severe local lesion. *Vomiting* and *high fever*, resembling the onset of scarlet fever, may initiate the attack; and within a few hours we may find extensive swelling at the angles of the jaws, of stony hardness, an offensive bloody discharge coming from the nostrils, accompanied with difficulty in opening the mouth. If the *throat* is examined, there will be found extensive swelling of the tonsils, even to meeting, the uvula and soft palate being edematous and covered with much sloughy-looking membrane. The *temperature* in severe cases soon reaches a point between 103° and 104° F. (40° C.), while the *heart-beats* become exceedingly feeble. In a day or two the cellulitis extends, the face becomes edematous, the skin pits all over the face, neck, sternum, and chest-walls. The patient becomes drowsy, cyanotic, and an erythematous rash may appear about the face, neck, and chest, while a purpuric rash is not infrequent. Death occurs in such cases within one week from toxemia. Cases of diphtheria septicemia have been recorded in the literature by Mahler[1] and others.

Nasal Diphtheria.—In all severe cases of pharyngeal diphtheria the inflammatory process is likely to extend to the nasal mucous membrane. In some cases the nasal mucous membrane is found to be the first involved; the exudate may spread to the tonsils, involving the back of the soft palate and pharynx as well. In many cases of nasal diphtheria no membrane may be found during life; there may be only a purulent discharge with blood, the presence of which in the nasal passage obstructs breathing, giving rise to a bubbling sound, and rendering sleep troublesome and noisy. Cases have also been reported of formation of pseudo-membrane in the nose with mild general symptoms, and from which organisms identical with diphtheria bacilli were obtained by culture. Sometimes the cases have recurring mild attacks of pseudo-membranous inflammation of the nose, while the bacilli may be constantly present. It is probable that these cases may give rise to infections of like nature, and even of true diphtheria. In nasal diphtheria the symptoms are quite as severe as in faucial diphtheria, and in cases in which the soft palate and tonsils are also involved the general symptoms, the depression, and also the albuminuria are apt to be well marked. In all cases of coryza with fever we should be guarded as to opinion, especially if an epidemic of diphtheria is prevalent at the time. The diphtheritic inflammation may spread from the nose to the conjunctivæ, with the formation of a false membrane, and much purulent discharge may escape from the eyes, the lids of which may be greatly swollen. In this place it is well to remember that in *measles* we sometimes have a form of membranous exudation occurring on the nasal mucous membrane, and as a primary disease—"rhinitis fibrinosis"—which is not always diphtheria. This disorder runs a favorable course, the membrane being less adherent than in diphtheria. Ravenel has collected 77 cases, and in 33 out of 41 cases examined bacteriologically the Klebs-Löffler bacillus was found. Constitutional symptoms were either slight or wanting.

Wound-diphtheria.—The bacillus will not live on normal skin, but when the skin is cut or bruised, as after blistering or an eczematous condition, and when a moist, raw surface is present, this germ freely

[1] *Berliner klinische Wochenschrift*, 1907, xliv., 1499.

flourishes. Granulations also form a favorable soil. The diphtheritic germs may be introduced into the system during an operation, such as an excision of the tonsils, or even a vaginal examination; and in newborn infants the granulating surface left after sloughing of the cord may become the seat of diphtheritic inflammation.

Laryngeal Diphtheria or Membranous Croup.—The exudate may appear first on the mucous membrane of the larynx, and in these cases the mucous membrane of the nose and pharynx may never give evidence of a false membrane. A close inspection of the posterior aspect of the palate and tonsils, however, may reveal a slight primary membranous formation in these situations. In laryngeal cases the first symptom is a *cough* of a *harsh, metallic, ringing character*, and never to be forgotten when once heard. The *temperature* may be slightly above normal, or even, in many cases, normal. The toxic absorption is slight, on account of the locality affected, and the constitutional symptoms are usually mild. The *local symptoms*, however, are very alarming, and result from laryngeal obstruction, there being marked *dyspnea* with retraction of the intercostal and supraclavicular spaces, and later of the epigastrium and lower chest, with an increasing *cyanosis*. The child is soon restless, is forced to sit up to breathe, and for the same reason bends forward with its head thrown back. In these extreme cases unless relief is soon gained the child dies of suffocation. In many instances a slower form of suffocation may result from the extension of the membrane downward to the bronchi.

Complications.—Local complications may be mentioned—*e. g.*, *hemorrhage* from the nose and throat in the more severe ulcerative cases. *Skin-rashes* are not unusual, especially *diffuse erythema*.

Broncho-pneumonia is the most serious pulmonary complication of diphtheria. It is is not produced by the Klebs-Löffler bacillus as a rule, but by the streptococcus or pneumococcus. Broncho-pneumonia usually terminates laryngeal cases that have been operated upon.

Albuminuria is a constant symptom (not a complication) of the disease (*vide supra*), and is almost as certain in establishing a diagnosis of true diphtheria as a bacteriologic examination. It is met with in both mild and severe cases, and the greater the amount of albumin the more severe the case. Acute nephritis not infrequently complicates diphtheria; it is usually not accompanied by edema or anasarca. It may set in with suppression of urine.

Dysphagia may, by its constant existence throughout the disease, produce a profound impression on the general nutrition. Involvement of the *conjunctivæ* is a rare but grave complication.

Otitis media occurs frequently, and may be a troublesome complication as well as a sequel. Snow[1] reports a case of diphtheria complicated with Escherich's pseudo-tetanus.

The most frequent **sequelæ** are anemia, chronic naso-pharyngeal catarrh, and peripheral neuritis and its associated paralysis.

Anemia may so prolong convalescence as to expose the child to some intercurrent disorder. The *chronic naso-pharyngeal catarrh* may be marked and offer a favorable ground for new diphtheritic invasion. *Paralyses*—*e. g.*, palatal and cardiac—may appear in the first and second weeks of the disease. Other forms of paralysis occur later. Par-

[1] *Amer. Jour. Med. Sciences*, Dec, 1902.

alysis usually is first seen when the child attempts to swallow, and the food, especially if liquid, is regurgitated through the nose. This is due to a paralysis of the muscles of the soft palate, which also produces a peculiar alteration of the voice. The paralysis may take a general form, such as is seen in multiple neuritis, the lower extremities being affected and the knee-jerk absent. It may extend to the external ocular muscles and cause squint, to the ciliary muscles and cause dimness of vision from unequal accommodation, or to the muscles of the trunk in general, producing widespread paralysis. The child, unable to hold anything, may stagger about as if intoxicated, so much so as to suggest the existence of a cerebral tumor. The disturbance of vision and the absence of the patellar tendon reflex has in adults led to a mistaken diagnosis of *locomotor ataxia.* Loss of taste, deafness, and a disturbance of sensation are not infrequent. Thus, paralysis is to diphtheria what dropsy is to scarlet fever—a proof positive of the disease. To make one step more, in many sudden deaths occurring in early diphtheria, we must recognize *paralysis of the heart* outside of all toxic influence. In these cases there occurs sudden disturbance of the vagus, which may be the seat of degenerative changes in some instances at least. The *prognosis* in post-diphtheritic paralysis after the third week is favorable, while the cardiac, pharyngeal, and diaphragmatic palsies beginning before the third week are serious. Myocardial weakness tends to supervene as a sequel. It is evidenced by the sudden accession of pallor, nausea, sometimes by vomiting, and also by weak heart-sounds and a feeble, broken, irregular pulse, etc., and usually leads to a fatal termination.

Diagnosis.—The diagnoses of a pharyngeal diphtheria is not difficult if an epidemic be prevailing. The false membrane on the fauces and the presence of albumin in the urine give us a practically certain diagnosis. The only unequivocal evidence of the disease, however, is the finding of the Klebs-Löffler bacillus in the membrane.

An immediate recognition of the disease is often possible from a smear-preparation of the exudate from the throat (see Fig. 14), the Klebs-Löffler bacilli being present in sufficient numbers to be readily distinguished by the microscopist. Park, who has had a rare experience with this affection, makes the following statement: "In cases in which the disease is confined to the larynx or bronchi, surprisingly accurate results can be obtained from cultures, and although, in a certain proportion of cases, no diphtheria bacilli will be found in the first, yet they will be abundantly present in later cultures. We believe, therefore, that absolute reliance for a diagnosis cannot be placed upon a single culture from the pharynx in purely laryngeal cases." When a bacteriologic examination cannot be made the practitioner must regard as suspicious all forms of throat affections in children, and carry out measures of isolation and disinfection. In this way alone can serious errors be avoided. Mistakes usually occur in the lighter types, many of which are in reality due to the Klebs-Löffler bacillus (Osler).

Differential Diagnosis.—From *follicular tonsillitis* we differentiate diphtheria by the seat of the membrane, that of the former being *in* the tonsils, while diphtheritic membrane is *over* the tonsils and *over* the soft palate. Moreover, in follicular tonsillitis the fever is high, the onset is sudden, and it is usually associated with gastric disturbance. Albu-

minuria is generally present in diphtheria, while it is present in follicular tonsillitis in exceptional cases only. Moreover, mild cases may not present albuminuria, or fail to show the presence of albumin until later in the disease. The histories of the two cases are quite different. (For differential diagnosis between diphtheria and follicular tonsillitis, see also Table, p. 756.) In many instances of so-called diphtheroid lesions the membrane is formed only by streptococcus pyogenes (*membranous angina*), and these cases are sometimes of an intense grade.

Croupous or *membranous angina* (a *streptococcus infection*) may offer some difficulty; yet in this disease there is no tendency to spread to the nasal mucous membrane or to the larynx; there is a diminished glandular enlargement; there is no albumin, and the onset is more sudden.

In *Vincent's angina* there is an absence of the formation on the surface of the mucosa of a thick false membrane; it is an ulceromembranous process. There is a deep and often widespread necrosis of the mucosa of the palate and tonsil. Bacteriologic examination shows the presence of a large number of atypic bacilli, which are often associated with a spirillum. According to H. W. Bruce[1] there is an absence of the diphtheria bacillus.

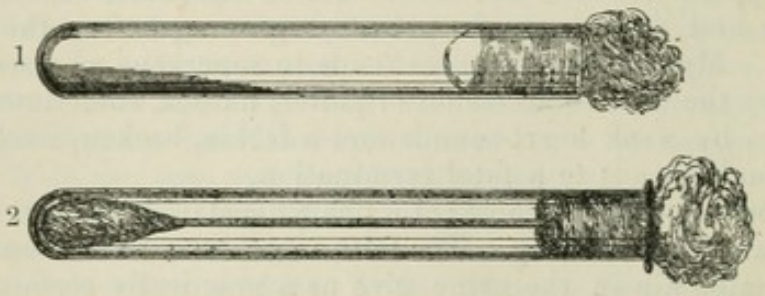


FIG. 14.—1, A tube of blood-serum; 2, a sterilized cotton swab in test-tube.

Rub the swab gently but freely against the visible exudate, and without laying it down, after withdrawing the cotton plug from the culture-tube, insert it into the latter, and rub that portion which has touched the exudate gently but thoroughly over the surface of the blood-serum without breaking its surface. Now replace the swab in its own tube, plug both tubes, and place them in the box provided by the health officials. This is to be sent to the bacteriologic expert. In laryngeal diphtheria the swab is to be passed far back and rubbed freely against the mucous membrane of the pharynx and tonsils.

Diphtheria frequently is associated with a rash, rendering it difficult to distinguish the condition from *scarlet fever;* but in diphtheria the rash is more truly an erythema, while in scarlet fever it consists of slightly raised points between which there may be an erythematous condition. The rapid pulse of scarlatina is of assistance in the discrimination. The glandular swelling and sloughy condition of the throat, however, closely resemble diphtheria, and a positive diagnosis without a bacteriologic examination is often impossible.

Prognosis.—Formerly diphtheria was at the same time the most prevalent and most fatal of the acute infections, the mortality being 30 to 40 per cent., although variable in different epidemics. The case-mortality from diphtheria has been very materially reduced since the introduction and wide use of antitoxin—certainly over 50 per cent. The remarkable diminution in the death-rate from laryngeal diphtheria has coincided precisely with the use of antitoxin. Of especially unfavorable prognosis are those cases that show large quantities of albumin in the urine, cervical glandular enlargement, excessive nasal discharge, rapid

[1] *Lancet*, July 16, 1904.

extension of the exudate, a necrotic membrane, vomiting, and partial or complete suppression of the urine. A sudden fall of temperature to a subnormal level and an irregular pulse, or bradycardia, are a bad augury. Recovery from a severe attack in which there are extreme depression and much albumin is unusual in a child under six years of age, though recovery may take place in apparently hopeless cases. The results of Morse's extensive observations are opposed to those of Bouchot and Dulinsay, who claim that the degree of leukocytosis is of prognostic value. The cases of neuritis invariably recover. The child is liable to suffer from the effects of the disease for years after apparent recovery.

The *causes of death*, in their order, are as follows: membranous croup or laryngeal stenosis; septic infection, which may be a slow death; sudden heart-failure—paralysis of the heart; bronchopneumonia, following tracheotomy or occurring during an advanced stage.

Treatment.—Prophylaxis.—The best preventive measures against diphtheria are a clean nose and mouth. The slightest appearance of a coryza must be overcome at once by the use of a mild antiseptic wash; all accumulations of crusts, dust, dried blood, etc., should be removed from the nose twice daily, especially in children attending school or during the prevalence of an epidemic. The child should be early taught to employ a small antiseptic gargle as a daily routine, using a weak solution of hydrogen dioxid or listerine. The teeth should be carefully cleaned daily, and all decaying teeth should be filled or removed. Since domestic animals, especially cats and dogs, may communicate the disease, they should be excluded from the sick room.

All cases of sore throat should be examined for the Klebs-Löffler bacillus, and, if it is found, the individual should be isolated; and all cases of diphtheria should be kept isolated until cultures taken from the throat or nose fail to indicate the presence of the specific germ. This is especially true in schools and asylums. Moreover, all persons exposed to this disease, and those caring for diphtheritic patients, should receive immunizing doses of antitoxin. Dzerjgowsky recommends the subcutaneous use of minute doses of toxin with a view to establishing active immunity. Bacteriologic examination of the throats of school-children is of the greatest aid in controlling epidemics. The fact that the Klebs-Löffler bacilli when found in healthy throats may not be active is no argument against isolation and antitoxin injections, because if the same germs were to find a broken or catarrhal membrane they would rapidly develop.

An unrecognized feature in the prophylactic treatment of the disease is seen in the uncertain period of convalescence. It frequently happens that long after all membrane has disappeared active bacilli may still cling to the throat. The persistence of the bacilli may be accounted for at times by assuming that the accessory sinuses of the nose may be involved. This condition may also continue for from two to six months, and even longer in deeply fissured tonsils; and the disease may be communicated by such throats in the act of kissing young children or adults with sensitive throats or with a broken buccal mucous membrane. For this reason the indiscriminate kissing of young children on the lips should be interdicted by the physician. Hewlett and Nankwell advise the use of endotoxin as an aid to the antitoxin treatment with a view to reducing the number of these diphtheria carriers.

Insufficient attention to isolation and disinfection of the milder cases explains the occurrence of many house-epidemics. The physician must, during his visits, wear a surgeon's apron or linen duster which has been steeped in a mercuric chlorid solution and allowed to dry. His hands and face should be washed in a similar solution on leaving the room.

Treatment of the Attack.—The treatment falls naturally under several departments: (*a*) the hygienic measures to limit the diffusion of the disease; (*b*) the local management of the throat to destroy early the toxic germs; (*c*) medication to antagonize the effect of the toxins, and eventually to overcome the complications and sequelæ.

(*a*) **Hygienic Treatment.**—The patient should be in a room well exposed to sunlight and fresh air, and superfluous furniture and hangings should be promptly removed. No stationary washstand should be allowed in the room, and Goodhart well says that many cases seem to have their origin in the proximity to foul-smelling drains. Even in mild cases the patient should be kept in bed throughout the attack. White and Smith, from a study of the heart complications in 946 cases of diphtheria, believe that the presence of murmurs and a slight degree of irregularity are no contraindications to getting out of bed at the end of two weeks, if the first sound is strong and the heart is not dilated. Patients who have been severely ill, or ill several days without treatment, should not be allowed out of bed before five weeks. The general comfort of the patient is enhanced by two daily sponge-baths of tepid salt-water or of alcohol and water.

Feeding.—Nursing infants may be fed on breast-milk obtained by a breast-pump, but should not be placed at the mother's breast (Holt). The feedings should be regular, yet lighter in quality and quantity than in health, remembering the fact that gastric disturbance is closely associated with diphtheria. The rule must be to pay every possible attention to the feeding. Milk in some form being our main dependence, it should usually be diluted, and for young children partially if not wholly peptonized. The greatest difficulty comes in the latter part of the disease, when the child is septic and most likely has a strong objection to being disturbed. At this time vomiting is most easily provoked and swallowing is rendered very difficult on account of the swelling and pain. We must not neglect the feeding even if it does cause discomfort, and here forced feeding by means of gavage is most valuable. Gavage is likely to be more successful with children under three years than rectal alimentation. In older children who object to the tube through the mouth, it may be passed through the nose with little difficulty, and gavage by this route, even in intubated cases, will be extremely satisfactory. Concentrated broths, meat-juice, and even milk-punch or raw eggs, may be given in this way.

(*b*) **Medicinal.**—*Alcohol* no longer holds a debatable ground in the treatment of diphtheria; it is the most powerful drug in our possession to offset the ravages of the disease on the nervous centers and for the control of the circulation. Stimulation should be commenced as soon as there is a reasonable certainty as to the correctness of the diagnosis, and by commencing early with whiskey or brandy we may prevent the depressing effects of the poison of diphtheria as seen in the pulse and general condition of the child. The indications for the free use of alcohol are marked prostration, feeble pulse, and a weak first sound of the heart. The

quantity must be adjusted to the age and gastric condition of the child, and usually one ounce (32.0) of good whiskey or brandy, well diluted, in twenty-four hours is sufficient for a child four years old. In very bad cases five or six times this quantity may be given, the only limit being the tolerance of the stomach. The stimulants should be mingled with the food, as the child may rebel against taking both food and stimulants.

Strychnin stands next to alcohol in importance in the treatment of diphtheria, and usually it is given in too small doses. For a child four years old gr. $\frac{1}{30}$ (0.0021) may be given every six to eight hours, and may be administered in little tablets by the mouth or hypodermically.

Digitalis does not hold an important place in the heart-weakness of diphtheria, and yet it is strongly indicated on theoretic grounds. Clinically, it has been found to have an unfavorable action on the stomach before its good influence can be had on the heart itself. The same may be said of camphor and ammonium carbonate. The aromatic spirits of ammonia is valuable for rapid effects in syncopal attacks. In cases of threatened heart-paralysis occurring late in the disease Holt has found nothing so valuable as morphin employed hypodermically, the drug being given in full doses and repeated every two hours, keeping the child under its influence for some days. In cases of diphtheria in which a murmur and slight arrhythmia develop, efforts at treatment should be concentrated on the general condition.

Internal medication should be minimized. Symptoms, as vomiting or diarrhea, are to be met with sufficient therapy only for their control.

(*c*) **Local Treatment.**—For the direct attack upon the membrane in the throat nearly all the remedies of the Pharmacopeia have been used. Gargling, swabbing, painting, spraying, and washing the throat, all have their advocates. Since the acceptance of the antitoxin treatment medical opinion has suffered a decided change as to the importance of local measures. The very best local application for pharyngeal or nasal diphtheria consists of hydrogen dioxid, diluted one-sixth, and used both as a gargle and spray as most convenient; this is usually sufficient in the early stage. Norikove[1] advises for infants who cannot use a gargle the administration of peroxid internally by the following formula: hydrogen dioxid, 5 to 7 c.c.; distilled water, 85 c.c.; syrupus simplex, 15 c.c. I have found a solution of mercuric chlorid, 1 : 1000, with an equal part of hydrogen dioxid, used as a spray, an excellent disinfectant and detergent. The tincture of iron and glycerin (one part of the former to two parts of the latter) is a valuable local remedy; it should be applied by means of a swab. The object of local treatment is a more thorough cleanliness. To avoid new lesions in making applications, the spray alone should be used, and for the nose boric-acid solutions or hydrogen dioxid, 1 : 10, will be most serviceable. Fackenheim's experience with a local spray of pyocyaneus convinced him that it is a valuable adjuvant to the serum treatment; it hastens the disappearance of the throat lesions and improves the general health. In this work the utmost tact and kindness must be maintained, for it is truly pitiable to force a struggling child. Warm, weak solutions, most thoroughly applied by means of the fountain-syringe, often have a better effect than the more frequent use of the hand-syringe or spray in cases of nasal diphtheria. Rendu advises in-

[1] *La Médicine*, Oct. 8, 1902.

halation of hot air as a supplementary measure by exposing patients to a temperature of 176° F. for five minutes at a time.

In laryngeal diphtheria the child should inhale an atmosphere laden with the vapor of slaking lime, or, whenever practicable, an atmosphere saturated with Löffler's solution (menthol 10 grams, dissolved in sufficient toluol to make 36 c.c., liq. ferri sesquichlorid, 4 c.c., absolute alcohol, 60 c.c.). J. Cordin warmly recommends mercurial fumigation for the relief of laryngeal stenosis. The development of the signs of actual stenosis, as shown by stridulous breathing, cyanosis, etc., furnishes an indication for either intubation or tracheotomy. According to my observations, the results of intubation have been quite favorable, and I would strongly recommend a trial of this procedure before resorting

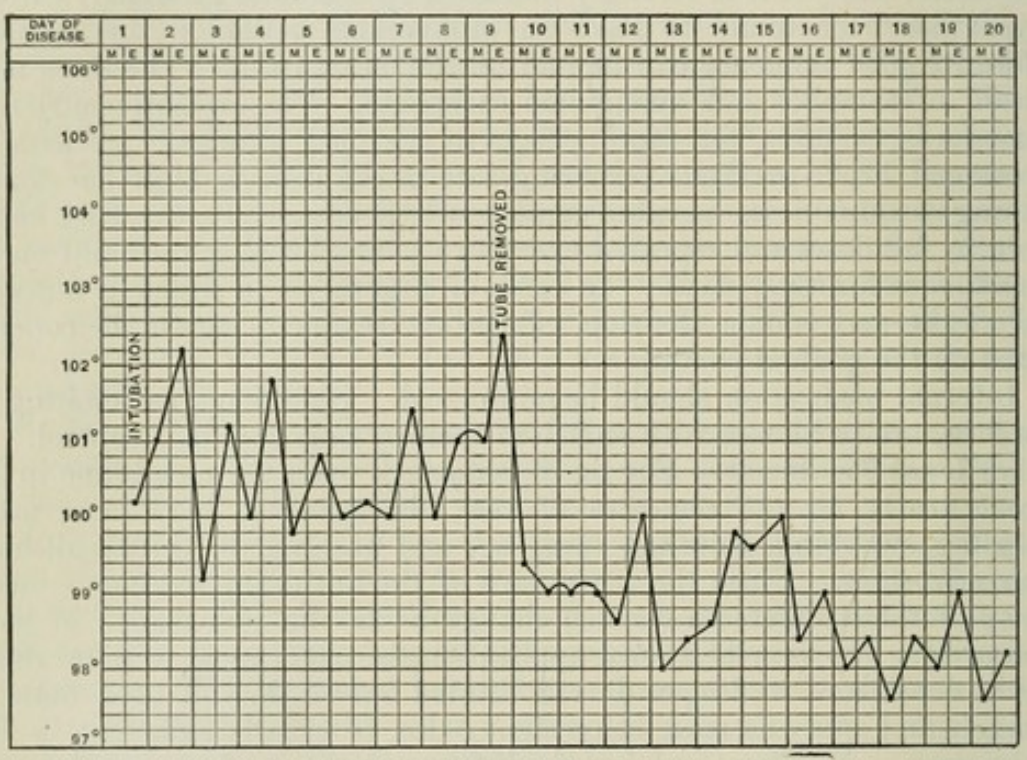


FIG. 15.—Temperature-chart of a case of diphtheria.

to tracheotomy (see temperature-chart, Fig. 15). To obviate the necessity for reintubation, vapor inhalations have been successful in my hands.

(*d*) **External Applications.**—External applications to the throat have no effect on the course of the disease. They are useful, however, in relieving the pain and the swelling in the lymph-glands. Careful massage of the neck with camphorated oil, as hot as the skin will tolerate, is very soothing; and soap liniment may be used in the same way, or, if much pain exists, chloroform liniment may be substituted. Poulticing for the relief of pain is not desirable, as it seems to favor suppuration. In older children the ice-collar has been used with good effect, and it soon brings grateful relief from the tension and subdues inflammation. Levinson recommends early lancing of suppurating glands to prevent a general septicemia. All manipulations about the child, however, should be carried on as gently as possible.

Serum-therapy; the Antitoxin Treatment.—This has now passed beyond the stage of experimentation. The general average mortality of diphtheria has been reduced by means of the antitoxin treatment to

about 10 per cent., and the greatest decrease has been shown to have occurred in children under six years of age. No physician should be guilty of failure to employ the serum in any case of diphtheria. The studies of Behring, Roux, Kitasato, and others, published in 1890, have demonstrated that the use of the blood-serum of the lower animals, artificially rendered immune against diphtheria, has a powerful healing influence upon diphtheria that has been contagiously or spontaneously acquired by man. The principle was first shown to be true of tetanus, and, late in 1892, Behring further showed that the blood of an immunized animal had the power both of protecting and of curing susceptible animals which had been inoculated either with the toxins or the bacilli of diphtheria. In preparing the blood-serum it is desirable to have a uniform strength or standard. One-tenth of one cubic centimeter of what Behring calls his normal serum will counteract ten times the minimum of diphtheria poison, fatal for a guinea-pig weighing three hundred grams. One cubic centimeter of this normal serum he calls an antitoxin unit. The serum prepared by his method is labelled in three strengths: No. I. is sixty times the strength of the normal serum; No. II. is one hundred times as strong; No. III. is one hundred and forty times as strong.

Dosage.—To a child of two years or over not less than 2000 to 4000 units should be administered at the first dose; hence solution No. I. is rarely employed at the present day. Should a favorable result not be attained, then, on the following day, a similar dose should be repeated, and a third dose if necessary. Massive doses should be employed at the outset in severe cases, in laryngeal diphtheria, and in advanced cases. The sites to be selected for injection are various. In very young children either the buttock or thigh is to be preferred, while in older children the flanks or subscapular spaces may be chosen as well. The injections should be made deeply into the subcutaneous cellular tissue, and the swelling which results should not be rubbed. It is to be emphasized that the best results are obtained from early injections. In laryngeal cases intubation should be combined with the serum treatment in suitable cases. The early use of antitoxin in this disease has greatly diminished the proportion of cases in which the usual complications, particularly paralysis, occurred previously.

In fortunate cases the influence of the serum soon becomes apparent. Within twenty-four hours the faucial swelling diminishes, the membrane exfoliates, the temperature falls, the pulse becomes slower and stronger, and the general condition of the patient quickly improves. In cases of moderate severity and when injections are employed early the improvement in the throat and the constitutional symptoms is very decided; and the earlier the case comes under treatment the better are the results. There are, however, some cases of great severity in which the antitoxin has been used early, yet has not shown any benefit. Kronig[1] has found that incising the hard, swollen process enhances the efficiency of the diphtheria antitoxin.

A danger in serum-therapy may be the development of local abscesses, which, if full antiseptic precautions be taken, must be rare indeed. I have escaped them altogether. Certain skin eruptions have been observed after injections, mostly urticarial, though sometimes scarlatiniform. The latter form has given rise to apprehensions of scarlatina. Widerhofer had one case which was isolated as measles, but never developed any

[1] *Jour. of the Amer. Med. Assoc.*, August 8, 1908.

symptoms other than the suggestive eruption. Rarely, joint-pains and swellings, with general prostration, supervene. Abnormal sensitiveness to horse-serum is found in certain persons, *e. g.*, coachmen and asthmatics. Two fatal cases have been reported—one [1] that of a healthy boy, five years old, the result of an injection of Behring's fresh serum as a preventive, dying within five minutes; the other occurred in Berlin.[2]

For establishing immunity in subjects exposed to infection the injection of 60 units (1 cubic centimeter of the No. I. serum) affords protection. In order to arrest the development of the disease during the period of incubation 100 units (1 cubic centimeter of No. II. serum) is probably sufficient. Rolleston urges rest in bed from three weeks after a mild angina to seven or eight weeks after a severe attack—as a prophylaxis against palsies. Adrenalin and strychnin have their advocates for the same purpose. Finally, for the serious circulatory disturbances which arise either during or after diphtheria, absolute rest and general management accomplish more than drugs.

SEPTICEMIA.

Definition.—Septicemia is a systemic infection due to a microbic invasion of the blood and tissues, with or without a detectable seat of infection. Sapremia is a toxemia of saprophytic origin (poisoning with chemical products), while *septic* intoxication is an affection due to the absorption of poisons from foci of suppuration. Mixed infections are common, hence it is clinically impossible to differentiate cases of pure sapremia, septic intoxication, and septicemia in most instances.

Pathology.—After death the body putrefies early. The macroscopic changes in the viscera are often wanting. The muscles present a brownish color-tint. The pia mater is generally congested, and, together with the nerve-centers, may be the seat of ecchymoses. The blood is dark ("tar-like"); its coagulability is diminished and, microscopically, it shows micrococci and bacilli. The organisms, however, are not so constantly found in the circulating blood as in the septicemia of mice or rabbits. The spleen is somewhat softened and its lymphoid elements more distinct. Ecchymoses are found in the serous membranes.

In protracted septicemia more marked alterations exist, and among them may be briefly enumerated the following: endocarditis (rarely ulcerative); gastro-intestinal catarrh (of the duodenum and rectum in particular) with punctiform extravasations; enlargement of the lymphatics and spleen, with softening of the latter; cloudy swelling of the liver (rarely the so-called emphysema of the organ due to putrefaction); edema and catarrhal inflammation of the uriniferous tubules; congestion, sometimes associated with edema of the lungs; and inflammation of the pleura, pericardium, and peritoneum, with ecchymoses.

Microscopically, the internal organs show numerous small foci of inflammation, some of which may be the seat of "coagulation-necrosis." Bacteria are found in abundance in various situations, such as the exudations, the capillaries of the inflammatory foci, and renal glomeruli.

Etiology.—Bacteriology.—Septicemia may be due to micrococci,

[1] *Jour. of the Amer. Med. Assoc.*, April 4, 1896. [2] *Med. News*, April 18, 1896.

which Koch has shown to be considerably smaller than pus-cocci, though no one form of bacterium has been found constantly present to the exclusion of all others. Besser, as the result of careful experiments, concludes that septicema is caused solely by streptococci, while Rosenbach and others have found both staphylococci and streptococci. In typhoid fever, pneumonia, gonorrhea, puerperal fever, and the like septicemia may be due to the organism causing the primary lesion. For example, Rosenow found the pneumococcus in 77 of 83 cases of pneumonia in which blood cultures were made. Doubtless in many instances of human septicemia the clinical manifestations are due partly to bacterial poisoning and partly to septic intoxication with the poisons (ptomains) developed by the organisms, and the ptomains probably kill the patient before the bacteria can propagate themselves throughout the system. Laboratory experiments teach us that in the lower animals septicemia can be produced both by chemical poisons and by bacterial infection, and these two types are observed in human beings. With reference to the bacterial form, Warren[1] states: "Whether this process is caused solely by the multiplication of bacteria, or is dependent in part upon the liberation of intensely powerful poisons, or is due to some ferment-like substance capable of reproducing itself, like the poison of the serpent, as are diphtheria and tetanus, much more extensive studies upon the human subject will be necessary to enable us to say."

Modes of Infection and Introduction of the Poison into the System.—(1) **Wounds**, either surgical or the result of injury, with which we have nothing further to do in this work.

(2) **Through the uterus,** following labor, miscarriage, or abortion. Generally in these cases there are accompanying local changes, but in a few the poison appears to pass the unguarded portals of the organ, while the latter exhibits nothing abnormal.

(3) The cases in which the poison gains entrance into the body **without obvious wounds** or raw surfaces are relatively more common. When the skin is quite natural, septic infection or intoxication cannot occur, but the slightest abrasion or cut, bed-sore, etc. may serve as a gate of admission. These slight lesions "may be almost completely healed by the time the severe symptoms of the disease are developed" (Strümpell).

(4) **Mucous membranes** often admit the virus, being less protective in nature than the skin. The numerous bacteria—benign and pathogenic—that are constantly present in the intestinal canal may also find in local lesions (as in typhoid fever, dysentery, etc.), or catarrhal inflammation even, points of lodgement and cause a systemic infection. To this category belongs that form of septic infection which follows gonorrhea. The so-called cases of "spontaneous septicemia" are also usually occasioned by absorption from the mucous surfaces.

Rheumatic or septic manifestations often follow attacks of *tonsillitis*, and it is probable that the tonsils are more frequently points of entrance for the organism than has hitherto been supposed (Wade, Bannatyne).

(5) **"Sepsis Intestinalis."**—This special form of poisoning is caused by canned meats, ice cream, sausages, and cheese. Vaughan, to whom

[1] *Surgical Pathology and Therapeutics*, p. 340.

we are indebted for the first description of "sepsis intestinalis," found in cheese a ptomain which he named *tyrotoxicon*, and which he regarded as the active agent in this group of poisoning cases. The symptoms are due, according to his statement, to poisoning by chemical substances, being instances of *sapremia;* but it may yet be found that the intestinal micro-organisms play a more or less prominent part in the process.

(6) Ogston[1] recognizes as one of the mildest forms of sapremia the sickness and nausea produced by a bad smell, which, he claims, is but a ptomain of putridity that may, under certain contingencies, produce serious symptoms. On the other hand, persons who are habitually exposed to bad odors (workers in sewers, in the dissecting-room, etc.) may acquire a considerable degree of immunity against poisoning of this sort. The fever in these cases corresponds in severity to the dose of the poison.

(7) **Septicemia** may be associated with or follow osteomyelitis.

Clinical History.—(1) **Symptoms of Sapremia.**—The fact that this form may occur without bacterial infection, either local or general, must be emphasized, but more frequently there will be either local infection or putrefactive changes with the production of a grave general condition due to the absorption of the poisonous chemical products. In certain other acute infectious diseases (diphtheria, tetanus, typhoid fever, erysipelas, etc.) the general symptoms are similarly engendered. Perhaps the most typical examples of sapremia seen by the physician are those due to tyrotoxicon and the unaccustomed inhalation of foul odors. At the beginning a chill may occur, but this is more generally wanting. In "*sepsis intestinalis*" marked *local symptoms* may initiate the attack, as nausea, vomiting, colicky pains, diarrhea, etc., and in all forms there is *fever*, the temperature often rising rapidly to 101° or 103° F. (38.3°–39.4° C.) and sometimes higher. *Prostration* and *anemia*, particularly the latter, may be prominent symptoms. *Microscopic examination* of the blood shows leukocytosis, and always a marked reduction in the number of red corpuscles. The hemoglobin, too, may be reduced.

Sapremia following childbirth is a most typical sub-variety, and, apart from the special history, the symptoms are much the same as those detailed above. It is the form most amenable to treatment, the removal of the cause being followed by a rapid disappearance of all symptoms.

(2) **Symptoms of True Septicemia.**—There is an *incubation*-period which is of variable duration, though usually averaging several days. The *onset* is more gradual than in the previous variety, although often marked by a *chill*. Accession of *fever* following surgical procedures, with headache, anorexia, prostration, sometimes vomiting and diarrhea, and especially dulness occasionally amounting to mild stupor, announce the affection: these symptoms should also excite suspicion in the absence of obvious causal factors. They become intensified, and now the attack may closely simulate certain other infectious diseases (typhoid fever, acute miliary tuberculosis, ulcerative endocarditis, etc.), the clinical picture as outlined presenting nothing characteristic. There are, however, more or less distinctive features, which will be considered seriatim.

(*a*) **The Fever.**—This is usually of the *continued* type, and tends to

[1] Warren, *loc. cit.*, p. 342.

increase in degree, fatal cases often terminating in hyperpyrexia. At the beginning the temperature may rise quite rapidly, and in some cases it may even be subnormal. Deep morning remissions may be observed, and the initial chill may be repeated.

(*b*) **The Circulatory System.**—The pulse is frequent, and near the end becomes very weak. In subacute cases characteristic lesions (endocarditis in particular) may develop, but are difficult of recognition, since they do not, as a rule, give rise to audible murmurs or other physical signs. In other instances soft murmurs may be heard, but it is indeed hard to discriminate these from functional sounds. Moderate leukocytosis (principally of polymorphonuclear variety) is observed, and the presence of micrococci in the blood during life has been demonstrated. A more or less severe secondary anemia is present. In toxic states associated with suppuration, *iodophilia* occurs (Cabot).

(*c*) **Gastro-intestinal System.**—The spleen may become perceptibly enlarged and gastro-enteritis is usually present, either in an acute form with vomiting and frequent serous discharges or, more often, merely with a diarrhea of moderate intensity (septic diarrhea).

(*d*) **Cutaneous Symptoms.**—Punctiform hemorrhages into the skin are of prime importance in the diagnosis. Occasionally more extensive ecchymoses appear, scarlatinal eruptions also showing themselves, but these are less characteristic. Among rare appearances, herpes, roseola, edematous inflammations, and faint jaundice may be observed.

(*e*) **Renal Symptoms.**—The lesions consititute the so-called "septic nephritis," the urine often containing a fair amount of albumin, epithelium, tube-casts, and red and white corpuscles.

Diagnosis.—(*a*) **Sapremia** can be distinguished by the history, the immediate appearance of the symptoms, their character, and by the prompt effect of the removal of the cause. The diagnosis requires a careful search for etiologic factors, though without the latter we can sometimes arrive at a correct conclusion by a process of exclusion.

(*b*) **True Septicemia.**—Here the existence of an incubation period, the continued fever, mental apathy, faint jaundice, splenic enlargement, and the characteristics of septic nephritis, all combine to form a well-defined group of phenomena. A careful blood-examination should be made for micrococci, etc., and cultures should be undertaken in spontaneous septicemia and associated forms (*e. g.* septico-pyemia). The surgeon should look to the condition of the wound if one is present.

Course and Prognosis.—The course may be brief, virulent attacks sometimes terminating fatally within forty-eight hours, this being especially true of sapremia when the dose of the poison is large. The gravity of the case in the latter form is in direct proportion to the amount of virus in the system, the outlook being good when the cause is removable, in true septicemia, however, the progression tending steadily to the end. The mildest types may rarely terminate favorably, but the effects are not dependent upon the dose, and the minutest quantity may lead to specific results in their fullest intensity. Septicemia of chronic course is somewhat milder.

Treatment.—Of first importance is the removal of the cause, and small wounds should be excised and the parts freely cauterized. The physician must support the patient's strength by a suitable dietary and by the judicious use of cardiac stimulants; the former should consist

mainly of liquids (milk, egg-white, meat-juice), and the latter of alcoholics, together with strychnin and ammonia. Of medicines, internal antiseptics (mercuric chlorid, creasote, etc.) richly deserve a trial. The fever calls for hydrotherapy. Quinine in large doses (gr. x every four hours) should be given, if well tolerated by the patient. To meet the renal conditions the free use of water, together with the least irritating of the diuretics, is to be advised. A powerful agency in eliminating the microörganisms and their toxic products is found in normal salt solution, which may be administered by hypodermoclysis ("washing the blood"). Not less than from one to several pints of this fluid are to be used daily. In the inoperable cases antistreptococcus serum, although of uncertain value, should be employed. If the blood-pressure is persistently low, adrenalin is valuable when *slowly* administered intravenously in the proportion of five minims of the 1 : 1000 solution to a pint of warm saline solution (Sajous). At first 1000 units of Marmorek's serum should be injected daily, later at longer intervals. Pearce states that polyvalent sera appear to give most favorable results.

PYEMIA.

Definition.—A disease of the blood invariably associated with secondary abscesses, and due to an absorption of pyogenic organisms.

Pathology.—The cadaver does not undergo putrefaction as early as in septicemia. Briefly considered, the pathologic lesions that fall within the physician's province arrange themselves under the following heads:

(1) **Thrombosis and Embolism.**—At first the veins leading to and from the seat of the local changes from which pyemia arises contain thrombi which may soften into a puriform material. Thrombi are also found in the lungs (a circulating embolus first finding lodgment in the pulmonary artery or its branches), liver, kidneys, spleen, and the cortical substance of the brain. *Fresh emboli* may be formed in the circulating blood. Suppurative phlebitis is almost constantly present.

(2) **Abscesses.**—These so-called metastatic abscesses are set up by septic emboli or result from the thrombi (chiefly pulmonary and portal), and are found in the lungs, liver, spleen, and kidneys. They are not large, but may coalesce and form cavities of the size of an apple. An original focus of suppuration may be the bronchial glands. The kidneys are the chief organs of elimination in this disease, and hence it happens that numerous clumps of micrococci (*infarctions*), producing miliary abscesses, are frequently seen in the regions of the Malpighian bodies. There are many other, though rarer, seats of abscesses, as the muscles, submucous and subcutaneous tissues, bones, the parotid gland, ovaries, and testicles.

(3) **Lesions of the Skin and of Mucous and Serous Membranes.**—At the post-mortem examination hemorrhagic extravasations and pustules are often visible in the skin. The mucous membrane of the alimentary tract is rarely affected, differing in this point from septicemia, though occasionally ulcers may be noted, and most commonly in the stomach near the pyloric orifice (in puerperal cases) and in the large bowel. Probably they are always secondary to the submucous miliary abscesses. The

serous membranes (pleura, pericardium, meninges of the brain, synovial membranes) may be the seat of purulent inflammation and of hemorrhagic extravasations.

(4) **Cardiac Lesions.**—Ulcerative endocarditis forms the chief morbid lesion (*vide* Ulcerative Endocarditis). Myocardial foci of suppuration may be present.

Etiology.—Bacteriology.—Experimental investigations have shown conclusively that the organisms usually responsible for this condition are the staphylococcus and the streptococcus. Whether the former or the latter be the agent of infection in the given case depends chiefly upon the condition of the tissues at the starting-point, especially with reference to the character of the local defensive processes; also, though to a lesser extent, the degree of virulence of the micrococci.

Other important pus-producing organisms are the gonococcus, pneumococcus, Friedländer's bacillus, bacillus pyocyaneus, bacterium coli communis, and micrococcus tetragenus. Pyemia may be caused by any wound or inflammation in which the pyogenic organisms are present.

Paths of Infection of the Body.—(*a*) Almost always the entrance is by the *blood-vessels*, the special varieties of bacteria that cause pyemia reaching the veins and producing thrombo-phlebitis. Less frequently they reach the arteries and produce thrombo-arteritis. From the former condition emboli may be disseminated throughout the system, while from the latter the emboli are arrested in the neighboring capillaries to which the tributaries of the vessel lead. Bacteria independently of emboli may be found wandering in the blood-stream.

(*b*) The *lymphatic system*, but here the bacteria meet with greater forces opposing their attempts to spread than in the blood-vessels, and hence it is a much rarer mode of propagation.

(c) In *spontaneous pyemia*, in which there is no wound to act as an infection atrium, we must presuppose the existence of either a trivial lesion, as in "spontaneous septicemia," or an area of lessened resistance. The latter may be produced by inflammation, by a contusion, and in other ways, and all that seems necessary is a lowering of the tone of the general system (Warren). I am certain that ulcerative endocarditis is not frequently the starting point, but is usually secondary to foci of inflammation elsewhere. The appendix is often the primary or original focus in this category of cases, micrococci localizing themselves here in consequence of a preceding disturbance of the circulation or catarrhal inflammation. I recollect one case in which no original abscess was found at the post-mortem.

Predisposing Causes.—(*a*) *Epidemic Influence.*—It has been proved by abundant experience that certain seasons are characterized by epidemic outbreaks of the disease.

(*b*) Cases have sometimes been noticeably more frequent in the early months of the year (February and March) than in other seasons.

(*c*) *Age and Sex.*—Males are more frequently affected than females, and most cases occur about the middle period of life or at the time of greatest danger from traumatism.

Clinical History.—Incubation.—The disease sets in within the first week after the reception of the wound or operation. The wound looks unhealthy, and phlebitis of the efferent veins is noted.

A most conspicuous symptom, and usually the first, is the *chill:* it may, however, be preceded for a variable time by *fever* of a continued or intermittent type. The fever of pyemia is of the suppurative type. Profound *prostration* develops early; the *skin* presents an icteroid appearance; and gastro-intestinal symptoms may appear, but are not prominent. The signs of abscess of the lung, liver, and other organs may develop in some cases, while in others the whole clinical picture is colored by the ill-defined characters of ulcerative endocarditis.

(*a*) **The Chill.**—This may be mild, though oftener it is quite severe. It is repeated throughout the course of the disease at somewhat irregular intervals, and rarely it may recur several times on the same day. Chills are most apt to occur during the daytime.

(*b*) **The Fever.**—A rapid rise of *temperature* accompanies the *chill.* The fever-curve is of the irregularly intermittent or profoundly remittent type, with intervening period, showing slight or marked variations, and as decided deviations may occur within a short space of time, a two-hour record should be kept. The temperature rarely falls to the normal level; it may do so, however, and remain there for one or two days. To explain the peculiarities of the curve in this disease we need only recall the great variety of pathologic processes before noted. With the sharp fall of temperature *sweating* occurs and leaves the patient more or less exhausted, though only temporarily so as a rule.

(*c*) **Respiratory System.**—Symptoms referable to the organs of respiration appear early. The pulmonary abscesses are usually latent, but may give rise to dyspnea, cough, and occasionally a purulent expectoration. *Pain* is present if they are superficially located, and under such circumstances the physical signs of cavity or of pleural effusion may be noted. The signs of *pneumonia* at one or both bases may also develop, the expectoration now becoming rusty.

(*d*) **Splenic and Hepatic Symptoms.**—The foci of suppuration in the liver are difficult of recognition unless they become large as the result of coalescence and are superficially located (see article Hepatic Abscess). Splenic infarction may also be safely diagnosed if there are pain and great tenderness (due to localized peritonitis) in the left hypochondrium, with progressive enlargement of the organ. In one case I detected distinctly crepitant sounds over the site of the spleen during life.

(*e*) **Cardio-vascular Symptoms.**—The *pulse* at first is accelerated, but moderately full and regular; later it becomes feeble, rapid (running), or even uncountable. Frequently cases in which ulcerative endocarditis develops are apparently of spontaneous origin. (*Vide* Endocarditis in the section on Diseases of the Heart.) Among the blood-appearances during life are *leukocytosis* and a rather marked reduction in the red corpuscles, with moderate poikilocytosis. Iodophilia is a frequent finding. Nucleated erythrocytes may be present. The blood-plaques are increased. Purulent pericarditis may occur.

(*f*) **Cutaneous Symptoms.**—The most prominent is a mild yet decided grade of *jaundice*, that is hepatogenous (?) in nature. Sweating has already been alluded to as a symptom, both during and after the febrile paroxysms. The skin finally shrinks from emaciation. *Skin-eruptions* are common, particularly erythema, purpura, pustules, pallor, and the general surface is often decidedly *hyperesthetic.*

(*g*) **Genito-urinary Symptoms.**—The urine is concentrated and urates are copiously deposited. There is *albuminuria*, which may be due to the pathologic changes or may be ascribable to the fever. The microscope discloses the presence of tube-casts, micrococci, pus- and (more rarely) blood-corpuscles. Peptone has been found in the urine.

(*h*) **Nervous Symptoms.**—The mind generally remains unclouded until an advanced stage is reached; then delirium sets in, and is followed by a terminal coma. Metastatic purulent meningitis, with its usual symptoms (hemiplegia, strabismus, ptosis, deafness, etc.), may appear.

(*i*) Symptoms may be presented by the **joints** and **bones**. Metastatic arthritis, usually suppurative, is a not unusual concomitant, and in some cases it is combined with similar involvement of the long bones. An acute osteomyelitis may be the only ascertainable source of the pyemia.

Septico-pyemia.—By this term is meant the combined presence of suppuration and a general intoxication (septicemia). The symptoms of pyemia (recurrent chills, copious sweats, metastatic abscesses, and early nervous symptoms dominate the scene in the majority of the cases. Some of these instances pursue a comparatively mild, chronic course.

Differential Diagnosis.—The disease is often confounded with malarial intermittent fever (*vide* p. 356), *acute miliary tuberculosis*, *malignant endocarditis*, and, more rarely, *typhoid fever*. Malignant endocarditis, the secondary condition, is, however, pyemic in nature. Typhoid fever is distinguishable by the Widal reaction, characteristic eruption, and course. A few points of contrast, by means of which *septicemia* and *pyemia* may be differentiated, are tabulated below:

Pyemia.	Septicemia.
Always associated with suppuration.	Suppuration may be absent, but there may be a sloughing wound.
Multiple chills.	A single chill, as a rule.
Irregularly intermittent fever-curve.	Continued type of curve.
Profuse sweats accompanying febrile attacks.	Absent.
Rapid emaciation and profound prostration.	Less marked.
Sensorium is rarely affected.	Sensorium seldom normal.
Hyperesthesia.	Absent.
Slight jaundice.	Less marked (very faint).
Metastatic abscesses.	Absent.

Prognosis.—Pyemia may kill after an illness lasting but a few days. On the other hand, it may become more or less protracted, so that a chronic form has been distinguished. In this variety the symptoms are milder in character, and recovery may rarely ensue.

Treatment.—So far as the physician's province extends, the treatment is identical with that of septicemia. For the sweating the best agents are aromatic sulphuric acid and atropin; the latter may be given with agaricin (atropin, gr. $\frac{1}{120}$—0.0005; agaricin, gr. $\frac{1}{8}$ to $\frac{1}{4}$—0.008 to 0.016), at bedtime. Prompt surgical interference must be resorted to not only with a view to asepsis of the primary wound, but also to evacuating the primary and all secondary foci of suppuration. Recently puerperal pyemia has been successfully treated in this way. The use of antistreptococcic serum has led to immediate improvement. The employment of normal saline solution by the method of hypodermoclysis aids in eliminating the peccant material.

ACUTE ARTICULAR RHEUMATISM.

(*Rheumatic Fever.*)

Definition.—An acute febrile disease, probably due to a diplococcus. It would seem to be a general infection, the commonest seat of the principal lesion being the joints, "but also involving the heart, both endocardium, pericardium, and muscle, occasionally the meninges and other structures" (Webster). Hueter first advanced the germ-theory to account for the disease, and, although the specific causal agent has not as yet been discovered, this view is the only one that offers a satisfactory explanation for the production of the lesions, the acute onset, the clinical course, and the complications of the disease. The frequent involvement of the joints in many diseases belonging to the acute infections may properly be regarded as supporting this theory. Strümpel points out the fact that in Leipsic, where articular rheumatism is one of the most frequent of acute diseases, at certain times there are only a few cases, while at others there is a striking increase in the number.

Pathology.—The disease does not show peculiar lesions, and, although the joints are the chief seats of invasion, still in many instances and even in aggravated cases, the changes presented are slight or altogether wanting. Usually the synovial membranes of the affected joints are injected and swollen, and their surfaces may be more or less coated with fibrin. The effusion, which in a majority of instances has been found sterile, is mainly serous, but contains fibrin and often leukocytes, and occupies the joints. A similar exudate infiltrates the periarticular tissues. The tendinous sheaths may also be inflamed; the cartilages in protracted cases may become eroded; and rarely a purulent exudate may be seen.

Fatal cases, except when death is due to hyperpyrexia, usually show the changes peculiar to endocarditis, pericarditis, or myocarditis, and less frequently those of pneumonia, meningitis, or pleuritis. The fibrin-factors of the blood are augmented.

Etiology.—**Bacteriology.**—Guttmann, Collin, and Sahli have found the staphylococcus in the articular exudate of patients suffering from complicated or recurrent cases of acute articular rheumatism, and Sahli is inclined to include the disease in the group caused by this organism. Netter, however, has found the streptococcus, and Lang a peculiar bacillus. Singer has examined 92 cases bacteriologically and discovered staphylococci or streptococci in the majority of these cases, and also post-mortem; they probably complicate acute rheumatism. Pierre Achalme[1] has described an organism which he found in the blood (*Thiroloix Achalmii*) of cases of rheumatism with cerebral complications. It is a bacillus somewhat resembling that of anthrax; it is readily stained with anilin dyes and by Gram's method, and is anaërobic. When inoculated into guinea-pigs, it causes inflammation of the serous membranes characteristic of rheumatism. Treboulet and Coyon[2] suggest that Achalme's bacillus is associated with only the severe forms of rheumatism, and they have found it in some other cases associated with a *diplococcus*, which latter was found in all other cases of rheumatism examined. The

[1] *Ann. Pasteur Inst.*, Nov. 1897. [2] *Bull. de la Soc. méd. des Hôp.*, Dec. 24, 1897.

diplococcus was facultative, anaërobic, and stained by Gram's method. The weight of authority, however, is in favor of the view that a diplococcus (*Diplococcus rheumaticus*) is the principal specific cause (*vide supra*). Streptococci of all bacteria have been most frequently found (Park) occurring as diplococci or short chains. The definitive cause of acute articular rheumatism, however, is still *sub judice*. In certain cases reported there was evidence of direct contagion.

Predisposing Causes.—(1) An *infective lesion* (septic wounds, attacks of angina, etc.) that has preceded for some time the appearance of the pain and articular manifestations may often be found, and this may be conceived to form a portal of entry for micro-organisms (Sacaze). The frequency with which an attack of tonsillitis precedes the development of acute articular rheumatism almost indicates a pathologic relation between the two diseases (Cheadle, Wade, Gerhardt, Packard). (2) *Seasons.*—The months of February, March, and April furnish the largest percentage of cases, though the disease is also quite prevalent in the remaining cold months; on the other hand, the disease may sometimes prevail in summer. Edlefsen and Newsholme have shown that the incidence is greatest when the ground-water is low. (3) "Catching cold" was formerly classed among exciting causes, but abrupt changes of temperature merely predispose to the disease. (4) *Climate.*—Rheumatism is most prevalent in temperate latitudes, being rare both in the cold and tropical zones. It is essentially an urban disease (Poynton). (5) *Occupation* is of importance, especially if it entail oft-repeated or prolonged exposure to the influence of wet and cold or to severe changes of temperature. Hence those who follow certain vocations are attacked with great frequency—*e. g.*, coachmen, laborers, sailors, and servant-girls. (6) *Age.*—Primary attacks are most common from fifteen to thirty-five years of age. Out of 655 cases, 80 per cent. occurred between the twentieth and fortieth years (Whipham). Cases are also rather numerous between ten and fifteen years, and I have met with 4 under the former age. Sucklings rarely suffer. (7) *Sex.*—Acute articular rheumatism is somewhat more common in men than in women, possibly owing to the fact that the former sex more often follows predisposing occupations. (8) *Hereditary influence* plays a causative rôle. (9) Conditions of ill health, particularly digestive and hepatic disturbances, seem to exert a slight effect. Preceding or accompanying the attack an infectious sore throat was noticed in 53 of 288 cases. (10) *Chronic endocarditis* renders its victims very prone to attacks of acute articular rheumatism, and some contend that the two diseases are etiologically one and the same. (11) *Choreic children* often develop rheumatism. Batton analyzed 115 cases; he found that within three years 11.3 per cent. of the children developed the disease, and after five years this total was increased to 20 per cent. (12) *Endemic and Epidemic Influence.*—In certain localities the disease is endemic, and epidemic incidence has been noted by McClymont, Newsholme, and others. House epidemics have also been observed.

An attack of acute articular rheumatism is not protective in character, but increases susceptibility. Of 288 cases, 45 per cent. of the patients had had one or more previous attacks (Thüs). In this respect the disease resembles certain other infectious diseases (pneumonia, erysipelas, etc.).

Clinical History.—Of the **incubation-period** nothing is known,

though *prodromata*, both local and general, may be observed. There may be malaise, slight fever, angina, laryngitis, etc., and last from a few hours to a day or two. The *invasion* is usually abrupt, with fever and synovitis, affecting one or oftener several joints, and a chill or a series of chilly sensations may accompany or precede the rise of temperature. The involved joints are tender, often red and swollen, and exhibit the local signs of a rapidly developed inflammation. *Pain* is a most prominent symptom. The medium-sized or larger *joints* (knee, ankle, and wrist) are first involved, and especially those of the inferior extremities; next the shoulder-, elbow-, and hip-joints; and lastly the fingers, toes, and intervertebral articulations. Quite unusual articulations may become implicated (*vide infra*). One of the chief peculiarities of the disease is in the fact that the joints that are affected are not all the seat of anatomic changes simultaneously, but that the process migrates from one joint to another from day to day, and often crosses from one side of the body to the other. Sometimes this occurs at longer intervals. Hence the number of joints involved at one and the same time may be either few or many.

In cases of average severity the general features are subordinate to the local symptoms. The fever is usually moderate, the temperature not exceeding 103° F. (39.4° C.), and the temperature-curve is of the irregularly remittent type, corresponding in severity with the joint-symptoms. Defervescence is by *lysis*. The skin is bathed in a copious perspiration which is not dependent upon a previous fall of temperature. Nervous symptoms are rarely observed.

The *general course* of the disease exhibits wide variations, both as to duration and intensity of symptoms, especially in children. It may not outlast several days, appearing with mild symptoms; on the other hand, cases sometimes persist for six to eight weeks. The latter instances are apt to show brief non-febrile periods, alternated with marked paroxysms, and similar cycles may be repeated. When the symptoms are distinct from the start the course may be briefer than when the features are of mild character. As will be seen hereafter, the disease frequently manifests complications, especially cardiac.

Leading Symptoms and Complications in Detail.—(1) **Joints and Surrounding Structures.**—As I have stated, pain is much complained of, and is greatly augmented by motion and by pressure of any sort. It may be out of all proportion to the degree of the anatomic changes. The joints affected are generally swollen (most markedly in the knees), and the swelling is due partly to effusion into the joint and partly to inflammatory edema of the periarticular structures. The sheaths of the tendons, the bursæ, and often the adjacent muscles and fasciæ exhibit inflammatory changes; hence it is usual to see an extension of the swelling for a variable distance from the joint, the backs of the hands often showing this to a marked extent. The skin may present a pink or rose-colored blush over circumscribed areas or taking the form of streaks.

In even *mild cases* there are usually two, three, or more joints involved, though it often happens that one bears the brunt of the disease, little complaint being made of others less severely implicated. Hence it should be a golden rule to examine carefully all the joints at each visit. Involvement of a single articulation (*monarticular rheumatism*) does sometimes occur, but the diagnosis of these cases offers great diffi-

culties. On the other hand, an existing polyarticular rheumatism may become centered in a single joint and there linger with great obstinacy.

In *severe cases* numerous joints may be invaded, with an involvement of the joints of the symphyses, of the jaw, of the ribs, and the sterno-clavicular articulations. Under these circumstances the patient assumes a dorsal decubitus, and seeks to relieve his excruciating pain by holding his limbs in a semiflexed position and absolutely motionless. If now an attempt be made to change his posture, he complains piteously of darting pains in the affected joints. The *fugacity* of rheumatic arthritis has already been alluded to.

The inflammation, however intense, may quickly subside in one joint, while at the same time an acute disturbance appears in another. Usually resolution is complete, no trace being left of former inflammation, though the disease may recur in the joints primarily involved. Suppurative arthritis may supervene, though rarely, and its occurrence points indisputably to mixed infection. This complication may lead to ankylosis—a sequela which does not belong to pure rheumatism.

(2) **The Cardio-vascular Symptoms.**—The *pulse* is quickened to 100 beats per minute or over, but is soft and full, and when complications arise it shows special characteristics which are described in appropriate sections of this work. In rare instances it is very rapid, feeble, and irregular, apart from cardiac involvement. The results of a careful blood-count show a high grade of *symptomatic anemia*, which may develop with marvellous suddenness. Moderate *leukocytosis* is also present. Diplococci have been found in the blood in severe cases.

Great importance attaches to the *cardiac affections* that so frequently accompany this disease. They may arise in any case, even the mildest, or at any stage of the disease, and hence the conscientious physician cannot afford to neglect the matter of closely and regularly examining the heart. It must be recollected that no special symptoms announce the development of cardiac disease. At first we may note an increase in the febrile movement, more or less palpitation, sometimes dyspnea, and precordial pains, which often do not amount to more than a sense of soreness. There may also be attacks of angina pectoris of apparently purely nervous origin (Strümpell).

(*a*) The most frequent cardiac manifestation is *acute endocarditis*, which is present in 25 to 30 per cent. of the cases. We are, however, sadly in need of reliable statistics upon this point. It usually takes the form of simple (verrucose) endocarditis, and affects most frequently the mitral valves. But, though usually indicated by an apical systolic murmur, it is hard indeed to eliminate the functional murmurs that may also develop in the course of this disease. Unless combined with the symptoms detailed above, the presence of a blowing systolic murmur does not afford trustworthy evidence of the existence of acute endocarditis. I have witnessed two instances in which endocarditis preceded the arthritic manifestations. Church and Cheadle[1] state that "in a large majority of cases, if no endocardial murmur is present during the first ten days of an attack, the endocardium escapes." While it rarely endangers life and may leave no trace, in the majority of instances the acute

[1] *Allbutt's System of Medicine*, vol. iv., p. 15.

endocarditis does not undergo complete resolution, but leads to sclerotic changes and terminates in incurable chronic valvular disease.

(*b*) Next in the order of frequency is *pericarditis*, which may or may not be combined with the former. In many cases the effusion consists of organizable lymph (often large in amount); less commonly it is serofibrinous and rarely becomes purulent or blood-stained. It is distinguished by its pathognomonic friction-sound, though also by other characteristic signs (*vide* Pericarditis).

(*c*) *Myocarditis* is often present to a slight extent in rheumatic endocarditis and pericarditis when these occur independently of each other, but to a more marked degree when endo-pericarditis exists. Hence it is less common than either endocarditis or pericarditis. The changes and symptoms occasioned will be discussed under Myocarditis. Here it should be pointed out that the condition weakens the cardiac walls and leads to dilatation of the ventricles (usually the left). Lees holds that dilatation of the left ventricle (greater or less) is always present and one of the earliest symptoms.

If we consider rheumatism an infectious malady, we can readily understand why the local manifestations should appear not only at the different articulations, but also in the cardiac structures and other viscera.

(3) **The Skin.**—Rheumatism produces copious *perspiration*. The sweat emits a sour odor and gives at first an acid reaction, though later it may be neutral, and rarely alkaline. The temperature-curve in most cases is not materially influenced by the sweats. Occasionally the drops in temperature and the free sweats are concurrent, but the latter symptom is apt to persist despite the oscillations in the temperature. *Sudamina* appear, often in extensive crops. Among other *skin eruptions* less frequently observed are forms of erythema (especially *E. nodosum*) and urticaria, which latter may be associated with purpura (*urticaria hæmorrhagica*). The association of the latter condition with polyarthritis is known as *peliosis rheumatica*, though, according to some writers, this is not rheumatic in nature. Cutaneous ecchymoses, and even extensive hemorrhages into the skin and from the mucous membranes—a general hemorrhagic diathesis—may also be encountered.

Subcutaneous Rheumatic Nodules.—In 1881, Barlow and Warner called attention to the fact that during and after acute articular rheumatism, particularly in children and young adults, small subcutaneous nodosities attached to the tendons and fasciæ may in exceptional instances be observed. These small nodules are rather firm, movable, and usually painless. The skin over them is simply elevated, with no traces of inflammatory action. They are most frequently found at certain points of election (fingers, wrists, edge of the patella, malleoli, and over the back of the elbow), though also seen less frequently elsewhere; they may disappear and after a brief interval reappear. On *microscopic examination* it is seen that round and spindle-shaped cells enter into their composition. Riess believes them to be of embolic origin. I met with one fatal case of the sort which occurred in a male aged forty-two years, in which acute articular rheumatism was also complicated with endo-pericarditis and pneumonia. Most of the nodosities were of the size of a bitter almond. Cheadle considers that the eruption of large nodules signifies persistent and uncontrollable cardiac disease.

(4) **The Fever.**—The fact that the fever fluctuates materially in this affection has already been noted. It remains to be pointed out that if suppuration occur as a complication, the fever may be of the hectic variety; also that rarely hyperpyrexia is suddenly developed, and with it marked cerebral symptoms (restlessness, delirium, and sometimes convulsions, finally merging into stupor) are, as a rule, though not necessarily, associated. This serious condition commonly develops about the beginning of the second week. In my case cited above, it began on the sixth day. Delirium usually comes on either shortly before or after the acute development of the *hyperpyrexia*. The pulse becomes excessively rapid and feeble and physical prostration extreme. The temperature may rise rapidly with slight interruptions until it touches 108° or 109° F. (42.7° C.), and as the fever reaches its maximum death usually ensues. The temperature may continue to rise after death. The cause of "hyperpyretic rheumatism" is not definitely known. It has been claimed that the intemperate are most apt to be attacked, but this belief is not corroborated by many clinicians. In a case of my own, however, in which pericarditis with hyperpyrexia occurred, the patient was an "alcoholic." The symptoms are probably due to an intense concentration of the poison upon the nerve-, and especially upon the thermal, centers.

(5) **The Muscular and Nervous Symptoms.**—It has been stated that the adjacent muscles and fasciæ may exhibit inflammatory changes. They may also show more or less swelling, and are often very tender to the touch, while in long-continued cases muscular atrophy ensues. The cause of this change is not clear, but the most likely view is that it results not so much from disuse of the muscles (the old theory), as from some trophic disturbance due either to the arthritis, or peripheral neuritis, or, to some extent at least, from extension of the rheumatic inflammation from the nearest articulation.

Mention has been made of the grave *nervous symptoms* that are attendant upon hyperpyrexia, but, independently of the latter condition, nervous phenomena may be present. There may be restlessness and sleeplessness (due to pain), but active delirium is exceptional in uncomplicated cases, and it is usually associated with a temperature of 104° F. (40° C.) or higher. In adynamic types, which are rare, low muttering delirium merging into stupor, and even coma, may be observed. Active mental symptoms are sometimes due to cerebral embolism secondary to acute endocarditis. When pericarditis is a complication, wild delirium, with or without hyperpyrexia, or the low muttering variety with stupor, is not unusual. The drunkard may develop *delirium tremens*. *Coma*, leading quickly to a fatal result, may develop without other previous or associated nervous symptoms, and DaCosta has reported cases in which a fatal coma was probably due to uremia. Rarely coma develops during the period of convalescence. *Convulsions* may be noted generally preceding the coma, though rarely as an independent symptom. *Melancholia* may arise in the course of the disease, but more frequently at its close. *Meningitis* must be numbered among the rarest of complications.

Chorea is a not infrequent sequel of this disease in children, and more rarely is associated with it. Of 554 cases analyzed by Osler, in only 88 were chorea and rheumatism associated. These instances may or may not be accompanied by acute endocarditis.

(6) **Pulmonary Symptoms.**—*Pleurisy* occurs, and is generally excited by, an extension of inflammation from the pericardium and pleura. The inflammatory process may be propagated through the diaphragm to the peritoneum. *Bronchitis* is sometimes present, but is rarely a part of the rheumatic morbid process; it is secondary, and is often occasioned by the co-operation of the factors that are at work in every disease in which enforced recumbency and great prostration coexist. *Broncho-pneumonia* may be produced. *Lobar pneumonia* rarely occurs, and is confined to aggravated cases, but pulmonary congestion is occasionally seen, and may prove fatal. Pulmonary complications also develop secondary to pericarditis, and especially to endo-pericarditis.

(7) **The Renal Symptoms.**—The *urine* is diminished in amount, is high-colored, and of high acidity and density. The standing specimen deposits urates. As in other infectious diseases, there is commonly present a slight febrile albuminuria, but acute nephritis is extremely rare. The chlorids are sometimes diminished, but rarely absent.

(8) **The spleen** is slightly enlarged in some cases. The *saliva* often has an acid reaction. (9) Inflammation of the *parotid gland* (rheumatic) was met with in 3 cases by Courtois-Suffit and Beaufume.

Clinical Peculiarities of Acute Articular Rheumatism in Children.—The arthritic symptoms in children are in abeyance, while endocarditis and pericarditis are predominant, and these cardiac conditions may appear before the joint-lesions are observed. Endocarditis follows the joint-lesions twice as frequently in children as in adults. Parsons lays stress upon reduplication of the cardiac second sound, audible at the apex only, as an indication of the development of endocarditis. This sign is to be distinguished from reduplication heard at the base—*e. g.*, in Bright's disease. Acid sweats are slight in children. Rheumatic tonsillitis is common, and may precede, accompany, or follow attacks of rheumatism in children. Erythema is a frequent concomitant, and is often mistaken for scarlatina. The febrile movement is brief and hyperpyrexia less frequent than in the adult. The nervous features are more marked, notably chorea, which Poynton[1] has emphasized as a symptom of rheumatism. Out of 217 cases of chorea, he found obvious heart disease in 122. Bareno[2] reports an instance in a newborn infant.

Diagnosis.—The acute development as a primary affection of polyarthritis with fever, early tonsillitis, sudden anemia, moderate leukocytosis, and fresh cardiac murmurs, is a symptom-complex on which an assured diagnosis can be usually based.

Differential Diagnosis.—*Pyemia* must be carefully separated. In pyemia, however, the general condition is graver, fever precedes the local manifestations, and the fever-curve is irregularly intermitting. Rigors also occur in pyemia at varying intervals, accompanied by a steep elevation of temperature—symptoms that are absent in rheumatism. Again, suppurative processes in the various viscera and skin and slight jaundice appear in pyemia. Rheumatic symptoms fluctuate greatly, while the pyemic do not.

The multiple swelling of the joints which develops after childbirth is to be regarded as *septic* in nature. In these cases arthritis leads rapidly to suppuration, with more or less destruction of the joints. *Gout* will

[1] *British Journal of Children's Diseases*, Feb., ix., No. 98.
[2] *Archives of Pediatrics*, Jan., 1902, p. 27.

be distinguished from rheumatism in connection with the consideration of the former disease (*vide* p. 432).

Monarticular rheumatism is with difficulty differentiated from a group of affections which simulate it closely. (1) The so-called *gonorrheal rheumatism* often affects a single joint, especially the knee; but in this disease there is usually a definite history of recent infection, and the local features (pain, swelling, etc.), unlike true rheumatism, are far more pronounced than the general. The course of gonorrheal arthritis is longer in duration, and is generaliy connected only with a single joint from the start; while acute articular rheumatism almost always begins as a polyarthritis, with subsequent fixation in one articulation. Cardiac complications are rare in the former disease.

(2) *Acute osteomyelitis* is generally single, and is sometimes mistaken for rheumatism, from which it differs, however, in the localization of the lesions in a single joint from the start, the greater prominence of the local symptoms, and in the implication of the epiphyses and the shaft of the affected bone rather than the joint, and in the graver general symptoms from the time of onset.

(3) There is a liability to mistake the *acute arthritis* of infants for rheumatism. This attacks by preference the hip or knee, and is purulent inflammation due to pyemia (Townsend), hence having no relation to the disease under consideration.

(4) *Tubercular arthritis*, particularly in children, has been confounded with rheumatic monarthritis. The former is less indurating, the swelling presented is less symmetric, the pain is greatest in the joint itself, and the course is far less acute than that of the latter.

(5) In the course of the *hemorrhagic diseases*, scurvy, purpura, and hemophilia, effusion into the joints, either hemorrhagic or serous in nature, occurs with great frequency and is associated with rheumatic pains. The differential diagnosis rests upon the tendency to hemorrhage, and in scurvy on the lesions of the gums. The absence of fever is usually decisive; unfortunately, it may be present in these joint-affections.

(6) *Glanders*, at the onset, may be mistaken for rheumatism.

Prognosis.—Recovery is the general rule. As in other infectious diseases, the chief danger springs from the great intensity of the type of infection, as manifested in hyperpyrexia with grave nervous symptoms, the development of the general hemorrhagic diathesis, etc.—happily rare occurrences in this disease. Certain complications, such as pericarditis, endopericarditis (especially common in childhood), pneumonia, etc., may render rheumatism grave or even hopeless, and rarely the endocarditis that complicates the disease is of the ulcerative variety and leads to fatal pyemia. Pulmonary embolism may occur and cause death.

The influence of personal factors may impede recovery, such as intemperate habits, great obesity, the existence of previous organic disease of the heart, or Bright's disease, etc.

Treatment.—(1) **Sanitary Environment, Diet, and Stimulants.**—The sick room should be well ventilated, and its temperature maintained at 65° to 70° F. (18.3°–21° C.), but draughts should be avoided. The patient should be lightly dressed in flannels and covered with a sheet of the same material. The *diet* should be liquid and nourishing, milk being the best food-article. Farinaceous matter, milk and Seltzer water,

buttermilk, egg-white, may be employed if milk cannot be taken in adequate amount. I begin the use of easily digested proteins soon after defervescence has occurred, but have immediate recourse to the earlier liquid or soft diet upon the return of pain and fever. An ordinary dietary is to be gradually resumed. *Stimulants* may be employed if indications for their use are present. The prompt treatment of tonsillitis among children and young adults is important prophylactically.

(2) **Internal Therapeutics.**—The bowels should be opened early with calomel, followed by salines. There has been of late a surprising unanimity among clinicians in commending the use of the salicylates in the treatment of this disease—more so than at any previous time since their introduction. They are employed in most of the larger hospitals, both in Europe and America. Differences, however, relating to the mode of administration and the particular salt to be selected still exist. Wood[1] favors ammonium salicylate, for the reasons that it is freely soluble, is rapidly absorbed, and when given in sufficient amount quickly produces the symptoms that mark salicylic action, while, in addition, it is less depressing than the other salts of salicylic acid. It is best given in milk and is usually well borne. My experience with this salt in acute articular rheumatism, though as yet somewhat limited, has been satisfactory. Until the present time sodium salicylate has met with more general favor than any other single salt of salicylic acid. The pure acid is also used, though not to any great extent at the present day. The amount given in twenty-four hours should not exceed 2 drams (8.0), while often 1½ drams (6.0) of the sodium or ammonium salicylate is sufficient. My method is to give gr. x (0.648) every two hours during the first day, or until the pain or other local features have largely disappeared; then the remedy is given at longer intervals, but not omitted entirely. If it be given in solution with an excess of alkali, it is least irritating to the gastric mucosa. In this manner fresh exacerbations are most probably averted. If the latter occur, however, larger doses must be instituted, so as to cut them short. Some recommend that the medicine be stopped as soon as the pain has been controlled. The hypodermic method of giving salicylates will prove of advantage in those patients who cannot take them by mouth. Lassere recommends methyl salicylate to relieve the pain. Some prefer salol to either the pure acid or the salicylates; in my experience, however, the use of this drug has not been followed by good results. Doubtless the reason for this lies in the fact that salicylic acid can neither be introduced into the system in sufficient amount nor rapidly enough in the form of salol.

Kinnicutt has recommended the employment of the oil of wintergreen, a salicylic compound which does not generally produce the unpleasant toxic symptoms so apt to be excited by the salicylates or salicylic acid. The dose is ♏x–xx (0.60–1.25), given in capsules or in milk, to be repeated every two hours. Salicin (gr. x—0.648, every hour, increased to gr. xv—0.972) is sometimes efficacious and invariably agrees; it is to be preferred to the salicylates in weakly individuals. Salophen, in daily doses of 1 dram (gr. xv—0.972, every four hours), may be substituted for sodium salicylate if the latter produces gastric disturbances after a few days' treatment; it is almost specific in its effects. Salophen

[1] *University Medical Magazine*, Jan., 1895.

passes through the stomach unchanged, to split into salicylic acid and acetylparalidophenol in the intestines. Sodium salicylate enemata (ʒj—4.0—of the salicylate and ♏x—0.60—of the tincture of opium in each injection) may be of advantage in certain cases. The remedy is slowly absorbed from the rectal mucosa.

The treatment with the salicylates or salicylic acid mitigates the fever, relieves the pain, and shortens the stay in bed by a few days, but does not curtail convalescence. The statistics of Williams go to show that the salicylic treatment also tends to protect against the development of cardiac complications, though it does not seem to influence the course of the complications once they are established. In my experience the alkaline treatment operates to obviate the occurrence of the heart-complications and shortens the period of convalescence, but exerts slight, if any, influence upon the fever-curve and pain. These facts led me long since to use, in addition to salicylates, an alkaline remedy, such as sodium bicarbonate, potassium citrate, etc., in sufficient doses to render, and then maintain, the urine of slightly alkaline reaction.

There are a few other remedies that should be referred to, and, although more or less serviceable, they are without specific influence. Among these is antipyrin, but safer and equally efficacious remedies have replaced this drug. Potassium iodid and the preparations of colchicum belong to this category; their effects are most beneficial in cases that drag on after the acute stage is over. Good results have been reported from the use of aspirin (gr. vij–xv—0.466–0.972, thrice daily) in both acute and chronic rheumatism. Stengel has noted improvement from the use of antistreptococcic serum in three cases of protracted recurring rheumatism. Menzer has successfully treated a number of cases with a serum made from streptococci of human origin. It is also indicated in cases showing a pyemic temperature (Chipman). Wolverton advises a mixed streptococcus and staphylococcus (*aureus* and *albus*) vaccine.

(3) **Local Measures.**—These occupy a subordinate place in the management of acute articular rheumatism. Their number is legion, but only a few of the more valuable can be adduced here. In mild cases the affected joints should be wrapped in cotton batting or in flannel. If the pain is severe despite the use of the salicylates internally, fomentations as hot as can be borne or hot cloths lightly wrung out of Fuller's lotion (sodium carbonate, ʒvj—24.0; laudanum, ℥j—30.0; glycerin, ℥ij—60.0; and water, ℥ix—270.0) are beneficial. As salicylic acid is absorbed through the skin, it may be used in the following formula:

℞. Acid. salicyl.,
Lanolini, *āā.* ʒiij (11.65);
Ol. terebinthinæ, ʒiij (11.25);
Adipis, ʒiij (11.65).
M. et ft. ung.
Sig. Rub over the affected joints and follow by wrapping in cotton.

Methyl salicylate, by local application, is of service. It is put on the skin over the affected joints drop by drop, and the joint then enveloped in gutta-percha tissue and a flannel bandage applied to it. Cold compresses and the ice-bag to the joints have been strongly ad-

vised, particularly by German authors. The affected joints should be kept at perfect rest, and this is best accomplished either by padded splints and a roller bandage or plaster casts. Blisters near the joints involved and the light application of the Paquelin thermo-cautery are sometimes serviceable. Taylor[1] has successfully employed currents of hot air, applied by means of an instrument (electro-thermogen).

The treatment of the *complications* will be considered under their appropriate headings. Should, however, hyperpyrexia occur during the progress of the affection, it is to be relieved by cold affusions, since large doses of internal antipyretics are of themselves dangerous. It may also be stated that the cardiac complications—endocarditis, pericarditis, and endo-pericarditis—rarely require special remedies. If marked cardiac asthenia appears, as indicated by the feeble first sound, the salicylates may be replaced by salicin, which is less depressing in its effect upon the heart. Cardiac stimulants may be required. A copious pericardial effusion calls for paracentesis (*vide* Sero-fibrinous Pericarditis). Gürich[2] succeeded in curing 98 out of 125 patients with articular rheumatism by tonsillectomy.

During convalescence the patient should not be allowed to get out of bed too early. My own rule has been to keep him in bed for a week after the temperature has returned to the normal and after the pain has disappeared, except it be during the hot season. These precautions are taken to avoid the occurrence of relapses. After the patient goes into the open air he should be told to avoid cold, and wet in particular. During this period iron is to be employed until the blood-examination fails to show anything abnormal. For the stiffness and swelling that sometimes persist, or disappear very slowly after the acute attack, massage and the application of hot water or warm baths seem to yield the best results.

SUBACUTE ARTICULAR RHEUMATISM.

THIS is, as a rule, a sequela of acute rheumatism, and may occur, though rarely, in persons who have not had a previous acute attack. Both the local and general features are of a mild type, but the course is apt to be prolonged into two, three, or more months. Usually the local symptoms are confined to one or two of the larger joints, with little swelling or redness, and the pain is slight except on movement. The temperature rarely exceeds 101° F. (38.3° C.), and at times may be practically normal. Though the course is prolonged, the joints usually return to their normal state; occasionally, however, the disease becomes chronic. As in the acute form, so in the subacute, anemia becomes well marked and cardiac complications are not uncommon, particularly when the disease occurs in children.

The **treatment** embraces, in addition to the usual antirheumatics, the use of iron, quinin, cod-liver oil, and, when practicable, a change to a warm climate. The affected joints demand hot applications and massage.

[1] *Lancet*, Nov. 26, 1898. [2] *Münchener med. Wochen.*, Feb. 8, 1910, lvii., No. 6.

GONORRHEAL ARTHRITIS.

Definition.—A septic synovitis caused by the gonococcus. It has no connection with true rheumatism. It usually manifests itself toward the close of an attack of gonorrhea, but it may develop during the active stage of the disease or at any period during the course of gleet.

Pathology.—The signs of ordinary synovitis are generally found in the affected joints, though, not rarely, the inflammatory process is periarticular (*gonorrheal tenosynovitis*). In these cases the inflammation may travel along the sheaths of the tendons for a considerable distance. Synovial effusion may occur, and rarely may be purulent, this being most frequent in gonorrheal inflammation affecting the wrist and hand. Gonococci have been found in the effusion, and it is now thought by many writers (Finger, Councilman, and others) that the gonococcus may be the only infective agent concerned in the morbid process. Others contend that the metastatic inflammation of the joints is due to the presence of pyogenic cocci. The disease is present in 2 per cent. of all cases of gonorrhea in males, and rarely occurs in females (Gaither); it may follow any urethral discharge or may be associated with menstruation or leukorrhea. C. Lucas has collected 23 cases of gonorrheal rheumatism in infantile subjects of ophthalmia.

Clinical Symptoms.—Two leading varieties, acute and chronic, are encountered. (1) *Acute Gonorrheal Arthritis.*—This may be very mild, amounting merely to slight fugitive pains about one or more joints, without swelling or redness (*arthralgic form*). The typical, acute form, however, presents the symptoms of a severe fibrinous or sero-fibrinous inflammation of a single joint, developing quickly. The pain is often violent; there is swelling of the joint with extension along the course of the tendons, and the condition is obstinate. Unless pus be present (a rare event) the constitutional features do not correspond in severity with the local. There are many instances in which the complaint begins as a *polyarthritis*, with subsequent concentration upon one or two of the larger articulations, especially the knees or ankles. Fibrinous ankylosis usually remains as the resulting condition. In infants, however, this condition is transitory, as a rule.

Complications and Gonorrheal Septicemia and Pyemia.—*Acute endocarditis* may be of gonorrheal origin, and undoubted instances are common. In the inflammatory products of this condition Hering has found the gonococci, as has also Councilman, in the heart-muscles (gonorrheal myocarditis). Rarely, gonorrheal endocarditis assumes the ulcerative or malignant form. As the result of invasion of the blood by the gonococci, *suppurative arthritis* may develop and form a part of *gonorrheal septicemia*. Instances of severe, rapidly fatal general infection in gonorrhea are probably always associated with foci of suppuration in the urinary tract (Osler). Among the widespread complications, *embolic*, *septic pneumonia*, and *iritis*, deserve special mention.

(2) *Chronic Gonorrheal Arthritis.*—This occurs (*a*) as a serous effusion (*hydrarthrosis*), and (*b*) as a chronic inflammation of the articular and periarticular structures (synovial membranes, bursæ, periosteum, and tendons with their sheaths). The former is usually monarticular, settling with especial frequency in the knees, and may be wholly painless. The latter is more or less painful—causes dense swelling of the

joint, and frequently of the structures for some little distance above and below the latter. Both forms lead to great restriction of motion. The os calcis may be the seat of gonorrheal periosteal inflammation with or without exostosis. It is sometimes called the painful heel of gonorrhea.

The **diagnosis** cannot be determined apart from the history of urethral infection, or the detection of the gonococci in the blood or the joint-effusion. For diagnostic purposes, Irons uses a "gonococci" of killed bacteria, suspended in glycerin, making inoculations after the method of von Pirquet, with a control. A papule is formed in a few hours, with surrounding hyperemia, disappearing by the third day. The reaction is classed as positive when over 5 mm. in diameter. The acute form is distinguished from *acute articular rheumatism* by the more intense pain, the extent to which the peri-articular tissues are involved, and the negative character of the general symptoms. The chronic variety must be discriminated from *chronic synovitis* due to other causes, and this often proves a difficult task.

Treatment.—I have never seen the slightest benefit from internal medication in gonorrheal arthritis, except possibly from the use of mercury. J. C. Wilson[1] has obtained excellent results from massive doses ℥x–1x—0.60–3.75 t. d.) of the syrup of iodid of iron. There is much evidence at hand to show that chronic gonorrheal arthritis is favorably influenced by the injection of gonococcus vaccine. Fuller[2] reports good results from seminal vesiculotomy.

Local measures, however, are of paramount importance. Absolute rest to the part is indicated, and the limb should be placed upon a splint; then after making an appropriate anodyne application (ungt. ichthyol. or ungt. belladonnæ), it should be bandaged as firmly as possible. In other instances complete immobilization in plaster-of-Paris dressing gives good results. In acute cases the patient should be anesthetized, and after the procedure, if pain be great, a hypodermic injection of morphin may be given. Dry heat, either sand-bags or the heating apparatus (oven), is useful. In *chronic forms* the aim should be to remove the effusion and swelling, and to restore the natural motility. For the latter two indications massage and passive movements are best. Hydrarthrosis may also be diminished by the use of the thermo-cautery, at intervals, and blisters. Willard urges removal of the infecting microorganism by arthrotomy and free irrigation with antibacillary fluids.

VARIOLA.

(*Small-pox.*)

Definition.—Variola is an acute contagious disease, characterized by its sudden onset and severe period of invasion, followed by a remission of the fever and an eruption of papules, which pass through the stages of vesicle, pustule, and scab. The stage of pustulation is accompanied by secondary fever. Variola runs a variable course, but on the whole has become milder far in character in recent years.

Historic Note.—Smallpox has existed from the earliest anti-

[1] Jacobi's *Festschrift*, 1900.
[2] *Medical Record*, New York, June 15, 1912.

quity in India, Africa, China, and other Eastern countries. During the thirteenth century (1241) it entered England, in the early part of the fourteenth Ireland, and in the latter part of the fifteenth Germany. In 1507 it was imported to America, and first appeared in the West Indies; a little later (1520) the Spanish troops conveyed the disease to Mexico, where it destroyed not less than three and a half millions of people. It was brought to the United States from Europe in 1649, and gained it first foothold in Boston, whence it progressed at intervals in a westerly direction to the western coast-line. During the Spanish-Cuban war the disease was transferred from Cuba to the Southern States, afterward spreading to many of the Northern and Western States. In the United States, for the years 1903 and 1904, there occurred 42,590 cases, with 1,642 deaths, and 25,106 cases with 1,118 deaths respectively (Wyman). Variola exhibits great variability in intensity in different epidemics.

Pathology.—The eruption of small-pox consists in an inflammatory cellular infiltration of the *rete mucosum*, and has four successive stages—(1) *Papular*, (2) *Vesicular*, (3) *Pustular*, (4) *Scab*.

(1) *The Papule.*—At first there is a hyperemia of the papillæ of the skin appearing as small red spots. These soon become round, discrete patches that may be rolled like shot under the skin, and then become elevated, owing to the increase in the cells in the *rete mucosum*.

(2) The *vesicle* appears at the apex of the papule, and results from a circumscribed elevation of the superficial layer of the epidermis in consequence of the mechanical pressure exerted by the fluid exudate, which is excited by peripheral inflammation. The vesicle is not unicellular, but is loculated (fibrinous reticuli), and contains serum, leukocytes, fibrin-filaments, etc. If a section of a vesicle be made early through the deeper layers of the *rete mucosum*, an area of coagulation necrosis is observed, due to the presence of micrococci (Weigert). The vesicle shows central umbilication, which corresponds with the necrotic area.

(3) The *pustule* is formed by the filling of the reticuli with leukocytes. Cellular infiltration and swelling of the true skin beneath the pustule occur, as a rule, as the result of diapedesis. Moreover, suppuration may involve the *cutis vera*, and as a consequence scarring results. In hemorrhagic small-pox the reticuli are occupied by an abundance of red corpuscles which have passed in from the adjacent blood-vessels, and may infiltrate the upper as well as the deeper layers of the epidermis surrounding the vesicles or pustules. The pustules may dry up, but commonly rupture, and in either case the result is (4) *scabbing*.

Recently Councilman, McGrath and Brinckerhoff have described the specific lesion as a focal degeneration of the stratified epithelium, accompanied by serous exudation and the formation of a reticulum.

The eruption has run an atypical and even abortive course in the cases occurring in recent years. An early maturation of the papules has been observed; in many cases they became solid, conical elevations with a small vesicle at the summit. W. M. Welch[1] states that the lesions seemed to involve only the outer epidermis.

The mucosa of the mouth, pharynx, and, rarely, the esophagus and the rectum may be the seat of a variolus eruption, and the plaques of Peyer may be somewhat swollen. The eruption also appears in the

[1] *Phila. Med. Jour.*, Nov. 18, 1899.

larynx, the trachea, bronchi, conjunctivæ and nasal mucosa, where ulcers rather than true pustules are seen.

Hemorrhagic small-pox presents extravasations occurring in the serous and mucous membranes, the connective tissue, the parenchyma of the various viscera, and also, though much less frequently, in the nerve-sheaths, bone-marrow, blood-vessel walls, and the muscles. In this form the spleen is firm (Ponfick, Osler), and the liver is sometimes enlarged and the subject of fatty degeneration. Hemorrhagic infarction of the lung occurred in 5 out of 7 cases examined by Osler.

Secondary Lesions.—The catarrhal inflammation of the larynx may extend in depth till it touches the perichondrium of the cartilages (perichondritis), and a croupous exudate in the larynx may often coexist with edema. Lesions are present in the lungs, some of them frequently (general bronchitis, bronchopneumonia), and others rarely (hypostatic congestion, lobar pneumonia), and *pleuritis* may be observed. Cloudy swelling, diffuse inflammation, and sometimes fatty degeneration of the *liver* have been noted; the *spleen* is enlarged and pulpy as a rule. The *heart* may show myocardial alterations—chiefly parenchymatous and fatty—and rarely endocarditis and pericarditis occur. The *kidneys* show cloudy swelling, an acute degeneration of the epithelium, more marked than in other infections, occurs. "An acute glomerulo-nephritis was found in 5 cases out of 54" (Councilman). Weigert found that at the commencement of the stage of suppuration the microscope revealed "small-pox cylindric masses" in the various viscera (coagulation necrosis).

Etiology.—Bacteriology.—The recent investigations of Councilman and his associates have resulted in the discovery of a protozoon in the epithelial cells of the lesions. There are two cycles of development, intracellular and intranuclear, the latter only occurring in small-pox. Transmission of these organisms by the dried epithelial scales may be responsible for the spread of the disease. These findings have been confirmed by Calkins and Howard and Perkins of Cleveland. M. Funck[1] found protozoa (probably the same organisms previously described by Pfeiffer) in all vaccinia pustules examined. They are usually from 1μ to 3μ in diameter, and larger cyst-like bodies filled with spores also occur. Iskigami[2] has also discovered protozoön-like bodies in the epithelial scales of the vaccine pustules, lymph, etc. Haushalter and Étienne[3] consider the hemorrhagic symptoms in small-pox due to secondary infection with the streptococcus, since they have found this organism in the blood of those dead of hemorrhagic variola. Widal and Sabrazes have also noted the streptococcus in autopsies upon small-pox cases.

Predisposing Causes.—The *receptivity* for variola is wellnigh universal, and among the few who have enjoyed immunity were three distinguished physicians—Diemerbroeck, Boerhaave, and Morgagni. It may be said that one attack confers permanent immunity, but exceptionally a second or even a third may occur. *Vaccination*, also, if successful, affords future protection against variola, but to this rule exceptions are not infrequent.

Age.—All periods of life are liable to the disease, but the very young are affected in a relatively larger proportion than older persons. During the entire puerperal stage there is an increased liability to the disease.

[1] *Deutsch. Med. Woch.*, Feb. 23, 1901.
[2] *Jour. Amer. Med. Assoc.*, Dec. 6, 1902; cited from Sei-l. Kwai (Tokio) xxi.
[3] Saunders' *Year-Book* for 1899.

It rarely affects the fetus in utero, and most babes even, who are exposed to the virus at the time of birth, will not take the disease if immediately and successfully vaccinated.

Sex is without influence.

Season.—In temperate climates, most cases occur during the winter months. On the other hand, in tropical countries it is said that the worst cases occur during the hottest months.

Race.—Among uncivilized peoples variola spreads with frightful rapidity, the negro and other very dark races being affected in larger numbers and more severely than whites, since they are not so generally vaccinated. A dread of the infection predisposes to its occurrence.

The Contagion; where Found; Modes of Conveyance and of Infection.—One case of variola is *prima facie* evidence of the existence of another, the poison having been transferred. The specific poison exists in the blood and in the secretions and excretions (most probably), but mainly in the pustules and dry scabs and in exhalations from the lungs and skin. The contagion is conveyed principally from the sick to the healthy by the dust-like particles of the dried scabs.

Modes of Infection.—(*a*) *Inoculation* with either the blood or the contents of the eruption or the dissolved dry scabs is followed by variola. (*b*) Contact with, or proximity to, a patient suffering from small-pox is very apt to convey the poison, with resulting variola in the person thus exposed. To what distance the contagion can be conveyed through the air is not known, but it is probably considerable; and all authors are agreed that it is one of the most infective diseases with which we are acquainted. It is contagious from the earliest active stage to the end of convalescence, and possibly even during the stage of incubation. (*c*) Transmission by *fomites* is common, the poison adhering to clothes, body- or bed-linen, etc., and evidence is not wanting to show that the poison is highly tenacious of pathogenic power. Its vitality is retained after death, and the room occupied by a patient, the bedding, and the articles of furniture all serve to convey the disease unless thorough disinfection be enforced. The *infection atrium* for the poison into the system is probably the *respiratory tract.*

Clinical History.—Incubation.—This stage varies with the mode of communication of the poison. If following inoculation, the symptoms appear in six or seven days; when originating from infection, usually in twelve days, though this stage may be either lengthened by a day or two or shortened to an equal extent. During a portion of this period complaint may be made of certain ill-defined symptoms, but these are usually absent. **Invasion** is sudden and accompanied by characteristic signs. These are—a *severe rigor, high fever, headache, and intense lumbar pains.* Instead of the usual severe rigor, repeated chills, extending over twelve to twenty-four hours, may occur. The symptoms of the onset have been milder in the recent outbreaks, although similar in character to the severer types of former epidemics. During the preliminary fever the *respirations* are accelerated, the *pulse* becoming decidedly more rapid, and there may be generalized bronchitis. The *tongue* is coated and slight pharyngitis may exist. There are anorexia, general vomiting, and constipation or rarely diarrhea. Restlessness, delirium, and stupor are the principal nervous symptoms observed. Infective *albuminuria* is common. In the female menstruation is apt to occur.

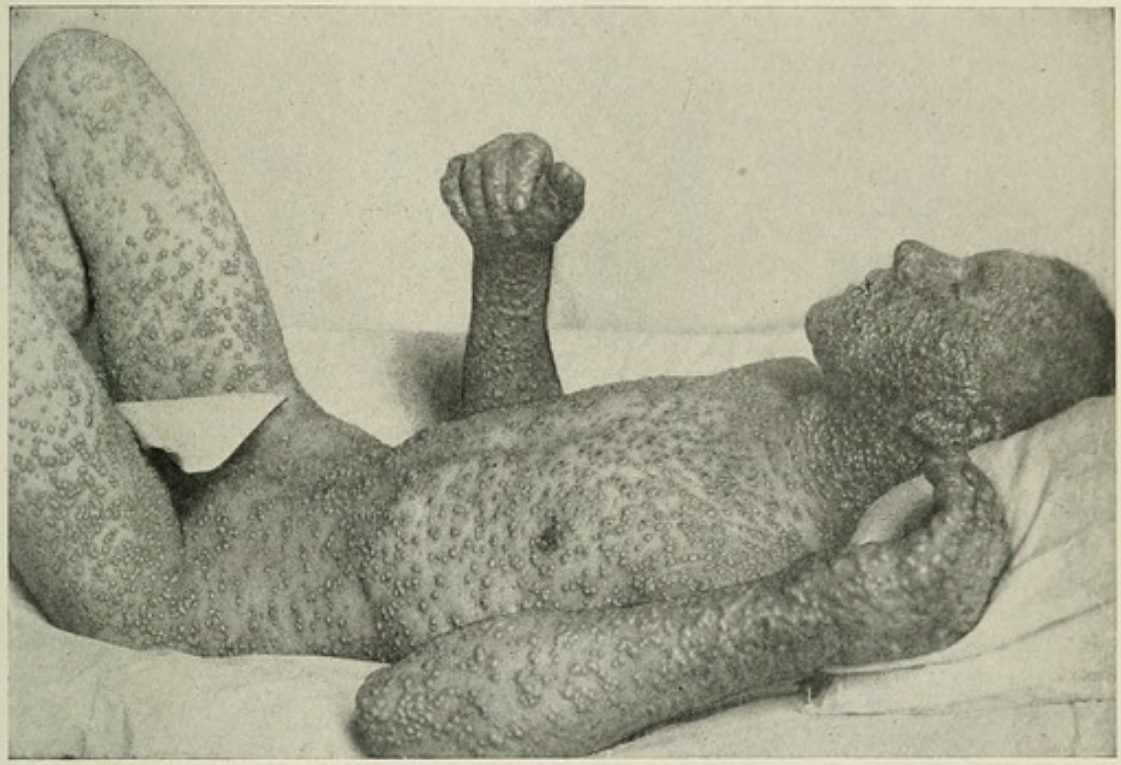
The **physical signs** referable to the lungs are few, and consist of a few dry and, later, moist râles, heard on *auscultation*. *Palpation* detects splenic enlargement. From the second day the so-called *initial rashes* may appear: (*a*) the *diffuse scarlatinous eruption*, which in no way differs from ordinary *scarlatina;* (*b*) the *measly eruption*, which may be diffuse and present a striking similarity to that of measles. Either associated with these or occurring independently there may be a hemorrhagic eruption (usually purpura), the petechiæ coming out by natural selection, mainly upon the hypogastric region or the inner surfaces of the thighs and in the axillæ (Simon). The initial rashes occur in a considerable proportion of cases (10–15 per cent.). The *stage of invasion* lasts three days as a rule. The temperature then declines rapidly, while at the same time the true *variolous eruption* appears upon the skin and mucous surfaces. It develops first upon the face, particularly upon the forehead and the hairy scalp, and spreads in a downward direction till it reaches the legs, where it last appears. The skin in the femoral triangle rarely shows the true variolous eruption. Each pock passes through the various stages noted in the pathologic description—viz., papule, vesicle, pustule, and scab; and when the stage of pustulation has been reached a secondary fever develops. During the following remission of fever the headache, lumbar pains, etc., subside. The fever of suppuration which then succeeds is accompanied once more by marked constitutional disturbances, particularly nervous derangements (wild delirium, etc.), and at this time complications are also apt to develop. On the eighth or ninth day of the eruption (the twelfth or thirteenth day of the disease) the pustules begin to dry up, forming yellow crusts; the redness and swelling of the skin subside; and two or three days later the scabs loosen and are thrown off. During this stage the fever again declines in company with the constitutional symptoms, and convalescence ensues. As previously stated, when suppuration involves the true skin scars are the inevitable result. The hair drops off sometimes, even to the extent of total alopecia, but is generally renewed.

The general *course* described above is that of an average case, but the cases met with in the recent epidemic appearances in numerous sections of this country have been unusually mild, and also manifested strange features; and to these the terms "modified" and "mitigated" small-pox may be appropriately applied. Although not all cases were equally mild, many of them were practically ambulatory, since they would not remain in bed after the eruption appeared, and this was also true of those unprotected by previous vaccination. There was little or no secondary fever and desiccation was rapid. Says W. M. Welch,[1] whose experience is unparalleled: "I must say I have never seen cases present, uniformly, so mild a type as during the present year (1899), nor have I been able to find in the vast amount of literature published on the subject any account of a similarly mild epidemic in this or any other country." During the last two years the disease has resumed a more typical course, both as to symptomatology and severity of type.

Leading Symptoms and Complications.—(*a*) **Eruption.**—The eruption in the more typical cases appears at the end of the third or on the fourth day, coming out first upon the forehead, particularly along the border of the hairy scalp, and spreading in a downward direction in

[1] *Loc. cit.*

PLATE I.



VARIOLA (Royer).

regular progression. It appears in the form of slightly elevated maculæ, which are at first of a pale red color, and later assume a darker red hue, resembling small fleabites. These increase in size during the next forty-eight hours, at the end of which period they are developed into (1) *papules*. The change of character is accompanied by intense itching and burning of the skin-surface. To the feel they are papular, like shot under the skin. The eruption is always most abundant upon the face and scalp, while the hands and fingers are the next most favored seats. At the end of the third day (the sixth day of the disease) the conical apices of the papules contain liquid, forming thus (2) *vesicles*. The latter increase in size till the entire papule is converted, at the same time acquiring more and more decidedly a central umbilication. Puncturing a vesicle does cause it to collapse, but allows only a small portion of its liquid contents to escape, owing to its reticulated character. As the vesicle increases in size its contents become opaque, and in three days more, or about the sixth of the eruption, the vesicles become (3) *pustules*. Umbilication now disappears, and the pustule looks full and well rounded, and is surrounded by a red border or "halo." If the pocks be close set, as on the face, wrists, and fingers, the intervening skin is inflamed and swollen and the itching and burning become almost intolerable. The pustules may coalesce along their edges, and thus the

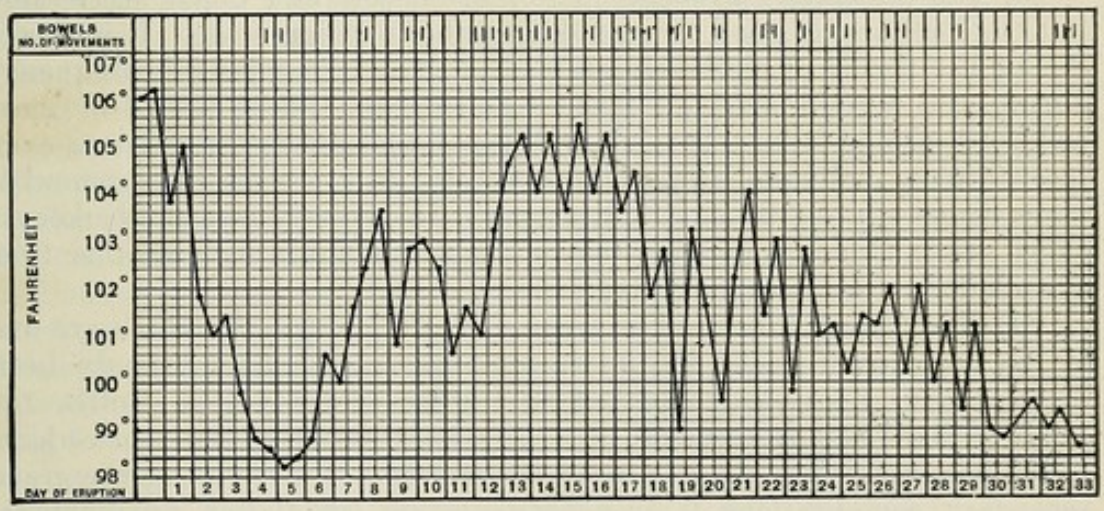


FIG. 16.—Temperature-chart of a case of variola, from a patient in the Municipal Hospital, Philadelphia. A. F——, aged three years; not vaccinated.

eruption becomes confluent. The eyes are closed as the result of swelling and tumefaction of the face, and the hands and feet assume a rounded, ball-like appearance. The face, as a whole, is markedly misshapen and is ultimately disfigured. When the pus is not liberated (a comparatively rare event), its desiccation begins on the ninth day (the twelfth day of the affection); if the pustule is ruptured earlier (as when confluence occurs, it begins at an earlier day. (4) The *scabs* now form, and remain until about the twelfth day of the eruption, and when pits or scars result they gradually fade until they remain as permanent whitish spots.

The eruption upon the mucous membrane develops simultaneously with that of the skin, and among favorite surfaces for its appearance are the mouth, tongue, soft palate, and pharynx (causing *dysphagia*), the nasal chambers (causing *coryza*), the larynx (causing *hoarse-*

ness), the trachea and bronchi (causing *bronchitis*). This mucous efflorescence does not proceed to the development of pustules, but forms ordinary ulcers as a consequence of early maceration of the superficial layers of the mucosa, and these ulcers also may become confluent.

The *skin* presents certain complications that are always secondary and are deserving of mention (erysipelas, abscess, gangrene, bed-sores).

(*b*) **The Fever.**—The temperature at the onset rises rapidly, and may touch 103° or 104° F. (40° C.) on the first day, its range being high and of the continued type during the invasion period. Evening temperature of 105° F. (40.5° C.) or higher may be observed, and in three days (or with the first appearance of the papules), the temperature remits, but does not intermit in true variola. It remains at a low elevation till the stage of suppuration is reached, when a fresh rise occurs. This secondary fever-curve is apt to show exaggerated points of elevation and deep remissions. The latter are generally the result of septic absorption (the fever of suppuration). Secondary fever, since the variolous infection has grown milder in type, is often slight or may be wanting (*vide supra*). This period lasts from one to three or four days. When desiccation of the pustules begins, defervescence also commences, and proceeds in a gradual manner by lysis. There may be a post-variolous rise, and if so, its presence is to be attributed to some sequel or other.

(*c*) **The Circulatory System.**—The *pulse* is soft and much accelerated (100 to 130) and of good volume during the invasion stage. It is slower during the period of remission, only to be greatly increased in frequency during the second stage of fever. During the latter period it may, owing to cardiac failure, become very rapid, and finally irregular or even intermittent. Simple *endocarditis* rarely, and *pericarditis* somewhat more commonly, occur as complications. In typical cases the leukocyte curve shows two exacerbations—one about the eighth day, another from the twelfth to the fourteenth day.

(*d*) **Respiratory Tract.**—The *laryngitis* and *pharyngitis* which are due to the presence of pocks in the respiratory mucosa have already been mentioned. *Laryngeal perichondritis* with edema of the glottis, the latter perhaps being the result of a direct extension of the pock-ulcers to the perichondrium, may arise; it is ominous. Chief among the grave secondary complications is *bronchopneumonia* (inhalation pneumonia); and *lobar pneumonia* also occurs, though rarely. *Pleurisy* is not infrequent, particularly as an associated condition in bronchopneumonia.

(*e*) **The Digestive System.**—The variolous efflorescence in the buccal and pharyngeal mucosæ may be an agency in predisposing to a secondary inflammation in adjacent organs—*e. g.*, suppurative otitis media, suppurative parotitis, pseudo-diphtheria, etc. *Palpation* almost always shows an enlarged spleen, and not infrequently an enlarged liver. The vomiting which is usual at the onset is due to a catarrhal condition of the stomach. Constipation is common, but diarrhea is also sometimes met with, being excited by a catarrh of the large intestine, and is especially common in children. The pocks may be found in the rectum and they sometimes excite dysenteric symptoms.

(*f*) **The Nervous Symptoms.**—The chief of these have been already pointed out. *Violent delirium* (previously alluded to) may be followed by fatal *coma*, and in children *convulsions* may be seen. Very rarely paraplegia has been observed during the attack, though it is more com-

mon during the convalescence, and is then due to different causes, such as peripheral neuritis and disseminated myelitis (Westphal). *Multiple neuritis* may be a sequel or the pharyngeal nerve may alone be affected, Among other conditions rarely arising during convalescence are insanity. epilepsy, aphasia, and hemiplegia.

(*g*) **The joints** may be swollen and painful after small-pox, and in rare cases periostitis may be observed.

(*h*) **Renal Symptoms.**—Welch and Schamberg[1] made analyses of the urine in 128 cases of variola, and found the presence of albumin in 65 per cent. and tube-casts in 45 per cent. ; they believe that the albuminuria in most cases is the expression of a structural change due to the small-pox poison. The clinical symptoms of variolous nephritis are mild as a rule. Hemorrhagic nephritis may occur, but it is rare.

(*i*) **The Special Senses.**—The pustules may form upon the conjunctivæ and eyelids, and several important conditions result from this variolous involvement of the eye—viz., conjunctivitis, keratitis, choroiditis, and panophthalmitis. Hebra met with ocular complications in 1 per cent. of 5000 cases of small-pox. Otitis media has already been mentioned.

Special Clinical Forms.—There are two unusual types of variola that are important in being severer than the moderate (discrete) form.

(*a*) **The Confluent Form.**—This is the result of an abnormally severe infection, and is less common than formerly. The *ushering-in symptoms* are very severe, and the eruption may appear as early as, or even before, the third day, when the temperature remits. The separate papules are vastly more abundant and close-set ; and after the stage of pustule is reached the face and hands present an uninterrupted area of suppuration. The deformity of the countenace is correspondingly pronounced. Naturally the *local symptoms* are intense and the fever and its concomitants are in exact proportion. The *nervous symptoms* often predominate. Salivation is frequent. The eruption may also entirely cover the mucous surfaces. The *lymphatics* of the neck may be greatly swollen—a circumstance that contributes to the facial disfigurement. The various complications previously adduced are of comparatively frequent occurrence, and following these a general pyemic process may develop. When death occurs it is usually preceded by the *typhoid state* (typhomania, tremors, a rapid, feeble pulse, dry, brown tongue, and diarrhea). On the other hand, if recovery ensues, it is tardy.

(*b*) **Black Small-pox.**—In this form the blood is much changed, so that hemorrhages into the skin, mucous membranes, and various viscera occur. It is important to distinguish several sub-varieties, as follows: (1) A *benign* form, in which blood is infused into the pustules when patients are allowed to leave their bed too early in convalescence. Here the condition is due to the effect of gravitation, and hence is confined almost solely to the lower extremities. (2) Doubtless the ordinary variolous eruption may become *slightly hemorrhagic* without aggravating the constitutional condition. (3) A *dangerous hemorrhagic* tendency may be manifested. During any of the eruptive stages—papular, vesicular, or pustular—hemorrhages may occur into the eruption, and, moreover, free bleedings may take place from the various mucous surfaces. The initial symptoms are usually intense, the eruption abundant, and in consequence of the hemor-

[1] *Phila. Med. Jour.*, Dec., 1902.

rhages collapse often occurs. The most serious complications, pneumonia, diphtheria, and nephritis (followed by uremia), are also apt to develop and terminate life. This and the subsequent sub-variety are truly anomalous. (4) A not uncommon form is met with in which the *acute hemorrhagic diathesis* develops during the period of invasion. Its onset is characterized by the usual symptoms intensified, and as early as the second day ecchymotic patches appear upon the skin surface and grow rapidly by peripheral extension, the mucous surfaces also showing more or less extensive ecchymoses. The variolous eruption rarely appears, though occasionally shot-like papules may be detected. The temperature may be slightly elevated, but is rarely high. Death often occurs before the time for the appearance of the characteristic eruption.

There are also varieties of small-pox that pursue an abnormally mild course. Of these (*c*) **varioloid** deserves first place. By this term is usually meant small-pox occurring in individuals who have been protected by a successful vaccination, but it may also be the result of natural insusceptibility. Hence variola and varioloid are one and the same affection. The *initial symptoms* of varioloid do not differ either in character or severity from those of true variola, but the general course of the attack is peculiarly prone to manifest irregularities. In the pre-eruptive stage an erythematous rash is very common, and its appearance is regarded by many as being of value in discriminating varioloid from variola.

When the *regular eruption* appears, the fever falls to normal and remains there. The rash comes out by the end of the first or on the second day, the papules being scanty, but may appear first upon the trunk, not the face. They are identical with the papules of variola, as is true also of the vesicles; but pustulation rarely develops, since resolution takes place, but, as a rule, before the latter stage is reached.

The *secondary fever* is either very slight or entirely wanting. The mucous surfaces are affected only slightly. Papules and vesicles may be found in close proximity; not so in variola. Desiccation begins between the fifth and seventh days of the eruption (the eighth and tenth of the disease), and hence, as compared with variola, the course is cut short and serious complications almost never occur. There has been noted the same marked tendency to extreme mildness of phenomena that characterize variola in the recent epidemics.

(*d*) An **abortive** form is occasionally observed. It is characterized the intensity of the invasion symptoms, but these subside, and the patient enters at once upon a stage of speedy recovery.

An exceedingly mild type may arise during seasons of epidemic prevalence of the disease, either with or without a scanty and undeveloped eruption; the diagnosis is made from the etiologic circumstances.

Diagnosis.—With a clear history and the presence of the characteristic features a positive diagnosis is a simple problem. But at any period before the papules are fully developed it may be confounded with certain other acute infections, notably cerebro-spinal meningitis, typhus fever, scarlatina, and measles. After the variolous eruption makes its appearance the disease may be confounded with impetigo contagiosa, pustular syphiloderm, and varicella. Councilman[1] advocates two methods to decide the diagnosis: one is by corneal inoculation on the rabbit, the other direct microscopic examination of the suspected lesion.

[1] Osler's *Modern Medicine*, vol. ii, page 295.

Differential Diagnosis.—In *typhus fever* the onset is very like that of small-pox. The former may, however, be distinguished by its peculiar etiologic factors, especially its origin by importation or its non-prevalence in the vicinity; the appearance of the eruption, first upon the trunk (chest and abdomen) in the form of maculæ, and later becoming petechial. Moreover, in typhus the temperature does not remit with the appearance of the eruption.

From hemorrhagic small-pox *typhus* is sometimes distinguished with great difficulty. In the former death often occurs before the eruptive stage is reached. In typhus shot-like papules are never detected, whereas they are sometimes found in hemorrhagic small-pox.

Hemorrhagic small-pox may be simulated by *cerebro-spinal meningitis*. If the history be not clear, lumbar puncture will settle the doubt.

Scarlatina may early be distinguished from the erythematous (scarlatinous) rash which often precedes the appearance of the variolous eruption; this is, as a rule, neither so intense nor so uniformly distributed over the skin-surface of the body as in true scarlatina. *Hemorrhagic scarlatina* may readily be confused with black small-pox.

The *macular stage* of the eruption may be confounded with *measles*. The absence of the characteristic prodromes and symptoms of invasion belonging to the latter disease, the redness and swelling of the conjunctivæ, the photophobia and marked coryza, the stubborn cough, and increased fever after the eruption appears, make the separation easy, as a rule. After the maculæ develop into hard, shot-like, conical papules the scales are turned in favor of variola.

Impetigo contagiosa presents no initial stage; it begins as vesicopustules (not papules) which appear "on the normal skin and are superficial and enlarge by peripheral extension, often attaining the size of a 10-cent piece and having a flat appearance" (W. M. Welch). The patient may infect new areas by scratching. Scars do not result.

Syphilis distinguishes itself by a milder initial stage, by the indurated base of the pustule, by the appearance in crops of the skin-lesions, and by the polymorphous character of the latter. There is neither umbilication nor characteristic pitting after the scabs fall, but a coppery hue.

To differentiate certain mild cases of discrete small-pox (in the non-vaccinated) and varioloid from *varicella* is difficult. In the table below, however, will be found contrasted points of distinction:

Variola.	Varicella.
History.	
Absence of previous attack.	Same.
Previous or existing case in the vicinity.	Traceable to previous or present case of varicella.
Not successfully vaccinated.	Negative.
Occurs at any age.	More commonly in childhood.
Characteristic pre-eruptive stage—rash on the third day.	Eruption not preceded by prodromes; develops more rapidly.
Sacral pain, high fever, and vomiting common.	Quite uncommon.
Eruption.	
Appears first upon the forehead, extending downward.	Appears first over parts covered by clothing. No regular procession over the body.

Variola.	Varicella.
Eruption.	
Vesicles uniform in size, umbilicated, and deeper seated.	Vary in size, sharply elevated, rarely umbilicated, and feel soft and velvety.
Eruption contains serum, later pus.	Only serum, giving pearly translucency.
Most abundant on face and fingers.	Most abundant upon back and lower extremities.
Various stages of eruption observed at points removed from each other.	Various stages side by side.
Pin-prick does not cause collapse of vesicles, being multilocular.	Does cause collapse, being unilocular.
Secondary fever usually present.	Absent.

Park found that monkeys are susceptible to inoculation with small-pox virus, whereas that taken from cases of varicella produced no result.

Prognosis.—The prognosis depends upon (*a*) the degree of severity of the type, the severer forms (confluent and certain of the hemorrhagic) being grave. The hemorrhagic variety, in which large ecchymoses suddenly develop, is almost invariably fatal, and often before the cases have advanced to the eruptive stage. The aggregate number of pocks that appear and the gravity of the infection are, as a rule, proportionate.

(*b*) The prognosis is modified by *individual peculiarities* (age, intemperance). Thus it is more fatal in the very young than in older subjects, more fatal in the intemperate than in the temperate, and so on.

(*c*) Complications increase the death-rate considerably. Of these, bronchopneumonia, lobar pneumonia, acute nephritis with uremia, septico-pyemic conditions, pseudo-diphtheritic angina, and pericarditis are most potent for evil. Among the foremost serious symptoms may be mentioned excessive vomiting, wild delirium, coma, a temperature of 106° F. (41.1° C.) or over, urgent diarrhea, and dysentery.

The death-rate has been computed to be between 15 and 30 per cent., varying, however, with each epidemic. Welch's statistics from the Municipal Hospital, Philadelphia, are as follows: In 2831 cases of variola, 54.18 per cent., while in 2169 cases of varioloid only 1.29 per cent. died. During the recent widespread prevalence of the disease in the United States the mortality rate was unprecedentedly low. Welch and Schamberg found the death-rate in unvaccinated persons 49.45 per cent. in the blacks and 44 per cent. in the whites.

Treatment.—The varied indications in the treatment of small-pox will be considered separately:

(1) **Prophylaxis.**—The rules that have been laid down elsewhere (*vide* Treatment of Typhoid Fever) for disinfection in infectious diseases must be rigidly enforced in this affection. Quarantine (*public and private*) must be secured if the deadly progress of small-pox is to be averted. Absolute isolation cannot be carried out successfully in private houses, and in view of this fact special, well-equipped hospitals should be provided for the reception of the disease. It is important also to remember that persons who have been afflicted with the disease cannot with safety to others resume their former places, either in the family or in society at large before they are completely convalescent. The best means of prevention, however, is vaccination (*vide* p. 200).

(2) **General Management.**—The room occupied by the patient should

be large and freely ventilated (an essential matter, though strong drafts are to be avoided), and all carpets, curtains, and articles of furniture not absolutely needful should be removed.

The *diet* should receive careful attention, and should be varied according to the stage of the affection. During the initial stage it must be restricted to liquid nourishment (milk, animal broths, etc.), and in addition cooling drinks, including ice, lemonade, and other of the various fruit-juices (diluted). During the stage of remission we may add soups, jellies, eggs, toast, and with the onset of the stage of suppuration a supportive diet, reinforced by the judicious use of stimulants, is an essential part of the treatment. Light forms of nourishment must now be given in definite quantities and intervals.

(3) **The fever and associated symptoms** during the invasion stage are best controlled by the cold or gradually cooled baths, which possess all the advantages in this disease that they command in typhoid fever. Cold sponge-baths, the ice-cap, or the cold pack may be resorted to if cold immersion baths are not accessible to the patient. The internal antipyretics must be given with a sparing hand, if at all, and only as antiseptic agents, on account of their depressing effects.

The therapy of this stage also embraces the treatment of certain symptoms. The vomiting may be incessant and exhausting, and chipped ice, champagne, dilute hydrocyanic acid, and cocain hydrochlorate should be tried in the order mentioned. If diarrhea be severe it should be checked (though neither wholly nor suddenly) by the use of arsenite of copper, the acetate of lead (gr. ij—0.1296) and opium (ext., gr. $\frac{1}{4}$—0.0162), in combination, or by bismuth salicylate (gr. v—0.324) and β-naphtol (gr. iij—0.1944). The nervous symptoms are usually restrained by the cold affusions, but occasionally a wild delirium may necessitate a combination of sodium bromid (gr. x–xv—0.648–0.972) with the deodorized tincture of opium (♏v—0.333), given every two or three hours. Very often the wise administration of stimulants removes all necessity for the use of further means of overcoming the nervous symptoms. The catheter must be used if retention of urine should occur. For the intense pains that belong to this stage no other remedy can be compared with morphin sulphate (gr. $\frac{1}{8}$ to $\frac{1}{4}$—0.008 to 0.016), to be administered hypodermically, and repeated if necessary.

(4) As previously stated, the **eruption** appears with the termination of the initial febrile period, and deserves the closest attention. The indications are twofold: (*a*) to limit the eruption as far as is possible, and (*b*) to modify its course, so that extensive suppuration and consequent disfigurement may be prevented. Ablutions with lukewarm water, to which may be added some antiseptic (carbolic acid and glycerin, or, better, a mercuric-chlorid solution—1 : 5000 or 1 : 10,000) will be found of great use. To prevent pitting many local applications have been used. Formerly, a common mode of treatment was to open the pustules as early as possible and touch them with silver nitrate—either in the solid stick or brushed over in a strong aqueous solution. The formula of Schwimmer, herewith given, gave excellent results in a case of my own:

℞. Phenolis, 4.0–10.0;
Ol. olivæ, 40.0;
Cretæ præparat., 60.0.
M. et ft. pastamolis.

It has been recommended to touch each pustule with carbolic acid, then to apply this agent in equal parts with the oil of thyme (Sansom). It is important that only a certain proportion of the pustules be touched at once. Welch and Schamberg recommend painting the surface with tincture of iodine. The parts must be kept aseptic, while irritation from scratching must be carefully avoided. Moore and Fingen have recommended the use of red curtains or shades to cut out certain chemical rays. N. R. Finsen has advocated the exclusion of daylight, especially the chemical rays, by means of a red light, the skin being rendered very sensitive to light by the small-pox infection. The supposed effect is to prevent pustulation, and hence the formation of pitting or scars. A saturated solution of potassium permanganate applied to the exposed regions has been recommended instead of Finsen's red-light treatment, which acts similarly in that it excludes certain chemical rays. But as the result of treatment of test cases by Schamberg, Ricketts, and Byles, the claims made for red light have not been substantiated. The daily use of scrub-baths, though severe, appears to prevent vesiculation and the further progress of the eruption, thereby avoiding pitting (S. M. Wilson). For the itching, Welch and Schamberg recommend an ointment containing 2 drams of sodium bicarbonate in 1 ounce of petrolatum.

During convalescence, warm baths, with the free use of carbolic soap, are to be given at intervals of two days until several baths have followed the separation of the crusts.

(5) **The Period of Remission of Fever.**—There are very rarely any symptomatic indications apart from those presented by the eruption. It is of first importance, however, to support the powers of the system.

(6) **The Suppurative Stage.**—All measures tending to support the strength of the patient are needed—the mineral acids, with the elixir of calisaya, quinin, strychnin, etc. Stimulants are often required, and it may become necessary to give them unsparingly, the character of the pulse being the physician's principal guide as to dosage. Gradually cooled baths of the usual duration or warm baths somewhat more prolonged give excellent results. The ulcers in the mouth and throat are best relieved by the use of a saturated solution of chlorate of potassium in water as a gargle or in the form of an atomizer spray. Ice allowed to melt in the mouth is valuable. Hemorrhages demand ergot subcutaneously. Internally, the tincture of the chlorid of iron, gallic acid, the mineral acids, or turpentine may be administered.

The *complications* are not numerous, and are for the most part secondary. By frequently changing the position of the patient when bronchitis is present, and by encouraging him to cough frequently, as well as by the timely use of stimulants and the proper care of the mouth, pulmonary complications can often be obviated. Should lobular pneumonia occur, the plan of treatment which is likely to meet with most success may be briefly put thus: Free stimulation with alcoholics and other cardiants, the assiduous use of cold sponges or gradually cooled baths, and nourishing foods. *Laryngeal perichondritis* with edema

of the glottis may suddenly demand tracheotomy. To avoid the development of *bed-sores* an air-cushion should be provided, if needful. Care should also be exercised to prevent ocular complications, and their occurrence demands supportive treatment. I have much confidence in the use of cold compresses, instilling into the eyes at the same time a solution of boric acid (gr. x to xv—0.648 to 0.972—to f℥j—30.0).

(7) **Special Modes of Treatment.**—These would be found to be numerous, were we to enumerate all of them, but only those based on the principle of antisepsis are worthy of notice. According to one plan, which has many advocates, antiseptic agents are administered internally. The remedies that have been most frequently employed in this manner, and with perhaps the most promising results are the sulphocarbolates, salol, sodium salicylate, carbolic acid, creasote, mercuric chlorid, and the sulphites. R. A. Woodson[1] adopted as a plan of treatment in the Holguin epidemic, daily scrub-baths, 1–2000 mercuric chlorid, and open-air treatment. Du Castel advises at the time of the eruption injections of ether morning and evening; during the day a solution containing 2 or 3 grains of the extract of opium is to be given in divided doses.

Kinyoun, Lundmann, and Béclére have used the serum from vaccinated subjects (human beings and the lower animals) or from variolous patients in advanced stages of the disease in the treatment of small-pox. The cases, however, are insufficient to warrant deductions.

Special Methods of External Medication.—Talamon recommends a mercuric-chlorid spray for small-pox vesicles and pustules as follows:

℞. Mercuric chlorid, gr. xv (1.0);
Tartaric acid, gr. xv (1.0);
Alcohol (90 per cent.), f℥jss (6.0);
Ether to make f℥jss (45.0).
Sig. To be applied as a spray three or four times daily for one minute.

It is essential to exercise the precaution to protect the eyes, which may be covered by layers of cotton dipped into a saturated solution of boric acid. Talamon advises the commencement of his method on the first day of the eruption, the application to be preceded with a vigorous washing of the face with soap, which may be rinsed off with boric acid and then dried with absorbent cotton. After the spray has been used the face should be covered with a layer of a 50 per cent. glycerolate of mercuric chlorid in order to keep the skin continuously aseptic. After the fourth day the number of sprayings per diem is gradually lessened, so that by the seventh day they may be discontinued; but the application of the glycerolate should be continued.

Talamon added, in the confluent and other grave forms of the disease, general mercuric-chlorid baths, lasting for three-quarters of an hour to an hour. The buccal and pharyngeal eruption is to be treated by gargles and lotions of boric acid.

Convalescence.—A furfuraceous desquamation may persist for some time; it is to be treated by applications of oils containing some disinfectant. Convalescence is not established until desquamation ceases.

[1] Saunders' *Year-Book*, 1901.

VACCINATION.

Historic Note.—One of the first steps in preventive medicine was the practice of inoculation as a method of protection against the infection of small-pox. It had been practised in China and other Asiatic countries for centuries, and Lady Montague, the wife of an English ambassador to Turkey, early in the eighteenth century introduced it into England, after which time and until vaccination was known, it was very extensively practised there.

Pus taken directly from a small-pox pustule was introduced beneath the epidermis, and the person inoculated developed variola, though in a milder form than when arising from ordinary infection.

The objections to this method were that it did not always produce a mild form of variola, a small percentage of cases having a fatal termination, and that, however mild the attack, other unprotected persons brought in contact with it were as liable to contract virulent small-pox.

In a paper published in 1798, Edward Jenner, a physician of Gloucestershire, England, and a pupil of John Hunter, first made known to the world the value of vaccination. Twenty years previous he had observed that persons employed in dairies, who were accidentally inoculated with cow-pox were insusceptible to the contagion of small-pox, and, after experimenting all these years, he became satisfied that inoculation with the vaccine lymph was a preventive against small-pox. After the publication of his paper he was subjected to ridicule and abuse by the profession, but through his persistence he was finally allowed to practise his method of vaccination in the wards of a hospital, and in the course of a few years it became generally recognized and was practised in France and America, as well as in England. Later, the method fell into disrepute for a time, owing to the fact that certain persons who had been vaccinated subsequently contracted the disease, it not being known then that a revaccination was necessary from time to time. At present it is generally held that successful vaccination imposes complete immunity against variola.

Vaccinia, or cow-pox, is a mild eruptive disease that occasionally occurs among cattle, a similar disease being produced in them by inoculation with the small-pox virus from man. It is communicable by contact only, and is usually carried from one cow to another by the hands of the milkers; hence being usually found on the udder or teats of milch cows. Since Jenner's time many theories have been advanced as to the exact nature of this disease in cattle, and at the present day the subject is still in dispute. It is now, however, generally conceded that if cow-pox is a distinct disease, originating only with the cow, the eruptive disease produced in this animal either by inoculation of small-pox virus from man or of "grease" from the horse is, at least in all essential respects, a disease not to be distinguished from primary or idiopathic vaccinia. Guarnieri has described certain parasitic organisms, the *Cytorectes Guarnieri*, found in corneal lesions produced by the injection of vaccine lymph. This observation has been confirmed by Pfeiffer and others, but the pathogenic nature of these protozoa has not been determined.

The *vaccine virus* consists either of the liquid contained in the vesicle or of the scab resulting from the desiccation of the pustule. The former is furnished from vaccine farms, of which there are several in this

country, is then dried on ivory points, and, if kept in a cool place, retains its virtue for a week or ten days, or, possibly, longer, but should be used as fresh as possible to ensure a successful result. It is also sometimes preserved in capillary glass tubes, sealed at both ends, or between glasses, and kept in this way it is less liable to infection through uncleanliness in handling. The scab from the cow is not used.

The Site.—The point usually chosen for vaccination is on the arm over the insertion of the deltoid muscle; but in girls, for cosmetic reasons, it is sometimes preferred on the leg, and the most common site is over the junction of the two heads of the gastrocnemius muscle.

Technique in Vaccination.—After the part selected has been rendered surgically clean, gently scrape the skin with an aseptic lancet or other instrument until serum begins to exude. If by too vigorous scraping blood should be drawn, it must be carefully dried with a piece of sterile cotton before the lymph is applied. Hutchins has recommended a method in vaccination of denuding the surface of the skin with a caustic in place of the lancet. A piece of cotton, as large as the spot to be denuded, is wet with liquor potassæ and laid on the skin for two or three minutes, after which the spot is wiped dry and the softened epidermis rubbed away with an ink-eraser, a piece of soft wool, or preferably a piece of sterile gauze cotton, when the vaccine is applied in the usual way. The chief advantage of this method is its painlessness.

The charged end of a vaccine point, covered with glycerinated virus and protected by a thick covering of paraffin, is now gently rubbed over the abraded spot and the limb left exposed to the air until the lymph has been dried upon it. It may then be protected by a piece of gauze strapped on it. *Humanized lymph* is still preferred by some, and when this is used the "arm-to-arm" vaccination is best. The lymph is taken from characteristic vaccine vesicle (from the fifth to the seventh day) of a healthy child and applied directly to the arm of another. The virus may be dried and preserved for use as in the case of bovine virus.

The *scab* resulting from a vaccine vesicle on a healthy child was formerly quite generally used, and it could be kept a long time without losing its virtue. It was sure in its action, and offered the advantage to the physician of being easily preserved; but it was more liable to become infected than the lymph when preserved in the usual way, and, since the vaccine farms are so conveniently located, lymph may be obtained from them at any time without delay. The possible danger of conveying syphilis or other constitutional disease from one person to another by means of humanized lymph should lead to its abandonment in favor of the bovine lymph. In recent years, however, bovine virus has been shown to be occasionally infected with tetanus, and that implantation of the tetanus germs at the time of vaccination may occur.

Period of Life for Vaccination.—It is usually advised to vaccinate infants within a few weeks or months after birth; but unless small-pox is prevalent, it is best to wait until the latter part of the second or the beginning of the third year, as the child has then passed through its teething period and will be better able to resist the effects (slight though they may be) consequent upon vaccination. If an epidemic be prevailing, vaccination should be performed during the first week or even the first day after birth; and pregnant women should receive prompt vaccination at any period of gestation if exposed to small-pox.

Time for Revaccination.—To ensure the individual against infection he should be revaccinated at puberty and every few years afterward, or at any time when small-pox is epidemic or is liable to become so.

Symptoms.—After vaccination no local or constitutional effects—except the slight irritation due to scarification—are noticed until the third day, rarely as late as the fifth or even sixth day, when a *small red papule* appears. By the fifth or sixth day a *vesicle* appears. By the ninth day it is fully developed, and, like the vesicle of variola, is filled with colorless lymph, is umbilicated, multilocular, and has a distinctly inflamed areola of deep red color, accompanied by heat, itching, and tenderness. By the tenth day this may extend an inch or two from the vesicle. Quite frequently the axillary or inguinal glands (depending upon the location of vaccination) are swollen and tender, and in a tubercular child they may go on to suppuration. After the tenth day all these symptoms gradually decline; the *pustule* dries up, and then forms a brown scab which is usually detached in the third or fourth week, leaving a permanent cicatrix. The course is more rapid in revaccinations.

Complications.—Occasionally one or more additional vesicles are formed at a little distance from the point of inoculation, and, rarely, there is a general vesicular eruption, due to absorption of the lymph. An *erythematous rash* about the sixth day is not uncommon. *Erysipelas* may occur as a complication, and, if it is prevalent in the house, vaccination should, as a rule, not be performed. Among other rare complications are tetanus (*vide* p. 313) and the hemorrhagic diathesis.

An ulcer may form which may be weeks in healing. Sexton noted marked improvement from the constant local application of antistreptococcus serum. *Eczema* and other skin affections are usually aggravated during the course of vaccination, and it is possible for syphilis to be inoculated with the vaccine virus. Any of these complications call for the usual treatment.

VARICELLA.

(*Chicken-pox.*)

Definition.—An acute, contagious disease, characterized by a cutaneous eruption of papules, passing into vesicles and pustules; also by slight fever and mild constitutional symptoms. For a long time it was confounded with varioloid, but its distinct character has now been recognized for many years. Complications and sequelæ are infrequent.

Etiology.—The virus is not transmitted by the inoculation of the vesicle contents, as a rule, although it has rarely been thus communicated. The specific poison has not been satisfactorily isolated, but it is suspected that certain protozoa are the direct cause. Positive proof, however, is wanting. Varicella may be transmitted by exposure to another case or possibly through the medium of a third person, the school and asylum being the most frequent points of its origin. It affects children of all ages, and usually one attack is protective. Doty and others have observed varicella in the adult. It closely resembles measles in its contagiousness.

Symptoms.—The *incubation* period is uniformly from fourteen to sixteen days. If there be a prodromal stage of the disease, certainly in the vast majority of cases it cannot be recognized, though a slight *fever* and general indisposition may be noticed for twenty-four hours before the appearance of the eruption. In many cases the *eruption* is the first symptom. This occurs in the form of small reddish puncta, from which rapidly develop rosy-colored maculations, and these become densely distended, transparent, or slightly yellowish vesicles of the average size of a split pea. The eruption *appears first* upon the upper part of the body, the chest and back, neck, scalp, and face (on the latter quite sparingly), and always upon the hairy scalp. Frequently the *vesicles* form on the mucous surface of the lips, inside the cheeks, on the tongue, palate, conjunctivæ, and in the progenital regions of both sexes. At times the glands of the throat become slightly enlarged and painful, the vesicles are superficial, the child has the appearance of having received a shower of boiling water, and the firm papule which precedes the variolous rash is altogether wanting. The vesicles are at first transparent, and their contents plainly show through their translucent roof-wall which is composed only of the stratum corneum of the epidermis. The contents of the vesicles become lactescent, and gradually seropurulent. The *areola* is most distinct when the vesicle is fully formed and fades as the latter dries. Desiccation begins at the apex of the vesicles. *Crusts* form, which drop off in from five to twenty days, depending upon the depth to which the skin has been involved. On the trunk, as a rule, no mark is left, but after the more severe attacks, when the true skin has been involved, scars remain, and frequently there is quite deep *pitting*. The marks are usually on the face when the skin has been unprotected. On the hands and feet the vesicles appear without having been preceded by a papule, and sometimes there is no areola, each vesicle resembling a drop of water upon a healthy skin. *Pustules* may develop in consequence of irritation or infection, as the result of scratching, or in feeble or poorly nourished children, and in unhealthy children deep ulceration may occur, lasting for weeks.

In *mild cases* only ten, twenty, or thirty spots may be found on the body, but in severe cases the skin may be almost covered in certain regions. The eruption, however, is never confluent. The *temperature* is highest on the second or third day, when the eruption is appearing. In mild, uncomplicated cases the thermometer registers 101° or 102° F. (38.8° C.) for two or three days at most, but in severe cases the temperature may be as high as 104° F. (40° C.). This is usually due to broken health prior to the acute illness. The temperature falls *gradually* as the rash fades, and presents a temperature-curve similar to that of measles.

There is usually neither coryza, cough, vomiting, nor diarrhea.

Complications.—*Erysipelas* occasionally acts as a serious complication in delicate children. It may develop about the pocks, particularly when they are deep and associated with some ulceration, and scratching with unclean fingers is its prime causal factor. In rare cases there may be necrotic inflammation about the site of the pox (*varicella gangrænosa*). *Adenitis*, mild and isolated, and *suppuration* in the deeper cellular tissue may occur.

Nephritis is infrequent, but may occur in carelessly managed cases. L. Ceof[1] has collected 40 cases of nephritis complicating varicella.

[1] *Arch. de Med. des Enf.*, Feb., 1901.

Varicella is also quite frequently complicated with other infectious diseases, and varicella, scarlet fever, and measles have been seen curiously blended in epidemic form. Ceof has reviewed the literature and found 40 cases of scarlatiniform eruption occurring in varicella. Varicella and measles, however, are more commonly associated.

The **diagnosis** of varicella offers no special difficulties. The eruption comes out slowly and in crops, so that papules, vesicles, and crusts may be seen upon the skin in close proximity. Again, it should be noted that the umbilication is due only to the fact that the drying up of the vesicle begins at the center. Varicella is distinguished from *urticaria* by the presence of fever, and from *eczema pustulosum* by the mild febrile symptoms of the latter, the discreteness of its pustular lesions, the absence of itching and of infiltration of the skin in patches, and by its tendency to symmetric development.

Variola and *varioloid* of infants are to be distinguished from varicella by the prodromal symptoms, and by the greater rise of temperature, though the distinction between mild varioloid and severe varicella will always tax to the utmost the skill of the keenest diagnostician (*vide* table, p. 195). The sooner it is understood that intermediate forms are likely to occur, which cannot be positively assigned to one or the other category, the better it will be for both the profession and the laity.

The **prognosis** in private practice is always favorable. Only in the slums or in hospital cases complicated by erysipelas, adenitis, gangrene, or nephritis may grave results be anticipated. The milder cases may, however, leave slight monuments of their existence in the form of one or more depressed cicatrices which may mar an otherwise beautiful face.

Treatment.—Isolation should be enforced in schools and in all institutions containing many young children. In private houses, unless the younger children are delicate, quarantine is unnecessary. The disease may be transmitted to others as long as the crusts are present, and hence isolation should be maintained until they have fallen off. In most cases constitutional symptoms of the disease are so mild as to require no treatment. It is best at the outset to place the child in bed for a few days, and sponge daily with warm carbolized water; the local itching may be allayed by sponging with a weak solution of carbolic acid or by the use of carbolized vaselin. When the crusts have formed, especially on the face, an ointment of zinc oxid containing ichthyol (2 per cent.) should be applied, and care should be exercised to keep the skin clean and to prevent scratching. In all cases the urine should invariably be examined several times during and following the attack.

SCARLET FEVER.

(Scarlet Rash; Scarlatina.)

Definition.—Scarlet fever, or scarlatina, is a self-limiting, acute, contagious disease, characterized by vomiting, fever (more or less typical), angina, and in twelve or twenty-four hours by a diffuse, punctiform, scarlet eruption, followed by membranous desquamation and, frequently, by nephritis. It is a disease of childhood, but may occur at any time of life.

Scarlatina is a widespread disease, though perhaps less universal than measles. It is endemic in all the large cities of the globe, and at intervals the cases multiply into more or less extensive epidemics. Smaller towns and rural districts are visited, and the epidemics are usually traceable to importation of scarlatinal poison, so that it may be stated that they never originate *de novo*.

Pathology.—There are no pathognomonic changes. When death occurs early the chief lesions are presented by the throat, while in addition engorgement of the viscera is noted, especially of the brain. The exanthem is rarely visible. In malignant types, however, in which the eruption is not seen during life, it makes its appearance rarely after death, and this aids in establishing the nature of the affection.

When death occurs at an advanced stage the lesions are those either of nephritis (with dropsy), or of septico-pyemia, or of inflammation of one or more of the serous surfaces (pleurisy, pericarditis, endocarditis, meningitis, etc.). Additional changes in the various viscera are, for the most part, identical with those met with in other acute infective diseases, and hence need not be described here. The blood is dark, fluid, and coagulates feebly, owing to a decrease in its fibrin factors.

Among other lesions which are more or less peculiar to the disease are (*a*) The *eruption*, which is a dermatitis of very mild grade. J. F. Schamberg[1] points out that the discrete vesicles sometimes seen originate in the hair-follicles or in the deeper layers of the *rete*, and contain a turbid leukocytic fluid. (*b*) *Scarlatinal angina*, which in its mildest form usually presents more or less hyperemia and a slight swelling of the mucosa of the tonsils, soft palate, and pharynx. In the severer grades the inflammation is phlegmonous (*scarlatina anginosa*), and sometimes terminates in ulceration. There is great swelling (especially of the tonsils), and the formation of abscesses is common. Extension of the purulent inflammation to the connective tissue of the neck produces marked induration, and more or less extensive abscesses may take place. Gangrene sometimes supervenes. (*c*) In certain epidemics a *membranous exudate* accompanies the *scarlatinal angina*, and this may or may not be truly diphtheritic. When it appears early it is non-diphtheritic, as a rule, and often due to the streptococcus; on the other hand, when it comes on late it often shows the presence of the Löffler bacillus. Schabad has, however, shown that bacilli taken from the throats of incipient cases of scarlet fever, although morphologically characteristic, have little or no virulence. There is also a malignant form of membranous scarlatinal angina, occasioned by a secondary streptococcic infection (Hirschfeld). (*d*) *The Nephritis.*—The renal lesions are included in the description of "Acute Bright's Disease."

Etiology.—The **bacteriology** of the affection is imperfectly known. The streptococcus pyogenes has been found in nearly all the inflammatory complications of the disease, especially scarlatinal pneumonia and angina, and some pathologists (Babés, Bergé, Klein) have held it to be the cause. Raskin and Mosny, however, believe that it is an example of mixed infection, the streptococcus being merely a secondary factor, and Charlton has shown it to be the cause of the unfavorable complications.

[1] *Proc. Phila. Path. Soc.*, Jan., 1901.

W. J. Class[1] first described an organism (*diplococcus scarlatinæ*) His researches have been confirmed by those of Gradwohl,[2] Jaques,[3] Page,[4] and others. The habitat of the diplococcus is not known, but it has been found in the blood, throat, epidermal scales, and urine of scarlatinal cases. The size of the organism is variable, and it stains with standard watery dyes easily, uniformly, and regularly (Gradwohl). Class[5] reports on his experiments to obtain an *antitoxin for diplococcus scarlatinæ*, in which he was successful. Sommerfield[6] has found the constant presence of streptococci in the tissues and blood. This may be the same organism as the Class coccus.

F. A. Mallory[7] has demonstrated the presence of protozoa (*cyclaster scarlatinalis*) in the skin in early stages of scarlet fever. They are best observed on the second day of the cutaneous rash, and usually occur within the epithelial cells, but have been observed also in the lymph spaces in the corium. The most striking form is a rosette-shaped body not unlike a similar appearance seen in the life cycle of the hematozoön of Laveran, and about one-third larger than the latter. Vipond claims to have found a bacillus in the enlarged axillary and inguinal lymphatics.

The *receptivity* for scarlet fever is not so great as in certain other exanthemata (*e. g.*, measles); hence in a household in which there are several children, some are apt to escape the disease, even though all have been equally exposed. The *virus* is probably contained in the excretions from the *throat*, *nose*, or *ear*, and in the epidermal scales thrown off from the surface of the body. It is also present in the blood.

Modes of Conveyance.—(*a*) The majority of the cases are produced by *contagion*, and I have observed that a single contact of a healthy child with a scarlet-fever patient suffices. The disease may also be transferred by persons who have been in the sick-room, while they themselves escape. Aaser[8] found, out of 3800 cases, 79 had been infected by discharged hospital patients from one to five weeks after cessation of desquamation ("return cases"). The source of infection in these cases is an abnormal secretion due to some local affection of throat, nose, or ear associated with discharge. (*b*) It is also communicated by *fomites*, and the poison of scarlatina contained in clothing retains its infective power for months. Again, any objects (furniture, utensils, library books, toys) which the patient has handled may serve to communicate the poison. (*c*) Infected dairies have been known to disseminate the poison and give rise to epidemics. (*d*) The infection may also be air-borne, though not for any great distance. (*e*) Behle reports an outbreak of human scarlatina in swine; and kine are potent to transmit it to man.

Mode of Infection.—Most probably the poison is *inhaled* into the throat, where infection usually occurs; but it may gain entrance to the body through the *alimentary tract*. Infection may also take place

[1] *Monthly Bulletin of the Chicago Dept. of Health*, March, 1899.
[2] *Philada. Med. Journ.*, March 24, 1900.
[3] *Bulletin N. W. Univ. Medical School*, March 31, 1900.
[4] *Journ. Boston Med. Sci.*, June 20, 1899.
[5] *Philada. Med. Journ.*, June 23, 1900.
[6] *Arch. für Kind.*, Jan., 1902.
[7] *Medical News*, May 14, 1904.
[8] *Nord. Med. Arch.*, 1903, Abt. II., Anhang 51.

through the blood, as is shown by the fact that children have been born in all stages of the disease. Artificial inoculation with the blood of scarlatina patients has resulted in more or less typical forms of the complaint. *Open lesions* predispose, but whether they are essential to infection is not known.

Predisposing Causes.—(1) *Age.*—The period of chief liability is from the second to the tenth year, after which it diminishes. It is rare under the age of one year, and especially so under six months. (2) *Recent wounds*—accidental or surgical—increase the susceptibility to the peculiar poison. (3) *Women in childbed*, for the same reason as (2); but care must be exercised lest this class be confounded with septic affections. (4) *Season.*—The autumn and winter months furnish the most cases. (5) Seitz believes there is evidence of a *family predisposition*, as 371 out of 800 cases occurred in 152 families.

Immunity.—Single attacks during the life of a person form a rule to which there are rather frequent exceptions.

Clinical History.—The **incubation-period** is variable, lasting, on the average, from ten to fourteen days (McCollam). It may rarely, however, be longer, although more commonly a briefer period, three to eight days.

The **invasion** of scarlet fever is generally quite *sudden* and, as a rule, active. The child feels uncomfortable, looks stupid, complains of *sore throat* and decided nausea, and in the great majority of the cases *vomits*. The tongue is furred. If he be very young, *nervous symptoms* are prominent, and the initial symptom may be a convulsion. The *pulse*, which is a strong diagnostic factor, is rapid and hard, reaching 140 to 160 at the very onset. The *temperature* rises quickly to 104° or 105° F. (40.5° C.), and remains high.

Eruption.—Within the first twenty-four or thirty-six hours the characteristic rash appears, and is, as a rule, first seen on the neck; there is no certainty about this, however, as it may first come out on the abdomen or back of the hands or on the thighs, and not be seen on any other part of the body. Frequently it is found on the dependent portions of the trunk. At first it is slight, but perfectly characteristic, and usually takes two days to mature. In mild cases it disappears within thirty-six to forty-eight hours, and at no time is more than a very fine rash, but when typical it cannot be mistaken, especially if accompanied by the premonitory symptoms. When seen from a short distance at the end of the first twenty-four hours of its appearance the whole body (except the face) is of a uniform bright scarlet color. If we examine more closely, we find that the eruption consists of a multitude of red points (*puncta*) that correspond to the hair-follicles. These points are surrounded by zones of erythematous redness, which, joining with one another, give a generally diffuse red appearance to the whole skin. Frequently, however, the rash consists of points representing the hair-follicles without the erythema, and in rough skins the rash may be more punctiform—that is, more strictly a condition of "goose skin." Sudamina are quite frequent. Pressure by the finger causes a pallor which at once disappears when the finger is removed. The patient's lips and chin are pale and in striking contrast with the vividly scarlet cheeks. In some cases the rash is patchy, especially on the limbs, and in these cases it may suggest measles, the patches consisting of clusters of fine papules or points with much surrounding

erythema, while normal skin is present between the patches. An intense continuous linear exanthem in the skin folds at the bend of the elbow (Pastia's sign) is supposed to be proof of scarlet fever. In severe cases the rash may be hemorrhagic in character, minute extravasations of blood taking place in the skin; this may occur even in mild attacks, and not be seen until after death, but more frequently it is seen in malignant cases. Purpuric patches are frequently found after death when even in life they do not appear. There is itching, which may be intense.

The rash is succeeded by a *desquamation* that will be extensive or slight according to the intensity of the fever. In *mild cases* the tonsils, palate, uvula, and pharynx are deeply congested, and the mucosa of the cheeks, palate, and tonsils may show the eruption. In *severer forms* the tonsils are red and inflamed, and covered with tenacious secretions, while minute yellow points corresponding to the tonsillar crypts are usually prominent. (*Vide* Malignant Scarlatina.) The nasal chambers are swollen, producing a free discharge, and the deeper cervical glands at the angle of the jaw are frequently enlarged. The *tongue* is coated with a thick, dense white fur (dead epithelium), and frequently shows a dry, glazed central band. In a few days the dead epithelium is cast off, clearing the tongue, when we have a red, clean, glazed tongue with greatly enlarged fungiform papillæ, giving us the *strawberry tongue* of classical history. The eyes are frequently swollen and the conjunctivæ injected. *Sleeplessness* and *mild delirium* often mark a typical case, suggesting a congested state of the meninges, but it is neither usual for the child to be violent nor for the delirium to continue long.

The *pulse* is usually a strong diagnostic feature, and is always hard, quick, and wiry, varying from 140 to 160; its rate is out of proportion to the temperature and the general condition of the child. *Leukocytosis* is noted; it develops early and is most marked in cases showing suppurative lesions. This leukocytosis is a true one, *i. e.*, an increase absolute and relative in the polymorphonuclear cells. With the onset of defervescence there is a constant eosinophilia (Tileston and Locke). The *temperature* in average cases reaches 104° or 105° F. (40.5° C.), and in severe forms it may touch 106° F. (41.1° C.), the nocturnal remissions being slight and defervescence gradual (*vide* Fig. 17). The *urine* is scanty, thick, and contains urates, with a small quantity of albumin.

Within one week, if no complications have occurred, the attack will have reached its height and the symptoms have begun to decline. The rash gradually fades, temperature falls, the tongue is less red, the throat less injected, and the child seems more natural. If at the end of one week the fever continues, it suggests one of the many possible complications, the most frequent of which are a throat or tonsillar ulceration, inflammation of the cervical glands, otitis, or acute nephritis (common). It must be well understood that no two cases of scarlet fever are alike.

Clinical Types.—Mild Scarlet Fever.—The premonitory symptoms are sometimes absent, the rash being the only indication of the complaint. There is neither vomiting nor fever to be recognized, and no tonsillar trouble of any importance, while the rash is neither uniform nor well marked. In these cases we must be very careful not to confound the eruption with urticaria or some of the many medicinal rashes.

During house epidemics when several children are affected it frequently happens that a child has sore throat and the "strawberry

tongue" without a development of the rash. This may also occur in adults, and is the so-called *scarlatina sine eruptione*. These very slight cases of the fever may be followed by the most severe attack of nephritis.

Here may be mentioned the so-called "*fourth disease*," in which the symptoms are said to resemble both German measles and mild scarlet fever and yet to be unlike either. It is to be recollected that "the symptoms may be caused by the poisons of both these affections acting simultaneously" (Dent).

Traumatic scarlatina is that form in which infection occurs in a wound. The eruption makes its appearance at the wound, and then spreads over the body; it is less severe in its course than ordinary scarlatina.

Malignant Scarlet Fever.—Death occurs usually by the end of the first week in severe cases, Drs. Ashby and Wright reporting a death within the first twenty-four hours (*atactic form*). In malignant cases such as usually occur among the unhygienic and delicate, the tonsils may be

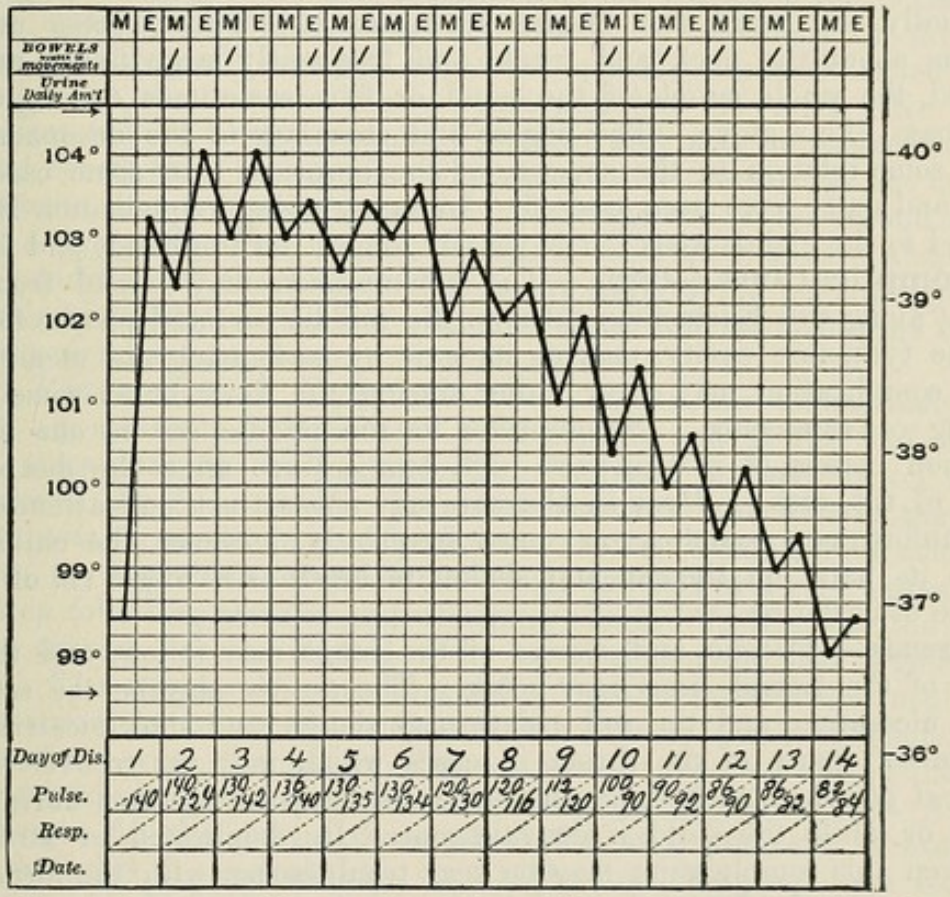


FIG. 17.—Temperature-curve of a case of scarlatina with favorable course—William C——, aged seven years.

covered by a membranous exudate, and the system quickly receive an overwhelming dose of the poison; death then results from septic causes (*anginose form*). In cases in which death occurs early a child soon becomes cyanotic, restless, or more frequently somnolent. In all these cases the temperature remains high—105° to 106° F. (41.1° C.), and very frequently 107° F. (41.6° C.). Diarrhea is frequently a troublesome factor in severe cases; coryza is very abundant; there is much

glandular swelling and cellulitis, the neck becoming enormously enlarged and hard, the skin dull and livid in color; the extremities grow cold; the heart gradually becomes irregular, losing a beat, and finally fails.

If life is sustained through such an ordeal, the tonsils slough and the lungs may eventually become the seat of a septic pneumonia. General septicemia is most likely to occur. In this condition the tonsils ulcerate, sloughing patches appear on the fauces, the glands about the neck become enlarged and doughy, and the nasal mucous membrane gives out a purulent secretion in abundance. The temperature may remit, but continues high; the urine is albuminous; pus wells from both ears; and thus the child is gradually consumed and perishes.

A third variety (*hemorrhagic*) shows at first cutaneous petechiæ which grow rapidly into large ecchymotic patches. Hemorrhages also take place from the mucous surfaces, epistaxis and hematuria being very common. Death, as a rule, follows in two or three days.

Desquamation.—By the end of the first week the rash commences to disappear, the skin is (or soon becomes) mottled, dry, and rough, and gradually the scarf skin begins to separate. This process usually begins about the neck and trunk, and frequently large flakes are detached, the whole cuticle of the hand or foot sometimes coming off in one mass like a glove. The degree and character of the desquamation bear some relation to the severity of the eruption. In some cases the hair and nails have been cast off. In many cases desquamation is prolonged to the eighth week; it is usually longest on the hands and feet.

Complications.—**Otitis.**—The inflammation may extend from the throat along the Eustachian tubes to the middle ear, and pus be formed in the tympanic cavity, making its exit by perforating the membrane. This complication may occur either during the fever or at some time during convalescence. Suppuration in the middle ear is one of the common causes of a continued high temperature after the disappearance of the rash. *Pain* in the ear may not attract our attention to this unfortunate complication; most frequently, however, the child will place its hand on the ear and shake its head, as if to get rid of some source of irritation.

Pyemia.—*Pyemia* and *abscess* of the lungs may follow, and thrombosis of the lateral sinus may occur. The *tonsils* may be the seat of deep ulceration, and the soft palate may slough and show cicatrization of the soft parts of the throat in cases which may yet recover. The *cervical glands* may become enlarged and suppurate, either during the fever or while the child is convalescent. In debilitated or strumous children this complication may be very troublesome, with the formation of deep ragged ulcers, slow to heal, and in rare cases exposing the larger blood-vessels. *Broncho-* or *lobar-pneumonia* may occur, and is most usual during the second week, being due to extension downward of the lesion from the throat. Pneumonia followed by *empyema* may also occur during convalescence.

Joint-affections.—(*a*) *Scarlatinal synovitis*, which occurs in 7 per cent. of cases, nearly always appearing from the fourth to the tenth day; in 72 per cent. of cases affecting the wrists (Marsden). Less commonly the small joints of the fingers, the elbows, the ankles, the knees, and soles of the feet may be affected. The trouble is fugitive, and seldom returns to the same joint, and is caused by the scarlatinal poison. (*b*)

Septic arthritis, met with in severe or fatal cases, is often associated with grave throat symptoms. In these cases the knees may be most severely affected, remaining swollen for weeks, and in unusual cases suppuration may take place and be followed by pyemia. (*c*) *Rheumatic synovitis*, which usually develops during convalescence. Two cases have occurred in my practice, complicated with simple acute endocarditis. Rarely tuberculous invasion of the joints occurs as a *sequel*. Here the scarlatina merely affords a predisposition to tuberculosis.

Nephritis.—No other complication of scarlet fever can equal *nephritis* in importance or interest, this condition always giving rise to anxiety in otherwise mild and hopeful cases. During the height of the fever there is commonly a transient albuminuria, and it is possible for the kidneys to escape without greater damage than occurs in other acute febrile affections. Independently of this febrile albuminuria, there are two forms of nephritis which it is important to bear in mind, though they have been frequently confounded:

(*a*) *Septic Nephritis.*—In severe forms of scarlet fever, when the throat symptoms include sloughing tonsils, involvement of the soft palate, and general adenitis, the urine quickly becomes loaded with albumin, but shows scarcely any blood and but few casts. No renal symptoms will be recognized, and if present they may be masked by the general condition of septicemia. There may be neither dropsy nor uremic phenomena, but the patient usually dies by the end of the second week, when a typical pyemic kidney is found containing minute abscesses. This condition of the kidney is only one part of the general pyemia, and merely illustrates the fact that this organ suffers during the course of the general inflammation.

(*b*) *Post-scarlatinal Nephritis.*—The kidneys are undoubtedly involved in an acute sympathetic inflammation, and at the end of the fever, more than at the beginning, are engaged in carrying off waste products of the fever itself. From the nature of the disease they are in an irritable condition and prone to take on inflammatory changes, just as the bronchial tubes and the lungs are left in a very susceptible condition following measles and whooping-cough. In this way the uriniferous tubules become choked up by the desquamation that is going on inside. The *number of cases* that suffer from post-scarlatinal nephritis varies according to social conditions, the nature of the epidemic, the season of the year, the nature of the treatment received during the disease, and especially the care received throughout convalescence. Ashby and Wright fix the rate of those who suffer at 6 per cent. of hospital cases, but this is, undoubtedly, too high, since hospital cases receive excellent care during convalescence as a rule. The usual *time* for this form of nephritis to occur is from the end of the second up to the fourth week, but it usually begins very insidiously. Traces of albumin may be found for a few days before the blood and larger quantities of albumin occur, but it is often impossible to date the commencement of an attack. Usually after the fever has subsided the patient for a few days feels well, but very suddenly grows restless, is feverish at night, is thirsty, has a quick, hard pulse, and passes a small quantity of dark-colored urine. The temperature almost invariably rises again and there is no relation between the pulse and the temperature. If care has been exercised, it will be found that the urine has been gradually diminishing

for several days, and a slight puffiness about the face frequently announces the beginning of the trouble. Later the face becomes pale and puffy, while there may be edema of the feet and scrotum, and some vomiting. Under favorable treatment improvement may take place, large quantities of urine may be passed, and the child resume convalescence. The nephritic symptoms may, however, deepen until *uremia* appears, the pulse becoming slow and wiry in character, the temperature subnormal, and the tongue dry and brown. Vomiting is now a frequent occurrence; diarrhea is not unusual; nose-bleed and hemorrhages from the various mucous surfaces, and muscular twitchings may be noted, and most likely the end may be reached in a general convulsion. After scarlet fever, kneeling for ten minutes in the lordotic position may produce albuminuria. Fatal results, however, are more frequent from *cardiac failure* than from the uremic convulsions. The constant effect of nephritis is to raise the blood-tension, followed by dilatation of the heart. Another not unusual result is *endocarditis* or *pericarditis*, with possible embolism.

Sudden death frequently occurs during the course of nephritis. The child may be doing well, possibly sitting up in bed and playing with its toys, when an attack of dyspnea occurs; the face becomes livid, the pulse disappears, and death quickly takes place. Death in such cases is due to a *dilated heart*, followed by edema of the lungs.

It is not unusual for a false membrane to form upon the *larynx*. This is not infrequently due to the streptococcus pyogenes, but the Klebs-Löffler bacillus is oftener found. A bacterial examination should always be made, and if the *diphtherial* nature of the infection has been determined the serum treatment should be employed at once.

Diagnosis.—A typical form of scarlet fever offers few difficulties in diagnosis. The period of incubation is short in comparison with that of any of the other exanthemata, particularly variola, measles, and varicella. The vomiting, associated with high fever, would also exclude the other eruptive diseases. The pulse in itself is strongly diagnostic, being quick, hard, and wiry, striking the finger at the rate of 140 to 180 per minute. The early sore throat and the intense hyperemia of the whole mucous membrane, associated with marked constitutional symptoms, make it easy to differentiate from measles, varicella, and variola. Leukocytosis occurs in this disease, but is not present in measles or varicella. Döhle[1] discovered certain inclusion bodies in the polymorphonuclear leukocytes from scarlatinal blood prior to the sixth day. This view is confirmed by Granger and Pole, but Bongartz and others state that it is not possible to consider the inclusion bodies pathognomonic for any one disease. The punctate eruption of scarlet fever is not found in any of the other eruptive diseases (*vide* table on page 219). If a child has never had scarlatina and the characteristic symptoms are present, a rapidly growing culture (taken from the throat) of the Class coccus from such a case determines the presence of scarlet fever, while its absence excludes the disease (Jacques).

The **differential diagnosis** embraces the discrimination of those rashes that follow the use of certain drugs (quinin, belladonna, potassium bromid and iodid, chloral, etc.). The characteristic invasion-symptoms (vomiting, angina, etc.) of scarlatina are absent; also the high fever

[1] *Centralbl. f. Bacteriol*, Nov. 23, 1911.

and frequent, hard pulse of the latter disease. *Drug-rashes* are seldom so vivid or diffuse as the eruption of scarlatina.

Scarlatina.	Acute Exfoliating Dermatitis.
Onset is sudden, with vomiting, angina, fever, and frequent, hard pulse.	Sudden, with fever only.
Eruption appears first on neck, face, and chest, soon becoming diffuse.	Appears first on trunk.
Duration, three or four days.	Duration, five or six days.
Desquamation begins after eruption has faded, often one week later.	Desquamation begins earlier, often before eruption has faded, and involves the hair and nails.
Ear and throat complications common.	Absent.
Nephritis is a common sequel.	Not so.
Relapses exceptional.	Relapses common.

The **prognosis** in regular, uncomplicated scarlet fever is almost always favorable, and, unless the treatment is unusually indifferent, the patient will recover. Severe types, however, and especially malignant scarlatina are very fatal. Complications arise that will most seriously endanger life.

The **treatment** of scarlet fever is that of the symptoms, together with an attempt at arresting the complications.

Prophylaxis.—The patient should be strictly *quarantined* in an upper room for at least eight weeks or until desquamation has been completed.

During *convalescence* hematinics are required to overcome the symptomatic anemia and debility. Preisich urges the importance of separating the convalescents from patients in the first stages, and of getting the mucosæ into a healthy condition before the patient is discharged. A competent nurse should be put in charge, and, whether a member of the family or otherwise, she should wear a washable dress, and should not mingle with the family except her clothing be changed or thoroughly disinfected. The room is to be stripped of all superfluous hangings and furniture. *Inunctions* are required as soon as desquamation commences, with a view to preventing the diffusion of the dried epidermal scales; and the best preparation for this purpose consists of cosmolin, menthol, and carbolic acid, ten grains each of the latter to one ounce of cosmolin after the plan of J. Lewis Smith. Carbolized water, 1 : 40, may be used to sponge the surface and may be agreeably followed by cocoa-butter. The naso-pharynx must also be kept disinfected.

The *disinfection* of the physician himself is important. He should generate chlorin gas by the following simple method, and allow it to permeate his clothes thoroughly before going into other families: A dram of powdered potassium chlorate is placed in a saucer, and a small quantity of hydrochloric acid added. The dish is then placed on the floor, and the physician stands over the vapor chlorid as it arises until it penetrates all his clothing. This, with the free use of the whisk and thorough hand-washing, renders him non-contagious and safe in entering any home or sick-room. The best method is to have in the patient's house a linen duster or surgeon's apron that has been dipped in a bichlorid solution and allowed to dry. This is slipped over the clothing before entering the sick-room, and is removed after leaving.

In the room, if the case be a severe one involving the throat, I keep the gas or an alcohol lamp burning under a small dish of water, so that steam is constantly generated. To the boiling water I frequently add

carbolic acid or oil of eucalyptus; this saturates the room very pleasantly, and at the same time tends to limit the extent of the contagion.

General Management.—The sick-room should be large and well-ventilated, and should be kept at a uniform temperature (68° to 70° F.—21.1° C.). A light flannel night-dress should be worn by the child, and the bed-clothing should be light as well. The *diet* should consist of milk, egg-white, and fruit-juices, and after the temperature has declined soft diet may be allowed. J. McCrae[1] insists upon twenty-one days' milk diet. A return to ordinary solid foods, especially proteids, must be made gradually during convalescence.

The evidences of heart-enfeeblement often arise and call for the judicious use of stimulants. This class of agents is remarkably well borne in this affection. To a child of four years I give one dram (4.0) of brandy or whisky every second hour, and often increase the dose as required. The preparations of ammonium, particularly the carbonate and the aromatic spirits, have also been warmly recommended. They should be administered in milk as the vehicle to prevent gastric irritation. Heart-failure is best treated by baths at 90° to 95° F. and oxygen inhalations (Ausset).

Special Treatment.—In the classical work of Thomas Watson, now over fifty years old, he hints in his treatment of scarlet fever "that, if the heat on the surface be very great and distressing, he should not recommend the cold affusion, but cold or tepid sponging would be very refreshing and beneficial." This sentiment finds its echo in most works on practical medicine at the present day. The physician must quietly but firmly insist upon the patient being sponged three or four times daily, according to the severity of the individual case, using carbolized water (1 : 60), mercuric chlorid (1 : 8000), or alcohol and water, at a temperature of 70°–100° F. (21.1°–37.7° C.). Systematic bathing and inunctions as above described protect the body from certain complications and sequelæ. The ice-cap may be combined with cool spongings. In extreme cases, with marked nervous symptoms and high temperature, the cold pack, with cold affusions applied to the head and nape of the neck, may be employed, and a description of the method of giving a cold pack may be found under the treatment of Typhoid Fever. A notable reduction of temperature may be secured from an injection of a pint or more of cool water containing 2 to 10 grains, according to the age of the patient, of sulphocarbolate of soda per rectum (de Voe). To eliminate the toxins, the baths should be aided by the administration of large quantities of water, and the bowels should be kept freely open.

In regard to the use of *internal antipyretics*, I prefer phenacetin for older children, combined with quinin in capsules. Acetanilid is better for younger children, and I generally give one-third as many grains as there are years in the child's life. When medicine can be exhibited in the form of capsules, I always prefer to combine it with quinin or strychnin to overcome the tendency to depression. Phenacetin and acetanilid act successfully in controlling the nervous element, relieving headache and fever, promoting diaphoresis, and inducing refreshing sleep. Acetanilid is much more prompt in its action than phenacetin, but its effects are not so lasting. These agents, however, are rarely required.

Internal Antiseptics.—Those remedies that are purely antiseptic, administered internally, have not given proof of their utility as yet.

[1] *Montreal Medical Jour.*, Sept., 1908.

The sulphocarbolates of zinc and of sodium, on account of their breaking up in the system and liberating carbolic acid, cannot be used in a sufficiently large dose to meet with success. The syrup of phenic acid is used by many physicians. Marmorek, and later Charlton, has used his *antistreptococcic serum* extensively, and, although it does not act as a specific, it prevents the serious complications and invariably renders the attack mild. Moser of Vienna has discovered a new serum which has given good results in a series of 400 cases.

The care of the *nose and throat*, and eventually of the *ears* will require all the skill of the medical attendant, and by commencing early in the case to give careful and constant attention to these parts we may prevent much trouble and danger later on. The attendant should use a small atomizer filled with warm water containing sodium bicarbonate (gr. xv–℥j—0.975–32.0). If decided inflammation should occur, a solution of hydrogen peroxid and cold water or glycerin (1 : 5) may be used, and then be followed by an oily preparation, such as liquid albolene containing menthol (a 5 per cent. solution).

If the patient cannot tolerate an atomizer, an application of the antiseptic oil directly to the posterior nasal spaces, by means of an aluminum applicator, may be made. Faithful attention to the removal and disinfection of the secretion from the nose and throat will prevent accumulation, and thus prevent regurgitation up the Eustachian tube with its associated ear-troubles. In this way diphtheria can also be prevented from gaining its full lodgement. For the appropriate treatment of this complication the reader is referred to the treatment of Diphtheria. It has been shown that the return cases (after return from hospital) are caused by the discharges from the nasal and aural passages. If pain in the ear should indicate the extension of the trouble up the Eustachian tube, we must redouble our efforts, even though the desquamation within the tube itself may be quite beyond the reach of our detergent wash.

The external auditory canal may become blocked by desquamating epithelium, and this must be removed by gentle sponging. If the tension of the ear-drum becomes very great, it must be punctured. The method of dropping laudanum and sweet oil in the ear is objectionable, as it serves as a nidus for a collection of dust, dirt, and dead epidermis.

Moser's antistreptococcic serum has been employed with doubtful results, but it should be used in streptococcic pseudomembranous affections.

Scarlatinal synovitis I have encountered in but a small proportion of cases, and then it was of a transient character. I am inclined to attribute this fortunate result to the faithful use of daily bathing and inunctions, long continued and at least until after completion of desquamation. Widowitz recommends the administration of from 1 to 8 gr. (0.06–0.5 gr.) of hexamethylenamine (urotropin) to prevent nephritis. Preisich administered the drug in 600 cases, nephritis occurring in 9.16 per cent., as against 13.6 per cent. of the cases in which the remedy was not used.

The specific poison of scarlet fever is peculiarly obnoxious to the kidneys, and is largely eliminated through them; and upon this fact hinges the scientific part of the treatment of this disease. Free bathing has the happy effect of vicariously eliminating the poison in a measure at least. In post-scarlatinal uremia venesection supplemented by saline infusion produces excellent results. (For the treatment of nephritis, see Diseases of the Kidneys, p. 1025).

"FOURTH DISEASE."

(*Duke's Disease.*)

This complaint was first described by Clement Duke[1] in 1900. The so-called "fourth disease" resembles both German measles and mild scarlatina. The *etiology* is obscure. The incubation period is from one to three weeks, and the time of transmissibility ranges from two to three weeks. *Prodromata* are often absent, though a slight febrile movement may precede the eruption by from six to twenty-four hours.

Cotton states that catarrhal symptoms of the faucial, oral, or ocular mucosa may rarely be present. Usually the postcervical and occipital lymph-nodes are palpable early in the attack. The *eruption*, which resembles that of scarlatina, appears first on the face or neck and spreads downward, quickly covering the trunk and portions of the extremities. Itching is absent, and the rash fades rapidly after two or three days without stain.

A fine, branny desquamation follows the disappearance of the eruption. The pulse is accelerated in proportion to the febrile movement, which is marked, but lasts only two or three days. Complications are rare, while sequelæ almost never occur.

The *discrimination* of Duke's disease from scarlatina is made with readiness by noting the absence of vomiting, of a pulse-rate out of proportion to the fever, the strawberry tongue, lamellar desquamation, and of the characteristic oronasal pallor. In *rubella* the rash is light in color and presents patches of irregular shape (*vide* p. 221). Corlett and Cole[2] state that aberrant forms of scarlatina should not be regarded as distinct affections, and that the consensus of opinion does not substantiate the claim for a fourth disease.

The *prognosis* is favorable and *treatment* that of mild scarlatina.

MEASLES.

Definition.—An acute contagious disease, characterized by an initial coryza, general catarrhal symptoms, fever in the earlier stage, followed by a peculiar papular eruption on the face and body.

Pathology.—In uncomplicated measles we have no pathologic lesions. The only post-mortem changes found, as a rule, are those of catarrhal pneumonia and acute nephritis. All the internal organs are gorged with blood, and minute hemorrhages are found on their surfaces. The skin presents the following histologic lesions: focal necrosis, with the formation of small vesicles, isolated necrotic epithelia, diffuse perinuclear vacuolation of cells of the epidermis and of the dermal glandular structures, with congestion, edema, swelling, proliferation of the endothelial cells, and a moderate increase of the large round-cells (Ewing).

Etiology.—Measles occurs in epidemics, although sporadic cases are common in the larger cities. There is an epidemic prevalence in large centers of population every eighteen months or two years, but the different epidemics vary in their extent and fatality. It generally happens that when once the disease enters a home, street, or small court, scarcely any

[1] *Lancet*, July 14, 1900. [2] *Jour. Amer. Med. Assoc.*, July 16, 1910.

one escapes who has not been protected by a previous attack. The susceptibility to measles in children is very great, except in the newborn, who seldom contract the disease. Biedert[1] found that only 14 per cent. of unprotected children escaped. In the Faroë Islands, under similar conditions, only 1 per cent. escaped (Madsen, Pannum). There is the same experience in schools and hospitals. The *epidemics* occur mostly in the fall and winter, yet the season has little influence. The poison is conveyed principally by *contagion*, less commonly by *fomites*. It is probably most contagious during the initial catarrhal stage.

Bacteriology.—Micrococci, *e. g.*, *streptococci*, are found in the secretions of the respiratory tract, but they have not been proved to be specific. Czajkamski[2] described motile bacilli, 2.5 to 5 micromillimeters in length, which did not color by Gram's method. They grew on glycerin-agar, bouillon, and blood-serum. Schottelius[3] found the *Staphylococcus pyogenes aureus* frequently in 40 cases of measles conjunctivitis, while in 40 fatal or very severe cases he found the streptococcus in 50 per cent. in the lungs and spleen. J. Goldberger and J. F. Anderson[4] have discovered an infective virus.

Immunity.—One attack of measles almost always exhausts the soil, but in exceptional instances recurrent attacks may occur.

Clinical History.—The period of **incubation** is from seven to fourteen days, and in inoculated cases from seven to ten days.

Catarrhal Stage.—The early symptoms are those of a *cold* with some *fever*. The child has marked coryza, watery eyes, sneezes, and has a dry, croupy cough. Frequently the symptoms are those of a *catarrhal laryngitis* and *bronchitis*, the fauces and tonsils being hyperemic, with abundant secretion, and there is conjunctivitis. The patient may be acutely ill, the *temperature* rising several degrees in the evening, and falling slightly in the morning; the fever continues high until the rash is fully developed. The *rash*, consisting of one or more distinct *papules*, may be seen on the hard palate fully twenty-four hours before it appears on the face. A transient prodromal eruption, which may be erythematous, truly scarlatiniform or urticarial, may rarely be observed.

The eruptive stage is very characteristic, and usually makes its appearance at the end of the *fourth* day. The neck, face, forehead, and trunk receive the eruption in the order of mention. The whole physiognomy of the child is so characteristically altered that a well-marked case may be diagnosticated at a glance. The face is flushed; the eyes are red and watery; a short, dry cough, frequently metallic in ring, is present; and the nose and cheeks are covered with crops of dusky-red papules surrounded by a zone of erythema which sharply contrasts with the normal skin between the patches. The rash on the face is both *discrete* and *confluent*, or may be arranged at times in small crescents, and in the course of a day or two the whole trunk is invaded, but in a slighter degree. By the fifth, and seldom the sixth day, the eruption has reached its height, and commences to fade, first on the face and neck, then on the body and limbs, followed by a *fine desquamation*. By the seventh or eighth day the rash is nearly gone, leaving a blue,

[1] *Jahrbuch für Kinderheilkunde*, vol. xxiv., p. 94.
[2] *Centralblatt für Bacteriologie*, vol. xviii., Nos. 17 and 18.
[3] *Münchener med. Woch.*, March 1, 1904.
[4] *Jour. Amer. Med. Assoc.*, Sept. 16, 1911.

mottled stain over the body. The temperature, which has reached 103° F. (39.4° C.) or even 105° F. (50.5° C.), falls when the rash is fully established—*i. e.*, on the fifth or sixth day—while the headache, the severe cough, and general features also subside with the fever. If the temperature continues high after the rash is out, we may look for some complication, such as pneumonia or acute nephritis (*vide* Fig. 18).

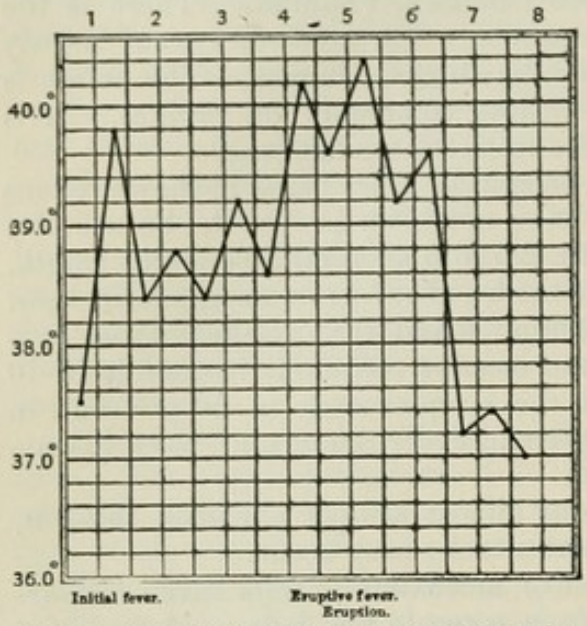


FIG. 18.—Temperature-curve of a case of measles.

An eruption first described by H. Koplik also occurs on the buccal and labial mucous membrane; it appears "as long as twenty-four hours, forty-eight hours, and even three to five days before the appearance of the skin exanthem." It is present before the signs of conjunctivitis appear, and when little or no fever is present. It was found in fifty-two consecutive patients in Koplik's clinic. This eruption consists of small, irregular spots of a bright red color, and in the center of each red spot is the interesting sign which Koplik has described, a minute, bluish-white speck. To see the latter requires a strong, glaring daylight, and they must be looked for by everting the mucous membrane of the lips and that of the cheeks. The spots may be few, and again they may be quite numerous. Widoweiz[1] and Cotter[2] fail to find the spots in about 10 per cent. of the cases, while Lepit[3] in an analysis of 327 cases, found them almost always present. Among recent writers, J. L. Hirst, C. C. Ross, and Rolly attach great value to Koplik's sign in the diagnosis. By recognizing this sign, measles patients may be early quarantined and institutional epidemics checked.

Complications.—In some epidemics the character of the disease is very severe, being marked by high fever (105° to 106° F.—40° to 41° C.), a dry, brown tongue, delirium and convulsions, and feeble heart-action, due to the intense hyperemia of internal organs—lungs, brain, kidneys, etc. The eruption may be petechial—*hemorrhagic measles.*

The main complications are presented by the lungs. The accompanying bronchitis tends to extend to the bronchioles, causing *broncho-pneumonia;* this occurred in 50 among 457 cases, 11.8 per cent. (Landis). The extent and seriousness of this complication are largely dependent upon the degree of the previous debility. *Lobar pneumonia* is rare.

Catarrhal or *membranous laryngitis* is frequent in the pre-eruptive stage or as a sequela. Quite rarely edema of the glottis occurs. *Ophthalmia* may occur in anemic and strumous children if strict eye-toilet

[1] *Wiener klin. Woch.*, 1899, No. 37.
[2] *Archives of Pediatrics*, Dec., 1900.
[3] *Med. Mod.*, 1899, No. 76; *Rev. Mens. des Mal. de l'Enf.*, July, 1900.

is not enforced. *Glandular involvement* may take place in the cervical glands. *Otitis* is frequent during desquamation, suppuration taking place in the middle ear and *meningitis* is rarely observed. *Cancrum oris* and *noma pudendi* may appear as complications of the disease. *Diarrhea* is frequent at the end of the eruptive period and as a sequela.

The health of the child often remains impaired for a long time after an attack of the measles: it is at this period that whooping-cough, diphtheria, nephritis, and, later on, *acute tuberculosis*, may arise. Tuberculosis very frequently gains entrance into the system from the existence of enlarged and cheesy bronchial and mediastinal glands. Biehler found pyelitis or pyelonephritis in 9 out of 147 cases. Nervous sequelæ rarely occur (hemiplegia, paraplegia). Certain eruptive diseases are rarely *concurrent*, as scarlet fever, variola, chicken-pox, and rubella. The cutaneous tuberculin reaction is absent for about one week in measles (von Pirquet).

Diagnosis.—Epidemics may be characterized by irregular forms of the disease, and the diagnosis of sporadic cases is often very difficult. We cannot recognize it by its dermal lesions, but by the prodromal symptoms, by the fall of temperature after the eruption is well out (differing here from scarlet fever), and by the character of the pulse, tongue, and desquamation. Koplik's early sign is usually present and is distinctive. For two or three days prior to the eruption the leukocyte count is increased (Hecker). During the latter part of the pre-eruptive and early part of the eruptive period, a distinct leukopenia occurs. A feverish period of four days, associated with catarrhal symptoms of the eyes, nose, and upper air-passages, a few papules on the hard palate, followed within twenty-four hours by a papular efflorescence on the face, will differentiate the disease from *variola*, *varicella*, *scarlet fever*, and *rubella*.

The accompanying table from Rotch will aid the discrimination:

	Measles.	Variola.	Varicella.	Scarlet Fever.	Rubella.
Incubation . . .	10 days.	12 days.	17 days.	4 days.	21 days.
Prodromata . .	3 days.	3 days.	A few hours.	2 days.	A few hours.
Efflorescence . .	Papules.	Macules. Papules. Vesicles. Pustules.	Vesicles.	Erythema.	Papules.
Desquamation .	Furfuraceous.	Large crusts.	Small crusts.	Lamellar.	
Complications and sequelæ . . .	Eye and Lung.	Larynx. Lungs.		Kidney, ear, and heart.	

The mortality differs according to the surroundings of the patient. In healthy children under favorable environment the mortality is practically *nil*, while in tuberculous and wasted children it is very large, this being especially due to complications and sequelæ. Infants may be born with the rash on them.[1] The disease is quite fatal when it follows other acute infections (*e. g.*, scarlatina).

Treatment.—Measles is a self-limited disease, and we are unable to shorten its duration, nor is there any means of producing immunity from the attack. The treatment is necessarily symptomatic; hence our efforts should be directed toward protecting the various organs that are most likely to become involved by complications, remembering at the same

[1] *Hem. Med. Chronicle*, May, 1890; *Brit. Med. Journal*, vol. i., p. 612, 1890.

time that the nose, ears, eyes, and throat are involved during the feverish stage, and that the skin is in a very susceptible condition.

The patient should be placed in a large, dark, well-ventilated room, with a uniform temperature between 68° and 70° F. (21.1° C.). He should remain in bed until the temperature has been normal for one week, and until the efflorescence has nearly faded and the desquamation is almost complete. The diet during the period of fever should be milk, bread, and light soups. Near the end of desquamation, if all symptoms are favorable, a more generous dietary may be allowed.

The bronchial cough, which may be very troublesome during the first few days, can be readily relieved by some simple expectorant and fever mixture, as—

℞. Potassii citrat., ℥ss (16.0);
Succi limonis, ℥j (32.0);
Tr. opii camph., ℥ij (8.0);
Syr. ipecac., ℥ij (8.0);
Syr. tolu., q. s. ad ℥ij (64.0).—M.
Sig. ℥ss–℥j every two or three hours, according to the age and condition of the patient.

For the coryza I have found that atomizing the nares with some oily vehicle (oleum petrolatum album, etc.) is advantageous.

The skin is in a state of great irritation, and from the commencement of the disease until the end of desquamation a daily warm bath (95° to 100° F.—35° to 37.7° C.) should be given the patient, and, after drying the body, cocoa-butter thoroughly rubbed over the entire surface. The child should live in an equable temperature for at least three weeks. For months he should be protected from sudden atmospheric changes in order to avoid respiratory troubles. If he be predisposed to tuberculosis, cod-liver oil and creosote should be prescribed for a period of two months or more. Otitis media may be avoided by frequent cleansing of the post-nasal spaces during desquamation.

Chatinière has suggested the treatment of measles by red light, simply hanging red curtains over the windows and other sources of light, and exposing the patient to these rays.

RUBELLA.

(*Rötheln; Rubeola Notha; German Measles; French Measles*).

Definition.—An acute contagious disease. It has no prodromal stage, and is characterized by slight fever, enlargement of the post-cervical glands, and an efflorescence upon the skin.

Etiology.—Rubella was not distinguished from measles and scarlet fever until about the middle of the eighteenth century. Since then considerable controversy has arisen at different times as to its nature, the theory being at one time strongly advanced that it was a combination

of these two diseases, as many of the milder cases have symptoms common to both. That there is a difference, however, in the character and course of these diseases has been proved beyond doubt to careful observers by the facts that rubella occurs independently of either measles or scarlet fever; that contagion from this disease produces a similar disease; that one attack affords immunity to subsequent seizures (although out of a total of 719 cases, second attacks were seen in 2.5 per cent.—Tobeitz); and that its onset and clinical course are characteristic.

Rubella may occur epidemically or sporadically. Although of undoubted microbic origin, the specific organism has not been isolated.

In hospitals or where persons are crowded and living under unhygienic circumstances the disease is very *contagious*, though probably less so than measles, and the epidemic will be quite general; but in family practice it is but slightly so, and the epidemics are limited, often being confined to a single household and attacking perhaps but one or two of the family. As compared with measles, the incidence shows a larger percentage of adults. As stated by Edwards, it is spread by the *cutaneous exhalations*, *breath*, *fomites*, and *clothing*, and is probably contagious from the period of incubation until far into convalescence.

Clinical History.—The **incubation** stage lasts from ten to sixteen days, though this period may vary and the disease appear three or four days after exposure. On the other hand, cases have been reported in which it was as long as twenty-five days. As a rule, the period of incubation is longer perhaps than in measles. The *stage of invasion* covers from one to three days.

For a period of a few days before the rash appears there may be noticed chilliness, pains in different parts of the body, a dull, heavy feeling, perhaps feverishness, sore throat, enlarged tonsils, and a slight bronchitis. Enlargement and induration of the postcervical glands is constant, while the anterior are also commonly enlarged.

Just before, or with the appearance of, the rash there is a rise in *temperature* to 99° or 100° F. (37.7° C.), or in severe cases as high as 103° F. (39.4° C.) or more. Again, the invasion symptoms may be absent or so mild as to escape notice, and the first sign of infection be the appearance of a *rash* which first shows itself on the face and extends downward over the body. In some cases the eruption does not follow the regular course, and is confined to one part of the body, and cases have been reported in which it only appeared on the roof of the mouth or on the tonsils. In other instances every part of the body, including the palms of the hands and the soles of the feet, may be covered.

The eruption consists of *papules*, is multiform, confluent, and of a pale or rosy-red color. The patches do not assume any regular shape or form, and the skin between them may become hyperemic and cause itching. The rash reaches its height on different parts of the body in succession, fading in one part while appearing in another. Its duration is from two to five days, and possibly longer in some cases.

A slight *desquamation* usually occurs, and a slight pigmentation of brownish color after the rash fades is frequently noticed, disappearing after a few days. The *temperature-curve* is variable, but as a rule it remains between 100° F. (37.7° C.) and 102° F. (38.8° C.) while the eruption is present. As mentioned above, sore throat is nearly always

present, with enlarged tonsils, a dry cough, and bronchitis. The glandular enlargement will also continue with the rash, and in severe cases the axillary and inguinal glands may become involved. The *pulse* varies with the temperature and respiration. Vomiting has been noticed as occurring during the eruption in severe cases.

After a period varying from three days to a week, with the disappearance of the rash, convalescence begins and the child rapidly regains its former health, and the whole course of the disease may be so mild that the patient cannot be persuaded to remain in bed.

Complications.—The most common are affections of the *respiratory tract* (pneumonia or severe bronchitis), and in some cases we have a *gastro-intestinal catarrh* of a troublesome character. *Diphtheria* or other contagious diseases may occur. A *relapse* is not uncommon, and may be as severe as the initial attack.

Diagnosis.—Rubella may be distinguished from *measles* by its less severe onset and course, by the absence of coryza, severe bronchitis, high fever, Koplik's spots and complications, by the lighter color, shorter duration, and more diffuse character of its rash, and the irregular shape which the patches assume. The presence or absence of an epidemic is an important factor in the diagnosis, and in cases occurring when there is no epidemic the diagnosis between this disease and measles of a mild type is difficult if not altogether impossible.

From typical *scarlatina* the diagnosis offers no difficulty. The absence of its initial vomiting, the strawberry tongue, the character of the rash (which in scarlet fever is erythematous), and the shorter duration and milder course of rubella, all render the diagnosis easy.

Rubella.	Erythema.	Urticaria.
Occurs first on the face.	On the hands and feet.	In wheals on arms and legs.
Enlargement of cervical glands.	No enlargement.	No enlargement.
At first no itching.	Burning pain.	Intense itching.
Contagious.	Not contagious.	Not contagious.
Microbic origin.	Reflex origin.	Gastric origin.

The **prognosis** in uncomplicated cases is invariably good but when the surroundings are unhygienic, or in cases in which the child has been delicate previously, it is more serious. Complications, especially pneumonia or diphtheria, may prove fatal, and in some cases the mortality reported has been as high as 9 per cent.

Treatment.—The treatment is simple and principally symptomatic. A mild cough-mixture, such as is recommended in measles for the bronchitis, nutritious but easily digested food, and medicine to regulate the bowels when necessary, fulfil all the indications for internal medication. As in measles, cool sponging should be resorted to before and during the rash; and, when the fever is high, a cool tub-bath, where practicable, will be found to reduce the temperature, quiet the patient, and hasten the appearance of the eruption. During convalescence, if the child does not rapidly regain his appetite and strength, tonics, such as tincture of nux vomica and syrup of hydriodic acid, are indicated.

The complications are to be treated as they arise, but the sponging should not be discontinued until the temperature becomes normal.

WHOOPING-COUGH.

(*Pertussis; Tussis Convulsiva; Keuchhusten.*)

Definition.—Whooping-cough is a highly contagious disease, characterized by a catarrhal inflammation of the respiratory tract, associated with a peculiar spasmodic cough, ending in a whooping inspiration.

Pathology.—There is no lesion that can be considered characteristic of whooping-cough, and none around which all the symptoms and complicating conditions are grouped. In the beginning there is catarrh of the nasopharynx, and this may be the only change coincident with the development of the characteristic cough. In advancing cases this nasopharyngeal catarrh becomes generalized by extension to the lachrymal ducts, the conjunctivæ, the Eustachian tube and the middle ear, to the glottis, trachea, large and small bronchi, and the air-vesicles. The more decided pulmonary lesions—emphysema, pulmonary collapse, pulmonary congestion and edema, and bronchopneumonia—are advanced pathologic conditions accompanying the later stages (W. W. Johnston).

The *postmortem* table does not give as much information as to the pathology except as to the sequences of the disease. In the early stages swelling and redness of the respiratory and digestive tracts will be found, together with a large quantity of viscid mucus.

Etiology.—The disease occurs in *epidemics*, yet occasionally may appear *sporadically*. Pertussis seems to have a tendency to occur in epidemics every two years, although in large cities the disease is generally endemic. Pertussis is directly *contagious*, though scarcely so in houses and school-rooms, unless it be for those of a specially susceptible nature. It is possible, however, for the disease to be propagated in schools, though not to the same extent as measles and scarlet fever. It seems that a more decided and prolonged personal contact must be made, as with members of a family, to ensure transmission. One close exposure in a susceptible child may be sufficient to ensure an attack. The germs seem to be located at first in the secretions of the respiratory tract, and are thus disseminated through the air, the disease being most highly contagious, therefore, during the paroxysms of coughing. Goodhart reports a case in which a third party conveyed the disease from one child to another, thus suggesting that the contagion is ponderable.

Predisposing Causes.—The influence of the *seasons* does not seem to have any effect, though perhaps fall and spring are the more frequent periods; the station in life, whether hygienic or unhygienic, does not modify the disease. *Bad ventilation*, however, may propagate the disorder, and cause additional cases by favoring the increase of germs in the immediate surroundings. The *previous condition of health*, especially of the respiratory mucous membrane, seems to possess some predisposing influence, weak, delicate children, with an irritable digestive tube associated with a catarrhal state of the respiratory passages, more readily contracting whooping-cough than those in robust health.

There is an intimate association between whooping-cough and *measles;* epidemics of measles are often followed by whooping-cough in the same sufferers. This is possibly due to the sensitive condition of the mucous membrane left by the measles, which is so favorable to the lodgement of the germs of pertussis; and the association of the two diseases must be more than

accidental. There exists a certain individual susceptibility, which, however, is not universal to whooping-cough, as well as to other infectious diseases.

Age exercises some influence on the development of whooping-cough, most cases occurring before the tenth year; after this time the frequency of the disease rapidly diminishes. West states that one-half of all cases develop under three years, but he must have based his knowledge upon an experience in hospitals and children's homes, as the experience of others does not sustain his statement. The disease occurs in adults but rarely, this being due partly to the fact that so many have suffered from it while young, and partly to a lessened susceptibility. It occurs frequently before the first year, and when it does it is the most fatal of all the diseases of childhood (Goodhart).

The *sexes* are about equally divided as regards susceptibility; many writers, however, seem to think that girls are more liable.

The *highway* of the contagion of whooping-cough into the system is evidently the respiratory tract, though this fact needs confirmation. Published cases of pertussis in the newborn would even seem to make its transmission possible through the fetal circulation, yet the reports are neither numerous nor satisfactory. One attack is usually protective for the rest of life, although exceptions to the rule may be found.

Nature and Bacteriology.—The true nature of whooping-cough has been thoroughly discussed, but is not, as yet, fully settled. Many writers claim it to be a simple bronchitis due to "cold," associated with a certain nervous habit or mimicry. The cough is started by the bronchial irritant and soon tends to become a habit, thus returning again and again, until it dies out in the oblivion engendered by more healthy and regulated discharges of nervous energy (Goodhart). It has been held that the disease is a lesion of either the pneumogastric, phrenic, sympathetic, or recurrent laryngeal nerves, or perhaps even of the medulla. If this ground be valid, whooping-cough is simply a neurosis. Eustace Smith says it is caused by the pressure of the enlarged tracheal and bronchial glands upon the terminal filaments of the pneumogastric nerve. Whatever the direct cause, the contagious character of whooping-cough, its appearance in epidemics, its incubating period, and the possible immunity from subsequent attacks, seem to prove beyond argument that it should be classed among purely infectious diseases.

Bacteriology.—Linnæus (to quote Dr. J. P. C. Griffith, in the *American Text-Book of Diseases of Children*) attributes pertussis to the presence in the nose of larvæ of insects. The researches of Afanassieff in 1887 have attracted much attention. This observer isolated a short bacillus, which he named the *bacillus tussis convulsivæ*, and of which he was able to obtain pure cultures upon various media. Animals inoculated upon the respiratory mucous membrane with these cultures exhibited some of the symptoms of the disease and developed catarrhal conditions of the respiratory tract, with a tendency to bronchopneumonia. These observations have been confirmed by others, and a toxin has also been reported as present in the urine of patients suffering from pertussis which is identical with that produced by Afanassieff's bacillus. Kuoloff believes that the parasite of whooping-cough is a specific micro-organism, a protozoon, and has found uniformly in the fresh sputa of patients ameboid organisms with spheric spores characterized

by concentric laminations.[1] Czaplewski and Hensel describe a short bacillus with distinctly staining rounded ends, and commonly occurring in pairs. It is found in sputum, both free and in pus-cells, increasing as the disease advances. It can be obtained in pure culture; and grows on any ordinary medium except potato. It resembles Koplik's bacillus. The latter is facultative-anaërobic. The organism is not found in the sputum during the prodromal stage.[2] Arnheim[3] found in fatal cases of pertussis Czaplewski's pole bacteria, which he was able to cultivate and which were pathogenic to white mice. Jochmann and Krause[4] have recently isolated a specific microbe and called it the *bacillus pertussis eppendorfii*, but their claims have not as yet received full confirmation. The careful investigation of Myer-Huni and of von Heroff indicate that the catarrhal inflammation is especially pronounced on the posterior wall of the larynx in the interarytenoid region, the so-called "cough region." Undoubtedly we have in whooping-cough an infectious catarrhal process which affects the mucous membrane controlled by the superior laryngeal nerve. Hence the value in many cases of purely local treatment.

The nature of the "whoop" has been frequently discussed to show the nervous origin of the disease, yet the infantile larynx is capable of responding to purely neutral stimuli owing to the flexible nature of the young cartilage. If we carry a young, sleeping child from a warm room out in the cool air, the same characteristic whoop may be produced.

Clinical History.—The period of **incubation** varies from four to fourteen days, according to the extent of catarrhal trouble in the child existing at the time. Goodhart gives several authenticated cases in which the incubation ended on the eighth day. In the beginning the symptoms are those of a slight bronchial cough, which has a tendency to be more pronounced during the night. After a few days the cough assumes an influenzal character, and at the same time it gradually grows metallic in ring and shows a laryngeal type. There is some fever present. There is a pronounced leukocytosis, with preponderance of the lymphocytes. This **catarrhal** or **feverish** stage lasts for a week or more, when it is followed by the **paroxysmal stage**, and these stages are divisions of the symptoms worthy of recognition, as the treatment in the first is not applicable to the second. Many authorities speak of a third stage as one of **decline**, which does not sharply occur, but includes the sequence of the disease. The **catarrhal stage** lasts about one week or ten days, during which the child is ill at ease, is feverish, and has a hoarse, dry cough. The symptoms may either be entirely laryngeal at first or bronchial, with a loss of appetite and broken rest at night. Auscultation at this time will reveal a few moist or dry râles in the larger bronchial tubes, but there is very little secretion. The cough seems to be out of proportion to the physical signs. As the catarrhal stage proceeds the cough commences to indicate its character by becoming more noisy, increasing especially at night. The physiognomy of the child commences to change, the face is swollen, the eyes suffused and watery, the under lids swollen and pink in color. This is one of the most decisive indications of the trouble, and may be recognized by a careful observer a few days before the "whoop" begins which stamps the disease and ushers in the second stage. The

[1] *Medical News*, Nov. 9, 1906.
[2] Saunders' *Year-Book* for 1899, p. 696.
[3] *Berlin. klin. Woch.*, Aug. 6, 1900.
[4] *La Semaine Medicale*, Aug. 21, 1901.

commencement of the **paroxysmal stage** is quite different from the easy and more constant coughing of the first stage. If the child is in bed, the onset of a paroxysm is usually quite sudden, but if he is up and playing, there is a period of restlessness, a premonition of the coming storm similar to the aura in epilepsy, and the child may even have time to run to his mother or nurse before the paroxysm comes on. Usually the paroxysms are induced by a quick inspiration, as during drinking, eating, or crying. The first (expiratory) part is short, and followed by a short whoop; this is very quickly followed by a long series of short expiratory efforts and a second and longer whoop, when the paroxysm may cease. In some cases a third and a fourth may quickly follow, until the child is quite exhausted. The paroxysms, whether short or long, generally terminate with vomiting or eructation of a quantity of stringy mucus. Food is ejected, and in most cases a little blood is mixed with the vomited mucus.

At this stage of the disease, if at all severe, the *countenance* of the child is characteristic, and so much so that a mistake is no longer possible: the features are swollen, puffy, and dusky in color; the eyes are injected, the lids swollen and pink; the skin livid, due to a minute ecchymosis of the smaller capillaries. In many cases there will be extravasation of blood beneath the conjunctiva, due to the violence of the congestive cough. If the chest be examined at this stage, it will tell but little, provided we have no broncho-pneumonia, though a few moist râles may be found scattered through the larger tubes.

The spasmodic stage of whooping-cough has no set *duration* and varies frequently in intensity. In severe cases it may consist of twenty to forty paroxysms during the twenty-four hours. Some spasmodic coughs are not accompanied by a whoop, and the absence of this sign may be noted in very young children, as well as in those that are very ill with broncho-pneumonia. Some children vomit after a coughing spell without the whoop.

It is frequently observed that long after the spasmodic spell has come to an end the paroxysms return again and again, perhaps years afterward, with almost characteristic features, evidently acting under the stimulus of some perfectly neutral catarrh.

Complications.—In severe cases the complications may be numerous. *Epistaxis* often occurs in children; *hemoptysis* when vomiting is frequent; *ulceration* of the frenum linguæ in violent coughing; *convulsions* in vigorous children; and *broncho-pneumonia*, *pleurisy*, *pericarditis*, *laryngitis*, and *hernia* in severe, prolonged coughing. Convulsions and broncho-pneumonia are alarming, and in young children a *profound stupor* may replace the convulsions. Eshner[1] states that peripheral neuritis is a rare complication of this disease. Interlobular emphysema has resulted from whooping-cough (Finch).

Sequelæ.—*Acute nephritis* frequently occurs, and is as severe as that found in scarlet fever. In a series of over 200 cases I have found the kidneys affected in 20 per cent. Knight found albuminuria in 66 out of 86 cases examined. *Emaciation* is a very important sequence of pertussis. All the viscera are liable to fatty degeneration, and nutritional changes open the door to cheesy, glandular altera-

[1] *American Medicine*, June 21, 1902.

tions, and eventually to a secondary tuberculosis. Atelectasis, by curtailing lung space, frequently brings about a general collapse, and this condition frequently explains the flattened chest found in young adults. Conversely, emphysema may be initiated by pertussis early in life. Emaciation may also be due to *mucous disease*, a chronic gastro-intestinal catarrh of long standing.

Prognosis.—Associated with its complications, pertussis is a very fatal disease, especially in children under two years of age. Dolan regards it as third in rank among the fatal diseases of England, where the death-rate per million is five thousand annually. The deaths occur chiefly among children of the poor and in bottle-fed infants.

Goodhart regards whooping-cough as the most fatal of all the diseases in children under one year of age. He places the mortality at 12 per cent., and thinks that this is not too high; his statement, however, is hardly warranted, as he includes the deaths from the many sequelæ which we cannot estimate. Ashby and Wright place the mortality at 7.6 per cent.

Differential Diagnosis.—Young infants usually do not "whoop," but cough spasmodically. Children with *pleurisy* or *pneumonia* do not whoop, yet we diagnose whooping-cough by the preceding catarrhal fever. From *influenza* in its early stages it is most difficult to differentiate the affection. The pink under eyelid has to me been the most certain sign. When the whoop appears and during the existence of an epidemic, however, the diagnosis may be rendered certain.

The diagnostic points prior to the whooping stage, enunciated by Eustace Smith are as follows: "If a child be made to bend back the head, so that his face becomes almost horizontal, and the eyes look straight upward at the ceiling above, a venous hum, varying in intensity according to the size and position of the diseased glands, is heard with the stethoscope placed upon the upper bone of the sternum. As the chin is now slowly depressed the hum becomes less loudly audible, and ceases shortly before the head reaches its ordinary position." It is true that we do not recognize the hum caused by the enlarged bronchial gland, but it occurs long after other symptoms are manifest.

I have for several years been able to place considerable value on the peculiar puffiness of the mucous membrane of the eyes and the swollen or edematous condition of the whole face and almost dusky color. This condition may exist for days before the catarrhal symptoms have extended throughout the respiratory mucous membrane. The cough at this stage may not be at all suggestive, but purely bronchial.

This symptom of fulness about the eyes suggests *measles*, and must be differentiated from it. As we are able to diagnosticate measles by its appearance first on the hard palate, so we may diagnosticate whooping-cough in its earliest stage by the characteristic swollen condition of the eyes and face. The diagnosis may be confirmed by leukocytosis and the presence of a sublingual ulcer.

Treatment.—The gravity of pertussis is scarcely appreciated either by the general physician or the public, and there is more criminal neglect in connection with whooping-cough than with any other disease.

Hygiene.—Throughout the whole course of the disease out-door life, as far as possible, should be encouraged, and if convenient a sojourn at the sea-shore will shorten the progress of the trouble and limit to a

great extent the number of sequelæ. Only the severe and complicated cases need to be kept in bed. It has been shown that the number of attacks is directly dependent upon the amount of CO_2 present in the atmosphere (Forcheimer). In cities the sufferer must be protected against the dust; this may be accomplished by the wearing of a veil in suitable cases.

Medicinal treatment is exceedingly unsatisfactory, although the therapeutic measures which have been advocated are boundless. The remedies most in use are the antispasmodics and the germicides.

Whooping-cough has a striking parallel in diphtheria, in that it has in its early stages a strong tendency to fasten itself upon the throat. How long this period exists we do not know to a certainty; yet there is undoubtedly a period in whooping-cough, as there is in diphtheria, long or short, in which the virus—if it could be recognized—could be destroyed and the disease terminated. To abort cases thus within two weeks is not unusual, and this explains the number of reported cures made by germicidal remedies.

I have notes of 2 recent cases in which the characteristic whoop commenced at once with the general catarrhal symptoms, and was cut short by a hydrogen-peroxid gargle. These 2 cases illustrate very clearly the fact that the germs of the disease will locate on the mucous membrane of the respiratory passages and bring about a nerve-discharge which ends in the characteristic whoop. In my treatment of this disease I find the greatest necessity for recognizing the affection early in the catarrhal stage. We must remember that the two stages are not sharply defined, and that either the one or the other may be lacking.

The drugs I have found most efficient in the catarrhal stage have been hydrogen peroxid for sterilizing the naso-pharynx, and belladonna and asafetida for the paroxysms.

To be more explicit, I will detail the methods of procedure in a family in which I have instituted my plan of thorough treatment: A child of four years attending kindergarten was brought to me with a suspicious cough. The history was given of an exposure of over two weeks prior. The child had coughed for a few days, more at night than in the daytime; was feverish during the evenings; showed slightly swollen eyelids, thus suggesting the nature of the impending trouble. I ordered hydrogen peroxid and pure glycerin in equal parts, which were well diluted and thoroughly sprayed through the naso-pharynx every four hours. The diet was light and digestible; out-door life was encouraged, except on windy days. At night the child was placed in a large, well-ventilated room, and over its cot was erected a mosquito netting, so as to prevent any unusual draught—a procedure which I have found highly beneficial. When the cough was fully established and was accompanied by eructations of stringy mucus, I commenced the exhibition of the mixture of asafetida ½ dram (2.0) every two hours. The record of the paroxysmal stage was as follows: The first week averaged six coughing spells per day; the second week averaged ten per day; the third week, four paroxysms; and the fourth and fifth weeks averaged about two paroxysms during the twenty-four hours. When the younger brother, but eight weeks old, commenced to show evidences of the disease, I first used hydrogen peroxid as in the older brother, and immediately followed it with asafetida. The case ended favorably.

My second choice is the tincture of belladonna, exhibited in doses of one drop for every year of the child's life, the doses being rapidly increased until toxic effects are reached. Then I gradually increase the amount as tolerance of the drug seems to be established. In very young children I have obtained good results from the use of a freshly prepared belladonna plaster placed between the scapulæ, and the physiologic action of the drug seems thus to be more constantly maintained. I have gained a decided advantage by an application of a 2 per cent. cocain solution directly to the naso-pharynx in a few bad cases. This treatment, however, does not preclude the use of hydrogen peroxid, which should be continued throughout the catarrhal stage. Irrigation of the nostrils thrice daily with a 1 : 40 carbolic acid solution has proved curative in its effects. Bradt declares that local treatment of the naso-pharynx tends to arrest the syndrome.

Bromoform was resorted to in fully 20 per cent. of my cases, and was a keen disappointment. The coal-tar products, pushed to the toxic limit, modified the disease but slightly. A drug that has almost reached the rank of a specific in my hands is the following: Atropin sulph., gr. j; aqua distil., ℥j. Each drop contains $\frac{1}{480}$ gr. atropin and this dose may be increased drop by drop until the full physiologic effect of the drug has been obtained. If this effect is maintained with the onset of the paroxysmal stage, much time is saved. This outline of the drug-treatment in whooping-cough has reference solely to the catarrhal and paroxysmal stages of the disease. Kilmer has advocated a tightly placed thoracic and abdominal belt, which has yielded great satisfaction. Goodson commends the use at the earliest moment of the continuous inhalation of creasote; he also advocates clearing the lungs of bronchitis as much as possible before using any special internal antispasmodic remedies. Graham advises a trial of the vaccine treatment.

Complications and Sequelæ.—Complications may be avoided by maintaining constantly the alkalinity of the body fluids. Sodium bicarbonate and the various alkaline waters are strongly indicated, and milk should be given in seltzer water.

Passalarqua[1] has employed diphtheria antitoxin successfully in 7 cases; it is especially indicated when bronchial or pulmonary complications exist. Diet of the simplest character and a uniformly quiet life must be maintained.

PAROTITIS.

(*Mumps; Parotiditis; Epidemic Parotitis.*)

Definition.—An acute contagious disease, characterized by an inflammation and swelling of the parotid gland, and occasionally by an involvement of the salivary glands, the testicles, and in the female the mammæ.

Pathology.—Opportunities for post-mortem examinations are rare, leaving in some doubt the pathologic course of the disease; but it probably begins as a catarrhal inflammation of the ducts, involving the

[1] *Rev. Française de Med. et de Chirurg.*, 1905, No. 11.

periglandular connective tissue. The inflammation is seldom severe enough or of such a nature as to produce suppuration.

Etiology.—Mumps is undoubtedly a constitutional or blood-disease with local manifestations. "It is a question," Goodhart says, "with mumps whether this disease shall be placed with the specific diseases or with those affecting the parts or organs with which the symptoms more particularly concern themselves."

The disease is no doubt of *microbic origin*, but the specific organism has not yet been isolated, and, while there has been some reason to believe that it is a bacillus, this has not been proved and is still doubtful. It is highly *contagious*, and at times, usually during the spring and autumn, becomes epidemic. It is communicated principally by the *breath* and *exhalations*, the greatest source of contagion being the salivary secretions. It may, however, be carried by a third person or by fomites, and is most liable to be communicated during the beginning of the attack, although the contagiousness continues until after the subsidence of the febrile symptoms. It occurs mostly among *children* and *young adults*, infants and old persons being rarely affected, while males are more liable than females. One attack usually gives *immunity* from a second attack in the same gland.

Clinical History.—The average period of *incubation* is fourteen days, but it may develop as early as ten or as late as twenty days after exposure. The *invasion* is marked by languor and a temperature from 101° to 103° F. (38.3°–39.4° C.), with possible headache and vomiting; the patient complains of pain at the angle of the jaw, and this is greatly increased if an acid (such as vinegar) is swallowed. With these symptoms is noticed a *pyriform swelling* of the parotid glands, the one on the left side usually appearing first, and the other one soon following. Occasionally cases are seen in which but one gland is involved, or the swelling may begin in both at the same time. This increases gradually until some time between the third and sixth days, involving the other salivary glands and causing marked disfigurement; the swelling fills the depression beneath the ear and extends to the cheek and neck, the most prominent part being just below, and pressing outward, the lobe of the ear. The *salivary secretions* are generally much increased, though there may be the opposite condition of marked dryness of the mouth. When the swelling has reached its height, pressure on the adjacent tissues causes a disagreeable sensation of tension, and chewing, swallowing, and even speaking, are at times painful and difficult. The *skin* over the affected part may be of a pale or of a dull-red color. Ringing in the ears and a dulling of the hearing is common. The *nervous system* may be affected, causing headache and delirium, or a low typhoid state may be present. The *duration* is about one week (six to ten days), after which time the swelling subsides, and by the tenth or twelfth day entirely disappears.

Diagnosis.—The diagnosis is easy, the nature and position of the swelling and the course of the disease being characteristic, while the fact that the tonsils are seldom involved prevents a diagnosis of acute tonsillitis.

Occasionally, however, in the course of *septic infection* or after operations, or owing to the extension of inflammation along the duct

from the mouth, the parotid gland becomes the seat of an acute inflammation at first hardly distinguishable from mumps. The existence of a possible source of infection, and the fact that the gland under these circumstances usually undergoes suppuration, should lead to the recognition of the true nature of the case.

Complications and Sequelæ.—Mumps, as a rule, runs a mild course without any serious symptoms, but occasionally complications arise. The most common of these are *orchitis* in the male, which may be followed by atrophy of the testicle; and *mastitis*, *ovaritis*, or *vulvo-vaginitis* in the female, especially after puberty. These complications appear after the subsidence of the swelling of the glands of the neck, only occasionally developing while the glands are still affected, though cases have been reported in which the disease first manifested itself by involvement of the sexual organs. This complication lengthens the course of the attack and increases the constitutional symptoms, but the rule is complete recovery. *Otitis media* sometimes occurs, and a lesion in the auditory nerve, with more or less deafness (which, unfortunately, may be permanent), has been observed. *Meningitis*, with active brain-symptoms, *facial paralysis*, *convulsions*, *albuminuria*, and *arthritis*, have all been noted in certain cases. Jacob and others report cases of mumps complicated with acute pancreatitis.

Treatment.—The patient should be kept in a well-ventilated room of even temperature, and in bed if the fever is at all severe, and should be isolated from those who have not had the disease. Either hot or cold applications to the swelling will often give relief, and support to the swollen gland by means of cotton and a bandage is very comforting. Saline laxatives may be given, and aconite or some simple fever-mixture at the beginning of the attack is usually indicated. These simple measures are all that are required in an ordinary case, while complications or unusual conditions must be treated as they arise.

TUBERCULOSIS.

Definition.—A chronic (less frequently acute) infectious disease, caused by the bacillus tuberculosis. This organism produces specific lesions, taking the form either of separate nodular masses or diffuse growths, infiltrating the tissues, while aggregations of these elementary tubercles give rise to large tubercular masses. Tubercles undergo caseation and sclerosis, followed in turn by ulceration (in consequence of secondary pyogenic infection), or, more rarely, calcification.

Historic Note.—Prior to the discovery, in the early part of the nineteenth century, by Bayle and Laennec, of the tuberculous new growth as a distinctive body, this disease had been studied chiefly from a clinical point of view. At this early period the disease was believed to consist chiefly of a suppurative process, and in its observation the physician was unaided by auscultation. Later, the tubercle was recognized as a small rounded nodule without any special histologic cha-

racteristics. Villemin in 1865 performed his epoch-making experiments, and the tubercle was no longer distinguished by its anatomic characters alone. Though the theory of the infectious nature of tuberculosis had been previously advanced by Buehl and others, it was first clearly demonstrated by Villemin's beautiful inoculation-experiments upon rabbits and guinea-pigs with particles of tubercular and cheesy substances, producing the characteristic lesions of tuberculosis. It then remained for Koch to discover (in 1881) the specific cause of the most important of all human ills—the tubercle bacillus. So soon as the specificity of the disease was definitely established it became clear that the associated inflammatory processes, that were formerly believed to be primary lesions, were secondary.

Geographic Distribution.—Tuberculosis prevails in almost every quarter of the globe, but is more prevalent in certain latitudes than in others. Thus, in general terms, it may be said to prevail more extensively in warm than in cold countries. Local conditions, however, exercise a more decisive influence in engendering predisposition than mere geographic position. It is of quite frequent occurrence in all densely populated municipalities, and more especially in the overcrowded sections of the latter; this fact explains why the inhabitants of cities of the North are but little less spared than those of the cities of the South. On the other hand, residents of mountainous countries, owing to the purity of the atmosphere and the elevation, are rarely victims.

General Pathology of Tubercular Lesions.—**Distribution of the Lesions in the Body.**—Tuberculous new growths elect, most frequently, the lung, and when the disease occurs in the adult this organ is almost invariably implicated. Next in frequency follow the larynx, intestines, peritoneum, urogenital organs, and the brain. The other chief viscera of the body (spleen, liver, heart, etc., particularly the latter) are less commonly the seat of tuberculosis. In children the lesions exhibit a different distribution, the favorite seats being the lymph-glands, intestines, bones, and joints. In them the distribution corresponds pretty closely, if we except the bronchial and mesenteric glands, to that of surgical tuberculosis.

The Elementary (Nodular) Tubercle.—This may be developed in any tissue to which the tubercle bacillus has found its way, and the presence of the bacillus is its sole distinguishing feature, since apparently identical bodies are produced by other micro-organisms—*e. g.*, certain of the worms (eggs of the distoma), actinomyces, aspergillus glaucus, aspergillus funigatus, and even as a result of irritation by certain foreign bodies (podophyllum). Various forms of pseudo-tuberculosis have been described, but all are due to micro-organisms that differ from the bacillus tuberculosis. Mallassez and Vignal described a form produced by a micrococcus occurring in a zoöglea; this was confirmed by Nocard, Eberth, and others. Charrin and Rogers have described still another form, in which they found bacilli about 1 μ long, actively motile, and growing freely upon ordinary media, but not growing upon glycerin and agar, and not liquefying gelatin.

The various stages in the development of a tubercle are—

(*a*) *Proliferation* of the fixed-tissue elements (connective tissue, endothelium of the capillaries, etc.) of the part infected, due to the local.

specific irritant action of the bacilli. These anatomic elements are transformed into epithelioid and giant cells. The epithelioid cells assume various shapes, chiefly rounded and polygonal; they have vesicular nuclei, and soon show tubercle-bacilli in their interiors. A certain proportion of the epithelioid cells, as the result of increase in their size and a repeated division of their nuclei, or by union of contiguous cells, become *giant cells*. The latter occupy the center of the tubercle, and also contain bacilli, the number of giant cells and of the bacilli being largely reciprocal. Thus, the giant cells are numerous in tubercular lymph-glands, joints, etc., in which the bacilli are relatively few; on the other hand, they are scanty in miliary tubercles, in which the bacilli are numerous—two facts supporting the view that giant cells display phagocytic action. Hektoen asserts that the giant cell is a living defensive agent.

(*b*) About the site of infection a *diapedesis of leukocytes* occurs in the nature of a defensive inflammatory process. At first the leukocytes are of the polynuclear variety and are quickly destroyed; but later mononuclear leukocytes (lymphocytes) appear. These latter resist the action of the bacilli, and I think their true function is a phagocytic one. The various forms of cells described are connected and surrounded by a reticular stroma "formed by the fibrillation and rarefaction of the connective-tissue matrix" (Baumgarten).

The fully-developed tubercles are small, nodular bodies whose diameters range from ½ to 2 or 3 mm. At first they are almost transparent, but soon lose this quality in consequence of the further changes described below. They are avascular bodies, and invariably undergo degenerative changes: (*a*) *caseation* and (*b*) *sclerosis*.

(*a*) *Caseation.*—This implies "coagulation-necrosis"—a destructive process proceeding from the center toward the periphery of the tubercle, and the result of the local action of the bacilli or their toxins. The cells are thus transformed into a uniformly yellowish-gray structureless matter. When the foci are numerous and closely set, fusion may occur, with the production of larger or smaller homogeneous masses (cheesy pneumonia). The latter may soften, resulting in the formation of cavities: this is due, usually, to secondary pyogenic infection, causing ulceration. Less frequently the cheesy masses undergo calcification or become encapsulated, and are then practically harmless.

(*b*) *Sclerosis.*—Preceding and during the time that cell-destruction is going on in the center of the tubercles the protective forces of nature are asserting themselves, though too often without avail. In the first place, hyaline transformation, with conversion of the cellular elements into fibrous tissue occurs. Frequently, now, the center of the tubercle is caseous and contains bacilli, while the peripheral parts are quite hard. Here the bacilli are incarcerated (*latent tuberculosis*). The fibroid change may pervade the entire tubercle. Again, the fibroid element in the tissues immediately surrounding the tubercle may be greatly increased and form new connective tissue, and this process be followed by secondary contraction, converting the tubercle into a firm fibrous nodule. The fibroid change in its completest development is observed in tuberculosis of serous membranes.

In every case of tuberculosis there is a battle for supremacy between

the destructive forces on the one hand and the resisting, conservative forces on the other hand. As mentioned above, limitation of the tuberculous process takes place by fibrous encapsulation. In the majority of instances, however, the bacilli fall upon a receptive, favorable soil, when nature's benign curative means fail and extension occurs by the appearance of secondary tubercles in adjacent tissues. The dissemination and transportation of the bacilli are effected principally through the lymph-channels and blood-vessels, although to some extent also by the phagocytic leukocytes. Again, infection may occur by actual contact of the affected organ with neighboring parts, the disease spreading by continuity. Lastly, lesions may be propagated by the movement of organs; thus localized peritoneal tuberculosis may rarely become generalized in consequence of the peristaltic movements.

Again, fusion of minute centers of infection or of miliary tubercles results in the formation of larger nodules or areas, which lead by a process of local extension to *diffuse tuberculous infiltration* (gray infiltration of Laennec). An entire lobe may become similarly involved (tuberculous pneumonia), and "there may also be a diffuse infiltration and caseation without any special foci, a widespread tuberculous pneumonia induced by the bacilli" (Osler).

The term "gray infiltration" is misleading, since the morbid changes differ in no essential manner from those described as occurring in the miliary or nodular tubercle. Moreover, the latter also presents a grayish appearance. The apparent difference between a miliary tubercle and diffuse tubercular infiltration lies in the fact that the latter displays a clearer tendency to spread by direct extension.

Associated Inflammatory Processes.—The tubercle bacilli excite associated inflammatory processes in the organs affected, and if the tuberculous lesions run a slow course a limiting wall of true fibroid induration circumscribes the area involved. By means of this induration the natural protective forces, either temporarily or permanently, check the progress of the local lesions, and the change is strictly analogous to the sclerosis that takes place in the peripheral parts of the elementary tubercle or immediately surrounding the latter, as in tuberculosis of serous membranes. On the other hand, when the tuberculous infiltration is less tardily developed the collateral reactive inflammation may show changes similar to those of catarrhal or croupous pneumonia (*vide supra*). Examination of the sputum, to determine the nature of a mixed infection, is of little value, since the sputum may show various organisms that have not caused any real infection, and that have entered the sputum from the throat or buccal cavity.

Etiology.—The Specific Cause and its Physical Characteristics.—In 1881, Koch discovered the tubercle bacillus, which is the sole cause of the disease. The bacillus is rod-shaped, straight or somewhat bent, and slender, its length equalling about one-third or one-half of the diameter of a red blood-corpuscle (Fig. 19). Its ends are slightly rounded, it is non-motile, and on the interior of the bacilli small colorless spots can be observed on microscopic examination; these clear spaces represent plasmolysis. Spores do not occur, except in mixed infection (*e. g.*, old cavities) due to symbiotic growth.

When stained the bacilli have a somewhat beaded appearance. The tubercle bacillus is one of the few varieties of bacteria that retain the anilin dye after washings with acids.

Biology.—The bacilli can be grown on culture-media, but not without difficulty, since they demand an even temperature between 98° and 100° F. (37.7° C.), or that of the human body. The best soil is blood-serum previously coagulated by heating and glycerin-agar. Over the surface of the medium gently rub tuberculous tissue, which is then allowed to remain on the surface. The growth of the bacilli requires about two weeks, when colonies appear as dry, grayish-white or grayish-brown, thin scales or masses on the surface of the culture-medium. From such cultures others may be grown on glycerin-agar or on the potato. The grass or butter bacillus has staining properties much like those of the tubercle bacillus, and since this organism is commonly found on hay and straw it should be carefully excluded in the study of milk, butter, etc. Both the lepra bacillus and the smegma bacillus resemble the tubercle bacillus in their manner of taking stains, but neither organism is capable of cultivation.

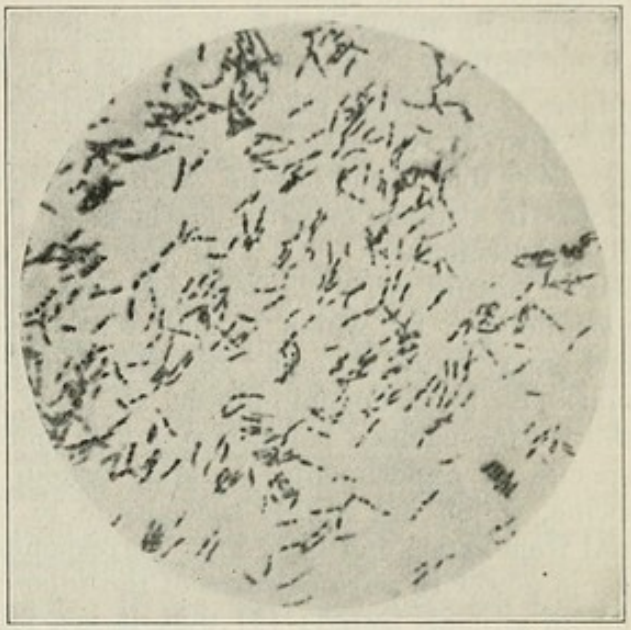


FIG. 19.—Tubercle bacillus in sputum (Fränkel and Pfeiffer).

Inoculations into the guinea-pig and other animals are succeeded in three to six weeks by the appearance of elementary tubercles—first, locally, and then in other organs of the body.

Chemical Products.—The growth of the bacilli is possibly attended by the formation of secretory products. Thus an albuminoid substance has been separated, and this when injected into the body of an animal produces slight fever. The albuminoid separated from cultures of tubercle bacilli is a nuclear proteid, and not a specific toxin. The constitutional features of the disease may be ascribed, in part, to the circulation of these poisons in the blood, but principally to the pus-producing organism.

Bovine Tuberculosis.—The disease is common among cattle (10 to 20 per cent.), and Koch first pointed out certain differences between the bovine bacillus and the bacilli of human and animal tuberculosis. Smith's[1] studies show that the bovine bacillus possesses the greater virulence. It is known that the human bacillus infects cattle with difficulty, while "the bovine bacillus infects animals, and probably also man, with great readiness" (MacFarland).[2] Koch,[3] in an address before the English Congress on Tuberculosis, said that man is rarely infected

[1] *Trans. of the Assoc. of Amer. Phys.*, 1896, xi., p. 78, and 1898, xiii., p. 417.
[2] *Text-Book Upon the Pathogenic Bacteria*, p. 331.
[3] *Jour. Compar. Path. and Therap.*, Sept., 1901.

with bovine tuberculosis. At the seventh International Congress on Tuberculosis (1908), Koch stated that he knew of no authenticated case of pulmonary tuberculosis in which bovine bacilli had been found repeatedly in the sputum. The results of the investigations made by the German Imperial Board of Health show that the dangers from the use of milk and other dairy products derived from cows with tuberculous udders is extremely slight.[1] The British Royal Commission found that one-third of the cases of tuberculosis in children under five years of age were due to the bovine bacillus, hence attention must be paid to this factor in connection with methods of prophylaxis (Woodhead). M. P. Ravenel concludes: (1) That the tubercle bacillus from bovine sources has in culture fairly constant and persistent characteristics of growth and morphology, by which it may tentatively be distinguished from that ordinarily found in man; (2) that cultures from the two sources differ markedly in pathogenic power, affording further means of differentiation, the bovine bacillus being very much more active than the human for all species of experimental animals tested, with the possible exception of swine, which are highly susceptible to both; (3) the tuberculous material from cattle and from man corresponds closely in comparative pathogenic power to pure cultures of the tubercle bacillus from the two sources for all animals tested; (4) that it is a fair assumption from the evidence at hand and in the absence of evidence to the contrary, that the bovine tubercle bacillus has a high degree of pathogenic power for man also.

Sources of the Bacilli.—The chief sources are the sputum of tuberculous patients and the dejecta of persons with tuberculous enteritis and infected meats and milk. The desiccated, germ-laden sputum is wafted into the atmosphere in the form of dust-like particles.

Distribution of the Bacilli.—The tubercle bacilli are found in a viable condition, both (*a*) inside and (*b*) outside of the body.

(*a*) *Inside of the Body.*—As before stated, the number of bacilli found in tuberculous growths varies within wide extremes. In general terms, it may be said that the more rapidly the process advances the greater the number of bacilli present. It must not be forgotten, however, that the activity of the tuberculous processes is intimately connected with the degree of resistance offered by the tissues. A chronic tuberculous focus may establish a fistulous connection with a vein or a lymph-vessel, and thus scatter the bacilli to the remotest parts of the body; and in such instances (as the direct effect of the original number of bacilli present) a chronic is quickly converted into an acute form of tuberculosis. Strauss[2] demonstrated virulent bacilli within the nasal cavities of healthy persons whose positions necessitated their association with, and frequent presence in rooms occupied by, tuberculous patients.

(*b*) *The Bacilli Outside of the Body.*—Tubercle bacilli can maintain their existence almost indefinitely outside the body. On the other hand, they probably do not develop or multiply under the usual external influences, but their vitality is extraordinary. Their destruction cannot be effected by freezing nor by desiccation, and they survive for months in water. Their power to resist chemical agents (nitric acid, etc.) is also very great, but they may be destroyed by boiling for four or five minutes or by exposure to the direct solar rays from four to eight hours

[1] A. Weber in Heft 10 of *Research Work in Tuberculosis.* [2] *Münch. med. Wochen.*

(Jousset). In milk they may be destroyed by heating in a closed pasteurizer for a period of twenty minutes at 140° F. (60° C.). Tubercle bacilli are undoubtedly present in all inhabited places, and they may be conveyed for long distances by means of water, food, and fomites. Willson and Rosenberger have shown that the presence of living tubercle bacilli in the urine and the feces is of importance in relation to the infectiveness of sewage and of drinking water.

The *sputum* dries and flies into the atmosphere in the form of dust, which not only floats in this medium, but also settles upon articles of furniture, the floor, the walls of living-rooms, hospital wards, draperies, clothing, bed-linen, etc.; and from these resting-places it may be conveyed back into the atmosphere. It has been shown, experimentally, that the dust obtained from the walls or from the air of rooms and hospital wards occupied by tuberculous patients is frequently, though not invariably, infected. It is the *in-door atmosphere*, laden with bacilli, that is especially liable to be dangerous. In places only rarely frequented by consumptives the dust is usually free from virulent bacilli.

Modes of Infection.—(1) **Inhalation of the Bacilli.**—Inhalation tuberculosis is, doubtless, less common than formerly supposed. There is some question as to the power of dust containing tubercle bacilli to infect persons and animals when inhaled. It has been demonstrated conclusively that when such dust-like material is mixed with the food, infection follows. In view of this evidence it is probable that the particles of sputum floating in the atmosphere are deposited in the mucosa of the nasopharyngeal ring and tonsils, gaining the lymphatics through these structures and passing to the cervical glands, thence to the apices of the lungs. Klebs and Flügge claim that infection may result from moist particles (salivary droplets) thrown off in coughing, and Boston[1] has demonstrated that in 75 per cent. of consumptives, with cavity formation, a fine spray containing tubercle bacilli is emitted during the acts of coughing, sneezing, laughing, and talking. Ravenel[2] has shown experimentally that tubercle bacilli may be disseminated by cows in coughing. It is highly probable that such spray magnifies the danger of infection from tuberculous cooks and bakers. Occasionally the bacilli attack first the upper respiratory passages (larynx, nose). Usually, however, primary infection takes place in the *smaller bronchi* in the apical area or, less frequently, in a *bronchus;* this is shown by the fact that healed lesions in persons dying of other causes are commonly met with in these situations in the dead-house. The *bronchial glands* may be found to present tuberculous lesions.

It has long been supposed that tuberculosis is a contagious affection; unlike small-pox, scarlatina, and other acute contagious diseases, however, tuberculosis is not transmitted by a single contact with a person ill of the disease. Flick and others have shown that persons who live in close proximity to affected persons frequently fall victims to the disease as the result of prolonged contact.

Flick's topographic study of phthisis in the Fifth Ward of the city of Philadelphia, extending over a period of twenty-five years, shows conclusively that consumption obeys the laws of infectious and contagious diseases. His researches furnish incontestable proof that tuberculosis is limited to

[1] *Jour. Amer. Med. Assoc.*, Sept. 14, 1901. [2] *Jour. Compar. Med.*, Jan., 1901.

centers, and each case owes its existence to previous cases in the same house or locality; that a house which has had a case of consumption will probably have others within a few years, and may have a large number of cases in rapid succession; and that approximate houses are considerably exposed.

The *contagious theory* of tuberculosis gains support from the fact that husbands have been frequently observed to contract the disease from their wives, and the latter, since they are more constantly confined in the house, to become infected yet more frequently from the former. Weber has observed the case of a tuberculous husband who lost four wives in succession, another who lost three, and four others who lost two each. In like manner, the statistical studies of Cornet, Niven, Baer, and others show that the disease spreads through factories, prisons, cloisters, and even among the physicians, nurses, and attendants in hospitals for the reception of tuberculous patients, producing a mortality-rate from this disease ranging from 45 to 75 per cent. Seventy-three per cent. of nurses up to the age of fifty die of tuberculosis (Whittaker). Those who are engaged in making the beds, dusting and sweeping the rooms of patients are most exposed; and, on the other hand, better hygienic living among these classes of individuals, and improved hygienic arrangements in prisons, institutions, and hospitals, have been found to reduce the death-rate decidedly. This result is to be accounted for as follows: (*a*) There is thus established a greater tissue-resistance to the bacillus tuberculosis on the part of the persons exposed; and (*b*) the germs are thus more widely disseminated. Obviously, then, in institutions in which the proper sanitary precautions are used there may be few if any instances; and from the records of the latter, facts opposed to the contagious theory of the disease can readily be furnished.

(2) **Infection by Swallowing.**—(*a*) That the milk of tuberculous animals contains the bacillus, and that the use of contaminated milk may infect the human subject, are well-established facts.[1] Gerlach and Klebs long since observed the occurrence of the disease in animals fed with milk from cows affected with the so-called "pearl disease." It is not even necessary that the animal infected should have tuberculous mammitis (Ernst), though some are of contrary opinion (Flick, Sidney Martin, and others). The exact frequency of this mode of infection is not known. Infected animals, especially cows and pigs, that suckle their young very frequently transmit the disease to the latter, the infection usually resulting in intestinal and mesenteric tuberculosis. The bacillus is, in this instance, swallowed and finds lodgment in the *primæ viæ*. Bang has even shown that butter made from the milk of tuberculous cows may be infectious (*vide* also Bovine Tuberculosis, p. 235). Human tuberculosis is entirely analogous, and hence the tuberculous mother is likely to transmit the disease to her suckling offspring. This explains, adequately, why abdominal tuberculosis is frequent in children.

(*b*) *The meat of a tuberculous animal* (*e. g.*, cow, pig, or fowl) may rarely be infectious, but the bulk of experimental evidence would seem to show that, unless the parts consumed are the seat of tuberculous deposit, infection does not follow. D. H. Bergey,[2] holds that the lower

[1] See the elaborate statistical studies of Dr. George Cornet: "Die Tuberkulose in den Strafanstalten," *Zeitschrift für Hygiene*, Bd. x, 1891.

[2] Saunders' *Year-Book* for 1899.

mortality from this disease shown by the Jewish race is ascribable to their careful *meat inspection*. Again, the possibility of contamination during the course of preparation for the market, and during transportation, must be recollected. The experiments of Aufrecht, Chauveau, Klebs, Parrot, Trappeiner, and others show that tuberculosis may be communicated by incorporating with the food the expectoration from tuberculous patients. The introduction into the stomachs of cattle and goats of a single quantity of virulent bacilli is followed regularly in from thirty to forty-five days by the development of tubercles at the tops of the lungs (Calmette and Guérin [1]).

(3) **Infection by Inoculation.**—Tuberculosis may be transferred by direct inoculation, as shown originally by Villemin's beautiful experiments upon the eyes of guinea-pigs. Infection may take place, though this is rare, through slight cutaneous lesions (cuts, fissures, excoriations), as the result of accidental inoculation of tuberculous matter. In this manner there is produced a local tuberculosis of the skin, as a rule. Rarely, the contagion is conveyed by the lymphatics to the glands in the vicinity. Persons who follow certain occupations are more or less liable to this mode of infection—*e. g.*, butchers, handlers of hides, dissectors of dead bodies, and, rarely, surgeons. Rare instances occur in divers ways (the bite of a consumptive, a cut from a broken spit-glass, or even from his pocket-knife, as I have seen in one instance).

The handkerchiefs, body- and bed-linen of the patient may infect by inoculation those who handle or wash them frequently, if they chance to have a fissure or excoriation upon the hand. No doubt *lupus* also arises in the same way. Czerny has reported 2 cases of infection by transplantation of skin; Collings and Murray, 3 cases by tattooing (?). The contact of the lips of tuberculous operators with surgical wounds (as in sucking the latter) may transmit tuberculosis, as in the performance of the rite of circumcision. Ravenel [2] reports 3 cases of accidental inoculation of the skin in man with the bovine tubercle bacillus.

(4) **Direct Hereditary Transmission.**—In exceptional cases the bacillus is found in the fetus *in utero*. In such instances the disease may remain latent, to break forth during childhood or later in life; and though the fetus itself may display no evidence of tuberculosis, the fetal viscera may yet be infective to guinea-pigs (Birch-Hirschfeld). Lehmann [3] has reported an undoubted instance of intra-uterine infection. The tuberculous mother died of tuberculous meningitis three days after the birth of her child, and the child lived twenty-four hours. In its spleen, lungs, and liver were found nodules resembling tubercles and containing tubercle bacilli in large numbers. Galtier has inoculated a pregnant animal with the disease, and found that the offspring was, in consequence, tuberculous at birth. The views of Baumgarten upon this question should be accorded careful consideration. This author believes that the contagion may be transmitted and become pathogenic at a variable period after birth—first, because the affection is very frequent in young children, even during the first months or weeks of life; and, secondly, because certain structures, not apt to be

[1] *Ann. de l'Inst. Pasteur*, 1905, vol. xix.; 1906, vol. xx., 609.
[2] *Proc. Philada. Path. Soc.*, October, 1900.
[3] *Berlin klin. Woch.*, July 9, 1895.

accidentally infected, are commonly the seat of tuberculous lesions in children—the bones and joints. After birth the bacillus may at any time either lose its vitality or take on a luxuriant growth. It is not known, however, in what percentage of these cases the lungs, intestines, peritoneum, and lymph-glands are free from tuberculous lesions. Küss disputes the theory of the latency of the tubercle bacilli, and contends that latent foci do not exist before the age of three months; that they are rare before the first year, when they mature progressively.

Two facts deserve to be here emphasized: First, that a child born of tuberculous parents is more receptive than one born of healthy stock; and second, that it is more liable to accidental infection.

The instances of direct transmission that have been traced occurred through *tuberculous mothers*. The observations of Csokor[1] upon hereditary tuberculosis in cattle also corroborate this dictum. Friedman,[2] on the other hand, has practically demonstrated the possibility of transmission of tubercle bacilli through the semen. Vignal[3] has shown experimentally that invasion by heredity is very rare.

(5) Dock and Chadbourne state that mixed modes of infection occur.

(6) Baldwin invites forcible attention to the danger of infection from the unclean hands of tuberculous patients.

Predisposing Causes.—(1) **Race and Nationality.**—The effect of nationality upon the receptivity to tuberculosis can be studied advantageously in America on account of the cosmopolitan character of the population. The tuberculous tendency on the part of Indians of this continent, even in the most favorable climates, is universally acknowledged, and the fact that the negro race is highly receptive to tuberculosis is also well known. Osler[4] gives the following corroborative statistics: "Of the 427 cases of pulmonary tuberculosis at the Johns Hopkins Hospital for the two years ending June 1, 1891, there were 41 cases in the colored—*i. e.* about 1 : 10. The ratio of colored to white of all patients in the wards has been 1 : 7." It is more than twice as common in the African as in the white, and still more prevalent with the Indian (W. L. Rodman). At present the number of tuberculous Indians is 120, 24 per 1000 population. Sears[5] found that in 200 cases of tuberculosis nearly 50 per cent. belonged to the first and second generations of Irish immigrants.

(2) **Hereditary Predisposition.**—The percentage of cases in which heredity can be traced has been variously estimated at from 10 to 40. As before intimated (*vide Direct Hereditary Transmission*), a child reared by tuberculous parents runs great danger of being infected accidentally; and again, a person living in an infected house (with or without the presence of a tuberculous patient) is very liable to become infected, whether his antecedents give a tuberculous history or not. It follows that a correct estimate of the number of cases of phthisis in which hereditary influence plays an etiologic part cannot be obtained. Too much importance has heretofore been attached to the influence of

[1] *Deutsche medizinal Zeitung*, Berlin, Jan. 29, 1892.
[2] *Deutsch. med. Woch.*, Feb. 28, 1901.
[3] *La Semaine médicale*, Paris, Aug. 1, 1892.
[4] *Text-Book of Medicine*, p. 204.
[5] *Boston Medical and Surgical Journal*, April 4, 1895.

inherited constitutional peculiarities to the exclusion of other potent factors, especially an infective environment. Moreover, a similar degree of predisposition may be acquired as the result of certain debilitating influences (childbirth, defective food-supply, close living- or working-rooms). An inherited tendency to tuberculosis is more unfailingly transmitted through the mother than the father. Multiple appearance is commoner in families with tuberculous parents (Dock and Chadbourne). Children begotten of parents who are drunkards, or who suffer from certain chronic incurable diseases (syphilis, cancer, etc.) at the time of the birth of their children, are liable to inherit a condition of the system that greatly increases morbidity, unless the tendency is overcome by a proper environment, together with systematic physical training during the first years of life. Moreover, persons who have the so-called tuberculous diathesis are frequent sufferers from catarrhal affections, especially of the respiratory organs. The latter condition forms a marked predisposing factor; yet, on the other hand, tuberculosis is met with in persons of robust figure.

The older authors of medical text-books describe two types of conformation—the *tuberculous* and the *scrofulous*. The latter has a heavy figure, thick lips and hands, large, thick bones, and an opaque skin; the former, a light figure, bright eyes, thin skin, oval face, and long, thin bones. The phthisical type of the chest will be referred to in connection with the physical signs of pulmonary tuberculosis. Here emphasis should be given to Cohnheim's view, which is for the greater part correct, to the effect "that the so-called phthisical habit is not an indication of a tendency to, but actually of the existence of, tuberculosis." Whilst the recognition of a pre-tubercular condition has its practical bearing, it must be recollected also that the term implies merely a "delicacy of constitution, incomplete growth, and imperfect development" (Fagge).

(3) **Previous Infectious Diseases.**—That there is no tendency to the transition of other diseases into tuberculosis, as was formerly supposed, cannot now be questioned in view of the undoubted specific nature of the latter disease. Tuberculosis is, however, embraced among the sequelæ of many acute infectious and chronic diseases—influenza, measles, pneumonia, whooping-cough, typhoid fever, cirrhosis of the lungs, and diabetes mellitus (the latter disease involving a predisposition to the former)—for the reason that they render the tissue-soil, especially that of the respiratory tract, more favorable to tubercular infection. Dock and Chadbourne have analyzed 100 cases of adult tuberculosis (bacillary phthisis); it developed rapidly after influenza in 16, and followed pneumonia in 9. It seems proper to mention here the fact that certain other diseases are thought by most writers to display an antagonistic effect (chronic valvular disease, pulmonary emphysema, etc.).

(4) **Age.**—This affects predisposition decidedly, though tuberculosis may occur at any or all times of life. Certain forms of tuberculosis are especially frequent in young children (meningeal, mesenteric, and lymphatic). Pulmonary tuberculosis is most common between *twenty* and *thirty*. It is more rare during early childhood and in the aged, and the cases that occur in young children are likely to be rapid in their progress. Tuberculosis in adults usually develops in an organism already infected.

(5) **Sex.**—Predisposition has but slight relation to sex. Females are,

however, somewhat more liable than males, and pregnancy in particular is a disposing factor. Again, when tuberculous females become pregnant the progress of the affection is accelerated, and even more so by the period of lactation. Regarding tuberculosis as being pre-eminently a house-disease, females are more exposed to contagion than males, because they are more closely confined in-doors.

(6) **Climate and Soil.**—Humidity of the soil and abundant atmospheric moisture increase the prevalence of tuberculosis. It is especially common in regions in which sudden variations of temperature, or protracted cold with dampness, prevail. This increase is most probably associated with a heightened vulnerability, due to an increased tendency to catarrhal affections of all kinds (Osler). It has been shown that proper drainage of marshy districts has diminished, to some extent, the frequency of this disease (Buchanan), and, on the other hand, mountainous districts are often remarkable for freedom from the disease.

Local Causes.—(1) **Occupation.**—Persons whose employment exposes them to different forms of irritating inhalations are particularly liable. In such, however, there is usually first developed a fibroid induration (*vide* Pneumonokoniosis), and the latter in turn is followed by pulmonary tuberculosis. The continual inhalation of an atmosphere laden with noxious particles, such as is met with in ill-ventilated and overcrowded working or living apartments, renders the tissues more vulnerable.

(2) **Bronchial Catarrh.**—An acute catarrh of the small bronchi prepares the soil for tuberculous infection. Frequently, however, this is the first step in tuberculosis, since the latter disease almost invariably begins as a local catarrhal process, involving the smaller apical bronchi. Here may be pointed out that gastro-intestinal catarrh (of protracted duration—H. M. King) increases the receptivity for tuberculosis.

(3) **Tubercular Pneumonia.**—In like manner, pulmonary tuberculosis may follow an unresolved pneumonia, but such cases are, as a rule, instances of tuberculous pneumonia primarily.

(4) **Hemoptysis.**—According to some authors, hemoptysis is potent in producing pulmonary tuberculosis. It is, however, certain that in most instances in which it appears to precede phthisis, and to exert a causative influence, it is in reality a symptom of existing tuberculosis.

(5) **Pleurisy** may be, though rarely, the starting-point of phthisis. Its predisposing effect may be attributable to compression of the lung, thus interfering with the respiratory excursions, or to the bronchitis which is frequently associated. Pleurisy sometimes initiates fibroid induration, which may then terminate in a tuberculous affection; but the fact is to be emphasized that a very large proportion of the cases of apparently primary pleurisy are tuberculous in nature.

(6) **Intrathoracic Tumor.**—Tuberculosis is often associated with intrathoracic tumors, and especially with aneurysm. Fehde[1] has reported 3 interesting cases of the kind.

(7) **Congenital or acquired contraction of the orifice of the pulmonary artery** predisposes markedly to tuberculosis. The lungs are often found to be undersized and ill-nourished from birth.

(8) **Trauma.**—Injuries to the chest-wall, with or without laceration of the lung, are frequently followed by pulmonary tuberculosis. The explanation of this association is to be found in the fact that trauma

[1] "Lungentuberculose mit Brusthöhlengeschwülste," *Inaug. Diss.*, Leipzig, 1894.

increases largely the susceptibility of the parts injured by diminishing phagocytic activity—the natural power of resistance. It is a familiar observation in surgical practice that after injuries to, or operations on, joints, tuberculosis, often acute, frequently ensues—in about 8 per cent. of the cases.

Tuberculosis of the Lymph-glands.

(*Scrofula.*)

Scrofula implies tuberculous infection, and scrofulous material inoculated upon susceptible lower animals, especially guinea-pigs and rabbits, invariably causes tuberculosis. The virus is, however, less virulent than that derived from other sources, and this explains the slow progress and often latent character of tuberculosis of the glandular system. A major predisposing factor is *age*, this form of tuberculosis preponderating in children. Hecker, from an examination of the records of the Munich Pathological Institute, found that in 147 cases of tuberculosis among children the lymphatics were affected in 92 per cent.; and in young adults tuberculous adenitis is not uncommon. It is rarely met with also during and after the middle period of life. The lesions generally remain limited to the glands first infected—*i. e.*, the cervical, mesenteric, etc., as the case may be—and this for the reason that the natural powers of resistance in the tissues are often able to oppose the march of the destructive forces. Another predisposing condition is an *acute* or *chronic catarrh* of the mucous membranes.

The cases are all divisible into two groups: (1) Local tuberculous adenitis, and (2) general tuberculous adenitis.

(1) **Local Tuberculous Adenitis.**—(*a*) **Cervical.**—This is the most frequent form, and is especially common among children.

Etiology.—Of 2035 persons examined by Valland, enlarged cervical glands were found between the ages of seven and nine in 96 per cent.; between ten and twelve in 96.1 per cent.; between thirteen and fifteen in 84 per cent.; between sixteen and eighteen in 69.7 per cent.; and between nineteen and twenty-four in 68.3 per cent. Tubercle bacilli were found in the cervical lymph-glands in about 68 per cent. of adults. Negroes are found to be more prone to the affection than whites.

Mode of Infection.—I have stated before that tubercle bacilli are sometimes found on the nasal mucous membrane of healthy persons. The presence of an acute or chronic catarrh of the nasopharynx may now lower the resistance of the tissue-cells, so that the bacilli may gain access to the lymph-current, and through the latter to the neighboring glands, setting up tubercular adenitis. The cervical lymph-glands, however, do not furnish a highly favorable soil for the growth and development of the bacilli, and hence the tendency toward latency.

The *tonsils*, owing to their free communication with the atmosphere, in which there is a wide diffusion of tubercle bacilli, may be primarily infected. Friedman suggests that primary tuberculosis of the tonsils is usually set up by infection through the food. But here also, as in the case of other glandular structures, there is a tendency for the affection to become encapsulated, for the reason that the tissue-soil after a prolonged contest generally gains the ascendency over the invading bacilli. The latter may, however, under certain favorable conditions, break down the

barriers opposed by nature and effect a lodgement in the cervical glands, or even become widely diffused through the economy. Thus Kinckmann in 64 autopsies found 25 cases of tuberculosis, in 12 of which the tonsils were affected.

A third mode of infection of the cervical lymph-glands is through the medium of slight injuries and abrasions of the skin or certain forms of skin-eruptions (eczema, etc.). These serve as doors of entrance for the bacilli, which find their way into the neighboring lymph-glands through the lymph-channels. Compared with infection from within, this mode is most probably much less frequent.

Symptoms.—The main feature is a *visible enlargement* of the affected cervical glands, chiefly the submaxillary. At first the glands are too small to be even palpated; later, they can be felt as small, firm tumors underneath the skin. By and by they appear as visible protuberances, ranging in size from that of an English walnut to that of a hen's egg or even larger. The *skin* over the enlarged gland is freely movable, as a rule; less frequently it becomes adherent—an indication of suppuration. When an abscess forms and is allowed to open spontaneously, there remains a chronic discharging sinus. Suppuration is attended with *fever*, *anemia*, and *emaciation*. In well-marked cases the separate tumors coalesce, forming large and irregular masses. The affection is usually bilateral, though almost invariably it is more marked on one side than on the other.

Not infrequently, in addition to the enlargement of the submaxillary, post-cervical, and supraclavicular glands, there is also involvement of the axillary, as was the case in a fatal instance in my own practice. The patient was a male child, eight years of age, who developed pulmonary tuberculosis. It may reasonably be assumed that the bronchial glands also become implicated, and may excite lung tuberculosis.

The *diagnosis* is based upon the history and the associated evidences (keratitis, conjunctivitis, eczema of the face, nasopharyngeal or bronchial catarrh), coupled with the glandular enlargement. Bacilli have occasionally been found in the purulent discharge from abscesses. Otis applies the tuberculin-test, and obtains positive reactions in 62 to 69 per cent. The Von Pirquet cutaneous reaction may be also employed.

The *course* of this affection is exceedingly slow, often extending over a number of years. Many cases, however, recover after timely surgical intervention. On the other hand, neglected cases are a menace to the life of a patient, since they may be followed by diffusion of the bacilli, with the development of a fatal form of disease.

(*b*) **Bronchial.**—Tuberculosis of the bronchial glands may be primary, or secondary to infection of the lungs, and it is commonly preceded by or associated with *bronchial catarrh*, which is its chief predisposing cause. The primary form is met with frequently in young children, the mediastinal lymph-glands being affected uniformly in 127 cases at the New York Foundling Hospital (Northrup).

The bronchial and tracheal glands are the receptacles for all foreign substances, including the tubercle bacilli that are not dealt with by the broncho-pulmonary phagocytes. After infection with tubercle bacilli the lymph-glands become swollen, tumefied, and are the seat of caseous change; later they may undergo calcification or proceed to abscess-for-

mation. The latter may rupture either into the lungs, into the trachea or the bronchi, or into a pulmonary blood-vessel.

Symptoms.—If a fistulous communication be established with the air-passages, *cough* and *expectoration* of purulent material, blood, and caseous matter containing bacilli will be noted.

Secondary infection of the lung may occur in this manner. When rupture takes place into a vessel systemic infection promptly follows. Tubercular adenitis involving mediastinal lymph-glands may also lead to infection of the pericardium and then proceed to tuberculous pericarditis.

(*c*) **Mesenteric** (*Tabes Mesenterica*).—This may be *primary* or *secondary*, the latter being common as a secondary infection to intestinal tuberculosis.

The former is rare, however, and the intestinal catarrh with which it is associated is doubtless tuberculous in the vast majority of cases. The mode of infection has already been pointed out. The lesions presented are similar to those met with in tuberculous bronchial glands.

The *symptoms* are not always distinctive, and may be entirely negative during the life of the patient; hence the condition is often incidentally discovered during the post-mortem examination. The *local symptoms* when marked are due in the main to an associated peritonitis. The abdomen is painful and more or less swollen. *Peritoneal effusion* is present, and sometimes sufficient in amount to be detected by the customary physical signs. Large and small *nodules* may sometimes be felt. *Diarrhea* is a marked and an obstinate feature and is usually due to tuberculous intestinal ulcers. *Fever* of an intermittent type is almost constantly present, causing emaciation, and the objective changes (pallor of skin, mucous membranes) due to anemia become pronounced. This form of tuberculosis may persist as a local condition, but there is danger of extension to other organs (pleura, lungs). On the other hand, in the adult pulmonary tuberculosis may be followed by involvement of the mesenteric glands without involvement of the intestines, and in such instances there occurs an extension by contiguity along the course of the lymphatics that pass through the diaphragm, and finally, in adults, primary tuberculous new growths may be met with in the mesenteric glands.

Diagnosis.—A probable diagnosis can usually be made if careful attention be paid conjointly to the symptoms, physical signs, and course of the affection. The detection in a child of a tumor which may be moderately hard, doughy, or even fluctuating will aid materially in the diagnosis, and will also afford evidence of tuberculous disease in other organs. The Von Pirquet cutaneous reaction will be found present.

(2) **General Tuberculous Adenitis.**—This term implies tuberculous disease of the lymph-glands throughout the body, with little if any involvement of other organs; it is a rare condition. The affection may begin as a local tuberculous lymphadenitis, nearly all the rest of the glands of the body becoming secondarily implicated. The primary seat of the trouble is perhaps most frequently the cervical lymph-glands, though in one instance observed by myself the mesenteric glands first became affected, the case terminating in pleuro-pulmonary tuberculosis.

Symptoms and Diagnosis.—There is *protracted fever*, the temperature being of the remittent or intermittent type. *Wasting* and *debility*

are progressive until the patient presents a decidedly puny aspect, while the lymph-glands that are accessible to inspection and palpation are more or less enlarged and manifest a marked tendency to *suppuration*. The affection is usually *chronic*, though very exceptionally it may exhibit an acute course. One of the chief dangers overhanging the sufferer in this affection is that, owing to liberation of the bacilli, the meninges or the lungs may become tuberculous; these cases may also eventuate in death from asthenia. Cases in which the glands are but little enlarged, while the *general features* are marked, are puzzling. On the other hand, when the superficial lymph-glands are greatly enlarged, the affection may bear a striking resemblance to Hodgkin's disease. Indeed, certain recent writers hold that generalized tuberculous adenitis and pseudo-leukemia are etiologically identical (*vide* Hodgkin's Disease, p. 483).

Acute Tuberculosis.

This form of tuberculosis is characterized anatomically by the rapid development of miliary tubercles in many and widely-separated parts of the body. In some instances the new growths are pretty evenly distributed through all the organs of the body, manifesting the clinical symptoms of an *acute general infection*. In other instances there is a tendency to centralization of tuberculous growths, as, for example, in the lungs (pulmonary variety) or in the meninges of the brain and spinal cord (meningeal variety).

Pathology.—The fact is to be emphasized that somewhere in the body there is an old tuberculous focus. Apart from this primary lesion, the anatomic changes consist in the widely disseminated miliary tubercles. Their most frequent seats are the lungs, liver, and spleen; less commonly, the marrow of the bones, the heart, the choroid, and the meninges. In some of the organs, particularly the meninges, lungs, etc., the tubercles may be readily perceived by the naked eye, while in others they frequently cannot be detected without the aid of the microscope. It must not be forgotten that in some of the more protracted cases the nodular tubercles may grow into foci of considerable size, ranging from that of a lentil to that of a pea.

Etiology.—This has been, in the main, given in connection with the general etiology of tuberculosis (*vide supra*), though a few special points remain to be adduced. The acute forms of tuberculosis are decidedly more frequent during infancy and childhood than during adult life, and with few exceptions the cases are secondary to a local tuberculous focus in one or more lymph-glands (tracheal, bronchial, mesenteric) or in the lungs. More rarely a pre-existing tuberculous focus in the kidneys, the bones, or the skin may give rise to the affection, as may the occurrence of certain other acute infectious diseases (such as measles, whooping-cough, and influenza) in children, and typhoid fever and lobar pneumonia (especially with delayed resolution) in adults.

Modes of Infection.—Most frequently there is established a fistulous connection between the local tuberculous focus and a vein, especially the pulmonary vein. Under these circumstances there may be large numbers of bacilli discharged into the blood-stream; but oftener only small

numbers of bacilli enter and subsequently multiply, inducing general infection (Ribbert and Wild[1]). A second mode of infection, though decidedly more rare than the above, is the rupture of a tuberculous focus into the thoracic duct, in which case the tuberculous material passes almost directly into the subclavian vein.

Clinical History.—That miliary tubercles may exist in many organs of the body (liver, heart, etc.) without giving rise to symptoms is a noteworthy fact. Cohnheim and Manz have discovered miliary tuberculosis of the choroid with the aid of the ophthalmoscope alone.

The following forms of the disease may be distinguished:

General Miliary Tuberculosis.

(*a*) typhoid form.

The **symptoms** are those of a general infection of the body, there being in most cases a period of *incubation*, during which the patient complains of malaise, headache, chilliness, feverishness, and increasing debility. Rarely, the *onset* is comparatively *sudden*. The reaction of the nervous system against the poison, which is now scattered to all parts of the body, is shown by such symptoms as the *fever*, which rapidly increases, a *rapid, feeble pulse*, and *mental dulness* or *delirium*. The *tongue* becomes dry, and sometimes also brown. The *respirations* are accelerated, and there is more or less *cyanosis*, with which symptom is associated a peculiar and *characteristic pallor* of countenance. Coincidently with the febrile exacerbations the *cheeks* wear a circumscribed blush. Among the rarer early symptoms is epistaxis. The patient soon becomes either profoundly prostrated or anxious: if, as sometimes happens, the course is protracted, *weakness*, *anemia*, and especially *emaciation* are well marked and assume diagnostic importance. These cases sometimes pass into the pulmonary or the meningeal form, the patients often succumbing speedily to such localized developments.

Fever.—The temperature usually pursues a high range, although there are a few cases in which the entire course is afebrile. Again, it occurs not infrequently that the temperature is normal or nearly so for a short period. The usual temperature-curve ranges at first between 102° and 104° F. (38.8°–40° C.), and then continues to rise, with the development of the serious general condition in a way exactly similar to that observed in typhoid fever. In many instances the fever is irregularly remitting, at least at intervals, if not so constantly. Thus, periods of irregular fever may alternate with others of continued, and later deeply remittent or distinctly intermittent, fever.

Nervous Symptoms.—In most cases the nervous symptoms are not prominent. In a smaller number headache, vertigo, delirium, and often stupor, become marked at an early stage and may persist. They are due to the general infection.

Circulatory System.—The pulse is small, and its rate is out of proportion to the fever, varying from 100 to 140 or higher. It may become irregular, particularly if the meninges be involved.

Respiratory System.—The breath is somewhat hurried and labored; there is a cough, but it is not annoying as a rule; and there is a slight expectoration, which is not characteristic. If there be present simul-

[1] *Deutsche medicinische Wochenschrift*, Dec. 30, 1897.

taneously in the lungs an old tuberculous focus, the expectoration may be more profuse and typical. The bacilli are also absent from the sputum unless an old tuberculous lesion exists in the lungs. J. Vogl states that individual tubercles can be demonstrated on the *x*-ray plate.

The physical signs are those of a diffuse bronchitis, though signs of consolidation or pleurisy may develop late in the course of the affection. Such signs, however, may be evidences of an old tuberculous affection.

Digestive System.—As before noted, there are anorexia and a dry tongue (symptoms due to the systemic infection), while vomiting may occur at the outset, and excessive thirst is common. The spleen usually becomes enlarged, though only to a slight extent, as a rule.

Ocular Symptoms.—The important symptom presented by the eye is the presence of choroid tubercles. Their demonstration is only possible with the skilled ophthalmologist. Their absence, however, does not militate against the diagnosis of this disease. Tileston has described an eruption, in cases occurring among children, which consists of scattered, discrete papules about the size of a pinhead, and on these are tiny vesicles with cloudy contents or minute pustules, followed by drying, with slight incrustation.

Diagnosis.—In the following table I have endeavored to contrast points of dissimilarity between this disease and typhoid fever:

Acute General Miliary Tuberculosis.	Typhoid Fever.
Family history of tuberculosis, or presence of an old focus.	Coexistent with an epidemic or following previous cases of typhoid.
Evolution of the disease not characteristic.	Evolution of the disease is characteristic.
Epistaxis rare.	Epistaxis a common early symptom.
Fever-curve of decidedly irregular type.	Temperature-curve of the continued type.
Pulse rapid, out of proportion to fever.	Pulse often dicrotic; slow in proportion to fever.
Respirations rapid and labored.	Respiration moderately increased.
Face dusky, with peculiar pallor.	No duskiness of face.
Abdominal symptoms are not suggestive.	Abdominal symptoms (stools, enlarged spleen, tympanites, etc.) suggestive.
No characteristic eruption.	The eruption (appearing in successive crops) is distinctive.
Von Pirquet reaction usually positive.	Widal reaction present.
Knee-jerk may be absent.	Knee-jerk never wanting.
Choroid tubercles may be detected.	Choroid tubercles absent.
Tubercle bacilli rarely demonstrable in the blood.	Cultures from venous blood show typhoid-bacilli. They may also be found in the stools and urine.
Hemorrhage from bowels exceptional.	Hemorrhage from the bowels common.
Perforative peritonitis absent.[1]	Perforative peritonitis often present.

The tuberculin test may prove an aid to diagnosis in cases pursuing an apyrexial course.

(*b*) PULMONARY FORM.

Though all gradations between the typhoid and the pulmonary types occur, the latter should be recognized and briefly described. It may develop *suddenly*, the ushering-in symptom being sometimes a *chill*, though more frequently there is a *premonitory period*, during which

[1] See also Differential Diagnosis of Typhoid Fever.

the general health fails materially. Some acute illness, as measles or whooping-cough, in which there has been marked catarrhal bronchitis, often constitutes the point of departure for this variety.

The *respiratory symptoms* are early prominent, and later preponderate in the clinical picture. From the start there is dyspnea, and this gradually increases until the respirations become rapid (40 to 60 per minute). When dyspnea becomes pronounced, the face presents a characteristic cyanotic pallor. The cough at first is moderately severe, but it soon becomes troublesome, being frequent and attended with a slight expectoration, which, however, is non-characteristic.

The **physical signs** are those of broncho-pneumonia, and the latter may or may not be preceded by the signs of generalized bronchitis. With the onset of consolidation there appear spots that yield either dulness or a tympanitic resonance on percussion, and broncho-vesicular breathing with numerous subcrepitant râles on auscultation.

The general symptoms are marked from the beginning. The *fever* is high—from 103° to 105° F. (39.4°–40.5° C.) or often higher. The pulse ranges from 100 to 140, is small, feeble, and sometimes irregular, and it may be more rapid still during the advanced stage of the affection (see Fig. 20). Cerebral symptoms rarely appear.

The **course, as a rule,** is more prolonged than that of general miliary tuberculosis, except in children, in whom it often runs an exceedingly acute course. As the end approaches the signs of suffocation are gradually intensified, and finally lead to a fatal termination.

Diagnosis.—The diagnosis is difficult; but a family history of tuberculosis, a knowledge of the pre-existence of a tuberculous focus or of an antecedent predisposing affection, will aid in its recognition. Tubercle bacilli are perhaps not demonstrable in the sputum unless an old tuberculous lesion is present. In doubtful cases, however, an attempt should be made to detect the bacilli in the blood. Occasionally either tuberculous meningitis or peritonitis supervenes, and aids in removing the doubt, and in a small percentage of the cases choroid tubercles are detectable. These points, together with the more marked general symptoms, will usually enable the clinician to distinguish this variety of tuberculosis from *non-tuberculous broncho-pneumonia.*

(*c*) CEREBRAL OR MENINGEAL FORM (TUBERCULOUS MENINGITIS).

This variety is of quite frequent occurrence, appearing in not less than 50 per cent. of the cases of miliary tuberculosis. When it develops, the symptoms referable to other organs than the meninges are in abeyance. With reference to the *etiology*, the fact needs to be emphasized that most cases occur between the ages of two and seven years; it may, however, be met with at any time of life. The affection frequently has its origin in tuberculous bronchial glands (Jacobi), and the history of a fall is common. A few cases have been found to be associated with erythema nodosum. Exceptionally, the meninges are primarily involved.

Pathology.—The chief site of the tubercles in children is the pia mater at the base of the cerebrum (basilar meningitis), while in adults the pia at the vertex is more apt to be involved. The membrane sur-

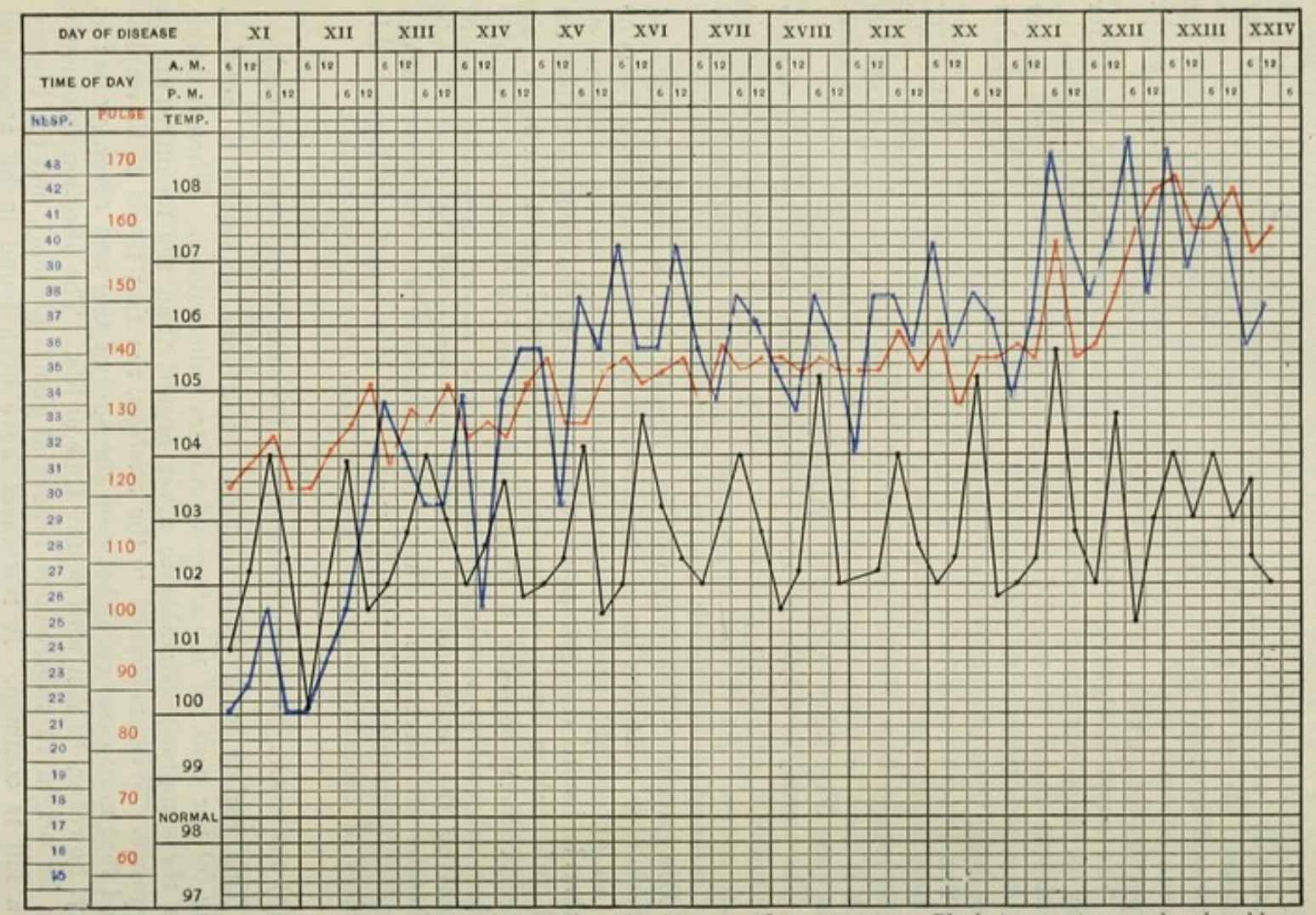


FIG. 20.—Chart of a case of acute pulmonary tuberculosis. Mrs. M——, aged twenty years. Black, temperature; red, pulse; blue, respirations.

rounding the tubercles may not be inflamed, there being a simple tuberculous deposit. On the other hand, more or less inflammation, with sero-fibrinous or fibrino-purulent exudation, is generally present in the region of the base. This exudate is usually abundant in the Sylvian fissures, and may find its way to the external surface of the hemispheres. It is gray in color, transparent, and gelatinous, and contains in its meshes the tubercles, which appear as grayish-white bodies, and which, in cases of equal severity, may be either numerous or scanty. They may be scarcely visible to the naked eye, but may vary from the size of a pinhead to that of a French pea. The branches of the Sylvian artery may be implicated, either owing to the direct pressure of the exudate or to the obliterating arteritis produced by a tuberculous infiltration. The pia looks like wet blotting-paper over the quadrangle at the base (Gray). Elsewhere it is thickened and opaque, though easily detachable. Osler says: "The arteries of the interior and posterior perforated spaces should be carefully withdrawn and searched, as upon them nodular tubercles may be found when not present elsewhere. In doubtful cases the middle cerebral arteries should be very carefully removed, spread on a glass plate with a black background, and examined with a low objective. The tubercles are then seen as nodular enlargements on the smaller arteries." Involvement of the chief vessels that nourish the walls of the ventricles and the ependyma, and stretch from the vermis cerebelli forward over the quadrigemina, explains the constant presence of a turbid fluid in the ventricles, with softening of their walls. As the result of undue intraventricular pressure the cerebral convolutions become more or less flattened, with effacement of the sulci. The cortex, to a variable depth, is generally the seat of red softening, and more rarely of white softening alone. The tuberculous infiltration involves the cranial nerves.

Histology.—The tubercles grow in the perivascular sheaths, which are often distended with lymphoid and epithelioid cells, and there is observed not infrequently a thrombosis of the arteries and of the venules of the pia, obliterating their lumen. The pia mater is gradually thickened through cellular infiltration, and in a small proportion of the cases the spinal meninges are similarly involved, chiefly in the cervical portion of the cord.

Symptoms.—There is a *prodromal period* which lasts one or more weeks, during which the patient (usually a child) is pale, peevish, has headache and photophobia, and grinds its teeth during sleep; the tongue is coated, appetite impaired, and there may be occasional vomiting, either propulsive or regurgitative. Constipation is present and may be marked. Among rare premonitory symptoms are slight hyperesthesia of the abdomen and a diminished urinary secretion. A tendency to *emaciation* is quite constant. These prodromal symptoms present variations as to their number and combinations in different cases. In few instances only is the onset acute. The symptoms usually indicate basic meningitis, and at first there is associated considerable mental excitement; later there are pressure-symptoms (caused by the exudate), with total loss of the mental faculties.

(1) **Stage of Cerebral Excitement.**—The *invasion* is generally gradual, or even quite insidious, its most characteristic phenomena being *severe*

vomiting, marked headache, and *chills followed by fever*. Certain other symptoms now arrest the attention, such as extreme irritability, screaming, and great obstinacy, and occasionally drowsiness appears early. When the onset is sudden the disease may be disclosed by convulsions, paralysis, wild delirium, or coma. The established disease exhibits certain distinctive features. The pain is often most excruciating, causing the child to utter short penetrating screams (hydrocephalic cry), and in rare instances the sharp cries may be continuous and lead to physical exhaustion. The headache is increased by light, noise, or movement. Vertigo is common; the pupils are contracted at this period; the face pales and then flushes; the pupils alternately dilate and expand; and the expression is sometimes sad, though more often stupid. Generally hyperesthesia or dysesthesia may appear, and there may be a slight mind-wandering at night, though active delirium is rare. *Tâches cérébrales* may be obtained, but are not characteristic. The patient is intolerant of every form of disturbance. All the symptoms of the prodromal stage are now aggravated; slight muscular twitchings and sleep-starts occur; the vomiting is apparently causeless, and may be frequently repeated; and constipation persists.

Fever is present, but is of slow development, and rarely rises higher than 102° or 103° F. (39.4° C.) in the evening. The *skin* is dry and harsh. The pulse is slow or moderately accelerated, but soon quickens to 120 or even 130, and later it may be irregular. At times the *pupils* are unequally contracted, and ptosis is usually an early sign.

(2) **Second or Transitional Stage.**—The symptoms of cerebral irritation now abate, the patient becoming more quiet, while mental dulness often supervenes. The vomiting and headache gradually subside, and the child rarely cries out. The abdomen is now distinctly scaphoid and the head occasionally retracted. Constipation is obstinate. The evidences of localized organic foci, such as slight twitchings of the muscles of the face, followed by strabismus, ptosis, or paralyses of the face or limbs, may appear. Generalized convulsions may occur, and muscular tremors and athetoid movements may appear. Both pupils (or one only) may be dilated as intracranial pressure develops; patchy flushing of the face is common. The respiration is now irregular and sighing.

(3) **The Stage of Paralysis.**—On account of the exudation the mental faculties are abolished, so that the patient is comatose, though convulsions or localized spasms of the muscles in different parts of the body (neck, back, limbs, etc.) may be observed. Optic neuritis develops, while the paralysis of the ocular muscles above noted deepens. The pupils are dilated, the eyes are partly closed, and the eyeballs at intervals slowly and alternately move in a lateral direction. Hemiplegia sometimes develops, and more rarely monoplegia, affecting the face or one of the extremities. There may be paralysis of the third nerve, with involvement of the face, hypoglossal nerve, and limbs on the opposite side (a combination of symptoms first observed by Weber), consequent upon a lesion localized in the internal inferior portion of the crus. Monoplegia of the right side of the face has been observed in a few instances, associated with aphasia. Exceptionally aphasia and brachial monoplegia have been combined. The temperature in the early part of

this stage usually rises to 103° F. (39.4° C.) or higher, but later it may drop to a subnormal level, and in rare instances as low as 94° F. (34.4° C.). Immediately preceding the fatal termination the temperature may rise to 106° or 107° F. (41.6° C.), the pulse becoming frequent, small, and irregular. Anesthesia comes on with general muscular relaxation.

Occasionally a *typhoid state* (great prostration, dry tongue, diarrhea, etc.) may develop, and Cheyne-Stokes respiration is almost invariably present, preceding the fatal event. Leukocytosis has been observed.

Ophthalmoscopic Examination.—The ophthalmoscopic appearances are—hyperemia of the disk, later the changes belonging to neuritis (swelling and striation) appear, and choroidal tubercles may be detected.

Diagnosis.—This is based: (1) On the reaction to tuberculin; (2) Examination of eyes, which present the characteristic appearance of the choroid coat (Jacobi). Macewen first pointed out that if the patient is caused to assume the upright position with the head inclined to one side, percussion over the pterion gives a tympanitic note which is indicative of internal hydrocephalus. Koplik found this sign present in 34 of 52 cases. *Post-basic meningitis* gives the same symptoms, and lumbar puncture is the only means of diagnosis. In tubercular meningitis the diplococcus intracellularis is not found. *Syphilitic meningitis* and meningitis due to *trauma* may bear a close resemblance to the tubercular form, but the history should prevent confusion.

Clinical Types.—(*a*) **Mild Type.**—The marked or alarming symptoms (tetanic rigidity of the muscles, convulsions, and paralysis) develop at a late period. In this class should be placed those cases in which the meningitis is but feebly indicated—*e. g.* when it is but a small factor in the condition of acute general tuberculosis.

(*b*) **Malignant or Rapid Form.**—This type is comparatively rare, occurring most frequently in adult life, while the lesions have their seat almost exclusively upon the convexity. The onset is marked by the most frightful tetanic convulsions, which precipitate a fatal termination in a couple of days.

(*c*) **Chronic Type.**—Cases pursuing a chronic course are rarely encountered, and the symptoms usually point to localized cerebral lesions (Jacksonian epilepsy, etc.).

Prognosis.—The disease lasts from two to four or five weeks, though chronic cases may continue for several months. When the convexity is implicated, however, the duration is only one or two weeks. It should be emphasized that frequently in the course of well-marked cases a decided remission in the leading symptoms occurs, so that convalescence is suggested; but this is deceptive, and is almost invariably followed by a renewal of the unfavorable features of the affection. A few cases only are recorded in medical literature as ending in recovery.

Freyhan has reported a case with recovery in which the diagnosis was proved by puncture of the spinal canal and the withdrawal of fluid, in the sediments of which tubercle bacilli were found. A. Jacobi has met with 2 cases that terminated favorably, and Leube has also reported a case in which the symptoms were characteristic, and at the autopsy, some years later, old tuberculous lesions were found in the meninges. It is to be recollected, however, that the course of tuberculous meningitis is probably uninfluenced by human agency.

Acute Pneumonic Phthisis.

(*Acute Phthisis; Florid Phthisis; Galloping Consumption.*)

This may be primary or secondary, the latter form being consequent either upon a localized tuberculous area in the lung, tuberculous pleurisy (acute or chronic), tuberculous peritonitis, or tuberculous disease of some other organ. Acute phthisis may occur at any age, though it is relatively more frequent in childhood and early adult life, but whether primary or secondary, the infection of the lungs is rapid.

Pathology.—Two forms may be recognized: (1) This reveals the appearances of an *acute lobar pneumonia*, one lobe only being implicated, as a rule, though sometimes the whole lung is involved. The process leads to a destruction of lung-tissue, so that a section may show the existence of cavities. The latter are usually small, while surrounding them may be seen tubercles in hepatized tissue, and here and there caseous masses of a yellowish-white color may be visible. These often indicate old or pre-existing foci. It is sometimes exceedingly difficult to distinguish a tuberculous croupous pneumonia from the ordinary form, and the most careful inspection may fail to reveal the presence of elementary tubercles in the acutely consolidated tissue. In cases in which this disease is suspected, however, the opposite lung, the bronchial glands, the peritoneum, and other organs should be carefully examined.

The lesions presented by cases that have run a long course are somewhat characteristic, though not always the same. If the case has had a duration of two or more weeks, apical softening with more or less extensive cavity formation often occurs. Less frequently, a lobe or an entire lung is found to be consolidated throughout, "and converted into a dry, yellowish-white, cheesy substance, in which condition it may remain till the end."

(2) *Presenting the Appearances of Broncho-pneumonia.*—This variety is more common than the previous, especially in children. The evidences of bronchitis affecting the finer tubes, together with consolidation of the lobules to which the tubes lead, are striking. As in ordinary broncho-pneumonia, so here, the solidified areas appear as grayish-red masses in the early stage, while later they are of an opaque-white. The products that fill the air-cells may caseate and break down, with the formation of irregular cavities that vary in size. When large areas are involved they are the result of the fusion of contiguous smaller areas of hepatized tissue. The trouble often begins in the upper lobes and spreads downward, though not infrequently the lower lobes are most extensively involved.

In not a few cases the masses are small, multiple, and widely disseminated throughout the lungs, and miliary tubercles in the lungs or pleuræ are associated with the broncho-pneumonic lesions before described. In nearly all cases signs of pleurisy may be noted, as is shown by pleural adhesions or by deposits of lymph on the pleura. The bronchial glands are also usually infected, and, particularly in children, are the seat of tuberculous processes.

Bäumler has called attention to a type of *tuberculous inhalation pneumonia* consequent upon hemoptysis, the blood and contents of the

cavities being drawn into the finer tubes in respiration. This form of broncho-pneumonic phthisis sometimes follows pulmonary tuberculosis in the early, though more often in its late, stage. On microscopic examination tubercle bacilli are found, though rarely in abundance, in the infiltrated masses and in the walls of the cavities.

Clinical History.—(1) **Acute Cases.**—Preceding the attack, the patient may have "taken cold" or have been in a run-down state; more often, however, he has been apparently healthy. The *onset* is sudden, marked by a *rigor*, *pain in the side*, *fever*, *cough*, and *systemic prostration*, and there may be *bronchial hemorrhage* which may last one or more days. The total amount of blood expectorated may be considerable. In the majority of cases the *expectoration* is mucoid at first, and then becomes rusty-colored, often containing tubercle bacilli, though at first they may be absent and, indeed, not appear until late in the disease. *Dyspnea* appears early, and may soon become extreme, and the fever quickly rises to 104° F. (40° C.) or over. It may be of the continued type, or it may early assume the remittent or hectic type, and with the latter forms of fever, which usually begin about the end of the first week, are associated *night-sweats* and *rapid emaciation*. The prostration of the vital powers is now extreme. The expectoration is more abundant, muco-purulent, and often greenish-yellow in color.

In the course of one or two days after the onset we obtain *physical signs*. Usually, as before stated, there are present the anatomic appearances of acute lobar pneumonia—viz., the complete consolidation of one or more lobes, which is usually followed by signs of softening, provided the patient survives the first week or ten days. The physical signs during the stage of consolidation are precisely the same as in lobar pneumonia. The signs of softening and of cavity will be given in detail below (*vide* Chronic Phthisis).

The *course* is usually rapid, occupying from two to six weeks on the average, though rarely cases that reach the stage of cavity-formation are protracted to three or even four months. Considering the brevity of the attacks, the extreme degree of emaciation (shown especially by the hollow cheeks and temples, pinched nose, and thin hands) is truly remarkable. The patient usually maintains a hopeful state of mind, notwithstanding the rapid downward course of the affection, and it may be admitted that recovery is possible. The parts involved are in such cases destroyed and replaced by fibrous tissue, and it should be remembered that the apex is oftenest involved. It may happen that consolidation only is present in the second lobe affected, while in the upper lobe one or more cavities have already been developed. The pleural crepitating friction is often audible before consolidation is complete.

Diagnosis.—The onset, symptoms, and course during the first week may be those of ordinary lobar pneumonia, but in some cases certain symptoms may arise which will excite suspicion of their tuberculous character in the early stage. Thus, hemoptysis rarely occurs in a pneumococcus infection, and the appearance of the patient, as well as his previous and family history, may also be of a confirmatory character. The points of discrimination have been fully set forth in the section on Lobar Pneumonia (pp. 123, 124).

(2) **Subacute Cases** (rarely acute).—The *onset* is less sudden than in

the former type, while the patient's antecedent condition may either be good or below the *standard*. At the beginning he has *repeated chills*, though *hemoptysis* may be the first symptom which indicates a pre-existing tuberculous focus. The *fever* rises high, and is apt to be irregular from the start; the *pulse* and *respirations* are rapid, and there is a *muco-purulent expectoration* which may either be profuse or scanty. Occasionally it is fetid, and the sputa may early contain *elastic* fibers and *tubercle bacilli*, though more often these are noted after the affection has become fully established. During the progress of the case, also, hemoptysis may arise. Later, drenching *night-sweats* increase the exhaustion and emaciation, which speedily reach an extreme degree, and soon or late a typhoid condition of the system is developed.

The physical signs are, at first, those of general bronchitis, with or without indications of pleurisy. Later, small areas of consolidation, which often increase in size, are indicated by impaired percussion resonance or dulness and by broncho-vesicular (rarely tubular) breathing, with subcrepitant râles. These signs may be unilateral, though more often they occur bilaterally. In many cases softening with cavity-formation ensues, with the usual physical signs of this condition.

Course and Duration.—For some time the patient may remain out of bed, although in most instances the disease constantly progresses. Less frequently there are exacerbating periods and remissions. Rarely these cases recover with a loss of more or less lung-tissue. Again, the condition may pass into chronic phthisis. It is important to recollect that the local lesions may become extensive, as the result of fusion of small consolidated masses, until an entire lobe is involved, and when this occurs the symptoms and course simulate those of the acute type. The *duration* ranges from two to eight weeks or more.

Diagnosis.—This variety is frequently confounded with non-tuberculous broncho-pneumonia, and the chief distinctions will be mentioned in connection with the latter disease. *Bronchiectasis* may be accompanied by emaciation, fetid expectoration, night-sweats, and the signs of cavity, and this disease has been mistaken for acute phthisis. Important in the recognition of the latter, however, are marked fever and emaciation. Moreover, the physical signs are more frequently referable to the apices, and the disease is more steadily progressive, running a shorter course than bronchiectasis. The sputum contains tubercle bacilli.

Acute Broncho-pneumonic Phthisis in Children.—The belief that the form of broncho-pneumonia that so frequently follows certain infectious diseases (measles, whooping-cough, etc.) is in the majority of instances tuberculous has been steadily gaining. Osler recognizes three groups of cases: (*a*) Those in which the child suddenly becomes ill while teething or during convalescence from fever, with high temperature, severe cough, and the signs of consolidation of one or both apices. Death may occur within a few days. To the naked eye the lesions do not appear to be tuberculous. (*b*) In this group the children show the ordinary symptoms of broncho-pneumonia, and the cases are more protracted, death occurring about the sixth week. (*c*) The child feels ill during convalescence from an infectious disease, fever, cough, and dyspnea being present. The intensity of the symptoms abates within a fortnight, and the physical examination shows the presence of diffuse bron-

chitis with scattered minute areas of consolidation. Many of these cases develop into chronic phthisis.

Chronic Tuberculosis.

(*Chronic Pulmonary Tuberculosis; Chronic Ulcerative Phthisis.*)

This form is much more common than the acute, the term embracing sub-varieties to which attention will be incidentally directed. Its most typical clinical form follows a mixed infection as a result of a septic element superadded at some time to the primary tuberculous infection.

The Causal Factors have been detailed under General Etiology.

Pathology.—The pathologic characters of tuberculosis in general have been already presented, but it will be necessary to describe briefly the special anatomic conditions met with in *chronic ulcerative phthisis.*

The post-mortem appearances of the lungs in chronic pulmonary tuberculosis are remarkable for their great diversity, not only in the extent of tissue involved, but also as to the character of the morbid processes. Often the associated lesions form no unimportant part of the picture. In nearly all fatal cases the most advanced and extensive lesions are found near the apex, and, as a rule, the entire upper lobe of one of the lungs is implicated. In addition, it is observed that the destructive process has extended to the lower lobe of the same side, and later to the apex of the opposite lung. Though both lungs are affected in fatal cases, they represent different stages of the disease. The case is very different in an old and cured tuberculosis of the lungs, such as is frequently met with in persons who have died of some other affection. Here the lesions may occupy but a small part of one lung, and usually near the summit.

Kingston Fowler has investigated the question of the points of election and paths of distribution of the lesions in chronic phthisis, and has found that the primary lesion is not, as a rule, at the summit of the upper lobe, but that it occurs from 1 to 1½ inches (3.79 cm.) below this point and near the postero-external borders. Favored by normal respiration, the lesions advance downward, so that on physical examination the first evidences of disease are to be found posteriorly over the lower part of the supraspinous fossa, while anteriorly the early signs are met with immediately below the middle of the clavicle, extending along a line running about 1½ inches (3.79 cm.) from the inner end of the second and third interspaces. The starting-point, though less frequently, may also be indicated by physical signs in the first and second interspaces below the outer third of the clavicle, with subsequent downward extension.

From personal observation of the post-mortem lesions of this disease, and from my studies at the bedside, I feel convinced that the initial lesion is frequently located anteriorly and near the apex, corresponding on the chest-walls to the clavicle and the supraclavicular spaces. This site has seemed to me to obtain more often on the right side than on the left. Kingsley has shown that when the lower lobe becomes involved the consolidation begins about 1½ inches (3.79 cm.) below its apex posteriorly, and corresponding externally to a spot opposite the fifth dorsal spine. From this point it spreads downward and laterally in a line fol-

lowing the border of the scapula "when the hand is placed on the opposite scapula and the elbow rests above the level of the shoulder." The middle lobe on the right side is usually invaded by direct extension from the upper. The seat of primary infiltration may even be the lower lobe, but this is rare. Cole[1] has found lesions at the root to precede parenchymal changes.

The relative frequency of involvement of the two sides varies according to different authorities. A careful analysis of my records and those of other observers show that out of a total of 1236 cases 726 occurred on the left side and 510 on the right.

In all cases the primary lesions are due to *tuberculous infiltration*, which at first is confined to certain lobules, though it may later involve extensive areas of lung-tissue (*tuberculous broncho-pneumonia.*) In most instances the starting-point of the morbid changes is in the smaller bronchi and also, according to Payne, the inside of the alveoli. Soon the bronchioles and the corresponding air-cells become blocked with inflammatory products. These areas then undergo caseation and present the usual opaque, grayish-yellow appearance, a cross-section of these yellow nodules showing the central bronchus usually plugged with exudate and surrounded by caseous matter. Softening and sometimes complete liquefaction, with expectoration or absorption of the altered morbid products, may take place, and this disintegration is associated with *ulceration* in the wall of the bronchus, consequent upon secondary pyogenic infection, and a resulting formation of small *cavities.* Ulcers may form in the bronchioles before necrotic processes supervene, and they are generally shallow, with sharply-defined edges. Recovery may ensue as the result of *calcification* with encapsulation of the cheesy masses, or the affected area may undergo *fibroid transformation*—a conservative process and one that may lead to actual cure. It often happens, however, that old and apparently healed tuberculous lesions undergo ulceration, when the calcareous masses (pulmonary calculi) may be dislodged and expectorated, and the more rapidly the caseous masses are formed the more liable are they to softening. Surrounding the healed areas the tissue may be the seat of atelectasis, though more often of emphysema. Destruction of lung-tissue also results from interstitial inflammation with the formation of new connective tissue, the latter in turn compressing and finally obliterating the alveoli.

Cavities (*Vomicæ*).—These result chiefly from progressive necrosis and ulceration. They are formed mostly by dilatation of the bronchi, whose walls are tuberculous and suppurating. But they may also arise independently of the bronchi. Cavities vary largely in number, size, and form. They are often multiple, though usually not far removed from one another, and unite as they increase in size. In this way large cavities, with irregular walls, involving the whole of one lobe and even an entire lung (except the extreme anterior margin), may be formed, and small pockets connecting with the bronchus may thus originate.

Vomicæ may be classified as (1) progressive and (2) non-progressive.

(1) The **progressive** are divisible into (*a*) New cavities and (*b*) Old cavities.

(*a*) *New cavities* have soft, necrotic, friable walls so long as the de-

[1] *Amer. Jour. Med. Sciences*, July, 1910.

structive processes are rapidly progressing, and the same state of things prevails in the cavities of acute phthisis. They may develop near a healed focus or near old cavities with limiting walls, and when situated near the periphery of the lung they may rupture into the pleura, causing pneumothorax.

(*b*) *Old cavities*, as a rule, have sharply-defined walls that vary considerably in thickness. At first they consist of a fibro-vascular zone, which has an inner suppurating surface; subsequently the lining of this zone is converted into an exfoliating membrane. The contents of vomicæ are muco-purulent or purulent, and often consist of a shreddy and sometimes a bloody fluid. Rarely they are gangrenous. Cavities also contain tubercle bacilli and other micro-organisms. Percy Kidd has studied the question of the relation of tubercle bacilli to tuberculous pulmonary lesions, and states that they are invariably present in newly-developed tubercles and fresh cavities, but frequently absent in old nodules. Trabeculæ composed of blood-vessels and remnants of pulmonary tissue often traverse the cavities. In old cavities excavation may be complete, not a vestige of normal or diseased tissue remaining in them, though the blood-vessels, many of which are beaded by small aneurysmal dilatations along their course, are the last to disappear. Their removal is effected by an obliterating inflammation. Rupture of these miliary aneurysms or the erosion of a large vessel gives rise to copious hemoptysis. Cavities having dense walls may also increase in size by encroaching upon the surrounding tissue, huge cavities often having thin, tense walls. But, wherever situated, they usually begin toward the summit of the upper lobe. Another common seat is the mid-dorsal region.

(2) **Non-progressive Cavities.**—Quiescent cavities are usually small, though variable in size, according to the stage at which the process of contraction is arrested. Medium-sized and large vomicæ do not become totally occluded. They may be multiple, though more often perhaps single, and associated with them may be observed dense, fibrous nodules representing healed foci. Their interior may be lined with a smooth, cuticular structure resembling mucous membrane.

Interstitial Pneumonia.—In the course of chronic phthisis interstitial inflammation of two sorts will most probably arise: (*a*) A consolidation excited by the tubercle bacilli themselves, and hence manifesting a *destructive* tendency; (*b*) A slowly-developed *interstitial pneumonia* which aims at *arresting* the progress of the affection. It develops in close proximity to caseous masses and around cavities. The new connective tissue thus formed in obedience to the well-known pathologic law tends to contract secondarily, and thus vomicæ are often partly, though seldom entirely, obliterated. The shrinking of the connective tissue may also result in compression, and finally in the destruction of pulmonary tissue, just as in a tuberculous inflammation. The process in this instance, however, is on the whole conservative and reparative.

Disseminated Tuberculosis.—*Miliary Tubercles.*—This form has for its chief characteristic miliary tubercles, which are scattered not only about the tuberculous area, but also throughout the rest of the lung, and usually in the lower lobe. Most of the tubercles undergo fibroid or fibro-caseous change. These minute, hard gray or grayish-yellow

nodules vary in size from a mustard-seed to that of a pea, and lung-tissue that is more or less studded with chronic miliary tubercles is apt to look pale, while the surrounding air-cells are *emphysematous*. The condition may lead to pneumonia, and the whole aspect then becomes altered. Here, as before described, fusion of miliary tubercles results in larger masses which become caseous, and hence the method of cavity-formation is identical with that observed in tuberculous broncho-pneumonia. In the disseminated form tubercles may also be found in many other organs than those indicated (pleura, trachea, larynx, bronchial and other lymphatic glands, peritoneum, spleen, kidneys, liver, brain, mucosa, testes, etc.).

Lesions of the Pleura.—This membrane is hyperemic and coated with fibrinous exudation coextensively with the affection of the parts in chronic ulcerative phthisis. The pleural membranes are only more or less thickened by organized adhesions, but in the latter and also in the pleura tubercles or cheesy masses may be found. Simple and other forms of pleurisy are met—sero-fibrinous, purulent, and hemorrhagic.

Lesions of the Bronchial Glands.—At first these are enlarged and edematous, containing tubercles, and later they present foci which often undergo purulent disintegration and sometimes calcification. Other lymphatic glands than these may be affected (mesenteric, etc.).

Lesions of the Larynx.—The larynx is frequently the seat of tuberculous infiltration and ulceration, particularly in certain parts, such as the vocal cords, posterior wall, and ary-epiglottidean folds.

Lesions of the Heart.—Tuberculous endocarditis is present in about 5 per cent. of the cases, and congenital stenosis of the pulmonary orifice is noted in not a few instances (Chevers). The right heart is often hypertrophied or dilated.

Other organs may present lesions in chronic phthisis, and these will be spoken of in connection with the clinical history.

Tuberculosis of the intestinal canal is a common though late lesion.

Amyloid degeneration of certain organs is a not unusual secondary event, especially of the kidneys, liver, spleen, and intestinal mucosa. Enlargement of the liver due to *fatty infiltration* is sometimes noted.

Clinical History.—The modes of invasion are quite diverse, but with few exceptions the onset is either (1) gradual or (2) abrupt, and, as a rule, the health has been previously undermined for a longer or shorter period.

(1) **Gradual Onset.**—(*a*) The disease often originates in a manner similar to *ordinary bronchitis*, and the symptoms of pleurisy are sometimes associated. Tuberculous bronchial affections often follow certain acute infectious diseases—influenza, typhoid, measles, whooping-cough—and in this form are rarely curable. The *physical signs* may be negative for some time, and then appear in the apex region, and the most characteristic grouping of physical signs during the incipient stage may be thus summarized: "Lagging" or defective expansion, as noted on inspection and palpation, a localized increase in the tactile fremitus, slightly impaired percussion-resonance, enfeeblement of the normal vesicular murmur, with (at a later period) prolongation and sharpening of the expiration. The fact that the lesions are commonly detectable in the suprascapular fossa must be remembered. At this period obvious *constitutional disturbances* are present (debility, fever).

(*b*) *Onset with Pleurisy.*—This may be sudden, as in an acute pleurisy with effusion, but often the latter condition develops insidiously. Of 90 cases of pleurisy with effusion, one-third terminated in chronic phthisis (Bowditch). It may begin as a dry pleurisy at the apex, either anteriorly or posteriorly, or the evidence of pleurisy may be associated with the more common bronchitic onset.

(*c*) *With Gastro-intestinal Symptoms.*—There is impaired digestion, and soon the patient becomes anemic, loses flesh, and is debilitated. Later, the first indications of pulmonary tuberculosis develop in the lungs. Close scrutiny of the data entering into the early history of cases of pulmonary tuberculosis usually reveals some perversion of the general health before distinctive pulmonary phenomena arise.

(*d*) *With indefinite peritoneal symptoms*, lasting for months or years.

(*e*) *With Laryngeal Symptoms.*—This is a rare form. It begins with hoarseness, more or less aphonia, and considerable cough; there is also a slight mucopurulent expectoration. Laryngoscopic examinations may detect tuberculosis of the organ, and tubercle bacilli may be found in the sputum before involvement of the lungs is discoverable.

(2) **Cases with Abrupt Onset.**—(*a*) The most important group under this category is heralded by the symptoms and signs of *pneumonia*, more commonly of the lobular variety. As compared with lobar pneumonias, these present peculiar features: the fever is irregular, the expectoration is more abundant, is blood-stained, and contains bacilli. The signs are usually located in the apical region. Resolution may occur, but recovery is not complete, and the condition may pass into chronic phthisis.

(*b*) *Onset with Fever.*—Chills and fever generally arise in the advanced stage of pulmonary tuberculosis, but these symptoms may also initiate the attack. There is no mistake in diagnosis more commonly made in malarial regions than to ascribe such cases to paludism.

(*c*) *With Hemoptysis.*—This symptom may invite attention to lung trouble. Müller states that hemoptysis was an early symptom in 170 of the 875 patients at the Davos German Sanatorium, and was twice as frequent in the male as in the female. The amount of blood lost is either considerable or repeated slight hemorrhages occur. In a great proportion of cases the clinical picture of incipient pulmonary tuberculosis is revealed, pursuing its accustomed course immediately after the occurrence of the hemorrhage. The physical signs may be latent for a time, and, while they are usually located in the subapical area, they may assume the guise of a pleurisy in the infrascapular region. A slight tuberculous lesion is present in these cases preceding the occurrence of the hemorrhage.

The **symptoms** are (1) *local* and (2) *general.*

(1) Local.—(*a*) *Pain.*—This is absent in many cases of chronic phthisis and in others it may be moderately severe. It is seated usually at the base, laterally or anteriorly, and not rarely there is pain of a lancinating character in the interscapular region in the early stages of the affection. This symptom is of diagnostic worth only after other forms of pain (rheumatic, neuralgic) have been excluded. The most common cause of pain is pleuritis, with or without pleuritic adhesions: it is increased on deep breathing and coughing. Intercostal neuralgia and pleurodynic stitches may also develop soon or late. Tenderness on

pressure with the right forefinger (algeoscopy), which causes the patient to exclaim or make a grimace, or merely a contraction in adjoining muscles, was present in 77.9 per cent. of 200 cases studied by Francke, while only one-third of these patients complained of spontaneous pain.

(*b*) *The Cough.*—This may be looked upon as an essential feature, though in a few instances it may be slight or even wanting throughout. Its severity bears no constant relation to the extent of the pulmonary lesions, but rather to the degree of sensitiveness of the patient. It is dry and hacking at the beginning, and, if the larynx be involved, the cough is marked and of a hoarse quality. It is most pronounced at certain periods of the day—viz., on lying down at night and on awakening from sleep. Paroxysms may occur after meals and induce vomiting. The cough is at times distressing and debilitating in its effects.

(*c*) *Expectoration.*—At the beginning the sputum is scanty and mucoid, rarely hemorrhagic, or it may be merely streaked with blood; later it may become muco-purulent, and the appearance of small gray or grayish-yellow flocculi first suggests the nature of the affection. With the onset of the stage of cavity-formation the sputum becomes more abundant and more distinctly purulent, and, after the formation of cavities of any size, airless, opaque, and nummular (coin-shaped) masses are expectorated. The latter are greenish-gray or greenish-yellow in color, and sink rapidly when discharged into water. They are often mingled with more or less bronchial secretion, and are sometimes observed in pure bronchitis. They may even be absent, and the expectoration be merely purulent. The opening of a fresh cavity may be followed by very free expectoration. The sputum is sometimes fetid, and exceptionally it is horribly offensive, varying greatly in amount in different cases and at different stages of the disease. In certain cases it is absent throughout the greater portion of their course, and is especially apt to be slight in children and old people. In such instances it may be impossible to collect sufficient sputum to examine for bacilli.

Microscopic examination discovers alveolar epithelium (particularly in the earlier stages), pus-cells, blood, fat-globules, elastic fibers, and *tubercle bacilli*, the detection of the latter being the most important factor in the diagnosis. It may be safely stated that the finding of bacilli in the sputum is *prima facie* evidence of chronic phthisis; on the other hand, however, their absence in the early stage does not exclude the disease. It is often needful to make repeated and delicate examinations of the sputa. It is also of the utmost importance to select for examination the small grayish masses that are usually to be found, since they early contain the bacilli. In tuberculosis in the aged tubercle bacilli are not always detectable in the sputum.

Method of Examining the Sputum.—"A small amount of the purulent portion of the sputum is spread in a thin and uniform layer on a perfectly clear cover-glass by means of forceps, needles, or the Ohse, which must previously be held a moment in the flame of a Bunsen burner or a spirit lamp, or by pressing a small amount of sputum between two cover-glasses, then sliding them apart. It is then dried in the air, or more quickly by holding the cover-glass with forceps some distance above the

flame of a burner or lamp. Finally, it is to be passed three or four times through the flame, and so 'fixed'" (Musser). Brown and Smith[1] recommend antiformin for the cultivation of tubercle bacilli directly from, and also to digest, the sputum.

The preparation may be stained with carbol fuchsin (basic fuchsin 1, alcohol 10, 5 per cent. solution of carbolic acid 90), either by dropping a few drops of the stain on the smeared side of the cover-glass and holding it above the flame until it steams, or by floating its face downward upon a watch-crystal containing the solution. It must then be decolorized either with a 30 per cent. solution of nitric acid, allowing it to remain until the red color has entirely disappeared (about fifteen seconds), and then washing and counter-staining with methylene-blue, or with Gabbett's solution (methylene-blue 2 gm., sulphuric acid 25 c.cm., water 75 c.cm.), in which it must remain until the red color has been replaced by a faint blue (thirty seconds or more). Instead of carbol-fuchsin, anilin gentian violet may be employed (add a saturated alcoholic solution of gentian violet to a filtered saturated solution of anilin until a metallic luster appears on the surface). The specimen may lie either several hours in a cold solution or a few minutes in one that is steaming. Decolorize with the nitric-acid solution ($\frac{1}{2}$ per cent.), and counterstain with rubin or a saturated aqueous solution of Bismarck brown. It is often much simpler to smear the sputum directly upon the slide, and then examine, when stained, without the intervention of a cover-glass. A much larger amount of sputum can thus be prepared.

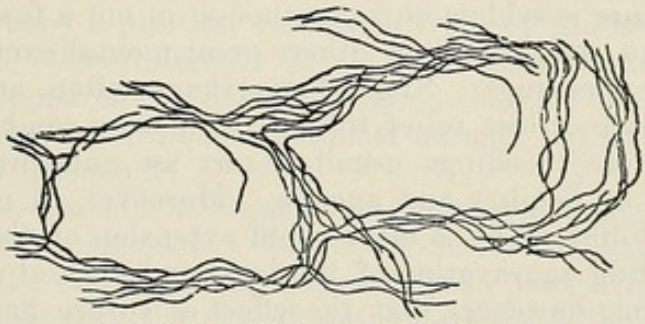


FIG. 21.—Elastic fibers (after Strümpell).

In the microscopic examination use a $\frac{1}{12}$-inch (2.11 mm.) oil-immersion lens and Abbé condenser. If carbol-fuchsin has been used in staining for the bacilli, and methylene-blue as a contrast, the former will be found as red rods in a blue field (background), while if gentian-violet has been used, the tubercle bacilli appear as dark violet rods, with all other bodies brown, if Bismarck brown is used for the contrast stain. There may be visible in the field a few bacilli only, particularly during the early part of the case. In the stage of cavity their number is usually increased, and sometimes they are quite numerous.

The demonstration of *elastic fibers* is also an important aid to diagnosis. Fenwick's method is the following: Boil the sputum with an equal quantity of a solution of caustic soda (gr. xv–$\bar{3}$j—0.972–32.0); pour the product into a conical glass and fill with cold water. The sediment is subsequently examined with care for elastic fibers.

The *form* and *appearance* of the elastic threads differ according to

[1] *Jour. Med. Research*, Boston, 1910, xxii., 517.

their special source. If they come from the alveoli, there is an interlacing of the fibers which may preserve the globular contour of the air-cells. If they come from the blood-vessels, they are single and elongated, or two or three of the fibers may be arranged side by side. Elastic tissue derived from the bronchi has a similar appearance.

The presence of elastic fibers furnishes incontestible proof that destruction of lung-tissue has taken place. To show that this loss of structure, however, is due to tuberculosis, we must exclude abscess (rare) and gangrene of the lungs—diseases in which it also occurs.

(*d*) *Hemoptysis.*—This symptom of phthisis will be spoken of under Diseases of the Lungs, but its importance as a diagnostic feature of this disease makes special reference to it here absolutely necessary. It is present in the majority of cases. Gabrilowisch [1] found that of 380 patients 213, or 56 per cent., had hemoptysis. The sputum may be merely blood-stained, or the hemorrhage may be excessive and prove rapily fatal, though hemoptysis is rarely the direct cause of death in tuberculosis. Slight hemorrhages are usually produced by mere hyperemia, and are most apt to occur during the early stages; while severe bleedings are produced by the erosion of a blood-vessel or rupture of a small aneurysm, and are most prone to occur during the stage of cavity. In certain cases hemoptysis is frequent.

A third or capillary form of hemorrhage may occur in phthisis with cavity-formation, and in this variety, which is of a rather frequent occurrence, the purulent sputum is uniformly stained with blood. It may also be nummular, but presents a reddish-brown or chocolate color. The ***exciting cause*** is seldom obvious, though in not a few instances aggravation of the cough, and in others great mental excitement, would appear to excite bleedings. Slight hemorrhages often, and severe ones rarely, afford more or less relief to the pulmonary condition. On the other hand, severe bleedings usually exert an unfavorable influence, being followed by debility and anemia. Moreover, in numerous cases hemoptysis is followed by a more rapid extension of the local lesions, with corresponding aggravation of the local and general manifestations. The fact remains, however, that the effect of severe hemoptysis upon the progress of chronic phthisis is by no means always untoward. In a case of my own there occurred periodically copious spontaneous bleedings (in spring and fall) for three years, which were as regularly followed by marked improvement for a period of three or four months. The physical signs of phthisis then developed. In a large number of cases of pulmonary tuberculosis the transition from warm to cold or cold to warm seasons corresponds with increased cough, hence with increased pressure in the pulmonary circulation; and so bleeding is also favored, particularly in those having a hemorrhagic tendency.

(*e*) *Dyspnea* is present, but is not a marked feature, as a rule, despite advanced pulmonary lesions. Perhaps the chief reasons for a lessened demand for oxygen on the part of the system are—first, the slow and gradual manner in which the lesions develop; and second, the pronounced bodily wasting. The *respirations*, however, are moderately increased in rate, averaging from 20 to 30 per minute, and this compensates admirably for the diminished breathing-space. The dyspnea may

[1] *Berliner klinische Wochenschrift*, Jan. 2, 1899.

be greatly intensified, however, as the result of intercurrent pneumonia, pleurisy, active exertion, or great mental excitement, and toward the close of fatal cases the most intense dyspnea may be manifested.

Physical Signs in the Stage of Consolidation.—*Inspection* gives most important results. The paralytic or phthisical thorax is generally presented to view. It is flat, particularly the upper half; the intercostal spaces are wide; the ribs slope at a sharp angle from the sternum, making the epigastric angle acute and producing elongation of the chest. The same sharp inclination downward from the vertebral column is observed laterally and posteriorly. The angle of Louis is prominent, and the depressions (supra- and infraclavicular, intercostal) are deepened, the costal cartilages being often prominent and the sternum, particularly in the lower part, sometimes much depressed or even concave (funnel-breast). The scapulæ stand out prominently and may be distinctly winged. A second type of paralytic thorax is narrow and long. Pulmonary tuberculosis may, however, arise in chests of apparently normal build. The paralytic thorax is often a resultant of developed phthisis. In subjects of obesity the phthisical thorax may be concealed. The deformity due to occupation, as leaning over a desk, may ape the paralytic chest, and, finally, it may be the result of extreme emaciation. With the development of phthisis the depressions of the side affected are relatively deeper, while the clavicle often stands out prominently.

Defective expansion is observed early, and usually at the apex of the side first affected; subsequently this may be more general, and finally bilateral. To note the motions of respiration with precision the examiner should occupy a position exactly in front of the median line of the patient's body. The difference in the movement of the two sides often becomes more apparent on deep respiration than on quiet breathing, and while at rest the respirations are almost normal, but exertion decidedly increases their frequency.

Palpation.—Testing the expansion by palpation gives better relative results than does inspection. To determine the comparative movements of the apices the extended hands should be so placed (by allowing them to diverge below) that the tips of the fingers touch the lower border of the clavicle, and then the patient should be asked to breathe deeply, though slowly. The expansion in the supraclavicular spaces is tested by standing behind the patient and using the tips of the fingers, or by allowing the two first fingers of each hand to pass parallel with the clavicles. In this way "lagging" over the apex will be the first symptom recognized, and may for some time be the only one. Palpation of the vagus nerve on the affected side elicits pain (Mays).

Tactile fremitus is early increased with oncoming consolidation, though it is normally more marked at the right than at the left apex. If there be thickening of the pleura, however, it is diminished, and if there be pleural effusion it may be absent.

Mensuration.—The difference between the measurement of the chest in inspiration and expiration in any person of average health should be not less than three inches, and a difference below two and a half inches points strongly to tuberculosis. The data thus gained are more important than the shape of the *thorax*.

Percussion.—Resonance is deadened more and more as consoli-

dation progresses. If the consolidated areas are minute, however, the percussion-note may be unchanged, and as the air-cells surrounding the latter are often emphysematous and relaxed, it may be somewhat tympanitic. The tympanitic sound and deadness may be intermingled, giving rise to the so-called tympanitic deadened sound. Slight dulness is, as a rule, noted first below the clavicle, though in not a few cases it is first detected above the clavicle. Impaired resonance, however, may be detected, first, in the supraspinous fossa, and less frequently in the interscapular space if the subject is not too stout, though slight dulness in the absence of other signs has little diagnostic value. The corresponding regions of the two sides must be compared during a held inspiration and also during a held expiration. The degree of dulness can sometimes be better estimated by comparing the apical note with that obtained lower down on the same side, allowing for the normal topographic differences of intensity. The latter method is especially applicable to cases in which both apices are involved. Light and single percussion blows must be used. As the lung-tissue becomes airless throughout an area of considerable size the note is deadened, until dulness is heard; finally, with extensive consolidation, the note may be wooden and the feeling of resistance increased.

Auscultation.—The vesicular breathing may be sharpened, owing to narrowing of the smaller bronchi, but more often perhaps it is diminished by the swelling and secretion. The corresponding regions on the two sides must be compared—first during quiet, and then deep breathing—and it should be remembered that prolonged expiration is an early and important diagnostic sign, at first being somewhat sharpened, and later distinctly bronchial. Tuberculous bronchitis may cause interrupted or jerking inspiration at the apex with or without crepitant râles. If heard elsewhere, it has small value. With lobular consolidation at different points in the region affected, the conditions favor the transmission of the bronchial sounds, but these are toned down by the remaining intact air-cells; hence there is "transition" or bronchovesicular breathing. With complete consolidation pure bronchial breathing is audible, and with the latter two forms of breathing crepitant or subcrepitant râles are heard. A clicking râle, although not common, is an almost conclusive indication when observed. Sometimes the first râles which accompany expiration have a low whistling sound; with liquefaction they become more moist, are louder (somewhat ringing), and often bubbling, and may be heard on inspiration and expiration. If scanty, they may be audible on inspiration only; they are increased by coughing. If the moist crepitant and subcrepitant râles, often due to concurrent bronchitis, be very numerous, the breath-sounds will be obscured, but after free expectoration their quality becomes appreciable.

Pleuritic friction-sounds may be heard, due to accompanying pleuritis sicca, and these may be audible before the bronchial râles reveal the disease. Friction-sounds and râles often occur together. *Pleuropericardial friction* is present when the "lappet" of lung over the heart is affected, while clicking râles, occasioned by the heart's systole, are audible when the same area is pneumonic. The vocal resonance increases with the progress of the consolidation, and when the latter is complete, *bronchophony* (rarely *pectoriloquy*) is present. In the subclavian arteries a systolic murmur is not uncommonly heard, the latter being supposed to be due to pressure exerted by the thickened pleura upon these vessels.

Physical Signs of Cavity.—*Inspection* shows a more marked retraction and a more decided lack of local motion than during the previous stage. The degree of shrinking is proportional with the extent of fibrous-tissue formation.

Palpation corroborates inspection as to lack of motion, and gives increased tactile fremitus if the cavity connects with an open bronchus and if it contains but little secretion. Excessive secretion interferes with conduction of sound.

Percussion.—Resonance is generally more or less impaired in consequence of the consolidation of the surrounding lung-tissue. The note may be somewhat tympanitic, but varies with the position of the cavities, the amount of fluid secretion contained by them, the condition of their walls, and the vibratory capacity both of the latter and of the individual thorax. Cavities of the size of a walnut situated in the apices usually give a distinctly tympanitic note, while cavities of the same dimensions, or even larger, in the lower portion of the lung do not. The metallic tone is especially noticeable over large cavities with smooth walls. The tympanitic sound may be deadened by closure of the connecting bronchus and by temporary filling of the cavities with secretion, and, again, if they are surrounded by thickened lung-tissue or by a large thickened pleura, there may be impaired resonance or absolute dulness even. Certain *special conditions* change the tympanitic sound over a cavity. Thus the note will be louder and exalted in pitch when the mouth is opened wide, and lowered when the mouth is closed (Wintrich's sign), there being dulness when the mouth is closed and tympanitic resonance when the mouth is open. If the cavity communicates freely with the bronchus, a tympanitic note may change in pitch with change in posture (Gerhardt's change of sound). If the patient changes from the dorsal to the upright position, resonance may give way to more or less flatness over the lower portion of the cavity, since the fluid contents of the latter are thus brought into contact with the chest-wall; this, although an almost certain sign of a cavity when present, is exceedingly rare. The so-called cracked-pot sound is often elicited over large parietal cavities with thin walls; but, since it also occurs in many other pathologic conditions, its diagnostic significance is subordinate. There may even be normal resonance if the cavity is covered by a layer of unaffected air-cells of considerable thickness.

Auscultation over small vomicæ with lax walls reveals *cavernous* (low-pitched) breathing, while over large cavities with tense walls (if parietal and communicating with a tracheo-bronchial column of air) it gives amphoric (higher-pitched) respiration. Moist râles (bubbling and gurgling, according to the consistency of the secretion) may be present, and these correspond in the main to the amphoric breathing, hence being heard most frequently over large, smooth-walled and peripherally-located cavities. The gurgling and slushing sounds caused by the air bubbling through the secretion in a cavity are always intensified by coughing.

The sounds of falling drops (metallic tinkling) may be heard over large vomicæ with tense, smooth walls containing thin secretion. *Pectoriloquy* and *amphoric whispers* are the vocal sounds heard over huge cavities. Whispering pectoriloquy was present in 55 out of 58 cases at

the Phipps Institute, but other pathologic conditions may cause this sign, "notably consolidation about a bronchus" (Landis).

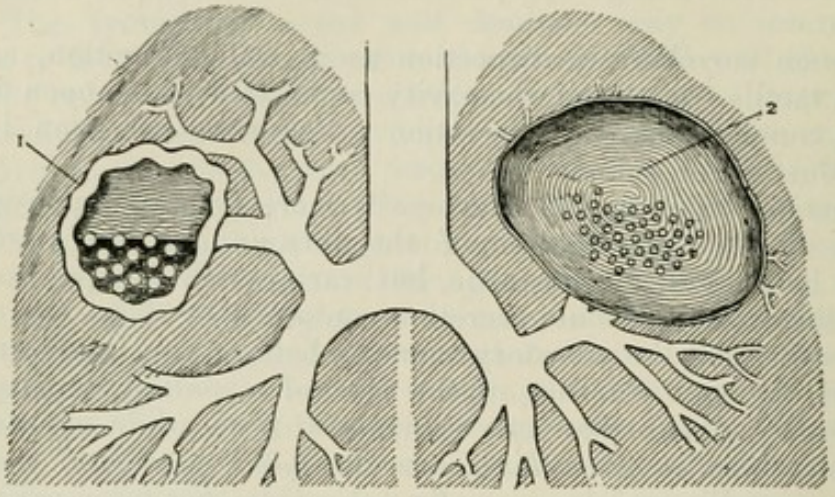


FIG. 22.—1. Small cavity near periphery, with thick relaxed walls, containing secretion and communicating with a bronchus (*vide* subjoined table). 2. Large parietal cavity, with thin, tense, smooth walls, communicating with a bronchus (*vide* table).

Physical Signs.	*Physical Signs.*
(*a*) Percussion-deadness on a strong blow, mere impairment of resonance on a light blow; Wintrich's interrupted change of sound, detectable only when patient is upright.	(*a*) Amphoric percussion-resonance, cracked-pot sound, and Wintrich's change of sound.
(*b*) On auscultation low-pitched cavernous (hollow) breathing; gurgling râles.	(*b*) On auscultation, high-pitched amphoric (musical) respiration and metallic râles.
(*c*) Pectoriloquy indistinct, owing to small size of cavity and the contained fluid.	(*c*) Amphoric (musical) voice and amphoric whisper.

General Symptoms.—(*a*) **Fever.**—Whilst the disease is progressing fever is a constant, significant, and, it may be, the earliest, symptom. If a two-hourly record be kept for a few days, from time to time an accurate conception of the course and type of the fever can be formed. In the first and middle stages the highest temperature occurs about 4 or 5 P. M., the lowest about 4 or 5 A. M. The fever may be continuous, remitting, or intermitting, and in a general way these types, in the order named, correspond to the stages of tuberculization, softening, and cavity-formation. Modified types, due to the fact that the lesions may simultaneously represent different stages, are also observed. Apyrexial periods are met with in the early as well as the late stages of chronic phthisis, and indicate cessation of the processes of tuberculization and caseation.

A *continued fever* is most apt to be met with during the initial period, the evening temperature sometimes registering but a degree higher than the morning. A similar curve may be presented at any later time if acute pneumonia supervene, though it is to be recollected that the remissions in such cases are usually greater than in primary lobar pneumonia.

A *remittent fever* is more common than the preceding type. It may be present from the start, but is oftener seen in the middle and less frequently in the advanced stages. It points to softening (see Fig. 23).

An *intermittent fever* is also frequent, and is invariably associated with cavity formation. The temperature may be intermittent from the

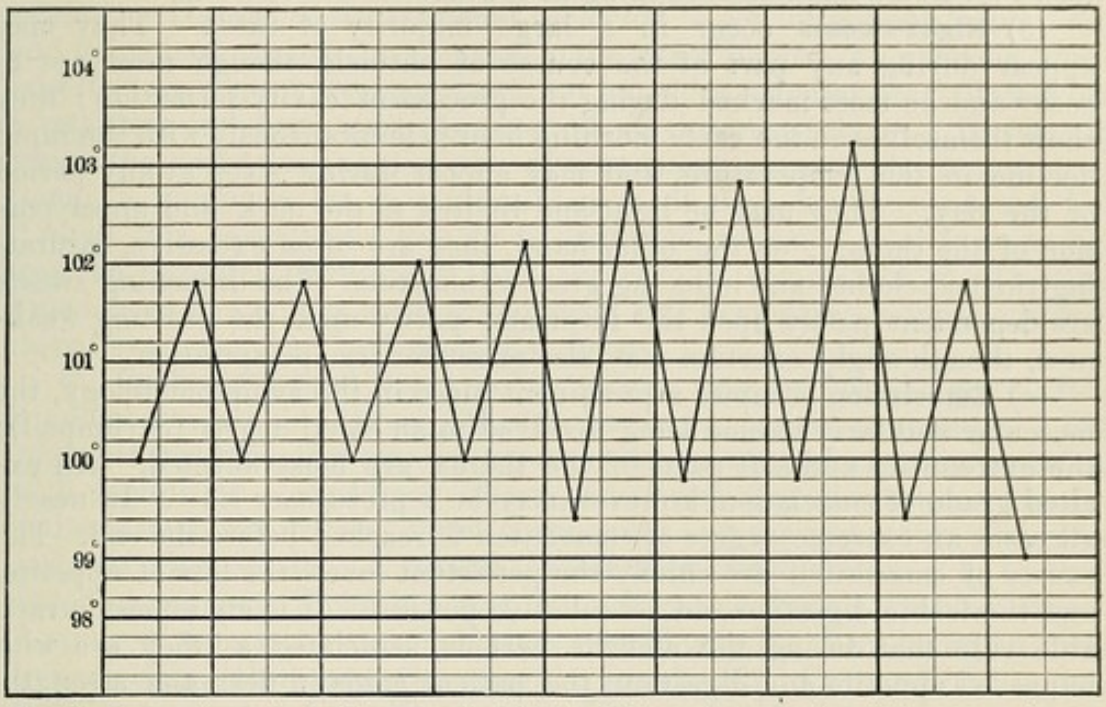


FIG. 23.—Temperature-chart of a case of phthisis. Quiescent cavity in right apex, and commencing excavation in left apex. Robert G——, aged 21 years; dyer.

start, suggesting malaria to the unguarded; but it is due to sepsis, the temperature rising during the day, beginning usually shortly before

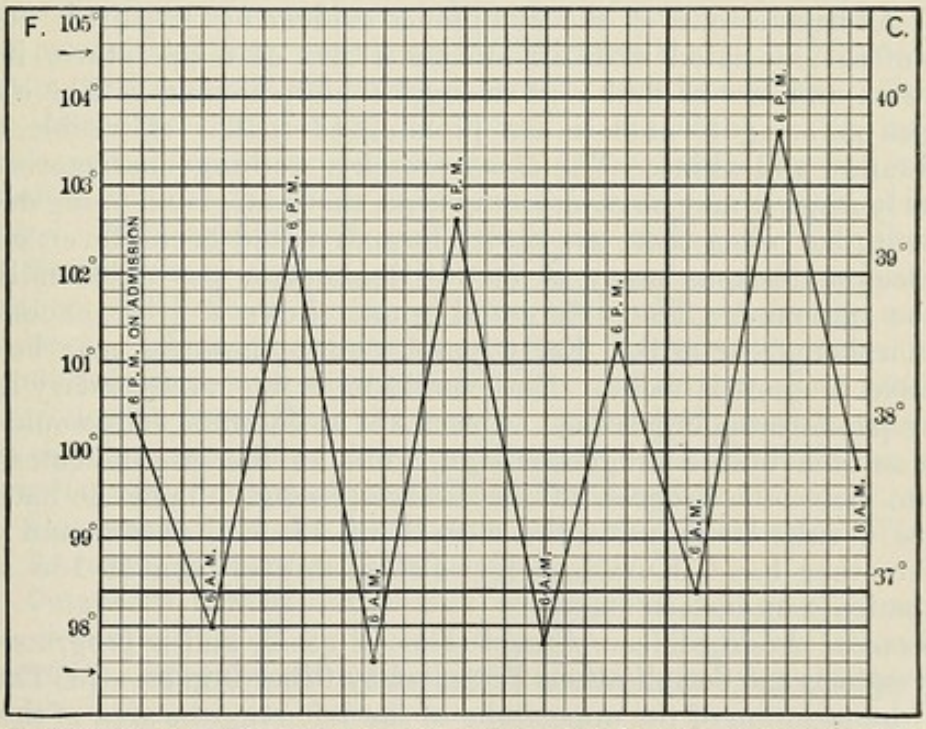


FIG. 24.—Temperature-chart of a case of phthisis. Cavity in left apex, giving cracked-pot sound, Wintrich's sign, etc. George C——, aged 22 years; glass-worker.

noon, and reaching its maximum at from 5 to 8 P. M. It now falls slowly until about 4 or 5 A. M., and then rapidly reaches the minimum—a sub-

normal point—usually at from 6 to 10 A. M. For a considerable portion of every twenty-four hours the temperature may be below the normal (see Fig. 24), sometimes dropping as low as 95° F. (35° C.).

(*b*) **Night-sweats** occur in a large majority of cases. They may appear during any part of the course of phthisis, though most apt to occur and be most marked during the process of cavity-formation; they show themselves in the early morning hours simultaneously with the rapid decline in the temperature, and may appear during sleep at any period of the day. They may be light and limited to the neck and upper portion of the thorax; on the other hand, they are often excessive, saturating the bed-clothes and inducing great exhaustion. The drenching sweats are dependent partly upon the fever and partly upon the existing weakness, though slight exertion may also engender free perspiration.

(*c*) **Emaciation** occupies a prominent place in the symptomatology, the muscular and fatty tissues being involved to an equal degree (Strümpell); the extremities and soft parts of the thorax are most affected. An exalted grade of emaciation, however, may be a precursory state. In nearly all cases an extreme degree of emaciation is reached before the end. The causes of emaciation are chiefly the persistent fever, the loss of appetite, and the feeble digestive and assimilative powers. It is an almost invariable rule that during the afebrile periods, associated as they are with improved appetite and digestion, the patient gains in flesh and strength. Unilateral atrophy of the muscles of the chest may be observed.

(*d*) The **pulse** is increased in frequency, is of good volume and regular in rhythm, though of low tension (soft). When suppurative fever sets in it becomes frequent and compressible, and the capillary pulse is often observed; rarely venous pulsation is seen in the hands.

(*e*) **Anemia** is one of the symptoms evidencing impaired nutrition. It is often associated with an afternoon rise of temperature, impaired digestion, and loss of flesh and strength (chloro-anemia). The objective changes pointing to anemia are pronounced (pallor of visible mucous membranes, and skin). The *blood* presents nothing characteristic. In the early stage it may be chlorotic in type, the hemoglobin being decidedly deficient; but when there are cavity formation and hectic fever, considerable leukocytosis, as many as 50,000 leukocytes per cubic millimeter, may be observed. The differential count shows a great excess of the polymorphonuclear cells. Early lymphocytosis, however, may be of considerable diagnostic value. The condition is due to secondary infection by the pus-forming organisms. Absence of eosinophile cells would appear to be an unfavorable prognostic sign, while an increase indicates a tendency to arrest the progress of the disease (Swan). Tubercle bacilli cannot, as a rule, be found in the circulating blood (Ravenal and Smith). On the other hand, Kurashige found them in every one of 155 tuberculous patients in various stages.

General debility is complained of in all cases, and is progressive.

Symptoms and Complications Presented by Other Organs.—(*a*) **The Heart.**—With retraction of the upper lobe of the left lung the area of the heart's impulse is obviously increased, particularly upward, so that pulsation may be visible in the fourth, third, and even second interspaces, near the sternum, while the normal apex-beat may be wanting. The physical signs noted may be rarely those of displacement of the heart to the right, while the necropsy may show the heart to be in its normal posi-

tion. Functional murmurs both at the apex and at the pulmonary orifices are often audible. In about 7 per cent. of the cases with murmurs, mitral regurgitation, dependent on weakness of the heart muscles, was diagnosticated.[1] Disease of the tricuspid segments is not infrequent in phthisis, and pulmonary stenosis predisposes to the latter disease. Conversely, there is perfect agreement among writers that left-sided valvular heart disease has a retarding influence upon the progress of chronic phthisis. In cases in which the valve lesions and the compensatory hypertrophy are proportional, a prognosis for an unusual length of days can be ventured, but "when this harmonious balance is disturbed an early fatal termination may be expected, principally from the cardiac complaints."[2] In combined cases dyspnea is more pronounced and hemoptysis a more common initial symptom than in non-cardiac forms.

(*b*) **Gastro-intestinal Tract.**—The *tongue* may be furred; more often it and the mouth and throat are red, showing increased irritability. The *pharynx* may be the seat of tuberculous lesions, which may interfere with deglutition. *Aphthous ulcers* and *thrush* may also arise. The appetite is impaired or lost; thirst is annoying and the symptoms of chronic gastritis often obtain. A catarrhal ulceration and dilatation may be associated conditions. Vomiting may be troublesome during the later stages. A study of the gastric secretion gives variable results, there being an early hyperacidity, while later the secretion is subacid. Croner found normal motility present in the early stages. The *causes* of gastric symptoms are not clear. The mucosa is the seat of venous engorgement, and thus occasions the catarrhal changes that are present in many instances. Anatomic changes may be absent.

The *intestinal symptoms* are important. During the early stage constipation is a frequent condition. Diarrhea is prone to appear at an advanced period, and may pursue an intermittent course. Occasionally it alternates with periods of "hectic fever," and late in the affection a watery discharge may develop (*colliquative diarrhea*). The intestinal lesions are of three sorts: (*a*) *catarrhal*, (*b*) *ulcerative*, and (*c*) *amyloid*. These often arise in the order enumerated, but may be combined in various ways. Hemorrhoids and anal fistulæ are among the complications.

(*c*) **Genito-urinary Organs.**—There is frequently an albuminuria that may either be febrile or due to chronic nephritis (*productive* and *non-productive*). *Chronic nephritis* is usually a late development; it gives rise to albuminuria, tube-casts in the urine, and dropsy. The total nitrogen excretion is lower than the normal. *Amyloid changes* may set in toward the close with their characteristic symptoms. Tuberculous *pyelitis* and *cystitis*, with the appearance of pus and blood in the urine, may develop. Hematuria may also result from temporary congestion. The testes should be routinely inspected (Osler).

(*d*) **Cutaneous System.**—*Cyanosis* occurs, but, being of a moderate degree, it is often veiled by a decided pallor. The *cheeks* often wear a "hectic flush," and the skin, late in the affection, is apt to be dry, harsh, and scaly. Among the cutaneous appearances are pigmentary stains over the chest (chloasmata phthisicora) and brown stains (pityriasis versicolor). Rarely, simple purpura and purpura hemorrhagica develop as

[1] "A Study of Murmurs in Pulmonary Tuberculosis," *American Journal of the Medical Sciences*, June, 1910, by C. M. Montgomery.

[2] *Amer. Jour. Med. Sci.*, Jan., 1902, by the writer.

late complications. The *hair* over the chest often becomes gray; that of the head and beard, long and harsh. The *finger-ends* are often bulbous (clubbed), with incurved nails, though this is not peculiar to chronic phthisis, and cracking of the finger-nails is also often observed.

(*e*) **Nervous System.**—The mental attitude is characteristically hopeful and buoyant, even in the advanced stages. Hence the patients are readily encouraged by the unscrupulous to believe that their condition is improving; they may be in an utterly helpless state, and yet confidently expect to recover. The *cerebral symptoms* are rarely marked, and the mind, as a rule, is exceptionally clear. Tuberculous meningitis and meningo-encephalitis may develop near the close. Focal lesions, due to the presence of tubercles, may produce forms of paralysis (aphasia, hemiplegia) according to their location. Rarely peripheral neuritis (usually an extensor paralysis of the leg) and insanity are observed. There may be early unilateral dilatation of the pupil, due to enlargement of the bronchial lymph-nodes on the corresponding side.

(*f*) **Chest-muscles and Mammary Glands.**—The former are abnormally irritable, and sometimes even painful on percussion, and the mammary gland is in rare instances hypertrophied, males suffering most; but, as pointed out by Allot, the affection is a chronic non-tuberculous mammitis.

Diagnosis.—The early recognition of chronic pulmonary tuberculosis often tests severely the diagnostic acumen of the physician. The general and local symptoms, including the physical signs, may afford merely a strong suspicion of the existence of phthisis, and in such instances repeated examinations of the sputum for the bacilli are imperative, and only when they are found is the diagnosis set at rest. Repeated staining of the sputum may be necessary for the detection of tubercle bacilli. It is also desirable to determine whether they are constantly present by re-examinations at intervals. There are cases in which the physical signs are obvious, yet the bacilli are either not detectable or only so after several examinations. An absence of the bacilli, however, does not justify a denial of the existence of phthisis, and is of little negative value. Philip and Porter conclude that tubercle bacilli are almost constantly present in the stools, whether the patient be expectorating bacilli or not. The symptoms of greatest diagnostic value are cough, expectoration, fever, progressive emaciation, and the constant presence of certain physical signs in the subapical region on one side (flattening of the chest, defective expansion, slight deadening of the percussion-note, enfeeblement of the vesicular murmur, prolonged expiration, with or without adventitious sounds). Skiagraphs that show the presence of tuberculous deposits and pleuritic exudates may, at times, give the earliest positive information in regard to these conditions.[1] Again, more reliable knowledge can be gained in the initial stage, if the lesions be deep seated, by the fluoroscope than by practising the physical signs. Thus enlarged bronchial glands and peribronchial infiltration are detectable.

The *tuberculin-test* is warmly commended by Trudeau, Otis, Klebs, and others. It is fairly accurate, and out of a total of 1470 injections in dubious cases, 71.9 per cent. reacted positively.[2] Its use should be

[1] For illustrative cases see "Diagnosis and Treatment of Prebacillary Stage of Pulmonary Tuberculosis," *The Journal of the Amer. Med. Assoc.*, Jan. 12, 1901, by the writer.

[2] "The Value of the Tuberculin-test in the Diagnosis of Pulmonary Tuberculosis," by the writer, *New York Medical Journal*, June 23, 1900.

limited to patients who have symptoms and signs of this disease, since latent tuberculosis gives the reaction, after other methods of diagnosis have failed us, and medium-sized initial doses are to be employed. A positive reaction demands an elevation of temperature to 101° F., and this rise usually occurs within twenty-four hours, but it may be delayed until thirty-six or even forty-eight hours. The possibility of reaction occurring in cases of syphilis, leprosy, chlorosis, hysteria, actinomycosis, and other affections will not lead to error if it is noted that such reactions are less intense. Calmette's ophthalmic reaction, which consists in dropping 1 to 2 mimims of a 0.5 to 1 per cent. solution of tuberculin into the eye, produces hyperemia of the conjunctiva (at times actual conjunctivitis) in from three or four to twenty-four hours without constitutional disturbance. The symptoms subside in from twenty-four to forty-eight hours. Von Pirquet applies the tuberculin with gentle friction to the slightly abraded skin. If the patient be tuberculous, a reaction occurs in from 6 to 48 hours; this is especially valuable in the diagnosis of tuberculosis among children. In adults, however, other signs of tuberculosis must be present to render it of any diagnostic worth. The opsonin test is useful in the diagnosis of early tuberculosis, the index to the tubercle bacillus being very low or very high, the former suggesting predisposition, the latter showing infection against which the resisting powers are raised in defense (J. C. DaCosta). Airlong and Courmont[1] describe a method of serum diagnosis, but from the reported trials of other observers its results are too irregular to be of value. A slight rise of the evening temperature (99.6° F.—37.5° C. or over) is, if associated with any disturbance of health, an almost infallible diagnostic symptom. In the more advanced stages of phthisis the diagnosis is rarely difficult.

In the very early stage the local condition may be obscured by the symptoms of impaired digestion, loss of flesh and strength, fever, and pronounced anemia (chloro-anemia, *vide* p. 270; also *Modes of Onset*).

Differential Diagnosis.—*Bronchial catarrh* is with great difficulty discriminated from beginning phthisis. If the temperature is elevated from 2 to 5 P. M., and not at all or only slightly above the normal night temperature in the evening, the probabilities are greatly in favor of tuberculosis (Barlow). In bronchial catarrh there is no dulness, and moist râles, that vary in intensity from one day to another, are heard equally on both sides. From time to time râles may also be heard at the bases in bronchitis. In phthisis one apex is more involved than the other, the moist sounds not being heard equally low, and after repeated coughs with subsequent deep inspiration the rales are more apt to remain than in ordinary bronchitis. In phthisis, also, there is a gradual loss of flesh and strengh, and repeated microscopic examination of the sputum will demonstrate the presence of the bacillus. A negative reaction, obtained repeatedly, from the Falk and Tedesko test,[2] is evidence that the disease process is limited to the bronchi, while a positive reaction indicates pulmonary involvement (tuberculosis). According to Armstrong and Goodman[3], however, this test is unreliable. If *hemoptysis* be the first symptom observed, then all other causes for the spitting of blood

[1] *Deutsche med. Woch.*, Nov. 29, 1900.
[2] *Wiener klinische Woch.*, Vienna, July 8, 1909.
[3] *Jour. Amer. Med. Assoc.*, May 27, 1911, p. 1553.

should be patiently excluded, unless the associated evidences of commencing phthisis are conclusive. Phthisis in the stage of cavity may be confounded with *bronchiectasis* (*vide* Diseases of the Lungs).

Pseudo-tuberculosis.—By this term is meant a distinct form of pulmonary infection caused by the streptothrix Eppingeri or a closely related species, and clinically resembling pulmonary tuberculosis. Warthin and Olney[1] report 5 cases, and point out that the frequency of occurrence, the symptomatology, and the therapeutics of this form of streptothricosis remain to be worked out. The clinical picture presented is that of tuberculosis or bronchopneumonia. The diagnosis, however, demands isolation of an acid-resisting streptothrix. The tubercle bacillus is absent, but streptococci and staphylococci are found in association, and some of the cases may be of primary streptococcus infection.

Fibroid Phthisis.

Definition.—Fibroid phthisis implies induration followed by contraction of the affected lung-tissue, due to an increase in the connective-tissue elements. There are cases in which it cannot be distinguished pathologically from chronic pulmonary phthisis, but they differ clinically. The majority of instances are primarily tuberculous, though manifesting a strong tendency to the formation of fibrous tissue—a conservative process; in other instances the fibroid change may be primary, followed by tuberculous infection (*vide* Pneumonokoniosis). The usual form arises variously as a sequel of other morbid processes, such as—

(1) Pneumonias, lobar (rarely) and catarrhal (commonly).

(2) Pulmonary lesions—tubercle in the stage of consolidation or cavity.

(3) Chronic tuberculous pleurisy.

(4) Bronchial catarrh from inhalation of irritants (steel-, coal-, or mineral-dust).

Pathology.—The process in the beginning is very often localized in one apex, and less frequently in the middle portion of the lung or in the bases. It may remain circumscribed, but more often it extends downward, and gradually invades the entire lung. It is unilateral. Secondary to the induration and contraction there is dilatation of the bronchi.

The lung-tissue is hard and dense, the alveoli being obliterated. It resists cutting and creaks, and the section presents a smooth, dry, gray, often marbled aspect, though the fibrous tissue may undergo caseation.

The pleura is thickened, as a rule, often to a marked degree, and its layers are adherent; the unaffected portions of the lungs frequently become emphysematous. The right ventricle is, as a rule, hypertrophied.

Symptoms.—These may be briefly stated, since they do not differ from those of cirrhosis of the lung (*vide* Diseases of the Lungs). The *onset* is insidious: a persistent *cough*, occurring in severe paroxysms in the mornings, and a *purulent expectoration* are for long the leading features. If bronchiectasis is present, the sputum may be fetid. *Dyspnea* is marked, particularly on exertion. *Fever* is slight or absent, hence emaciation progresses slowly or may even be absent. The *physical signs* are identical with those of fibroid induration of the lung (*vide infra*).

The **course** of this disease is long, ranging from ten to twenty or even thirty years, and both lungs may become involved. Again, as in chronic

[1] "Pulmonary Streptothricosis," *Amer. Jour. Med. Sci.*, vol. cxxviii., No. 4, pp. 637–649.

pulmonary tuberculosis, prolonged suppuration may lead to amyloid changes in the liver, spleen, kidneys, and intestines. *Dropsy*, due to secondary dilatation of the right ventricle, often closes the scene.

Differential Diagnosis.—*Chronic bronchitis* may be mistaken for fibroid phthisis. In the latter disease, however, there are unilateral retraction and the signs of consolidation or of an apical cavity, and the sputum-test may settle the doubt.

Complications of Pulmonary Tuberculosis.—*Lobar pneumonia*, and less commonly *lobular pneumonia*, may develop and cause a fatal termination. In a study of 100 cases H. M. King found the principal complications of a non-tuberculous character were lobar pneumonia and nephritis.

Erysipelas may arise in the course of chronic pulmonary tuberculosis, though the proportion of cases is not formidable. Out of 1165 cases of erysipelas, 15 coexisted with pulmonary phthisis.[1] Some contend that its occurrence in this disease may be beneficial, but my own observations show that the gravity of both conditions is increased.

Typhoid fever may rarely be met with in sufferers from chronic phthisis. Out of a totality of 249 autopsies in cases of typhoid fever, only 19 (7.6 per cent.) showed the presence of tuberculous lesions.[2] This contradicts the opinion that typhoid fever predisposes to tuberculosis.

Chronic nephritis and pulmonary tuberculosis are often found in the same subject, and with these arterio-sclerosis is quite commonly combined. Intercurrent acute hemorrhagic nephritis may develop.

Chronic endocarditis, particularly of the tricuspid segments, may also occur in phthisis, and from time to time cases of valvular heart-disease are reported, in which it is evident that passive congestion must have existed for some time before the tuberculous condition developed. The old doctrine of the mutual antagonism between disease of the left heart and pulmonary tuberculosis finds support from these cases, as in a large proportion a tendency to encapsulation of the tuberculous lesions exists.

Course and Duration.—Both as to course and duration this disease exhibits unusual variations. If not promptly treated during the incipient stage it frequently progresses with more or less rapidity toward the grave. It is common, however, to observe periods during which the disease is arrested or improved. Generally, the improvement, though followed by an exacerbation, endures for a long time, and permanent cures, even in the advanced stage, are by no means rare. The duration of pulmonary tuberculosis varies exceedingly, though from the collective investigations of different authors and from all the statistics available I find the average duration to be about three years. The late Austin Flint long ago directed attention to the innate tendency of a considerable percentage of the cases to spontaneous recovery—a fact that simply indicates a victory for nature's silent defensive processes in the struggle for supremacy.

In fatal cases death is by (*a*) *gradual asthenia* (most frequently), with retention of consciousness until the end approaches.

(*b*) *Complicating conditions* (bronchitis; pneumonia; pleurisy; pneumothorax; amyloid degeneration of the intestines, liver, spleen, kidney; Bright's disease; diabetes, etc.).

[1] "Points in the Etiology and Clinical History of Erysipelas," *Journal of the American Medical Association*, July 2, 1893.

[2] *Amer. Jour. of the Med. Sci.*, May, 1904, by the writer.

(*c*) *Tuberculosis of other organs*, particularly the meninges, intestines, and genito-urinary tract.

(*d*) *Hemorrhage*, due commonly to rupture of an aneurysm in the lung-cavity; less frequently to erosion of a large vessel. Fatal hemorrhage may, when the vomica is of large size, occur without hemoptysis, as in a case of Roland G. Curtin's at the Philadelphia Hospital.

(*e*) *Syncope.*—Though of comparatively rare occurrence, there are a number of events that may lead to sudden, fatal syncope—*e. g.* hemorrhagic embolism or thrombosis of the pulmonary artery, pneumothorax, thoracentesis for pleural effusion, walking about in a moribund state, etc.

(*f*) *Asphyxia* often closes the scene in acute pneumonic phthisis, and rarely in chronic phthisis complicated with pneumo-thorax, or with a large undiscovered or neglected empyema, or with sero-fibrinous pleurisy.

Tuberculosis of the Alimentary Tract.

(1) **Lips.**—Whilst tuberculosis of the lip is quite rare, the possibility of its occurrence must not be forgotten. It assumes the form of a small ulcer, and the diagnosis is made by an examination of the labial mucus. It is usually associated with laryngeal or pulmonary tuberculosis. In *diagnosticating* the condition, chancre and epithelioma must be excluded, the former by the history, and the latter chiefly by a microscopic examination for tubercle bacilli.

(2) **Tongue, Palate, and Tonsil.**—The work of Orth, Hanan, Schlenker, Kruckman, and others has shown that the tonsils, owing to their frequent inflammation, serve as the door of entrance of the tubercle bacilli. The fact that tuberculosis of the tonsils has repeatedly been found, and when other lesions of the disease were absent, points to the not infrequent occurrence of primary tuberculosis in this site. The infiltrated areas often present small grayish spots, but the appearance of the ulcers is not characteristic, frequently bearing a strong resemblance to epithelioma and to the syphilitic ulcer. The *diagnosis* demands either inoculative experiments or a microscopic examination of the oral mucus, the latter being oft repeated if necessary. E. D. Smith records 5 rare cases of tuberculous ulceration of the soft palate.

(3) **Pharynx and Esophagus.**—Both miliary tubercles and ulcerative lesions may rarely arise on the posterior wall of the pharynx by direct extension from laryngo-pulmonary tuberculosis or as the result of secondary inoculation. The chief symptoms occasioned are the excessive secretion of pharyngeal mucus and muco-pus, and painful deglutition. Tuberculosis of the esophagus is extremely rare.

(4) **The Stomach.**—Tuberculous lesions of the stomach are of exceptional occurrence. Marked gastric symptoms, however, are common, and they may be due to involvement of the larynx. I have been able to find reports of 4 cases of tuberculous gastric ulcer in addition to the 12 collected by Marfan.[1] The ulcers may be single (as in Musser's case) or multiple (as in Osler's case). The *symptoms* are not characteristic, but hematemesis occurring in patients suffering from tuberculosis of other organs should excite a strong suspicion of the existence of ulcer. Pain coming on soon after meal-time is more marked in tuberculous ulcer than

[1] *Paris Thesis*, 1887.

in ordinary gastric lesions. Perforation may take place. Four cases are recorded in which the pylorus was found encircled with a flat, granular ulceration, operated on under the diagnosis of carcinoma (Alexander[1]). The process was isolated and the symptoms all pointed to pyloric cancer.

(5) **Intestines.**—The lesions may be (*a*) primary or (*b*) secondary.

(*a*) Primary tubercle of the intestines is chiefly met with in children, for the reason that they are more likely to swallow the tubercle bacilli with their food, and especially in milk. The intestinal route of infection is, according to my own observation, more common in adults also than is supposed. Many cases during life present the features of both intestinal and peritoneal tuberculosis, and it is often impossible to determine which of these was the primary condition; and the same difficulty arises when the cases come to autopsy. I have never seen an instance (post-mortem) of intestinal tuberculosis in which the peritoneum and mesenteric glands were not involved to an equal degree.

(*b*) The secondary variety occurs in more than one-half of the cases of pulmonary tuberculosis, the chief seats of the lesions being the lower part of the ileum, the cecum, and the upper part of the colon. The rectum is also the seat of secondary tuberculosis in a small proportion of the cases of chronic phthisis and it may be rarely a primary seat of the affection.

The morbid process begins in the solitary glands in Peyer's patches, where at first grayish, firm tubercles grow and form little prominences. These caseate, becoming yellow in appearance, and then soften and disintegrate, producing ulcers. Osler thus describes the characteristics of the tuberculous ulcer: "(*a*) It is irregular, rarely ovoid or in the long axis, more frequently girdling the bowel; (*b*) the edges and base are infiltrated, often caseous; (*c*) the submucosa and muscularis are usually involved; and (*d*) on the serosa may be seen colonies of young tubercles or a well-marked tuberculous lymphangitis." In all *acute* cases the surface-lesions show little tendency to repair (Senn).

In *chronic* cases attempts at healing are the rule; and the cicatrices are extensive and often pigmented, and as they undergo contraction may produce incomplete or even complete stricture of the bowel. At a point corresponding to the seat of the ulcers local peritonitis invariably develops. The serosa is thickened and adherent, and the ulcer may penetrate through this coat without causing perforative peritonitis, while rarely a fistulous connection is established between the different parts of the intestine.

Symptoms.—In children the symptoms are those of a protracted catarrh of the intestines, or they may be absent. Among prominent features are diarrhea, colicky pains, and the presence in the stools of pus, blood, and particles of mucus resembling sago-grains. In many cases there is constipation, which may be due either to peritonitis or cicatricial stenosis. The general symptoms are irregular fever, wasting, and a lack of development; they are especially valuable for diagnosis.

In adults intestinal tuberculosis generally gives rise to symptoms similar to the above, and when they arise in the course of pulmonary phthisis they are highly significant. If diarrhea be present, it stubbornly resists treatment, and it must not be forgotten that it may also

[1] *Deutsches Archiv f. klinische Med.*, Berlin, lxxxvi., Nos. 1–3, 1906.

be due either to catarrhal colitis or to amyloid change, both of which processes may be associated with chronic phthisis. Constipation is common and often marked, and local tenderness and colicky pains are complained of frequently. The pulmonary signs, however, may be in abeyance.

If the abdominal and general symptoms are such as to excite suspicion of this disease, then a rigid physical examination of the lungs should be made. The chief seat of the lesions may be for a long time in the cecum, or in the appendix, when the symptoms—both local and general—will be those of appendicitis.

The *diagnosis* of primary intestinal tuberculosis is beset with special difficulties. Sawyer[1] has in special instances demonstrated the presence of clusters of tubercle bacilli in the rectal mucus, and in this way the recognition of intestinal tuberculosis at an early date, or before diarrhea sets in, is rendered possible. The mucus is obtained after placing the patient in a position as if to examine for piles, and directing him to bear down as though at stool, by gently removing a small quantity from the everted membrane with a sterile loop. It is then spread upon a clean cover-glass and treated exactly as sputum in the ordinary examination. The same method is applicable to cases of secondary intestinal tuberculosis, but here the history and associated tuberculous lesions usually serve to remove all doubt.

Tuberculosis of the Serous Membranes.

General tuberculosis of the serous membranes *secondary* to pulmonary and intestinal tuberculosis is of common occurrence, and that a *primary* form of tuberculosis of the serous membranes also occurs is undoubted. Unfortunately, accurate means of discriminating the secondary from the primary form are wanting, since often in the secondary variety the primary lesions in other organs are insignificant.

The anatomic alterations resemble those of ordinary inflammation of these structures, plus the presence of nodular tubercles. The latter may be observed, as a rule, only over small, scattered, circumscribed areas, though not infrequently they are both numerous and diffuse (general miliary deposit). The effusion is in most instances sero-fibrinous, though sometimes it becomes purulent, and not uncommonly it is hemorrhagic. Most instances of so-called hemorrhagic pleurisy are due to pleural tuberculosis.

Clinically, cases are divisible into (1) acute serous membranous tuberculosis and (2) the chronic form. The *acute* form results from inoculation of the peritoneum or pleura, induced by limited foci in the bronchial, tracheal, or mediastinal lymph-glands, or in the Fallopian tubes in women. The *chronic* type is apt to result from a direct extension of a tuberculous process from some organ adjacent to the pleura or peritoneum, though it may attack the serous membranes primarily. Belonging to this class of diseases are two groups of cases: those attended by sero-fibrinous or sero-purulent effusion and the presence of caseous masses, and those in which there is a tuberculous deposit with increased density and great

[1] *Medical News*, May 23, 1896.

thickening of the pleural layers, and slight exudation. The pericardium may be similarly involved.

(*a*) **Tuberculous meningitis has** been described fully in the present section (*vide* Miliary Tuberculosis).

(*b*) **Tuberculous Pleuritis.**—This subject will be referred to in the section on Diseases of the Pleura. Its import, however, is such that brief special consideration is demanded, and from a clinical view-point the cases may be grouped under two heads—namely, *acute* and *chronic tuberculous pleurisy.*

The *acute* form often has a sudden onset, the initial symptoms being a rigor or repeated fits of chilliness, a stitch-like pain in the side affected, shallow, catching breathing, a cough, and fever. The ushering-in symptoms sometimes suggest lobar pneumonia, and a fatal termination is not uncommon, though apparent recovery or a transition into chronic tuberculous pleuritis also occurs.

Chronic tubercular pleurisy is vastly more common than the acute form, and it is sometimes primary, though more often secondary to pulmonary tuberculosis. In all cases of the latter disease in which the periphery of the lung becomes involved the visceral layer of the pleura is invaded. This leads to plastic pleurisy with adhesion, and the membranes contain disseminated tubercles, or to sero-fibrinous tuberculous pleurisy. As above stated, the effusion may be hemorrhagic and may also become purulent. When the tuberculous pulmonary focus perforates the pleural sac, pyopneumothorax is produced. In tuberculous pleurisy, as opposed to simple pleurisy, there is usually an absence of leukocytosis.

Symptoms.—The *onset* is very insidious and often unnoticed. There may be few symptoms, and yet a physical examination reveal a large sero-fibrinous exudate. The cough and other symptoms are frequently due to a coexisting tuberculosis of the lungs, and the presence of subcrepitant and dry râles is strongly confirmatory of tuberculous pleurisy. By and by the evidences of pulmonary tuberculosis are of diagnostic importance, or the supervention of acute general miliary tuberculosis makes clear the nature of the case. The *subacute variety* with effusion may terminate, after absorption of the exudate, in chronic adhesive pleurisy with great thickening of the membrane. The latter may also originate as a primary proliferative process.

(*c*) TUBERCULOSIS OF THE PERICARDIUM.

The morbid lesions are analogous to those of tuberculosis of the pleura. The effusion may be enormous on the one hand or insignificant on the other, and it is often hemorrhagic, while in the chronic form there is marked thickening of the membrane with the deposit of tubercles and cheesy masses. The affection is less common than tuberculosis of the pleura, yet not so rare as was formerly supposed, and occurs in the acute and chronic forms.

Acute tuberculous pericarditis is rarely a primary affection, and, as a rule, originates secondarily to pulmonary, pleural, or glandular tuberculosis. It is especially prone to arise in tuberculosis of the bronchial and mediastinal lymph-glands, and, as the latter condition is frequent in young children, so tuberculosis of the pericardium is relatively frequent at this period, though it may occur at any time of life. Pericardial

tuberculosis also results from direct extension from a contiguous focus. The symptoms will be detailed in the discussion of Pericarditis. In the *diagnosis* of the affection the history and any associated tuberculous processes detectable must be taken into account, and a point of some diagnostic value rests in the fact that tuberculous pericarditis does not show the usual inflammatory leukocytosis.

Chronic Tuberculous Pericarditis.—This may be a part of the general tuberculosis of the serous membranes, or it may follow an infection of the bronchial and mediastinal glands (most frequently), lungs, pleura, or peritoneum. Cases of primary origin also occur, but they are exceedingly rare, the neighboring lymph-glands being generally involved. This form is also dependent upon direct extension from the spine and sternum.

From personal observation I am convinced that the cases naturally fall under two heads, when considered clinically: those without effusion, in which the pericardium is adherent; and those with more or less effusion. The former are the more frequent, though often entirely latent, the adherent pericardium leading to hypertrophy of the heart, followed sooner or later by dilatation. The signs are therefore those of adherent pericardium, with the occasional difference that the dulness may extend higher up over the sternum, in consequence of the presence of firm, cheesy masses at the base of the heart and also encircling the aorta. The smaller group of cases (in which the effusion is present) resembles dilatation of the heart in its clinical manifestations. I recall one instance of this sort that occurred in a male aged about sixty years at the Episcopal Hospital, the autopsy revealing extensive pulmonary tuberculosis and chronic tuberculous pericarditis, with the presence of eight ounces of hemorrhagic effusion.

(*d*) TUBERCULOSIS OF THE PERITONEUM.

This is dependent upon infection by means of the bacilli circulating with the blood, or upon extension of tuberculous inflammation or ulceration from adjacent organs. In 11 per cent. of 3405 autopsy records Cummins [1] found there was peritoneal involvement. Mention has already been made of the fact that the intestines are often invaded by tuberculosis, and that the serosa is quickly involved in such instances. The condition may rarely be primary. This involvement may remain circumscribed and undergo spontaneous cure if the intestinal lesion cicatrizes, as postmortem findings frequently indicate, but in extensive peritoneal involvement spontaneous resolution is out of the question. These cases may be subdivided into acute and chronic. The *very acute cases* are those forming a part of acute general miliary tuberculosis, or due to perforation into the peritoneal sac from adjacent organs, and Adlebert's classification is as follows: (*a*) The ascitic form, (*b*) the ulcerous form, and (*c*) the fibroid form. Though these groups do not present sharp clinical distinctions, the courses they run vary considerably, as do the results of treatment. In the *ascitic* form the exudate is purulent or sero-purulent, and is often encapsulated. In the *ulcerous* the tuberculous new-formations, which may be quite large, undergo caseation and ulceration, the latter process being progressive, so that it may perforate the walls of the intestines. This and the ascitic variety may be combined.

[1] *University Med. Bulletin*, December, 1905.

In the third or *fibroid* form the peritoneal surfaces are adherent. There is little exudation; the tubercles may be numerous and diffuse, or found only in scattered localized areas. The lesions may represent the concluding stage of acute or subacute tuberculous peritonitis.

Etiology.—Most cases are produced by extension of tuberculous inflammation from adjacent organs, and of 107 cases analyzed by Phillips the lungs were involved in 99, the pleura also in 60, and the bowel in 80. Children are frequent victims to intestinal tuberculosis, and the bacilli often reach the peritoneum through the intestines, as they are also apt to do in adults suffering from chronic phthisis. Extension from the pleura to the peritoneum is frequent (pleuroperitoneal), but from the pericardium is rare. In females the starting-point is often the Fallopian tubes (Mayo, Murphy), and in either sex it may be the appendix.

Predisposing Factors.—*Age.*—During the period from fifteen to forty years the incidence is most frequent, although it is not uncommon in children under ten years, nor between the fortieth and fiftieth years of life. Subsequently, it rapidly decreases in frequency. I agree with Osler in stating that in America negroes are more prone than whites.

Sex has a tolerably potent disposing influence. Abdominal surgeons have taught us that the disease occurs more frequently in females than males, owing to the fact that the Fallopian tubes are a favorite seat for primary tuberculous infection. The ratio based upon sex is as 3 to 2 in favor of females.

Symptoms.—Some cases develop *abruptly* with *severe symptoms*, as fever, marked constitutional disturbance, rapid small pulse, abdominal *pain*, *vomiting*, and sometimes *diarrhea*. The *temperature* may be quite high (103° to 104° F.—40° C.), or it may be only slightly elevated even in the worst cases. There follow quickly such symptoms as *anemia*, *marked emaciation*, and a pronounced *typhoid condition*. The signs of *peritoneal effusion* (rarely large) are soon in evidence, and are attended sometimes by a suppurative type of temperature, sweats, etc., indicating the presence of *pus* in the peritoneal sac. A few cases are unattended by effusion, and here nodular masses are palpable, while on auscultation friction-sounds may be audible in the umbilical region. *Tympanites*, due to intestinal paresis, is common in cases having an acute onset.

The *acute stage* may be absent, the affection then being marked by slight local and general symptoms (low fever, anemia, slight belly-pains, and a sense of distention). The skin is sometimes pigmented, and usually in patches. There are not a few instances in which the affection is latent, and in one case of this sort with ill-defined general symptoms pigmentation of the skin first directed my attention to the peritoneum.

The **physical signs** of moderate ascites frequently, and those of enlarged mesenteric glands sometimes, are present. These conditions are often combined in children, constituting the so-called *tabes mesenterica*. I cannot conceive of the occurrence of this association of symptoms without simultaneous involvement of the peritoneum, and doubtless co-involvement of the latter membrane and intestines usually occurs. Hamman emphasizes the great frequency with which more than one serous membrane is affected (multiple serosites). The tuberculous new growth in the peritoneum may also form a distinct tumor not unlike that produced by glandular enlargement, while the intestinal coils

with their now thickened walls are sometimes knotted together so firmly as to simulate a dense new growth. The exudation may be loculated owing to adhesions between peritoneal layers of the intestinal coils, etc., producing a localized tumor varying in size and position. Such sacculated exudations most frequently occupy the pelvic or umbilical regions, though they may also be found elsewhere in the abdomen. They may be multiple, and are not infrequently too small to be recognized by the physical signs, being often discovered during laparotomy. On the other hand, they may occupy a large portion of the abdomen. An *omental* tumor of characteristic elongated form (produced by a shrinking and curling up of this membrane) is demonstrable, its long axis generally taking a transverse direction just above the umbilicus. Gardiner has observed this tumor to disappear by spontaneous resolution in children.

The dry, fibrous variety, which is not infrequent, is often latent, and the condition may be general or localized. It is decidedly more frequent in adults than in children. The *symptoms* are far from characteristic. Among local features are pains, abdominal distention (giving rise to a tympanitic note on percussion), tenderness on pressure, and sometimes a tumor-ridge extending across the upper abdominal region. Among general symptoms are usually anemia and emaciation, with or without fever. Indeed, the temperature may be subnormal, and these cases may show a tendency to spontaneous recovery.

Diagnosis.—Unless tuberculosis of other organs can be demonstrated the diagnosis is often impossible. This is particularly true in cases in which there is no abdominal pain, which is the most important local symptom, nor tenderness. Fever and the presence of a tumor, especially if the latter be elongated and lies transversely in the umbilical region, are important aids; but if tuberculosis of the lungs, pleura, pericardium, appendix, and the tubes, in women, can be excluded, the rectal mucus and the urine should be examined for tubercle bacilli. From the *acute form* several affections must be discriminated:

(*a*) *Internal Hernia.*—This comes on suddenly; the pain is strictly localized and paroxysmal; stercoraceous vomiting appears in a few hours; the constipation is absolute, and tympanites is marked, but ascites is absent.

(*b*) Similar symptoms belong to *volvulus* and to the quick incarceration of loops of intestine under bands of adhesions; on comparison they will be seen to differ from those of acute tuberculous peritonitis.

(*c*) *Enteritis* is discriminated *from acute tuberculous peritonitis* by the presence of copious mucous discharges, and by the absence of associated tuberculous lesions, peritoneal exudate, tumors, and the phenomena of the typhoid state.

Chronic tuberculous peritonitis often closely simulates *cancerous peritonitis*, owing to the fact that the elongated omental tumor may be met with in both, associated with effusion, abdominal pain, and slight fever. In carcinoma, however, there is an absence of the tuberculous history and lesions, and the presence, sometimes, of a gradually increasing tumor of primary growth, the slowly oncoming intestinal obstruction from pressure, and the cancerous cachexia. Moreover, tuberculous peritonitis occurs more commonly in younger subjects, and is more apt to be interrupted by periods of improvement, followed in turn by rather alarming symptoms. The tuberculin test is to be used in dubious cases.

Locular exudations must be distinguished from *ovarian tumors*, and here the history, together with tuberculous lesions elsewhere in the body, the occurrence of febrile attacks, and intestinal disturbance with pain, are of great diagnostic significance. Such cases should be examined by a gynecologist, since, however expert the examiner, when the saccular exudations are located in the pelvic region an exploratory laparotomy must often decide the nature of the condition. Finally, it must not be forgotten that the vast majority of cases of chronic peritonitis are tuberculous.

Tuberculosis of the Liver.

The liver was formerly overlooked in many instances of tuberculosis, because the lesions, particularly in acute tuberculosis, are often microscopic. In the chronic disseminated variety, however, grosser changes are observed, the organ being slightly enlarged, pale, and fatty, and presenting an irregular surface like that of an orange. On section, the parenchyma cuts with great resistance, being very dense (tuberculous cirrhosis). Minute gray and larger yellow masses are seen, especially just under the capsule, and small cavities, the result of a breaking down of the cheesy masses and containing pus and bile, are also observed. These changes are most pronounced about the bile-ducts.

Etiology.—The liver is implicated in all instances of acute miliary tuberculosis. It is also involved secondarily in chronic tuberculosis of the lungs, pleura, peritoneum, spleen, lymphatics, etc.

Symptoms.—This is a common condition, the organ being appreciably enlarged and its surface presenting irregular, palpable prominences. The clinical features of *perihepatitis* and *peritonitis* are often found in combination. Ascites may be present, but is rare.

Here may be mentioned that occasionally the spleen seems to be the primary focus of tuberculosis.

Tuberculosis of the Genito-urinary System.

(1) **Tuberculosis of the Kidneys.**—This may be primary or secondary, the secondary form being the more common, and it may be either unilateral or bilateral.

Pathology.—The process begins in the calices and apices of the pyramids (papillæ), thence proceeding to the pelvis of the kidney, so that early the condition may be pyonephrosis. The morbid changes then extend to the ureters, and sometimes to the bladder and prostate, and instances are even met with in which the process seems to have crept from below upward, starting from the bladder or prostate. The tubercles pass through the usual stages of caseation, necrosis, and suppuration, and destruction of the renal tissue to a greater or lesser degree occurs, with the formation of cysts containing cheesy material in which lime-salts may be deposited. When the process invades the kidneys through the blood, it may be limited largely to the cortical layer and give rise to nodular tuberculosis with caseous masses, yet with little loss of renal substance. H. A. Kelly[1] believes that the infection of the kidney is almost always hematogenous. While it is difficult to judge of the relative ages of the lesions in different organs, I cannot escape the conviction that in a

[1] *British Medical Journal*, June 17, 1905, p. 1319.

small group of cases renal tuberculosis is an *ascending* process and follows ureterocystic tuberculosis. Although both kidneys are finally involved in most instances, for a considerable period the disease is unilateral. Hallé and Motz found one kidney alone affected in 89 out of 132 cases. In acute miliary tuberculosis, both kidneys show disseminated tubercles. Caseation and necrosis, however, seldom occur.

Etiology.—Of disposing factors *age* and *sex* deserve especial mention, most cases occurring during middle life, though they are by no means rare both at an earlier and a later period. The disease is more frequent in males than females.

The bacilli reach the kidneys with the blood-stream, producing primary renal tuberculosis (hemogenic infection), through the lymphatics (lymphogenic infection) and direct extension from adjacent structures.

Symptoms.—In many cases there are either no renal symptoms or none until a late stage is reached, but the symptoms of pyelitis are usually present. *Pyuria* may be the only symptom for a long time, and this symptom, according to certain authorities, points directly to cystitis. When the latter condition is present, however, the micturition becomes frequent and there is vesical tenesmus. *Pain* in the side chiefly affected is complained of, and is sometimes not unlike renal colic; *hematuria* is not rare, and it may be the initial symptom. Braasch found hematuria in 60 per cent. of 203 cases, and bladder irritability in 86 per cent. Cystoscopic examination may show the blood to be of renal origin (Tuffier). It is useful also in showing the state of the bladder-mucosa. The demonstration of tubercle bacilli in the urine, especially if arranged in S-shaped groups, is diagnostic (Frisch). When the bacilli cannot be found, inoculation-experiments upon guinea-pigs and rabbits furnish an accurate criterion, though it must not be forgotten that tubercle bacilli may find their way into the urine from more distant tuberculous foci. Catheterization of the ureters may determine which kidney is involved. Tubercle bacilli are not found in the urine in the miliary form. *Polyuria* is sometimes present, as well as *albuminuria;* the urine may also show *tube-casts* (rarely) and pus-cells. Macroscopic cheesy masses are occasionally found. Roentgenographs after injections of 20 c.c. of a 10 per cent. solution of collargol are of much diagnostic value.

The *general features* are often marked, but not until the affection becomes advanced, chills, fever of a suppurative type, emaciation, and increasing debility being the principal symptoms. A good general appearance often accompanies an extensive lesion. Associated tuberculous lesions, especially of the lungs, are constantly observed.

Physical Signs.—Inspection may show a tumor-like prominence on the side chiefly affected, though rarely of large size. Renal tumor was palpable in but 20 per cent. of Braasch's cases (*vide supra*). Palpation often detects tenderness, and the outline of the organ may be defined by careful firm pressure with the finger-tips.

Diagnosis.—It is difficult to discriminate *calculous pyelitis*. In the latter, however, the pain is severer, the tumor-mass larger, and the hemorrhage more frequent than in tuberculous nephritis. The discovery of tubercle bacilli or the demonstration of tuberculosis of the lungs or other organs would remove all doubt. The tuberculin test may be used. Chevassre[1] recommends the antigen reaction of Debré and Paraf.

[1] *Presse Medicale*, February 28, 1912, xvii., 173.

(2) **Tuberculosis of the Ureter and Bladder.**—This is almost always secondary to tuberculous disease of the pelvis of the kidney above, or of the deep urethra, testes, or prostate below. When primary, as rarely happens, the process extends from ureters to bladder. The *symptoms* are those of chronic cystitis, and in all cases in which no other cause for the latter can be found the primary tuberculous lesion must be sought for and the urine carefully examined for bacilli. The smegma bacillus, sometimes present in normal urine, can be distinguished by decolorizing with absolute alcohol, which will take place in about two minutes, while with the tubercle bacillus a very much longer time is required. Others say this is not sufficient, and that only their methods of culture-growth or inoculation will distinguish them. A catheter specimen should be obtained if possible (Ogden). With the development of ulcerative lesions hemorrhage is apt to arise.

(3) **Tuberculosis of the Vesiculæ Seminales, Prostate, and Testes.**—The prostate gland and testes are frequently invaded in genito-urinary tuberculosis, and the vesiculæ seminales somewhat less frequently. The morbid process leads to the formation of cheesy nodules, which may, though comparatively rarely, disintegrate, causing excavations or perforation. Rarely, the tubercle does not pass through the stage of caseation, but merely shows the presence of numerous embryonic cells.

Etiology.—The condition is usually secondary, but the existence of primary tuberculosis in these organs cannot be denied. Testicular tuberculosis may begin at any period of life, and is of rather frequent occurrence in infants. When it occurs in the latter, it is part of a more general tuberculous infection, and is in many instances undoubtedly congenital. In some cases it may be a late hereditary affection.

Symptoms.—In the *testicle*, tuberculosis, as a rule, induces a painless, protracted orchitis, though when cavernous lesions occur the symptoms are more acute. In *prostatic tuberculosis* the bladder is highly irritable, there is great distress felt in the thigh and groin, and micturition is very painful. Catheterization, particularly if the urethra (as is very rarely the case) is the seat of tuberculous ulceration, causes most excruciating suffering, and there may be signs of stricture. *Rectal palpation* detects in the prostate firm nodules varying in size from a pea to a bean, together with enlargement of the organ.

Diagnosis.—The diagnosis of tuberculosis of the prostate is easily made from the vesical symptoms, the presence of tuberculosis in other organs, the result of rectal examination, and the detection of bacilli in the urine. Syphilitic involvement of the testicle is sometimes excluded with difficulty; in the latter disease, however, the surface of the swollen organ presents greater irregularities, and is even less painful than in tuberculosis. The absence of the history of syphilitic infection and the presence of tuberculosis in other organs, particularly in the uro-genital system, are valuable points in the discrimination.

Tuberculosis of the Fallopian Tubes, Ovaries, and Uterus.

Tuberculosis of the tubes in women is a not infrequent condition, and may be primary.

Etiology and Pathology.—The tubes, as a result of infiltration,

are thick, hard, and bound down by false membrane. Their ends are generally closed, but the intervening portion is dilated, and contains mucus, pus, and cheesy material. A catarrhal salpingitis is generally in association. *Uterine Tuberculosis* is rare, and its origin is usually attributable to similar involvement of the tubes.

The disease is most common during the period of greatest sexual activity, but young children may suffer (*vide* literature of Hennig), and in them the ovaries and uterus may be implicated without participation of the tubes, as in cases reported by Gusserow. At any period of life the lesions may be microscopic; they usually, however, excite marked local peritonitis, which may become general, with the development of ascites. The process may extend to the vagina.

Diagnosis.—The age, family history, and signs of the tuberculous diathesis must be noted. The disease does not distinguish itself from other tubal tumors by anything characteristic on bimanual palpation. Cases occur with ascites and also without, and in the latter variety plaque-like thickening of the subperitoneal tissue is an aid to diagnosis. The uterine secretions should be examined for bacilli in all obscure cases. Ashton advises an exploratory incision or puncture and examination of the contents of the peritoneum or tubes for bacilli.

Tuberculosis of the Mammary Glands.

This is rare; the affected glands present fistulæ and ulcers, with induration of the organ and retraction of the nipple. Warden[1] reports the finding of 58 authentic cases in the literature. Nearly 90 per cent. of the patients were females, and most cases developed in the third decennium. The *symptoms* are sharp and lancinating pains radiating to the arm, and tumor, the latter consisting of one or more nodules. Pyogenic secondary infection, leading to obstinate fistulæ, is common. The axillary glands are often enlarged. A positive *diagnosis* rests crucially upon the finding of the bacilli in the pathologic secretions.

Tuberculosis of the Brain.

Pathology.—Tuberculosis of the brain occurs in two forms, one of which, acute tubercular meningitis, has been previously described, while the other is a chronic tuberculous infection, usually localized, of the meninges and cortex, and causing meningo-encephalitis. Very rarely the membranes remain intact. The so-called solitary tubercle is an irregularly round mass, varying in size from a small pea to an apple or even larger. It is generally single, though sometimes there are two, and rarely even three, nodules. The tubercle may be imbedded in, and be contiguous with, the brain-substance, or may be separated from the latter by cysts. The peripheral zone is formed largely of connective tissue, is lighter in color (often translucent), and may contain miliary tubercles, while the central portion, which is cheesy as a rule, may liquefy and thus form a small cavity containing a purulent-looking material. They are seen with greatest frequency in the inferior portions of the brain.

The new growths may compress the longitudinal sinus, inducing thrombosis; they may interfere markedly with the circulation, causing cerebral

[1] *Medical Record*, October 1, 1898.

softening; and, finally, they may excite acute tuberculous meningitis. Tuberculosis of other organs is usually found as an associated condition.

Etiology.—The disease occurs with especial frequency in young subjects, and, according to the statistics of Pribram, in about three-fourths of the cases before the fifteenth year. The symptom-picture is identical with that of brain-tumor (*q. v.*).

Tuberculosis of the Spinal Cord.

The lesions are those of solitary tubercle of the brain. It is an extremely rare condition, and almost invariably secondary. (For symptoms, *vide* Spinal Tumor and Meningitis.)

Tuberculosis of the Heart.

(*a*) *The Myocardium.*—Tuberculous myocarditis, though comparatively rare, is more common than has been supposed. It may be primary, although practically always secondary to a focus in some other tissue, and transmission to the heart generally occurs by way of the lymphatic system. Infection through the agency of the pericardium is also common, and rarely it may be by the blood. Three *pathologic* varieties (here mentioned in the order of relative frequency) are recognized: (*a*) Large tubercles; (*b*) Miliary variety; and (*c*) Diffuse form, or tubercular infiltration. Generally speaking, the smaller nodules are found usually in the ventricles, and the larger masses in the auricles, chiefly the right. In miliary tuberculosis scattered gray granulations or semitransparent areas are formed. The literature furnishes a total of 72 cases, nearly one-half of which have occurred in persons under fifteen years of age, and is quite rare after the forty-fifth year. The *diagnosis* is exceedingly difficult, and rarely possible. In addition to the suspicious features, such as syncopal attacks of short duration or sudden collapse, with comparative well-being in the intervals, that may be present, the existence of generalized tuberculosis and pericardial tuberculosis, one or both, are essential to a diagnosis. Death may occur suddenly.[1]

(*b*) *The Endocardium.*—True tuberculous endocarditis is a rare condition. It is most apt to occur in acute miliary tuberculosis. The endocardium is to an unusual degree resistant to the tubercle bacillus. In tubercular invasion of the mediastinal glands the endocardium may become involved by extension of the morbid process. Infection of the endocardium also takes place through the blood supply to the heart structure. Vegetations occur on the valves, and in cases in which the lesions are of the ulcerative variety secondary pyogenic infection probably exists. Clinically, the cases of tuberculous endocarditis are extremely difficult of recognition. The history of the case, however, may be of diagnostic significance. "If it can be shown that the cardiac affection developed subsequent to undoubted pulmonary tuberculosis, and if rheumatic and other forms of infectious endocarditis can be eliminated, and especially if there have been neither previous arteriosclerosis nor fibroid degeneration of the viscera, then a reasonably certain diagnosis of tuberculous endocarditis, given the usual signs and symptoms, can be

[1] "Tuberculous Myocarditis," *Journal of the American Medical Association*, Nov. 1, 1902, by the writer.

made."[1] Of 1232 cases studied by N. G. Seymour, 62, or 5 per cent., were complicated by cardiac disease, of which 25 were cases of mitral incompetency.

Tuberculosis of the Arteries and Veins.

This may arise from extension of an adjacent tuberculous process into the vessel, as in chronic phthisis. It causes infiltration of the arterial wall, resulting in thrombosis, or the vascular tubercles may caseate and soften, thus leading to hemorrhage. In tuberculous meningitis the arterial lesions are conspicuous. The perforation of a vein by an old focus is followed by a distribution to all parts of the body of numerous bacilli and the production of acute miliary tuberculosis. Infection of the arteries may also occur through the blood. Of 1778 cases of pulmonary tuberculosis, thrombosis occurred 19 times, most commonly in the veins of the lower extremities (H. Ruhl and Hierokles).

General Prognosis.—The prognosis is best reached as in other infectious diseases—namely, by taking into account (*a*) the severity of the type of the disease; (*b*) the presence or absence of frequently associated diseases or complications; and (*c*) the numerous circumstances connected with individual patients.

(*a*) **The Severity of the Disease.**—Though there are no accurate criteria, we may judge of the severity of the disease by its progress, by the result of proper treatment, and from certain symptoms. If the fever be high, the prostration marked, and the local lesions rapidly advancing, we may safely infer that the disease is of aggravated type. With these certain other considerations are closely connected—the stage of the affection and the extent of the local lesions. Thus at an early stage the prognosis is more hopeful than at a late period, and, similarly, when the lesions are strictly localized at one apex it is more hopeful than when they have reached the stage of extensive cavity-formation or are bilateral. As already stated, a certain proportion of the cases manifest an inherent tendency to spontaneous arrest or even cure, and this may occur even after the stage of excavation has supervened. Notwithstanding this truth, however, it is well to make in all undoubted instances of the disease a guarded prognosis. A common error is the mistaking of a temporary for a permanent arrest of the tuberculous process, and in the natural history of the affection the fact was emphasized that its course was interrupted by periods of comparative comfort and noticeable improvement, followed by sharp exacerbations.

(*b*) **Associated Diseases and Complications.**—These unfavorably modify the prognosis—marked toxic symptoms (*e. g.*, rapid pulse, high temperature, rapid emaciation), chronic nephritis, gastric complications, intestinal and laryngeal involvement. Some of the accidents of the disease may precipitate a fatal result (*vide* Modes of Death). The appearance of intercurrent acute pneumonia is indicative of danger. The detection of secondary infective agents in the sputum renders the outlook worse. Other complications have been detailed in the Clinical History.

(*c*) **Circumstances Connected with Individual Patients.**—(1) A feeble, delicate constitution, either acquired or inherent (tuberculous diathesis), increases the morbidity of tuberculosis.

(2) When the fever subsides and the patient gains flesh and strength,

[1] *American Journal of Medical Sciences*, January, 1902, by the writer.

the outlook at once brightens. Maintenance of the weight curve while the patient is in his usual environment is a favorable indication. Konzelmann has observed a number of instances in which a tuberculous process of the lungs healed under the influence of a pleural effusion, the theory being that the affected lung is given rest. The temperature and local symptoms often promptly subside. A high polymorphonuclear percentage gives a bad prognosis, while an increasing lymphocyte percentage denotes favorable progress. The course of the disease is unfavorable in cases showing tubercle bacilli in the blood (Hilgermann and Lossen).

(3) *Hygienic Surroundings.*—When the hygienic regimen under which the patient lives is the best, the prospect is more hopeful than when it is faulty. A proper diet aids favorable progress, while a defective one often turns the scales against recovery. Equally influential for good is a pure atmosphere, while, *per contra*, a vitiated one is injurious.

(4) *Age.*—In young subjects from five to fifteen years of age tuberculosis often pursues an acute course and the mortality-rate is exceedingly high. Chronic tuberculosis may, however, form a sequel, and under appropriate surroundings may lead to recovery. In chronic phthisis "the younger the patient the shorter the duration." I have observed that patients who give a history of pleurisy early in life do not bear chronic phthisis well. Naegeli [1] found, as the result of 500 autopsies at Munich,

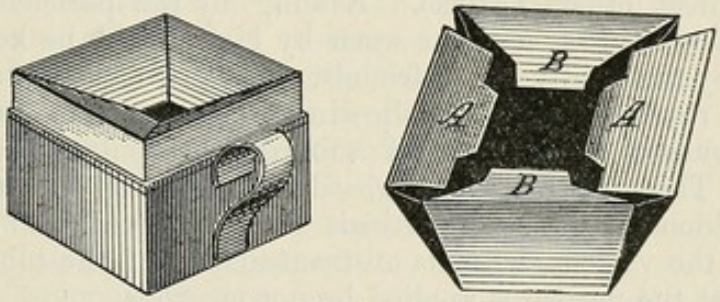


FIG. 25.—Pasteboard spit-cup for receiving infectious sputum. When used the pasteboard can be removed from the steel frame and burned.

that in persons over thirty years of age every one had been more or less successfully attacked by the bacillus tuberculosis; but from the fact that most deaths had occurred in subjects under thirty, he concludes that the adult body is, as a rule, well able to resist the attack. "The absence of a tuberculous family history has but slight, if any, favorable significance" (Bonney). During old age pulmonary tuberculosis is usually more or less latent, and, owing to coexistent emphysema and chronic bronchitis, pursues a slow course.[2]

(5) The gravity of tuberculosis may be determined with some degree of accuracy by the use of creasote in gradually ascending doses. Hence this agent has a prognostic value.

(6) Römer and Joseph [3] claim to have demonstrated beyond question that one infection with the tubercle bacillus confers a certain protection against subsequent infection.

TREATMENT OF TUBERCULOSIS.

Prophylaxis.—(1) This embraces thorough and prompt disinfection of the sputum as the best preventive element. To this end the patient must be taught to expectorate at all times into a spittoon or spit-cup which

[1] *Hyg. Rundsch.*, 2, 1901.

[2] A physician should not neglect to examine the sputum in suspicious cases for bacilli.

[3] *Beiträge zur Klinik der Tuberkulose*, Wurzburg, xvii., No. 3, p. 281.

contains a proper disinfectant solution, and when the breaking-down stage has arrived portable flasks (*e. g.* Dettwiler's) containing an antiseptic solution must be worn by the patient, even while out of doors. Stokes and Schmitz advise a combination of alkaline solution of sodium hypochlorite (antiformin) and phenol. Afterward the sputum is to be destroyed by boiling or burning and the spit-cup sterilized. The sweat of tuberculous patients should be removed at intervals, and the surface of the body bathed with appropriate antiseptics.

(2) **Isolation.**—After the stage of softening is reached the patient should invariably occupy a separate apartment, since, despite great care, the room and bed occupied by the consumptive become in time a source of infection. Hence, unwashable hangings and upholstered furniture, as well as other objects that facilitate the harboring of the bacilli, should be removed from the sick-room. The floor of the apartment should not be carpeted, but may in part be covered with rugs that can be frequently taken up and shaken in the open air. For like reasons, special hospitals and sanatoria for the treatment of the tuberculous poor are a necessity. Flügge's important researches (*vide supra*) show that phthisical patients should wear a mask day and night, that should be removed only for eating and to expectorate. Tuberculous patients in the infectious stage of the disease should be retired from occupations in which they may infect others (Flick). Kissing by the patient must be prohibited and all things used or worn by him should be kept apart from those used by the family or his friends. The prevention of auto-infection, which often results from the swallowing of sputum, is most important.

(3) **Compulsory registration** of tuberculous (pulmonary) patients is desirable. This insures thorough disinfection by health officers of houses in which deaths from phthisis have occurred, and serves to cut off many of the varied channels of transmission of the tubercle bacillus, provided that the measures applied be not rigorous.

(4) **Government Inspection of Dairies and Slaughter-houses.**—This is the serious business of the State, and, since infection through food, especially milk, is quite common in infants, skilled veterinary inspection of dairies is of prime importance. Of the greatest benefit would be the killing of all tuberculous cattle, and of less though decided efficacy the confiscation at the abattoirs of all carcasses that present marked lesions.

(5) The **popularizing of information** relating to the dangers of, and the means of stamping out, this great scourge. This may be in part accomplished by mural placards, stating simple, plain facts about the way in which the disease is spreading. Armaingaud suggests the placing in the homes of the people printed matter in a form suitable for preservation.

(6) **The Removal of Known Predisposition to the Disease.**—The tuberculous diathesis, whether inherited or acquired, must be overcome, if at all, by vigorous measures or by better hygienic living. In attempting to remove the phthisical tendency the physician must place chief reliance upon the most favorable environment attainable. The value of a change of residence—from the city to the country, the seaside, or the mountains, in selected cases—cannot be overestimated. It often renders predisposed persons immune. For some, and particularly young subjects, an equable climate (Southern California or Florida), that will enable them to live an out-door life is to be preferred. Attention to the food must not be forgotten. Milk and raw eggs are excellent and should be used freely.

Daily sponging of the neck and thorax with cold water is beneficial, and appropriate light gymnastics should be instituted if the subject be old enough. In-door occupations are to be forbidden, and the ventilation of living- and bed-rooms must be looked after carefully.

Tuberculosis is apt to develop especially in children while convalescing from acute fevers, and hence during this period the child should be strengthened by vigorous feeding, pure air, and tonics. In children predisposition often results from obstructions in the nose and from persistently enlarged tonsils; and they should be promptly removed. All local foci of tuberculosis in children—glandular, osseous, and articular—must be attacked surgically.

Treatment of the Disease.—The treatment of tuberculosis, regarded as a parasitic disease, presents two leading indications. One has reference to the destruction of the specific cause, the tubercle bacilli, by the use of antiseptic inhalations or of some parasiticide taken internally. Of the numerous substances used by inhalation, few have given satisfactory results, this being largely due to our inability to convey them to the smaller bronchi in a sufficient degree of concentration. They are best adapted to, and most efficacious in, cases in which the larynx is involved.

The inhalation of antiseptic substances may be accomplished in various ways—by inhaling vapors, by the use of the steam atomizer, or by some form of "respiration-inhaler." I have long employed the Robinson inhaler, the sponge of which is moistened with a few drops of a mixture made of equal parts of creosote, chloroform, and alcohol, the patient wearing the inhaler when not eating or sleeping. Unfortunately, most patients object to the constant use of this instrument. When hemoptysis is present, turpentine may be added to the above mixture. The chief among other antiseptics thus employed are carbolic acid, terebene, terpin hydrate, thymol, formalin, and oil of peppermint.

The most common, because least objectionable, mode of introducing this class of substances is by internal administration. According to the results reported from all quarters of the world, creasote thus employed alone enjoys the confidence of the profession; and in common with numerous other observers I have found its continued use to be followed by lessened cough and expectoration, lessened fever, and by a lessening or cessation of the night-sweats, with a gain of strength and weight as the natural consequence. It must be borne in mind that the dose is to be gradually increased to the point of gastric tolerance, which in my experience rarely exceeds 15 to 20 drops (0.999) three times a day.

Following, in the main, the practice of Trudeau, who has used this drug quite as extensively as any other American physician, after reaching the point of tolerance I gradually reduce the dose to and maintain it at 5 or 6 drops (0.333), three times daily. Among the best vehicles are hot milk, hot water, and diluted alcohol. Recently I have ordered it in capsules, which the patient himself fills at *the time of using*, and have found it a popular and ready mode of administration. When creasote is not well borne by the stomach and its inhalation is seriously objected to by the patient, it may be given by enema, the dose being 20 to 30 drops (1.332), in peptonized milk or mixed with a little egg-white. It has also been employed hypodermically in a 10 per cent. solution in oil of sweet almonds, the dose of which is 1 dram to 1½ drams (4.0–6.0). Lastly, it has in rare instances been employed by inunction.

Guaiacol, particularly in the form of the carbonate, has of late been quite extensively employed in place of creasote, of which it is the chief active principle. It may be administered in pill or capsule, the dose being slightly less than that of creasote. It is well tolerated by the stomach, and is broken up in and absorbed from the intestinal canal.

Among other remedies prescribed for their supposed parasiticidal effect are arsenic and mercuric chlorid, but they are clearly inferior to creasote.

I am of the opinion that all antiseptics used internally in this disease have for their chief influence a modification of the soil-conditions on which the growth and multiplication of the bacilli depend. They are, in truth, of great value in fulfilling the second leading indication of treatment, which is to overcome the bodily receptivity for the specific bacillus, or to aid the natural defensive processes in limiting the destructive work of the latter. All forms of tuberculosis, however, may heal spontaneously in any stage, especially the local varieties so common in children, affecting the lymph-glands, joints, and bones.

Old pleuritic lesions, a large proportion of which are tuberculous in nature, are constantly met with at autopsies in persons dying suddenly of other diseases. Spontaneous recovery is seen oftenest in cases that have not progressed to the stage of cavity formation. Indeed, in the instances in which vomicæ of considerable size have formed, cicatrization or complete cure is out of the question, though they may become encapsulated (quiescent). The percentage of cases in which encapsulated and obsolete lesions have been observed at the postmortem table in persons dying of all causes differs widely with the statistics of different observers. If we consider the cases that are latent from an early period in life, together with those of all ages after childhood, it is doubtless true that in more than 50 per cent. of the human family the bacilli effect a lodgment. Since about 14 per cent. of the deaths from all causes can be ascribed to tuberculosis, there must be manifested a strong tendency to limitation and healing.

In removing the diathesis medicines are unquestionably of less value than the hygienic treatment, the latter in the widest sense of the term aiming to reinforce Nature's efforts at spontaneous recovery, and embracing four main elements: (1) Climate; (2) Feeding; (3) Special Remedies; (4) Treatment of Leading Symptoms.

(1) **Climate.**—The all-powerful influence of environment has already been pointed out. Experience and observation have shown that certain climates, selected with particular reference not only to the stage of the affection, but more particularly to the individual, are useful modifying influences of the tissue-soil. In any case of tuberculosis that climate is most suitable in which the patient "feels well, eats well, sleeps well, and gains flesh and strength" (Delafield). Until the patient finds such a climate, or if he finds no single climate to produce these results, he should travel from place to place, unless special contraindications (excessive debility, etc.) exist. If active tuberculosis has existed, the stay in a suitable climate should not be less than two full years.

The climatic requisites for a consumptive are (*a*) purity of air, (*b*) equability, and (*c*) abundant sunshine. Less beneficial, though important, are (*d*) dryness and (*e*) altitude.

(*a*) *Purity of Air.*—This requirement is of paramount importance,

and thus is explained the fact that mountain air and that of the virgin forest are so helpful in phthisis. Forests, and particularly pine-groves, favor atmospheric purification, since they generate ozone, which oxidizes the impurities contained in the air.

(*b*) *Equability* has reference to the absence of rapid variations of temperature. On the whole, a relatively low is better than a high temperature, the former being stimulating, and the latter sedative, in effect. It should be pointed out that forests also greatly favor the quality of equability,[1] both as to temperature and relative humidity. They tend to maintain an almost unvarying degree of moisture in their vicinity, thus minimizing the diurnal variations of temperature—a point that is of far greater importance than the question of seasonal variations. Forests intercept and temper the bleak winds of winter, while by their shade and leaf-surfaces they afford a cooler temperature in summer.

(*c*) *Abundance of sunshine* is demanded by the consumptive. The advantages of sunshine are obvious from the observations made by Munn[2] in the year 1892, when in Denver there was sunshine in 62 per cent. of the possible hours during which it could occur. A *dry* atmosphere has advantages, but that dryness is not an essential element is shown by the fact that patients often do well at places having comparatively high relative humidity, such as Florida, Southern Georgia, Southern California, and the resorts on the south coast of England. The *rarefied* atmosphere of high altitudes, on account of its stimulating effect upon the respiratory function, aids in producing good results, but the pulmonary changes induced (enlargement of the air-cells, with augmentation of the size of the chest) make it necessary for patients to remain for the rest of their lives. That it is not an essential factor is shown by the excellent results obtained in the ofttimes purer atmospheres at lower levels. Cases in which hemoptysis is severe and of frequent occurrence, those complicated with weak hearts, and neurasthenic subjects should not be sent to the high altitudes.

The essential climatic factors mentioned are found in certain American and European resorts. Of the former, the Adirondack region, Colorado, Arizona, and New Mexico are especially to be mentioned, combining as they do in winter a uniform cold, much sunshine, and purity of atmosphere. A camp- or tent-life in the open air is strongly advocated. According to my own experience, the Adirondacks meet the indications best in early cases or in patients who have strength enough to lead an outdoor life, and in whom the breaking-down stage is not too far advanced. Some cases, in the early stage, also do well at Thomasville, Ga., Southern California, and at Lakewood, New Jersey. Some of these resorts possess the added advantage of affording an opportunity of gaining a livelihood. Among foreign resorts, Davos possesses about the same advantages as may be met in Colorado, New Mexico, and the Adirondacks, while the resorts in Southern Italy and France are comparable to Southern California, Southern Georgia, Florida, and the Bermudas in this hemisphere. Good culinary and home comforts are considerations of no less importance than the climate.

Briefly, the atmosphere of forest resorts possesses certain unmistakable advantages for this group of sufferers. Hence they should be sent into the neighborhood of the nearest forest in mild latitude (if they cannot enjoy the advantages of more remote resorts), where reasonably good

[1] *House-plants as Sanitary Agents; Sanitary Influence of Forest Growth*, p. 312, by the writer.

[2] *Medical News*, Aug. 18, 1894.

food and other comforts of life are obtainable. The superior value of the highly ozonized and terebinthinized atmosphere of the pine-groves in largngeal tuberculosis cannot be too strongly emphasized.

Sanatorium Treatment.—While it is essential to send patients to suitable resorts, the most satisfactory results are obtained from the combined climatic and sanatorium treatment. Sanatoria are warmly advocated by Trudeau, Knopf, Bowditch, and others. Of 237 early cases, treated in the Sharon Sanatorium, 81 per cent. were known to be alive and in excellent health for at least one year after leaving the institution.[1] They should take the form of cottages and pavilions. The principal advantages offered are due to a rigid system of hygiene under the close supervision of competent medical officers. There are *four groups of cases* among the middle and lower classes that require institutional treatment:

Group I.—The numerous cases that have progressed to an advanced and practically hopeless stage and the acute forms. These require every comfort and kind care, such as can be furnished by *special hospitals* for consumption in a healthful urban locality.

Group II.—Incipient cases among the pauper element. For these, sanatoria located close to large municipalities, though with special reference to such factors as purity of atmosphere and protection from chilly blasts, by natural elevations or the woodland, should be provided.

Group III.—Phthisis pulmonalis among the middle and working class, or persons having small means. The members of this group will find themselves compelled to depend principally upon private philanthropy, and probably to some extent also upon semi-State institutions; they need sanatorium treatment in the best climates, and there is no valid reason why the combined sanatorium and climatic treatment should not be attempted, since such an undertaking could be made almost self-sustaining.[2]

Group IV.—"A settlement for patients with arrested consumption where they can be employed on work adapted to their strength" (Powell[3]).

Among *home sanatoria* are the Adirondack Cottage Sanatorium, the Sharon Sanitarium, near Boston, the Loomis Sanatarium, at Liberty, N. Y., the Winyah Sanitarium, at Asheville, N. C., the White Haven Sanitarium and Mont Alto in Pennsylvania. *Foreign sanatoria* are to be found at Falkenstein, near Frankfort-on-the-Main, Goerbersdorf, and Hohenhonnef. Solaria, in connection with city hospitals for advanced cases, would, I am certain, yield gratifying results. Home sanatoria can be readily improvised by stocking living apartments with growing plants. The beneficial influences arising from the presence of the latter are ascribable to two functions—the generation of ozone and transpiration.[4] Tuberculosis dispensaries and classes are highly recommended in the treatment of tuberculosis among the poor and persons having small means. The class method is useful as an object lesson to teach the essentials in the home that are taught to the individual in a sanatorium (Wood).

Open-air Treatment at Home.—This method is now widely practised. It is of inestimable value to patients who must perforce be treated at home. They are kept constantly in the open air, and for the most part

[1] V. Y. Bowditch and W. A. Griffin, *Jour. Amer. Med. Assoc.*, Dec. 24, 1912, 2132.

[2] "Sanatoria and Special Hospitals for the Poor Consumptive and Persons with Slight Means," by the writer.

[3] *Lancet*, Jan. 6, 1906.

[4] Ibid, by the writer, p. 168.

at rest. At night the bed-room windows should be open, even in severe weather. Indeed, sleeping in the open air on a veranda, porch, or the roof, is to be advised and encouraged, and ingenious contrivances have been invented whereby the patient can occupy a bed out-of-doors at all seasons of the year. With warm clothing, abundance of good food, especially raw eggs and milk, and a careful regimen, surprising results are obtained even in large cities. In my opinion, however, most tuberculous patients, at all events, require the rigorous discipline of a sanatorium for a variable period of time so that they may acquire proper habits of living. Such a sanatorium for the reception of indigent patients should be situated in their home climate. The experiment has already been made in Chicago and other cities with complete success. When the temperature is above 100° F. (38.7° C.) the patient should be kept at rest. Flick, Minor, and Coleman are of the opinion that tuberculosis patients can be successfully treated in their homes and other places than sanatoria.

(2) **Feeding.**—The diet should be both nutritious and generous. Too close attention cannot be bestowed upon the feeding. Above all, when the remedies prescribed (cod-liver oil, creasote) embarrass in the slightest degree the function of the stomach they must be stopped.

Such albuminous articles as milk, eggs, flesh, fish, and fowl, together with an abundance of fats, should be taken. The hydrocarbons are urgently needed, but they must be taken with care lest they derange the digestive function. Over-alimentation with raw eggs and milk is strongly advised. The eggs are to be slightly beaten and stirred into the milk and the quantity is to be increased until from eight to twelve eggs and as many glasses of milk are taken daily. One-half of this amount may be used during the morning hours and the other half during the evening hours. At mid-day a generous meal composed of easily digestible solids is allowed. In advanced cases it is often needful to resort to a rigid system of feeding, giving a small quantity of food, such as milk, meat-juice, egg-white, and the like, at brief intervals. The French method of forced feeding deserves a trial if there be absolute loathing for food. It consists of first washing out the stomach with cold water, and then introducing the following mixture thrice daily: 1 liter of milk, an egg, and 100 grams of very finely powdered meat. As a rule the patient cannot be induced to swallow this, and it then must be poured through a stomach-tube. In a minority of the cases the appetite is ordinarily keen, often as a result of change of air, and these usually pursue a favorable course. The following combinations will be found useful in assisting the appetite:

℞. Sodii bicarb., ʒjss (6.0);
Tr. nucis vomicæ, fʒijss (10.0);
Glycerini, fʒj (3.75);
Inf. cascarillæ, q. s. ad fʒiv (120.0).
Sig. ʒij (8.0) t. i. d., in water, fifteen minutes before meal-time.

Other simple bitters and mineral acids may be tried, and there are some cases in which the judicious use of stimulants, particularly wines and malt liquors, aids the appetite and digestion materially. The chief indications for the exhibition of alcohol are loss of appetite, feeble digestion, and weak, rapid action of the heart. Brandy or whisky in the form of milk-punch may be given freely in the advanced stage. Strych-

nin is a valuable remedy in the later stages. Lavage has helped some of my cases immensely. Lastly, an orderly method and sound judgment must be brought to bear in arranging the diet and drink.

(3) **Special Remedies.**—The treatment of tuberculosis by mercury has been widely adopted. Wright[1] recommends the succinimidum, and the details follow: "One injection of hydrargyrum succinimidum, grain $\frac{1}{5}$, is given every other day until 30 injections have been given. Then injections are discontinued and potassium iodid, grain iij to x, is given well diluted with water one-half hour after meals for two weeks. Then potassium iodid is discontinued and no medicine is given for one week. Injections are then resumed as follows: One injection every other day until 30 injections have been given, on alternating injection days giving hydrargyrum succinimidum, grain $\frac{1}{5}$ and $\frac{1}{10}$, respectively. After the thirtieth injection the same course of potassium iodid is given as followed the first series of injections; then a week free from medication. The injections are then resumed again, the succinimide, grain $\frac{1}{10}$, being given every other day until 30 injections have been given. By the end of this third series experience will direct any necessary further treatment."

Cod-liver oil is another special remedy of great value. It may rarely cause further impairment of the appetite and digestion, or set up intestinal disturbances, when its effects are harmful. The commencing dose should be small (ʒj—4.0, once or twice daily, to be increased after a time to ʒij—8.0, two or three times daily). It should be taken about mealtime. When the oil is not well borne, it may be given in combination with an alkali (lime, soda). As a substitute for cod-liver oil, cream, preferably Devonshire, may be tried (ʒij to ℥ss—8.0 to 16.0, three times daily).

The *hypophosphites* are especially serviceable in a certain proportion of the cases. The dose is ʒj to ij (4.0–8.0) thrice daily, after food.

Arsenic is warmly advocated for its general influence in this disease. The dose should be small, so that it may be given for a long time without interruption. As sodium cacodylate, its use has increased of late. Jacobi speaks highly of digitalis in tuberculosis in children.

Iodoform- or *europhen-inunctions* are warmly commended by Flick, who asserts his belief that incipient cases almost always can be cured in this way, and that advanced cases can be improved. Crofton advises the use of iodoform intravenously (gr. $\frac{1}{4}$ to j—0.0162 to 0.0648, dissolved in ether with a little liquid paraffin added) two to five times a week.

Serum-therapy.—The definition of this term embraces also the employment of toxins and modified toxins produced in various media for the establishment of artificial immunity. Koch's tuberculin, Rosenbach's tuberculin, and Paquin's antitubercle-serum have been highly recommended as remedies in tuberculosis by certain clinicians. Dr. Trudeau prefers antitoxic immunity, and considers tuberculin habituation its essential feature and the best guide to dose; this requires long-continued and almost imperceptible progression in dosage until a maximum is reached without causing general or local reactions. A. E. Wright recommends Koch's tuberculin, claiming that in patients so treated the formation of opsonins is stimulated, as shown by a remarkable increase in the phagocytic index. Penrose[2] first obtains a good tolerance dosage of tuberculin, then administers a course of succinimid of mercury, and later alternates the two remedies with a view to using larger doses of tuberculin

[1] *Jour. Amer. Med. Assoc.*, Nov. 28, 1908. [2] *New York Med. Jour.*, June 11, 1910.

without risk. Vaccines made from associated organisms isolated from the sputum are of marked value in the treatment of tuberculosis.[1] Escoyer claims 53.8 per cent. of radical cures from the use of Cuguillere's serum.

Brauer, Wellman,[2] and others recommend artificial pneumothorax in the treatment of selected cases of pulmonary tuberculosis. A limited involvement of one apex may present an indication for either surgery, or the injection of nitrogen into the pleural cavity which may be the more effective (Mumford). Bayle claims to have cured 75 per cent. of 172 patients with spleen organotherapy.

The advent of an acute disease may arrest and cure a tuberculous process. Thus, the symptoms and signs of advanced tuberculosis have disappeared after an attack of virulent small-pox and acute rheumatism (Harris and Beales). Hysteria also exercises an ameliorating effect upon pulmonary tuberculosis, according to the observations of Gibotteau,[1] who advises against treatment of the former disease in tuberculous persons.

Treatment of the Acute Forms.—The treatment of acute tuberculosis is an expectant one. The special measures recommended above should be tried, but are rarely effective, and a change of climate is inadvisable. Supportive measures, such as stimulants and nutritious aliment, are required. The medicinal treatment must be adapted to the acute febrile condition, but all depressants are to be avoided. Special symptoms may be relieved in accordance with general principles.

In *renal tuberculosis*, recent experience confirms anew the importance of prompt nephrectomy. When this is impossible, for any reason, courses of mineral waters may prove useful. Castaigne reports 5 cases in which he used tuberculin or Spengler's immunizing bodies, or both, with ultimate success. Barney recommends epididymectomy in cases of tubercular epididymitis. In tuberculous peritonitis, Hoffmann reports good results from opening the peritoneal cavity, evacuating the fluid, if present, and painting the peritoneal surfaces of the gut and abdominal wall with 10 per cent. tincture of iodine. In *tuberculous adenitis*, the Röntgen rays should be employed early.

(4) **Treatment of Leading Symptoms.**—(*a*) *Cough.*—This is often quite annoying. The special cause or causes of the coughing should be determined before any attempt is made to treat it. When attributable to catarrhal irritation of the upper air-passages, it is best treated by topical applications. The following substances may be inhaled: compound tincture of benzoin, combined with paregoric or carbolic acid; formalin; creasote, alcohol, and chloroform in equal parts. For local applications by means of the spray sedatives and narcotics should be preferred, and a solution of cocain is sometimes most efficient. The cause may be found in pleurisy or pleuritic adhesions, and for this condition counter-irritants, as iodin, sinapisms, etc., may be used. Pleuritic coughs often demand codein or even morphin in moderate-sized doses. The cough is in most instances occasioned by the tuberculous bronchitis, and to a lesser extent by the vomicæ. Cough-mixtures as usually formulated are apt to disorder the digestive function, and in so far as they have this effect they are positively harmful. Syrups should be omitted from their composition. Creasote by inhalation is the remedy *par excellence* for tuberculous bronchitis, combined with spirits of chloroform and alcohol.

[1] See also *New York Med. Jour.*, Nov. 11, 1911, by G. Sanders.
[2] *Beiträge zur Klinik der Tuberkulose*, Würzburg, Dec. 10, 1910, p. 2100.
[3] *The Practitioner*, October, 1894.

When expectoration is copious, preparations of terebene, terpin hydrate, and tar may be resorted to; and when the cough becomes distressing, I employ codein (gr. $\frac{1}{8}$–$\frac{1}{4}$—0.008 to 0.016, every three or four hours) in the form of a granule. In the later stages morphin is allowable, since it is at this time that constant coughing or severe paroxyms of cough, if not restrained lead to utter exhaustion. Heroin, in doses of gr. $\frac{1}{6}$ to $\frac{1}{12}$ (0.01–0.005), three or four times a day, acts beneficially in allaying the cough that accompanies phthisis. Stimulant expectorants may be needful, and ammonium carbonate in the infusion of wild-cherry bark is perhaps most efficacious: a few drops of the deodorized tincture of opium or spirits of chloroform may be added.

(*b*) *Fever.*—Creasote has found a new field of usefulness in the treatment of the fever of tuberculosis. In my experience at all events, the cases in which it has been used, as above indicated, have shown a greatly diminished febrile movement. Cold or tepid spongings of the body at intervals of one, two, or three hours, according to the intensity of the fever, should be tried. Internal antipyretics are rarely advisable, since during the period of high temperature the cardiac action is much enfeebled; but if urgently called for, the following may be employed: acetanilid (dose gr. ij–iij—0.129–0.194), phenacetin (gr. iij–v—0.194–0.324). These are to be administered about two hours before the commencement of the daily rise in temperature, and repeated every three or four hours if necessary. Other antipyretics worthy of trial are the mineral acids and zinc oxid, but not quinin, which has utterly failed in my hands. Keeping the patient at complete rest when there is fever is of the utmost importance, though he should be wheeled into the fresh air for as long a time as possible during the day.

(*c*) *The Night-sweats.*—Among remedies that control the sweats most successfully may be mentioned—atropin (gr. $\frac{1}{120}$–$\frac{1}{60}$—0.0005–0.001); zinc oxid (gr. ij–v—0.129–324); sulphuric or gallic acid; muscarin (♏iij–vj—0.399 of a 1 per cent. solution); agaricin (gr. $\frac{1}{8}$–$\frac{1}{4}$—0.008–0.016). Sponging with equal parts of alcohol and tincture of belladonna is very effective, but my own best results have been derived from the use of atropin (gr. $\frac{1}{120}$–$\frac{1}{90}$—0.0005–0.0007) in combination with agaricin (gr. $\frac{1}{8}$—0.008).

(*d*) *Secondary Anemia.*—Bullock and Peters recommend subcutaneous injections of citrate of iron (0.05 gm. injected daily). Barlow and Cunningham advise the subcutaneous or intramuscular injection of arsenic or iron, or of the two in organic combination.

(*e*) *Dysphagia* may be a troublesome symptom, especially from involvement of the larynx, and it is best met by local applications of a solution of cocain in glycerin and water (gr. x to ℥j–0.648 to 32.0), thrice daily before meals. In advanced cases I have resorted to hypodermic injections of morphin (gr. $\frac{1}{8}$—0.008) before meal-time.

(*f*) *Gastric Disturbance.*—In nearly all cases of phthisis dyspeptic symptoms come on sooner or later, and for this gastric disorder nothing is so important as a proper regulation of the diet. Perhaps the medical treatment of the stomach symptoms has been dealt with at sufficient length, save that of vomiting, which may come on after meals and constitute a distressing concomitant. Those remedies giving the best results may be adduced as follows: cerium oxalate (gr. v–viij—0.324–0.518), in capsules before meals; calomel and soda in fractional doses; hydrocyanic

acid (♏ij–iij—0.133–0.199); and chipped ice with brandy sprinkled over it, taken at short intervals, but especially shortly before meal-time.

(*g*) *Diarrhea.*—The most important factor in the treatment of this symptom is a properly restricted dietary. Alum whey, mutton and chicken essence are of service, but curds of milk, beef-tea, and solids are not suitable. Of the numerous medical measures that have been employed, the most useful are bismuth subgallate, lead acetate, opium, thymol, salol, benzo-naphthol, and naphthalin. To these may be added the following acid diarrhea-mixture, each dose containing—

℞. Acid. acetici dil.	♏x	(0.666);
Morphinæ acetat.,	gr. $\frac{1}{8}$	(0.008);
Plumbi acetat.,	gr. j–ij	(0.0648–0.1296).

Complications when they arise must be dealt with according to accepted therapeutic principles.

LEPROSY.

(*Lepra*).

Definition.—A chronic, contagious disease, caused by the bacillus lepræ. It is distinguished by constitutional depression and, pathologically, by tuberculous masses in the muco-cutaneous surfaces, and by changes in the nerves.

Historic Note.—In 1889, Morrow stated that in India alone there were certainly not less than 150,000 lepers, while at present it is estimated that there are over 250,000. Its geographic distribution probably covers more than one-third of the entire surface of the globe. It is common in Africa, Brazil, in the East, and in Norway. In the Sandwich Islands the disease is of comparatively recent origin, and yet of great and increasing prevalence, a leper settlement having been established consisting of more than 11,000 cases. Leprosy is not unknown in America, and in Mexico it has existed ever since the time of Cortes (Morrow). Blanc states that there are 75 to 100 lepers in Louisiana alone. It was introduced into California and Oregon by the Chinese, and into Illinois, Iowa, Wisconsin, and Minnesota by Scandinavian immigrants. It has been imported from the Sandwich Islands to *Salt Lake City*, and from Normandy to Tracadie on the Gulf of the St. Lawrence, where the "disease is limited to two or three counties which are settled by French Canadians" (Osler). Sporadic cases have been met with in most American cities. The Commission on Leprosy reported in 1902 the records of 278 cases, of which 145 were native born Americans. The disease appears to be lessening in the United States.

Pathology.—The bacilli grow and develop in clusters in the tuberculous nodules in the skin and in the anesthetic and pigmented areas, residing within the epithelioid cells and leukocytes. These so-called lepra-cells are probably derived from the lymphatic vessels or capillaries, having been transformed by the bacilli. Surrounding the granulomatous masses is a layer of connective tissue. The bacilli are also found in the lymphatic glands, the spleen, and liver, but rarely in the blood. The nodular tumors form projections from the skin-surface, and, being poorly supplied with blood-vessels, they soon undergo caseation and absorption

or are obliterated by dense connective tissue (*facies leontina*). The pus-organisms generally exercise an influence in causing suppuration with ulceration, which may manifest a marked destructive tendency. Similar changes occur in the internal organs or in the mucous membranes.

Nerve-lesions are induced by the presence of the bacilli within and around the nerves. Here they set up an irritation with hyperesthesia (neuritis), leading to atrophy, with marked degenerative changes.

Etiology.—**Bacteriology.**—In 1880 Hansen discovered the bacillus lepræ, since proved to be the special agent of the disease. It strongly resembles the tubercle bacillus, but differential stains have been suggested by Unna and others. Bordoni-Uffredozzi was able to cultivate a bacillus which differed from the lepra bacillus in its morphology, although staining in a similar manner. His results have been confirmed by Czaplewski. Inoculation experiments on animals have not as yet succeeded.

Predisposing Causes.—Everyone is susceptible to leprosy. E. B. Goodhue, however, claims that a natural immunity exists. The disease is most frequent between the twentieth and fortieth years, and is rare in childhood. Sex and latitude have little if any influence. Hereditary transmission probably influences about one-fortieth of the instances (Zambaco). Heredity is denied by both Hansen and Raminez. As pointed out by Bidenkap, leprosy is often rare in large cities, even though prevalent in the surrounding rural districts.

Modes of Infection.—The disease is transmitted by contact; but Widal and others, who have studied the disease as it exists in the Hawaiian Islands, think that leprosy is contagious only by inoculation. Long's experiments point to transmission by means of the bed-bug. Morrow's view, that, like syphilis, leprosy is generally transferred by sexual intercourse, receives support. Hansen holds that the infection atrium is unknown; he thinks it probable, however, that the mouth and nasal cavities are the avenues of entrance. Sticker also regards the nasal mucous membrane as the primary focus, and finds in it constant lesions. The bacillus has been found in the floors and walls of houses in leper colonies, and also from the urine and even the milk of patients.

Clinical History.—Two forms are recognized, the *tubercular* and the *anesthetic*, but neither of these runs its entire course without developing into a third or mixed form.

The *incubation* is usually long (three to five years—Hansen). It may rarely be shorter or much longer. Vague *prodromes* are present for years (drowsiness, chilliness, recurring attacks of fever, debility).

(1) **Tubercular Form.**—In the first stage there is a patchy, cutaneous *erythema* with a slight hyperesthetic elevation of the affected areas (*macular leprosy*). These are oftenest seen on the face, the extensor surfaces of the arms and knees. They may vanish and leave the skin pigmented and anesthetic, and later the pigment may disappear, while white spots of corresponding size remain (*lepra alba*).

When the disease progresses less favorably tuberculous *nodules* (dusky red or almost brown in color) develop in addition to anesthesia. The small ones soon disappear, while the large ones are either absorbed or break down and ulcerate—changes which, as they advance together with the slow healing process, produce marked deformities. The *skin* is greatly thickened and presents a scaly surface, and there is loss of substance in certain parts, while others are enormously enlarged (eye-

brows, nostrils, lips, etc.). Among the many symptoms pointing to involvement of the mucous membrane are *ozena*, *hoarseness* or even *aphonia*, and the signs of *inhalation-pneumonia*. Blindness often ensues as the result of extension of the process. To ulcers extending deeply into the mucosa of the pharynx and larynx, death may often be ascribed.

(2) **Anesthetic Form.**—In this variety the *local symptoms* point usually to implication of the nerves. At the onset there are *pain* and *patchy hyperesthesia*, while minute bullæ, due to trophic changes, put in an appearance on the arms and legs. The *muscles* supplied by the branches of the affected nerve-trunk waste, and the superficial nerves feel thickened and nodular. *Bright-red patches* of vaso-motor congestion appear and soon become anesthetic, while the maculæ disappear. *Anesthesia* may proceed without the latter eruption. Dry, yellowish-white, scaly patches upon the trunk and extremities are also visible. Early their centers alone are anesthetic, but subsequently the loss of sensation spreads even to healthy portions of the skin.

Trophic alterations reach an extreme degree. Bullæ appear, and, bursting, leave perforating or destructive ulcers, usually upon the extremities. As the result of absorption, wasting, and necrosis great deformities are produced. The hands often take on a claw-like form, and the fingers and toes may disappear (*lepra mutilans*).

Diagnosis.—The early diagnosis rests upon the presence of patchy erythema with hyperesthesia, followed by the development of anesthesia, with a disappearance of the muscular eruption. Nodular neuritis is pathognomonic of anesthetic leprosy. Scrapings of the skin lesions frequently show the specific bacilli. In the advanced stages of either form confusion could scarcely arise. The nodular form of *tubercular syphilis* is distinguished by the distribution of the lesions, the history, the frequent sensory nerve-lesions, and by incising the tubercle and compressing serum from it—when lepra bacilli are found in the exudate—bacilli may be found in the nasal secretion. Zambaco and others have claimed that *syringomyelia* and *Morvan's disease* are in most cases but forms of leprosy; but this has been disputed by Hoffman, Schlesinger, and Sahli. Syringomyelia depends on lesions of the central nervous system, while leprosy has its nervous lesions in the peripheral nerves. The first symptoms in syringomyelia are localized usually in the upper extremities, while in leprosy they are generalized. In leprosy the tactile sense is usually lost, in syringomyelia usually not lost. Shoemaker and Boston[1] report an advanced case where lepra bacilli were found in the blood, and collected reports of 20 similar cases from the literature.

Prognosis.—Leprosy runs a very chronic course, lasting sometimes two, three, or more decades. The prognosis as to the final issue is hopeless, but the patient may live in comparative comfort for many years before the ravages of the disease cause great mutilation.

Treatment.—Certain diseases are supposed to exercise a retarding effect on leprosy (erysipelas, pneumonia, variola, phthisis). *Antagonistic inoculation*, however, as practised by Beaven Rake and others, has been practically negative in its effects; and the same is true of the treatment by Koch's tuberculin. The disease has thus far resisted all methods of treatment. Matthews[2] treated 7 cases representing both kinds of leprosy

[1] *Proceedings of the Philadelphia County Medical Society*, Jan., 1903.

[2] "Treatment of Leprosy with *x*-rays and High Frequency," *Indian Medical Gazette*, Aug., 1908.

with *x*-rays and high frequency, and concludes that it is the only method which has produced any real effect on the progress of the disease. *Internally*, chaulmoogra oil has been employed with excellent results, the dose being from 1 to 2 drams (4.0–8.0). It is sometimes administered in pearls (each containing ℥iij to v—0.199–0.333), in ascending doses, until the limit of tolerance is reached. Surgical interference may become necessary. Manson advises free excision if only one tubercle, and no signs of a general invasion, be present. Segregation of lepers has been instituted in certain localities with encouraging results. Calmette's antivenomous serum, while not a true antidote, may produce a marked temporary improvement or even cure in uncomplicated cases. The Nastin treatment (dose 1 c.c. by injection) has given variable results. Rost advises weekly injections of vaccine.

GLANDERS.

(*Farcy.*)

Definition.—An infection of equine origin, caused by the bacillus mallei. Two forms are recognized—*true glanders* and *farcy*.

Pathology.—The characteristic lesions are new growths (granulomata, according to Virchow), which are usually nodular in character, though they may be diffuse. These masses soften and form ulcers when they occur on the nasal mucosa, and abscesses when they are situated subcutaneously. Microscopically, the nodular tumors are composed of cells—lymphoid and epithelioid—together with the specific bacillus.

Etiology.—The morbid changes above described are caused by a specific organism, the *bacillus mallei*, which resembles closely the tubercle bacillus, though it is a little thicker as well as shorter. It is nonmotile. It can be readily grown, and as readily inoculated into horses, in which it produces the disease with every characteristic symptom. Perhaps the simplest method of staining the bacillus mallei "is to treat a cover-glass preparation with warm carbol-fuchsin (preceded by acetic acid), and then wash it off with a 2 per cent. solution of nitric acid."

Modes of Infection.—The virus is, as a rule, transferred directly from the infected animal to man, hence the disease occurs almost invariably among *males* and persons who come in contact with horses (hostlers, coachmen, soldiers, veterinarians, and farmers). Transmission from man to man has been observed, but rarely. The medium of conveyance is either the *pus* or the *nasal secretions*, which may drop or be blown from the animal's nostrils upon a wound in the skin or mucous membranes, however slight, and be absorbed.

Immunity.—The disease is rare in man because of natural immunity. Singer has produced artificial immunity by intravenous injections of sterilized cultures of the glanders bacillus.

Clinical History.—The duration of the *incubation-period* is from three to five days, and rarely longer. Both glanders and farcy may be *acute* or *chronic* in their course.

(1) **Acute Glanders.**—At first the signs of inflammation develop at the point of infection, *lymphangitis* and swelling of the adjacent lymphatic glands being associated. *Fever* and other evidences of general disturbance soon appear, and at the end of two or more days the nasal mucosa

becomes implicated, ulcers forming, from which a fetid muco-purulent (sometimes blood-streaked) discharge takes place. *Nose-bleed* is common. Later an *eruption* comes out on the face, the trunk, and the extremities, particularly about the joints. It is papular, quickly becoming pustular, and the pustules may dry up while fresh papules are developing—a characteristic feature. The *face*, particularly the nose, now swells, and a bluish-brown tumor covered with vesicles appears. Implication of adjacent mucous membranes—conjunctivæ, pharynx, mouth, etc.—is usual, and less frequently the bronchial and gastro-intestinal mucous membranes are involved. The ulcerative processes may extend to the bones, setting up necrosis. True arthritis occurs in 10 per cent. of the cases (H. Morel). Broncho-pneumonia is a common complication.

(2) **Chronic Glanders.**—A rare disease with mild but vague general symptoms, as muscular and arthritic pains, fever at intervals, asthenia, and progressive wasting, and the local features of nasal catarrh, with a bloody muco-purulent discharge. Cough may be present.

(3) **Acute Farcy.**—In this form the virus is inoculated into the skin, which presents the chief symptoms, the nasal condition being in abeyance or absent. The primary lesion is of an aggravated type, accompanied by numerous cutaneous boils and abscesses, often following the line of the lymphatics. Their favorite seat is in the vicinity of the joints. The constitutional symptoms simulate those of acute pyemia.

(4) **Chronic Farcy.**—Granulomatous tumors, resulting in abscesses, constitute the chief clinical peculiarity. The abscesses are situated primarily in the subcutaneous tissues, and often near the joints. As a rule they open spontaneously and discharge, first a thick, creamy pus, and later a thin, fetid material. They sometimes form distinct ulcers, extending in depth until the bones are involved.

The *general symptoms* simulate those of chronic glanders, the fever-curve being of the hectic type. In advanced cases emaciation and prostration become extreme. The *duration* varies from ten to eighteen months, though death may result earlier from some associated disease.

Diagnosis.—The diagnosis cannot be made without a clear history of contact with an animal known to be affected with the disease. In doubtful instances some of the suspected material should be injected into the peritoneal cavity of a male guinea-pig. Pus is soon formed in the tunica vaginalis testis and from it bacillus mallei may be recovered in pure culture. One of the products of the bacillus mallei is so-called "mallein," which has been used by Nocard and others as a diagnostic agent in animals. Its injection into horses suffering from glanders is followed by a febrile reaction. Schindelke found that a reaction of 3.5° F. (2° C.) is almost positive proof of glanders; while a rise of 1.25° F. (1° C.) is suspicious.[1] Wade recommends the complement-fixation test supplemented by the agglutination test on all negative serums.

Differential Diagnosis.—Cases of acute glanders have been mistaken for *variola;* but the history of exposure, the mode of onset, nasal symptoms and the course of the eruption all differ from those of the latter disease. *Pyemia* may be eliminated by the history of exposure and inoculation experiments. The chronic forms must be distinguished from *tuberculosis* and *syphilis.*

Prognosis.—Acute glanders and acute farcy are almost invariably

[1] Saunders' *Year-Book* for 1896, p. 1013.

fatal. The chronic forms, however, and particularly chronic farcy, end in recovery, under appropriate treatment, in nearly one-half the cases.

Treatment.—The primary lesion should be dealt with surgically, and thorough disinfection followed by cauterization is highly recommended. Bayard Holmes advocates the opening of fresh abscesses and the scraping out of old ones under an anesthetic. A supporting plan of treatment, by generous feeding and judicious stimulation, is to be adopted, and the symptoms are to be met as they appear. The product, "mallein," has been recommended as a specific, but its curative properties have not yet been demonstrated. Bristow reports a case of human glanders treated by an autogenous vaccine, with recovery.

ACTINOMYCOSIS.

(*"Big-jaw," "Lumpy-jaw," etc.*)

Definition.—An infectious disease of cattle, less frequently of man, caused by the ray-fungus (actinomyces), which grows in the tissues, developing a mass with secondary chronic inflammation and metastatic growth as well as a secondary pyemic infection.

Historic Note.—In 1877 Bollinger gave the first description of the ray-fungus, which he had observed in the disease known as "big-jaw" in the ox. Israel of Berlin discovered the fungus in man one year later. In 1879, Ponfick showed clearly that actinomycosis in man and cattle was one and the same disease. Murphy, who described the first case of actinomycosis hominis in America, states that up to the present date more than 500 cases have been reported.

Pathology.—A *macroscopic* mass is produced, consisting of a central fungous mass from which threads of mycelia radiate in all directions, producing the ray form of growth. Individual growths are of the size of a millet-seed, but their aggregation may result in masses as large as an orange; they are of a sulphur-yellow color and of tallowy consistence. Induration and infiltration may extend far into the surrounding tissues.

Microscopically, the little or single ray-like tumors show straight or wavy branching filaments (*supra*). Their development is accompanied by the growth of dense adjacent connective tissue. In addition, abscesses containing yellow granules in the pus occur, but these are secondary. In man the lesions consist of nodular growths with secondary abscess formation, differing from those described as occurring in beasts.

Bacteriology.—The organism of the disease belongs to the cladothrix variety of fungus, and may be cultivated, though with difficulty. The finer threads may readily be stained with anilin colors. The club-shaped projections, however, do not take these stains. Pus from whatever source should be examined for the actinomyces even though cocci are present. Rabbits and cows have been successfully inoculated. Pyogenic organisms are commonly in association.

Modes of Infection.—Infection generally takes place in young subjects through the mouth, teeth, and pharynx; and rarely the infection atrium is the air-passages or the skin. The infecting microbe is generally introduced with the food or drink, and Bostroem, from a study of 32 cases, concludes that the poison enters the economy by means of the ingested grains of some cereal (barley).

Clinical History.—(1) Oral Actinomycosis.—The patient often com-

plains of *toothache*, *dysphagia*, and of *difficulty in opening the jaw*. The latter symptom may be owing to induration of adjacent muscles, and is a very characteristic sign (Partsch). At the angle of the jaw a swelling appears, which quickly passes into suppuration; later it opens (first externally, then into the mouth) and discharges pus containing little yellow masses. If not properly treated, extension of the process takes place in a downward direction, even to the abdominal organs.

The *upper jaw* may be the primary seat of infection, and if so the base of the skull may be perforated and the disease attack the meninges and brain. Bollinger has seen primary actinomycosis of the brain. In these instances caries of the spine may result from extension.

(2) **Pulmonary Actinomycosis.**—I am satisfied that primary pulmonary actinomycosis is comparatively rare, although Karewski and Butler have each recently reported an instance. In Butler's case the disease followed an injury by a falling board. The disease begins with *pain in the side*, often the left, due to *pleurisy*. There are *cough* and a peculiar (fetid) *expectoration*, together with general wasting. A microscopic examination of the sputum, if made with care, reveals the *actinomyces*. In some instances the symptoms are identical with those of *disseminated tuberculosis* of the lungs (Brigidi), though generally the disease is unilateral. There is irregular fever, due to suppuration.

The **physical signs** may be those of chronic bronchitis merely; but there are, in not a few cases, extensive destructive changes of variable character (abscess, broncho-pneumonia, etc.), which modify the signs accordingly. In primary pulmonary actinomycosis an extension to adjacent organs and also metastatic growths and abscesses occur. Wood and Eshner[1] found the so-called sulphur granules in a pleural exudate.

(3) **Intestinal Actinomycosis.**—The condition may be *primary* or *secondary*. The organism grows upon the mucosa of the intestine and excites a proliferation of the underlying connective-tissue cells, and the formation of submucous nodules. The latter ulcerate, and perforation of the serous coat of the bowel may occur, inducing peritonitis. Pericecal abscesses have been formed in like manner.

The **symptoms** point to intestinal catarrh, there being some gastric disturbance, with recurring attacks of diarrhea. The actinomyces has been detected in the stools. Secondary metastatic growths (rarely) and abscesses may arise in other organs (liver, spleen, ovaries, etc.). The visceræ may also be the primary seat of infection.

(4) **Cutaneous actinomycosis** rarely occurs. The skin presents chronic suppurating ulcers which show the presence of the ray-fungus, and the condition bears a close resemblance to a lupus patch.

Diagnosis.—This rests solely upon the finding of the actinomyces. The wooden hardness of the tissues beyond the borders of the ulcers or sinuses, the hardness of the neighboring muscles in oral actinomycosis, and the yellow granules in the pus are all significant, but merely corroborative. To detect the actinomyces, says Warren, sections may be stained with Ziehl's carbol-fuchsin from fifteen minutes to half an hour, and then decolorized in a 1 per cent. picric-acid solution until the whole section has a yellow appearance. Dehydrate and mount. The fungus appears as a brilliant red aster, while the surrounding tissues are yellow. The points mentioned above will serve to distinguish this disease from

[1] *Medical Record*, June 4, 1910.

tuberculosis, *syphilis*, *chronic pyemia*, and *sarcoma*. Widal[1] differentiated actinomycosis by the sero-reaction in 8 cases.

Course and Prognosis.—The course is chronic. Mild cases may recover in from six to nine months or earlier, the oral form being perhaps the most favorable. Pulmonary actinomycosis may terminate in recovery, though rarely. Death usually results from pyemia, amyloid degeneration, and wasting.

Treatment.—This is mainly surgical. The removal of the parts involved and disinfection with acid-sublimate solution are the best measures. Billroth in a case of abdominal actinomycosis communicating with the bladder effected a cure by the use of fifteen tuberculin injections. Kinnicutt and Mixter have used vaccine made from actinomycotic pus in 8 cases with encouraging results. Internally, the potassium-iodid treatment, as first recommended by Thomassen in 1885, and recently emphasized by DaCosta,[2] is often attended with success when decided iodism is produced.

ANTHRAX.

(*Malignant Pustule; Splenic Fever; Wool-sorter's Disease, etc.*)

Definition.—An acute, infectious disease, caused by a special bacillus and clinically accompanied by the development of a characteristic pustule (boil) and blood-poisoning (*external anthrax*). The disease likewise affects the gastro-intestinal tract and the lungs (*internal anthrax*). Both forms are derived principally from the herbivora, it being especially prevalent among sheep and cattle. The occurrence of anthrax in the United States is much more frequent than has been held to be the case.

Pathology.—Post-mortem rigidity is marked. The blood is dark and thick and coagulates poorly, and in it, particularly in the spleen, as well as in the liver, kidney, and lungs, one may find the spores.

Besides the local lesions of the skin (*i. e.* ulceration, gangrene, edematous infiltration), and besides the degeneration of the heart, kidneys, and liver that is common to the severe and rapid infectious diseases, the especially striking lesion is the constant and great splenic enlargement.

The bowel may show hemorrhagic infiltration and gangrene, and the mesenteric and retroperitoneal glands may be enlarged and hemorrhagic.

Etiology.—Bacteriology.—The special agent is the *bacillus anthracis*. Gratia and Jonne give as the microscopic characteristics of anthrax, as seen in the blood, the following: (1) The anthrax bacillus has the form of a rod of a length varying from 5μ to 20μ, and in breadth from 1μ to 1.5μ. It is broken up into short articulations from 1.5μ to 2μ long, placed end to end like the sections of a tenia, the ends of each articulation being slightly swollen, giving the appearance of a bamboo cane; (2) clear spaces, appearing like a biconcave lens, exist between the ends of the articulations, and result from the slight concavity of these ends; (3) a capsule, often distinctly marked, surrounds the rod, seeming to form a protoplasmic support for the individual articulations. These threads of anthrax bacilli stain best with Löffler's blue. They grow readily on various media (agar, gelatin, potatoes, etc.) into inter-

[1] *Bull. de l'Acad. de Med.*, Paris, May 10, 1910.
[2] *Proc. of the Assoc. of American Physicians*, 1900.

lacing thread-like filaments which distinctly show spore-formation, the threads assuming the appearance of strings of beads. They resist desiccation, many of the germicides, and boiling water even for a few minutes. Inoculations are followed by the production of the pustule of anthrax. Conradi[1] affirms that it is highly improbable that the anthrax bacillus produces a *toxin*.

Modes of Infection.—The *virus* (spores) gains entrance into the human body through the skin (slight wounds, abrasions, or scratches), the intestines (with food), or through the lungs (rarely). The sting of insects (mosquitoes, flies) may also transfer the poison to man.

Predisposing Causes.—Occupation is most influential: persons who come into direct contact with infected animals (hostlers, butchers, shepherds), and workers in factories who handle the hair or hides of such animals, being liable.

Immunity.—Pasteur's well-known protective inoculation with attenuated virus has been extensively practised in anthrax localities, with very favorable results. Peterman, however, reinvestigated the question of immunity by the albumose of anthrax, and found it without protective action, except in the case of cultures on ox-serum, which, when injected in large quantities into the veins, conferred temporary immunity.

Clinical History.—The period of incubation is from one to three days. Two leading clinical types are distinguished:

(1) **External Anthrax.**—(*a*) *Malignant Pustule.*—At the point of infection (the hand, arm, neck, or face, or other exposed part) a small *papule* first appears, and develops into a *vesicle* of considerable size with bloody contents. This vesicle breaks, leaving a characteristic dark-bluish or black *scab* (anthrax), and encircling the primary vesicle an areola of miliary vesicles may be noticed. The base of the original vesicle now becomes swollen and indurated, and this brawny edema spreads rapidly to the adjacent tissues until an extensive area is involved. The neighboring *lymph-glands* may or may not be inflamed; if so, they are apt to be connected with the pustule by red lines (lymph-vessels, veins).

Severe *general disturbances* accompany the local disorder in the course of a couple of days, and comprise fever, decided prostration, sweats, splenic enlargement, and delirium tending toward coma. If recovery occur, the edematous swelling subsides and the black scab is cast off. In unfavorable instances collapse develops, and the case ends fatally between the fourth and eighth days. In such instances intestinal symptoms (diarrhea) or nervous phenomena of aggravated type may attend.

(*b*) *Anthrax Edema.*—In a certain proportion of the cases the *systemic infection* is out of proportion to the *local disturbance*, the latter consisting of an edematous swelling without the presence of an eschar. The eyelids (commonly), lips, tongue, and upper extremities may be the seat of extensive swelling, though there is no change in the color of the skin. This is a dangerous condition, and may result in gangrene.

(2) **Internal Anthrax.**—(*a*) *Intestinal Mycosis.*—In this form certain general, indefinite symptoms are the primary features, such as headache, pains in the limbs, anorexia, languor. Soon acute gastro-intestinal features supervene, sometimes preceded by a chill. As a rule, vomiting occurs, followed by abdominal pains and diarrhea, and the stools often become bloody. Hemorrhage may also occur from other outlets.

[1] *Zeitschrift für Hyg.*, June 14, 1899.

Other symptoms, as dyspnea, marked cyanosis, and restlessness, are noted, followed sometimes by stupor, general convulsions, or spasms of single muscles or groups of muscles. There is moderate fever, and the spleen is enlarged. Death is preceded by collapse.

Interesting *epidemic* outbreaks of internal anthrax have occurred, due both to drinking-water derived from infected wells and also to diseased meat. Murisier has related the history of an epidemic in which 200 persons fell ill after eating meat from a certain cow. The animal was quartered by a butcher who had previously slaughtered an ox afflicted with anthrax, and had not disinfected his instruments; four days after this 25 persons were attacked by the disease.

(*b*) *Wool-sorters' Disease.*—This occurs among the operatives in factories in which imported wool or hair, mostly from Russia and South America, is sorted, and to produce the typical affection the infection must be swallowed or inhaled in the form of dust. *Mixed cases*, or those showing both external and internal anthrax, may be met with among workers in curled-hair establishments and the like. The *onset* is sudden, with a chill that is accompanied by pains in the back and legs, prostration, and a sharp rise of temperature to 102° or 103° F. (39.4° C.). The *local* symptoms may either be chiefly pulmonary or gastro-intestinal. The former consist in dyspnea, chest-pains or feelings of constriction, cough, and rarely the physical signs of bronchitis; the latter comprise vomiting and a diarrhea that is followed by collapse, with marked lividity. Nervous symptoms, delirium, convulsions, or coma are often prominent in serious forms; but a fatal ending may occur while the mind is unclouded. The *course* ranges from one to five days.

(*c*) *Rag-pickers' Disease* ("Hadernkrankheit").—This has been identified by Eppinger as the same form of disease as "wool-sorters' anthrax." It occurs among the rag-sorters in the paper-mills near Graz. Infection occurs in the respiratory tract. The symptoms observed are high fever, followed by collapse, with depression of the body-heat, painful and paroxysmal cough, cyanosis, very weak heart, together with the signs of pleuritic effusion and consolidation of the lung.

Diagnosis.—The history (occupation, etc.) and the appearance of the malignant pustule in external anthrax leave little room for doubt. The diagnosis, however, should be confirmed by an examination of the contents of the pustule for the presence of bacilli, and if found they should be cultivated and inoculated upon a guinea-pig or rabbit.

Internal anthrax may be suspected if the more characteristic pulmonary or gastro-intestinal symptoms, together with those of systemic intoxication, develop in persons whose occupation entails exposure. In doubtful cases the presence of bacilli in the blood must be shown.

Prognosis.—In external anthrax occurring in healthy persons the disease often pursues a favorable course; moreover, radical surgical measures have decreased the death-rate decidedly. Internal anthrax, however, is a deadly affection. As regards "wool-sorters' disease," those who survive for one week usually recover (Bell).

Treatment.—Prophylactic measures embrace the sterilization and destruction of the hair, hides, wool, etc., of infected animals as well as the cremation of their bodies. Subsequent disinfection of the infected premises and the prohibition of grazing in infected pastures are matters of the utmost importance. In the carbuncular form, if seen early, the

best treatment is excision of the affected area, including a considerable amount of surrounding skin. In the edematous variety, early excision followed by cauterization is indicated. If impossible, as is the rule, injections of carbolic acid in a solution of water and glycerin (1:10) into the surrounding tissue have given the best results. Hallopeau recommends that in order to prevent extension the neighboring structures be bathed with a 10 per cent. solution of carbolic acid (first dissolved in alcohol) in oil or glycerin. Internally, stimulants, antiseptics, and nourishing food constitute our chief reliance. In internal anthrax efforts at treatment avail nothing. Several sera have proved valuable in the treatment of anthrax, the best being that of Sclavo, which is obtained from the sheep or ass (Emery).

HYDROPHOBIA.

(*Rabies.*)

Definition.—A specific, infectious disease peculiar to carnivora and to a less extent to herbivora, which may be communicated to man by direct inoculation. It is characterized by slight fever, spasm of the larynx and pharynx, delirium, a short stage of paralysis, coma, and, in the great majority of cases, a fatal termination.

Pathology.—The facies, pharynx, and esophagus may be congested, the latter organ being sometimes markedly edematous; pulmonary congestion has also been noticed. The mucous membrane may show here and there points of hemorrhage, and Fitz has observed blood-extravasations into the perivascular spaces of the brain. Soft thrombi may fill the cerebral vessels, especially the veins, while the blood has a dark color and its clots lack firmness.

Balzer, Benedikt, Kolesnikoff, and Schaffer made studies of the changes in the nervous system. Later, Babes described the "*tubercles rabiques*," which consist of pericellular accumulations of embryonal cells, the latter finally taking the place of the destroyed cell. More recently Van Gehuchten and Nèlis discovered lesions in the cerebro-spinal and sympathetic ganglia; they "consist in the atrophy, the invasion, and the destruction of the nerve-cells brought about by new-formed cells derived from the capsule, which appears between the cell-body and its endothelial capsule. These new-formed cells increase in number, invade the protoplasm of the nerve-cell, and finally completely occupy the entire capsule." Rarely, the kidneys may show cloudy swelling.

Etiology.—Pasteur has found the poison abundantly present in the nerve-centers, and has transferred the disease by taking bits of brain-substance or medulla derived from an infected animal and inoculating them into healthy subjects.

Bacteriology.—The micro-organism of the disease has not yet been determined, though Spenelli, Rivolta, Foll, Ferran, and others have described a bacillus. Memmo [1] believes he has established its claims as the specific organism, and reports successful production of the disease in dogs, rodents, and birds, with the typical differences characteristic of each.

The usual *mode of infection* in man is through the bite of a rabid animal, the virus being contained principally in the saliva, and in an immense majority of cases (about 90 per cent.) the dog is the offending

[1] *Centralbl. f. Bakt.*, Abt. i., Bd. xx., 17, 18.

party. The cat, wolf, cow, and horse also suffer from the disease, and in rare instances they communicate the disease to man. The skunk is also liable, and its bite has often transmitted rabies, especially to persons sleeping in the open air or in tents which the animal can enter. The virus gains access to the system through the broken skin.

Susceptibility to the poison exists in about one-half the instances in which persons are bitten by rabid animals, though in some cases this apparent immunity may be owing to slight or even non-infection.

Clinical History.—The *incubation-period* lasts from six weeks to three or four months, though in young subjects and in cases in which the infection is severe the symptoms develop earlier. Certain *prodromal symptoms* are manifested, as a rule, and generally last only a day or two; I have, however, seen two instances in which melancholia, due probably to the dread of what might follow, showed itself immediately after the reception of the bite and persisted. The usual premonitory symptoms are headache, loss of appetite, sleeplessness, great depression of spirits, and sometimes darting pains that radiate from the seat of the bite. The adjacent lymph-glands may become swollen, and slight difficulty in swallowing is experienced.

Following the *invasion* are two stages: (1) **The Stage of Excitement.**—The patient wears an expression of the most intense anxiety. Hyperesthesia is present and attains to a marked degree, and the special senses exhibit the keenest vigilance, a noise or a draft of air often causing great psychic disturbance or a violent reflex spasmodic contraction of the larynx. Quite early the mere sight of water is dreaded by the patient, and forms a characteristic feature of the disease. This symptom has given the name hydrophobia to the disease, and springs from the fear of inducing a painful spasm of the larynx. The patient has thirst which he cannot assuage. There may be maniacal excitement, and the spasmodic contractions of the larynx may become so strong as to excite urgent dyspnea, with the emission of curious sounds. The muscles of the mouth may also exhibit convulsive movements, causing the patient to make snapping sounds; these, however, are secondary. There is associated great restlessness, with frequent lateral rolling of the head, and foaming saliva may be ejected from the mouth. The symptoms occur in paroxysms, and during the intervals the patient is generally free from excitement. There is fever as a rule, the temperature ranging from 100° to 102° F. (37.7°–38.8° C.) or over, but it may be absent; the pulse is moderately accelerated and is sometimes irregular, and toward the end of this stage the reflex spasms of the respiratory apparatus develop spontaneously. Mental aberrations and melancholia may ensue, and often lead to suicidal tendencies.

(2) **The Paralytic Stage.**—In the concluding stage the patient passes into actual unconsciousness or coma, without spasms. This lasts from twelve to eighteen hours, ending in death.

In man there is a *paralytic form* of rabies, but it is rare as compared with the delirious or psychic type. Thirty cases have been reported by Gamaléia, and it is apt to follow deep and multiple bites. The paralysis begins near the part bitten, and spreads until it becomes general, finally involving the respiratory centers. In rodents quiet madness ("dumb rabies"), without maniacal excitement, is the rule.

Diagnosis.—The hyperesthesia, the fear of water, the reflex spasms

on attempting to swallow, accompanied by dyspnea and great mental agitation, form a very characteristic grouping of symptoms. Bits of brain-substance or medulla of the rabid animal that has inflicted a bite should be quickly obtained, and a subdural inoculation of a rabbit be made. If virulent, the paralytic form of the disease will ensue in from fifteen to twenty days. Ravenel and McCarthy,[1] following the method[2] of Van Gehuchten and Nèlis, conclude that when present the capsular and cellular changes in the intervertebral ganglia, taken in connection with the clinical manifestations, afford a trustworthy means of diagnosis of rabies in the animal. When these changes, however, are absent (as happens in early stages of the disease), rabies cannot be excluded. *Hysteria* may be misleading, but here the previous history suffices.

The name *lyssophobia* has been given to cases that simulate, but have no relation to, hydrophobia, and Mills has advanced the warning that, however suggestive the symptoms following a dog-bite, the given case cannot be assumed to be a case of hydrophobia until other possibilities are excluded. It is highly probable that there is a form of hydrophobia which is the result of the wide publicity given to genuine and suspected cases alike. The characteristic symptoms may be present, but the affection does not develop. This so-called *pseudo-hydrophobia* appears only in neurotic and hysteric subjects. Recovery is the rule. Burr reports an interesting case of the kind that occurred in Osler's clinic, attended, however, with recovery.

Prognosis.—Few if any cases of rabies in man recover if the disease be allowed to develop.

Treatment.—**Prophylaxis.**—Upon the reception of a bite thorough disinfection, followed by cauterization of the wound with caustic potash, or, better still, excision, if important structures be not involved, is a measure that can be quickly carried out. The wound is then to be kept open for a period of four or five weeks. Dudley advises that a tourniquet should be applied if the bite be on an extremity. Systematic muzzling of dogs is to be encouraged and advised.

Preventive inoculation as perfected by Pasteur is a precautionary measure of the utmost importance. He showed that the virulence of the virus which is obtained from the nervous system undergoes modification by passage through animals. Thus the potency of the virus is increased by its inoculation from rabbit to rabbit (by placing bits of spinal marrow beneath the dura mater), the period of incubation at the same time growing shorter, till at last it is but seven days. On the other hand, the virulence is decreased or attenuated as the result of similar experiments upon the monkey. Pasteur also found that if fragments of the spinal cord were suspended in a dry atmosphere they lost

[1] *Proc. Path. Soc. Phila.*, March, 1901.

[2] This is as follows: The ganglion is put at once into absolute alcohol, in which it is left for twelve hours, the alcohol being changed once. It is transferred for one hour to a mixture of absolute alcohol and chloroform; next put for one hour into pure chloroform; then for one hour into a mixture of chloroform and paraffin, and lastly in pure paraffin for one hour. The sections are put in the oven for a few minutes, then passed through xylol, absolute alcohol, and 90 per cent. alcohol, after which they are stained for five minutes in methylene-blue according to Nissl's formula, differentiated in 90 per cent. alcohol, dehydrated in absolute alcohol, and cleared in essence of cajuput and xylol. Ravenel and McCarthy found that the capsular changes were best brought out in sections stained by hematoxylin and eosin. Since these latter changes are the most essential diagnostic features in the sections, they suggest that material unfit for the Nissl method will still show the capsular changes when stained by hematoxylin and eosin.

gradually their virulence and finally became inert. From these an emulsion is prepared which is employed in the antirabic inoculations in man. In this way he secured a virus of known and reliable strength, and with this he could readily render the dog refractory by inoculating with very weak virus; then, by increasing from day to day the virulency of the inoculations, complete immunity was established.

Protective Inoculation.—"The patients are first inoculated with a cord fourteen days old, and the inoculation is repeated daily for nine days, each time with a cord one day fresher. In winter the oldest cords used are five days old, and in summer cords that have been drying for four days are also employed" (Warren).

For patients who have been bitten on the face, hands, or bare feet, as well as for those who have been bitten long before commencing treatment, the special preventive method, the so-called "intensive treatment," is applicable. Briefly, this consists in eliminating some of the inoculations of intermediary strengths, thus lessening the number of injections, and also in administering the latter at shorter intervals than in the usual method of treatment. The success of the Pasteur method is universally attested. Pottevin gives the following summary of figures from the Pasteur Institute: From 1886 to 1894, 13,817 persons were bitten, with a mortality of 0.5 per cent. In the New York Pasteur Institute, 313 West Twenty-third Street, under the directorship of Paul Gibier, of 1367 cases treated during the decade ending Jan. 1, 1900, 19 died—a mortality of 0.66 per cent. The patients should be sent to the Pasteur Institute at once, since delay diminishes the protective power of the inoculation.

The established affection defies all known methods of treatment. Our aim should be to diminish the intensity of the painful spasms and the psychic disturbances. The patient should be isolated from sounds, light, and excitement of every sort. Food, as a rule, must consist of nutrient enemata, though by the local application of cocain the sensitiveness of the throat may be diminished sufficiently to enable the patient to take liquid nourishment (Osler). For controlling the spasms chloroform by inhalation is most effective; chloral internally and morphin hypodermically may be of advantage. The patient's anxiety is best relieved by a cheerful demeanor on the part of the attendants.

TETANUS.

(*Trismus; Lockjaw.*)

Definition.—An acute, infectious disease caused by the tetanus bacillus. It is characterized by painful spasms, affecting first and chiefly the muscles of the jaw and neck (*trismus*), and secondly those of the trunk, especially the extensors of the spine and limbs (*opisthotonos*). Two varieties are recognized: (*a*) idiopathic (?); (*b*) traumatic. In certain institutions and certain localities it occurs endemically, and among newborn children and the colored race it may prevail epidemically (*trismus neonatorum*). The incidence of the disease, however, is decreasing.

Pathology.—No constant post-mortem lesions have been found. The virus acts principally upon the nervous centers of the medulla

and the cord, producing inflammation (and sometimes softening) of the gray substance of the cord. According to Brown-Séquard, the characteristic lesions are consequent upon an *ascending neuritis* starting from the wound, and it is true that the nerves often present traumatic lesions with redness and swelling of the neurilemma. Tetanus neonatorum often shows inflammation of the umbilicus.

Etiology.—Bacteriology.—In 1884, Nicolaier discovered the bacillus of tetanus, and in 1886, Rosenbach first found it in man. It is a long, slender rod, at one end of which appears a swelling due to the formation of a spore in that locality, thus giving the organism an appearance like that of a pin or drumstick. The bacilli are easily stained by Abbott's method, and are purely anaërobic. Pure cultures can be made, but with difficulty, since they will not grow in the presence of the smallest amount of oxygen. If pure cultures are injected into animals, typical tetanus follows. Brieger has obtained two poisons from sterilized cultures of the bacillus in the pure state, and termed them "tetanin" and "tetano-toxin"—both most virulent poisons in the minutest quantity. These alkaloidal substances produce the tonic convulsions; hence tetanus is purely toxic in nature—an intoxication. The bacilli are most probably limited to the point of infection, and here develop the *toxin*, which "is carried mostly along the nerves to the spinal cord" (Stintzing).

Tiberti whose experiments corroborate those of Meyer and Ransom, found that the toxin is transported to the nerve-centres through the plasma of the nerve-fibers, but that the normal integrity of the axis cylinders to effect the conduction is preserved.

Modes of Infection.—In the outer world tetanus bacilli are found to be both numerous and widely distributed. They abound in the earth (garden-soil in particular), putrefying liquids, manure, in rubbish, and dust of streets and houses, etc. The fact that the bacillus of tetanus is anaërobic explains why it is most apt to follow punctured and contused wounds. An analysis of 1201 cases by the writer and A. C. Morgan[1] affords convincing proof that every case is the result of the introduction of the tetanus bacillus through a lesion of the skin, however minute it may be, and that so-called idiopathic or "rheumatic" tetanus does not exist. The presence of the bacillus in vaccine has apparently been the cause of some recent cases. The *locality* of the injury is most commonly on the extremities, particularly on the hands and the feet, although the figures of Anders and Morgan (previously cited) indicate the great susceptibility of all portions of the body to the poison.

Certain Prediposing Causes.—1. Males are more susceptible than females (*e. g.*, out of 981 cases the former sex made up 79.3 per cent.), although males are more exposed to infection. 2. The robust are more receptive than the weak, and the nervous than the lymphatic. 3. Season. In 687 cases the seasonal occurrence was recorded by Morgan and myself and indicated that tetanus is more prevalent in the hotter as compared with the colder months of the year. The maximum number of cases occurred in July (4th of July tetanus). 4. Age. An analysis of 583 cases, with reference to liability according to age, gave 229 cases, or 39.3 per cent. from the fifth to the fifteenth years of life, 145 cases, or 24.9 per cent. from the fifteenth to the twenty-fifth years, while there were 86 cases, or 14.8 per cent. between twenty-five and thirty-five years. After the fiftieth year only 14 cases occurred.

[1] *Journal of the Amer. Med. Assoc.*, July 29, 1905.

Immunity.—Behring and Kitasato have rendered animals immune by the injection of cultures of the bacillus after the addition of iodin trichlorid to diminish their strength, and this serum has been successfully used to protect others against tetanus.

Clinical History.—The duration of incubation depends upon whether the given case pursues an *acute* or a *chronic* course. In acute tetanus it lasts from one to two weeks, while in chronic the first symptoms usually appear after the second week. In so-called idiopathic tetanus the symptoms appear shortly after exposure to the special causes.

Symptoms of Acute Tetanus.—(1) Mild prodromal symptoms (languor, headache, etc.) may precede the more intense characteristic phenomena, which develop gradually. At first the patient complains of stiffness and tension in the muscles of mastication and back of the neck, and soon tonic spasm of the masseters renders the facial muscles more or less immobile and locks the jaws (*trismus* or *lockjaw*). The rigidity of the cervical muscles is shown by the retraction of, and by attempts at raising, the head. The physiognomy is distinctive; it is immobile, the forehead being often wrinkled and the corners of the mouth retracted, producing a peculiar smile (*sardonic grin*). Next the muscles of the body become rigid, first the trunk (*orthotonos*), and then the spine is bent or bowed and the convexity presents anteriorly (*opisthotonos*). Lateral arching of the body also occurs, though rarely (*pleurosthotonos*). The belly-muscles are hard and board-like, and their contractions may throw the body forward (*emprosthotonos*). The arms generally remain movable, but the legs may be rigidly extended. The position of the body is one of constant rigidity, but from time to time convulsive seizures of variable duration occur, causing most agonizing suffering, thoracic oppression, dyspnea, and more or less cyanosis, due to interference with the respiratory function (especially spasm of the glottis). Sharp, lancinating pains occur at the base of the chest. "Convulsive dysphagia" (as in hydrophobia) is rarely observed. These spasms are usually reflex. The reflexes are increased. Rostowzew thinks that Kernig's symptom is an early and constant one in tetanus. The intellect remains clear. Profuse perspiration is a significant symptom.

Fever of a moderate degree is generally present. The temperature, however, may suddenly leap to 110° or 112° F. (43.3°–44.4° C.), forming an ominous symptom, these extreme elevations of temperature being probably due to paralysis of the centers that regulate bodily heat. Conversely, fever may be absent throughout the attack, and a brief postmortem rise of temperature be seen. The *pulse* is quickened, and in the worst cases may become very rapid (140 to 160 beats per minute), small, and irregular. The *urine* may be suppressed or its passage impeded by the muscular contractions. The bowels are constipated.

(2) **Chronic Tetanus.**—The same symptoms are manifested as are seen in the acute form, but the condition does not progress so rapidly. In some instances the symptoms soon become aggravated, to be followed, however, by periods of decided relief from the painful spasms, so that during the latter the patient's strength can be maintained by means of stimulating food, and intervals of partial freedom from the excruciating pains grow longer in favorable cases, until finally the period of convalescence may be reached. *Relapses*, however, are common.

(3) **Cephalic tetanus** (first described by Rose) usually follows injuries to the head (face). Its most characteristic symptoms are rigidity of the

masseter muscles, spasm of the pharyngeal muscles, causing dysphagia, chronic contraction of the muscles of the neck and abdomen (rare), and paralysis of the facial nerve on the same side as the injury. The latter symptom is due to local infection by a toxin. Recovery takes place in about 25 per cent. of the instances, according to Willard's statistics.

Diagnosis.—In view of the usual history, the predominating feature—trismus—together with the early appearance of rigidity at the back of the neck, will, as a rule, render the diagnosis a simple one.

Strychnin-poisoning must be eliminated, in which the following table will assist:

Tetanus.	Strychnin-poisoning.
Reception of a wound, generally followed by a period of incubation.	Ingestion of strychnin, followed immediately by the symptoms.
Begins with lockjaw; later spreads downward (the arms and hands escaping).	Begins with gastric disturbance or a tetanic contraction of the extremities. Hyperesthesia of the retina occurs and objects look green.
Reflex spasms not present at the outset.	Violent convulsions present from the onset.
Rigidity is persistent, except in the chronic form.	Intervals of complete relaxation occur.
The course is prolonged into days or weeks.	Course is brief, terminating in death or recovery.
Cultures made from the discharges of the wound show the bacillus tetani.	Examination of the gastric contents shows strychnin.

Tetany gives rise to a prolonged spasm affecting the extremities (hands in particular) and the larynx, with intermissions; it is also characterized by a peculiar posture, and occurs chiefly in the young.

Hydrophobia is discriminated from tetanus by the history of a bite from an animal, by the predominance of the reflex spasm of the respiratory apparatus, by the intensity of the psychic disturbance, and by the absence of lockjaw and opisthotonos.

Course and Prognosis.—"Acute tetanus or that which developed within ten days gave a total of 568 cases and a mortality of 74 per cent. On the other hand, 211 cases lasted over fifteen days, with only 18 deaths, or 8.5 per cent. mortality" (Anders and Morgan). Death results from asthenia, heart-failure, or asphyxia (during the paroxysm). According to Richter's statistics, 88 per cent. of military cases are fatal. In the so-called idiopathic cases the mortality-rate is under 50 per cent. Chronic tetanus gives a less grave prognosis than does acute. There is a direct relation between the duration of the incubation period and the mortality-rate.[1] In the newborn recovery is so rare that when it occurs the diagnosis may be called into question.

Treatment.—In traumatic cases the wound must be disinfected and thoroughly cauterized. The agents employed must be brought in contact with every portion of the wound, so that punctured wounds must first be laid open. Excision of the wound, and even amputation, may be advisable in some cases. The fact that the deadly poison is developed at the site of infection gives to the local measures supreme importance in the treatment. The application of lipoid substances (*e. g.*, Peruvian balsam salve) is found to delay the incubation period (Brockenheimer).

The patient should occupy a secluded room with little light and a

[1] *Journal of the Amer. Med. Assoc.*, July 29, 1905.

carefully regulated temperature. A single nurse will suffice, and all sources of external irritation should be avoided. A nourishing *diet* is demanded, and rectal feeding must be instituted as soon as it is found that food cannot be administered *per oram*, or the food may be introduced by means of a small stomach-tube or catheter passed through the nostril. Stimulants hypodermically should not be spared when the heart's action becomes quick and feeble. The spasms are best controlled by chloroform-inhalations, and during the intervals the patient should be kept under the influence of morphin, administered subcutaneously. Kintzing[1] reports excellent results from a solution of pure phenol (10 per cent. strength) in sterile water. Of this solution, the adult dose employed was 10 drops diluted, by hypodermic injection deep into the muscles, and repeated every three hours in the beginning, increasing the interval as improvement manifested itself. Among other capital remedies are chloral hydrate and Calabar bean. The former may be exhibited by rectal injection (gr. xl—2.59 at a dose), to be repeated at intervals of six or eight hours until the spasm is overcome. The heart, however, must be carefully guarded. Rarely, chloretone, potassium bromid, curare, nitrite of amyl, belladonna, and cannabis indica are useful.

Tetanus-antitoxin has been recommended for the *cure* of the disease, and is prepared in both fluid (antitoxin serum) and dry form. A dried preparation (which does not deteriorate) is also obtainable from Merck and his agents in the form of tubes containing from 4 to 5 grams each; at the time used it may be dissolved in water or in glycerin. Of Tizzoni's dried antitoxin 2.25 grams are to be given at the first dose, and 0.6 gram at subsequent doses. As shown by recent experimentation, antitetanic serum may prevent further invasion, but it cannot cure infection that has already reached the spinal cord and brain. The dose, as recommended by Copley,[2] should be large (30 c.c. at once, to be repeated at least every six hours until improvement is seen). Behring insists upon giving the serum not later than twenty-six hours after the commencement of the attack. Hoffmann[3] records recovery in 14 out of 16 cases injected with the serum, intradurally. Torres recommends the injection of 120 c.c. of tetanus antitoxin *intravenously*, to be repeated twice in the twenty-four hours, a third injection of 100 c.c. on the second day and this repeated on the following days, if symptoms be still present. Magnesium sulphate (2 c.c. of a 25 per cent. solution) may be injected into the spinal canal after the removal of an equal quantity of cerebrospinal fluid.

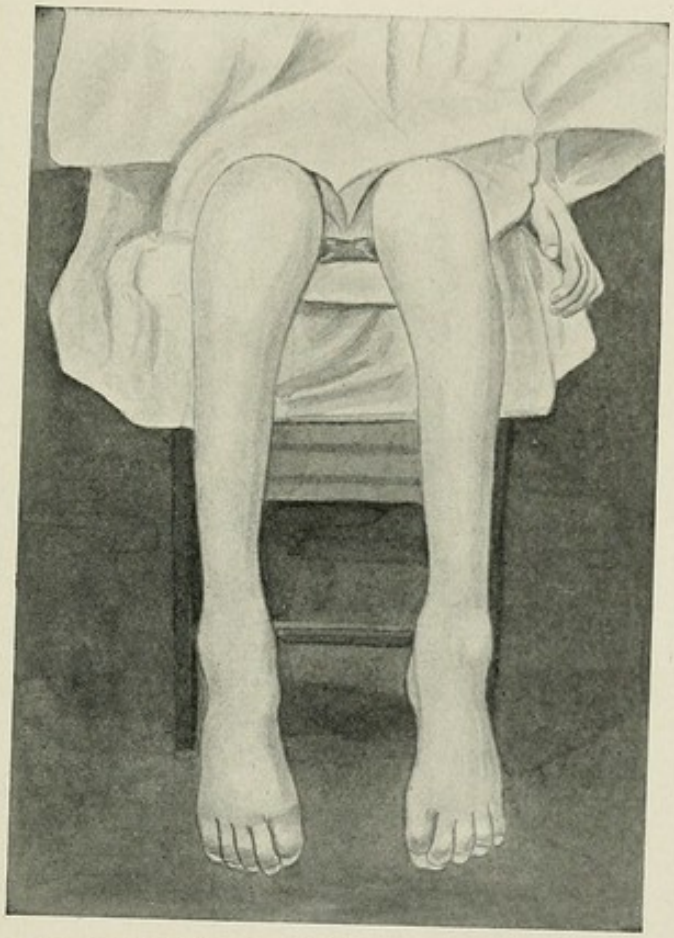
Prophylatic injections of 500 antitoxic units should be used at once after disinfection of the primary focus. Baccelli advises subcutaneous injections of carbolic acid. The dejecta should also be thoroughly destroyed as the tetanus bacillus has been found in the intestinal tract.

BERI-BERI.

(*Endemic Multiple Neuritis; Kakke;* "*Weak legs.*")

Definition.—Beri-beri is a specific disease characterized clinically by fever, muscular weakness followed by muscular atrophy, pain, tenderness, paresthesia, gastro-intestinal disturbance, tachycardia, and often general anasarca. It is not certainly contagious.

[1] *New York Med. Jour.*, Dec. 23, 1911. [2] *Brit. Med. Jour.*, Feb. 11, 1899.
[3] *International Clinics*, vol. ii., S. 20, p. 17.



BERI-BERI (Herzog, in *Philippine Journal of Science*).

Historical.—Beri-beri, first recognized by Strabo among the soldiers in the Roman armies while occupying Arabia (24 B. C.), was strangely enough not grouped with the infections until the beginning of the nineteenth century. At this period the subject began to receive the serious attention of Dutch and (a little later) of Anglo-Indian writers, and investigators. As stated by Osler, however, we may date the modern study of the disease from Malcolmson's monograph, published in Madras in 1835. It remained for Sheube and Baelz to point out that the principal morbid lesions are those of a multiple peripheral neuritis.

Distribution.—The disease is endemic in tropical and subtropical countries, but may occur epidemically on shipboard, in prisons, and in armies. Instances of epidemic prevalence in armies are numerous, *e. g.*, in Arabia, India, and the Philippines. Birge has reported an outbreak in which 11 out of 13 of a ship's crew were attacked, and Bondurant described the epidemic that occurred at the State Hospital for the Insane at Tuscaloosa, Ala., while J. J. Putnam observed cases among the New England fishermen. The principal habitats, however, are certain parts of Asia, namely, Japan, China, India, the Malayan Archipelago, the Dutch Colonies, and the Philippines. In all of these countries the cases may multiply under favorable conditions into extensive and devastating epidemics. In England and along the Pacific Coast, among the Japanese and Chinese, it is not uncommon at the seaports.

Pathology.—The essential feature is the changes in the nerves; these are inflammatory and degenerative, involving the medullary sheaths and axis-cylinders. In addition to the peripheral nerves, the pneumogastric and phrenic may be affected. Degeneration in the muscles also occurs, and, not uncommonly, serous effusions.

Etiology.—Although beri-beri is most probably an infectious disease, the specific cause still remains in doubt. Both the bacillus of Hamilton Wright and the coccus of Okata and Kokubo have been shown to bear no causative relation to the disease. Beri-beri is the result of a protozoan infection (Hewlett and de Korte[1]). Davis, of Shanghai, showed a direct relation between the presence of bed-bugs and beri-beri among Chinese prisoners.

A second leading theory ascribes the disease to polished rice and fresh fish. Fraser and Stanton's[2] experiments on chickens and human beings indicate that polishing rice removes something (pericarp?) necessary to maintain health on a strict rice diet. Chamberlain and Vedder[3] have induced a disease somewhat resembling beri-beri in children nursing from mothers suffering with beri-beri by the administration of rice-polishing extract. The striking diminution in the number of cases in Japan following the introduction of an improved dietary would seem to support the food theory, but the number of cases of beri-beri that developed during the Russo-Japanese War (in the Japanese army) did not exceed 75,000 to 80,000.[4]

Predisposing Causes.—(*a*) The principal disposing condition is overcrowding, more particularly when combined with antihygienic surroundings. This fact explains the localized outbreaks in armies, asylums, ships, and the like. (*b*) Certain climatic factors (*vide* Distribution, *supra*),

[1] *Journal of Tropical Medicine and Hygiene*, October, 1907.
[2] *Philippine Jour. of Science*, 1910, v., 55 to 64.
[3] Editorial, *Jour. of the Amer. Med. Assoc.*, June 15, 1912.
[4] M. Herzog, *Philippine Journal of Science*, Feb., 1908.

as heat and humidity, favoring the development of beri-beri; hence natives of tropical countries and imported coolies suffer most. (*c*) *Age and sex.* More cases occur among males than females, and the decade from 15 to 25 years furnishes a large proportion of the cases.

Symptoms.—The period of *incubation* is not definitely known, but is probably from ten days to one month, or even longer. Prodromata are commonly present; they are *thoracic oppression*, *epigastric pains*, *anorexia*, *headache*, and a slight febrile movement. Rigors rarely occur.

Four clinical varieties are recognized:

1. **Atrophic Form.**—This is characterized by *muscular weakness*, slowly developing, leading to paralysis of the lower limbs and trunk, rarely extending to the arms, head, and neck. Atrophy of the affected muscles quickly ensues, with loss of the deep reflexes. The extensors are more profoundly involved than the flexors. There are *pain* and *tenderness* in the muscles and over the nerve-trunks. The electrical reaction of degeneration is present. Sensory phenomena are constant, such as zones of anesthesia and paresthesia over the affected parts. Slight dropsy may arise. In cases of the paralytic form that recover, convalescence is protracted.

2. **The Wet or Dropsical Form.**—The earlier or later development of general anasarca with effusion into the serous sacs, characterizes the wet form. The swelling may be enormous and obscure the wasting, which, however, is less marked than in the atrophic variety. The urine contains no albumin and the edema is firmer than that of nephritis. *Dyspnea*, *cardiac palpitation*, and *tachycardia* are commonly present.

3. **The Acute, Cardiac (Pernicious) Form.**—This serious type may develop acutely either as a primary affection or secondary to a mild form of the complaint. The predominating features are *cardiac palpitation*, *marked dyspnea*, and indications of progressive cardiac failure. A moderate *leukocytosis* is usually present; this was true of my cases. The urine may be scanty or suppressed, while the presence of indican in large amounts may be noted. The *duration* may be brief, not exceeding twenty-four hours, but oftener, perhaps, extending over several weeks.[1]

4. **The Mild or Rudimentary Form.**—The initial symptoms may be *catarrhal* in nature, to which are soon added the characteristic features—*pain*, *weakness in the legs*, *paresthesia*, *cardiac palpitation*, and possibly *malleolar edema*. Mild cases may be the forerunners of the types previously described, including acute pernicious beri-beri. The disease is often associated with malaria, the result of a blood examination in 4 cases in my care having shown the plasmodium in 3, or 75 per cent.

Diagnosis.—This offers no practical difficulty except in sporadic cases, in which the circumstances under which they arise (*e. g.*, the country or region from which the patient may have come), are unknown. The epidemic form is easily recognized. The grouping of the symptoms of peripheral neuritis with edema, absence of deep reflexes, and threatening cardiac dilatation, leave little room for doubt in any case.

Differential Diagnosis.—Other forms of infectious polyneuritis are distinguished by the absence of the peculiar endemic or epidemic status, the visceral symptoms, the edema, and of the transudation in the serous sacs. In *alcoholic neuritis* the peculiar history and such characteristics as the

[1] "Beriberi, with Report of Cases," *The Medical Bulletin*, by the writer.

prevalence of painful features and trembling are noted; in *diphtheritic multiple neuritis* the *velum palati* is involved.

Course and Prognosis.—The course is interrupted by periods of aggravation and apparent pauses, and on leaving the beri-beric centres, all symptoms may disappear. The *prognosis* is mainly dependent on the intensity of the infection, the presence or absence of associated diseases and the circumstances of the individual patient. The particular variety present in the case in hand influences greatly the outlook, *e. g.*, the cardiac or pernicious form being highly threatening to life. Again, the anatomic seat of the nerves implicated decidedly affects the prognosis.

The *mortality* differs with the seasons, locality and individual epidemics. In Japan the death-rate is only 12.5 per cent., while among the Chinese and Brazilians it is much higher.

Treatment.—1. **Prophylaxis.**—It is most probably a fact that under certain compulsory conditions—individual susceptibility, overcrowding, and a warm, moist climate—the usual hygienic measures will not prevent the outbreak and spreading of this disease. Under these circumstances, removal to a non-contaminated locality alone suffices. The foregoing facts were strikingly confirmed during the recent Russo-Japanese War, when the rigid execution of ordinary sanitary means succeeded in limiting serious outbreaks of typhoid, typhus, scorbutus, and dysentery in the Japanese army, while beri-beri proved a veritable scourge (*vide supra*). Removal of early cases to special hospitals, followed by rigorous disinfection of the houses and rooms in which they have been should be systematically carried out during an epidemic season.

Certain *hygienic measures*, such as systematic feeding with easily digestible proteins, exposure to fresh air without undue fatigue, should be advised. A change to a milder and drier climate is usually efficacious, if practicable. In severe cases rest must be enjoined.

2. **Medicinal Treatment.**—Various methods have been advised, such as the early free use of the salicylates (Baelz), venesection, and free purgation. While all of these are useful in suitable cases, there is not one that is applicable in every instance. The product known as "oryzanin" is thought to possess specific potency by certain Japanese investigators. In cases in which serious cardiac dilatation supervenes, venesection for its immediate effect is often effective in saving life. Many of the most distressing symptoms in acute forms (dyspnea, pain, nausea,) are benefitted by the use of morphin hypodermically. The dropsy of the cardiac cases requires rest and saline laxatives, followed by digitalis (♏v–x, of the tinct. every third hour). For the so-called cardiac seizures, nitroglycerin or inhalations of the nitrite of amyl are recommended. The atrophied muscles should be treated with electricity and massage, and strychnin with tonics is indicated for the same condition.

MALTA FEVER.

(*Mediterranean Fever; Rock Fever; Undulant Fever.*)

Definition.—A protracted infectious disease, caused by the *micrococcus melitensis*, and characterized clinically by irregular fever, copious sweats, rheumatoid pains, and frequent relapses.

History.—Malta fever was described clinically by Burnett in 1816 as a type of remittent malarial fever, but it was first depicted as a specific disease by Marston in 1859. It is *endemic* in Malta, and from time to time is encountered there, and at other Mediterranean ports, in *epidemic* form. Owing to observations made by Wright on the serum reaction, this disease has been shown to exist in India, Hong Kong, the United States, the West Indies, and Brazil. Kinyoun first suspected the presence of Malta fever on this side of the Atlantic, along the coast and in the islands of the Gulf of Mexico. Musser and Sailer[1] recognized the affection in Philadelphia in a soldier who had come from Porto Rico. No essential *pathologic lesions* have been identified with the disease. Hughes[2] noted an enlargement of the spleen and of the mesenteric glands, and in grave cases, bronchitis and broncho-pneumonia.

Etiology.—**Bacteriology.**—The *micrococcus melitensis* (Bruce) has been found in certain tissues (the spleen in all fatal cases), and is readily recognized morphologically and by culture. Bruce, in 2 cases, and Hughes, in 4, reproduced the disease in monkeys by the inoculation of pure cultures of the organism. Antihygienic conditions increase morbidity. There is no special liability according to age. Goat-herders are markedly predisposed.

Modes of Infection.—(*a*) By the "absorption of urine secreted by cases of Mediterranean fever, and this is one way in which workers in hospitals become infected" (Horrocks). (*b*) It is extremely probable that human beings are infected by the bites of infected mosquitoes—*culex pipiens*, *stegomyia fasciata*. (*c*) By the absorption of infected goats' milk from the alimentary canal. Gentry and Ferenbaugh have obtained the positive serum test in 34 per cent. of the goats examined.

The **incubation-period** lasts from a few days to twenty or thirty.

Symptoms.—The disease is of *slow* and *gradual* development, and the features simulate those of beginning typhoid fever. Headache, boneache, anorexia, malaise, and slight fever (often preceded by shiverings); the face may be congested, and epistaxis may be present. The bowels are constipated, and the stools may be blood-streaked. The spleen is always enlarged and frequently painful, particularly on pressure.

Three classes of cases are recognized: (1) A *pernicious* type which is rare and generally fatal (Hughes) and needs no further description here; (2) an *undulant* type, characterized by exacerbations of temperature at pretty regular intervals; (3) a *continued* type, in which a continuous fever persists for weeks and even months. The *fever* is of a remittent type, with undulating course, and perspirations lasting one, two, or three weeks; this, after an apyrexial period of two or three days, is followed by a relapse, with rigors, high fever, delirium, and increased prostration.

The *relapse* frequently lasts from five to six weeks, and then, after a week or two, a second relapse may ensue; symptoms somewhat similar to the first—rigors, intermittent form of fever, extreme prostration, and general rheumatoid symptoms. The latter may be so well marked as to prohibit muscular movements of any kind. The case now either terminates in recovery, or, after the lapse of one or even two months, there may be a repetition of the whole symptom-complex. In *grave cases* the temperature is continuous, and death may occur in hyperpyrexia (Hughes). The temperature range is often markedly irregular, hence its comparative

[1] *Phila. Med. Jour.*, Dec. 31, 1898. [2] *Annales de l'Institut Pasteur.*

uselessness, as claimed by Craig, from a diagnostic point of view. A polynuclear leukocytosis is present in Malta fever. Certain *complications*, as touches of pleurisy and pneumonia, rarely appear.

Diagnosis.—From the use of pure cultures of the special organism in the blood of Malta fever patients gives a typical agglutination. According to Negre, this test is only reliable for the *micrococcus melitensis* when the serum is heated to 56° C. for half an hour. Thus the affection is with ease and certainty distinguished from *typhoid fever* and erratic forms of *malaria*. In no other manner can it be discriminated from typhoid fever in the earlier stages. The presence or absence of the Widal reaction will assist in the differentiation. If *malaria* is suspected, the blood should be examined microscopically. Many cases present hacking cough and physical signs of lung congestion, or even consolidation, and, as a consequence, are confounded with *incipient tuberculosis*. The serum test will remove all doubt. Malta fever not infrequently, in its mode of onset and the symptoms present during the first few days, resembles *lobar pneumonia* (Craig). The absence of rusty sputum, stabbing chest pains, and the milder character of the cases, however, are an aid in excluding pneumonia. The polyarthritis with fever has led to confusion with *acute articular rheumatism*. *Pyemia* must also be excluded.

Duration and Prognosis.—Soldiers show an average stay in the hospital of ninety days (Bruce); obstinate cases, however, may last six months. Most cases pursue a chronic course. The death-rate is low—about 2 per cent. Death is generally due to hyperpyrexia.

Treatment.—This should be sustentative or supportive, in view of the uncertain, protracted course. Nourishing liquids and, usually, stimulants are required. Dalton allows solids, such as eggs, rice, and bread, in addition to 2 to 3 quarts of milk. The bowels should be moved daily. Fever is to be combated by the application of cold (cold bath, wet pack, or sponging). Methylene-blue (gr. $\frac{3}{4}$—0.05, two or three times daily) is considered the best remedy available (Audibert and Rouslacroix). Bassett-Smith[1] reports two series of cases treated with vaccine prepared from cultures of *Micrococcus melitensis*, freshly isolated from the spleen during life, with gratifying results. Tonics, coupled with a change of climate, favor convalescence. Hematinics are especially indicated during this period to overcome the well-marked secondary anemia.

PROBABLE INFECTIOUS DISEASES.

MUSCULAR RHEUMATISM.

(*Myalgia.*)

Definition.—A common, painful disease of the muscles and of the structures to which they are attached (fasciæ and periosteum), probably due to an attenuated form of the virus of acute articular rheumatism. Leube contends—and very properly, I think—that muscular rheumatism is a general disease with local symptoms. The latter may be seated in different parts of the body, and in this way give rise to a number of leading sub-varieties, and it may either accompany acute and chronic

[1] *Journal of Tropical Medicine and Hygiene*, May 15, 1907.

rheumatism or it may be experienced as an independent disease. I have also met with several instances in which it followed joint-rheumatism, and Leube has seen it precede the latter. Certain authors believe that the affection is a neuralgia of the sensory nerves of the muscles.

Pathology.—In fatal cases (these are exceedingly rare) the affected muscles show a swelling of the fibers and more or less granular change. In long-standing cases there is an atrophy of the muscles, due to trophic disturbance. Strauss describes circumscribed nodules in the muscles.

The changes are essentially those of myositis. In the acute form there is often an extensive round-cell infiltration of the connective tissue, with swelling and partial degeneration of the muscular fibers and the formation in them of vacuoles. In the chronic form there is a proliferation of the interfascicular connective tissue.

Etiology.—Among the disposing influences the most important are —(1) The *rheumatic diathesis* (appropriate soil); (2) *Heredity;* (3) *Exposure* to cold, damp, and strong air-currents, especially after heavy exercise or during free perspiration; (4) *Sex*, owing to the more frequent exposure of men while following their occupations; (5) *Age*. It is met with at all ages, but acute and subacute forms most frequently occur among children and young adults, while the chronic form generally affects elderly persons; (6) *Previous attacks* increase the susceptibility to the disease. (7) Lumbago may be reflex in character, due to hemorrhoids, enlarged prostate, and intestinal irritation.

Symptoms.—In the majority of instances the clinical symptoms are local. Out of 200 cases Leube found *fever* in about one-third, the temperature rarely exceeding 102° F. (38.8° C.) for two days in duration. In one-sixth of Leube's cases there was a cardiac murmur that disappeared under treatment in one-half of this number. *Pain*, which which is sometimes sharp, lancinating, and paroxysmal, while in other cases deeply seated, dull, and constant, is troublesome. It is aggravated at night by contraction of the affected muscles, by weather-changes, and by pressure. In long-continued cases pressure with the broad side of the hand usually affords relief. The *duration* ranges from a few hours to several days or longer. The rheumatic nodules are common in the shoulder- and calf-muscles. The cases in which the symptoms tend to persist or recur with changes in the weather may be termed *chronic*.

Leading Clinical Varieties.—(1) **Lumbago** (*Myalgia Lumbalis*).—This is the most common form, and may be taken as the type of the myalgias. The *onset* is sudden, sometimes intensely so, and the lumbar muscles are exceedingly painful and sensitive. *Motion*, such as stooping or turning the body or rising from the sitting position, causes intense exacerbations of pain. The affection occurs most frequently in laboring-men, its course being brief, as a rule, and recurrences frequent. Erben, from a study of 200 cases of lumbago, finds that the trouble is principally an affection of the lumbar vertebræ, or a neuralgia of the cutaneous nerves.

(2) **Pleurodynia.**—This term implies involvement of the intercostal muscles, and less frequently of the pectorals and the serratus magnus. It is unilateral, and oftener affects the left than the right side, and causes untold suffering, since it is constantly aggravated by the normal respiratory excursions. The pain is also intensified by pressure, reaching, etc., and by movement of the trunk, sneezing, and coughing. Similar symptoms may be occasioned by *traumatism* in which the fibers

of the thoracic muscles are lacerated, and there is also great danger of confounding pleurodynia with *costal periostitis* and with *pleurisy*.

(3) **Torticollis** (*Myalgia Cervicalis*).—Here the muscles, some or all, on one side of the neck, and at times the throat, are implicated. The head is held toward the affected side, so as to relax the group of muscles involved, and on attempting to turn it the patient rotates his entire body in a pivot-like manner. The complaint is frequent in young persons.

(4) **Cephalodynia.**—By this term is meant rheumatism of the head-muscles of the scalp and fasciæ. It may be either *general* or *local*, being sometimes limited to the frontal, temporal, or occipital muscles. The *pain* is severe and greatly increased on motion of the scalp.

(5) Other terms descriptive of localized forms of muscular rheumatism are employed: (*a*) *Omodynia* (myalgia of the deltoid); (*b*) *Dorsodynia* (involvement of the muscles of the upper part of the back, etc.); (*c*) *Abdominal rheumatism* (myalgia of the muscles of the abdomen); (*d*) *Rheumatic myositis* of the extremities.

Diagnosis.—This is assured by the etiologic influences and the presence of pain, which is greatly increased by muscular contraction. The presence of fever does not exclude the affection. It differs from *neuralgia* in that there are no painful points, and in that firm pressure with the broad hand often affords relief. On the other hand, in *gonorrheal rheumatism* the plantar fasciæ are commonly involved and the patient complains of pain in the head. *Dermato-myositis* must not be confounded with muscular rheumatism. Unverricht first distinguished the former from the latter, showing that there are present pain and swelling of the muscles, as in muscular rheumatism, but additionally redness (erythema) and hyperesthesia of the skin, while the joints usually escape. Of general symptoms, the chief are fever and physical prostration. The spleen is enlarged, and angina and hemorrhages have been noted. The disease is obviously infectious, probably septic in nature, and may rarely prove fatal. Dermatomyositis, unlike muscular rheumatism, is more common among women, especially servants, than men. *Abdominal rheumatism* has been mistaken for appendicitis.

The **prognosis** is good, the disease never endangering life, though a person may be incapacitated for work by muscular rheumatism.

Treatment.—Severe and acute forms demand the use of opiates internally and anodyne and hot applications externally. When cases are seen early, morphin, administered hypodermically, may serve to relieve the pain and cut short the disease. In acute cases the salicylates and other antirheumatic remedies are to be employed. Hot fomentations give comfort, and the Turkish bath may end the attack if it can be used sufficiently early. The hot-water bag, sponging with water as hot as can be borne, or dry heat in the form of bags filled with heated salt or heated hops, will all do good service. For the dull pain which is so distressing in some cases of torticollis the affected muscles may be covered with flannel, over which a warmed flatiron may be passed for a few minutes. This is an efficient expedient. For lumbago acupuncture is highly commended. Needles of from three to four inches (7.5–10 cm.) in length (ordinary bonnet-needles, sterilized, will do) are thrust into the lumbar muscles at the seat of the pain and withdrawn after five or ten minutes (Osler). Schmidt recommends local injection of 5 or 10 c.c.

of physiologic salt solution for the relief of pain. Blisters have been recommended, but I have tried them without beneficial effects. In subacute and obstinate cases I have recently obtained good results from the use of a 20 per cent. ointment of salicylic acid freely rubbed into the skin. Active friction with anodyne and stimulating liniments (the latter when pain is not great) is worthy of trial. Massage and electricity (constant current) are sometimes efficient, and in chronic cases potassium iodid, guaiacum, and arsenic (the latter in small doses) should be tried. The same measures of prophylaxis are to be adopted as in chronic rheumatism. The general health must also be looked to, every endeavor being made to maintain the proper quality of blood and perfect nutrition.

CHRONIC ARTICULAR RHEUMATISM.

Definition.—An affection of the articular structures which develops slowly and gradully and may have the same etiology as the preceding forms. Rarely it is a sequence of acute or subacute attacks.

Pathology.—The joints, as a rule, do not show pronounced gross lesions, there being some degree of synovial injection and also some, though not much, effusion. Inflammatory thickening of the articular and periarticular structures (capsule, ligaments, sheaths of the tendons, etc.) with contraction, is noted, and is a change which deforms and stiffens some joints to a certain extent. Superficial erosions of the cartilages may also be witnessed, and muscular atrophy supervenes. The probable causes of these important changes have been pointed out in connection with the latter disease. When the shoulder-joint is the seat of chronic inflammation, this muscular atrophy (affecting chiefly the deltoid) reaches its highest degree of development.

Etiology.—(*a*) *Age* predisposes to the affection. Though it may appear at any age, the greatest number of cases is furnished by the years from forty to sixty. (*b*) *Sex* exerts a slight influence, the disease being observed most frequently among females. (*c*) *External agencies*, as poverty and occupations which entail exposure to cold and dampness. (*d*) *Heredity* may operate to favor its development.

Symptoms.—The involved joints may not present any visible evidences of disease, and perhaps the most prominent local symptom is *pain*, increased often at night as well as by approaching cold or damp weather. Both the larger and smaller joints are involved, though the former to a greater degree, and yet, though usually multiple, the disease may be limited to one joint (knee, hip, shoulder, etc.). The joints are somewhat *swollen*, as a rule, at times slightly reddened, tender upon pressure, and their mobility is generally restricted. Pain and *stiffness* are most marked in the morning hours (after rest), and often largely disappear with each returning evening (after use). All the local symptoms are subject to exacerbations and remissions. A peculiar *crepitation* may be elicited on applying the hand over the affected joints during motion, and eventually ankylosis, with some degree (usually slight) of distortion of the joints, may occur.

The *general features* are usually conspicuous by their absence. No fever is present, and, in most instances, there is no serious impairment of the general health. On the other hand, as the result of constant suffering, a wretched general condition with marked anemia and debility

may finally be reached, such patients often passing sleepless nights and suffering severely from dyspepsia. Chronic endocarditis may develop along with the chronic articular changes—a not uncommon association, though frequently the history of a previous attack of acute rheumatism is also obtainable, to which the endocarditis may be attributed (for the differential diagnosis of this disease *vide* Arthritis Deformans).

Prognosis.—Full recovery is, with but few exceptions, out of the question. A cure may rarely be effected if the case come under appropriate treatment in the incipient stage. The disease, however, rarely shortens the duration of life, though it may do so by interfering with the nutritive processes, the latter effect resulting from loss of sleep (due to pain) and inability to take active exercise.

Treatment.—(*a*) **The local measures** hold first place. The affected joints should be enveloped in flannel at all times, and underneath the latter may be applied cold cloths, and the whole covered with oiled silk. On the other hand, sponging the joints frequently with hot water relieves the pain and stiffness. Bier, Reed, and others, employed a hot-air treatment with good effects. Blisters are efficacious in removing effusions. In the absence of synovial effusion the thermo-cautery is to be preferred to blisters, and for the swelling and stiffness massage with passive movement affords excellent results. Massage is also valuable when atrophy of the adjacent muscles exists; and in these so-called "rheumatic paralyses" electricity is an important help. Iodin and stimulating liniments are more or less serviceable.

(*b*) **Hygienic Measures.**—The diet should be abundant and nourishing; it may embrace milk, eggs, the lighter forms of meat, fats, farinaceous articles, and cruciferous vegetables; wines and alcohol may be permitted. Dietetic abuses, however, tend to aggravate the arthritic condition. The patient should adopt and continue moderately active exercise until compelled to omit it on account of the advancing joint-lesions. Cold spongings of the skin-surface, followed by active friction, have a good effect in that they lessen cutaneous sensitiveness.

(*c*) **Internal remedies** do not control the morbid process directly, although arsenic, iodin, potassium iodid, guaiacol, and other agents are much used for this purpose, but their effects are usually limited, and never brilliant. It should be our aim to maintain the general health at a maximum level by the employment not only of the sanitary means before alluded to, but also by tonics (iron, quinin, strychnin, etc.). A course of cod-liver oil is the most serviceable form of internal medication.

(*d*) **Hydrotherapy** is an important adjuvant to the treatment.

The thermal springs whose waters are alkaline or contain sulphur, and of which the hot springs of Arkansas and Virginia, and the Richfield Springs, New York, furnish good examples, have been strongly advocated, and sometimes prove curative in their effects. I have seen excellent results from the methodic use of hot-water baths at a constant temperature (100° to 105° F.—37.7° to 40.5° C.), combined with passive motion and careful manipulation of the affected parts. Every precaution must be used to avoid exposure to cold or draft during and after the baths, which should not be prolonged beyond ten minutes.

Mountain Fever.

(*Mountain Sickness*).

The term "mountain fever" should be regarded as applicable only to a condition produced by the action of a rarefied air upon the organic functions. There is no definite *pathology*. Aron's investigations show that the intake of oxygen is diminished at high altitude.

The **symptoms** are a much-quickened pulse, urgent dyspnea, headache, vertigo, and at times nausea and vomiting. There is a subfebrile movement, the temperature touching 100° F. (37.7° C.) or even 101° F. (38.3° C.). Thirst is present and the appetite is lost. Malaise and a sense of exhaustion on attempting exertion are experienced. Hemoptysis has been noted, but rarely. The effect upon the human economy of high altitude varies with the extent of the differences in individual reserve nerve-force. Rest and acclimatization will almost invariably restore healthy function. Oxygen inhalations are advised (Aron).

The "mountain fever" of the older writers is almost universally conceded at the present day to be typhoid fever modified by the effects of extreme altitude.

Rocky Mountain Spotted Fever.

Historic Note.—This disease has been known in the valley of the Bitter Root River, in Western Montana, during the past twenty years. Rock Creek and Bonito, nearly twenty miles distant from Bitter Root Valley, have furnished a limited number of cases.

Mountain spotted fever has also appeared in the valleys of streams situated in the mountainous sections of Northwestern Nevada, Southern and Western Idaho, and in Northern Wyoming.

Predisposing Causes.—*Climate.*—Mountain spotted fever has not been observed south of 40° or north of 47° N. lat., and epidemics are most prevalent at elevations ranging from 3000 to 4000 feet.

Season.—The disease prevails exclusively during spring and early summer months.

Occupation, Age, and Sex.—Persons who are compelled to be in the open air and among the woodlands and farming districts are most likely to become infected.

In Anderson's[1] analysis of 121 reported cases, 76 were males and 45 females. Most of the cases occurred between the fifteenth and fiftieth years of age.

Parasitic Origin.—A series of investigations, conducted by Anderson, showed that the Pyroplasma hominis, a parasite closely allied to the Pyrosoma bigeminum (known to cause Texas fever in cattle) and found within the body of the red blood-corpuscles, is the infecting parasite. He further believes that the disease is transmitted to man through the bite of ticks (Dermacentor reticulatus) common to infected districts.

Incubation.—The period of incubation varies from three to ten days, seven days being the rule. During three or four days of this period the patient experiences slight chilly sensations, malaise, and nausea.

Clinical History.—The disease is ushered in by a distinct chill, which is followed by a rapid and continuous elevation in the temperature,

[1] *Hygienic Laboratory Bulletin*, No. 14. "Public Health and Marine-Hospital Service of the United States," p. 8.

with slight morning remissions, until the tenth to the twelfth day, and in fatal cases it reaches 104° to 106° F. In favorable cases the temperature reaches the maximum from the eighth to the tenth day, after which there is a gradual decline to the normal by the fourteenth day.

Following the chill the patient experiences pain in the back and loins, soreness of the muscles, the limbs are moved with difficulty, and there is always slight, and at times severe, nose-bleed after the first week of fever; the tongue is heavily coated at the center and base, while its edges and tip are red; nausea and vomiting are common, and persistent constipation is the rule. The conjunctivæ are at first congested and later assume a yellowish tinge; the urine is febrile in character, being diminished in quantity and containing a moderate amount of albumin and also renal casts; the respirations range from 25 to 60 per minute, and it is not uncommon for the patient to develop bronchitis from the third to the sixth day of the fever; the liver and spleen are enlarged. The pulse is weak and rapid, being out of proportion to the temperature. The mind is, as a rule, clear even in the severer forms.

Eruption.—The eruption is rather characteristic; it appears on the third to the fourth day, on the wrists and ankles, from which points it spreads to the arms, legs, forehead, back, chest, and, lastly, to the abdomen.

The spots are at first bright-red maculæ, varying from the size of a pin's point to that of a pea. In the severer forms of the disease these maculæ become dark and later assume a purplish tinge. They begin to fade at about the sixth day, and lose their petechial character with the decline of the fever at or about the fourteenth day.

Blood.—During the course of the disease the red blood-cells show evidence of destruction, and the white cells may be slightly increased in number. The hemoglobin may gradually fall to 50 per cent.

Diagnosis.—Mountain spotted fever is to be differentiated from *cerebro-spinal meningitis*, *peliosis rheumatica*, *malaria*, *typhoid fever*, *mountain fever*, and *purpura hemorrhagica*. The eruption is similar to that of *typhus fever*, but the spleen is less habitually enlarged, and it runs a shorter course with a more abrupt onset and termination of the fever.

Prognosis.—Of 121 cases occurring in the Bitter Root Valley district, 84 were fatal (Anderson). In other districts the rate of mortality may reach 90 per cent. Death usually occurs between the fourteenth and eighteenth days of the disease, and may result from complications, among which pneumonia deserves special mention.

Treatment.—The treatment is ordered to meet the indications presented by each individul case. L. B. Wilson and Anderson have suggested the use of quinin hypodermically. Morphin, in the form of Dover's powders, is usually required to relieve the intense pain and soreness. Hot sponge-baths are of value in relieving the temperature. The *diet* should consist of milk, broths, soft-boiled eggs, and soft toast. H. T. Ricketts recommends the eradication of the tick in infected areas as a prophylactic measure. Heinemann and Moore found that serum from horses recovered from spotted fever has a protective value; it can be concentrated as in the case of diphtheritic antitoxin.

WEIL'S DISEASE.

(*Acute Febrile Jaundice; Fiedler's Disease.*)

Definition.—An acute febrile disease, probably specific in origin, and characterized by jaundice, remittent fever, and muscular pains. It usually runs a definite course and terminates by lysis.

Pathology.—During the comparatively recent studies of the post-mortem lesions occurring in this disease very little has been noted. The liver and spleen are sometimes the seat of an active hyperemia, and occasionally some gastro-intestinal irritation is present. The cortical substance of the kidneys is swollen and mottled, and the epithelium of the tubules and glomeruli shows cloudy swelling.

Etiology.—The special organism of the disease is unknown; indeed, it may be an acute febrile jaundice of varied etiology. Jaeger claims that it is due to infection by the *bacillus proteus fluorescens*. Cockayne believes that it may be due to some biting insect. Certain French authorities consider the disease a ptomain poisoning.

Predisposing Causes.—Among these may be mentioned the following:

(*a*) *Age.*—The age of the patient usually varies from twenty to forty years. A. Holz records a case in a woman fifty-one years old.

(*b*) *Occupation.*—*Butchers* are most commonly affected. Workers in ditches and sewers or those exposed to foul water are particularly prone to infection.

(*c*) *Sex and Season.*—Most of the recorded cases occurred in males and during the summer months.

(*d*) *Locality.*—The cases have appeared in groups, in both rural and urban localities.

Symptoms.—The disease is usually ushered in by a *chill*, followed by *fever*, *headache*, and *pain* in the muscles, joints, and epigastrium. *Jaundice* usually appears on the second day, and may either be slight or very intense; if it be due to obstruction, the stools are gray-colored, showing the absence of bile. The fever is of the remittent type, running from ten to fourteen days and terminating by lysis. Nausea, vomiting, and diarrhea may rarely occur. The *liver* and *spleen* are often enlarged, the latter being tender on pressure. The *urine* is febrile, high-colored, and often shows the presence of albumin, with tube-casts, and sometimes blood (hemoglobinuria). In grave (but rare) cases *cerebral symptoms*, such as delirium, convulsions, and coma, may occur and prove fatal.

The **diagnosis** rests on the acute onset, fever, pains in the muscles, joints, and epigastrium, nephritis, and icterus. *Schlammfieber*, which prevailed mainly among young persons who had worked in the recently flooded districts near Breslau during the summer of 1891, and assumed epidemic proportions, has not been satisfactorily classified. Müller shows its resemblance in many respects to Weil's disease, which may occur at times without jaundice (?).

Prognosis.—The prognosis, both as to life and recovery, is good. W. E. Hughes, notwithstanding, records two cases that proved fatal within forty-eight hours of the onset.

The **treatment** is purely symptomatic. The *diet* should be fluid, such as milk, broths, and the like. Hydrotherapy is indicated in the more toxic cases. The muscular pains may be relieved by warm stupes and fomentations.

FEBRICULA.

(*Simple Continued Fever; Ephemeral Fever.*)

Definition.—A brief febrile attack, unattended with definite local lesions, and of varied, often indeterminate etiology. A true ephemeral fever is one that lasts about twenty-four hours, while the term simple continued fever or febricula is given to cases lasting a longer period.

The cases are diversified with reference to their **etiology** and clinical relations, but may be roughly grouped under several heads:

(*a*) A large group of cases in which a *gastro-intestinal disturbance* is the only assignable cause. The latter may be due to cold or more often to errors in diet (particularly the use of tainted food-stuffs), accompanied by absorption of ptomaines, or it may assume the form of gastro-intestinal catarrh met with in young children.

(*b*) Undeveloped or abortive forms of the *infectious diseases* (typhoid, influenza, rheumatism). These affections, particularly during times of epidemic prevalence, may run a brief course without manifesting any of their distinctive characters. This is particularly true of the abortive types of typhoid, and other acute infections. Again, diseases that ordinarily manifest a characteristic eruption (*e. g.*, scarlet fever, measles, erysipelas) may run their course without doing so, or the eruption may escape observation.

(*c*) It may follow *exposure* to the summer sun or *excessive heat* (?), or *exhaustion of the nervous system.*

(*d*) It is not infrequently the result of a slight and unnoticed *localized inflammation* (tonsillitis, bronchitis, lymphadenitis, etc.).

(*e*) The *inhalation of sewer-gas or other noxious vapors* (such as emanations from decomposing organic matter) may produce an aberrant form of the fever (*vide* Septicemia).

Symptoms.—It is to be remembered at the outset that a single symptom, peculiar to all cases, is the *fever.* The *onset* is generally sudden, and especially in ephemeral fever, but it may be gradual; if sudden, there is rarely either a chill or vomiting, while in neurotic children a convulsion may occur. The temperature ascends quickly to 102°–103° F. (39.4° C.) or over, pursues the continued type, and at the end of one, two, or more days subsides abruptly by crisis. There are accompanying symptoms, many of which are due to the fever, such as headache, hebetude, mild delirium, flushed countenance, a full, rapid pulse, anorexia, constipation, scanty, high-colored urine, and, not rarely, herpes labialis. Defervescence may be attended with critical sweats, diarrhea, or a copious flow of urine. *Special types* (*e. g.*, cerebral, gastric, gastro-intestinal) may be observed, due to the predominance of the symptoms presented by individual organs or systems.

In another class of cases the access of simple fever may be less sudden, the maximum level attained being somewhat lower and the attending phenomena less acute and pronounced. Da Costa[1] has described cases belonging to this category. The *course* is more protracted, though

[1] *Transactions of the Association of American Physicians*, vol. xi., 1896.

rarely exceeding a week or ten days, and the defervescence is not so abrupt. So-called *thermic fever* is at the present writing believed by Guitéras, who first described it, to be due to a special, though as yet unknown, organism.

The **diagnosis** necessitates the exclusion of other acute fevers. The affections from which it is most difficult to distinguish febricula are *typhoid fever*, *remittent fever*, *scarlet fever*, *incipient tuberculosis*, *larval pneumonia*, and *meningitis* (in children). In febricula, however, there is an absence of local manifestations and of physical signs pointing to consolidation of the lungs; characteristic skin-eruptions are also absent. Tyson points out that in cases in which there is splenic enlargement (rare) the resemblance to typhoid is close, and the diagnosis may have to remain in doubt until settled by the Widal test or by time. The cases must also be discriminated from the fever which sometimes attends chlorosis and certain nervous disorders.

The **prognosis** is good.

Treatment.—Few cases require treatment other than rest in bed and liquid nourishment for several days. Cooling drafts internally, and mild forms of hydrotherapy (spongings, ice-caps) externally, are indicated. If traceable to gastro-intestinal disturbance, a laxative usually proves beneficial and effective. It should be followed by intestinal antiseptics. Unless it is clear that the given case is non-infectious and non-contagious, isolation of the patient should be ensured.

MILK-SICKNESS.

Definition.—A peculiar infectious disease, occurring both in man and in the lower animals, when it is known as "trembles." The disease is unknown east of the Alleghany Mountains, but throughout many of the Western and South-western States it formerly prevailed very extensively, with fatal effect. It has, however, been almost exterminated as the result of denudation of the forests and the advancing cultivation of the virgin soil. It still prevails in parts of North Carolina (Osler), and until very recent times has been seen in certain parts of Illinois.

No peculiar pathologic lesions have been described.

Etiology.—It is believed to be due to a special poison derived from the earth, but as yet we are ignorant of its exact nature. Phillips claims to have found a spirillum in the blood.

Modes of Infection.—The disease attacks cattle most frequently (especially unweaned calves), horses, sheep, goats, and less often many undomesticated animals; wherever trembles prevails among cattle, milk-sickness is met with in man. It is thought that the poison is communicated to man in the milk, butter, and cheese, or in the flesh of infected animals.

Among disposing factors are the *seasons*, the disease being most frequent in the late summer and autumn. It is most common in adult life.

Symptoms.—The period of *incubation* may be short or long in duration, and *prodromata*, such as headache, anorexia, languor, and oncoming fatigue, may be noted. These symptoms increase in severity, and are soon eclipsed by the more characteristic features—nausea and vomiting, a hot pain in the stomach, and a peculiar fetor of the breath. There is an unquenchable thirst, a swollen, tremulous tongue, and abso-

lute constipation. Fever is present, but it is slight, and the surface-temperature is often below the normal. The nervous symptoms include restlessness, merging into mental dulness with marked indifference, and the latter condition passing in grave cases into a stupor that may deepen into actual coma. Convulsions may arise or the patient may drop into a fatal typhoid state.

The **diagnosis** rests chiefly upon the history (particularly upon the coexistence of "trembles" in cattle) and the exclusion of other acute intoxications.

The **prognosis** is generally favorable, though a fatal termination due to asthenia may occur within a few days of the time of the onset.

Treatment.—*Prophylaxis* consists in the avoidance of those foods that act as bearers of the disease. Apart from the use of supporting measures (appropriate diet and stimulants), we can attend only to the symptomatic indications. Medicated enemata should not be omitted.

MILIARY FEVER.

(*Sweating Sickness.*)

Definition.—An infectious disease, characterized by copious sweats and a vesicular (miliary) eruption. In certain countries it has prevailed epidemically (France, England, Italy, Germany), and in 1887 a severe epidemic occurred in France. Schaffer[1] reports the occurrence of a recent epidemic in an Austrian province in the spring of 1893, lasting for nearly three months. Out of 5079 persons (the total population of the district), 159 suffered, as follows: 17 men, 14 women, and 128 children. At the present day it seems to be met with only in Picardy, in a few other French provinces, and throughout a limited area in Italy.

Neither have definite *pathologic lesions* nor the *specific exciting* cause been found. Among *predisposing influences* the following have been noted: (*a*) Most epidemics occur in spring and summer; (*b*) It is more common among women than men, and most frequent during the middle period of life. A large percentage of the entire population of an invaded district (usually limited in area) is attacked.

The **symptoms** that characterize miliary fever are *fever* with its usual accompaniments, irritation of the skin, epigastric oppression, copious and persistent sweating, followed, on the third or fourth day of the disease, by an *eruption* (due to profuse sweatings) of miliary vesicles. A. Weischelbaum[2] has shown by serial sections through sudaminæ that the fluid in the latter is not due to retained secretions in the sweat glands, but is always of an inflammatory nature.

The vesicles burst, and within forty-eight hours scaly desquamation is generally completed. In severe types the nervous phenomena (delirium, etc.) are grave in character; hemorrhages may occur, and at times fatal collapse may follow. *Relapses* are not uncommon.

The **prognosis** is affected largely by the character of the epidemic, the average death-rate being 8 or 9 per cent.

Quinin has met with almost universal favor as a remedy, but the expectant plan of **treatment** is the most appropriate, the indications being fulfilled as they arise. The sweating may demand atropin.

[1] *Wiener med. Blätter*, 1893, No. 32. [2] *Zeit. f. Klin. Med.*, 1907, lxii., 21.

FOOT-AND-MOUTH DISEASE.

(*Epidemic Stomatitis; Aphthous Fever.*)

Definition.—An acute infection of certain lower animals (cattle, sheep, pigs, goats), caused by a micro-organism as yet undiscovered, although Klein has described a micrococcus. It is characterized by fever, by the appearance of vesicles and ulcers in the mucosa of the mouth, in the furrows about the feet and on the udder, and by the rapid development of asthenia and marked emaciation. Though a disease of mild character, its territorial range is so vast as to entail untold loss to European countries. Young animals or sucklings perish in great numbers on account of the deteriorated quality of the milk, which assumes a yellowish-white appearance and has a bitter, nauseating taste.

During epidemics of foot-and-mouth disease the poison may be transferred to man, in whom the disease is known as *epidemic stomatitis*, the poison generally being transferred by means of milk. Boiling the latter destroys the virus, but rarely the infection may be transmitted through butter and cheese made from the milk of infected cattle. Communication by inoculation (while milking) may also occur. The disease does not seem to be transmissible through the meat of diseased animals.[1] In America a few instances only of transference from animals to man are recorded.

Symptoms.—The *incubation-period* lasts from three to five days. A rigor may mark the onset or merely slight shiverings, followed by fever and malaise, and soon vesicles, such as are described under Aphthous Stomatitis, appear upon the tongue and inner surface of the lips. The mouth is hot, the mucosa reddened and swollen, and salivation is present. A form of miliary eruption that may become pustular may also appear on the skin-surface, and particularly on the fingers and hands. Hemorrhages have been observed in severe epidemics.

The **diagnosis** is made with ease if the disease be prevailing at the same time among lower animals. The peculiar coincidence of the eruption in the mouth and extremities, sparing the rest of the body, has not been noticed in any other eruptive disease (Whittaker).

Course and Prognosis.—The course is mild and ends in about one week, the disease being very rarely fatal.

Treatment.—*Prophylaxis* requires the use of milk from healthy animals (cows or goats), together with measures looking to the care of the stables and isolation of diseased cattle. A reliable method of immunization against foot-and-mouth disease has not as yet been discovered.[2] For *treatment* the reader is referred to the article on Aphthous Stomatitis.

GLANDULAR FEVER.

Definition.—By this term is meant an acute infectious disease of children, characterized by adenitis affecting the lymph-glands of the neck, especially the anterior cervical.

[1] Zuell's translation of Friedberger and Fröhner's *Pathology and Therapeutics of the Domestic Animals.*

[2] Siegel, "Experiments in Immunization against the Poison of Bites and Scratches." Quoted in the *Philadelphia Med. Jour.*, January 28, 1899.

History.—A detailed description of glandular fever was first given by E. Pfeiffer, in 1889, under the name of *Drüsenfieber*, but it had probably been previously described by Filatow, of Moscow. Donkin, Fischer and Dawson Williams, in England, and J. Park West have given excellent descriptions of the disease.

Pathology.—The anterior cervical lymphatic glands are involved first, and it is "probable that the infection finds its point of entrance through either the tonsils or the pharyngeal mucous membrane" (Williams). The adenitis may also affect the inguinal and axillary glands.

Etiology.—The special micro-organism of the disease is unknown, although Burns has isolated the staphylococcus aureus. The complaint occurs usually in the form of house-epidemics. West, of Ohio, however, has described the most widespread epidemic hitherto recorded. There were 96 cases in 43 families, and rarely did a child exposed to the infection escape. The disease usually occurs during childhood; the ages of West's cases ranging from seven months to thirteen years. A. E. Roussell has reported four cases, one occurring in an adult. Most cases occur between the months of October and May, inclusive. According to Hand, the weight of clinical evidence tends to variation in the etiology in different cases (*e. g.*, it is often one of the protean manifestations of influenza).

The incubation-period lasts usually from five to eight days.

Symptoms.—The *onset* is *sudden*. The child holds the neck stiffly, since movement causes pain; there are anorexia, nausea, and less commonly vomiting, the bowels are constipated, and often there is abdominal pain. The child may complain of pain and swelling; an examination of the pharynx may show some chronic enlargement of the tonsils, and in some cases injection of the pharyngeal mucosa, actual pharyngitis being rare. The temperature oscillates from 101° to 103° F. (38.3°–39.4° C.). Nervous symptoms (delirium, hebetude) are rarely observed.

The *glandular enlargement* becomes obvious on the second or third day, and in most cases is observed first on the left side, then, after a few days, on the other side of the neck also. The glands vary in size from a bean to a hen's egg, and are painful on palpation. They rarely suppurate. Other groups of glands (axillary, inguinal) may be successively involved. Cough and dyspnea may point to involvement of the bronchial and tracheal glands. The mesenteric glands were enlarged in 38.5 per cent. of West's cases. Splenic enlargement occurs in 50 per cent. of the cases, while the liver is increased in size in almost all cases. There is a leukocytosis varying from 18,000 to 25,000. The average *duration* is sixteen days (West). Among *complications* may be mentioned hemorrhagic nephritis, bronchitis, and otitis media.

Diagnosis.—The recognition of glandular fever embraces the exclusion of such affections as *tonsillitis*, *pharyngitis*, and *influenza*, in the course of which adenitis might arise. Griffith[1] has reported cases resembling glandular fever in which influenza was probably the sole disease present.

Prognosis.—Recovery is the rule.

Treatment.—The course of the disease is probably uninfluenced by treatment. Locally, cold compresses and fomentations are useful. Internally, West advises castor-oil in the early stage, followed by minute doses of calomel (gr. $\frac{1}{12}$ to $\frac{1}{10}$) twice or thrice a day.

[1] *Univ. Med. Magazine*, October, 1900.

PART II.

ANIMAL PARASITIC DISEASES.

PARASITES OF MAN.

The human species furnishes a habitat for many varieties of parasites. Protozoa, including the Amebas and Infusoria, Plathelminthes, Nematodes, Leeches, Arachnoids, and Insects. Some infest the body surface, while others find their locus in the intestines, bone marrow, vascular system, muscles, brain, genital apparatus, or solid viscera.

AMEBIC DYSENTERY.

(*Amebiasis.*)

Definition.—A colitis, usually chronic, though it may be acute, caused by the *Entamœba dysenteriæ*, often leading to abscesses of the liver.

Etiology.—This disease is caused by the *amœba dysenteriæ* (Councilman and Lafleur) or the entamœba hystolytica. The amœba dysenteriæ is a unicellular, motile organism, in size 3 to 7 times the diameter of a red blood-corpuscle (15 to 30 micromillimeters). Its protoplasm consists of two zones—an outer colorless (ectosarc) and an inner granular zone (endosarc), with a visible nucleus and one or more vacuoles. This micro-organism was first described by Lambl (1859), but it remained for Lösch, and especially Kartulis, to show its close association with dysentery. The amœba dysenteriæ is occasionally found in healthy individuals, and also in other bowel affections than dysentery (mucous enteritis, simple diarrhea, proctitis due to engorgement), and two species are recognized —a virulent entamœba hystolytica and a benign form, entamœba coli. Walker's studies, however, indicate not less than ten species. The ameba is found not only in the discharges, but also in the pus from the secondary liver-abscesses. Flexner[1] affirms that bacterial association probably has much influence on the pathogenic powers of the amebæ. The principal causative *rôle* in the production of this form of dysentery has been ascribed to the pyogenic cocci by Tancarol, Ascher, and others. The disease is much more prevalent in adult males.

The **mode of transference** of the ameba is not definitely known, though the principal source of the dysenteric germs is most probably the drinking-water. The disease is feebly communicable by contact.

[1] *Jour. Amer. Med. Assoc.*, Jan. 5, 1901.

Pathology.—The lesions are almost always situated in the large intestine, although rarely the ileum is also invaded. The first visible change is a hyperemia of the mucosa, most marked in the descending colon and rectum; but the changes which produce the characteristic dysenteric ulcer begin with infiltration and swelling of the submucosa, followed by necrosis, which involves the overlying mucosa with its epithelium (Kruse and Pasquale). How the amebæ reach the submucosa has not yet been observed. The infiltrated circumscribed areas are oval or hemispheric in shape, and project above the level of the surrounding mucosa. The submucosa presents a grayish-yellow appearance, and is soon thrown off in the form of a slough.

The ulcers take various shapes—chiefly irregular, and less frequently round or oval. Their edges are ragged and undermined, and the floor, which is more or less covered with pultaceous material, is rough or crater-like, and formed by the musculature or the outer serous coat of the intestine. From the manner in which the ulcers are formed it is obvious that cellular infiltration (followed by necrosis) may occupy the submucosa for a greater or less distance beyond the borders of the ulcers. In this way fistulous channels may be produced beneath the mucosa and connect two or more ulcers. Usually this ulcerative process affects only certain portions of the large gut, especially the flexures—hepatic and sigmoid—and the rectum; but it may be general, and I have seen an instance of this kind. Cases are not uncommon in which the ulcers are so numerous as to include almost the entire mucosa of the colon.

Healing is attended with the development of fibrous tissue along the edges and in the base of the ulcer, and secondary contraction of this new connective tissue is often productive of colonic stricture, which is usually either partial or irregular. The cases that come to autopsy often show diphtheritic inflammation as a secondary or terminal condition.

The *microscope* reveals proliferation of the fixed connective-tissue cells (rarely pus), and the presence of amebæ in the walls and the base of the ulcers, in the lymph-spaces, and rarely in the blood-vessels.

The liver may be the seat of prominent lesions. These are (*a*) *abscesses*, which may be single or multiple. The single or large solitary abscess is usually situated near either the upper convex or the lower concave surface, while the abscess-cavity is formed in a manner similar to the intestinal ulcers. The area affected is at first infiltrated; it then becomes necrotic, and finally more or less liquefied. Upon the full development of the first stage the part invaded is a grayish-yellow pultaceous mass, but in the second or necrotic stage the abscess contains a yellowish or greenish-yellow, spongy material with beginning liquefaction. The contents of the mature abscess consist of a greenish- or reddish-yellow purulent material and of remnants of liver-tissue. The walls of the recent abscess are irregular and ragged, those of an old abscess being dense and fibrous, and a section of the abscess-wall shows an inner necrotic zone, a middle zone (in which there is great proliferation of the connective-tissue cells, compression and atrophy of the liver-cells), and an outer zone of intense hyperemia (Osler). The contents of the abscess show either few or many amebæ, and only rarely pus. When pus is present it is due to a secondary infection by the

pyogenic germs. The amebæ probably gain access to the liver by metastasis from the intestinal foci. Cultures are generally sterile.

(*b*) The parenchyma of the liver may be the seat of circumscribed necrotic spots, due to the action of the toxins formed by the amebæ.

The lungs sometimes show changes similar to those in the liver; they are the result of direct extension of the hepatic abscess through the diaphragm into the lower lobe of the right lung. The kidneys often present the lesions of acute parenchymatous nephritis (Craig).

Clinical History.—The mode of *onset* is *gradual* except in a small proportion of the cases, in which it is sudden with well-marked symptoms. When, as generally happens, it is insidious, the initial symptom is often a trivial diarrhea. The affection is then characterized principally by *intermissions* and an exacerbating *diarrhea*, the liquid stools containing necrotic tissue of a grayish-brown and sometimes yellowish-gray color. The latter are often bloody and mucoid, later becoming fluid. The *number* of discharges per day is exceedingly variable, although in most instances they range from six to eight or ten daily.

Microscopic examination of the feces during the exacerbations discloses amebæ that are almost invariably endowed with motion, though usually not when the stools have become formed. Tenesmus is not a prominent feature in most cases, and may be entirely absent. Colicky abdominal pains, nausea, and vomiting are rare.

General Symptoms.—Fever is usually present, but it is slight and exhibits marked variations. In certain instances, however, the temperature is below the normal. From the time of onset there is gradual, progressive loss of flesh and strength, and anemia becomes well marked.

Complications.—The most common is *hepatic abscess*, and as the result of perforation of the diaphragm may arise secondary abscess of the right lung. Authors are not agreed as to the frequency of occurrence of liver-abscess (see p. 922) in amebic dysentery, but it is certainly comparatively rare in this country, not exceeding, perhaps, 3 per cent. of the cases. In the tropics it occurs in 20–25 per cent. of the cases. *Peritonitis* may result from perforation of a dysenteric ulcer, causing death. The point of perforation may, however, be in the rectum, when *periproctitis* is the result; or it may be in the cecum, when *perityphlitis* is the sequel. In tropical or subtropical countries the disease is often complicated with malaria. The presence of an intermittent fever is not, however, sufficient to warrant the assumption that malaria complicates dysentery; we must be able to demonstrate the presence in the blood of the *plasmodium malariæ*. In pyemia and in suppurative processes generally—conditions sometimes met with in dysentery—the temperature-curve is often distinctly intermittent. *Typhoid fever* is a rare complication. The *typhoid state* is met with, and pyemia and septico-pyemia may appear late. Pylephlebitis, pericarditis, endocarditis, pleuritis, nephritis (common), and rheumatoid pains in the joints are observed.

Diagnosis.—The slow course, marked by intermissions and exacerbations of the bloody, fluid stools, the mild general symptoms, apart from emaciation and debility, are salient features, but an assured recognition of the affection demands a microscopic examination of the stools. Cases have been recorded by Councilman and Lafleur in which the diagnosis rested upon amebæ being found in the sputa; this was explained by the existence of an hepato-pulmonary abscess, which had

discharged through a bronchus; the intestinal symptoms were negative. Similar cases have been reported by L. Napoleon Boston[1] and others.

Prognosis.—The mortality-rate in certain epidemics has been frightful, particularly among soldiers in the field (amounting to 70 per cent.). In sporadic cases the danger to life is less, the mortality-rate in temperate climates being not over 5 or 6 per cent. The complications which render the prognosis unfavorable are,—peritonitis, hepatic and pulmonary abscess, pyemia, broncho-pneumonia, malaria; death may be due to hemorrhage or peritonitis, but in a preponderating proportion of the cases to asthenia. A dangerous degree of debility is indicated by great nervous depression; a cool, clammy skin; a sunken, pinched facies; a dry tongue; a feeble, rapid pulse; and by low muttering delirium.

Course and Duration.—The average duration ranges from eight to ten weeks in uncomplicated cases; the disease can, however, be cut short by appropriate treatment. It manifests an innate tendency to pursue a chronic course, interrupted by frequent exacerbations or true relapses, and convalescence occupies a long period of time in consequence of the profound anemia and debility that supervene.

Treatment.—The diet should consist of easily digestible and nutritious solids, as raw oysters, eggs, rice, fowl, fish, and the like, in small quantities. Milk should also be freely allowed. It may be necessary to restrict the diet to fluids if diarrhea be well marked. During convalescence a return to a mixed dietary is to be adopted in a gradual manner.

A judicious hygienic regimen calculated to maintain assimilation is especially valuable. Rest in bed, combined with gentle, systematic massage, may be necessary in severe cases; in other and lighter cases graduated exercise in the open air and rest are serviceable. The medical treatment is by ipecacuanha, in the form of salol-coated pills. Not less than 30 grains at a single dose are to be given on the first day. "Subsequently the amount is to be diminished by 5 grains *per diem*, so that by the sixth day only 5 grains of the drug are administered. During the next week or ten days a nightly dose of five grains must be allowed."[2] Rogers[3] recommends the subcutaneous injection of $\frac{1}{2}$-grain doses of emetin hydrochlorid. Beck advises the treatment of dysentery with ipecac through the Einhorn duodenal tube, especially in acute cases, as much as 1 to 2 drams being introduced at one sitting. Colonic injections of warm solutions of quinin hydrochlorid (strength 1 : 1000 to 1 : 5000) have proved effective in the hands of most clinicians. Leroy, of Memphis, has used formalin similarly (1 : 1000), with almost specific effects. Copper arsenite internally (gr. $\frac{1}{100}$–0.00067) and hot instillations of copper sulphate solution (1 : 10,000 to 1 : 6000) have been found valuable (Storck[4]). Musgrave[5] prefers thymol, which he gives by enema. A small class of cases do not yield to either the ipecacuanha treatment or rectal lavage; they demand "appendicostomy and systematic, thorough irrigations through the appendix." Recurrences will yield to the same means, and they can sometimes be prevented by promoting the repair of the blood and tissues during the intervals.

[1] *Jour. Amer. Med. Assoc.*, April 26, 1902.

[2] "The Treatment of Amebic Dysentery, Especially by Appendicostomy," by J. M. Anders and W. L. Rodman, *Jour. Amer. Med. Assoc.*, February 12, 1910.

[3] *Brit. Med. Jour.*, 1912, 2695, 405.

[4] *Jour. Amer. Med. Assoc.*, Jan. 6, 1912.

[5] *New Orleans Med. and Surg. Jour.*, May, 1911.

FLAGELLATA.

MASTIGOPHORA.

During the motile period of their existence these organisms possess one or more flagella attached to either or both ends in the various forms, and some of them also possess an undulating membrane, the trypanosomes being the best exemplars of this latter group. The body of these parasites is very small, many with rounded anterior portion, pointed posteriorly; others, spindle-shaped. They are nucleated, often have vacuoles, and some contain chlorophyl.

Trichomonas vaginalis lives in an acid medium. It is not found in normal vaginal secretion, but in vaginal catarrh with acid secretion; it may occur at any age from childhood to advanced life. It is a specific parasite of the female generative tract, though, rarely, it has been found in the urine of men, probably introduced through coitus. It is not known how they gain entrance to a woman. Alkaline solutions destroy them, as does cold.

Trichomonas intestinalis and *pulmonalis* are met with in stools, urine, sputum, secretions from mouth; but these forms are not pathogenic.

Lamblia (*Cercomonas*) *intestinalis*—a monad commonly met with in intestinal discharges; not believed to be pathogenic. Cercomonads have been found (*C. hominis*) in discharges of cholera patients and (*Bodo urinarius*) in urine.

Balantidium coli (*Paramœcium coli*) is found in discharges from obstinate cases of colitis; also in mucosa, and even submucosa, of rectum and colon. Stokvis has found it in the sputum of a case of pulmonary abscess. The pig is believed to be the source from which man is infected.

TRYPANOSOMIASIS.

SLEEPING SICKNESS.

The trypanosoma is a flagellated hematozoön common to the lower animals, and has been found in man. Trypanosoma hominis is a minute, colorless, translucent, active protozoön, tapering toward its extremities, the anterior of which displays a long flagellum. The body of the organism is finely granular. It is found free in the plasma. Trypanosomes have been known for over sixty years, but their pathologic import was first pointed out by Evans in surra, a disease of horses and cattle in India, *trypanosoma Evansii*. In May, 1901, Forde found the organism in the blood of an Englishman suffering from an irregular chronic fever, at first thought to be malaria. Six months later Dutton found and recognized the nature of the organism in the blood of this same patient, though about ten years before, Nepven, a French observer, had seen the same or a similar parasite in man, this being the first time that man was found to be subject to infection from trypanosomes. Dutton suggested the name *trypanosoma gambiense* for the parasite, and trypanosomiasis for the disease. Trypanosomiasis is engrossing a large proportion of the professional attention at the present day of Europe, Asia, Africa, Australia, and even America. Castellani[1] found the trypanosoma in

[1] *Lancet*, June 20, 1903.

the spinal fluid, obtained by lumbar puncture, in 20 out of 34 cases of sleeping sickness, but Bruce first showed the pathologic relationship between sleeping sickness and trypanosoma, and that the agent of transmission is the tsetse fly of the genus *Glossina palpalis*. This fly is not found in America.

Trypanosomes have been found associated with many diseases of man and animals, and Manson believes that three of these—*i. e.*, the *trypanosoma Brucei*, a tsetse-fly disease, causing nagana in horses and cattle; *trypanosoma Evansii*, and the trypanosome of mal de caderas—are closely allied species, if not identical. Recently Broeden discovered in the Congo country a trypanosome which is pathogenic for cattle, rats, guinea-pigs, and monkeys, and the infection is essentially like that produced by other trypanosomes.

Symptoms and Diagnosis.—Early the *skin* may appear nearly normal, but Ford and Manson have described blotches of erythema, isolated areas of edema, especially of the eyelids, and, later, general edema and cachexia. The *tongue* is red and raw. There are wasting, general malaise, and decided weakness of the lower limbs. The *pulse* may reach 120 beats per minute. Tachycardia and muscular weakness are the rule. Fever may develop at intervals or may occur daily for an indefinite time, ranging from 100° to 104° F. (37.7°–40° C.). Lethargy is the dominating feature of these cases. In addition to the cutaneous symptoms, which may resemble leprosy, there are restlessness, difficulty in speech, delirium, Cheyne-Stokes respiration, and coma. *Splenic enlargement* and tenderness were present in Ford's case. The lymphatic glands are enlarged and contain trypanosomes. An irregular remitting *fever* is a leading symptom of the first stage. There is general *anemia* of the simple chlorotic type. Ophthalmoscopic examination may show mottling of the fundus. The Liverpool School of Tropical Medicine maintains that gland palpation is the most efficient means of diagnosis of human trypanosomiasis, other causes for the glandular enlargement being absent.[1] The parasites (*trypanosoma hominis*) are numerous in the blood during the febrile periods. (For technique necessary for its recognition and staining, see Malaria.) *Sleeping sickness*, which is due to a lymphatic infiltraton of the brain with small mononuclear cells, probably constitutes the second or final stage of human trypanosomiasis. There is a marked rise of temperature in the evenings. There are mental dulness, headache, general weakness, and somnolence, merging into coma later.

Treatment.—Koch advises the sacrifice of every animal whose blood is found to contain the parasite. He has found arsenic to be a specific in the treatment. The methods adopted by Great Britain and Germany to prevent trypanosomiasis are: segregation, notification, and measures for dealing with animals serving as carriers. Dr. Daniels informs me that atoxyl in ascending doses has been found effective. Commencing with gr. j, every second day, the dose is increased to gr. iss at the end of one week, to gr. ij at the end of another week, then gr. iiss to gr. iij every second day for a year or more after all trypanosomes have disappeared. This treatment may be followed by a course of mercury. Excellent reports from the use of Ehrlich's remedy (arseno-phenyl-glycin) have been received, but is still on trial.

[1] J. L. Todd, *The Journal of Tropical Medicine and Hygiene*, October 15, 1908.

FEBRILE TROPICAL SPLENOMEGALY.

(*Dumdum Fever, Kala-azar, Piroplasmosis.*)

Definition.—It is a chronic disease, characterized by anemia, irregular fever, emaciation, pigmentation of the skin, enlarged spleen, and by a protozoön organism, which is found in the spleen, liver, bone-marrow, lymph-glands, adrenals, testicles, intestinal and cutaneous ulcers, and inflammatory exudates, and only very rarely in the blood.

Tropical splenomegaly, known also by its native Indian name, kala-azar (black fever), from the pigmentation of the skin, has also been termed Dumdum fever in Indian medical circles, after Dumdum, a military station near Calcutta. The disease is met with in India, Assam, China, Egypt, and Africa. Epidemics move forward very slowly—about 14 miles a year—the disease clinging to a place for almost five years and then disappearing.

Etiology.—In 1885 Cunningham, and in 1901, Firth, called attention to certain minute bodies to be found in the protoplasm of the cell exudate of the base of the Oriental sore or Delhi boil. In November, 1900, Leishman found these bodies in smears from the spleen of a case of Dumdum fever in a soldier invalided home from India. In the winter of 1902–3 Major Leishman noted these same organisms in smears of blood and internal organs from a trypanosome-infected rat. In May, 1903, he published these observations, and suggested that the organisms were trypanosomes. Soon after Donovan found them in fluid obtained by splenic puncture from an Indian, hence the name, Leishman-Donovan body. Low, Manson, Rogers, Bentley, and others have since found them. They are minute, oat-shaped, oval, or round bodies, with faintly staining protoplasm, but deeply staining chromatin masses, usually placed at opposite poles of the cell. Rogers succeeded in cultivating these bodies in citrate of soda solution, typical flagellated organisms resulting—the proof of their nature. Unlike the usual type of trypanosomes, the flagellum is attached to the end of the body at which the micronucleus is situated, and it does not possess an undulating membrane. It probably escapes from the body of the infected individual in the discharges from cutaneous or intestinal ulceration, and in all likelihood the intermediate host is some biting insect.

Predisposing Causes.—One-third of the cases occur under twenty years of age, and the Hindus were more frequently affected by the disease than the Mohammedans, the proportion being about 4 to 1 (Brahmachari).

The Oriental sore—Delhi or Bagdad boil, a local infection, without constitutional symptoms—is apparently due to the same trypanosome as tropical splenomegaly, but it is not fatal, and one attack, as a rule, gives immunity. Manson says it has been noted that Oriental sore is peculiar to camel-using countries, and if this really be due to the same Leishman-Donovan body as kala-azar, that a reduction in virulence of the organism has been attained by passage through the camel, as is the case with small-pox in its passage through the cow. The inference is, therefore, that the virus of Oriental sore should be employed in an attempt to protect against kala-azar. The disease prevails at all ages, in both sexes, and shows a predilection for the natives and old residents.

Symptoms.—There is fever, irregular in type, generally remittent, often comparatively long intermittent periods, the whole extending over some months. The fever may occur in ague-like attacks. A dirty, sallow, anemic appearance of the cutaneous surface is noted, and occasionally areas of pigmentation. Enlargement of the spleen and liver

occur early, the former being an invariable accompaniment, while the latter is less constant. Dyspnea, emaciation, progressive and, finally, extreme weakness, and more or less edema are present. Leucopenia, in which the proportion of white or red corpuscles may be less than one to one thousand with relative low polymorphonuclear counts, is almost diagnostic of the disease. Cutaneous and intestinal ulceration develop various hemorrhages or purpura, and these, in an extremely emaciated individual with a large protuberant belly, make a picture fairly characteristic. Death often results from some intercurrent affection. Among the commoner complications are pneumonia, pulmonary tuberculosis, abscesses, and splenalgia due to infarcts in the spleen.

Prognosis.—Manson regards the disease as absolutely hopeless; he has never seen a case recover. Rogers places the mortality at 96 per cent. Donovan gives an equally gloomy prognosis.

Treatment.—Tonic and hygienic. Quinin is of no special value, but may be employed with iron and arsenic. When possible, segregate infected cases, since no other known method of prevention exists, and, as we have seen, once developed, it proceeds to a fatal issue.

PSOROSPERMIASIS.

Psorosperms belong to the lowest form of protozoa. They are also known as *sporozoa*, and, because of their parasitic relation to cells, as *cytozoa*. A common form occurs in the muscles of the pig (*sarcocystis Miescheri*). The *amœba coli* of amebic dysentery belongs to the protozoa. Various coccidia may occur in man (*e. g.*, *sarcocystis hominis*) to produce the disease indicated by this heading.

(*a*) *Internal Psorospermiasis.*—In man, hepatic disease similar to that found in the rabbit is produced by the *coccidium oviforme*. The tumors formed by the coccidia may be palpable, and the liver may be quite tender. Some chilliness and fever, malaise, and stupor passing into coma have been observed. Death was caused on the fourteenth day in a case admitted to St. Thomas's Hospital (Osler). The necropsy showed whitish neoplasms in the peritoneum, omentum, kidneys, pericardium, liver, and spleen.

In the intestinal variety of *internal psorospermiasis* nausea and vomiting, diarrhea, and the typhoid state may be manifested. Involvement of the kidneys has caused hematuria and frequency of urination.

(*b*) *External Psorospermiasis.*—*Cutaneous psorospermiasis*, one form of which was formerly called *keratosis follicularis*, is characterized by lesions at first of a hard, crusty, papular type, later becoming confluent, and situated on the face, lumbo-abdominal, and inguinal regions. These papillomatous growths contain either parasitic sporozoa, or, as suggested by Montgomery and others, parasites that belong to the blastomyces.

In carcinoma, epithelioma, and Paget's disease of the nipple coccidia are readily found in and between the pathologic epithelial cells, but whether they have an etiologic bearing upon these malignant affections is still a matter of uncertainty.

Prophylaxis consists in cleanliness and care in preparing such food vegetables as spinach, lettuce, cabbage, and other greens that may possibly be contaminated by the excreta of the lower animals liable to psoroform-infection. The **treatment** of psorospermiasis is symptomatic, though rectal injections of a solution of quinin (1 : 5000 to 1 : 1000) may be tried.

MALARIAL FEVER.

(*Chills and Fever; Fever and Ague; Swamp Fever.*)

Definition.—An infectious, non-contagious disease caused by the hematozoön of Laveran. It is characterized by splenic enlargement, brief febrile attacks which recur periodically, melanemia, and a tendency in protracted cases to irregular fever and extreme anemia. The following sub-varieties will be discussed: (I.) *Intermittent fever;* (II.) *Pernicious intermittent;* (III.) *Remittent fever;* (IV.) *Malarial cachexia;* (V.) *Masked intermittents;* and (VI.) *Malarial hematuria.*

Historic Note.—There are few diseases with which the profession has been acquainted longer than with malaria, and chief among the earliest known hotbeds of this disease were the city of Rome, the Pontine marshes about the latter, and the swamps along the lower Danube. It is pretty generally believed that the prevalence of the disease long has been, and still is, diminishing. This view is fully corroborated by my own observations upon the cases from four leading hospitals of Philadelphia. The drying of marshy districts of a malarious character, thereby rendering them unsuited to the development of the mosquito, is the cause of this decreased prevalence.

New England, once a region in which the disease was very prevalent, now affords few cases. Again, in the southern portion of the United States, where the severer forms of malaria prevailed extensively in the past, a marked tendency to progressive reduction in the number of cases has also been observed. In foreign lands (England and Continental Europe) the constantly decreasing prevalence and virulence of this disease have been noted by numerous careful observers. The relation between malaria and the mosquito is suggested in the writings of such ancient authors as Columella and Varro. The peasants say "in such a place there is much fever because it is full of mosquitoes." Shepherds returning from the European mountains to their cabins smoked them to drive out the mosquitoes. The sheep occupied the cabins at periods when the famished mosquitoes inflicted their bites upon these animals, after which they showed little tendency to bite man (an ancient prophylaxis). Mbù is the term used in Eastern Africa for both malaria and the mosquito.

In 1848 Noth, of America, maintained that both yellow fever and malaria were transported by the mosquito, and King, in 1883, showed that malaria was transmitted in this manner. In 1891 Laveran recognized the mosquito as an intermediary host of this parasite. Similar views were held by Pfeiffer and Koch in 1892 and Bignami in 1894.

Pathology.—The chief and most constant morbid lesions are attributable to the direct effect of the malarial parasites upon the blood. The symptomatic anemia (often quite pronounced) results from the destruction of red corpuscles by the parasites. There is a marked tendency to an accumulation of pigment in the blood and in certain of the internal organs, particularly the spleen and liver. To account for this

DESCRIPTION OF PLATES III. AND IV.[1]

The drawings were made with the assistance of the camera lucida from specimens of fresh blood. A Winckel microscope, objective ⅟₁₂ (oil immersion), ocular 4, was used. Figures 4, 12, 22, 24, and 42 of Plate III. were drawn from fresh blood, without the camera lucida.

PLATE III.

The Parasite of Tertian Fever.

1.—Normal red corpuscle.
2, 3, 4.—Young hyaline forms. In 4, a corpuscle contains three distinct parasites.
5, 21.—Beginning of pigmentation. The parasite was observed to form a true ring by the confluence of two pseudopodia. During observation the body burst from the corpuscle, which became decolorized and disappeared from view. The parasite became almost immediately deformed and motionless, as shown in Fig. 21.
6, 7, 8.—Partly developed pigmented forms.
9.—Full-grown body.
10–14.—Segmenting bodies.
15.—Form simulating a segmenting body. The significance of these forms, several of which have been observed, was not clear to Drs. Thayer and Hewetson, who had never met with similar bodies in stained specimens so as to be able to study the structure of the individual segments.
16, 17.—Precocious segmentation.
18, 19, 20.—Large swollen and fragmenting extracellular bodies.
22.—Flagellate body.
23, 24.—Vacuolization.

The Parasite of Quartan Fever.

25.—Normal red corpuscle.
26.—Young hyaline form.
27–34.—Gradual development of the intracorpuscular bodies.
35.—Full-grown body. The substance of the red corpuscle is no more visible in the fresh specimen.
36–39.—Segmenting bodies.
40.—Large swollen extracellular form.
41.—Flagellate body.
42.—Vacuolization.

PLATE IV.

The Parasite of Æstivo-autumnal Fever.

1, 2.—Small refractive ring-like bodies.
3–6.—Larger disk-like and amœboid forms.
7.—Ring-like body with a few pigment-granules in a hyaline, shrunken corpuscle.
8, 9, 10, 12.—Similar pigmented bodies.
11.—Amœboid body with pigment.
13.—Body with a central clump of pigment in a corpuscle, showing a retraction of the hemoglobin-containing substance about the parasite.
14–20.—Larger bodies with central pigment clumps or bloebs.
21–24.—Segmenting bodies from the spleen. Figs. 21–23 represent one body where the entire process of segmentation was observed. The segments, eighteen in number, were accurately counted before separation, as in Fig. 22. The sudden separation of the segments, occurring as though some retaining membrane were ruptured, was observed.
25–33.—Crescents and ovoid bodies. Figs. 30 and 31 represent one body, which was seen to extricate slowly, and later to withdraw, two rounded protrusions.
34, 35.—Round bodies.
36.—"Gemmation," fragmentation.
37.—Vacuolization of a crescent.
38–40.—Flagellation. The figures represent one organism. The blood was taken from the ear at 4.15 p. m.; at 4.17 the body was as represented in Fig. 38. At 4.27 the flagella appeared; at 4.32 two of the flagella had already broken away from the mother body.
41–45.—Phagocytosis. Traced with the camera lucida.

[1] These illustrations are reproduced by permission from the article by Drs. Thayer and Hewetson in *The Johns Hopkins Hospital Reports*, vol. v., 1895.

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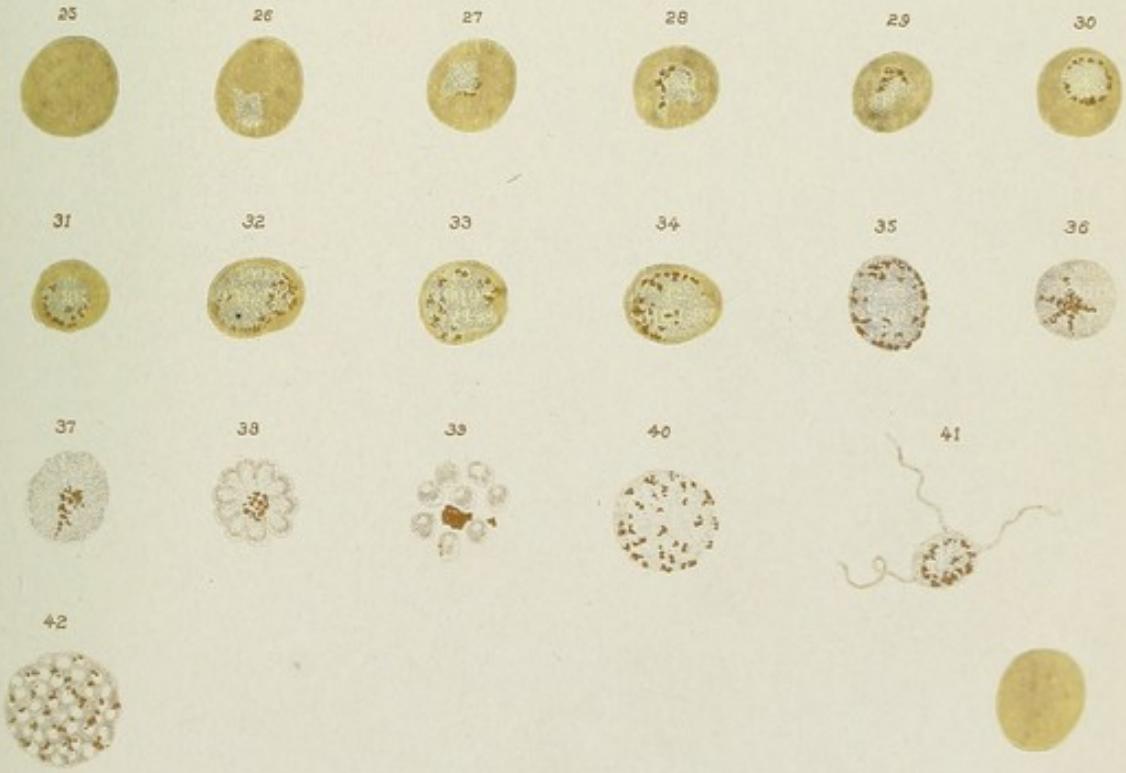
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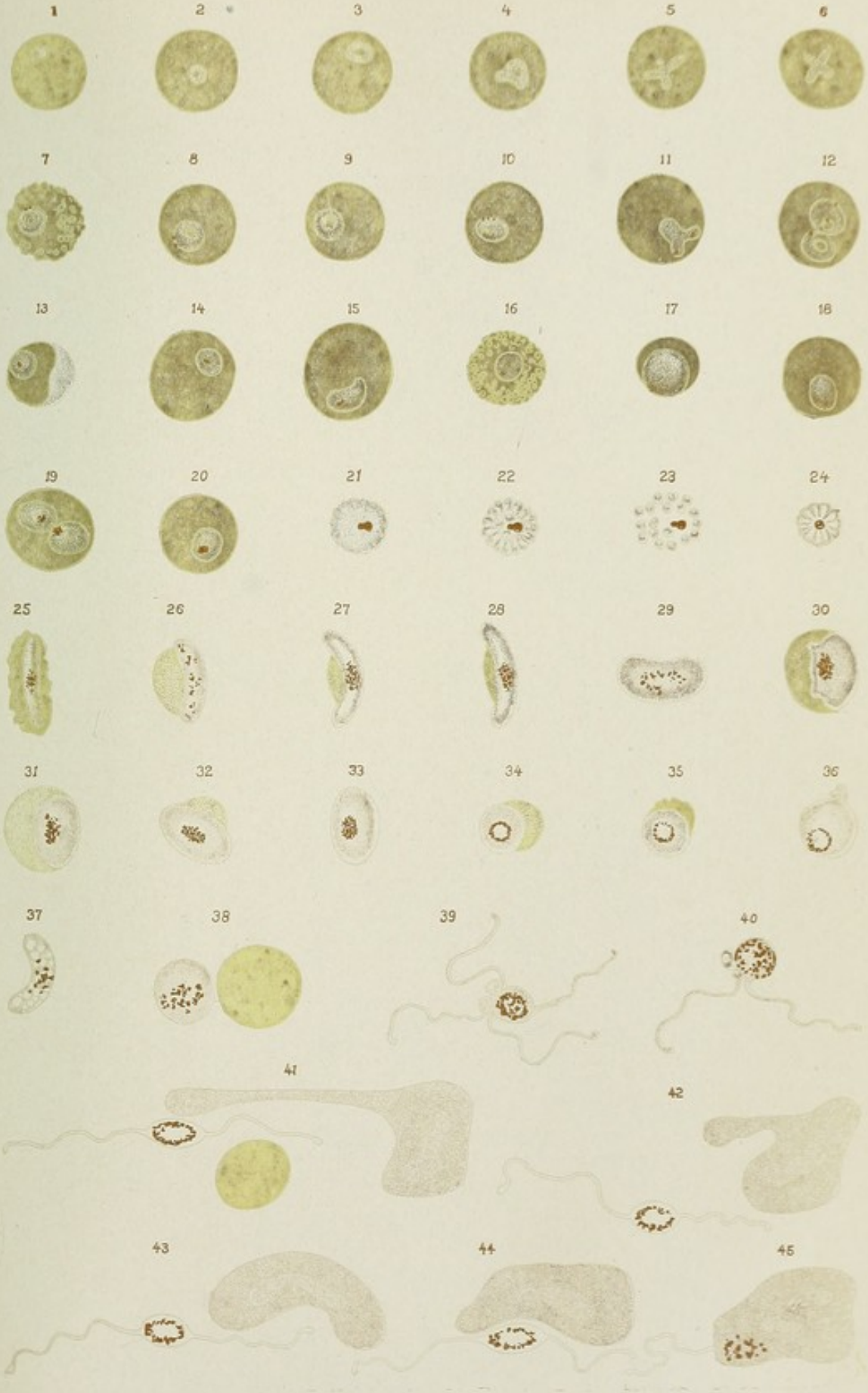
The Parasite of Tertian Fever.



The Parasite of Quartan Fever.



The Parasite of Aestivo Autumnal Fever.



is the fact mentioned in the description of the amebæ (*infra*) that the hemoglobin of the blood is converted into melanin (pigment) by the organisms. The phenomena of the disease are, at least in part, referable to the toxic action of this pigment. The malarial parasite also engenders a *toxin* which may be in part responsible for the morbid lesions of the disease.

The spleen is engorged with blood, and at first is swollen (chiefly during the febrile paroxysm), but it soon becomes permanently enlarged ("ague-cake"). A rare accident in intermittent fever is rupture of the spleen. Hemorrhagic infarcts are occasionally presented by this organ.

The liver is also engorged, but not to the same extent as the spleen.

The heart-chambers may be found to be acutely dilated. Neuritis has been observed by Gowers, Bamstark, Ewald, and V. P. Gibney. W. G. Spiller[1] reported a case that showed partial sclerosis of the motor tracts, and recent hemorrhages within the left internal capsule (*vide* Pernicious Malarial Intermittent, also Remittent Malarial Fever).

Etiology.—Parasitology.—Our knowledge of the malarial parasite may be discussed under five heads: (1) Discovery of the Plasmodium malariæ in the blood of persons suffering from the disease. (2) Its developmental cycle in man (sporulation), as shown by Golgi, in 1885. (3) The discovery, by MacCallum, of its method of sexual fertilization. (4) Its fertilization and development in an intermediate host (the mosquito), as first pointed out by Surgeon-Major Ross. (5) The observations of the Italian school, showing its method of re-entering the tissues of man.

(1) In 1880 Laveran discovered the malarial parasite, but it was not until 1883, when Marchiafava, Celli, and Golgi published their confirmatory investigations, that the parasitic origin of the disease was accepted.

(2) Laveran and Golgi observed that certain parasites, especially those found in estivo-autumnal fever, developed into peculiar crescentic bodies (gametocytes). Other more rounded, ring-like bodies were seen to display abnormal agitation in from ten to fifteen minutes after being withdrawn from the body, followed by the discharge from the protoplasm of several filamentous bodies or flagella (microgametocytes). The latter were seen to separate from the parent-cell, after which they were observed swimming independently in the blood.

(3) The significance of both the crescentic and flagellated bodies was first described by W. G. MacCallum, of the United States, in 1897. While studying the life-history of the "halteridium," it was noted that a limited number of ellipsoid bodies were produced, corresponding to the crescentic bodies seen in human malaria. Certain of these bodies were hyaline, others granular, and it was the former variety only that developed flagella. A flagellum, on swimming away from the mother-cell, was seen to display peculiar agitation on approaching one of the granular bodies (crescentic). One of the flagella was seen to enter the granular body and effect a symbiosis with it (*sexual fertilization*). Fertilization was followed by a short rest, after which the granular body assumed a worm-like form, and then swam slowly away, its pointed end directed foremost and trailing behind it pigment particles, which had been situ-

[1] *Amer. Jour. Med. Sci.*, Dec., 1900.

ated within its protoplasm. Later MacCallum was able to confirm these investigations by a microscopic study of the blood from a case of malaria (estivo-autumnal) in man, the flagella being formed after exposure to the air. In human malaria sexual fertilization takes place in the mosquito's stomach or middle intestine within the first twelve hours.

(4) Manson correctly supposed that the mosquito sucked blood from malarial subjects. Surgeon-Major Ross,[1] of Liverpool, began his investigations in India, in 1895, by endeavoring to determine what became of the parasite after fertilization in the mosquito's stomach. During August and September, 1897, two members of the species *Anopheles maculipennis*, bred from the larvæ, were fed on the blood of patients containing crescents, and he found that peculiar spheroidal cells developed on the walls of their stomachs, which convinced him that "these cells constituted the long-sought mosquito stage of the parasite" (*zygote*).

In 1898 Ross studied the "zygotes of protozoma" of birds. He found that they attached themselves to the outer coats of the mosquito's stomach. The zygote grows rapidly, without movement or change in form, protruding into the insect's body-cavity. Later its capsule becomes easily perceptible and the cell-substance is seen to divide into from ten to twelve "meres." In from one to three weeks, depending on the external temperature, the zygote matures, when each mere contains a number of delicate, "thread-like" blasts.

The next step in the development of the parasite is the rupture of its capsule, setting free these "thread-like" blasts within the insect's body; they are then carried by the blood's currents to all its tissues, more particularly into the insect's salivary gland. The common duct of the salivary gland of the mosquito (genus Anopheles) passes along the middle stylet of the proboscis, opening at its extremity, and a portion of the secretion of this gland is poured into the wound caused by the insect's bite. In the human body the blasts return to the amebulæ, with which the life-history of this parasite began.

(5) In 1898 Grassi found three chief species of the mosquito in malarious localities, the Anopheles claviger being constantly present. Manson gives 32 members of the family Anophelinæ, which have been shown, with more or less precision, to be hosts of the malarial parasites. Banks has shown conclusively that *Myzomyia ludlowi* should be added to the list.

In Nov., 1898, Bastianelli, Bignami,[2] and Grassi conducted a series of experiments, by feeding mosquitoes the blood from persons suffering from estivo-autumnal fever, confirming the findings described by Ross, in Aug., 1897. These investigations showed the *mode of infection;* that healthy mosquitoes become infected by sucking blood from malarial patients, and that in from eight to twenty-one days such insects may infect healthy men by their bites. One mosquito may infect many persons, and may possess this power for an indefinite period, "since not all of the germinal threads escape from the venomosalivary gland."[3] Neither the common house mosquito (genus Culex) nor the Anopheles nigripines takes part as an intermediary host for this parasite. The fact that mos-

[1] *Brit. Med. Jour.*, Dec. 18, 1897.
[2] "Malaria and Mosquitoes," *Lancet*, Jan. 13, 1900.
[3] *Jour. Amer. Med. Assoc.*, Feb. 3, 1900, A. Woldert.

quitoes (Anopheles claviger) are known to occupy non-malarious districts proves the innocence of the uninfected insects. A single case of malaria transported to such territory often results in an epidemic.

The *malarial parasite* of Laveran belongs to a subclass of the protozoa known as hematozoa (*hæmameba*). Of the latter, three varieties, corresponding with the three leading clinical forms of the affection, have been distinguished in man, and the evolution of two of these parasites at least takes place within the red blood-corpuscles. Manson describes five species which he classifies as follows:

Benign { Tertian, Quartan } do not form crescents.

Malignant { Quotidian—pigmented, Quotidian—unpigmented, Tertian } form crescents.

They enter the red cells in the form of small, non-pigmented plasmodia, exhibiting ameboid motion, and then feed upon their host, transforming, at the same time, the hemoglobin of the latter into dark pigment-granules as they develop. The special varieties of the malarial parasite as observed in microscopic studies of the blood of human beings will be described separately.

(1) *The Hæmameba Causing Tertian Intermittent Fever.*—This begins its asexual cycle of evolution in the red blood-corpuscles as a small hyaline ameba. Its development is attended with the appearance in its interior of fine, brown, motile granules in the form of pigment, and when matured it about equals the size of a normal red corpuscle. It now assumes a spheric form, the pigment collecting centrally, and sporulation into fifteen to twenty or more segments follows. The tertian parasites are exceedingly numerous in the blood, and pass through the various stages of their life-cycle almost simultaneously, the sporulation of an entire generation occurring within the space of a few hours (Golgi). The occurrence of the malarial paroxysm follows the process of sporulation, which is attended, most probably, with the development of a *toxin*, and the symptoms of the disease may be attributable chiefly to the effects of the latter. The red corpuscle that includes the parasite becomes enlarged and decolorized as the latter develops. The parasite of tertian intermittent runs its cycle in about forty-eight hours. Hence infection by a single generation would result in sporulation every second day, followed by the malarial paroxysm. Quite commonly, infection by two groups of parasites occurs on successive days, and, since each has a definite period of evolution, a daily malarial paroxysm is the result (quotidian intermittent). Multiple infection with this parasite may occur, but with great rarity.

(2) *The Hæmameba Causing Quartan Fever.*—This cannot be distinguished from the tertian parasite at the beginning of its asexual career, but later differences are clearly perceptible. Its ameboid movements are more deliberate, and its pigment-granules are coarser, darker, and also less motile than those of the tertian organisms. Unlike the latter, it does not attain the size of the red corpuscles, and during sporulation the segments (five to ten in number) encircle in an orderly way the central pigment-mass or clump, "rosettes" of great beauty thus being formed.

The red blood-corpuscle that harbors the quartan parasite contracts upon its destroyer, appears shrivelled, and its color changes at the same time from the normal to a deep greenish or bronzed tint. It sporulates about seventy-two hours after it enters the red corpuscle; hence, if only one group of parasites be present, febrile attacks occur every fourth day —quartan intermittent. On the other hand, double quartan infection results in paroxysms on two successive days, followed by an intermission lasting one day, while triple infection, or the presence of three groups, causes daily paroxysms—the quotidian intermittent. Infection by more than three groups of the quartan parasite may occur, but is very rare.

(3) *The Hæmameba Causing Estivo-autumnal Fevers.*—The endogenous cycle of this variety is evolved, chiefly, in certain of the internal viscera, and the microscopic examination of the blood in the various stages of the disease does not always give a positive result, as in benign tertian and quartan. The organism invades the red blood-corpuscle, but to what extent is questionable. It is a quite small hyaline body, its size at maturity scarcely equalling one-half the dimensions of the red corpuscle, and it accumulates a few fine pigment-granules. The parasite may be found in the later stages in the blood from certain internal viscera, as the spleen. After the condition has lasted a time characteristic oval and crescentic bodies, which are more or less refractive, may be observed in the fresh blood. These so-called "sickle-form bodies" show central rods and clumps of coarse pigment, and are connected with the malignant type of malarial fevers. Ring-form bodies, and, at times, the signet-ring forms, are observed. The red corpuscle, at whose expense the parasite develops, assumes a brassy-green hue, becoming shrivelled and crenated.

It would appear from the studies of Manson, Marchiafava, Bignami, and Surgeon Craig[1] that two varieties of parasite are concerned—quotidian and tertian forms of autumnal fevers (*vide* table, p. 345).

The parasites of tertian estivo-autumnal fever are larger than the quotidian parasite, and during the hyaline stage the signet-ring form, sluggish ameboid movement, clear-cut refractive outline, and the occurrence of one organism in a blood-cell which is not wrinkled are observed; during the pigmented stage, the ameboid movement and fine granular motile pigment. Segmentation takes place outside the corpuscle. Crescents are large, slender, and deeply pigmented (see Plate IV., p. 346).

The quotidian parasite is smaller, at times actively ameboid, and more than one parasite may occupy a single red cell, which is usually wrinkled. Their pigment is motionless, and usually in the form of short rods. Unpigmented parasites also occur (Manson). Crescents are small, plump, and often present a double outline. Segmentation occurs within the red corpuscle.

Development of Flagella.—Some of the crescents become ovoid with scattered pigment; this in turn becomes more or less spherical, the pigment forming a central ring; "this finally approaches the periphery, the whole parasite becomes violently agitated, throwing out flagella, which have a wave-like motion, many of which break away" (Wright).

Predisposing Causes.—(1) **Soil.**—Fresh-water marshes favor the development of malaria, and are most fruitful in influencing its growth

[1] *New York Med. Jour.*, Dec. 23, 1899.

when located near the coast and tainted with salt water. Again, marshy districts affording luxuriant vegetation are notorious as malarial foci. Keeping in remembrance the foregoing facts, we can readily see why malaria is unusually prevalent in certain countries (chiefly tropical), and why it is chiefly confined to the low-lying estuaries and the deltas of rivers. The same facts explain satisfactorily why certain districts which were very liable to the affection should have become, as the result of denudation of the virgin soil and its subsequent drainage and cultivation, entirely free from the complaint. Epidemics following the upturning or the removal of the surface of the virgin soil are probably due to importation of the disease (or infected mosquitoes), and are common on the frontier of the South and West.

(2) **Climate.**—Malaria is more prevalent in tropical and subtropical than in temperate climates, and more common in the latter than in the polar zones. Hence it occurs more frequently in the southern than in the northern States of our own country.

(3) **Rapidly growing trees** dry the soil by absorbing enormous quantities of water. In the Roman Campagna extensive experiments have been made with the eucalyptus tree, and districts protected in this manner becoming almost entirely free from malaria in a few years, the environment being unsuited to the mosquito.

(4) **Seasons.**—In temperate latitudes most cases are developed in the autumn, the maximal period corresponding with the month of September. This dictum is based upon 4841 cases of malaria gathered by the author from the records of the leading Philadelphia hospitals.[1] Cases that develop before the "Anopheles claviger" makes its appearance (in June) are possibly relapses. In the tropics the case seems to be different, and two maximum periods—spring and autumn—obtain. Statistics from the hospitals of Rome, collected from 1864 to 1898, show the maximum number of cases to occur in August, September, October, November, and July, respectively, and in June the minimum number.

(5) Persons occupying the upper stories of a house or living on elevations are affected with relative infrequency, for the reason that mosquitoes are always found near the earth's surface, where the air-currents are feeble. This fact also explains nocturnal infection.

(6) **Race** exerts little influence, but in the United States negroes are slightly less susceptible than are the whites.

(7) **Sex** is without effect when men and women are equally exposed. Cases are, however, vastly more frequent among males because of their increased liability to mosquito bites while following certain occupations (agriculture, marsh-draining). The 5044 cases collected by me gave the numerical proportion of 6 to 1 in favor of males.

(8) **Age.**—Children are more susceptible than adults.

(9) The disease may flare up after either an accident or surgical operation.

Immunity.—There are individuals immune from malaria and experimental malaria. An individual may present this property after a mild fever has been cured by quinin. Maurel has shown that when living in a malarious district whites may in time show marked immunity. By the use of methylene-blue and euchinin an immunity may be established against the inoculation of from 1 to 2 grams of estivo-autumnal blood.

[1] *Univ. Med. Mag.*, May, 1897.

Incubation.—According to Bignami and Bastianelli, the period of incubation for experimental malaria is: Quartan, 15 days; spring tertian, 12 days; estivo-autumnal tertian, 5 days. The administration of potassium bromid, potassium iodid, arsenic, carbolic acid, antipyrin, and phenocoll may result in a longer period. Angelo Celli has seen spring tertian show incubation of 22, and the estivo-autumnal tertian 17 days.

Epidemiology.—Estivo-autumnal fevers are rare in their recurrence, while mild tertian and quartan prevail with each new spring, and the first cases of tertian are noted to occur in the same houses in which the last recurrences of these fevers appeared. After the first cases there is a lapse of from seventeen to eighteen days, after which the epidemic spreads. The life and habits of the Anopheles have a direct bearing upon epidemics—"either the first cases of these fevers in July are recurrences of a previous infection, or the very first cases of these fevers in July are primary" (Celli). "Both hypotheses are possible. In both we have to deal with a contagion circulating, so to speak, between the temporary host (man) and the definitive host (mosquito), a contagion which, by means of the blood of the relapsing cases of the preceding year, is transmitted by the agency of mosquitoes, and starts the epidemic of the following year." There are many interesting questions not yet explained.

(I.) **Intermittent Fever.**—**Symptoms.**—The clinical history presents itself under two heads: (*a*) the paroxysms, and (*b*) the manner in which the paroxysms recur.

(*a*) *The Paroxysms.*—There may be premonitions lasting from one to several days, and most significant, yet not distinctive, are headache, pain in the nape of the neck, yawning, a yellowish complexion, and a slight splenic enlargement. In a large proportion of the cases, however, the onset is abrupt. Typical paroxysms present three stages—*chill*, *fever*, and *sweating*. The chill is intense, causing shivering, and often chattering of the teeth. Malaise is marked, the skin is cool and pale, face slightly cyanotic, and limbs painful. This stage usually occurs in the morning hours, but the time of onset is not constant; its duration, also, varies greatly, generally lasting from one to two hours. The internal temperature rises rapidly; the pulse is small, rapid, and of high tension.

The *hot stage* succeeds the chill, and, in striking contrast with the first stage, the face wears a decided flush and the skin is burning hot to the touch. The temperature continues to rise, but not so rapidly as in the first stage. Its maximum level, usually from 104° to 106° F. (40° to 41° C.), is soon reached, and may either be maintained uniformly for several hours, or the curve may show two small summits if the temperature be recorded frequently (Fig. 26). The pulse is full and bounding, except in the rare instances in which acute dilatation of the heart ensues, when it is quite feeble and sometimes irregular. The length of the second stage is from three to six hours. The temperature generally begins to decline before the close of the febrile stage.

When *sweating*, which soon becomes profuse, sets in, the symptoms of the hot stage are promptly relieved. The temperature falls by crisis, touching the normal level in a few hours; the decline, however, is less

rapid than the rise at the beginning of the paroxysm. The fall may be unbroken by any fresh elevations of temperature, though more often the latter occur. Less frequently defervescence occurs by steps, the temperature falling one or more degrees, and remaining at the new level for a short period; then dropping again about an equal distance, and so on until the normal is reached. Usually, following the paroxysm, the temperature becomes subnormal (about 97° F.—36° C.). The length of the typical malarial paroxysm ranges from eight to twelve hours.

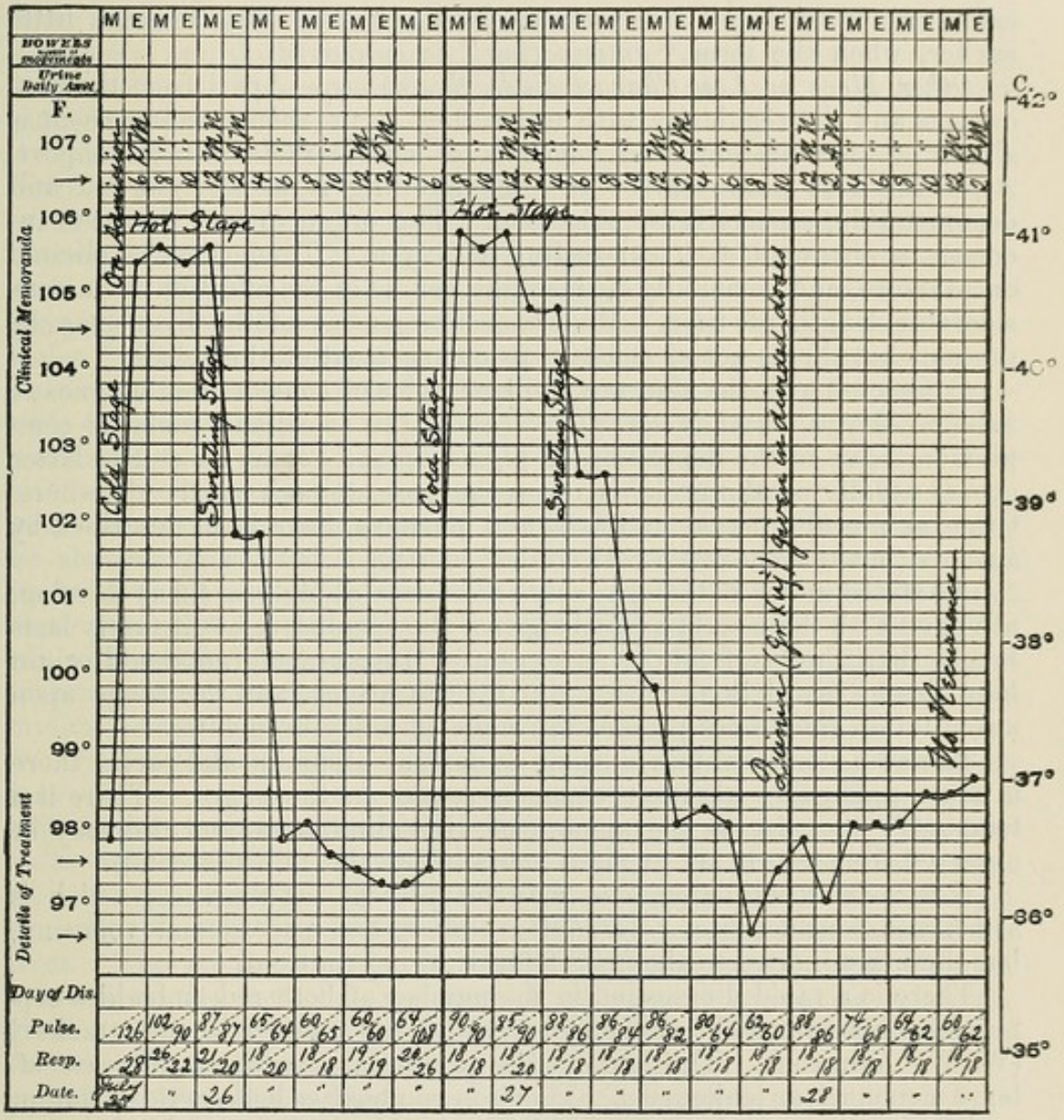


FIG. 26.—Temperature-curve in a case of double tertian fever. C. F. C., aged forty-one years.

(*b*) *The Manner in which the Paroxysms Recur.*—The special characteristic of this form of intermittent is the regularity with which the paroxysms recur in cases that are not under treatment. The intermission, or time between two successive paroxysms, is most frequently twenty-four hours (quotidian intermittent fever); almost as often it is forty-eight hours (tertian intermittent); and less frequently it is seventy-two hours (quartan intermittent). If there be two paroxysms on

one day—a rare occurrence—the term "double quotidian" is used to designate the case. Of the above types, as stated in the life-history of the parasite, three only—the *quotidian* (malignant), the *tertian*, and the *quartan*—have been clearly distinguished. The quotidian ague (the most frequent clinical variety) is generally due either to double infection by the tertian parasite or the quotidian parasite (single infection), and rarely is it to be attributed to the presence of three groups of the quartan parasite, resulting in daily sporulation. It sometimes happens that the paroxysms recur a couple of hours later each successive day, when it is called a "*retarding*" intermittent fever, or they may recur a little earlier, when the term "*anticipating*" is employed.

Other More or Less Characteristic Symptoms.—Apart from the paroxysms and the regularity with which they recur, *splenic enlargement* is almost always present, and hence is of considerable clinical import. After the first paroxysm or two the swelling is usually marked and demonstrable, especially by palpation. The organ can be shown to increase in size with each succeeding paroxysm. Tenderness is elicited on pressure, and commonly outlasts the course of the affection for a considerable length of time. Moderate enlargement of the liver may be present, but this is not so constant as enlargement of the spleen.

Connected with the *skin* are two symptoms of considerable diagnostic value: (1) a *yellowish-brown discoloration*, the so-called "malarial complexion," due to the deposition of pigment; and (2) *herpes*. The latter occurs usually on the prolabia or on the nose, though rarely elsewhere. Other skin-eruptions, as urticaria and purpura, have been described by authors, but they have no real clinical worth.

As stated under Pathology, *acute dilatation of the heart* may develop, attended with the usual physical signs of this condition, but it rarely lasts longer than the brief febrile paroxysm. Murmurs of functional origin may also be heard in the heart during the attack, and the *lungs* upon auscultation sometimes present the signs of a dry bronchitis.

The *urine* may contain a small amount of albumin, and rarely there is acute nephritis—a rather common sequel in the negro. There is a temporary increase in the amount of urea eliminated, and this may be observed from two to six or eight hours before the chill (Jaccoud).

Gastro-intestinal symptoms may be present, as diarrhea, which is sometimes considerable, *catarrhal jaundice*, and paroxysmal vomiting, but these are limited to the graver forms of intermittent.

There is a rapid diminution in the number of both red and white corpuscles, proportionate "to the severity and the number of the attacks" (W. W. Johnston). The leukopenia is associated with an increase of large mononuclear leukocytes. Thomson emphasizes leukocytic variation at different times of day, correlating these with the stage of development of the parasites; he also found that the more numerous the parasites, the greater the leukopenia, whereas a small number on sporulating cause a leukocytosis. The hemoglobin is reduced and granules of pigment are found in the plasma.

Clinical Varieties.—Besides the typical attacks, *mild* or *rudimentary* forms are met with, these either being due to slight infection or appearing as the remnant of cases of usual severity after active treatment. The separate stages of the febrile attacks are not well marked, and one or more may be missing; thus the chill may be absent (dumb ague).

In *children* there is no rigor noticeable. They grow pale, the mucous membranes often being slightly livid during the chill, and the paroxysms may be initiated by a convulsion or other nervous phenomena. Acute nephritis is a rather frequent sequel in white children.

(II.) **Pernicious Malarial Intermittent.**—This truly serious form occurs chiefly in highly malarial districts, and rarely also in the widespread regions in which the simple variety prevails. In the United States it is encountered most frequently in the Southern and Southwestern States. In this form of malaria the parasites of estivo-autumnal fever are constantly associated. The paroxysms do not recur with strict regularity, and the primary paroxysms are rarely pernicious in character.

Pathology.—This type of malaria may arise (1) as a fresh infection, and (2) as a reinfection.

(1) *Infection.*—The blood is more or less hydremic, and the blood-disks are in all stages of disintegration. The *spleen* is considerably swollen, soft, and its parenchyma is turbid and lake-colored, all its tissue elements being more than naturally pigmented. Upon microscopic examination pigment-granules and red corpuscles containing parasites and phagocytes are observed, particularly in the pulp adjacent to the arterioles. The *liver* is enlarged, soft, and turbid, and pigmentation occurs, but it is also microscopic. In the minute vessels phagocytes and parasites containing pigment are perceptible within the red corpuscles, and numerous small necrotic areas have been observed. The *kidneys* show microscopic pigmentation, most marked in the vicinity of its blood-supply. Minute areas of cell-death are sometimes seen. The *brain* may be abnormally colored, assuming in severe cases a chocolate tint, and in mild types a lighter hue. The brain-tissue is often anemic, and more rarely edematous. Occasionally there is congestion. The capillaries are literally blocked with phagocytes and blood-disks more or less disintegrated (containing parasites), and perivascular infiltration and minute hemorrhages may rarely occur, producing a focal lesion.

(2) *Reinfection.*—The blood is often extremely hydremic. The *spleen* may or may not be much enlarged, and is usually quite firm, with a well-marked pigmentation that is obvious to the naked eye. The *liver* is, as a rule, moderately increased in size, and is somewhat indurated, while macroscopically it is seen to be deeply pigmented. The changes presented by the *kidneys* differ in no essential manner from those of the liver. The microscopic appearances of the liver, spleen, and kidneys, apart from the fact that the amount of pigment present is relatively greater, are entirely analogous to those met with when a fresh infection occurs. Pigmentation of the lung is also common.

Clinical Varieties.—Three varieties merit description:

(*a*) *Congestive Chills* (*Algid Form*).—These are accompanied by raging gastro-intestinal symptoms (vomiting, purging, etc.), inducing systemic collapse, which simulates to a nicety the algid stage of cholera. The temperature of the interior of the body is much elevated. True dysenteric symptoms may arise, and sometimes jaundice, followed by grave nervous symptoms, may be a secondary development. The intellect is unclouded, as a rule. This condition is to be discriminated from yellow fever, with which it has frequently been confounded. The parasites in this affection center in a special manner in the gastro-intestinal

mucosa, in the vessels of which they may be seen in unusual numbers, sometimes forming distinct thrombi. In the United States this is the most common among the pernicious forms.

(*b*) *Hemorrhagic Pernicious Malaria.*—In this form the chill is severe and prolonged, and during the hot stage the urine is bloody and scanty, containing considerable albumin, with bloody, epithelial and granular casts. Hemorrhages from other outlets of the body (mouth, rectum, vagina, nares, stomach) may also occur, together with larger and smaller cutaneous ecchymoses, and the yellowish-brown malarial complexion is intensified. The mind may remain clear, although the patient is restless and anxious. Urinary suppression may ensue, and uremic toxemia be superadded; the greatest dangers being cardiac failure, uremia, and delirium (or coma independently of the latter). Death is rarely the direct consequence of hemorrhage. Brem believes this form to be due to an hemolysin produced by the malarial parasite.

(*c*) *Comatose Form.*—The chill may be absent. Grave cerebral symptoms, as acute delirium or sudden coma, seize the patient violently. The hot stage is attended with high fever, and if the patient survives the paroxysm, the violent nervous symptoms either disappear suddenly with the appearance of the sweating stage, or may outlast the latter by several hours. Primary paroxysms rarely prove fatal, but recurrences bring imminent danger. This variety is due to an inordinate localization of the malarial parasites in the brain, where they form complete thrombi, and induce pathologic lesions in the adjacent structures.

(III.) **Remittent or Continued Malarial Fevers (Estivo-autumnal Fever).**—On account of the intensity of the gastro-intestinal symptoms this variety is also termed *bilious remittent fever.* Its severity exceeds that of intermittent malarial fever. It prevails for the great part in warm and truly tropical climates, though it is also seen in its milder forms in temperate climates. The estivo-autumnal parasites previously described are the specific cause of the disease.

Pathology.—Melanosis of the spleen, liver, and brain is generally observed; on the other hand, in rare instances in which the specific parasite had even been demonstrated during life, the internal organs were found to be non-pigmented on autopsy. The degree of the pigmentation depends upon the length of time that the patient has been infected, as well as upon the frequency of reinfection. The *spleen*, if it be a fresh infection, becomes swollen, but is soft; in protracted cases it becomes permanently enlarged and firm. Microscopically the pigment is seen to be most abundant in the splenic pulp and within and around the splenic veins. The *liver* is enlarged in like manner. The pigment that is found in the form of granular masses in all the hepatic tissue-elements (especially Kupffer's cells, vessels, vessel-walls, and perivascular tissue) gives to the organ a bronzed appearance ("bronze liver").

As in pernicious malaria, so in this affection, the *brain*, and particularly the gray matter, is in long-standing cases of a dark brown or almost black color. The *arterioles* are often found stuffed with phagocytes and blood-disks which contain pigmented parasites. Punctate hemorrhages may occur in the brain. The *kidneys* are pigmented and may show "a severe acute degeneration of the cortical tubule cells" (Ewing). Massing of the parasites in the renal capillaries may occur. Other organs and tissues of the body, including the *lymphatic glands and the skin*,

become more or less deeply pigmented. The *blood* shows marked hydremia, with partly or wholly degenerated red blood-disks in abundance.

Symptoms.—There may be *prodromal* symptoms, such as headache, anorexia, and epigastric oppression, lasting a day or two, but these signs are variable. There may be daily or bi-daily paroxysms of fever which resemble the ordinary quotidian and tertian intermittent forms, with this difference, however, that the febrile paroxysms are of longer duration (twenty hours or more). Both the rise at the onset and the decline at the end of the paroxysm are more gradual than in true intermittent malarial fever, and the initial chill may even be wholly absent. The febrile attacks are often "anticipating," the succeeding paroxysm beginning before the elevated temperature of the preceding touches the normal level, giving rise to a remittent type of fever which often exhibits considerable irregularity. The remissions may become shorter, producing finally a continued type of curve—*continued malarial fever.*

In *typical cases* of remittent fever a chill generally occurs at the onset, but is less severe than in malarial intermittents. Shortly after the chill the temperature rises rapidly, so that in ten or twelve hours it may reach 104° or 105° F. (40.5° C.). The *pulse* is full and accelerated to 100 or 120, and there is rending headache. Nausea and vomiting are common; oppression in the epigastrium is intense, and there is well-marked tenderness in the latter region. The *spleen* is found to be enlarged on palpation. *Nervous symptoms* (delirium, coma, etc.) may develop speedily, and rarely a mild bronchitis may also arise.

About midnight the *remission* in the temperature and sweating begin, in consequence of which the headache and gastric symptoms largely disappear. The *temperature* usually drops to 100° F. (37.7° C.) by the next morning, to be followed by a new exacerbation of fever, which commences about noon of the second day. The same symptoms now repeat themselves. The affection has usually, by this time, reached its acme, and the temperature may have risen to 106° F. (41.1° C.). Grave *nervous symptoms* may also have appeared. The *urine* is diminished in amount, often slightly albuminous, and acute nephritis is observed in 4.7 per cent. of the cases (Thayer); while either a slight or marked hepatogenous jaundice may appear. Urriola[1] states that the presence of malarial pigment in the urine is a pathognomonic sign. A. C. Smith[2] reports instances of bubo (inguinal) as a complication. *Herpes labialis* is quite common. The nocturnal remission again ensues, and in the *mild types* or in those brought promptly under suitable treatment the febrile paroxysms grow briefer, resulting in an intermittent form of fever. The course of light cases is run, usually, within two weeks.

In *severe types* or in neglected cases the separate febrile paroxysms grow longer until the remissions become slight and simulate continued fevers. These are the cases that are distinguished by the same symptoms as those that mark typhoid fever, save only the eruption and the Widal reaction. The course of the attack, if not properly treated, prolongs itself to three, four, or more weeks, and the salient features of pernicious intermittent may suddenly appear and the disease may terminate life. On the other hand, *mild forms* of the continued type also occur, and these yield promptly to the specific—quinin.

[1] *Interstate Med. Jour.*, Jan., 1912. [2] *New York Med. Jour.*, June 22, 1901.

(IV.) **Malarial Cachexia.**—This is an exceedingly *chronic* condition, and is usually a remnant of one of the acute forms. When the latter are not properly treated, they are apt to drag on, and assume the characteristic features of chronic malarial cachexia. The condition may, however, be chronic from the start in truly malarial localities.

The **symptoms** are varied both in character and in intensity. There is *fever* at intervals, but chills do not occur, and the temperature-curve is typical neither of remittent nor intermittent fever, although it may approximate either the one or the other. Again, the fever is sometimes wholly irregular, though its range is not high, and it seldom exceeds 103° F. (39.4° C.). The *skin* often presents the dirty yellowish-brown complexion to a marked degree. The spleen is enormously enlarged and indurated, and hypertrophy with hardening of the liver may also be pronounced. The *blood* is profoundly anemic, the count in one of my cases showing but 1,300,000 red corpuscles per cubic millimeter.

Many of the local and general symptoms are dependent upon the well-marked *anemia*. Among *general features* may be mentioned debility, frequent sweatings, and dropsy. *Nervous symptoms* may also be noticeable, and chief among these are tremors, neuralgia, palsies, vertigo, wakefulness, and nervous palpitation of the heart. Among the rarest concomitants of this condition is paraplegia. *Malarial neuritis* is met with and presents most of the features common to other toxic forms of neuritis. Slight cough and dyspnea evidence the presence of mild *bronchitis;* and anorexia, nausea, diarrhea, and other symptoms of *chronic gastro-intestinal catarrh* are observed. The joints and voluntary muscles may be painful. *Hemorrhages* from the various mucous surfaces and into the retina are common; and I have seen one case in which spongy, bleeding gums, with numerous petechiæ, pointed to the existence of associated scorbutus. *Tuberculosis* finally developed and carried off the patient. *Chronic dysentery, fatty degeneration of the heart,* and *chronic nephritis* may develop and prove serious sequelæ. These cases do well, generally, if the patient can be removed permanently from the malarial district. In long-standing cases the spleen does not return to its natural dimensions. Complete recovery, however, may be expected.

(V.) Masked Intermittent.—This presents itself in much the same forms as chronic malarial cachexia, but with the important difference that there is no fever. This type comprises a long list of conditions, at the head of which stands *neuralgia*, most frequently involving the supraorbital branch of the trigeminus. Often a striking periodicity is observed, the painful paroxysms usually beginning in the morning and terminating in the late afternoon hours, the patient's sufferings increasing steadily in intensity until just before the close of the attack, when they suddenly abate. Among other nerves implicated with relative frequency are the occipital, the intercostal, and the sciatic. Except the blood-appearances be characteristic or unless the attacks yield promptly to quinin, a certain diagnosis of malarial neuralgia should not be ventured. Craig,[1] out of 395 cases of latent and masked malaria, found the estivo-autumnal parasite in 275; they appeared as a small hyaline disc or ring-form within the red blood-corpuscle. The parasites, however, have been observed in all stages of growth, even undergoing segmentation. Craig

[1] *Amer. Med.*, Oct. 29, 1904.

believes that the latency can be accounted for by the fact that the few organisms present do not generate sufficient toxin to provoke characteristic symptoms. Masked intermittents may assume the forms of paresthesia, anesthesia, convulsions, or paralysis; they may also appear under the guise of edema, hemorrhages from the various mucous outlets of the body or into the skin, diarrhea, dysentery, dyspepsia, bronchitis, pneumonia, appendicitis, etc. But, since these affections may all obey the law of periodicity, we should not pronounce in favor of malarial infection unless they yield readily to the therapeutic specific, or the parasite is found.

(VI.) **Malarial Hematuria and Hemoglobinuria.**—I have previously described a hemorrhagic form of pernicious intermittent in many cases of which hematuria is a prominent symptom. Boisson,[1] in 3 cases of hemoglobinuric fever, occurring in soldiers attacked with malaria in Madagascar, found great reduction in the erythrocytes, while 7 out of 10 red cells contained parasites. I have observed several instances of malarial hematuria in the Kensington district of Philadelphia, where the milder forms of malaria prevail. Hematuria in its severest form is seen with the approach of cold weather (Jones). It is rare in the negro. Young in both sexes and males over puberty are most apt to suffer. The blood shows pigmented parasites (forming rosettes), and sometimes crescents and pigmented leukocytes.

The **symptoms** consist of a mild cold stage, a subfebrile temperature to which is added hematuria, or more often hemoglobinuria. The paroxysms may recur daily, bi-daily, or at longer intervals, and in severe forms the hemoglobinuria may be continuous, with aggravations at definite intervals. Suppression of urine may appear early, accompanied by uremic features, *e. g.*, coma, nausea, vomiting, diarrhea. The lumen of the renal tubules may be occluded by plugs of granular material derived from the hemoglobin. The *diagnosis* demands the demonstration of the malarial parasites in the blood, and of the hemoglobin in the urine. Tyson recommends Teichmann's (hemin crystals) test to show the presence of hemoglobin. The earthy phosphates are precipitated, filtered out, and a small portion placed on a glass slide and carefully dried. A minute granule of common salt is carried on the point of a knife to the dried mass and thoroughly mixed with it. Any excess of salt is then removed, the mixture is covered with a thin glass cover, a hair interposed, and a drop or two of glacial acetic acid allowed to pass under. The slide is then carefully warmed until bubbles begin to make their appearance. After cooling, hemin crystals can be seen by the aid of the microscope, and are easily recognizable by an amplification of 300 diameters. Chemically they are hydrochlorate of hematin.

The so-called blackwater fever is an intoxication due to repeated attacks of malaria, in which "some exciting cause produces a sudden hemolysis" (Prout), and quick spontaneous disappearance of the malarial parasites (Plehn). Other observers (Sambon, Macay) regard hemoglobinuria as a specific disease. Bass and Johns found that calcium salts added to culture-mediums caused hemolysis of the infected as well as the non-infected cells of the blood of the sufferer. The leading characteristics are irregular paroxysms of fever with rigor, bilious vomiting, jaundice, hemoglobinuria, and nephritis. This form occurs in the Philippines, in Germany, and other countries.

[1] *Rev. de Méd.*, May 10, 1896.

According to Frank A. Jones, obesity occurs among persons coming from a climate free from malaria to the Mississippi's delta. They neither have chills nor manifestations of chronic malaria. "The obesity subsides rapidly by changing from a malarious to a non-malarious climate."

Complications.—The author's analysis of 1780 cases of malaria (intermittents and remittents) showed complications in about 10 per cent. The more common among these were: Enteritis (16), nephritis (14), rheumatism (10), typhoid fever (8), lobar pneumonia (5), jaundice (5), and dysentery (4). The opinion of the profession is divided upon the question: "Has pneumonia any special connection with malaria?" According to the results of my collective investigations, pneumonia is rarely associated. Craig affirms that malaria may present typical symptoms of pneumonia, probably owing to a localization of the malarial parasite in the capillaries of the lungs. Thayer's studies show that the frequency of albuminuria and nephritis in malarial fever is somewhat below that observed in the more severe acute infections.

Typhoid fever is a complication of malaria, according to these researches, but the relationship between these affections cannot be close.

Diagnosis.—(1) Of Intermittents.—This is difficult, unless the brief febrile paroxysms, with their characteristic stages and other diagnostic features (enlarged spleen, malarial complexion, herpes), together with the rigid periodicity of the paroxysms, be present. Residence in a malarial district is confirmatory. The only unquestionable method of diagnosis is provided by a microscopic examination of the fresh blood. If this cannot be made an early diagnosis is rarely possible until the peculiar manner of recurrence of the paroxysms is established.

Differential Diagnosis.—Non-malarial affections, exhibiting an intermittent form of fever, are often mistaken for malarial intermittents. Of these, (*a*) *pyemia* is very apt to be thus confounded. It will be observed, however, that the chills occur at more irregular intervals, and that prostration is more profound during the intervals between the febrile exacerbations. The etiologic factors and place of residence are also to be considered. The blood should be examined microscopically, and, if this be impossible, the therapeutic test will, as a rule, remove any doubt. *Leukocytosis* is common in pyemia and absent in malaria.

(*b*) *Acute tuberculosis* and, more rarely, *incipient chronic tuberculosis* may present a febrile movement in no way differing from quotidian intermittent, except that in the former the pyrexia develops in the afternoon instead of the forenoon, as in the latter. A clear history, the associated local and general symptoms, along with the results of a careful physical examination, usually render tuberculosis probable and distinguish it from malarial intermittents. In tuberculosis the chills recur despite the use of quinin, and this is not the case in malaria.

(*c*) *Ulcerative endocarditis* may exhibit an intermittent pyrexia, but the history is different, and the associated features are more numerous and decidedly more grave. A blood-examination reveals leukocytosis—a distinguishing feature. Again, quinin is without effect. The irregular forms of intermittents are difficult in the extreme to diagnosticate. If, in suspected cases of "erratic" malaria, quinin is resisted, we cannot feel certain of our diagnosis unless we obtain the microscopic evidence of the presence of the malarial parasite in the blood.

(2) The diagnosis of **remittent fever** would be easily made if it did not sometimes bear a strong resemblance to typhoid fever. Its certain recognition demands the detection in the blood of the estivo-autumnal parasite. In *typhoid fever* the history points to a more gradual onset, the remissions are less marked, and epigastric oppression is wanting. Again, typhoid has its characteristic eruption and gives the sero-reaction. (For diagnosis from hepatic abscess, *vide* p. 926) Chronic malaria must be differentiated from *chronic biliousness* or *enterogenic intoxication.*

Method of Examining the Blood for the Malarial Parasite.—The finger or lobe of the ear is carefully cleansed, and then slightly cut with a sharp lancet. The first drop of blood is wiped away and the second collected on the center of a clean cover-glass, which is immediately placed upon a clean slide and the blood allowed to spread in a thin film, and examined immediately through an oil-immersion objective. It is all-important that the blood be perfectly spread between the surfaces of the slide and cover-glass, in order that the corpuscles do not rest one against the other. In the fresh specimen one is able to detect the parasite during all its developmental stages seen in man, but the best time is either just before or during the chill. If the blood of estivo-autumnal fever be exposed to the air a short time and then mounted in this manner, it is likely to display flagella. If desirable to preserve the specimen or if impossible to make the microscopic examination at once, smears should be prepared by laying another cover upon the first, allowing the blood to spread in a thin layer, and then sliding them apart quickly and drying in the air. If permanent specimens are desired, Wright's modification of Romonowski's stain is to be preferred. The specimen should be covered with the solution and allowed to stand two minutes. To stain, add 3 or 4 drops of distilled water and allow to remain two or more minutes, when the specimen is washed with water, dried, and mounted in Canada balsam. The organisms appear as small blue bodies, often containing pigment. For the crescent and oval forms, often difficult to find, it may be advantageous to allow a drop of blood to dry upon the cover-glass without spreading, fix, and then wash with dilute acetic acid; wash thoroughly with water and stain as before. The hemoglobin of the red cells is dissolved, and only the white cells and the parasites remain visible.

Prognosis.—Uncomplicated cases of intermittent fever under proper treatment generally recover. In certain malarious regions and seasons pernicious types are prevalent. *Primary pernicious attacks* are moderately dangerous, while recurrences are highly so. The mortality-rate in this variety is between 20 and 25 per cent. In *remittent fever* death may be due to asthenia, particularly when the type is severe and when wrong notions as to treatment prevail. Suppression of urine, followed by uremic symptoms, hemorrhages, and intense jaundice are grave complications.

Treatment.—**Prophylaxis.**—The investigations cited above show that an individual ill of malaria is a source of danger in a community, and should be promptly protected from mosquitoes, and then treated. The homes, and more particularly the sleeping-apartments, of persons residing in paludal regions, should be protected against invasion by mosquitoes. The use of wire netting is to be advised for this purpose. Caps to which the same material is attached may be worn out

of doors. Methods for destroying the mosquito (adult female and larvæ) should be adopted. In rooms this is best accomplished by fumigation; in the outer world the breeding-places (*e. g.*, marshes) must be found and then removed by thorough drainage and covering water-barrels and privies. The larvæ are most effectually suffocated by sprinkling petroleum upon the water, to the surface of which they rise to get air.

Koch states that gametes are often found in children and that many persons harbor the parasite without manifesting active symptoms; he advises prophylactic doses of quinine in malarial localities.

1. For **intermittent malarial fever** there is an almost infallible remedy in quinin. "When shall its use be commenced?" is a pertinent question. It would certainly seem highly desirable to check the course of the disease as soon as possible, and especially since transmission of the simple intermittents into the pernicious forms may occur if the disease be not arrested. At the present day specific treatment is often delayed in order to give full opportunity for making a blood-examination with a view to completing the diagnosis. There is no decided advantage in commencing the use of quinin during the first paroxysm, when the blood may be examined; but on finding the case to be one of malaria, quinin should be administered after the paroxysm, so as to prevent a recurrence. For like reason, if the history at the physician's first visit, combined with the symptoms presented, make the diagnosis of intermittent malaria reasonably certain, and there is no opportunity to examine the blood microscopically, the principal antiperiodic remedy should be commenced at the close of the paroxysm. The quinin cures malaria by acting directly upon the intracorpuscular hematozoa (the young forms).

During the *paroxysm* we should aim to make the patient comfortable. He is to remain in bed, is to be well covered, and external heat applied during the cold stage; and he is to be lightly covered, given cooling drinks and cold spongings during the hot stage.

During the apyrexial intervals the patient may leave his bed, provided that he feel strong enough, and, as before intimated, the specific remedy is given during the afebrile period. Certain authors recommend that the entire daily quanitity be given at one dose from four to six hours before the succeeding paroxysm is expected, the object being to surcharge the blood at the time when the hematozoa sporulate. The total amount per day required to destroy both the asexual and sexual parasites is from 16 to 20 grains (1.036 to 1.296) for a period of three weeks, in most temperate climates. When this fails, more may be given—24 to 30 grains (1.555–1.944). My own practice has been to administer immediately after the close of the sweating stage gr. iv or v (0.259 or 0.324), repeating the same dose a few hours later, and the remaining 8 or 10 grains (0.518 or 0.648) (or one-half the daily dose) six hours before the time for the next paroxysm. I have thus escaped the slight toxic symptoms (tinnitus, deafness, nausea, etc.) which are apt to follow single large doses. The remedy should be administered in the form of the hydrochloride, in capsules. After the attacks cease to recur quinin should be continued in amounts of 6 to 8 grains (0.388 to 0.518) daily for several days. If quinin cannot be taken *per os* it may be tried by enema or by suppositories in appropriately large doses. In young subjects I administer the quinin by suppository.

The physiologic effects of the drug can be quickly obtained by administering it hypodermically. Hence, if there be no time for absorption from the stomach (four hours being the shortest period it is safe to allow), the drug should be thus employed. For this purpose the more soluble salts (hydrobromide, hydrochloride) of quinin are to be preferred to the ordinary and more insoluble sulphate.

Many preparations of cinchona other than the salts of quinin may be tried, and among these cinchonin administered in the same manner as the latter is the best substitute. Some contend that the sulphate of quinidin has antiperiodic power, almost equal to quinin. In prolonged cases the salts of quinin and other preparations of cinchona sometimes lose their specific influence, and arsenic is then to be employed, either alone or in combination with the former agents. The dose of the arsenic, beginning with ♏iv (0.266) t. i. d. of Fowler's solution, must be increased until its physiologic effects are produced. Arsenious acid often does even better service than Fowler's solution. Administered as above indicated, this remedy is most efficacious in malarial cachexia and masked forms of intermittents; it may be combined with iron and quinin. Atoxyl, either alone or associated with quinin, is capable of bringing about rapid improvement in health, especially in cachexia and chronic forms of malaria. In cases of malaria that are resistant to quinin (often the quotidian forms), methylene-blue has been found extremely active and serviceable. Cardamates believes that it is indicated only when quinin is contraindicated, as in hemoglobinuria or in pregnancy, when abortion is feared. It attacks the adult forms of the parasite. Werner recommends salvarsan (0.6 to 0.7 gram) in cases in which the parasite is resistant to quinin. While in charge of the out-patient service of the Episcopal Hospital, Philadelphia, I employed in chronic malarial cachexia, with very satisfactory results, the sulphate of cinchonidin in daily doses of gr. xxx–xl (1.944–2.592). In this class of cases Warburg's tincture ʒss (16.0), three times a day, has been warmly recommended. Splenectomy has been recommended in intractable forms, but it is to be performed only as a *dernier ressort.*

2. **The Treatment of Pernicious Intermittents.**—(*a*) *Prophylaxis.*—By treating all ordinary intermittents actively after the first paroxysms the occurrence of pernicious forms can be obviated. Not to pursue this course in malarial seasons and localities is next to criminal.

(*b*) *The first pernicious attack* must be treated immediately, and there is not a moment to be lost. Hence in all varieties of pernicious intermittents quinin should be administered hypodermically until the patient is fully cinchonized—a condition that must then be maintained for several days. In all varieties stimulants are to be used freely if the heart's action becomes feeble, and the patient is to be well nourished throughout. There are other details, though of relatively minor importance, and they vary with the individual forms. Thus in "congestive chills" external warmth is useful, and morphin combined with atropin should be given hypodermically, this combination tending to allay gastro-intestinal symptoms as well as to warm the extremities, and meeting really important indications. Rectal feeding must be resorted to should the stomach refuse to retain nourishment. In the *comatose form* the nervous symptoms are most successfully combated by prompt and energetic

antiperiodic treatment, together with vigorous stimulation and feeding, since they are due to the intensity of the infectious process.

(*c*) During the apyrexial period every effort must be made to prevent a recurrence of the paroxysm, and to this end the patient must be kept fully cinchonized until the time for the next paroxysm is over.

3. **Treatment of Remittent Fever.**—The mode of treatment in this form differs somewhat from that appropriate for intermittents. At the onset a mild mercurial is advantageous (calomel gr. ¼ (0.0162) every hour for three doses), followed by a saline laxative (Rochelle salts, ʒij; 8.0). During the febrile exacerbations cool spongings of the body, coupled with the use of the ice-cap, are serviceable. The gastric symptoms demand chipped ice by the mouth or small doses of cocain, and a mustard plaster externally. Immediately after the first remission sets in quinin must be exhibited, and large doses are now indicated (gr. xv (0.972), to be repeated at 8 or 9 A. M.). A third and even a fourth dose of the same size may be required. The exacerbations of fever generally yield to this remedy, but if, as rarely happens, they do not, then small doses of pilocarpin (gr. ⅛ to ⅙; 0.008 to 0.010) may be administered hypodermically during the height of the fever. This causes free sweating in many instances, and in consequence renders the remission more marked and more prolonged; thus, in short, rendering the course of the affection speedily favorable. The heart must be guarded.

A case that has been allowed to run on for one, two, or more weeks is often greatly benefited by the use of Warburg's tincture, as before recommended, for several days, when quinin may be re-employed. The patient, especially if the case be protracted, must be vigorously fed, and *per rectum* if it cannot be accomplished by the mouth. In typical cases, which are promptly controlled by quinin, stimulants are rarely needed, or at least not until the convalescent stage is arrived at. In severe and neglected cases the indications for their employment may be presented early, and they should then be given, the physician conforming to the same rules as in typhoid and other acute infectious diseases. The renal congestion and anuria are to be met by internal diaphoretics (pilocarpin, etc.) and by saline laxatives. Most efficacious, perhaps, is a combined hot-water and steam bath. The patient is placed in hot water, and then a blanket is put around the neck, its free ends being allowed to extend over the edges of the tub.

4. **Treatment of Malarial Hematuria.**—Whether or not quinin is to be employed in hemorrhagic pernicious malaria is a question involved in doubt. The statistics of M. Brady indicate a tremendous advantage in discontinuing quinin as soon as blood shows in the urine—a reduction of mortality from 24 to 6 per cent. Forcheimer also holds that if in an attack of black-water fever the administration of quinin is followed by hemoglobinuria, the quinin should be withheld. Under these circumstances the use of methylene-blue has given favorable results. This remedy is best administered in doses of gr. iss–iiss (0.097–0.162) every third hour, and it should be continued in somewhat diminished dosage for a week or longer after the cessation of fever. Cholesterin has been found to arrest paroxysmal hemoglobinuria. Pringsheim advises five intramuscular injections of 5 c.c. each of a 10 per cent. emulsion of cholesterin in eleven days.

DISTOMIASIS.

(*Trematodiasis.*)

VARIOUS forms of trematodes, including the distomata, may become parasitic in man.

Distoma Hepaticum (Liver-fluke).—Among the more common varieties of trematodes or flukes, is the distoma hepaticum or liver-fluke, a parasite found in animals (horse, goat, ass, sheep, rabbit) and accidentally ingested by man.

It is almost 30 millimeters (1.1 inches) in length, and inhabits the biliary passages of the animal, and from them is discharged into the intestinal tract and evacuated with the feces. Under certain conditions of temperature and moisture, a ciliated embryo escapes from the egg, and is ingested by a gasteropod or snail (*limnæa truncatula*), in which it undergoes development into a sporocyst, that in turn gives origin to *radiæ* or parent nurses. These give birth to daughter-radiæ or *cercariæ*, which leave the gasteropod or snail and attach themselves to aquatic plants, where they are in turn eaten by animals.

Symptoms.—When present in sufficient numbers in the bile-passages the liver becomes greatly enlarged, with the occurrence of jaundice and ascites that may prove fatal. Other symptoms may also be present; thus pain was prominent in 41 out of 100 cases reported by Kurimato in Japan, and heart-murmurs were present in 42 of those cases.

Late in the disease the liver may become nodulated and terminate in atrophy.

On inspection in well-marked cases, a peculiar barrel-shaped bulging is sometimes seen, extending over the hepatic area, with tense abdominal walls over the enlarged liver. This is a pathognomonic symptom of hepatic distoma. An endemic form occurring in Japan has been described; it is characterized by marked emaciation, diarrhea, hepatic enlargement, and often by ascites.

The **prognosis** of distoma hepaticum is absolutely fatal and the **treatment** is merely palliative, though it may run a course extending through many years, often with intermissions, even apparent recovery, later to relapse.

Among other trematodes may be mentioned (*a*) *distoma lanceolatum* (found also in cattle); (*b*) *distoma crassum*, which is larger in size than the preceding; (*c*) *distoma sibiricum;* (*d*) *distoma pulmonale* (*D. Ringeri*); (*e*) *distoma spatulatum* (*endemicum*); (*f*) *amphistomum hominis;* (*g*) *distoma hematobium* (Bilharz). Two of these deserve extra, though brief, mention.

Parasitic Hemoptysis (*Distoma Pulmonale*).—This is caused by the *Paragonimus Westermanii* first described by Manson (1880) and Baily (1880) in man. The lung fluke worm has also been found in the tiger (originally by Kerbert), hog, dog, and cat. The disease is extremely prevalent in certain provinces of Japan and China. Elsewhere it is usually mistaken for pulmonary tuberculosis. Stiles and Hassall[1] have discussed the whole subject. The parasite is 8 to 16 mm. long, 4 to 6 mm. broad, and 2 to 5 mm. thick. It is found encysted,

[1] *Annual Report of the Bureau of Animal Industry*, 1899.

usually two individuals in each cyst, with eggs, and its habitat is the lungs of mammals. It enters its final host (man, etc.) either encysted or as a free-swimming *cercaria*. *The mode of infection.*—Eastern writers look upon the drinking-water supply as the source of infection, and this view has much in its favor. The disease has been found in hogs throughout various sections of the United States by Stiles; this suggests the possibility of widespread infection in America.

Predisposition.—Most cases occur between the ages of eleven and thirty years. Out of 58 sufferers, 38 were farmers (Stiles).

Symptoms.—In the usual form (lung infection), *cough* is common but not constant; the sputa are similar to those of lobar pneumonia, although they may be absent from time to time. Free *blood-spitting* often occurs at intervals. Jacksonian epilepsy may supervene from metastasis to the brain.

Diagnosis.—This rests upon the detection of the eggs in the sputum. Place a drop of the bloody sputum on a slide, and upon it a cover-glass. On microscopic examination the red color will be found due to both red blood-cells and large dark-brown, thick-shelled, operculated ova, which vary from 80 to 100 μ in length, and from 40 to 60 μ in breadth.

The **prognosis** depends upon the number of the *parasites present*, *the age of the patient* (the young and the old bearing the disease badly) and the presence or absence of complications. Pulmonary tuberculosis is an unfavorable complication.

Treatment.—Prophylaxis embraces care regarding the drinking-water, and the collection and disinfection of the sputum as in pulmonary tuberculosis. The patient should be sent to healthy non-infected areas. There is no special *medical* treatment.

Distoma Hematobium (*Bilharzia hematobia; Blood-flukes*).—This hematode is a narrow worm with anterior abdominal sucking-disks. The male is shorter and thicker than the female; the former being 4–15 mm. ($\frac{1}{6}$–$\frac{3}{5}$ in.) long; the latter, about 20 mm. ($\frac{4}{5}$ in.). It prevails mostly in Egypt, Cape Colony, and other parts of Africa, and its entrance into the human body is now believed to be through the skin of those who bathe frequently in the African rivers, in many of which it abounds. It is not unlikely that, as formerly held, infection may also occur in many cases from drinking the impure water of the rivers. The parasites or their ova are found in the bladder, the pelvis of the kidney, and the veins (portal, mesenteric), most rarely the pulmonary.

The **symptoms** are hematuria, with some pain during urination. The last few drops of urine voided only contain blood, although rarely hemorrhage is more extensive and then the entire bulk is blood-tinged. Cystitis often occurs, with resultant thickening of the bladder wall. The ova become nuclei for vesical stone-formation.

Proctitis may result when the parasites lodge in the rectum, in which case mucous and bloody stools with tenesmus result. Ova of the parasites are found in the urine. No serious systemic disturbances occur in bilharziosis except, rarely, profound anemia from loss of blood. There is slight leukocytosis with increase in the eosinophile and large mononuclear cells. Prophylaxis as regards drinking and bathing in African waters should be exercised. Fouquet affirms the value of the extract of male fern internally in this form of distomiasis.

CESTODES.

ECHINOCOCCUS DISEASE.

(Hydatid or Bladder-worm Disease.)

THE tænia echinococcus is the smallest tape-worm of our domestic animals, and lives between the villi in the small intestine, especially in the larger breeds of dogs. It has a length of from 4 to 9 mm. ($\frac{1}{6}$ to $\frac{1}{3}$ in.), and consists of only three or four sections, the last one of which is mature. The rostellum projecting from the small head has thirty or forty hooklets arranged in a double row and a quadruple sucking apparatus. Thousands of eggs are contained in the mature segment. The intermediary hosts for the larvæ are rarely man, the horse, and the sheep, but more often the hog and ox.

Life History.—The ova, embryos, or the proglottides even, of the adult tenia are voided by the dog, and in various ways, later, are ingested by man. The dog first becomes infected by eating the echinococcus cysts of some animal that harbors the larval form of the tenia, and the matured teniæ appear in from eight to ten weeks. The liberated six-hooked embryos burrow through the intestinal wall or enter the portal vein; they pass into the solid viscera and muscles. There they develop into the larval form and cause the formation of echinococcus cysts.

In the development of echinococcus cysts, about four weeks after the ingestion of the bladder-worm eggs, small nodules appear, about 1 mm. ($\frac{1}{25}$ in.) in size. In about five months the cyst-walls consist of two layers, an external layer and an inner, granular, parenchymatous layer (or endocyst), containing a clear liquid. As the reaction to the irritation caused by the parasite and its cyst increases, a fibrous investment forms around them. At this time, also, small daughter-cysts, or vesicular buds, form the minor granular layer of the mother-cyst, and contain the heads of the larvæ. They are soon set free, and may themselves give rise to other or granddaughter-cysts in a similar way. These really become the breeding capsules of little cellular outgrowths that form the scolices or heads of future teniæ. They show the four sucking disks and a circle of hooklets. Each scolex, when taken into the intestine of the dog, develops into an adult bladder-worm or tænia echinococcus. This endogenous mode of cystic growth is common in man (*E. hydatidosus*). In animals the so-called exogenous cyst development is the more common in which the primary cyst-buds push out between the cyst wall and then develop externally. A third variety is the multilocular echinococcus (*E. alveolaris*, Buhl), affecting principally the liver. A large, hard tumor is seen that on section shows a firm connective-tissue framework surrounding alveoli that average a small pea in size. These alveoli contain small echinococcus cysts with thick, laminated walls. They may contain scolices or hooklets, and sometimes they are sterile. The echinococci may be situated in the lymph-channels and bile-ducts (Zenker).

The pure hydatid fluid is colorless, limpid, neutral in reaction, and has a specific gravity of 1005 to 1012. About 96 to 98 per cent. is water, and sodium chlorid, carbonate, and sulphate; traces of sugar (dextrose); cholesterin and uric acid are found among the constituents.

Among the changes that an echinococcus cyst may undergo the commonest is that of the *death of the echinococci*, as from diminished nourishment due to intense proliferation of daughter- and granddaughter-

cysts. The contents become thickened, putty-like, or granular, and even calcified. Remnants of these obsolete cysts, such as the chitinous substance of the old and outer wall-layer and hooklets, may be found. Sometimes *rupture* of the cyst occurs, with serious consequences to the patient; or the peritoneum daughter-cysts or free scolices may be disseminated and grow. Or *perforation* into the respiratory, digestive, or urinary tracts and discharge of daughter-cysts and hydatid fluid may take place. Lastly, *suppuration* and the formation of large hepatic abscesses may ensue, either spontaneously or on account of septic instruments used for tapping the cysts.

Etiology.—Carelessness in the feeding and the keeping of dogs is the primary source of hydatid disease, and the preparing of food where dogs are allowed to roam about, and so on, accounts for the majority of cases. Females are more often affected than males, and children and young adults seem to be oftener affected than those older in years.

As regards the *geographic distribution*, echinococcus disease prevails most extensively in Iceland, where man and dog live closely together. In Australia, also, many persons are affected. It is not so common in Europe, Asia, or Africa, and in America it is rare.

Organs Affected.—The tenia echinococcus has an undoubted predilection for the liver. "Of 1806 organ-infections, the following organs were the most frequently affected: liver (1011), lung (147), and kidney (126)" (Stiles). The brain and spinal cord, spleen, bones, muscles, the heart, and blood-vessels are involved with uncertain frequency.

Symptoms.—Hydatids of the Liver.—Unless the cystic tumors compress the portal area or the biliary passages, or invade the neighboring viscera, subjective symptoms may be entirely wanting. Not infrequently echinococcus sacs, partly calcified, have been found *postmortem*, not having produced any symptoms during life. Gradual but progressive loss of flesh and strength with the presence of a fluctuating tumor may be the only symptoms present until late in the disease. If the cysts attain a large size, a sensation of dragging, and of pain even, is often present; as a rule, however, pain is absent throughout the course of the disease. If the tumor displaces the diaphragm upward and compresses the lung, cough and dyspnea result. In some cases the sac has ruptured into the bronchi, and given rise to cough and to expectoration of the fluid and vesicles.

If the portal veins and bile-duct are compressed, splenic enlargement from passive congestion, ascites, and jaundice will occur, these symptoms being more common when the cysts are multilocular. Rupture may occur into the intestines (colon), into the pleura or pericardium, causing pyothorax or pyo-pericardium, or into the inferior vena cava, causing fatal pulmonary embolism.

Fever is usually absent throughout, unless the contents of the sac become converted into an abscess; then rigors or chills, fever (hectic in type), and sweatings occur, with jaundice (more or less intense) and rapid emaciation.

Not infrequently the cyst-wall becomes partly calcified and the contents are reabsorbed.

When rupture occurs, unless the contents be evacuated through the respiratory, alimentary, or urinary tracts or externally, symptoms of collapse develop and are followed by death. Toxic erythema or urticaria may follow rupture of cyst.

The **physical signs** give on *inspection* fulness or bulging in the right

hypochondriac region, especially if the cyst be single, of large size, and situated anteriorly.

Palpation confirms inspection and shows a fluctuating mass or masses. A trembling impulse is felt sometimes on deep palpation, aided by light percussion over the opposite side of the cyst, constituting the so-called "hydatid thrill." This sign cannot always be elicited, but when present is pathognomonic of the disease. The remainder of the liver shows uniform enlargement. The spleen is often palpably increased in size from passive congestion.

Percussion reveals, in addition to the hydatid fremitus, an increased area of dulness to the left or posteriorly, depending on the location and extent of the growths. If the left lobe be involved, the line of flatness may extend across the sternum to the left hypochondriac region. If the cysts are multiple and on the antero-inferior surface, the stomach may be displaced toward the left and dulness may extend across the epigastrium; if posteriorly, the pleural cavity may be encroached upon, causing an increased area of flatness upward in the postero-axillary line. Frerichs claims the line of dulness posteriorly in hydatid disease to be a curved one, whose convexity is upward.

Auscultation gives, according to Santoni and others, a short sharp booming sound when the tumor is percussed, that may be likened to one produced by striking a membrane stretched over a metallic frame.

Diagnosis.—In the entire absence of subjective symptoms and of characteristic physical signs, the diagnosis is impossible. If the cyst be of sufficient size to give fluctuation and the liver be irregularly enlarged, with an absence of fever, pain, and marked emaciation, the disease may be strongly suspected. The only certain demonstration of the condition is the discovery of the characteristic hooklets or heads in the aspirated or discharging contents of the cyst. Among the conditions that may be misdiagnosed for hydatid disease are—(*a*) Dilatation of the gall-bladder, (*b*) hydronephrosis, (*c*) right-sided pleurisy with effusion, (*d*) syphilis of the liver, (*e*) carcinoma, (*f*) abscess, and (*g*) cirrhosis.

Hydatid Cyst.	Dilatation of the Gall-bladder.
Previous history negative, except the companionship of dogs.	A previous history of having passed biliary calculi is often present.
Pain and jaundice usually absent.	Attacks of biliary colic followed by jaundice either are present or enter into the previous history.
Enlargement in any direction, depending upon the location of the cysts.	Enlargement is always in one direction—downward and posteriorly.
Hydatid thrill may be present.	"Hydatid fremitus" never present.
Less so.	The tumor is somewhat movable.

Hydatid Cyst.	Hydronephrosis.
The history is negative (*vide supra*).	There is a history of renal calculi or of vesical inflammation.
Urinalysis is negative.	Urinalysis reveals evidences of renal disease.
The tumor is most prominent over the hepatic area, and is associated with enlargement of the liver.	The tumor is most prominent in the flank and iliac fossa. If extending to the right hypochondriac region, it *does not* move with the liver.
The duration is indefinite and uremia rare.	The duration is short; a termination in uremia is common.

Hydatid Cyst.	Pleurisy with Effusion.
The onset is slow; pain and fever are absent.	The onset is sudden, and violent pain is present, with fever and dyspnea.
The presence of a fluctuating mass in the hepatic area, *not changing with the position of the patient.* Hydatid fremitus is present, but no bulging of the intercostal spaces.	The presence of effusion, beginning at the base of the chest and gradually extending upward—*changing with the position of the patient* and accompanied by bulging of the intercostal spaces.
Aspiration reveals a clear yellow liquid of low specific gravity without albumin, but chlorids, sugar, and hooklets.	Aspiration gives a cloudy, turbid liquid, containing albumin and flakes of lymph with high specific gravity.
The disease invariably runs a chronic course.	The disease generally runs an acute course.

For a differential diagnosis from (*d*), (*e*), (*f*), and (*g*) I would refer the reader to the discussion of the several diseases (*vide* Diseases of the Liver).

Echinococcus of the Respiratory Organs.—The lung has been the seat of the larvæ quite frequently, and instances have been noted especially in North Germany and Australia. The right lower lobe has been the seat of predilection, though sometimes the pleura is the primary source of trouble. There are pain in the chest, cough, dypsnea, perhaps arching of the overhanging thoracic region, signs of a pleural effusion, a tympanitic note above the prominence, hemoptysis, and the pathognomonic expectoration of hydatid disease. The general condition may or may not be seriously affected. Perforation into the pleural sac by pulmonary echinococci may be followed by empyema, and, later, by perforation of the chest wall. The heart may be dislocated. Compression of the lung may produce gangrene.

The *diagnosis*, in the absence of the characteristic sputum, is to be made from phthisis and a pleural effusion. Their location at the base of the chest may serve to differentiate hydatid cysts from phthisis, as well as the absence of marked emaciation. The characteristic curved upper boundary of dulness in pleural effusion and the change of the boundary upon changing the patient's position will serve to distinguish this affection. Pleural echinococci sometimes cause great compression of the lung and a barrelling of the chest on one or both sides. The pain may be quite sharp, and the respiratory murmur either distant or altogether absent.

Echinococcus of the Mediastinum.—Hare has collected 6 cases of hydatid disease among 520 cases of mediastinal tumors.

Echinococcus of the Heart.—Most of the cases have shown involvement principally of the right side of the heart.

Echinococcus of the brain and spinal cord should not be confounded with cystic degeneration of the choroid plexuses. J. H. Lloyd found 19 distinct cysts in the lateral ventricles and one occupying the fourth ventricle. The symptoms of cerebral hydatids are those of tumor, persistent and intense cephalalgia, vomiting, psychical disturbances, convulsions, amblyopia, and "choked disk," and sometimes paralysis. Hydatid disease may develop inside the dura mater, or it may penetrate from without and destroy the vertebræ before they compress the cord to a great degree. The symptoms are those of a compression myelitis.

Echinococcus of the Spleen.—About 40 cases of involvement of the spleen have been described. The organ may become greatly enlarged and be mistaken for that due to malaria, leukemia, etc. The hydatid thrill may be detected.

Echinococcus of the Kidneys.—More than 100 cases have been observed, mostly in Germany and France. The cyst may be as large as in hydronephrosis. Many of the cysts are of the exogenous form of growth. As a rule, one kidney only is affected, and generally the left one. Abdominal and thoracic compression symptoms may be caused, and bulging is often present in the lumbar region in marked cases. This may be punctured as an aid in the diagnosis. Rupture into the pelvis of the kidney and the discharge of the smaller cysts may give rise to renal colic and to the discharge of the cysts with the urine. More rarely, rupture of a suppurating cyst may take place in the loin.

Echinococcus of the peritoneum is rare as a primary condition. Echinococci have also been located in the bladder, prostate, testicle, ovary, uterus, great omentum, mesentery, pancreas, arteries, lymphatics, thyroid gland, muscles, bones, joints, parotid gland, orbit, and mamma.

A peculiar **complication** of echinococcus cysts is the occasional development of urticaria. It has been noted especially shortly after the puncture of a cyst, and this is somewhat diagnostic when it appears.

The **prognosis** is generally grave both as to life and cure, although some cases of hydatid disease of the liver have lasted for more than ten years.

The character of the changes in the cysts and their mode of termination influence the prognosis. Thus, the occurrence of suppuration is to be dreaded. Spontaneous cures have been noted in a few instances.

Treatment.—As in most of the other parasitic diseases, prevention is more or less effectual, and a cure is difficult or impossible. Infection of the dog should be avoided by preventing its gaining access to possible sources of hydatid disease, as the raw flesh of animals, especially in the form of meat-scraps around slaughter-houses. In order that human beings may not be affected, dogs should not be carelessly handled or allowed to be where they may come in contact with food and drink in any way, whether meat or eggs, vegetables, fruits, or cereals. Cleanliness in keeping dogs and in the proper preparation of food are essential in regions where hydatid disease is prevalent.

Medicines cannot reach the parasites in man, situated as they are in larval form encysted in the various tissues and organs of the body. Whenever the cyst becomes large, accessible, and the cause of troublesome symptoms, surgical measures may be resorted to. Among these are, simple tapping, tapping with aspiration, and with the subsequent injection of various substances (as iodin and zinc-chlorid electrolysis), and incision with drainage. Excision of the liver cysts has been practised by Raggi, Pozzi, Tansini, and others, but its practical value is still undetermined. Should suppuration occur, treat as an abscess.

TÆNIÆ OR TAPE-WORMS.

Natural History.—Tape-worms are found in the intestine of man, and are the matured or completely developed larvæ or cysticerci from the muscles and solid viscera of animals. Different varieties of cysticerci develop from the ova of the respective varieties of teniæ. These tape-worm eggs, after having passed out of the bowel, may be taken into the systems of various animals by various modes, entering the circulation, it may be, and becoming fixed within the solid tissues, especially the muscles. In about two or three months pea-sized cysts develop, and from the cyst-walls there gradually forms a new tenia-head, called a

scolex, or nurse. The worm-cysts, popularly termed "measles," constitute the cysticerci. Remaining in the tissues, they die and become calcified in from three to six years (Strümpell). But, if taken into the stomach by the eating of raw or partially-cooked meat, a tape-worm develops from the scolex. The maturation of the segments of the tape-worm commences several months after the fixation of the scolex in the intestine. In the natural life-cycle of a tape-worm the usual order of lodgement may be reversed. Thus man instead of a lower animal may become the host of the tenia eggs, which in turn may find their way into the solid viscera and muscles to develop into cysticerci. Again, this same order may be brought about by "auto-infection." The tape-worm has a ribbon-like form; although it has a number of segments and joints, giving it a link-belt appearance. When matured, these segments, or *proglottides*, develop male and female generative organs.

Varieties.—Tænia Solium (*Pork Tape-worm*).—This worm is rarer in America and also in Europe than formerly. It develops in the small intestine after the ingestion of raw or underdone "measly" pork. This worm does not necessarily exist singly, as its name would indicate, although such is usually the case. It ranges from 2 to 4 meters (6 to 13 feet) in length. The head is rounded, pin-head in size, and is succeeded by a thread-like neck and by gradually shortening and widening segments. Four suckers and a projecting circle of twenty-six long and short hooklets arm the head of the tenia. The mature ones become detached, and are passed with the feces. They are about 1 centimeter ($\frac{2}{5}$ in.) in length and from 6 to 8 millimeters ($\frac{1}{4}$–$\frac{1}{3}$ in.) in breadth, and about 1 meter (39.36 in.) from the head they are "approximately quadrilateral" in shape. These proglottides are bisexual. The female matrix occupies the middle of each proglottis, and is provided with from eight to fourteen irregular, tree-like branches on each side. The male generative organs are small vesicles in the anterior portion of the segment. The sexual opening is situated on one side, near the middle. The ovarian or uterine apparatus of a mature segment contains myriads of thick-shelled eggs, each one of which has an embryo with six hooklets.

Tænia Mediocanellata (*Saginata*).—The beef tape-worm is sometimes called the "unarmed tape-worm," since the head possesses sucking disks, but no hooklets. It is more common in this country and even in some of the European countries, as England. Longer than the tenia solium, being 4 to 10 meters (12 to 30 feet) in length, its segments are also thicker and larger, measuring from 16 to 88 mm. ($\frac{2}{3}$ in.) long, and from 8 to 10 mm. ($\frac{1}{3}$ in.) broad. The head of the worm as well as the ripe ovum is also slightly larger and proportionately thicker. The ovarian branches are more numerous (eighteen to thirty in number) and divide more dichotomously than those of tenia solium. Proglottides are also found in the stools, where they sometimes exhibit a crawling motion that has caused them to be mistaken for individual parasites. Cysticercus saginata has never been observed in man.

Bothriocephalus latus (*Fish tape-worm*, *Tænia lata*) occurs most commonly in Russia, Switzerland, Holland, and the German Baltic provinces. It is the longest cestode, measuring from 6 to 10 meters (20 to 30 feet). The head is club-shaped, unarmed, and has two lateral longitudinal grooves as suckers. The segments may be distinguished from

those of the preceding varieties named by their marked breadth and shortness, also by the centrally situated, tortuous ovarian rosette, and the sexual orifice near the center of the abdominal surface of each proglottis. The ova are larger than those of the pork and beef tape-worms, though thinner-shelled and with a sort of lid at one end. They develop only in fresh water. From them is formed an embryo with vibrating cilia and six hooklets. Pike and other fish swallow these embryos, which develop into cysticerci in the muscles, peritoneum, and solid viscera. The eating of measly fish, raw or partially cooked, thus favors the development of this tape-worm in the human intestine.

Symptoms.—Tape-worms may develop in man at any period of life. D. J. Milton Miller met with one in a child a few months old who had been fed on expressed beef-juice. Contrary to what has been supposed in days gone by, there are no absolutely diagnostic symptoms of the presence of tape-worm that can be relied upon. Indeed, the existence of a tape-worm in the bowel may not be suspected even because of the total absence of indicative, subjective sensations. On the other hand, teniæ may cause considerable local distress and impairment of the general health. Because of this fact a knowledge of the existence of tape-worm in certain neurotic subjects leads to an inordinate description of symptoms that exist mainly in the workings of a morbid imagination.

Alimentary symptoms of tape-worm may be as follows: anorexia alternating with a voracious appetite, constipation alternating with diarrhea, colicky pains in the abdomen, indigestion, nausea, and vomiting. Certain foods (herring, garlic, sour foods) increase the colic-like pains, others decrease them, as milk, eggs, and oils.

General symptoms of the teniæ may be added, as lassitude, inappetence, mental uneasiness, worry and irritability, depression of spirits, some physical prostration, and even emaciation. Various *reflex symptoms*, such as pruritus of the nose and anus, vertigo, migrain, tinnitus aurium, palpitation, visual disturbances (even temporary amaurosis), unequally dilated pupils, chorea, and epileptiform convulsions have been attributed to these parasites. But, on careful inquiry, adequate causes for some of these symptoms may be found in other associated morbid conditions. The bothriocephalus, however, may cause anemia, often very grave, even fatal. The blood-picture, in fact, is identical with that of pernicious anemia, as Schaumann's study of 38 cases has shown, and as was the case in three Finnish sailors seen by W. E. Robertson. This, in all likelihood, is due to some toxin elaborated by the worm. The blood-findings are otherwise unique among the verminous parasitic diseases in that they are the only class in which eosinophilia does not occur.

Diagnosis.—This is always to be made by the discovery of tenia segments or ova in the underclothing or stools. The doubtful presence of suspected tape-worm may be cleared by the administration of a suitable purgative, which will usually suffice to bring away portions of the worm in the dejections. I would here add a special warning lest mucous casts or shreds or vegetable structures (as of onion) be mistaken for tape-worm.

The diagnosis of the variety of the tape-worm is made by a careful scrutiny of the segments. Those of the tenia saginata are larger and fatter than, and their generative apparatus is unlike that, of tenia solium.

Hypochondriasis can be excluded by repeated examinations of the

stools, especially after the exhibition of cathartics, and by the uniform failure to detect portions of tape-worm or tenia eggs.

Prognosis is favorable. Indeed, teniæ saginata may exist at all ages and for years without any danger to the patient. Tenia solium, however, is attended with danger on account of the possibility of its causing cysticercosis.

Treatment.—*Prophglaxis.*—The way to avoid acquiring a tape-worm is to use none but well-cooked meats; this applies to beef and pork in particular. The use of pure drinking-water is of no little importance also. The proglottides of the tenia should always be burned, and not thrown where they may be taken into the bodies of other animals, as the cow or hog, and then be allowed to propagate. Governmental inspection of the meat-supply in abattoirs should be rigidly enforced.

Curative.—Before administering the chosen anthelmintic, the patient needs to undergo a "preparatory treatment." This has for its object the starvation of the parasite, so as to weaken its hold upon the intestinal mucosa. This is specially necessary in the case of tænia solium, in which the cephalic hooklets are obstinately and firmly fixed to the membrane, and since a cure cannot be said to have been effected unless the head be dislodged with the dejecta. For about two days prior to giving the remedy the patient should be restricted in diet to milk, light soups, a little white bread, and the like. Meanwhile, the bowels should be purged gently once or twice, after a simple enema.

In the evening preceding the day on which the drug is to be exhibited, a saline cathartic should be given to empty the bowel as completely as possible. The following morning no breakfast should be allowed, and before noon the selected anthelmintic should be administered. If the worm does not come away in a few hours, and an intense sense of pressure is felt in the abdomen, a brisk purge is indicated. The worm should be passed into a bowl containing warm water.

There are several very efficacious anthelmintic drugs to choose from. Prominent among them is male fern. Given to an adult in doses of ½ to 1 dram (2.0–4.0) of the ethereal extract, and followed in several hours by a calomel and a saline purge, it usually succeeds in bringing away the tenia. Schilling gives in the morning, fifteen minutes after a breakfast of coffee with Zweiback, this formula:

℞. Fresh ethereal extract of male fern, ℥ij;
Powdered jella, gr viiss;
Simple syrup, q. s. ad ℥j.
Sig. Shake well and take at one dose.

If evacuation of the bowels be delayed, an enema of warm water is indicated. Another valuable remedy is pelletierin, the active principle of pomegranate; the tannate may be prescribed, dose 1 to 1.5 gm., in capsules; or, a decoction of the pomegranate bark may be used, in combination with male fern, as in the Leipsic formula (Strümpell):

℞. Granati radicis corticis, ℥iv–v (128.0–160.0);
Aquæ, Oij (1 liter).
Mix and macerate for twenty-four hours,
and boil until reduced to f℥v (148.0).
Add: Oleoresinæ aspidii, ℥j (4.0).
Sig. To be taken in three or four doses, at short intervals.

Pepo in emulsion or in a sugary paste (about two ounces—64.0—and deprived of the envelopes) is at once a useful and harmless remedy.

Another effective vermifuge is kousso (Brayera anthelmintica). An infusion of half an ounce (16.0) of the flowers to one pint of water and mucilage of acacia is made, a wineglassful of which may be taken every half hour. The Germans recommend sometimes the agreeable, though more expensive, Rosenthal's "kousso tablets." Enough of these to make 15 grains (0.972) may be taken within one hour, with *café noir* or lemonade. Koussin (the active principle) in doses of 30 to 40 grains (1.94–2.592) has also been recommended, but should not be given to pregnant women, as abortion may be produced. Among other remedies of value as vermifuges may be mentioned kamala (1 to 3 drams—4.0–12.0—of the powder and hairs, in wine or water), oil of turpentine ($\frac{1}{2}$ to 2 ounces—16.0–64.0—in emulsion or milk), and thymol. The combined use of such drastics as croton oil renders the action of the anthelmintic drug more certain at times.

Although the head of the tenia may not be detected in the stools along with the body of the worm (and such is usually the case), a cure usually follows nevertheless, since, on account of its smallness, it may easily escape notice, and also from the fact that the head often dies and thus loses its hold upon the membrane, being carried away with the feces. On the other hand, if after the lapse of several months from the removal of a tape-worm, segments again appear in the stools, it may be inferred that the head was not dislodged or that another worm has developed. In cases where the tenia seems to redevelop with remarkable frequency and obstinacy it may happen that the head and neck are well protected beneath one of the valvulæ conniventes.

After the removal of the tape-worm—a weakening procedure, as a rule—the condition calls for supportive measures. The diet should not be too heavy for a time, but nutritious and easily digestible.

TÆNIA NANA.

This is the smallest tape-worm in man (v. Siebold). It varies from 8 to 20 mm. ($\frac{1}{3}$–$\frac{4}{5}$ in.) in length and from 0.5 to 0.7 mm. ($\frac{1}{50}$ in.) in width. The head has four suckers, a rostellum, and hooklets. The segments are yellowish, short, and broad. It is more common than is supposed. It is believed by some observers that, occurring in children, as it commonly does, this parasite is the cause of *epileptiform convulsions* and *enuresis nocturna*. Thousands of worms may be found within a cubic centimeter of fecal matter. *Hymenolepis nana fraterna*, which develops in rats without intermediate host, is regarded as being identical with the T. nana. Persons infected should occupy separate beds until cured. Male fern is the only remedy which has thus far been useful in expelling this worm (Stiles).

TÆNIA FLAVOPUNCTATA.

(*Tænia Diminuta; Tænia Leptocephalata.*)

Tænia diminuta is a very small cestode, 20 to 60 mm. ($\frac{4}{5}$–$2\frac{1}{2}$ in.) in length, with a small club-shaped head and nearly a thousand segments. The cysticerci inhabit such insects as the *asopia familiasis* (caterpillar and cocoon); the *anisolabis annuli* (belonging to the orthoptera); and the coleoptera *axis spinosa* and *scaurus striatus*. Man has been infested a

number of times, probably by taking food containing these infested insects.

Tænia Madagascariensis and *Tænia serrata* are other forms rarely found in man.

NEMATODES.

Helminthologists include in this class the cylindric worms, certain varieties of which are among the most common entozoa that infest the human body and inhabit the intestines.

ASCARIASIS.

Ascaris Lumbricoides (*Round-worm*).—**Natural History.**—This species resembles the common earth-worm, and is the most frequent in occurrence of all the parasites. It usually appears in children between the ages of three and ten years. The round-worm inhabits the upper portion of the small intestine, and occurs singly or in numbers. Its body is round, fusiform, and marked with fine transverse striæ. It has a yellowish or reddish-brown color, and measures in the female from 7 to 14 inches in length (17.5–35 cm.), and from 4 to 8 inches in the male (about 20 cm.), its thickness being about that of an ordinary goose-quill. The cephalic extremity of the worm has three oval papillæ, furnished with fine teeth; the caudal extremity is straight in the female and curved in the male.

Lumbricoid worms develop from ova, which are about .05 to .06 mm. long, elliptic, dark-reddish in color, and have a thick, resisting envelope; they occur in the feces. The eggs obtain entrance into the human intestine most probably through drinking-water and food.

The round-worm sometimes, though rarely, migrates from the small intestine. It has been vomited, and it has also crawled into the pharynx, mouth, and nares, and has been withdrawn thence by the patient's fingers. It has even passed into the larynx and trachea, causing fatal asphyxia or pulmonary gangrene. The Eustachian tube and biliary ducts may be invaded with such serious symptoms as perforation of the membranum tympani and hepatic abscess.

Symptoms may be absent, and yet the worms be found repeatedly in the stools. Existing symptoms are indefinite, and point simply to an irritative condition of the bowels. Some writers ascribe them to toxins elaborated by the worms. Serious symptoms may, however, result from the migration of the worm, as into the biliary passages, Eustachian tube, or larynx. Fever is not a necessary concomitant. Lumbricoid worms may give rise to any or all of the following symptoms: colicky pains, nausea, vomiting, indigestion, diarrhea (sometimes), restlessness, irritability, anorexia, itching of and picking at the nose, disturbed sleep with grinding of the teeth, salivation, and nervous twitchings. Very nervous children may manifest epileptiform convulsions, choreic movements, dilated pupils, vertigo, cephalalgia, mental disturbances, and even contractures.

Complications.—The development of jaundice will indicate obstruction of the bile-duct, in cases in which the worms have been found in the feces. Intestinal obstruction from coiled worms has occurred. So also, suffocative symptoms coming on, especially at night, in a child with worms, may be due to a migrating lumbricoid. Perineal abscesses

and inflamed herniæ that have perforated externally sometimes discharge the ascaris lumbricoides.

Diagnosis.—This is positively determined only by discovering the worms or ova in the stools.

The **prognosis** is good, unless serious complications arise (*vide supra*), when it should be guarded accordingly.

Treatment.—*Prophylaxis.*—The water used for drinking purposes should be obtained from the purest sources.

Before giving an anthelmintic, it should be borne in mind that no good result can be certainly obtained unless the gastro-intestinal tract be nearly deprived of food for from twelve to thirty-six hours, so that the toxic action of the drug used may be exerted directly upon the unprotected worm.

Santonin is at once the most efficient and the most easily administered remedy. It may be given in doses of gr. ¼ to 1 (0.0162–0.0648) of the crystals to a child, or from gr. ij to iv (0.1296–0.2592) to an adult, in the form of a troche, before breakfast. A little milk or other light nourishment may be allowed, the troches being continued once or twice daily for two or three days. This treatment is to be followed by a brisk purge, preferably gr. j to iij (0.0648–0.1944) of calomel. I have sometimes combined small doses of calomel with the santonin in a troche, and with good effect. Xanthopsia or yellow vision, spasms, and even convulsions, and saffron-colored urine may follow the use of santonin in cases of idiosyncrasy or overdose of the drug. Oil of wormseed (chenopodium) in doses of five to ten drops, in emulsion, capsules, or on sugar, may also be used with benefit. Another favorite remedy with some is the unofficial fluid extract of spigelia and senna, to be given in from 1- to 3-dram (4.0–12.0) doses. Finally, the fluid extract of spigelia alone (1 to 2 drams—4.0–8.0), followed by a brisk purge, may bring away dead worms.

Oxyuris Vermicularis (*Seat-, Pin-, Thread-, or Maw-worm*).—**Natural History.**—The *ascaris vermicularis*, as this worm is also called, inhabits the colon and especially the rectum. It is a small worm, as several of the commonly-used terms signify, and frequently it occurs in great numbers, sometimes agglutinated with mucus into feculent balls. It is most common in children, though found not rarely at any period of life. The female oxyuris is whitish in color and about ten or twelve millimeters (one-half inch) long, the male being about three or four millimeters (about one-sixth of an inch) in length. Oxyures develop from ova in about two weeks after the ingestion of the latter. The eggs are irregularly ovoid, about $\frac{1}{500}$ in. (0.05 mm.) in length, and tenacious of life. By the time the embryos have reached the cecum, they are sexually mature, and when the female arrives in the rectum, immense numbers of eggs are deposited that mature into great numbers of worms, the latter being discharged with the feces. Sometimes the worms crawl out of the anus.

Infection with the ova may take place through water and food (green, uncooked vegetables and fruit) that have come in contact with the hands of infected persons. Scratching the anus will permit of the reception of oxyuris eggs under the finger-nails (Zenker and Heller), and in careless, ignorant, and uncleanly persons the possibility of such an auto- or re-infection should be recognized and avoided.

Symptoms.—*Pruritus ani* (itching of the anus), sometimes burning pain, and tenesmus, with restlessness and disturbed sleep, are the commonest symptoms of the presence of this parasite. The itching is always worse at night, and may be paroxysmal. An herpetic or eczematous eruption around the anus should arouse suspicion, particularly in children, of the presence of the oxyuris in the rectum, and it accounts for the intense itching. Anorexia and anemia, rectal irritability, and "nervousness" may be associated. It is believed that the migration of the worms into the vagina may excite vulvo-vaginitis, pruritis, and leukorrhea, and that habits of masturbation may be induced in both girls and boys by the sexual irritation caused by the worm. Inspection of the stools will reveal, in positive cases, the whitish, thread-like parasites.

Diagnosis.—The pruritus, indicating rectal trouble, will direct the physician's attention to the anus, where the oxyures may be seen; if not found, their discovery in the feces or the discovery of the eggs by microscopic examination will suffice.

The **prognosis** is good, and proper treatment is always effective, though occasionally exceedingly refractory cases are encountered.

Treatment.—The exhibition of anthelmintics and purgatives, such as recommended for destroying and removing the lumbricoid worm, may be effective against seat-worms also, in reaching those lodged in the bowel above the rectum. C. W. Stiles[1] states that the adult worm lives in the small intestine and should be driven into the large intestine by an anthelmintic before local injections are given. Ashford recommends betanaphtol in 2-dram doses. The larval forms may be killed by methylene-blue in pills—18 to 24 one-grain pills daily for five days. This treatment is to be taken three times, ten days apart. Attacking the oxyures directly, however, by means of enemata is rational treatment.

The rectum should be well emptied of feces, so that the worms may be exposed to the action of the medicament injected, and for this purpose enemata of cold water, either simple or with salt or soap, may be resorted to. Injections containing the decoction of quassia (1 or 2 ounces—32.0 to 64.0—of the powder or chips to the pint—½ liter—of water) are nearly always curative. Other useful remedies are carbolic acid, turpentine, tannin, vinegar, camphor, potassium sulphid, and the oil of eucalyptus. The injections should be repeated once or twice daily for at least ten days.

Rectal irritation may be allayed by injections of laudanum and starch-water (gtt. iij–v to the ounce—32.0). Anal itching is often amenable to carbolized vaselin, at bed-time, or to belladonna ointment.

Ascaris Alata.—This is another name for the *ascaris mystax*, a species of worm found in the intestines of the dog and cat, and occasionally in man. It is a slender worm, with a closely-rolled spiral tail and a wing-like projection on either side of the head. The female is about 6–7 centimeters (2.7 inches), the male about 4 centimeters (1.75 in.) in length. Scarcely ten instances, however, have been recorded in which this parasite has occurred in man.

Trichocephalus Dispar (*Ascaris trichiura*).—**Natural History.**—This so-called whip worm measures about four or five centimeters (2 inches) in length, and is characterized by the very slender, hair-like appearance of the anterior two-thirds of its body, in contrast to the

[1] "Proceedings of the Amer. Soc. of Tropical Medicine," *N. Y. Med. Jour.*, Apr. 18, 1908.

thick posterior portion, which is more or less straight and blunt-pointed in the female, but rolled into a spiral in the male. Its particular habitat seems to be the cecum, though sometimes it is also found in the colon. It may exist in great numbers. Europeans appear to be infected with the parasite more commonly than Americans.

Propagation is effected by the microscopic eggs, which are ovoid, hard, nodular, brownish, and about 0.05 mm. ($\frac{1}{500}$ in.) in length.

Symptoms.—It is not certain that the parasite causes any symptoms.

The diagnosis is made by detecting the microscopic ova in the feces.

The **prognosis** and **treatment** are not called for.

UNCINARIASIS.

(*Ankylostomiasis; Hookworm Disease.*)

Ankylostomum Duodenale (*Dochmius Duodenalis*).—**Natural History.**—This parasite belongs to the family of *strongylidæ* of the nematoid worms. It was discovered in Milan, in 1838, by Dubini. The length of the female is from 8 to 18 mm. ($\frac{1}{2}$ inch), and of the male from 6 to 10 mm. ($\frac{1}{3}$ inch). Its body is thread-like, with a conical-shaped head, and a large, bell-shaped mouth surrounded by a horny capsule, and possessing four hook-like teeth, ventrally situated, and two smaller, vertical teeth on the dorsal side, by which the worm fixes itself to the mucous membrane. A bulbous-like swelling exists at the tail end of the male worm. It inhabits the jejunum and duodenum. The eggs are found in muddy water, or in warm moist earth, and there liberate the embryos. These develop into larvæ, which soon enter the dormant state, remaining quiescent for an indefinite period until they are taken into the human stomach through drinking-water, food, dirt ("dirt-eaters"), or, more commonly, dirt that has collected upon the hands and about the nails. Probably direct infection through the skin, as first shown by Loos, is the usual mode of infection, however, and Allen J. Smith and others have regarded the subtropical dermatitis known as "ground itch" as an expression of this mode of infection. Loos has shown that on the completion of the exogenous phase of the embryo, the parasite enters through the skin, generally of the feet and legs, by contact with soil contaminated with the ova of the ankylostoma. Carried by the blood stream to the lungs it passes into the air vesicles, then into a bronchus, to the trachea, esophagus, and stomach, and finally to the small intestine. Here sexual characters develop in the parasites, reproduction ensues, and the ova are deposited in the bowel. They do not multiply within the intestine.

Predisposing Causes.—(*a*) *Geographical Distribution.*—The parasite is found in Italy, Egypt, India, Philippines, Germany, Belgium, Switzerland, and in England was found by Haldane in miners in Cornwall. B. K. Ashford[1] (U. S. Army) has shown that a large percentage of all cases of anemia occurring in Porto Rico are induced by this parasite. H. F. Harris has found the ankylostoma prevalent along the Gulf of Mexico and in the southeastern section of the United States. The Rockefeller Sanitary Commission, after a survey made to determine the degree and extent of hookworm infection, showed that hookworm disease belts the earth in a zone about 66 degrees wide, extending from parallel 36 north to parallel 30 south latitude. Not less than 58 per cent. of the earth's estimated population is in the infected area. The importation of

[1] *New York Medical Jour.*, April 14, 1900.

infected Italian, Hungarian, and Polish laborers may be accountable for the propagation of the parasite in the United States. Stiles has studied the parasite of American origin correlatively with that known to foreign observers, and suggests the term *Necator Americanus*. (*b*) *Sex.*—Males and females are infected to the same extent. (*c*) *Age.*—The greatest infection occurs between the age of six and sixteen years (Wells).

Pathology.—The ankylostomum is probably nourished by the plasma of the blood it sucks from the intestinal vessels, though this is denied by Loos. It is found *postmortem*, sometimes, in the mucous or even submucous coat, rolled up in a little blood-cavity. Ecchymoses, containing a central opening through which blood can ooze, are the usual result of the worm's action. Chronic catarrhal enteritis is usually associated. Hypertrophic dilatation of the heart is observed.

Symptoms.—The chief symptom of the condition is progressive anemia (secondary), and the skin is a pasty yellow or dirty gray color, called in the southern part of the United States "Florida complexion." When the number of ankylostoma embryos introduced into the intestine is large, the anemia may develop acutely; when but a few are introduced, the withdrawal of blood is more gradual, and *chronic anemia* develops. I think, however, it may be safely affirmed that the anemia is not wholly due to blood-sucking, but that some poisonous substance is given off by the worm, of the nature of a hemolytic toxin. The impoverishment of the blood has been so profound as to simulate a pernicious anemia.

Ashford found the red cells to vary between 700,000 and 3,525,000 per c.mm., and the hemoglobin between 10 and 55 per cent. *Leukocytosis* is not a feature of uncomplicated cases; the polymorphonuclear cells may show slight reduction, and the lymphocytes a moderate increase. Eosinophilia is common in this as in many parasitic diseases, and may reach 40 per cent. or more. In mild cases, however, eosinophilia may not be available for diagnosis, in which case the feces should be examined for eggs. The red cells are pale, of irregular size and outline; normoblasts are plentiful, and less often megaloblasts are found. This parasite is the cause of "Egyptian chlorosis," first described by Griesinger. Ankylostomiasis is not uncommon in tropical countries. In Italy it has been termed *tunnel* or *mountain anemia;* in Belgium it is known as *brickmaker's anemia;* again, it occurs among workers in coal mines—*miner's cachexia.*

There may be, in addition, slight gastro-intestinal disorder (anorexia, colicky pains, nausea and vomiting, and constipation alternating with diarrhea). In cases marked by an acute development of anemia considerable general weakness, dyspnea, and dropsy may ensue. There is no loss in weight; but swelling of the feet and ankles, sleeplessness, headache, faintness, palpitation, and scanty perspiration are common symptoms. The renal function is maintained and slight fever may develop. Corneal ulcer is not uncommon. Lemann[1] describes infantilism in uncinariasis.

Physical Signs.—The areas of the apical cardiac impulse and of cardiac dulness are increased. Various murmurs—hemic—may be heard, and palpitation and dyspnea are common. In those affected the face is peculiarly dull, expressionless, and, owing to the marked metabolic disturbance, the growth of young subjects is greatly hindered.

Diagnosis.—This is made by finding the eggs or mature worms in the feces. The former are oval-shaped, about 0.05 mm. ($\frac{1}{500}$ in.) in length,

[1] *Archives of Internal Medicine*, Chicago, August, 1910.

and have a much thinner shell than the ova of the round worm. Ova are seen with a $\frac{1}{6}$ objective and are commonly entangled in the mucus that escapes with the feces. Suspicious specimens, if negative, should be centrifuged, Bass'[1] method being preferable for the purpose. "If in doubt as to the diagnosis of the eggs, they may be hatched out in twenty-four to forty-eight hours and the characteristic larvæ looked for" (Dock and Bass). In cases of pronounced anemia, in which the cause is obscure, the patient's dejections should be carefully examined.

Duration.—The disease may last for months or for several years.

Prognosis.—If left untreated, the affection may end fatally. Intense anemia, obstinate diarrhea, and profound nutritive disturbances constitute symptoms of grave import. Properly treated, the prognosis is favorable, although the subject remains a carrier.

Treatment.—*Prophylactic.*—Workmen in mines, tunnels, and brick-yards, and in tropical localities especially, should be warned not to drink the water close at hand without previous boiling and then cooling. Stools infected with ankylostoma ova should be carefully disposed of and efforts at prevention of further pollution of the soil be made. The feet, legs, and hands should be protected against contamination with infected soil.

Medicinal.—Anthelmintics to kill the ankylostoma and purgatives to remove it from the intestine are indicated. The Permanent Commission for the Suppression of Uncinariasis in Porto Rico employ repeated doses of thymol and betanaphtol, preceded and followed by a saline. Brauch[2] administers 30 gr. (2.0 gm.) of thymol in powder at 4, 6, 8, and 10 A. M., on an empty stomach, followed by an ounce of castor oil at 6 P. M. The State Board of Health of Florida recommends the following dosage: Under 5 years of age, up to 8 grains; 5 to 10 years of age, 8 to 15 grains; 10 to 15 years of age, 15 to 30 grains; 15 to 20 years of age, 30 to 45 grains; 20 to 60 years of age, 45 to 60 grains; over 60 years of age, 45 grains. The condition of the heart, the degree of debility and anemia should also be considered. Manson sounds a note of warning concerning the use of thymol. He says alcohol should never be given at the same time nor for some hours after a dose of thymol, as the drug is soluble in alcohol and may then exercise its toxic action on the host as well as on the parasite. After this routine, nourishing food, fresh air, iron, and tonics are to be given.

TRICHINIASIS.

(*Trichinosis.*)

The parasite that gives rise to this affection is *trichina spiralis.*

Natural History.—The mature male worm is 0.8 to 1.5 mm. ($\frac{1}{20}$ in.) long and the female 2 to 4 mm. ($\frac{1}{12}$–$\frac{1}{6}$ in.). The head is pointed and unarmed, and the neck is long and more slender than the body, which has a round blunt end. The worm is viviparous. It inhabits the intestines of such animals as the rat, dog, cat, hog, and man.

The embryo or muscle trichina is about 0.6 to 1 mm. ($\frac{1}{25}$ in.) long, and lies coiled up in a spiral form within an ovoid capsule in the sarcolemma-sheath of muscle-fiber. The life-history begins with the larval state of the trichinæ encysted in the muscles. When this flesh is eaten by another animal, or by man, the larvæ are liberated during the digestive process. Passing into the intestines, they reach the adult stage in from two to four

[1] "Hookworm Disease," Dock and Bass, pp. 175, 176. [2] *Brit. Med. Jour.*, March 5, 1904.

days, being then sexually mature, and in five to seven days more they produce hundreds of living embryos.

The intestinal trichinæ become fully grown, and then usually die in from four to five weeks. The female trichina may bring forth several broods of embryos during her life-period in the intestine. The living embryos leave the intestine at once, and invade the muscles through various channels—principally along the connective-tissue routes—so that the symptoms of muscular irritation develop in from seven to ten days after eating the trichinous meat. The embryos attain to maturity (larval form) in about two weeks after entering the muscular tissues. Their presence causes a mechanical irritation that results in the formation of a fibrous capsule in from four to six weeks. In man it probably becomes encysted at a later period than in the lower animals, as shown by the accompanying illustration, taken from a case under the immediate observation of Dr. L. Napoleon Boston (Fig. 27). Usually but a single worm is found within one capsule, though occasionally three or four are seen. The encapsulated trichinæ may live many years in the muscles. With increasing age the capsules become thicker and may be the seat finally of calcareous infiltration. In the hog, calcareous infiltration of the capsule is the exception, hence the difficulty, even impossibility of seeing them with the unaided eye.

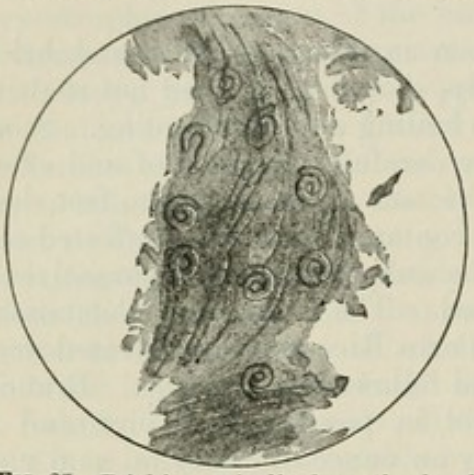


FIG. 27.—Trichina spiralis from the head of the right gastrocnemius muscle three weeks after the first symptoms appeared (Queen obj. ⅔; eye piece No. II).

Pathology.—The diaphragm is most thickly infested with the larval trichinæ. Next in order are such trunk-muscles as the intercostals and abdominals, then the muscles of the neck, including the larynx, head, eyes, and extremities. Up to the seventh week of the disease the intestinal trichinæ may be very numerous. *Microscopically*, the muscles show "the changes characteristic of acute myositis" (Fitz) after the fifth week. The trichinous cysts in the muscles may be seen with the naked eye as small, grayish-white, opaque, "oat-shaped" specks, longitudinally disposed in the meat-fibers.

Sources of the Trichina.—The trichina was first found in pork—the usual source of trichiniasis in man—by the late Joseph Leidy. Recent investigations show that the live trichinæ may be found in the fatty as well as the fleshy portion of pork. The pig is infested by eating trichinous rats, trichinous pork, or human or porcine excrement containing the embryos of propagating intestinal trichinæ. The rat may be the original host of the parasites, or it may itself become infected by older rodents eating their fellows, or by eating trichinous pork or human or porcine excrement.

As to the frequency of the infection of hogs, it may be said that about 2 per cent. were found to be trichinous, according to Salmon's report (1884), of nearly 300,000 examinations of American pork. In Prussia, according to Eulenberg's statistics, the ratio is decidedly less varying—from 1 to 2160 hogs (1876) to 1 to 1817 (1889). "The dissecting-room

and *postmortem* statistics show that from ½ to 2 per cent. of all bodies contain trichinæ" (Osler).

Of course, man, as a rule, becomes infected by eating raw or partially cooked pork containing living muscle-trichinæ (larvæ). Eating raw ham and sausages, a habit common among the Germans of Prussia (particularly during picnics), and in some parts of the United States where German emigrants have settled in large numbers, explains the comparative frequency of this disease in such localities. About 900 cases have been reported in the United States in the past forty-five years (Beecher). Trichiniasis has occurred in epidemic form in North Germany, France, Spain, Russia, the Scandinavian countries, and in several of the northwestern United States.

Symptoms.—*Postmortem* examination often reveals the presence of unsuspected muscle-trichinæ.

In well-marked cases of infection *gastro-intestinal disturbances* appear on the second or third day after the ingestion of the affected meat. Vomiting, diarrhea, and colicky pains in the abdomen may be present. The diarrhea sometimes takes on the characteristics of a choleraic attack or may be followed by obstinate constipation.

Extreme "muscular weariness" and bodily fatigue often occur for several days before the embryonic parasites can have begun to wander into the muscles. On about the tenth to the fifteenth day, when migration usually commences, *chills*, followed by a temperature of 101.5° to 104° (38.6° to 40° C.) and marked *myositis*, come on. The muscles are stiff, tense, painful on pressure and motion, and somewhat swollen. The flexors of the extremities are particularly sore and often firmly contracted, causing the knees and elbows to be acutely bent. *Mastication*, *deglutition*, and *phonation* may be difficult and painful because of the involvement of the muscles of the jaws, pharynx, larynx, and tongue. Intense *dyspnea* is frequent on account of the involvement of the diaphragm and intestinal muscles. The *temperature* shows marked remissions in most cases, and may even be subnormal. The *pulse* varies with the temperature.

Edema is characteristic in nearly all of the cases. It appears on about the seventh day after the infection, and begins in the face (frontal region), usually being noted first in the eyelids, and extending thence to the extremities and trunk during the height of the muscular symptoms. It may last for several days, then disappear for several days or a week, and reappear. Ascites even has been observed. Œdema of the larynx and bronchial catarrh, the latter rarely leading to broncho-pneumonia, may also supervene and add to the gravity of the dyspnea. Profuse sweating may last for several weeks. Miliaria, urticaria, acne, furunculosis, herpes, and pruritus may occur as skin-manifestations. Insomnia, headache, a temporary loss of the tendon-reflexes, and dilatation of the pupils (Rupprecht) have been noted among the nervous symptoms. Prolonged cases show a marked degree of emaciation and anemia. T. R. Brown[1] found a decided increase of the eosinophiles in the blood, amounting to 37 per cent. This discovery has been confirmed. W. T. Howard, Jr.,[2] noted eosinophiles in the muscle-lesions, but failed to find an increase of these cells in the circulating blood. Opie[3] administered trichina spiralis to the guinea-pig, and found that a resulting mild infection

[1] *Johns Hopkins Hospital Bulletin*, 1897, vol. viii.
[2] *Phila. Med. Jour.*, December 2, 1899. [3] *Amer. Jour. Med. Sci.*, March, 1904.

stimulates the eosinophile cells to active multiplication, but severe infection causes their destruction. A marked, absolute leukocytosis is the rule.

There is little doubt that the "muscle symptoms," varying with the site of the muscle attacked, can be explained on the basis of a reactionary inflammation, a true myositis existing about the parasitic cysts as Brown has shown, but the general symptoms are probably—in part at least—due to some toxic emanation from the parasite itself.

Complications, as a typhoid state, hypostatic pneumonia, and pleurisy may appear. Albumin, with casts, are found in the urine.

Recovery is effected in mild cases within two weeks, while in the severe ones from six weeks to several months may be occupied.

Diagnosis.—The following symptoms are regarded as pathognomonic: sudden swelling of the face, coming on after the patient has suffered for several days from muscular soreness; loss of appetite, fever, and profuse sweats (Böhler); painful, tender, and "rubber-like" hardness of the muscles, with difficulty in movement; semiflexed extremities; gastro-intestinal catarrh, with a red, dry, coated tongue; dyspnea, diarrhea, and edema of the extremities following the subsidence of that first noticed in the face. Friedreich emphasizes hoarseness, and the late Dr. Packard rapidity of respiration without evident cause.

Differential Diagnosis.—*Meat-* and *sausage-poisoning* may be distinguished from trichiniasis by the more rapid course of the former, the dry throat and skin, jaundice, visual disturbances, and the absence of edema and muscular symptoms.

Direct examination of the passages and of the muscles may be resorted to. The discovery of the parasites in the pork a portion of which has been eaten by the sick establishes the diagnosis. A low-power microscope should be used to examine the intestinal mucus for the trichinæ. Light purgation should precede this endeavor. Harpooning such muscles as the biceps for the purpose of removing some muscle-fiber, or directly incising a small portion under Schleich's method of infiltration-anesthesia, may permit of a positive diagnosis.

Acute rheumatism, *cholera*, *typhoid fever*, and *acute polymyositis* (*pseudo-trichiniasis*) may at times resemble trichiniasis. Epidemics of the parasitic disease are more readily diagnosed than an isolated case.

Prognosis.—This depends upon the number of parasites ingested and upon the number of embryos generated in the intestines. Marked early diarrhea is favorable. The prognosis should be guarded, as death may occur as late as from the fourth to the sixth week. Of 357 cases collected by Packard, the mortality was 24.07 per cent.

Treatment.—Prophylaxis is of supreme importance, both as to the infection of the hog and the danger of eating infected pork. Care should be exercised in the feeding of swine, and the destruction of rats should be made as complete as possible in and about the styes. Pig-excrement should be removed and burned, and feeding with milk, bran, grain, and vegetables should be forced upon all keepers of swine.

Rigid inspection of the meat-supply, as is done in Germany, should be carried out by government sanitary officers. Decidedly the safest and most efficient way to prevent trichinosis is to thoroughly salt, smoke, and cook the pork that is to be used. Putrefaction does not kill the parasites.

The treatment of those who have eaten trichinous meat should be by a prompt evacuation of the bowel, especially within the first twenty-four

hours, as after the embryo young have been brought forth and have passed into the muscles no known treatment is successful in attacking them. Calomel is one of the best drugs, and active purgation usually follows its use in large doses, succeeded by salines; rhubarb, senna, sulphur, aloin, and large doses of oil or glycerin may also be tried. In combination with the purgatives some anthelmintic (male fern, santonin, thymol) should be used. The encysted or larval parasites are not accessible to treatment, although picric acid has been recommended. The symptoms to be met are the great muscular pains, insomnia, and weakness, which is often severe in protracted cases. Prolonged hot baths.

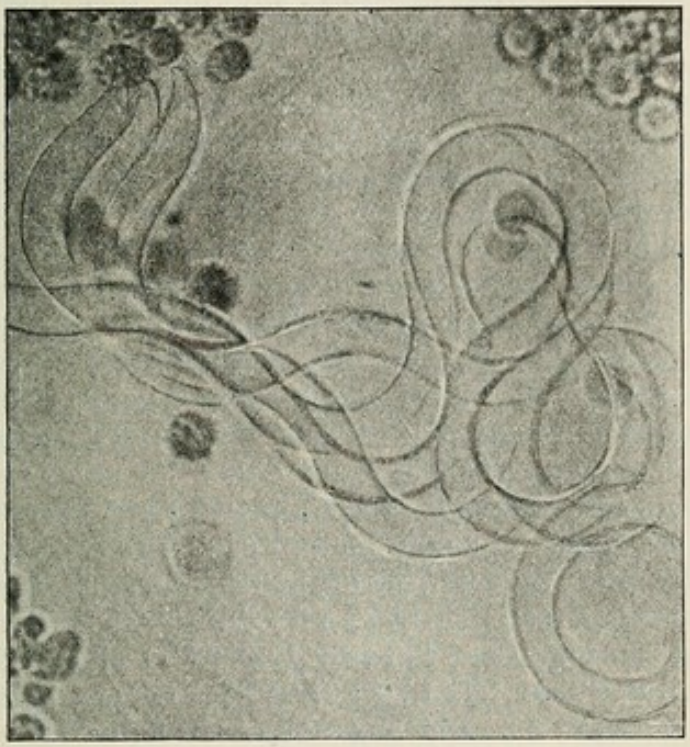


FIG. 28.—The movement of a single filaria during a series of four successive instantaneous exposures. The length of each exposure was one-fifth of a second, the entire series occupying less than five seconds. The magnification is to eight hundred diameters, with a Zeiss one-twelfth homogeneous immersion lens (F. P. Henry).



FIG. 29.—Filaria alive in the blood. Instantaneous photomicrograph. Four hundred diameters magnification. Four millimeters Zeiss apochromatic (F. P. Henry).

anodyne embrocations, with hypodermics occasionally, may prove useful for the first; bromids, chloralamid, and the like for the second symptom; and a concentrated liquid diet, strychnin, peptonoids, and the like for the last. Massage, electricity, and stimulating applications, as chloroform liniment, may be required during convalescence and for some time thereafter to combat the muscular weakness, soreness, and stiffness.

FILARIASIS.

(*Filaria Sanguinis Hominis.*)

There are several varieties of filariæ that may be found in human blood. The two principal ones are the *filaria sanguinis hominis nocturna* and *filaria sanguinis hominis diurna*. But two other distinct species of nematode worms infest the blood of tropical man according to Manson. These are *filaria perstans* and *filaria demarquai*. Of these various forms the *filaria nocturna* is the most important and the best known. The adult forms, male and female, found only in the lymphatics, are

called *filaria Bancrofti;* the embryos, which alone are found in the circulating blood, are termed filaria nocturna. The first is a white, opaline thread-like worm, tapering toward the ends, which latter, however, are blunt. The male is 83 mm. (3.2 inches), the female 155 mm. (6.1 inches) long. The second worm is known only in embryonic form, and is distinguished by granulations in the axis of the body. This is the filaria diurna, of which the adult form is said to be the *filaria loa*, now known to be the cause of Calabar swellings. Manson found them in the blood of Congo negroes, but only during the daytime. On the other hand, the nocturnal filaria is found only at night, or, if the host be either by habit, necessity, or choice, a day sleeper, during this time, showing, then, that there is some condition of the body during quietude that is conducive to the appearance of the filaria in the blood (Granville). Manson observed that during the diurnal temporary absence of the filariæ from the cutaneous circulation they are found principally in the larger arteries and the lungs. This "filarial periodicity" is a striking characteristic. Inversion cannot be induced in cases of filaria diurna.

Filaria philippinensis is also worthy of mention. Ashburn and Craig conclude: (*a*) That the complete development of *Filaria philippinensis*, discovered by them in 1906, has been followed in the mosquito, *Culex fatigans;* (*b*) that the *Filaria philippinensis* is distinguishable from other filaria "both in the blood and during the developmental cycle within the mosquito"; (*c*) that as regards the time of its occurrence in the blood, the organism presents no periodicity.

The embryos of filaria sanguinis hominis are produced by the female in great numbers, and are so small that they readily pass through the capillaries. According to Manson, who, in 1877, found the larvæ in the stomach of a female mosquito, it is probable that after filling itself with the blood of an infested man during sleep, the mosquito seeks stagnant water, dies, and the larvæ are set free. In this way it may happen that man takes in the embryos through the drinking-water. More recently it has been shown that the filaria, once in the stomach of the mosquito, sheds its delicate envelope, then pierces the wall of the mosquito's stomach and lodges in the thoracic muscles. Here it undergoes further developmental changes during two weeks, then finds its way into the proboscis, to be discharged into the blood of the human host. They find a permanent seat in the lymphatics of the human host, mature, and bring forth young, which may again infest the blood by passing through the lymph-ducts into the thoracic duct and general circulation.

The geographic distribution of filariasis is limited mainly to the tropics and sub-tropics. It is most common in Brazil, the West Indies, Mexico, the Southern States, Southern China, India, Egypt, a part of Australia, and the southern Pacific islands, where it is endemic.

The **symptoms** of filariasis are in abeyance until some obstruction and inflammation of the lymph-channels is caused by the parasite. There are several conditions or endemic diseases produced. **Elephantiasis arabum** is believed by Manson to be the effect of these parasites, in a certain proportion of cases at least. In specimens of night-blood from 88 Cochin Chinese he found filariæ in 21; 14 specimens came from patients with elephantiasis, and only 1 showed filariæ. This latter fact, he explains, is to be expected, since, in order to give rise to elephantiasis (due to an infarction of the lymphatic glands connected with the diseased areas), the adult filariæ

must lie on the distal side of the glands, which makes it impossible for the young filariæ to pass into the general circulation. "Therefore the person least likely, in a filarial district, to have filariæ in his blood is one who is the subject of elephantiasis."[1] Embryos can be demonstrated in the blood as soon as the adult filariæ reach maturity and begin to discharge them, *i. e.*, before the usual obstructive symptoms appear (Rivas and Smith[2]).

Hematochyluria and Chyluria.—The patient passes a white, opaque, milky urine, occasionally bloody, with a clotty sediment. This may be intermittent, and normal urine may be passed for many weeks before chyluria or hematochyluria reappears. There may be at the same time a slight degree of polyuria. Under the microscope, fat granules and white and red corpuscles are seen. The lively, wriggling embryo filariæ may also be discovered in the urine, as well as in the blood at night. There is a dilatation of the lymph-vessels in the kidneys alongside of the tubules, and in the abdominal lymph-plexuses. Sometimes a little vesical irritation and straining during urination may be caused by the endeavor to pass chylous blood-clots. The thoracic duct above the diaphragm has been found impervious (Stephen Mackenzie).

Lymph-scrotum and lymph-vulva have been caused by the filariæ. The parts are greatly swollen, thickened, and contain distended lymphatics filled with a turbid and either milk-white, salmon-colored, or blood-red coagulable liquid that is discharged upon puncturing the varices. The filaria is not always found in the exuded lymph. The inguinal and femoral regions are often enlarged. An erysipelatous inflammation of the parts is not infrequent in these cases, and may be ushered in by a chill and high fever, lasting a day or two, and ending with a profuse sweat.

The filariæ have been found in ascites (Winckel), in hemoptysis, and in the feces (Yamane, Japan). Worms killed by blows or other injuries are often absorbed, but may act as an irritant and cause abscesses.

Treatment.—Healthy subjects must protect themselves against mosquito bites. Filtering, boiling, and storing the drinking water in mosquito-proof receptacles are important measures. Thymol, in from 1- to 5-grain (0.0648–0.324) doses, given for from two to eight weeks, has caused the disappearance of the larval filariæ in several cases. Methylene-blue appears also to have produced a cure in a case of chyluria reported by Flint. Henry, however, states that he has "given this drug in larger doses than were used in the case reported by Flint, and for a much longer period, without the slightest effect upon the parasite."[3] The adult filaria seems to be beyond the reach of any known medication that will not prove dangerous.

DRACONTIASIS.

(*Guinea-worm Disease.*)

The parasite is the *filaria* or *dracunculus medinensis* or *persarum*, common in the tropics of Asia, Africa, and America. It is usually solitary, and measures from 50 to 100 cm. (20 to 40 in.) in length and about 2 mm. ($\frac{1}{12}$ in.) in diameter. It is cylindric, whitish, with blunt papillated head, and a sharp, curved tail. The body is nearly filled by the uterus, which contains innumerable embryos, which, after maturation of the worm,

[1] *British Med. Jour.*, June 2, 1894. [2] *Southern Med. Jour.*, October, 1912.
[3] *Med. News*, May 2, 1896.

escape shortly after contact with water in the form of a milky fluid. The process of emptying the uterus takes from two to three weeks. This accomplished, the worm dies. It is then taken into the stomach and intestines of man through the contaminated drinking-water. The female enters the intestines by way of the mesentery and brings forth its young, which pass into the connective tissue of its human host. The male worm is unknown. The worm has an inexplicable affinity for the subcutaneous and intermuscular tissues of the feet and legs, where it attains full development.

Symptoms.—Wherever the parasite is situated, it may often be felt coiled up under the skin, which at that point becomes red and fluctuating like an abscess. When opened, either surgically or naturally by the worm, the head appears through the aperture. The favorite spot for perforation is the dorsum of the foot, though sometimes it extrudes from the legs, rarely the thighs, and very rarely from the thorax and abdomen.

Treatment.—Prophylaxis in regard to the drinking-water and as to bathing where the intermediary host of the dracunculus—the cyclops—has its habitat is essential for safety.

The treatment embraces the surgical measures necessary to remove the worm and to promote the healing of the irritated tissues. Roth claims that after incision the application of carbolic acid (1 : 15) causes the worm to be removed in two or three days. Native Indian physicians commend highly the local application of the leaves of the "amarpattee" plant.

OTHER FILARIÆ.

Among other filariæ that have been found in man are the following: The *filaria immitis*, which causes hematuria and has been found in the portal vein, whilst the ova were discovered in the ureteral and vesical walls; *filaria labialis*, found in a lip pustule; *filaria lentis*, found in a cataract; *filaria trachealis* and *bronchialis*, seen in the trachea, bronchioles, and lungs; *filaria hominis oris*, observed by Leidy in the mouth of a child; *filaria loa*, noticed in the tropics among negroes, its habitat being beneath the conjunctiva. Recently L. N. Boston found *filaria mermus* (according to Wardell Stiles, to whom he referred them) in a cavity in the centre of an apple. They are believed to be parasites of the apple worm, but whether pathogenic, is not known.

OTHER AND UNCOMMON NEMATODES.

Eustrongylus Gigas.—This parasite is exceedingly rare in man, but has been found in many of the carnivora and in some herbivora. It is supposed that fish act as the intermediary host for the larvæ. The worm is enormous in size, the female being from 25 to 100 cm. (10 to 40 in.) in length. It is a red, cylindric parasite with blunt-pointed ends. Its seat is the kidney, which it may destroy, causing hematuria and the presence of the eustrongylus ova. Dr. John McKenna has recovered an adult eustrongylus from a child whose urine had long contained ova.

Anguillula stercoralis or **strongyloides intestinalis** occurs in the stools of certain tropical endemic diarrheas. It is common along the Gulf of Mexico. The parasites are oviparous, and the eggs may be taken through the drinking-water. They have been found in the biliary and pancreatic ducts, as well as in various parts of the intestines. The administration of thymol or male-fern is to be recommended.

Echinorhynchus moniliformis occurs in rats, and in one case, that of a Sicilian, reported by Calandruccio, the ova were found in the feces.

PARASITIC ARACHNIDA.

Pentastoma Tenioides.—This parasite is an inhabitant of the nasal fossæ of the dog or horse, though it may also occur in man both in this and in the larval form. The ova are ejected during sneezing, and are then ingested by man. The larvæ are found in the liver, lungs, and kidneys.

Sarcoptes (*Acarus Scabiei*).—This insect produces the skin affection known as "the itch," or *scabies*, an affection more common in Europe than in America, where it constitutes only about 4 or 5 per cent. of all cases of skin disease. It is most prevalent among the poor and the unclean. The female is visible to the naked eye, and is about 0.5 mm. ($\frac{1}{50}$ in.) in length; the male is about 0.25 mm. ($\frac{1}{100}$ in.). Both are nearly as broad as they are long.

The parasite penetrates the skin and lives in a burrow or *cuniculus* that it makes for itself. The female lives in the end of the burrow, which may contain a number of ova, and appears as a minute, brownish-black, dotted, sinuous line, situated chiefly in the cutaneous folds, where the skin is mostly delicate, as between the fingers. Secondary skin lesions, due to scratching, are common. Sulphur ointment, well rubbed in after hot bathing, is usually quite efficacious.

Sarcoptes scabiei hominis is a variety of the preceding that infests other animals (cat, dog, cow, horse, wolf, goat, camel, etc.). Occasionally it may gain an entrance into man's skin, but dies simultaneously in the human host, although many invasions may occur.

Leptus Autumnalis (*Harvest Bug*).—The most common of several varieties is a mite of a reddish color, having six legs armed with claws and sharp mandibles. It arises among low bushes and thus appears about the ankles and legs. It partially penetrates the skin, boring only far enough with its short, thick head to procure nourishment. Artificial dermatitis may be produced by the irritation of scratching. Mercury, sulphur, and naphthol ointments suffice to destroy the parasite.

Demodex Folliculorum (*Comedo Mite*).—This minute parasite may be expressed from swollen sebaceous follicles of the nose, cheek, and other parts of the face. It has a worm-like body with very short legs, and is only about 0.2 to 0.4 mm. ($\frac{1}{60}$ in.) in length. It is not known to produce acne, as was formerly supposed.

OTHER PARASITIC INSECTS.

PEDICULOSIS.

(*Phthiriasis.*)

Lice or **pediculi** live on and attack the skin. Three forms are found on man: *pediculus capitis*, *pediculus corporis*, and *pediculus pubis*.

The *pediculus capitis* is whitish or grayish in color, about 1 mm. ($\frac{1}{25}$ in.) long (male), and has six legs under the front part of the body. The

oviparous female is nearly twice as long as the male, and lays from fifty to eighty eggs on the hairs within a week. These ova, or "nits," mature in from three to eight days. Itching is the most prominent symptom, and an eczematous eruption above and behind the ears and in the neck is often associated. "Plica polonica" was a phrase once used to designate the matted condition of the hair in extremely dirty, crusty, and long-neglected cases of head-lice. Secondary adenopathy of the cervical lymphatic glands is a common feature in neglected cases.

Pediculus Vestimentorum (*Corporis*).—This louse inhabits more often the clothing than the body itself. It is larger than the head louse, and, like the latter, moves slowly. The nits are found with difficulty on the fibers of the underclothing. It sucks blood through a proboscis inserted into the sweat pores, and after withdrawing leaves a minute hemorrhagic speck. Irritation of the skin is produced, and in old cases, as in filthy tramps, the skin becomes scaly and quite pigmented (vagabond's disease). The efforts at scratching are almost frantic, and after a cure is effected parallel white lines, the remains of scratch-marks, followed by atrophic changes, may be visible, as in a case that I reported.[1]

Pediculus or Phthiriasis Pubis (*Crab-louse*).—This parasite is not limited to the pubis, but attacks also the hairy region in the axilla, on the chest, and may even reach the beard and eyebrows. It clings firmly to one or two hairs close to the skin. Its six legs with strong claws are placed closely together at the anterior part of the ovoid body.

Treatment.—The hair should be cut short where the head-lice and nits are abundant. Saturating the hair and scalp with kerosene oil for twenty-four hours usually kills the parasites. *Body-lice* may be destroyed by scalding the underclothing and hot-ironing carefully about the seams. A hot soap-and-water bath is sufficient for the body, and sedative and antiseptic ointments may be useful adjuvants. Mercurial and beta-naphthol unguents usually suffice in treating for *pediculus pubis*. Prof. J. V. Shoemaker affirms that naphtol is a remedy that meets the indications presented by all forms of the disease; he prepares it as follows:

℞. Beta-naphtol, ʒj (4.0);
Cologne water, fʒiv–vi (120.0–178.0).—M.

Cimex Lectularius or Bed-bug.—This too well-known parasite is flat, brownish-red in color, and from 2 to 5 mm. ($\frac{1}{12}$–$\frac{1}{6}$ in.) in length. It infests beds and public vehicles, emitting a disagreeable odor. It is a blood-sucker, and causes considerable itching, local irritation, and urticaria even in some persons, while others are unmindful of their attacks. Sulphur fumigation and mercuric chlorid applications to the harboring places of the bed-bugs are effectual destructive agents. Saturated sodium bicarbonate solution will relieve the burning and itching.

Pulex Irritans (*Common Flea*).—This "ubiquitous" parasite is from 2 to 4 mm. ($\frac{1}{12}$–$\frac{1}{6}$ in.) in length, black or (when filled with blood) brownish-red in color, having six legs, the hind ones of which are relatively very large and powerful, enabling it to jump many times its own height. A flea's bite causes a sharp sting, and leaves a slightly raised red spot with a dark, pin-point center, the site of penetration of the biting apparatus of the insect. Treatment is the same as for the preceding insect.

[1] *International Clinics*, vol. iii., third series, p. 769.

Pulex Penetrans ("*Jigger*").—This parasite, also called "sand-flea," is indigenous to the West Indies, South America, and the Southern States. The impregnated female penetrates the skin, and especially that of the feet, for purposes of ovulation. As the distention with the eggs occurs, swelling, pain, and even ulceration may appear. The sand-flea is a small, egg-shaped insect, about half the size of an ordinary flea, brownish in color, and exceedingly resistant to crushing force. *Prophylaxis* in regard to foot-wear is necessary. Essential and antiseptic oils may also be put on the feet or stockings.

Ixodes (*Wood-tick*).—There are several varieties of tick- or wood-louse that may attack the human skin, among which *ixodes albipictus* is supposed to be the most common. *Ixodes ricinus* and *ixodes bovis* are found on horses and cattle. They are blood-suckers, adhering to the skin very firmly, and wheals may be produced by them. A drop of turpentine, or of some such essential oil as anise or rosemary, will cause them to loosen their hold.

Dermanyssus Avium et Gallinæ.—These bird- and fowl-insects are small and grayish-white in color, and may attack the human skin and cause eczematous eruptions, owing to the scratching induced by the irritation.

Culicidæ (*Mosquitoes and Gnats*).—The blood-sucking *mosquito* (*culex auxifer*), so well known, may also transfer to human beings the filaria sanguinis hominis and the plasmodium malariæ.

The *gnat* (*culex pipiens*) is very troublesome during certain seasons, particularly along water-courses and in wooded districts. Its bite is quick, sharp, and stinging.

The **hirudo** (leech) is a parasite that sometimes attaches itself to bathers. In the tropics it has been known to cause severe bites and inflammation. A remarkable case of hemoptysis is on record in which a leech was found attached to the larynx, below the cords.

The bites and stings of bees, wasps, spiders, and ants have been known to cause considerable inflammation, edema, and blood-poisoning.

Estridæ (*Bot-flies*).—These may become parasitic in man in the larval form. Species of the *hydoxerma* and *dermatobia*, that infest the skin of the horse, ox, goat, etc., have also been observed among the Central and South American Indians. They burrow beneath the skin of the abdomen, scrotum, and other regions.

Muscidæ (*Common Flies*).—Common flies affect the skin of man by depositing eggs in wounds. The ova hatch within twenty-four hours sometimes, and the dipterous larvæ may swarm to make the so-called "living" wound or sore (*myiasis vulnerum*). The larvæ or maggots do not penetrate the tissues, however. The principal flies that infest wounds are the flesh-fly (*sarcophila carnaria*), the blow-fly (*calliphora vomitoria*), the screw-worm fly (*compsomyia macellaria*), and the ordinary house-fly (*musca domestica*).

Internal myiasis may also be caused by swallowing the ova of these flies. The larvæ may thus be vomited or defecated.

Epidemic urticaria is often caused by the migration of the caterpillar (*cnethocampa*). Among other parasites that attack man and inhabit particular regions are the following: The *simulium reptans*, or creeping gnat of Sweden; the *seroot-fly* (*zimb*) of Abyssinia; the *ixodes carapato*, a virulent bed-bug in Brazil; the *hæmatopota pluvialis* (Clegg) of the West Highlands.

SYPHILIS.

Definition.—A chronic infectious disease communicable from person to person by direct or indirect contact with a specific virus, or by heredity. According to its clinical course, it is characterized by five periods: (1) Period of primary incubation—the time which elapses between contact with the poison and the appearance of the chancre. (2) Period of secondary incubation—the time which elapses between the appearance of the initial lesion of the disease (the chancre) and the development of its cutaneous manifestations. (3) Period of secondary symptoms (skin eruptions). (4) Intermediary period characterized by the absence of lesions, although evidences of existing dyscrasia can still be found. (5) Period of tertiary symptoms. The hereditary form of the disease is transmitted at the time of procreation by the sperm virile, by the ovum, or by both. Prince Morrow[1] points out that the important lesions of the disease are those that occur in the internal organs—*visceral syphilis.*

General Pathology.—(*a*) **Primary Lesion of Chancre.**—This appears at the site of inoculation, and is characterized by infiltration of the connective tissue chiefly with round cells of the same type as those seen in recent granulations. There is sclerosis of the small blood-vessels, chiefly involving the adventitia of the arterioles. The neighboring lymphatic glands soon undergo hyperplasia and induration.

(*b*) **Secondary Lesions.**—Macular and maculo-papular eruptions are frequent, and, with the mucous patch, show round-cell infiltration of the connective tissue and blood-vessels similar to that found in the chancre, with plasma cells and leukocytes. The favorite sites for mucous patches are the mucocutaneous junctions (mouth, anus, etc.). Other lesions of this stage are general adenopathy, alopecia, and pharyngitis.

(*c*) **Tertiary Lesions.**—These are circumscribed inflammatory products known as gummata. They appear in the connective tissue, bones, periosteum ("nodes"), skin, muscles, brain, liver, lungs, kidneys, heart, testes, etc. The gummata, though usually sharply circumscribed, may take the form of diffuse infiltrations and vary in size—from a pin's point to a hen's egg. Usually firm, they may be soft, and tend to form ulcers. Their color is grayish, and on section they show a caseous, semi-opaque center, with a fibrous, translucent periphery.

Microscopically, the gumma consists of lymphoid cells, plasma cells, leukocytes, and epithelioid cells, in which fatty degeneration and softening result in the formation of a pasty mass. The mass thus formed may either be absorbed or persist; but in most instances coagulation-necrosis occurs in the center, with conversion of the peripheral zone into fibrous tissue. Gummata of certain structures (skin, mucous membrane, bones, and cartilages) often lead to destructive ulceration and sloughing.

General Etiology.—**Parasitology.**—Schaudinn and Hoffman[2] described two spiral micro-organisms; one, from the deeper layers of the chancre, condylomata, and lymph-glands, the specific organism, or the *treponema pallidum;* while from the superficial part of the lesions, the non-specific *spirochæta refringens.* The *treponema pallidum* in length varies

[2] *Deutsch. med. Wochenschr.*, May 4, 1905.
[1] *Medical News*, March 23, 1901.

from one to six times the diameter of a red blood-cell, in width from unmeasurable thinness to $\frac{1}{2}\ \mu$. Metchnikoff and Roux[1] have demonstrated this organism in acquired syphilis of man and in experimental lues in the monkey and ape.

Predisposing Causes.—Since acquired syphilis originates only by inoculation, it is obvious that a break in the cutaneous or mucous surfaces is essential to infection, such as a slight abrasion, fissure, or laceration, etc., particularly of the genital mucosæ. Other surfaces may also be the seat of infection, as the lips, hands, etc.

Susceptibility to the virus is universal, and no age is exempt. *Reinfection* is exceedingly rare, but does occur.

Contagion of Syphilis.—The blood of a syphilitic during the secondary period, and the secretion from the chancre or any of the secondary lesions, are contagious, the lesion at the point of inoculation always being a chancre. The physiologic secretions, saliva, sweat, milk, and semen, do not convey the virus, unless contaminated with the discharges from some of the lesions of the primary or secondary stage. The semen is able to infect the embryo and, in turn, the mother. There is experimental evidence to show that the gumma is infectious.

Modes of Infection.—(1) In a great proportion of the cases (about 70 per cent.) syphilis is transferred by illicit sexual intercourse.

(2) *Accidental Inoculation.*—This is not uncommon. (*a*) Most frequently it is accomplished through the pernicious custom of indiscriminate kissing (lip-chancre), and I have personal knowledge of not less than 8 instances in which infection has occurred through labial contact. In Russia from 75 to 80 per cent. are acquired in this manner from popular customs.

(*b*) The site of inoculation may also be the mouth and tonsils, the virus being conveyed during the low practices of sexual perverts or by kissing. The wet-nurse may infect the mouths of suckling babes, or, *vice versa*, the infant may infect the nipple of the nurse.

(*c*) The obstetric finger may become infected. Three instances of the sort have come under my own observation, and Fournier gives the details of 40 cases of primary syphilitic infection of the hand. In 30 of these the malady was acquired in medical practice (4 obstetricians, 20 general practitioners, 3 students, and 3 midwives). Montgomery[2] states that chancre of the finger is peculiarly frequent in physicians.

(*d*) Humanized vaccine virus may rarely transmit the disease.

(*e*) Accidental infection has, at times (though very rarely), taken place in a variety of other ways—*e. g.*, handling foul rags from the hospital ward, by bed-clothing, drinking-cups, the pipe and cigar, tattooing, etc.

Krafft-Ebing found that out of 3455 cases $15\frac{6}{10}$ per cent. were of extra-genital origin. The lesion was upon the lips in 51 per cent.

(3) *Hereditary Transmission.*—Paternal transmission (through the semen) is much more common than is maternal, the period of greatest danger being immediately after the father has become infected or during the time of the secondary manifestations. The first-born, if the father be syphilitic, is apt to show well-marked lesions. Appropriate treatment of a syphilitic parent lessens the danger of transmission very

[1] *Bulletin de l'Académie de Medecin*, Paris, May 16, 1905.
[2] *Jour. Cutan. Diseases*, April, 1905.

materially, however, and in such instances there is little tendency to transmission shown after the third year. On the other hand, a syphilitic father or mother may beget healthy offspring, the infant having acquired some immunity which protects it from its mother (Profeta's law). Syphilitic children are also common to infected women. In the majority of instances of hereditary transmission, however, both parents are syphilitic, and under these circumstances the liability to infect the offspring is much augmented. A woman who has become infected after conception may bear a syphilitic child; though the latter may, on the other hand, escape infection.

Allusion may here be made to Colles's law—that a woman who bears a syphilitic child enjoys, owing to a sort of protective vaccination with the specific virus, perfect immunity, and this in the absence of all signs of the affection. Coutts[1] dissents from this opinion.

Clinical History of Acquired Syphilis.—(*a*) **Primary Stage.**—The typical *initial lesion* (chancre) appears about three weeks after infection, and is followed soon by swelling and induration of the nearest lymphatic glands. The primary sore begins as a *red papule*, which rapidly reaches its maximum, and then undergoes a central necrosis with the formation of a *small ulcer*. The adjacent structures become hard or cartilage-like—a characteristic to which the lesion owes its name of "hard chancre." A small chancre may often escape detection, especially if it be situated inside the meatus. When situated upon a mucous membrane it is always a *chancrous erosion*, which may be so mild and of such brief existence as to come and go without the knowledge of its bearer. Particularly is this the case in the female. The *general symptoms* are negative in this stage.

(*b*) **Secondary Stage.**—This is announced about six weeks after the appearance of the infecting chancre by *moderate fever* (100°–101° F.—37.7°–38.3° C.), exceptionally higher, accompanied by languor, headache, bone-pains, impaired digestion, and a slight degree of prostration. There is angina, with hyperemia of the fauces and hard palate. The blood shows a marked reduction in hemoglobin with some diminution in the number of red cells. *General lymphatic enlargement* is seen, especially significant in the post-cervical and epitrochlear glands.

Skin eruptions are of many forms. The *erythematous* or *roseolar* is the earliest and most common, coming out abundantly upon the trunk (especially the chest), buttocks, thighs, and forehead. Another early variety is the *papular*. The papules are small or large, hard, and appear on the face, trunk, and flexor surfaces of the extremities.

Mucous patches may appear on the visible mucous surfaces (angles of mouth, tongue, tonsils, pharynx, vulva, vagina, penis, and around the anus), and are among the early and constant lesions. The distribution of these early syphilids is symmetric; their outlines are rounded; their color like that of a slice of raw ham ("coppery"); they are polymorphous; and, as a rule, they excite neither pain nor itching.

Other and later-appearing eruptions may be *pustular*, and *tubercular*. These show a tendency to bunch in certain areas, and hence are less diffuse than the afore-mentioned eruptions; and are not symmetrically distributed on the body.

[1] "Hunterian Lectures," *Lancet*, 1896, No. 3889.

Other frequent symptomatic conditions arise during this secondary period, such as alopecia, laryngitis, iritis, choroiditis, retinitis, and epididymitis (more rarely). The hairs of the eyelids and eyebrows may fall off and the finger-nails become brittle.

The *secondary* symptoms last from two to three months (the usual duration) to a year or more, and are followed by a period of apparent good health lasting for an exceedingly variable interval (from a few months to many years) before the tertiary stage sets in. During the secondary stage the symptoms may be severe, mild, or even absent. There is a *late secondary* syphilis, the symptoms appearing a variable number of years after the primary lesion. Fournier states that late secondary phenomena occur most often in cases that have been well treated.

(*c*) **Tertiary Stage.**—As I have already stated, the secondary period is generally followed by a variable interval of freedom from symptoms, but to this rule there are numerous exceptions, and among not uncommon occurrences may be witnessed the appearance of tertiary symptoms during the secondary stage. As stated by R. W. Taylor, "By far the most potent and frequent cause of tertiary syphilis is the absence or insufficiency of treatment during the secondary stage." Belonging to the third stage are certain skin-eruptions, especially the characteristic *rupia*, which first appears in the form of pustules that break and form ulcers that are covered with dry, laminated crusts "like an oyster-shell." To this stage also belongs the *tubercular* variety, affecting the face, back, and legs, and very commonly the elbows, and rarely other portions of the bodily surface. These eruptions involve the true skin, and in healing leave scars, but, unlike the secondary cutaneous lesions, they are neither infectious nor contagious, are not, as a rule, symmetric, and are more liable to be attended by itching. A purpuric syphilid (blood-extravasation form) is also met with in this stage. True *gummata* may develop in the skin and subcutaneous tissue, and these break down and form kidney-shaped ulcers which tend to spread in a serpiginous manner. On healing (a process that is accomplished with difficulty), scars result. Gummata may occur in the mucous membrane and pass through the stages of ulceration and cicatrization. When situated in the larynx or trachea, their healing is attended with narrowing of the organ, and when in the lower bowel or the rectum, dysenteric symptoms, followed by actual stenosis, may result.

In the muscles gummata occur and form small hard tumors. They may also cause periostitis and death of the bones, especially of the nose, palate, and skull; "nodes" are thus formed, which are situated chiefly upon the tibia and the skull in larger or smaller numbers, and also, though less frequently, upon other bones. These are exceedingly painful, particularly at night, and are very tender. They may be true gummata, but more often, if not absorbed, they either become ossified or undergo fibroid change, while in rarer cases they suppurate. Chronic enlargement of the lymphatics and of the testicle, with little tendency to suppuration, may be noticed. The pregnant female is apt to abort or miscarry. Fever of any type may attend this stage.

Gummata also occur in the internal organs (*visceral syphilis*), and of the latter I shall speak presently, taking up separately some of the various organs and systems of the body. *Amyloid degeneration* is frequently caused by the acquired form, particularly syphilis of the rectum in women, but very rarely by the congenital.

Malignant Syphilis.—By this term is meant a virulent and a fatal form of the malady which is fortunately rare. The various stages manifest themselves early, and especially the tertiary, as on the forty-fifth day in a case of Mauriac. The course is rapid and the condition resists all forms of treatment. Roussel narrates a case in which death occurred about one year after the commencement of the disease.

Clinical Symptoms of Hereditary Syphilis.—These may, though rarely, be identical with those of acquired syphilis, if we except the chancre.[1] Occasionally the characteristic symptoms are present at birth. On the other hand, in the vast majority of instances, they appear between the first and fourth months of life (*infra*). The symptoms of *inherited syphilis* may be grouped according to the time of appearance. Kassowitz[2] states that one-third of all children procreated of syphilitic parents are born dead, and of those born living 24 per cent. die within the first six months of life.

(1) **In the New-born.**—There is a lack of physical development. The babe may be *greatly emaciated*, it has snuffles, and singultus occasionally sets in soon after birth. Skin-eruptions are rare, except *pemphigus neonatorum*, which appears as bullæ on the palms and soles; among exceptional cutaneous phenomena are gummata around the radio-carpal articulations, palmar psoriasis, and a fleeting roseola. Ulcers and fissures (*rhagades*) may be noticed around the outlets of the body (mouth, anus, etc.); the osseous system may show hyperostoses of the long bones; and the liver and spleen are enlarged. Comby reports 8 cases of pseudo-paralysis due to syphilis in the new-born.

(2) **Early Post-natal Symptoms.**—Most subjects of syphilis hereditaria are born *plump* and *without taint*. Symptoms appear in the majority of cases not later than the third month.

The first symptom is generally *coryza* (syphilitic rhinitis), which is betrayed by a sero-purulent or bloody discharge and a peculiar form of *obstructed breathing* (snuffles), rendering nursing difficult. The coryza may in some cases be preceded by singultus lasting ten or twenty days (Carini), and ulcers may form in the nose, leading to necrosis of the bones and producing at last a sunken and deformed nose which is highly significant. The coryza may extend to the middle ear and cause otitis media, with deafness and otorrhea as the chief symptoms. The *skull* may approach the natiform in shape, and the signs of diaphyso-epiphyseal inflammation develop.

The *cutaneous symptoms* appear early. The skin has a tawny hue, and an erythematous eruption of the nates and genitals is frequently seen; this is patchy, with well-defined margins, and has the characteristic coppery color. In the same localities papules may appear, while pemphigus may attack the palms and soles. Syphilitic onychia may be present, and the lips and angles of the mouth often show fissures that are of real diagnostic worth. Other symptoms are ulcerations of the skin and mucous surfaces, falling of the hair, and a moderate glandular enlargement.

Enlargement of the spleen is a frequent characteristic symptom, and, according to White and Martin,[3] of greatest importance "when noticed

[1] With prenatal syphilis we are not concerned.
[2] *Vererbung der Syphilis*, Vienna, 1876.
[3] *Genito-urinary and Venereal Diseases*, 5th ed., 1902.

early—the first three months after birth—since at this period enlargement of the spleen due to rachitis can hardly come into question."

Swelling of the liver may also be present, but is of little diagnostic import. Syphilitic infants occasionally manifest a hemorrhagic tendency. At birth bleeding from the umbilicus may occur; later, into the subcutaneous tissue and from the mucous membranes (gastro-intestinal, vaginal, nasal, etc.). Hecker[1] considers an examination of the umbilical cord important for the early recognition of syphilis in the offspring of syphilitic parents; if the microscope shows characteristic changes, time may be gained for treatment; "these changes range from a decided endarteritis or periarteritis or phlebitis to a simple round-celled infiltration of the blood-vessel walls or the surrounding tissue." As pointed out by Osler, these cases must not be confounded with Winckel's disease.

Among *nervous symptoms*, restlessness, sleeplessness, and a harsh, shrill cry which may be almost constant for days together and due most probably to darting pains, are the chief. Anemia and other evidences of syphilitic cachexia soon supervene.

(3) **Late Symptoms.**—The symptoms of syphilis hereditaria tarda may be arranged in groups (Fournier):

(1) *Those Indicated by the General Appearance.*—There is a retarded general development, as shown by the small stature, undeveloped muscles, the graceful form, and infantile appearance at ages varying from four to twelve or more years. The skin has an earthen tint, and the hair may be scanty and late in its appearance on the face and genitals.

(2) *Skin-cicatrices.*—Cutaneous scars, particularly if multiple and extending over a circumscribed area, are important diagnostic signs. Their form is usually round or serpiginous, and their chief location the mouth, nose, soft palate, and lumbo-gluteal regions.

(3) *Lesions of the Skeleton.*—The natiform skull, "with a transverse enlargement, lateral bulgings, and the flattening in the middle," is almost pathognomonic. Asymmetric and hydrocephalic skulls are also to be considered, in many cases, as signs of hereditary syphilis, as is a sunken and deformed nose. The thickened, "sabre-shaped" tibia, due to gummatous periostitis, is capital evidence of the disease, while the chicken-breasted thorax is significant.

(4) The *testicles* show an arrest in development (infantile testicles). This is a sclerotic atrophy.

(5) *Hutchinson's triad*, under which title come (*a*) the Hutchinson teeth; (*b*) ear-conditions; (*c*) affections of the eye.

(*a*) *The Hutchinson Teeth.*—The teeth may be late in appearing, and the dental arch may be malformed, the teeth presenting various irregularities in form and condition (dental dystrophy).

The incisors, especially the superior median of the second dentition, are notched, and show a thinness of the free edge, an atrophy of the summit, and crescent-shaped erosions. Fournier[2] calls attention to the absence of one, two, or more teeth in a great number of cases.

(*b*) *Ear-conditions.*—Otorrhea, secondary to naso-pharyngeal catarrh, has already been mentioned, and, in addition, at or about the time of puberty an incurable form of deafness may develop speedily, without the presence of pathologic lesions to explain the same.

[1] *Jahr. f. Kinderh.*, Bd. li, Heft 3.
[2] *Gaz. hebdom. de méd. et de chir.*, January 18, 1900.

(*c*) *Affections of the Eye.*—These are interstitial keratitis and iritis, affecting both eyes successively.

Visceral Syphilis.

Syphilis of the Brain and Cord.—Pathology.—The most characteristic and not infrequent lesions are: (1) *Diffuse Gummatous Meningitis.*—This occurs most often in the pia, extending to either the dura or brain-substance. It is seen as patches of round-cell infiltration with sclerosis of the blood-vessels. In the cord the same changes are found.

(2) *Gummata.*—Their usual situation is in the membranes, more often the dura, extending to the brain secondarily. Rarely the brain-substance only is affected. Their size varies from that of a millet-seed to that of an egg, and they present irregular contours. They are single or multiple and are usually situated either in the cerebral hemispheres or on the pons, and rather superficially, connecting directly or indirectly with the dura or pia mater. In gummata of average size a cut-section shows caseation in spots which are connected and surrounded by firm, translucent, gray or reddish-gray, fibrous tissue; and the more irregular surfaces and the irregular caseation serve as important distinctions from tuberculous tumors. When, as is usual, the gummata touch the membranes, meningitis—subacute or chronic, with much thickening—is combined. Gummatous growths may attack the cord. They seldom attain to a large size.

(3) *Endarteritis.*—This important lesion of syphilis may result in aneurism, hemorrhage, or narrowing and obliteration of the lumen of the blood-vessels. As a consequence of the latter, areas of softening and secondary degeneration occur, varying in size with the distribution of the affected vessel. Thrombosis in cerebral arteries may be found. Similar vascular lesions occur in the cord.

Etiology.—Cerebral syphilis is usually a late (tertiary) manifestation, appearing on the average three or four years after infection, but it may appear much sooner. After twenty years it is rare. Taylor[1] has pointed out that syphilis of the nervous system is likely to appear in persons of a neurotic or neurasthenic constitution, particularly in those cases where the treatment required for the secondary period of the disease has been neglected or insufficiently carried out.

Symptomatology.—*Imbecility* and *idiocy* may be due to inherited syphilis, but they are probably too often attributed to this cause. The other features simulate those of the acquired form.

The symptoms of the acquired form are with few exceptions referable to three affections: (*a*) epilepsy, (*b*) brain-tumor, and (*c*) paralysis.

(*a*) *Epilepsy* coming on after the thirty-fifth year, not dependent upon alcohol nor uremia (p. 1205), is usually due to the ravages of syphilis, and a careful search for traces of scars and of the entire body surface for bone-lesions, etc., should be instituted. Convulsions of the epileptic type have also occurred during or just before the advent of secondary symptoms. The appearance of the disease may be preceded by psychic disturbance, headache, dizziness, and loss of memory. Hysteric manifestations may also be presented, being probably provoked by the specific lesions. On the other hand, a protracted torpor which may last for a few

[1] R. W. Taylor, *Venereal Diseases.*

days or as many weeks may develop. While in this stuporous condition the patient may wander aimlessly about. In one of my own cases periods of marked mental excitement, that persisted for three or four days, alternated with periods of almost complete insensibility of about equal duration.

(*b*) *Brain-tumor.*—The symptoms pointing to brain-tumor will be discussed under this head in the section on Nervous Diseases. The syphilitic nature of the cerebral growth cannot be determined with certainty except in the presence of a clear history of syphilis—congenital or acquired—and the characteristic symptoms or traces of the primary, secondary, or tertiary lesions.

It must be remembered that the secondaries are either sometimes absent or go unnoticed, and if the patient has had a primary sore, the presence of the characteristic symptoms of brain-tumor (headache, optic neuritis, convulsions, etc.) make the existence of specific nerve-lesions highly probable. The chancre may also be overlooked or denied, and it is in such instances as the latter that the occurrence of convulsions in persons over thirty should excite suspicion.

(*c*) *Paralysis.*—This may take the form of hemiplegia, due usually to cerebral thrombosis if of sudden advent (p. 1170), or if of gradual development to gumma (p. 1185), or of general paralysis (*dementia paralytica*). The relation that these affections bear to syphilis will be indicated in its appropriate place in this work in the description of Nervous Diseases. The fact may here be pointed out that syphilis may induce precisely the same changes met with in general paralysis of the insane (p. 1198).

The cranial nerves, especially the third, fourth, and sixth, are liable to be involved in syphilitic basal meningitis (p. 1121).

The history of syphilitic infection, together with symptoms of spinal tumor (p. 1156), points to *gumma* of the cord. Syphilitic myelitis usually develops in five years after the infection, and may pursue an acute or subacute course, though oftener it takes the form of chronic myelitis (pp. 1134 and 1137). The latter attacks by preference the lumbo-dorsal section of the cord—a fact corroborated by the character of the symptoms, and in most cases is not a true inflammation, but is a softening due to thrombosis in some of the spinal arteries. A type described by Erb, and known as Erb's syphilitic spinal paralysis, presents rather characteristic symptoms. These consist of slowly increasing weakness and stiffness of the lower limbs, with increased reflexes and sometimes paræsthesia of legs, back, and anal region. There is also some inco-ordination, and Romberg's symptoms may be present. The gait is of the ataxic paraplegic type (p. 1152), and incontinence of urine and fæces is usually present. Sensory paralysis is absent. A peculiar feature is that at rest the rigidity of the limbs is not marked, but becomes so when attempts to walk are made. The lesion is probably a thrombosis of the vessels supplying the posterior columns and pyramidal tracts.

General Diagnosis.—The onset in nervous syphilis may be acute or subacute, and the symptom-complex embraces a multiplicity of phenomena, there being an especially erratic distribution of the ocular and other attending palsies and early marked impairment of the mind, all occurring, as a rule, in *early adult life.* The symptoms, while they may simulate any of the various system and general diseases of the nervous system, are apt to present some atypical feature, and their development is frequently preceded by violent headache and somnolence. They are also frequently

more or less transient and shifting. Examination of the cerebrospinal fluid for pleocytosis and increase in globulin, as well as the Wassermann test in both blood and fluid, is of service in doubtful cases (pp. 1149 and 1201).

Prognosis.—In acute cerebral syphilis, with stupor, this is bad. In other forms complete recovery may occur, but it is well to remember that where actual destruction of nerve-cells and fibers has taken place that removal of the lesion will not restore them, therefore it should always be guarded as to how much restoration of function will occur.

Syphilis of the Liver.

In my experience the liver, with comparative frequency, bears the stress of visceral syphilis. Syphilis of the liver occurs more frequently in men than in women, and, according to Peiser,[1] appears most frequently in from five to fifteen years after date of infection.

Pathology.—The lesions may be thus classified: (*a*) **Diffuse Syphilitic Hepatitis.**—This is met with chiefly in congenital cases. Though its occurrence in adult life has been questioned by some, I have seen an instance in an adult who died of cerebral hemorrhage. The liver is uniformly enlarged, firm, and resists the cutting-knife. Its color is grayish-yellow. The microscope shows a marked increase in the connective tissue and a cell-infiltration throughout. From intense, focal cellular infiltration miliary gummata may result; these undergo contraction, diminishing somewhat the size and altering the shape of the organ.

(*b*) **Gummata.**—These may be seen in congenital cases (chiefly the miliary gummata). As seen in the adult, hepatic gummata are disseminated nodules, with the usual central, cheesy mass surrounded by a zone of grayish fibrous tissue and varying in size from a hazlenut to an apple. They form separate tumors, whose favorite seats are the convex surface of the organ, especially near to the suspensory ligament, and in the region of the portal vessels. They are usually tertiary lesions, and appear a number of years (two, three, or four) after infection. These so-called syphilomata in the advanced stage contract, and the liver will be found smaller than the normal. Deep furrows due to contracting fibrous bands traverse the organ in different directions and divide it into lobes of various dimensions. Gummata frequently undergo fibroid change, but more rarely they soften and liquefy (Wilks). On the other hand, before contraction occurs the liver is increased in size and the gummata form protuberances on its surface.

(*c*) **Gummatous Arteritis.**—Briefly, this may affect both the portal vein and hepatic artery, though syphilitic endarteritis is situated chiefly in the smaller branches of the latter.

(*d*) **Perihepatitis.**—Here Glisson's capsule is thickened, owing to augmentation of its connective-tissue elements. From the latter there dip into the hepatic tissue cicatricial bands, particularly along the portal canals, which may change somewhat the shape of the organ. Section shows admirably the pale scar-like tissue (*vide* Diseases of the Liver).

Clinical History.—The affection may exist without symptoms. In the *congenital form*, however, we have signs of hepatic enlargement, with icterus, the spleen being likewise large and firm, as a rule. The history and associated lesions are necessary to a certain diagnosis.

[1] *Die Leber syphilis brochure*, Leipsic, 1886.

In the *adult* syphilis of the liver does not usually attract attention until the gummata interfere with the portal circulation. As they undergo contraction they tend to occlude some of the portal branches, or they may, on account of their situation, exert pressure upon the vena porta itself. In either event the evidences (ascites and splenic enlargement) of portal obstruction will develop as in alcoholic cirrhosis. The gastro-intestinal symptoms common to the latter disorder are also present, and obstructive jaundice may supervene, though it is, comparatively speaking, rare. Pain, usually localized to some particular spot over the right hypochondrium, is sometimes complained of, and may be quite severe, while pressure over the painful area elicits great tenderness.

Physical Examination.—In the early stage, while the organ is enlarged, flattened, irregular protuberances may be detected by the palpating fingers. These nodules, or large, round masses, invade especially the left lobe as compared with the right. At a more advanced period ascites may interfere with palpation, and in such cases an aspiration of the fluid will enable one to feel the syphilomata. Finally, in the stage of contraction the results of palpation are obviously negative.

There is a group of cases in which the clinical picture is that of *advanced amyloid disease* of the viscera. The liver and spleen are enlarged, the urine is increased in amount and contains albumin and tube-casts, and finally dropsy supervenes.

Diagnosis.—This rests upon the etiology, the presence of scars on the skin-surface, bone-lesions (irregularities of the tibial surfaces), or other evidences of the ravages of the disease, and upon moderately good general health. The most important local symptoms are the hemispheric prominences on the surface of the liver and the localized pain. The diagnosis between syphilitic disease of the liver and *echinococcus-cysts* is sometimes extremely difficult. R. Lennhoff has noted in a number of cases of echinococcus-cyst that on deep inspiration a furrow forms above the tumor, between it and the edge of the ribs.

The clinical findings resemble those of cancer of the organ. I have contrasted the main dissimilar points in the subjoined table:

Syphilis of the Liver.	Cancer.
History of heredity or of infection.	Of heredity or of primary growth.
Occurs congenitally, or, if acquired, at any age.	Never congenital. Usually occurs after the age of forty.
Often accompanied by symptoms of tertiary syphilis—alopecia, rupia, etc.	Often preceded by the primary growth pylorus, uterus, mammary gland.
Jaundice and ascites are common, especially the latter. No cachexia.	Jaundice and ascites are rare. Marked cachexia.
The margin is markedly irregular, and neither nodular nor umbilicated.	Often the margin reveals the presence of umbilicated nodules.
Recovery may follow, or the affection may last for years.	Always fatal. Duration usually from a few months to a year.

The course and the *results* of antisyphilitic treatment are of value for diagnosis. The course is slow and often interrupted, while appropriate treatment may lead to recovery, as in three of my cases.

Syphilis of the Alimentary Tract.

The lesions in the mouth have been for the most part considered. In the tongue gummata often develop. A decidedly fissured appearance

of the organ and whitish scar-like patches upon the surface may be observed in syphilis, but have no essential connection with that disease. Perforation of the palate, due to tertiary lesions, is not rare. Gummata also appear on the posterior wall of the pharynx and lead to ulceration, which may cause fatal hemorrhage by erosion of adjacent large blood-vessels (internal carotid, etc.). The walls of the esophagus may also be invaded, resulting usually in stenosis.

The stomach-walls may be infiltrated and, rarely, ulcerated. Einhorn, Fournier, and others have met gastric ulcer in syphilis; it was cured by the specific treatment. Syphilitic tumor of the stomach may rarely occur; the symptoms are those of malignant growth, resembling cancer, but curable. Syphilitic ulcers may appear in the intestines. The condition may lead to perforation and peritonitis; more often to stenosis.

Gummatous infiltration of the rectum is a somewhat frequent, severe, and clinically important affection. It is much more common in women than in men, taking place in the "submucosa above the internal sphincter." It has frequently caused a fatal result in persons who failed to show post-mortem specific lesions in other viscera, and hence it is to be classed as one of the ravages. The result of the gummatous infiltration is the production of a funnel-shaped stenosis of the rectum which narrows from below upward. Above the stenosis, and directly dependent upon it, there is dilatation of the rectum and the descending colon. Here may also be found ulcers—some specific, and others the result of mechanical pressure exerted by the fecal accumulations.

Symptoms.—The clinical features are for the most part those of a gradually induced *stenosis* of the rectum. At first there may be hemorrhages, suggesting internal hemorrhoids. The action of the bowels is irregular, and is followed shortly by a tendency to dysenteric diarrhea, with pains, tenesmus, and scanty stools containing mucus and pus. Prolapse of the rectal mucosa may occur, and, owing to the presence of small hemorrhoids, the true nature of the case may be overlooked. The disease is most distressing, and leads slowly and gradually to extreme emaciation and asthenia. Death may be due to the latter or to some complication (perforative peritonitis, etc.).

Diagnosis.—This may be aided by a clear history of associated syphilitic symptoms or of specific lesions, including amyloid degeneration. In tubercular ulcer other undoubted evidences of tuberculosis are found. Carcinoma is usually situated higher up the rectum than gumma and more often forms firm adhesions to surrounding parts. Final diagnosis would depend on microscopic examination of an excised portion.

Syphilis of the Lungs.

While undoubted cases occur, syphilis of the lungs is rare indeed.

Pathology.—The cases are pathologically divisible into three forms: (*a*) Gummy tumors; (*b*) Interstitial pneumonia; (*c*) Fetal pneumonia.

(*a*) **Gummy Tumors.**—These appear as yellowish-white scattered nodules, varying in size from a cherry-pit to a hen's egg. Their centers are dry and caseous-looking, and their peripheral zones fibrous. They are relatively thicker set near to the root of the lungs. Cicatricial bands may be seen connecting not only the separate nodules, but stretch-

ing outward to the thickened pleura. Such growths may undergo ulceration, thus forming a cavity that rarely attains to large measurements; or, on the other hand, in favorable cases the fibroid changes and cicatrization may lead to recovery. A primary lesion is atrophy of the alveolar walls, with hyaline degeneration of the capillaries (Councilman). Broncho-pneumonia (syphilitic?) may be associated.

(*b*) **Interstitial Pneumonia.**—This is a fibrous infiltration, showing a predilection for the right lung. Its chief seat is the root of the lung, whence it extends along the bronchi and vessels, and usually involves a part of one or more lobes. Occasionally its starting-point is the pleura, from which the process advances along lines corresponding to the interlobular tissue. Bronchiectasis may be noticed. Gummata may also be associated, or may have been present and been practically obliterated during the process of cicatrization.

(*c*) **Fetal Pneumonia** (*Virchow's White Hepatization*).—This is peculiar to the new-born, in which miliary gummata first occur, followed by hepatization of large zones or an entire lung. The chief changes are an infiltration of the alveolar walls, while the air-cells are filled with epithelium; on section the tissue presents a grayish-white appearance.

Symptoms.—From what has just been stated it is clear that a certain limited number of cases present symptoms and signs that simulate ordinary ulcerative phthisis, but do not show bacilli in the sputum. There is another group of cases in which the picture presented to view is almost identical with that of fibroid induration, though usually giving a distinctly syphilitic history. I am not prepared to say that there is an acute syphilitic broncho-pneumonia analogous to acute pneumonic phthisis, though I fail to see any reason why this may not occur.

Diagnosis.—If a suspected case is treated early, the result may serve to corroborate the diagnosis, which is at first far from being final.

Bronchiectasis, dependent upon syphilitic peribronchitis or interstitial pneumonia, cannot be discriminated from other forms of that disease except there be a clear history of infection, and unless associated scars or active syphilitic lesions coexist. *Pulmonary tuberculosis* cannot be distinguished from *pulmonary syphilis* without a careful microscopic examination of the sputum. Moreover, it must not be forgotten that these affections are often combined. The suspicion of syphilis should always attach to lesions beginning in the lower parts of the lung, and slowly progressing without the production of fever (Taylor).

Syphilis of the Spleen.

Pathologically, syphilis of the spleen is to be classed with the general adenopathy of the disease. According to the statistics of Sée (relating to hereditary syphilis) and of Avanzini and Schuchter (relating to acquired syphilis), in about 25 per cent. of the cases of secondary syphilis hypertrophy of the spleen may be noted. This augmentation begins from two to four weeks after the appearance of the chancre, and gradually increases, persisting throughout the secondary period; it is not, however, observed during tertiary stage. It is often accompanied by localized pain—syphilitic pleurodynia (Besnier). Gummata are rare.

Syphilis of the Circulatory System.

The Heart.—The pathologic divisions are—(*a*) *Gummata*, which attack chiefly the walls of the left ventricle. They are usually encysted.

(*b*) *A Fibro-sclerotic Myocarditis.*—The process begins in the perivascular tissue and proceeds from the vessel walls outward (Mracek). It is diffuse, as a rule, and leads to narrowing of the lumina of the coronary arteries and their branches or to aneurysmal bulgings, but the pathologic effects of these lesions are seldom detected clinically. Sudden death may occur.

(*c*) *Syphilitic Endocarditis.*—The changes are of the fibro-sclerotic variety, and not of the acute verrucose type. The symptoms to which the lesion gives rise are depicted under Organic Valvular Disease.

Syphilis of the Arteries.

Two forms are recognized: (*a*) *Obliterating Endarteritis.*—Here the syphilitic product consists chiefly of proliferated subendothelial tissue, which encroaches more and more upon the lumen of the vessel—a fact to which the disease owes its name. This so-called "Heubner's degeneration" is not peculiar to syphilis, but, as Osler says, "if, however, there are gummata in other parts, or if there be gummatous periarteritis in adjacent vessels, the process may be regarded as syphilitic.

(*b*) *Gummatous Periarteritis.*—The arteries most frequently involved are those at the base of the brain. Charcot describes a condition which he calls "syphilitic periarteritis," where the tunics of the arteries are infiltrated with tumors or nodosities which the microscope showed were the result of an acute arteritis producing infiltration of connective-tissue cells into the tunica media.

Syphilis of the arteries has an important etiologic bearing upon atheroma and aneurysm (*vide* Diseases of the Arteries).

Syphilis of the Kidneys.

Renal syphilis belongs chiefly to the tertiary stage, though it may appear in the secondary.

Pathology.—(*a*) *Amyloid degeneration* is a common renal lesion.

(*b*) *Chronic interstitial nephritis.*

(*c*) *Gumma.*

Symptoms.—Except in the case of amyloid degeneration the conditions are impossible of correct diagnosis. Wagner describes a special form which he calls *acute syphilitic glomerulo-nephritis.* Clinically, it is characterized chiefly by hematuria, and ends rapidly with uremia.

Syphilis of the Joints.

The following division of the affection is made by Hutchinson:

(1) *Synovitis* appears during the secondary stage, but soon clears away under appropriate treatment, leaving no traces behind.

(2) *Perisynovial gummata.*

(3) *Arthritis*, due to osseous nodes or gummata in the neighborhood of the joints.

(4) *True Chronic Synovitis.*—This is the most common form of syphilitic arthritis.

(5) *Syphilitic chondro-arthritis* (Virchow).

The last four forms belong to the tertiary lesions.

Symptoms.—It is to be borne in mind that a joint-affection that does not yield to specific treatment is not necessarily non-syphilitic.

Perisynovial gumma attacks frequently the tissues around the knee-joint; it is very chronic in its course and is more common in women.

Arthritis due to osseous nodes has a special diagnostic feature in the severe nocturnal pains. The fourth form of syphilitic arthritis (true chronic) is the most common among the types due to acquired syphilis, while the symmetric synovitis of the knees occurring about puberty is perhaps peculiar to the congenital cases.

Syphilis of the Testicles.

The lesions are of two forms: (*a*) *Gummata.*—These produce hard, usually nodular, swellings, either single or multiple, and of moderate size, that occupy the substance of the testicle and sometimes the epididymis.

(*b*) *Interstitial Orchitis.*—This is a fibro-sclerotic change that leads to slow, gradual atrophy. Though bilateral, it is usually more marked on one side than the other. *Epididymitis* occasionally develops as a late secondary lesion. It is usually unilateral, painless, and quickly disappears under treatment. In the tertiary stage gummata may develop.

Diagnosis.—In gummatous orchitis the swelling of the testicle is painless, smooth, globular, dense, and heavy, with no tendency to involvement or ulceration of the overlying skin.

In *tuberculous disease* the history and associated lesions differ from those of syphilitic orchitis, and the head of the epididymis is generally affected. Atrophied testicles may be due to congenital syphilis. In such instances typical scars, eye-affections, and the characteristic physiognomy are usually to be noted. Hydrocele may owe its origin to the same cause. Atrophy of the testes may lead to impotency and sterility. Such instances are not to be mistaken for the results of metastasis in *mumps.*

General Diagnosis of Syphilis.—Perhaps sufficient has been said regarding the importance of obtaining a correct statement with reference to the primary infection. On failure to find evidence of a genital chancre, an examination for extragenital primary sores must be instituted, even among children. The *treponema pallidum* may be obtained from the serum of the tonsil in from 80 to 90 per cent. of patients suffering from secondary untreated syphilis (Campbell).[1]

The striking characteristics of the cutaneous manifestations of secondary syphilis are, first, symmetrical distribution; second, polymorphous character; third, non-inflammatory nature, and fourth, raw-ham or dark red color. In this connection two facts need to be emphasized, first, that a syphilitic eruption, either macular or papular, never causes troublesome itching; and second, that a patient with a syphilitic eruption may experience itching due to another cause—namely, eczema or scabies.

Inherited syphilis may be diagnosticated on the appearance in a child under five months of snuffles and the characteristic skin-eruptions.

[1] *Jour. Amer. Med. Assoc.*, May 14, 1910.

Syphilis hereditaria tarda may be recognized either from a retrospective view or from the presence of active lesions and symptoms.

Tertiary manifestations of acquired syphilis embrace these points: 1. The consideration of the fact that obscure cases in general and atypical symptom-groups are often due to the syphilitic taint. 2. Direct information or proof, as the result of careful inquiry, to show that the primary and secondary stages (either one or other, or both) have transpired. 3. The evidence presented by the patient and to be obtained by the careful objective examination of the eyes (for iritic adhesions, etc.), throat and skin (for scars), bones (for necrosis and nodes), and the testes. 4. Certain symptoms are significant, such as nocturnal pains, paralysis of the single cranial nerves, double deafness without otorrhea, etc. 5. The therapeutic test may aid in doubtful cases.

The presence of scars constitutes a most important factor in making a retrospective diagnosis. Recent scars are pigmented, and exhibit a slow, progressive clearing up, until, from four to eight years after infection, they are wholly decolorized, pearly white in color, and smooth. On the other hand, as pointed out by Hyde, eczemato-varicose scars remain stationary. These scars are apt to be found on the scalp and on the anterior surfaces of the legs. They may be single or multiple, and may exhibit certain defined shapes (semilunar, dumb-bell, etc.).

Justus's blood-test for syphilis, which consists in a sharp, transient reduction of the hemoglobin after the administration by inunction or hypodermic injections of mercury, is not pathognomonic, but an aid in diagnosis. It occurs in conditions other than syphilis (Christian and Foerster).[1] Both inherited and acquired syphilis can now be recognized by the serum reactions of Wassermann and Klausner—in from 90 to 95 per cent., according to Butler.[2] Obviously, the Wassermann reaction is of the greatest necessity and diagnostic importance in cases in which syphilis is not recognizable by the ordinary methods of examination—*e. g.*, cerebral or spinal syphilis, syphilis of bones and the internal organs. The reaction being a systemic one, it follows that it is indicative only of the presence of active syphilis. On the other hand, a negative Wassermann reaction does not imply that the *treponemæ* are absent in lesions of a fibrous character, but may mean their temporary disappearance from the lymphatic and vascular structures. It is clear that treatment should not be discontinued simply because the Wassermann reaction is negative.[3] Schmidt recommends that in syphilis of the nervous system, the Wassermann reaction should be done with the cerebrospinal fluid as well as with the blood-serum. The Weil test[4] is more sensitive than the Wassermann in the later, untreated stage of the disease. The cholesterin reaction is valuable as a control to the Wassermann (Matlack[5]). Robinson contends that the Noguchi luetin reaction (intradermic injection of killed cultures of *treponema pallidum*) is specific for tertiary syphilis.

[1] *Univ. Med. Mag.*, Nov., 1895.

[2] *Jour. Amer. Med. Assoc.*, Sept. 5, 1908.

[3] Wassermann's reaction is quite complicated, and for the details of the method itself, the reader must consult special works on diagnosis.

[4] This test is based upon the finding that the blood of syphilitic patients is more resistant to the hemolytic action of weak solutions of cobra venom than is the blood in other diseases, with the possible exception of carcinoma (*vide N. Y. Med. Jour.*, Jan. 6, 1912, p. 23).

[5] *Monthly Cyclopedia and Med. Bull.*, Phila., July, 1912.

General Differential Diagnosis.—Numerous affections and conditions—local and general—are liable to be confounded with syphilis. Mere allusion to some of these common errors of diagnosis can be made here, while others must be omitted altogether:

(*a*) *The primary sore* of the lip has been mistaken repeatedly for cancer. The history and symptoms of syphilis, together with the therapeutic test, must clear up the doubt.

(*b*) Certain *skin-eruptions* (lichen, psoriasis, papular eczema, measles, etc.) may be mistaken for the eruption of secondary syphilis. J. V. Shoemaker[1] details the differential diagnosis in a recent article, which the reader who desires full information may consult.

(*c*) Care must be exercised lest the *specific eruptive fevers*, especially the pustular stage of small-pox, be mistaken for secondary syphilis.

(*d*) The syphilitic arthritis which may develop at the onset of the second stage must be discriminated from *rheumatic arthritis*—an easy task if only the attention be drawn to the primary lesion and the characteristic secondaries in cases of the former disease.

(*e*) Syphilis in the tertiary stage may simulate *chronic gout* or *rheumatism*, and unless there is definite evidence of syphilis on the one hand, or typical rheumatic symptoms and history on the other, the diagnosis may remain indefinitely uncertain. The therapeutic test may aid.

(*f*) *Periosteal nodes*, like those occurring in syphilis, may follow vaccination, small-pox, typhus and typhoid fevers. Here the history and associated phenomena furnish reliable data for discrimination.

(*g*) *Carcinoma of the tonsil* has often been diagnosed, and the tonsils have been excised when really the seat of a syphilitic lesion.

(*h*) Janeway[2] asserts that *chronic syphilitic fever* and *tuberculosis* are not rarely confounded.

Treatment.—(*a*) **Prophylaxis.**—To prevent the transmission of hereditary syphilis infected persons should not marry within four years after the appearance of the primary sore. "Marriage should also be prevented when the patients have not been subjected to a thorough and prolonged treatment" (Porter). If at the end of the third year the patient presents a mucous patch, he must wait one year longer.

A healthy mother may safely be allowed to nurse her syphilitic child owing to her acquired immunity (Colle's law). Should she be unable to suckle the child its prospect of survival is greatly diminished. Wet-nurses should not be employed for syphilitic children, but may be for non-syphilitic, even when the mother is affected. If syphilis appear in the mother during pregnancy, antiluetic treatment should be begun and persisted in even after apparent recovery. After the birth of the child treatment should be continued, if the child be nursed by the mother, with a view to medicating the milk.

As has already been stated, the most frequent mode of infection is irregular and illicit sexual congress, and it follows that absolute moral purity would go further toward the prevention of this widespread malady than any sanitary code or legal restrictions. Physicians cannot too strongly advocate continence. Should prostitution be regulated and controlled by the state? Experience has shown that but a slight control is exercised over the spread of syphilis in countries where system-

[1] *Medical Bulletin*, November, 1893.

[2] *American Journal of the Medical Sciences*, September, 1898.

atic regulation of prostitution is attempted by the state. I am of opinion that the state should maintain some form of sanitary regulation and control, but, unfortunately, to render this efficient demands that prostitutes shall be officially registered. Such a sanitary supervision should consist in the examination of every prostitute at least twice a week, including a microscopic examination of the uterine and vaginal secretions, and the sending of every diseased prostitute to a hospital with a special department for such cases.[2] Palmer suggests that the female offender is usually not aware of the existence of a primary sore, while the male is; hence the latter should undergo inspection also. Inspection of prostitutes, however, unless rigid and careful, is absolutely valueless. Chancres are often concealed from view in the vagina or upon the lateral aspect of the os uteri. The maintenance of legal brothels, however, is not here recommended, either from a moral or hygienic standpoint. Experiments have shown that the application of calomel ointment within an hour of inoculation is preventive of infection in man (Metchnikoff and Roux).

(*b*) **Medicinal Treatment of Hereditary Syphilis.**—For syphilis of the new-born, mercury by inunction or in the form of calomel (gr. $\frac{1}{10}$—0.064. t. i. d.) or gray powder (gr. $\frac{1}{2}$—0.0324, t. i. d.), is to be employed. If these babies must be hand-fed the issue is almost unexceptionally bad.

When the first symptoms appear at the second or third month the above method of treatment is generally successful. Among the poorer classes no objection is made to mercurial inunctions, and these are often preferable. The ointment may be rubbed into the armpits, thighs, or sides of the abdomen, which should be covered with a flannel roller. The parts must be kept clean; and the mouth washed after nursing with a 3 per cent. solution of boric acid. Shaw prefers to treat infantile syphilis by inunctions, because of the digestive disturbances usually following the internal administration of mercury to children. *Syphilis hereditaria tarda* is best treated by the use of potassium or sodium iodid. To the iodid may be added mercuric chlorid in suitable doses, though the latter may sometimes disagree (Roberts). In addition to the specific therapy, tonic measures are usually indicated.

(*c*) **Treatment of Acquired Syphilis.**—There is a specific plan of treatment which should be commenced as soon as the appearance of the secondaries has set the diagnosis of the given case at rest. This is the use of mercury, and rarely of potassium iodid also. The instances in which the latter alone is to be administered are rare. Fournier's "chronic intermittent treatment" of syphilis—which consists in continuous medication for two or three years with mercury and iodin alternately—is warmly advocated by some syphilographers; but the continuous mode is, in the opinion of most specialists, of greater advantage to the patient. Unless mercury disagree or the patient is exceedingly susceptible to its physiologic effects, I use it persistently during the secondaries, and later at intervals until the end of two years. It is a protracted course, and a protracted course only, of the specific treatment that suffices if we would obviate the dread ravages that otherwise are so apt to appear. I usually employ the protiodid (gr. $\frac{1}{8}$–$\frac{1}{3}$—0.008–0.021, three times a day), and later the biniodid (gr. $\frac{1}{30}$–$\frac{1}{24}$—0.0021–0.0027, three times a day). We should begin by giving one pill three times daily, and increasing one pill each day until the premonitory symptoms of ptyalism appear (ten-

derness when the teeth are knocked together, and ropy saliva); then the pills should be reduced ½ or ⅓, depending upon the number taken. By this procedure the physician is able to ascertain for each case the largest dose of mercury that can be given without harm. Hutchinson recommends the gray powder given in pill-form, combined with Dover's powder (āā gr. j–0.0648), this pill to be taken from four to six times daily. A well-known mixture, prescribed in dispensaries, contains mercuric chlorid and potassium iodid in combination.

Inunctions of mercurial ointment (ʒss—2.0, night and morning) produce excellent results, and it is advisable in cases in which the syphilids yield unsatisfactorily to internal dosage to suspend the latter at intervals of six or eight weeks and give a course of twenty inunctions. White advances the view that in the later stages, with the involvement of the deeper tissues, the combined use of inunctions over the affected region with potassium iodid internally often seems to have distinct advantages as compared with the administration of the "mixed treatment" by the mouth. It is necessary to omit the inunction once in seven or eight days for one day, and to take a warm bath to aid in the elimination of the mercury.

The hypodermic use of mercury in syphilis is to be adopted only when very prompt action of this agent is desired. Several preparations are used, and whether these are soluble or insoluble is a matter of little moment. The bichlorid takes first place, the dose being gr. ¼ (0.0162), in 15 to 20 drops of water, twice a week. Calomel probably holds second place (dose, gr. j—0.0648—in 15 drops of glycerin, twice a week). Among other preparations employed are the albuminate of mercury and gray oil. All injections must be made deeply into the muscles. The subcutaneous injection of sterilized serum for the blood of lambs and calves has been successfully practised by Tommasoli.

The method of fumigation has gained favor in the treatment of syphilis, particularly in institutions on the Continent. Lane recommends that calomel (ʒiss—6.0) be put in a china bowl about half filled with water; a spirit lamp is placed under this, and the patient, "sitting above it wrapped in a cloak, has a deposit of mercury settle all over his body as the calomel is sublimed." He should remain wrapped in the cloak for one hour, take a fumigation once daily, and remain indoors. From six weeks to three months are necessary to effect a cure.

It is almost universally agreed that the new preparation, "606," offers incredible possibilities in the treatment of syphilis, and yet the immense majority of writers are of the opinion that it should not be used in every instance of the disease. The classes of cases in which its employment would appear to be entirely justifiable are—(*a*) recent cases, in which mercury or iodides have not been used; (*b*) those in which mercury and iodides have failed; (*c*) where the Wassermann reaction confirms the diagnosis in suspicious cases, and also where it discloses hidden syphilis. The question of its permanency of curative action has not, as yet, been definitely settled. The effects of the remedy must vary with the stage of the disease, the size of the dose, and its methods of administration.

The mode of introduction into the organism is important, and Fordyce states that the order of effectiveness is as follows:

"1. Intravenous injection of a very dilute alkaline solution.

"2. Intramuscular injection of an alkaline solution.

"3. Acid solution (monochlorhydrate or dichlorhydrate).

"4. Neutral emulsion (intramuscular or subcutaneous)."

The method of injecting the drug in neutral suspension or emulsion subcutaneously gives less permanent results than by the other methods, although it produces the least amount of pain. The technic for 2, or intramuscular injection of an alkaline solution, is as follows: Take a graduated cylinder with ground-glass stopper, add "606" salt; immediately add 15 c.c. hot water, shake vigorously until every particle of the salt is dissolved; then add 2 c.c. normal sodium hydrate (NaOH) solution; a precipitate occurs. Then continue to add sodium hydrate solution in very small quantity, shaking vigorously after each addition, until the solution begins to clear; then drop by drop, until we have a clear solution. This should be neutral or slightly alkaline; if the cylinder does not contain 20 c.c. of solution, sterile water is added up to that amount. Then 10 c.c. of this solution is injected deep into the buttocks on either side, always taking care to cleanse the parts with soap, water, and iodin.

The intravenous method gives the most satisfactory results and the preparation of the solution employed follows: "Into a graduate holding 250 c.c. drop 10–20 c.c. of sterilized water. Add the required dose of '606,' and mix thoroughly until there is a clear solution; add sterile water or, better, normal salt solution to the 100 c.c. mark; then add pro 0.1 of '606,' 0.7 of normal sodium hydroxid solution, and mix thoroughly until the precipitate is thoroughly redissolved. If after thorough mixture the solution is not clear, add a few drops of the sodium hydroxid solution to produce this, and then add sufficient normal salt solution to make 200–250 c.c. The fluids used are all to be warm. The alkaline mixture is then ready for injection. The Cassel syringe and apparatus supplied for this purpose are preferable, for by their use the dangers of introducing air are reduced, if the operator continues cautious and follows the directions given in the original paper of Schreiber."[1]

The dose to be administered varies, according to different clinicians, from 0.3 to 0.7 gramme subcutaneously or intragluteally, while 0.3 to 0.5 gramme is used intravenously. In cases in which the combined intravenous and subcutaneous or intragluteal methods are employed, as much as 0.9 gramme should be used. McDonagh has found that three to seven injections are necessary to cure most cases of syphilis.

The remedy is contraindicated in organic valvular affections of the heart, advanced arteriosclerosis, locomotor ataxia, non-luetic retinal and optic lesions, chronic Bright's disease, the acute infections (including bronchitis), pulmonary disease (except tuberculosis), and advanced diseases of the brain and cord.

The teeth should be cleaned thrice daily. *Hygiene* plays no mean rôle in the successful management of syphilis. The *diet* must be liberal, though green vegetables and fruits are not to be taken. Alcohol and tobacco are the two great enemies of the luetic.

Auxiliary measures, when other lesions are associated, are important. At present, atoxyl and sodium cacodylate as remedies promise much. If syphilis occur in a tuberculous subject, it is of great value to add the potassium iodid to the mercurial, and, if active tuberculous lesions are present, cod-liver oil and creasote as well. Anemia and debility call for

[1] Schreiber, *München. med. Wochenschr.*, 1910, No. 39.

iron and a tonic plan of treatment generally. Attention should be given to the stomach, bowels, kidneys, and other organs. At all times it should be borne in mind that the patient, as well as the disease, is to be treated.

In women the iodids should be suspended during menstruation if the flow of blood is excessive, but not the mercury. Says Mauriac: "During pregnancy specific treatment is well tolerated, and often requires to be pushed to a point a little short of intoxication for the good of both the mother and the child, close watch being kept upon the kidneys, suspending treatment at the first sign of albumin."

(*d*) **Treatment of Tertiary Syphilis.**—For most tertiary manifestations, including visceral syphilis, we have a therapeutic specific in potassium iodid. This should be used alone, the inunctions of mercury being added if the iodid fails to produce the desired result. I give the potassium iodid in a saturated solution, one minim being equal to $\frac{3}{4}$ grain of the salt. I use gr. x (0.648) t. i. d. at the first dose, and increase the latter 1 grain (0.0648) each day until the manifestations for which it has been prescribed disappear or iodism is induced. It is best given in milk. In cases showing cerebral symptoms it is to be cautiously used, and it is then my custom to combine the iodid with potassium bromid.

In hepatic syphilis the mercurials are usually combined with iodids from the start, and particularly calomel if there be ascites or jaundice.

In nervous syphilis, especially in the graver forms, I begin with large doses (gr. xx—1.296, three times a day), and augment as above indicated. The limit of doses depends upon the effect produced. I have often found sodium iodid to agree better with the stomach than the potassium salt. Mercury should be administered preferably by inunction in combination with the internal use of the iodids in all forms of nervous syphilis. Among unpleasant effects are coryza, conjunctivitis with edema of the eyelids, salivation, and certain skin-eruptions (erythema, urticaria, etc.). In this form of syphilis the specific treatment is made more effective by attention to hygienic measures—fresh air, appropriate diet, and rest.

SPIRILLOSIS.

(*Febris Recurrens; Relapsing Fever.*)

Definition.—An acute infectious disease caused by the spirillum of Obermeier, and characterized by febrile periods which usually last six days, and are separated by a febrile period of the same duration. Manson suggested the term *spirillosis* for this disease, since "relapsing fever" covers "a number of infections, spread probably by a corresponding number of previously unsuspected ticks or other blood-suckers."

Historic Note.—The first accurate account of this affection was published in 1739, though it is known to have prevailed in Europe and Ireland prior to that period. During the next century numerous epidemic outbreaks, more or less extensive, occurred, and in 1844 the disease made its first appearance in America at the Philadelphia Hospital, being brought by immigrants from Ireland. Subsequently small groups of cases occurred, and were reported by Flint and others, and in 1869

it prevailed considerably in Philadelphia (where it was studied especially by E. Rhoads and William Pepper) and in other large cities of the country. This was the last appearance of the disease in the United States.

Pathology.—The solid organs of the body present no characteristic anatomic changes, though when death occurs during the febrile period the various viscera (heart, liver, kidneys) are the seat of cloudy swelling, and sometimes of hemorrhagic infarct and extravasation. The *spleen* shows the most constant alterations, being enlarged, but in size it exhibits a great variability. Infarction is frequent, and the lymphoid element of the bone-marrow often shows hyperplasia.

Etiology.—**Bacteriology.**—In 1873, Obermeier discovered in the blood of patients suffering from relapsing fever a special organism, the *spirillum Obermeieri*, until recently classed with the bacteria, but now placed by Schaudinn and others with the flagellate genus trypanosomata. It is a delicate filamentous organism of spiral form and much elongated, its length equalling four to six times the diameter of a red blood-corpuscle (Fig. 30). Examined under the microscope during a pyretic period, it is seen to exhibit active motion among the blood-cells, this motion being spiral and following the long axis of the organism. It is aërobic, and may best be demonstrated in *dry blood* by staining with Wright's stain. It is apparent in the blood only during the paroxysms, and Dr. Van Dyke Carter's careful studies have shown that by inoculation of the blood containing spirillar organisms the disease may be conveyed to new or old subjects. Shortly before the crisis the spirilla disappear from the blood, and are, as a rule, absent during the whole of the succeeding apyrexial period. After death they are found in all the organs, but they have not been cultivated successfully on artificial media.

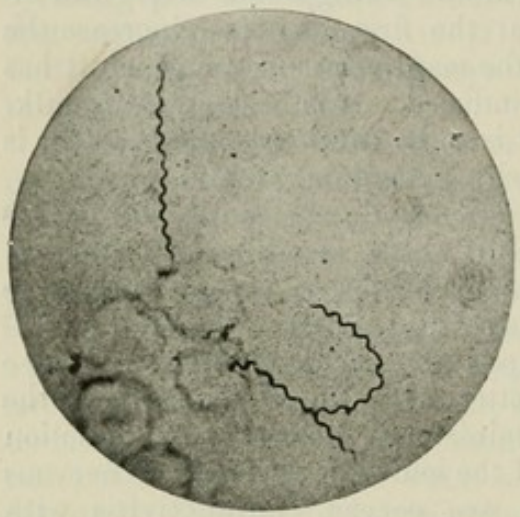


FIG. 30.—Bacillus of relapsing fever (from human blood); × 1000 (Günther).

Predisposing Causes.—*Age.*—The complaint is most common in young adults between fifteen and twenty-five years.

Sex.—A larger proportion of males than females is affected.

Famine and antihygienic surroundings are potent predisposing causes.

Mode of Infection.—Tiotin's studies indicate that the medium of transmission may be through suctorial insects (as bedbugs). Mackie[1] observed an epidemic of relapsing fever in which the pediculus corporis played a part in the transmission of the disease. A well-marked percentage of the lice taken from the infected ward contained multiplying spirilla. During epidemics nurses and physicians are frequent sufferers, and there is some evidence that the disease may be conveyed by fomites.

Clinical History.—The **incubation period** ranges in its duration from four to ten days, though sometimes it is even briefer; and in this stage certain symptoms (malaise, fugitive pains) may appear.

The **invasion** is *abrupt*, often occurring on awakening in the morning,

[1] *British Medical Journal*, Dec. 14, 1907.

and commonly the attack is ushered in with a severe *rigor*, though there may be only a repeated slight shivering. The chief accompanying symptoms are frontal headache, vertigo, severe pains in the loins and limbs, and marked prostration. The *temperature* rises soon, and often rapidly, reaching 105°–106° F. (41.1° C.) on the first or second day. The *skin* is dry and pungent, and presents very soon either a "characteristic dirty-yellow color" or a distinctly bronzed appearance. The *cheeks* are flushed, the eyes sunken, and profuse perspiration often takes place, in consequence of which sudamina are frequently observed. Other forms of eruption have been described, but none that are either constant or characteristic. In certain epidemics *herpes labialis* has been noticed. At first the *tongue* is moist and coated with a yellowish-white fur, and later it may become brown, dry, and fissured, with sordes on the teeth. *Ulcerative stomatitis* has been observed occasionally, and catarrhal pharyngitis and mild tonsillitis may be evidenced by pain on swallowing. Among the *earlier* symptoms are excessive thirst, anorexia, nausea, and vomiting. The vomitus may be yellowish-green, green, or even black in color, and consists partly of bile (rarely, also, blood) and gastric secretions. Constipation often precedes invasion, and is apt to continue throughout the attack.

The *pulse* rises rapidly with the temperature, though the normal ratio between the two is not maintained. The pulse is full and strong, and its beats number from 100 to 140 or more per minute; but in serious cases it becomes weak, irregular, or even intermittent, while at the same time the heart-sounds grow more and more indistinct. Hemic *murmurs* may be audible. The *nervous* manifestations are not of a grave character, but the headache persists, is severe throughout, and the patient is restive and sleepless. Delirium is not common, excepting only in rare cases toward the crisis, and the intellect remains clear, as a rule. The *urine* presents the ordinary febrile characteristics, and may contain albumin and casts. It also contains bile-pigment when jaundice is present. The *respirations* are accelerated, and urgent dyspnea may precede the crisis.

The **physical signs** during the febrile paroxysms are few. The epigastric region and the nerve-trunks are tender to the touch, while the skin-surface and certain muscles are often hyperesthetic. *Palpation* detects a variable degree of enlargement of the spleen and liver, and the signs of bronchitis, of lobular pneumonia, and of hypostatic congestion of the lungs may be present. The symptoms above detailed persist with slight daily fluctuations of temperature until the crisis.

The Crisis.—This occurs from the fifth to the seventh day, and rarely as late as the tenth. It is sometimes heralded by a critical rise of temperature, the mercury touching 108° F. (42.2° C.), but evidenced chiefly by a rapid fall of temperature (within twelve hours) to or below the normal, with profuse sweating. Coincidently, all other symptoms disappear with marvellous rapidity. The critical sweat may be replaced by diarrhea, intestinal hemorrhage, metrorrhagia or epistaxis, and after the lapse of a day or two the patient expresses himself as being well.

During the *intervals* between the paroxysms the skin may exhibit a faintly jaundiced tint; there may be trivial evening exacerbations of temperature, particularly if complications be present and outlast the fever stage; and the spleen is evidently enlarged. There may be, rarely, but a single paroxysm. As a rule, at the expiration of the second week, a recurrence of all the active symptoms of the primary attack

occurs, including the rigor and fever. Quite frequently a third pyrexial stage takes place, and rarely a fourth or even fifth.

The **duration** of the first relapse is briefer than the primary pyretic stage, and if there be subsequent relapses, each succeeding one is separated from its predecessor by the usual apyrexial period, but is briefer and lighter. Hence, should a fourth or a fifth febrile period occur, it is, as a rule, quite rudimentary. The relative duration and severity of the different febrile periods, and their manner of recurrence, are best appreciated by a glance at the temperature-chart (*vide* Fig. 31).

Complications.—These are not frequent. At the head of the list stands *lobar pneumonia*, and next comes *broncho-pneumonia*, which is always secondary. Rupture of the spleen may occur. Other complications are septico-pyemic processes, hemorrhagic nephritis, hematuria, iritis, irido-choroiditis, parotitis, laryngitis, entero-colitis, and neuritis. In pregnant women abortion may take place. Epistaxis has been noted, and may prove dangerous. As the result of the very high temperature and toxemia, the heart may become suddenly paralyzed.

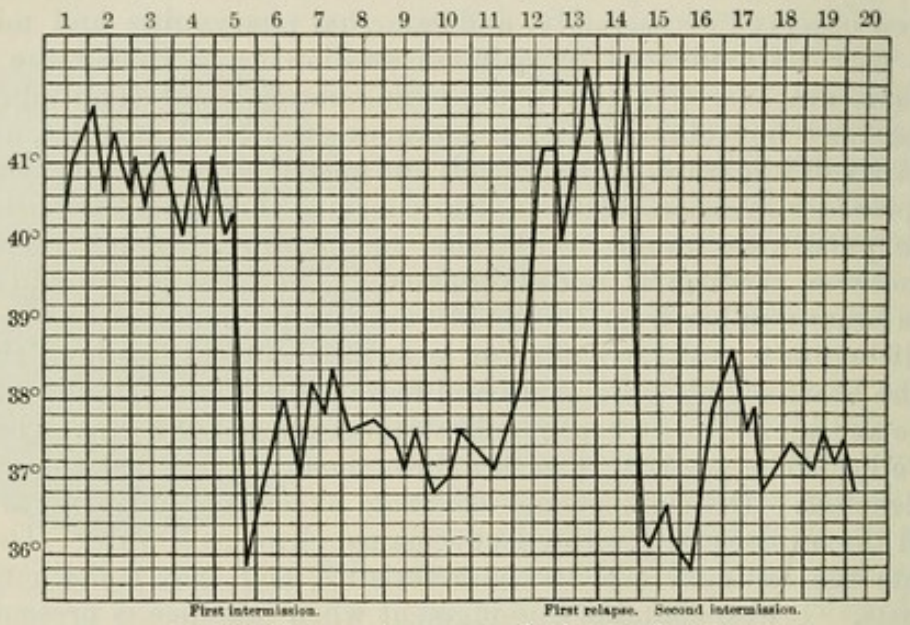


FIG. 31.—Temperature-curve of relapsing fever.

Clinical Varieties.—The difference in the general course of cases in different epidemics, and even in the same one, is, for the most part, the direct result of the varying degrees of intensity of the infection. Thus very *light* or even rudimentary cases occur in which the whole course may be made up of one or two brief febrile periods, and their resemblance to ordinary intermittents may be close. The so-called "*bilious typhoid*," which is a form of relapsing fever, occupies the other extremity, being of malignant type. The symptoms are greatly intensified; but more often, perhaps, the condition early merges into a typhoid state, to which are added certain grave features and complications (marked icterus, hematemesis and hemorrhages from other outlets of the body, uremia, sudden collapse, etc.). Septic and pyemic processes, including infarctions, are common accompaniments, and the outcome is frequently unfavorable. Novy and Knapp believe the African form, or tick fever, to be due to the *spirillum duttoni*.

Diagnosis.—The prevalence of an epidemic in which the cases present similar symptoms; the sudden onset; the course and intensity of the fever with its concomitants; the termination by crisis on or about the seventh day; and the peculiar manner of repetition of the fever-attacks after an afebrile period of equal duration,—are points that distinguish relapsing fever from other affections which simulate it more or less closely. Additional symptoms that are of special value for diagnosis are—enlargement of the spleen and liver, a negative character of the nervous and a prominence of the gastric phenomena, and jaundice. To be able to state that relapsing fever is positively present the *spirocheta Obermeieri* must be found in the blood, and this is particularly true in the earlier cases of an epidemic, before they have passed through their typical relapses. To demonstrate the presence of this parasite in the blood during the fever-stage is not a difficult task. A drop of blood obtained from the finger-tip is to be examined microscopically without previous dilution. On account of their size and motility the spirilla can be readily detected, and usually the attention of the examiner is first arrested by the peculiar joggling movements of the red blood-corpuscles. Then the real disturbing agents appear as slender spirals with a snake-like motion. Their identity may be confirmed by staining with anilin colors, and, in exceptional cases, by injecting them into the blood of the monkey, in whom they produce the disease.

Differential Diagnosis.—*Typhus fever* may be mistaken for relapsing fever, since both have the same predisposing causes, both prevail epidemically, both are characterized by an abrupt onset, with or without prodromes, and by a continued type of fever. In relapsing fever, however, the eyes are clear but hollowed, the cheeks are flushed, and there is a dirty-yellow tint of skin; in typhus the eyes are injected, the pupils contracted, the face wears a stupid, inanimate expression, and the characteristic maculopetechial eruption. In relapsing fever, delirium and stupor are rare, the period of fever briefer, while the blood shows the presence of the spirillum. In typhus relapses are the exception. *Yellow fever* resembles relapsing fever in its general course, but in the former the stage of remission is both briefer and more incomplete. Yellow fever presents a stage of collapse with black vomit, and jaundice is more intense. The spirilla may be detected in the blood, and there is marked splenic enlargement in relapsing fever.

Pel and Ebstein have described a *febrile condition* which sometimes occurs in pseudoleukemia and simulates that of relapsing fever; but it may be distinguished by the absence of the spirilla from the blood, the general enlargement of the lymphatic glands, liver, and spleen, and the fact that the pyrexial periods do not tend to grow shorter.

Prognosis.—The prognosis of relapsing fever is good, but of "bilious typhoid" it is bad indeed. Apart from the type, we must consider, in this as in all other acute infectious disease, the number, character, and frequency of occurrence of the various complications. As stated, these are few, infrequent, and mostly benign. Among those signalizing danger are severe hemorrhages (epistaxis, metrorrhagia, hematemesis, etc.), premature labor, signs of uremia and syncope, marked jaundice and excessive vomiting, and urgent diarrhea. Perhaps the most frequent causes of death are pneumonia and acute hemorrhagic nephritis.

Individual circumstances render the prognosis more grave—as the want of good nursing, privation, a previously enfeebled system, and old age.

The **duration** depends upon the number of paroxysms, since the latter are of definite length. In most cases there is but one relapse, and in this event the disease lasts from eighteen to twenty days.

Treatment.—Thorough disinfection and isolation must be carried out in relapsing fever. The general management, including the time and use of stimulants, must be based on the same principles as are employed in typhoid fever. The fever, as well as the nervous and other symptoms, is to be opposed by the cold or gradually cooled bath, employed as indicated in the article on the treatment of the latter disease. Cold spongings, with the ice-cap or the cold pack, may be substituted for the baths in special cases. Internal antipyretics may be reserved for use in cases in which the temperature is very high and the above-mentioned means are impracticable. Small doses of phenacetin (gr. ij to v—0.1296 to 0.3240) or acetanilid (gr. ij to iij—0.1296 to 0.1944) are to be administered, at the same time guarding the heart, and the signs of collapse must be promptly met by the free use of stimulants (strychnin, alcoholics, ammonium, etc.). Vomiting often induces marked debility, and calls for the use of ice or iced champagne and small doses of cocain, morphin, or dilute hydrocyanic acid, preceded by a mercurial laxative. Counter-irritation over the epigastrium is also useful. For the intense muscular pain, restlessness, and sleeplessness nothing is so good as morphin given subcutaneously, and Dover's powder may be employed if the pain be of moderate severity. During the intermissions the patient should be kept indoors for ten days or more, lest exposure or sudden exertion predispose him to a relapse. Solid food may now be gradually resumed, and tonics judiciously given. H. Löwenthal[1] treated 131 cases with antispirochetic serum (obtained from a horse that had been treated with blood containing the spirochetæ) with but 1 death. Ardin-Delteil, Negre, and Reynaud feel sure that a specific remedy for relapsing fever has been discovered in salvarsan, which should be employed in massive doses (0.6 gm.–gr. x). The treatment of *relapses* differs in no way from that of the first febrile period.

[1] *Deutsche med. Woch.*, October 27 and November 3, 1898.

PART III.

CONSTITUTIONAL DISEASES.

DIABETES.

(*Diabetes Mellitus.*)

Definition.—A nutritional affection, attended by an abnormal amount of sugar in the blood, and characterized clinically by persistent glycosuria, by polyuria, and by a progressive loss of flesh and strength.

Pathogenesis.—This is still undetermined. Post-mortem lesions of different organs and structures of the body have been met with in diabetes—a fact that has given rise to a variety of views.

(1) That it is dependent upon *organic disease of the pancreas*, especially granular atrophy, or upon marked functional disturbance of this organ. It has been shown experimentally that extirpation of the pancreas is followed by diabetes, and that if one-half the gland remains glycosuria does not result.

It may safely be assumed that total loss of function always, and partial loss sometimes, leads to diabetes. Lepine and Martz have been able to produce a glycolytic ferment by treating the pancreas after their own special method, which need not be detailed here. It is probable that the pancreas, and particularly the cells forming the islands of Langerhans, furnishes an internal secretion containing a glycolytic ferment. This ferment is identical with that which is contained in the blood, and in the presence of which glycogen is assimilated. Croftan [1] has placed the origin of the blood-ferment in the leukocytes, and believes it, with the pancreatic ferment, to be identical with trypsin.

(2) The *suprarenal glands* have assumed a rôle of much importance since the valuable researches of Herter.[2] He found that a solution of adrenalin chlorid injected into dogs or painted on their pancreas, caused marked glycosuria. Other reducing substances acted in a similar manner. On the other hand, excision of the left suprarenal gland with ligation of the blood-vessels of the right gland, caused a reduction in the percentage of sugar in the blood. He concludes that "the suprarenal glands make a secretion which is capable of stimulating the pancreas in such a way as to call forth an increased conversion of hepatic glycogen into sugar." And further "it seems that this disturbance in metab-

[1] *Amer. Jour. Med. Sci.*, April, 1902. [2] *Medical News*, October 25, 1902.

olism (glycosuria) is in some way dependent on interference with the oxidative activities of the cells of the pancreatic gland."

(3) If the glycogenic function of the *liver* be interfered with materially, diabetes follows. This may result from organic hepatic disease or a faulty nervous system. Puncture of the floor of the fourth ventricle will also cause glycosuria, and section of the pneumogastric nerve is followed by paralysis of the hepatic vessels, disappearance of glycogen from the liver, and saccharinuria.

(4) The so-called *alimentary glycosuria* has frequently been induced experimentally by Miura and others. It results from the ingestion of more carbohydrates and peptone than can be stored in the liver as glycogen, so that some of the latter finds its way into the hepatic vessels with resulting glycosuria.

(5) The administration of *phloridzin* produces glycosuria both in animals and man. There are two views as to the cause of phloridzin diabetes: (*a*) that the kidneys, owing to the action of the phloridzin on the renal epithelium, eliminate the sugar from the organism; (*b*) that an excessive formation of glucose occurs (generally held).

(6) *The Microbic Theory.*—Ernst and others have observed all the forms of fungi, but as yet no etiologic relationship has been shown.

(7) Another view regarding diabetes is that the carbohydrates of the food are converted into fat by the protoplasmic action of cells in the intestinal villi, and enter the system in the same way as do fats taken as such. The surplus carbohydrates that escape the action of the cells of the villi are transmuted into glycogen in the liver. The glycogen stored in the liver obviously forms fat also, since this organ has some fat-forming function, and there are thus two barriers preventing the carbohydrate matter from entering into the blood, and if either is deranged, hyperglycemia with consequent glycosuria results.

The influence of the nervous system is undoubted, and Pavy[1] claims that the disease is essentially a neurosis affecting a particular part of the vaso-motor system. In light cases the carbohydrates are not warehoused in the liver and muscles, and the excess of glycogen not burned up in the tissues, so that from the storage reservoirs (liver and muscles) the blood is supplied with an excess of grape sugar. In severe cases the carbohydrate moiety of the proteids of the food or tissues furnishes the sugar excreted in the urine (Stengel).

Pathology.—The *pancreas* in more than one-half the instances shows morbid changes. Opie's[2] researches, since confirmed by other observers, indicate that the important lesions are those affecting the islands of Langerhans. These peculiar structures appear to have a different function from that possessed by the other pancreatic cells, and are probably the source of the internal secretion of the pancreas. In *chronic interstitial pancreatitis* of the *interlobular* type, the islands of Langerhans are affected only late, and glycosuria is rare. The changes following occlusion of the pancreatic duct by calculus, growths, etc., are of this variety. In the *interacinar* type the cells forming the islands

[1] *Lancet*, May 3, 1906.

[2] *Jour. of Exper. Med.*, V., No. 4, 1901.

of Langerhans are affected early, hyaline degeneration of the capillaries may be seen, and there is frequent and early glycosuria. In Herter's[1] experiments the injection of fatal doses of adrenalin was followed by granular degeneration of the islands of Langerhans.

Acute necrosis of pancreas, primary cancer, and diffuse cancer may cause glycosuria, but rarely. The *liver* is often enlarged and fatty, particularly the zones corresponding to the distribution of the hepatic artery. According to French writers, there is a diabetic cirrhosis of the organ (*cirrhose pigmentaire*), the pigment being derived from destroyed blood-cells. *Microscopically,* the liver-cells are found to be enlarged, nucleated, and globular in outline. Rindfleisch holds that these changes are most striking in the peripheral portion of the lobule.

The Kidneys.—A well-marked chronic interstitial nephritis, with fatty degeneration, is often present. The tubal epithelium and the vessels of the Malpighian bodies may show a hyaline change. More commonly the appearances are those of an ordinary catarrhal nephritis.

Nervous System.—In rare instances organic disease of the medulla (tumors, sclerosis, etc.) is found. Changes in the posterior columns of the cord have been noted, and a peripheral neuritis, simple or multiple, is commonly seen. The so-called *diabetic tabes* is generally supposed to be due to multiple neuritis. Extreme hyperemia and edema of the meninges was found in all of 8 cases of diabetic coma (Hanssen).

The Lungs.—The commonest lesions in the lungs are gangrene following pneumonia and the so-called diabetic phthisis. Fatty emboli are found in the pulmonary vessels.

The Heart.—Arterio-sclerosis with cardiac hypertrophy is often met with, but does not constitute a peculiar lesion.

The Skin.—Cutaneous pigmentation (diabetic bronze of the French), more or less uniform, has been reported in 9 cases (Hanot and Chauffard). It is associated with hypertrophic cirrhosis of the liver.

The Stomach.—Dilatation and, according to Jacobson, marked catarrhal changes are common in the early stage.

The Blood.—The normal proportion of sugar in the blood (0.15 per cent.) is increased, though there is no immediate connection between the percentage of sugar in the blood and the urine in diabetes. Both in experimental and pathologic diabetes hyperglycemia may be marked, with moderate or slight glycosuria, and Lépine has shown that diuretics diminish hyperglycemia by increasing the glycosuria. The blood-plasma contains much fat. It is probable that the albuminoid matters in the blood may produce glucose. Glycogen probably exists in the blood-corpuscles, and not in the plasma, "where it would be destroyed by the diastasic ferment" (Dastre), and it is a normal element of the blood, apparently belonging to the leukocytes (Huppert and Czerny). The alkalinity of the blood is diminished, probably owing to the presence of oxybutyric acid. The corpuscles show no special alterations.

General Etiology.—(*a*) *Heredity* is generally believed to exert a predisposing influence, since cases are observed to succeed one another in the same family. (*b*) *Season* also exerts an influence, diabetes appearing more frequently in the months of March, April, July, and Novem-

[1] *Medical News*, May 10, 1902.

ber (Davis). (*c*) *The male sex* suffers much more frequently than the female. Wegeli, however, found in 107 cases that children of both sexes were affected in an equal proportion. (*d*) *Age.*—Most cases occur between thirty-five and sixty years of age. Infantile diabetes is rare, and occurs most frequently about the age of five, though it has been met with under one year. (*e*) *The Hebrew race* is especially susceptible. The colored race rarely suffers, although of a series of 77 cases 8, or 10.3 per cent., were in negroes (Futcher). (*f*) *The better classes* of society furnish most instances, and particularly that large element composed of neurotic subjects. (*g*) *A nervous shock* or strain or prolonged mental anxiety acts as a predisposing cause. (*h*) *Occupation.*—The urine of 607 individuals engaged in manual labor that required great muscular and respiratory activity showed no sugar in any case; while the urine of 100 individuals engaged in intellectual work of a more or less fatiguing character, but always intense and sedentary, showed sugar in 10 of the cases (Worms). (*i*) *Obesity* predisposes, somewhat, particularly to the lipogenic form. (*j*) *Certain chronic diseases—e. g.* syphilis, malaria, gout—predispose. (*k*) *Pregnancy* has a slight though decisive influence. (*l*) It sometimes follows *acute infectious diseases.* (*m*) *Locality.*—Diabetes mellitus is, comparatively speaking, rare in America, although Hare's statistics indicate that diabetes is becoming more prevalent. In certain other countries (Normandy, India, France) diabetics appear to be constantly increasing in number, the mortality in Paris having more than doubled from 1883 to 1892, inclusive. The disease is much more frequent in cities than in rural districts. *Contagion.*—Among 770 cases, Senator saw 9 instances of man and wife suffering from the disease. In a series of 5,000 cases 1.8 per cent. of conjugal diabetes occurred (Schram).

Special Etiology.—Under this head may be arranged the following groups of cases: (1) *Diabetes* due to *pancreatic disease.* (2) Cases occasioned by *hepatic disease* (*organic and functional*). (3) Those comparatively rare instances caused by *disease of the brain* (*tumors, sclerosis, or irritative lesions of the diabetic center*) *and spinal cord.* (4) Diabetes following *traumatism*, and especially injuries to the head. Not infrequently it occurs after injuries to other parts of the body, such as the spine, sacral region, abdomen, etc. In 212 cases of traumatism of the head Higgins and Ogden found 20 cases of glycosuria, though only a small proportion of the cases (2) exhibited a permanent glycosuria. Ebstein,[1] after an exhaustive study of 6 of his own cases and of 44 gathered from literature, concludes that there can be no question of the direct causal relation of traumatic neurosis and diabetes.

Clinical History.—For the sake of accuracy and convenience of description, the cases will be divided into the acute and chronic forms.

1. **Acute Diabetes Mellitus.**—The instances are few and the course is, as a rule, rather subacute than acute, manifesting a predilection for the young and middle-aged. The *onset* is more abrupt than in the chronic form, but the characteristic features do not differ from those of the latter. Many of the cases due to pancreatic disease are of this class. Exceptionally, acute diabetes occurs in the aged.

2. **Chronic Diabetes.**—The symptoms are evolved *slowly* and *gradu-*

[1] *Deutsche Arch. f. klin. Med.*, April, 1895.

ally, as a rule, and prominent among prodromal conditions is dyspepsia or chronic gastric catarrh. We may note certain nervous disorders, such as headache, mental irritability, moroseness, and insomnia, with or without gastro-intestinal symptoms. The patient may suffer merely from general debility and malaise, and either frequent micturition, polyuria, or unnatural thirst is apt to be noticed. Rarely, diabetes has an *abrupt onset*, as after an injury, a sudden nervous shock, or a chill. With the development of the affection the polyuria and thirst become marked, the appetite keen, and glycosuria appears. In spite of the enormous quantities of food taken, progressive emaciation and debility attend.

Leading Symptoms and Complications in Detail.—(1) *The Urinary Symptoms.*—The daily amount of urine varies from four or five pints to as many gallons. In mild cases and in intercurrent febrile attacks it may be slightly, if at all, increased in quantity. The twenty-four-hour specimen should be examined at once, lest yeast-cells develop and cause the sugar to disappear. Its color is pale and its specific gravity ranges from 1020 to 1050, rarely being as low as 1015; it has an acid reaction, a sweetish, aromatic odor, and a distinctly sweetish taste. Sugar is present, the amount varying from ½ to 1 to 2 per cent. in mild cases, to 5 or even 10 per cent. in severe attacks. The amount eliminated in the twenty-four hours varies from five ounces to a pound or more.

Other forms of sugar than glucose (inosite and levulose) may be contained in the urine, and glycogen has rarely been found. The urine may also contain fermentation-products (acetone, diacetic acid, beta-oxybutyric acid). Acetone strikes a Burgundy-red color on the addition of the chlorid of iron. Hirschfeld's studies upon the excretion of acetone in diabetics show that in severe forms an increased amount is excreted, while other writers regard it as being benign. Diacetic acid is probably of graver significance than acetone, whilst the presence of β-oxybutyric acid is a danger-signal of diabetic coma (*vide infra*).

The *urea* is increased, Kaufman finding it in the blood of diabetic dogs to be doubled. *Uric acid* is either normal in quantity or increased, but a large amount of ammonium is present, indicating an increase of organic acids. The phosphates may also be present in greatly increased proportion (Ralfe), and in such cases the glycosuria may be more or less intermittent. This has been described as a special variety—phosphatic diabetes. Lipuria may be present and creatinin is increased.

Slight albuminuria, often with an intermittent tendency, is common even in the early stages, and is not of grave significance. Well-marked nephritis with its characteristic phenomena may develop, though usually in advanced diabetes; and if albuminuria be marked, the amount of sugar excreted may be considerably diminished. The development of chronic interstitial nephritis, however, is not a favorable complication, as some have supposed. Arteriosclerosis may be observed, and pyelonephritis (rarely) and cystitis (not rarely) may appear as complications. A marked reduction in the percentage of dextrose may accompany the development of intercurrent febrile affections. As the result of fermentative processes in the bladder gases may form (*pneumaturia*).

(2) *Digestive Symptoms.*—Although a general feature, *thirst* may be discussed under this head. This symptom may be most distressing,

necessitating the drinking of large quantities of water at frequent intervals both by night and by day. The amount of water taken stands in direct relation to the amount eliminated. Notwithstanding the fact that the increased amount of water is needed to dissolve the sugar, cases of confirmed diabetes are met with in which thirst is not marked. Cases are also encountered in which the amount of urine is large and the percentage of sugar excreted very low. The cause of the unusual thirst is probably an increased systemic demand for liquids.

The *appetite* is abnormally large and sometimes almost insatiable (bulimia), and there may be an intense craving for carbohydrates. I have, however, met with instances of diabetes in which the appetite was not inordinate. Considering the quantity of food consumed, the digestion is often surprisingly good, but the association of dyspepsia and diabetes is by no means an uncommon one. The stomach may be found enormously dilated at times, yet functionating normally. There is constipation, though brief intervening attacks of diarrhea may occur.

The *tongue* is generally dry, large, often presenting a rough and fissured surface, and it may either be coated or red and glazed. The gums sometimes swell, and may ooze blood. The saliva is scanty and its reaction persistently acid, while the salivary secretion may show sugar on testing. The teeth decay, and aphthous stomatitis or thrush may attack the oral cavity.

The *liver* is frequently somewhat enlarged, though the biliary secretion usually is not disturbed; jaundice may, however, arise as a complication. Marie has given a description of *pigmentary "hypertrophic cirrhosis* with diabetes mellitus," of which only 9 undoubted cases have been published. It appears late in adult life, and, in addition to the symptoms of diabetes mellitus, slight ascites, considerable hypertrophy of the liver and spleen, with brown or even gray-black cutaneous pigmentation, are among the chief features noted. There is no true icterus as a rule, but the urine is highly colored and contains bile-pigments. Bernoulli[1] reports 41 cases of so-called bronze diabetes.

(3) *Cutaneous Manifestations.*—Diabetic urine, on account of the sugar it contains, has irritant properties, and often produces in the female *pruritus vulvæ*, a troublesome symptom and one that should excite suspicion of this disease. In the male, *balanitis* often occurs, due to the effect of the decomposing urine, and from the same cause the genitals and adjacent cutaneous surfaces may be the seat of *eczema*, particularly in women. General pruritus, due to irritation of sensory nerves by the glycemia, may be observed. The skin is usually harsh and dry, though rarely copious perspiration may be observed, and particularly if phthisis be a complication. The hair often falls off, and in one of my cases *onychia* with shedding of the nails occurred. Among the commonest of the early cutaneous symptoms are *furuncles* and *boils*. Later large *carbuncles* often appear. *Gangrene* (especially of the feet) due to arteriosclerosis is not infrequent, and *edema*, arising independently of nephritis, is not uncommon. Morris has reported 21 cases of xanthoma diabeticorum.

(4) *Nervous Symptoms.*—Diabetic coma is the most important symp-

[1] *Correspondenz-Blatt für Schweizer Aerzte*, Basel, July 1, 1910.

tom, marking a fatal termination in more than half the cases. It is of most frequent occurrence in instances showing rapid wasting and in the young, and is heralded by a fruity odor in the exhaled breath and in the urine. The polyuria and glycosuria lessen, while acetonuria increases as a rule. The tolerance for the carbohydrates is increased (Hirschfield). The cases may be brought under six heads:

Group 1. To this belong abortive forms that terminate in quick recovery. This process may be repeated several times at intervals, and at last a fatal coma may supervene.

Group 2. Perhaps the largest group, in which the diabetic coma follows some form of exhausting exercise. It may end fatally in a few hours or, though less frequently, in three or four days.

Group 3. This is a comparatively small class, and is characterized by collapse of the circulation (small, rapid, feeble pulse, cyanosis, etc.), leading to coma. It is induced either by over-exercise or by intoxication. I have seen 2 typical instances, but feel that it may be questioned whether most of these cases should be classed as diabetic coma.

Group 4. Without previous dyspnea or distress there appear such symptoms as headache and signs of intoxication, and these are followed quickly by deep and fatal coma (Frerichs).

Group 5. Here diabetic coma is preluded by symptoms of some localized disorder, such as gastro-enteritis, pharyngitis, pneumonia, gangrene, or carbuncle. The attack sets in with headache, delirium, distress, and dyspnea both inspiratory and expiratory. Cyanosis may develop early, and, if so, cardiac failure precedes the coma. The duration is from one to five days. This group, which was first described by Frerichs, may have a different onset, and I have seen two fatal cases, one attended by carbuncle, the other with gastric symptoms.

Group 6. Hirschfeld has recently described a class of cases in which we find, in old persons, a moderate glycosuria and coma supervening under the influence of gangrene or carbuncle.

The *causes* of diabetic coma are still obscure. Hirschfeld points to insufficient nutrition from an exclusive meat diet as a factor. Kussmaul believed diabetic coma to be due to acetone. Kulz, Stadelman, and others have more recently found β-oxybutyric acid in the urine, and this is now generally held to be the immediate excitant of diabetic coma (which is an acid-intoxication). This acid results from an increased destruction of the proteids. Lastly, coma must sometimes be of uremic origin, and Herrick[1] emphasizes the fact that casts are very common. Cases that follow suppuration and gangrene may be septic in nature.

Peripheral neuritis is common. The most frequent form is *diabetic tabes*, indicated by an absence of the knee-jerks, darting pains, paresis of the extensors of the foot, and by the steppage gait. Other symptoms may be numbness, tingling, and certain trophic disturbances—shedding of the nails and perforating ulcer of the foot. R. T. Williamson found the knee-jerk absent in 25 of 50 cases recorded; and in 18 of 21 cases of diabetic coma. Schupfer[2] attributed absence of patellar reflex to toxic effects in most cases. Neuralgia

[1] *Jour. Amer. Med. Assoc.*, January 26, 1901.
[2] *Soc. Lancisiana Roma*, January 24, 1898.

may be a troublesome symptom, particularly when it is of the symmetrical sciatic type, and it points to neuritis. The same is true of paraplegia, a condition that may be met. Herpes zoster may be observed.

Psychopathia (*e. g.* irritability of temper, hypochondriasis) may sometimes be present, and temporary hemiplegia has been noted.

(5) *Special-sense Symptoms.*—Not infrequently cataract develops, leading to blindness. Its cause is not clear. Transient ptosis and strabismus are seen, and among other ocular conditions are optic-nerve atrophy, iritis, retinitis (often due to associated nephritis), and hemorrhage. Amaurosis is rarely observed. Among the aural symptoms I would mention otalgia, otitis media, and mastoid disease.

(6) *Muscular Symptoms.*—In diabetics there is a tendency to cramps, especially in the calf of the leg, that appears during the night and on waking in the morning. Unschuld found it present in 33 out of 109 cases. Another variety of cramps that may appear at any hour of the day may occur with the so-called "gastric crisis." In these attacks colicky pain in the epigastrium with vomiting and fever attend.

(7) *Respiratory System.*—Serious pulmonary complications may appear in the advanced stages. The most frequent is pulmonary tuberculosis, which has the customary termination, and does not differ from the usual form of the disease. A second, quite frequent complication is gangrene (circumscribed or general). The peculiar offensive odor of the expectoration may be wanting here. A serious form of *pneumonia* (lobar or lobular) sometimes occurs, and may terminate in gangrene.

(8) *Circulatory System.*—The *pulse* may be of natural frequency and tension. In other cases it is somewhat slow, and the tension may be increased; this is often due to an associated arterio-sclerosis. The heart is sometimes quite weak. The rate of the pulse, therefore, varies greatly: it may be slow (brachycardia), not exceeding 40 or 50 beats per minute, or it may be accelerated. Dyspnea, a tendency to syncope, and gastric disturbance may be seen in combination. Symptomatic anemia is present, and, in most cases, a relative lymphocytosis.

(9) *Sexual Symptoms.*—Impotence may be an early symptom; it is often of great diagnostic significance. Diabetes may be acquired during pregnancy: *per contra*, the diabetic may conceive, though rarely, and bear a healthy child; but death of the fœtus occurs in about one-half of the cases. Premature delivery occurs in many cases (26 per cent.). After delivery the condition is generally aggravated.

(10) *Constitutional Symptoms.*—Usually there is a constantly increasing loss of flesh and strength. In the mildest types, however, good bodily nutrition and a fair degree of strength may be maintained. When emaciation is progressive the polyuria is apt to be proportional. The *temperature* is at first normal, later usually subnormal, though intercurrent febrile attacks, due to complications, are often witnessed.

Clinical Varieties.—(*a*) **Infantile Diabetes.**—Heredity, traumatism, and convalescence from severe acute infectious disease are the chief causes. The type is severer and the course shorter than in adults. A comparatively mild chronic form, however, is rarely met in children.

(*b*) **Pancreatic Diabetes.**—This is a grave variety, and may present evidences of pancreatic involvement. There may be epigastric pain; the fats are poorly assimilated; and the physical signs may rarely point to pancreatic growth. Fitz noted that out of 166 cases treated in the Massachusetts General Hospital fatty stools were not recorded in any instance. Marked polyuria and great thirst may be absent.

(*c*) **Alimentary or Lipogenic Glycosuria.**—This is caused by dietetic errors, and especially by excesses in eating and drinking, combined with physical inactivity. Block experimented on 50 patients, and found that the amount of grape-sugar that could be given before glycosuria appeared differed widely in different diseases. Frequently the smallest quantity was required in *nervous diseases*, and particularly in cerebral affections and hysteria. This form of the disease is often a temporary affair. The percentage of sugar in the urine is usually small.

Prognosis.—In acute diabetes the *duration* varies from a few days to eight or ten weeks, while in chronic diabetes the course ranges from one or two to five or even ten years. When the disease commences in the declining period of life, the course is longer still. The severe forms are generally fatal, and occur, as a rule, at an early period of life and in persons with an hereditary taint. The mild types and those that occur later in life offer a more hopeful prognosis, and in certain cases the withdrawal of all carbohydrates from the diet will cause the sugar to disappear from the urine. Of the special varieties, *alimentary glycosuria* is altogether favorable in its course, *traumatic diabetes* somewhat less so, while the prognosis of the *pancreatic form* is quite unfavorable.

Stout persons bear saccharine diabetes better than lean. Diabetes in gouty subjects often pursues a favorable course. Pre-existing affections may render the prospect gloomy, and certain complications indicate grave danger (coma, phthisis, gangrene, pneumonia, cardiac weakness, nephritis). Of 108 such cases, 64 per cent. terminated fatally (Wegeli), and between the ages of four and five years 20 out of 29 cases perished. The appearance of β-oxybutyric and diacetic acids in the urine is of serious omen. Cases showing heredity give an increased mortality-rate.

Diagnosis.—Diabetes is distinguishable by means of (1) its causal influences and its pathologic antecedents and relations; (2) its gradual onset, by certain suspicious symptoms (*e. g.*, debility, impotence, symmetrical sciataca, cataract, furunculosis); (3) the persistent presence of glycosuria, polyuria, and, later, acetonuria and albuminuria; (4) the inordinate thirst and appetite; (5) cutaneous boils, carbuncles, gangrene, pruritus vulvæ, balanitis; (6) neuritis (especially double sciatica), diabetic tabes, and coma; (7) muscular cramps; (8) special complications; and (9) the long course with slowly progressive asthenia and wasting.

In suspicious cases, even before the discovery of sugar in the urine, grape-sugar may be administered for diagnostic purposes. If glycosuria result, the cases are to be treated just as in pure diabetes. Transient glycosuria, however, is not the genuine affection. Grape-sugar must be eliminated for weeks, months, or years (von Noorden).

Blood-test.—Williamson's blood-test, depending upon the power of diabetic blood to change a warm alkaline solution of methylene-blue to a dingy yellow color, has decided value. It occurs constantly.

Treatment.—1. **A properly regulated diet** is of the first importance. Such food-articles as contain starch or sugar (honey, sugar, ordinary flour or bread, biscuits, rusks, toast, arrow-root, oatmeal, cracked wheat, potatoes, tapioca, sago, peas, beans, turnips, carrots, parsnips, asparagus, artichokes, squashes, beets, corn, rice, hominy, the stalks and white parts of cabbage, lettuce, broccoli, figs, grapes, prunes, apples, pears, bananas, jams, syrups, sweet pickles, chocolate, cocoa, liquors, and sweet wines) are either to be prohibited or restricted to definite quantities, as will be pointed out below. Among articles to be forbidden are also the livers of animals, mollusks (oysters, etc.), and the inside meat of crabs and lobsters. The chief diet must be nitrogenous, and my own plan is to first note the effect of a rigid dietary as follows:

(*a*) *Animal food:* Fresh meats, poultry, game, bacon, ham, fish of all kinds, including crabs and lobsters (except the inside meat of the latter). Fatty substances in large quantities (℥viij—256.0—daily), with a view to restricting nitrogenous destruction, are highly commended by Klemperer. The free use of butter is urged, while eggs, cream-cheese, curds, and buttermilk are also allowed.

(*b*) *Vegetables:* Sour-kraut, lettuce, sorrel, mushrooms, water-cresses, spinach, chicory, celery, cucumbers, mustard-cress, and pickles of various sorts (except sweet). Soya bean, in which the starch and fermentable carbohydrates are removed, or *sarton*, which contains 35 per cent. of vegetable protein, is recommended by Von Noorden and Lampe.

(*c*) *Bread:* The crust of a French roll, first recommended by Flint. Ebstein has recently very highly recommended aleuronat bread; it contains a large proportion of vegetable albumins. The so-called **No. 1 gluten biscuit**[1] is the only form of gluten bread made in this country that does not contain nearly as much starch as the white flours (Tyson). Mosse and Sawyer[2] find that potatoes, steamed with their skins on to retain the potash salts, are often well borne in diabetic glycosuria.

(*d*) *Fruits:* Lemons, oranges, and nuts (except chestnuts).

(*e*) *Beverages:* Milk enough for cooking purposes; tea and coffee, sweetened with glycerin or saccharin; alkaline mineral waters (Saratoga-Vichy, Seltzer-water), simple water with some brandy, and acidulated drinks; Bass's ale, in which all the sugar is converted into carbonic acid and alcohol, and certain acid wines (claret, Rhine).

This strict diet usually causes the sugar to diminish greatly in amount, and in many cases to disappear entirely. If the patient keeps well-nourished and strong, carbohydrates should not be added, since there is no toleration for the latter. On the other hand, Pilosohof[3] found that a small amount of carbohydrate (two apples) added to the albumino-fatty diet caused a diminution of sugar and acetone. In advanced cases with acidosis a carbohydrate diet is indicated; it consists of protein, gm. 60, carbohydrates, gm. 300. One or other of the pure starches, as potato, rice, and oatmeal, are used for the purpose. Again, inulin and levulose are recommended, and possess the advantage of passing directly into the blood unchanged; this form of carbohydrate is epecially indicated in cases in which pancreatic disease is coexistent. An exclusive oatmeal diet for a few days, to be alternated with a diet consisting of flesh

[1] This is made by the Battle Creek Sanitarium Co., of Battle Creek, Mich.
[2] *Brit. Med. Jour.*, March 5, 1904.
[3] *Roussky Vratch*, November 25, 1906.

greens, butter, cheese, and the like has been advocated. Pari and others believe that the starch of the oatmeal has a specific action in this disease. The glycosuria and polyuria must not be relieved at the expense of the general strength of the patient. Von Noorden claims that a non-carbohydrate diet improves tissue-metabolism, and recommends a rigid albuminoid diet at intervals of a few months. S. Solis Cohen recommends levulose as a form of sugar that can be assimilated without augmenting the excretion of glucose. Lactose has been found to give similar results. As a substitute for the latter agents a small amount of ordinary bread (which contains 55 per cent. of starch) or potatoes may be allowed. Strauss has given inulin (℥iij ss—100.0 per day) mixed with the food, and has found it to be well tolerated, the patients increasing in weight. The effects upon the general condition of the patient (body-weight), as well as upon the glycosuria (ascertained by a daily quantitative estimation of the sugar in the urine), are to be carefully noted, and the proportion of carbohydrates may be increased gradually until the limit of the system's ability to assimilate them is found. A more generous dietary is allowable only after the sugar has been absent from the urine for a couple of months, and then it is to be adopted in a gradual manner. A skimmed-milk diet has been recommended by Donkin, Tyson, and others. Disque and others recommend vegetable days, one or two a week, in the treatment of diabetes.

2. Next to an appropriate diet stand certain directions as to **proper hygienic living**: (*a*) All forms of mental excitement and worry must be avoided; (*b*) moderate and regular physical exercise aids metabolism, and is thus directly useful; massage may be substituted for active exercise when the latter is prohibited on account of weakness; (*c*) the diabetic requires a temperate and equable climate; (*d*) a daily tepid bath if the patient be feeble, and a cold bath if he be strong, are to be commended; (*e*) flannels should be worn next to the skin all the year round; (*f*) the living and sleeping apartments must be thoroughly ventilated; (*g*) the teeth must receive careful attention in order to prevent caries.

3. The **medicinal measures** play a subsidiary part in diabetic therapeutics and opium is our best antiglycosuric drug. It is not necessary to employ it in all cases, but it may be tried if the dietetic and hygienic treatment fails to effect a cure. Opium seems not only to exert an influence over the polyuria and the excretion of sugar, but it almost invariably lessens the intense thirst and conduces to refreshing sleep. The drug is well tolerated by diabetics. The commencing dose may be gr. j (0.0648) three times daily, and later increased to gr. v (0.324) or even to gr. x (0.648) three times daily. If morphin be employed, we may begin with gr. ¼ (0.0162) and increase the dose to gr. j (0.0648) or more three times daily. Pavy warmly advocates the use of codein (gr. ½–iij—0.0324–0.1944, three times a day). My own best results have been obtained from the use of the latter remedy in the form of the sulphate, in ascending doses, commencing with gr. ¼ (0.0162), three times a day, and augmenting the dose by gr. ¼ (0.0162) every second day until gr. ij—0.129 (rarely more) are taken thrice daily. Codein possesses the advantage of being less constipating and less likely to disturb the digestive function than either opium or morphin. In patients of a full habit the alkaline waters exercise a valuable influence—Bethesda,

Carlsbad, and Vichy of France have long had a reputation. For the foreign water our native alkaline waters may be substituted, especially the Saratoga-Vichy. While these are valuable adjuncts, they are without the curative effect that is claimed for them by certain authorities.

Among other therapeutic agents that have been employed are the following: the solution of the bromid of arsenic, ♏iij–v (0.199–0.333), three times a day, after meals—in some cases a useful adjuvant to the treatment above outlined; potassium bromid, gr. xx (1.296), three times a day, approximating in efficacy the latter remedy. Fowler's solution, in ascending doses until mild toxic effects appear and then gradually reduced, is preferable to the opium in non-nervous cases; antipyrin (gr. x—0.648), three times a day; sodium salicylate, gr. xv (0.972), and aspirin, gr. v (0.324), three times daily, lessen the formation of sugar; and strychnin, gr. $\frac{1}{30}$ (0.0021), three times daily, is an almost invariably useful remedy. Rudisch[1] observed that methylbromid, or sulphate, of atropin in increasing dosage renders diabetics tolerant of larger quantities of carbohydrates. Horowitz recommends the Bulgarian bacillus, which liberates free lactic acid in the alimentary tract, the latter interfering with the rapidity of the starch digestion.

The treatment of diabetes by *fresh pancreas* or by dry or glycerin extracts has been generally unsuccessful. Fitz, however, mentions a case in which remarkable improvement followed the exhibition of raw calf-pancreas. These preparations have been employed to supply the ferment (internal secretion) essential to the assimilation of sugar. R. Lépine has obtained from the fresh pancreas, from saliva, and from the diastase of malt a glycolitic ferment by a method which, he tells us, still requires to be perfected. This agent he has used in 4 cases of diabetes with a fair degree of success. Williams tried grafting sheep's pancreas in diabetics in 2 cases, but the results were unsatisfactory.

Thyroid extract, in small doses, was followed by immediate improvement in 2 of my cases; it is indicated in alimentary glycosuria.

4. **Symptomatic Treatment.**—Most symptoms demanding therapeutic interference the competent physican is prepared to meet by following general rules. Balint[2] gives a sugar solution in diabetic acidosis—100 to 150 gm. of sugar by proctoclysis daily, with favorable effect. The sugar is absorbed into the general circulation at once instead of being obliged to go by way of the portal system. The management of diabetic coma, however, will be briefly discussed. Klemperer urges the use of fatty substances in large quantities as the best means of restricting nitrogenous destruction, and thus preventing the condition to which diabetics so frequently succumb. When a disgust develops for fats a substitution-method of treatment consists in administering alcohol (℥iss—48.0—per day). Alcohol in *small* quantity checks waste (Hirschfeld). When indications of coma arise, carbohydrates should also be added to the diet. Foster states that an oatmeal diet is the best method of treating acidosis.

The coma is almost certainly due to intoxication with beta-oxybutyric acid, and treatment with alkalies has given the best results. When an attack threatens, sodium bicarbonate, or, preferably, sodium citrate, should be given in large doses (℥iij—96.0 daily) until the urine becomes

[1] *Medical Record*, 1909, xxvi., 1093.
[2] *Berliner klinische Wochenschrift*, Aug. 21, 1911.

alkaline. In the attack, the intravenous injection of the same remedy is to be used freely. Normal salt solution by hypodermoclysis may be tried. Oxygen should be inhaled, and strychnin, digitalis, or ether may be given hypodermically. Prolonged tepid baths with occasional douching have seemed to produce beneficial results, and are worthy of a trial. Elimination from the bowels is to be increased. The *x*-rays projected over the hepatic region have caused decrease in glycosuria.

DIABETES INSIPIDUS.

Definition.—A chronic nervous affection, characterized by constant thirst and an excessive flow of urine, which is free from sugar and of low specific gravity.

Pathology.—No characteristic lesions have been noted, though some degree of enlargement of the kidneys, together with sacculation, due to pressure backward upon the renal structure by the enormous quantities of urine in the bladder and ureters, has been observed. The ureters and pelves of the kidneys may be dilated, and the bladder, owing to constant over-distention, may be hypertrophied. The nervous lesions are not peculiar to simple polyuria. Most important, perhaps, are the tuberculous and other tumors about the floor of the fourth ventricle.

Etiology.—(*a*) Diabetes insipidus is often induced by *nervous influences*—shock, fright, etc. In many cases it follows injuries to the head, but also, more rarely, to injuries of other parts of the body. Tuberculous and other lesions in the vicinity of the floor of the fourth ventricle may produce polyuria. It has also been caused by paralysis of the sixth nerve, with or without meningitis. Frank holds that overactivity of the pituitary gland may be a cause. (*b*) It may occur during convalescence from *acute infectious diseases*. I have seen two instances after influenza in young subjects. (*c*) *Intemperance*, especially the consumption of inordinate quantities of malt liquors, proves a cause. In several of my own cases the amount of urine passed was out of all proportion to the quantity of fluid ingested. (*d*) *Heredity*.—Weil found in four generations of a certain family, consisting of 91 members, that 23 exhibited continuous polyuria—all, however, remaining in good health. (*e*) *Age*.—The disease is relatively more frequent in childhood and early adolescence than is diabetes mellitus. Of 70 cases collected, 22 were under ten years of age, and 13 between ten and twenty (Roberts). Diabetes insipidus may be *congenital*. (*f*) Most cases occur in males as compared with females. (*g*) Diabetes insipidus may be, though rarely, a sequel of diabetes mellitus occasioned by traumatism to the head.

Nature of the Affection.—We are totally ignorant of its true nature, though the facts discovered by Bernard, that either a puncture at a certain spot in the floor of the fourth ventricle or section of the vagus causes polyuria, go to show that it is of nervous origin. It is true that the disease may come on in persons apparently in robust health without discernible causative agencies. In many instances, such as organic affections of the brain or abdominal tumors, the condition is purely symptomatic, and these are probably not to be classed as cases of gen-

uine diabetes insipidus, which is a vaso-motor neurosis, usually of central, though sometimes of reflex, origin.

Clinical Symptoms.—The *onset* is *gradual*, as a rule, but when it follows a fright or traumatism it may develop quickly. There are two main symptoms—the passage of an enormous quantity of limpid urine, and the constant thirst. The daily *amount* of urine varies from 20 to 60 pints (10–30 liters); it is transparent, and the specific gravity is low (1001 to 1005). While the percentage of solids is lessened, the total is usually about normal, and may even be increased. *Albumin* and *sugar* are rare, but in a few cases inosite has been detected. The act of micturition is of very frequent occurrence, and the quantity of urine passed at each sitting surprisingly large. The persistent *thirst* necessitates frequent drinking, but the voracious appetite seen in diabetes mellitus does not mark this disease, in which the appetite is only slightly increased. As a result of the polyuria the skin and mucous membranes are abnormally dry, as in genuine diabetes. But, unlike the latter affection, a fair degree of bodily nutrition is maintained as a rule. The saliva and other digestive secretions are scanty, and this, together with the good appetite, is a fact which explains the disturbances of digestion sometimes met with. The tolerance of the system to alcohol is often phenomenal. Associated nervous phenomena are frequently observed, such as neurasthenic symptoms, insomnia, and chorea.

Prognosis.—The majority of instances proceed to recovery sooner or later, while others pursue an almost endless course—forty or even fifty years in duration—and the patient meanwhile retains his general good health. There is a small group of grave cases that are due to organic diseases either of the brain or abdominal organs (tuberculosis). Death may also be occasioned by some intercurrent affection.

Diagnosis.—The clinical recognition of diabetes insipidus rests upon—(*a*) the enormous amount of urine passed; (*b*) its low specific gravity; and (*c*) the absence of sugar and albumin.

Differential Diagnosis.—Among affections that must be differentiated are *diabetes mellitus*, which has a single point of resemblance—namely, the polyuria; *hysteric polyuria*, which is transient and accompanied by other hysterical manifestations; and *chronic interstitial nephritis*, which generally distinguishes itself by the presence of albumin and hyaline casts in the urine, arterio-sclerosis, and cardiac hypertrophy.

Treatment.—The amount of drinking-water is to be moderated in a gradual, cautious manner, and the patient should be warned not to exceed his actual necessities. I find also that methodic physical exercise and a diet poor in salt and albumin reduces the diuresis. Galvanism has its advocates.

Of medicines, nervines, especially valerian and its preparations, are useful in the idiopathic variety of the complaint, and may be given in the form of the ammoniated elixir (℥j–ij—4.0–8.0) three or four times daily. The valerianate of zinc, quinin, and iron may be variously combined, according to the indications presented by special cases. Ergot and gallic acid have long enjoyed a high reputation in this disease. The commencing doses should be moderate, and then be increased until full physiological doses are employed, this method often bringing about ad-

mirable results. Antipyrin, acetanilid, the bromids, and arsenic have been extensively employed and lauded by different writers in the treatment of this affection. My own best results have been attained by the use of ergot. The potassium iodide is much vaunted. Next to this agent the bromids and acetanilid, given alternately at intervals of a couple of weeks, have been found to be most useful. In one case, Herrick obtained excellent results from lumbar puncture. If a primary disease, *e. g.*, syphilis, exists, it must be met on intelligent general therapeutic principles.

ARTHRITIS DEFORMANS.

(*Rheumatoid Arthritis; Rheumatic Gout.*)

Definition.—A chronic disease, characterized by progressive changes in the arthritic structures (cartilages, synovial membranes, etc.) and by osseous periarticular formations, producing great deformity. The affection may rarely be acute in its course.

Pathology.—Among early gross changes there may be an effusion into the affected joints, but this disappears later. The cartilages are absorbed, the process beginning centrally, where there are both the maximum amount of friction between the opposed cartilaginous surfaces and the minimum blood-supply. Disappearance of the cartilages is naturally followed by contact of the ends of the bones, the latter becoming polished and resembling ivory as the result (eburnated). The friction between the bony extremities may lead to absorption of the latter.

At the periphery, where pressure is slight or even absent, the cartilages become greatly thickened in consequence of persistent irritation, and later become ossified, forming osteophytes which overlie the articular surfaces. These may lock the joints. Bony nodules may also be formed from the periosteum of the bony shafts.

Almost simultaneously the *synovial membranes* become inflamed, a proliferation of their cells taking place, and this exudate may undergo organization and rarely ossification. Later the *capsule* and the *ligaments* are thickened, causing a restriction of movement of the affected joints and producing pseudo-ankylosis. Less frequently they soften and weaken to such an extent that often partial, and sometimes complete, dislocation of the joints ensues; but displacement of the ends of the bones, amounting even to complete luxation, may also be due to absorption. This is often observed in the head of the femur, producing the so-called *morbus coxæ senilis*. Muscular wasting occurs and may be profound. Neuritis has been noted.

The *histo-pathologic* changes consist in cell-proliferation, with fibrillation and softening of the matrix of the cartilages, followed by absorption due to pressure. At the margin, however, proliferation of the cells leads to massive nodulation.

Etiology.—The *nature* of the disease is still dubious, though the old view, that it is *closely* connected with rheumatism on the one hand or gout on the other, should be abandoned. J. K. Mitchell long since maintained that rheumatoid arthritis is of neurotrophic *origin*, being espe-

cially dependent upon affections of the spinal cord, and without stopping to adduce all of the facts that tend to support this theory, the following deserve mention: (1) Diseases of the cord (locomotor ataxia, etc.) are known to cause arthritic conditions; (2) The character of certain causal factors, such as nervous shocks, griefs, etc.; (3) The symmetry of the joint-deformities; (4) The time of occurrence; and (5) Noticeable trophic disturbances that are frequently associated. Falli[1] autopsied 4 cases, 2 of which were typical, and in the latter lesions were found in the anterior horns of the spinal cord, atrophic in the first case, but degenerative as well as atrophic in the second. According to Falli, not all cases of arthritis deformans are to be interpreted as instances of nervous disease. The *microbic* theory of the disease claims a small but increasing number of supporters.

Bacteriology.—Dor claims to have succeeded in finding a definite organism. He also claims to have reproduced the disease by injecting cultures directly into the blood of rabbits, and considers the germ an "attenuated culture" of the *staphylococcus pyogenes aureus*. v. Dungern and Schneider isolated after death from the mucus of the gall-bladder, and also from the exudate in the joints, small diplococci that did not resemble the organisms previously described by Blaxall and Schüller. Injections of the cultures into the knee-joint of rabbits resulted in the production of lesions similar to those observed in the patient.

Predisposing Causes.—(*a*) *Nervous shocks*, mental worry, and deep grief. (*b*) *Females* are more frequently victims than are males, the proportion, according to the statistics of Garrod, being about one to five in favor of the former sex. To account in part for its greater frequency in women is the fact that sterility and certain ovarian and uterine complaints seem to exert a strong etiologic influence. (*c*) *Age* exerts a decided influence. It is most frequently contracted in the third decade of life, though it has been noted as late as the end of the fifth. It occurs also in children, though rarely. Out of 307 cases treated in the Devonshire Hospital during 1892, only 2 per cent. manifested the disease before the age of ten. (*d*) *Heredity* has been traced in some instances, and in many a *family tendency* to joint-affection. (*e*) Though it occurs in all classes of society, the *poor* or those exposed especially to debilitating influences are more liable than the *rich*. (*f*) *Infectious diseases* may have an influence. (*g*) Ewart[2] recognizes some mixed conditions in which rheumatoid arthritis may supervene on the gouty diathesis.

(1) **Symptoms of the Chronic Form.**—At first one joint, usually of the hand, is slowly involved; soon the corresponding joint on the opposite side is attacked. These may recover apparently, but are soon reinvaded and grow progressively worse. The affected joints slowly enlarge, and are moderately painful, particularly on movement. *Pain*, however, may either be slight or even absent, or severe, if the synovial membrane be involved. There is neither redness nor tenderness, as a rule, but on palpation an effusion, variable in extent, is generally detectable. The *course* during the early stage is often marked by periods of improvement, alternating with exacerbations in the local symptoms, and

[1] *Il Policlinico*, Dec., 1894.
[2] *International Medical Magazine*, April, 1899.

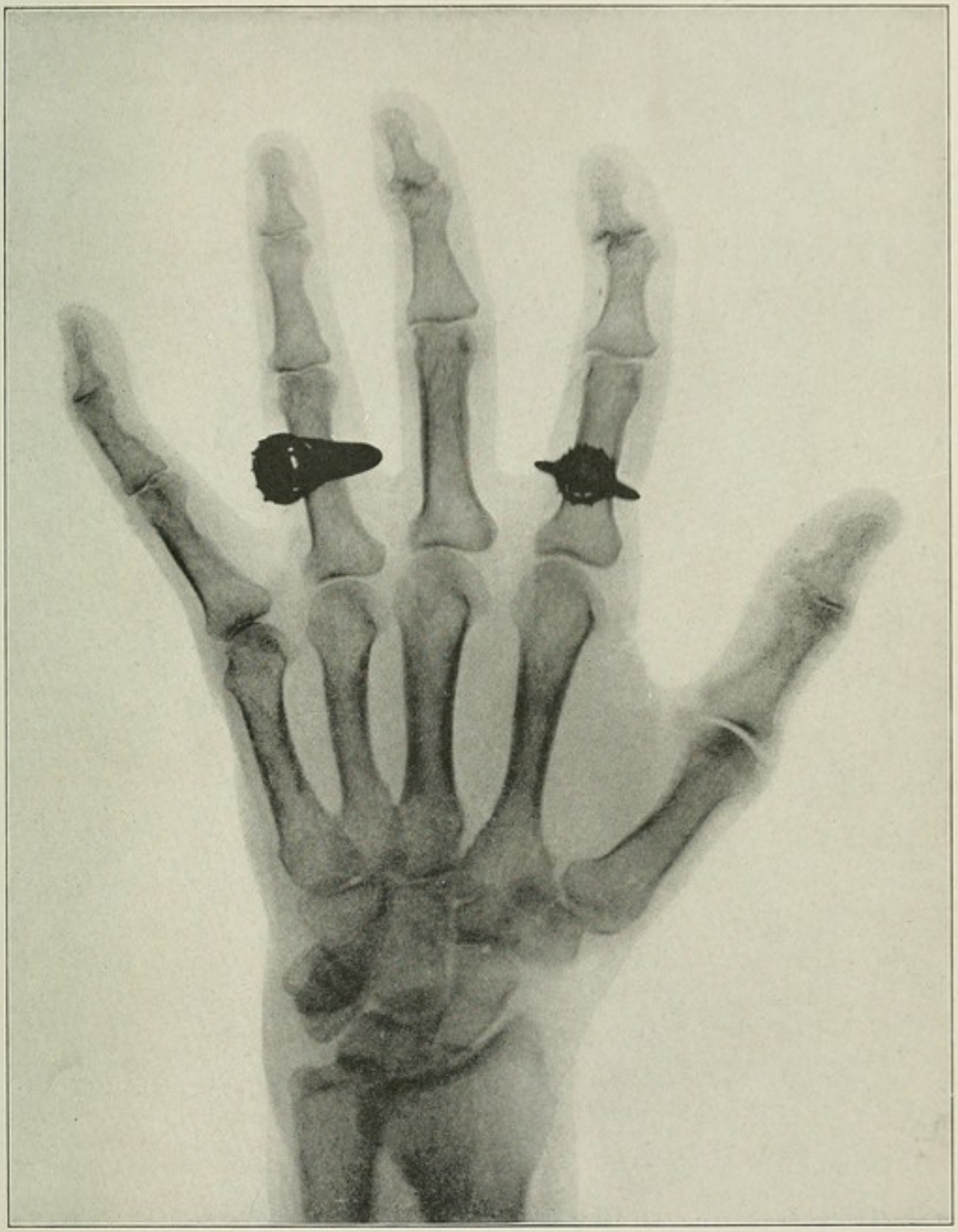


FIG. 32.—Hand of M. R., aged fifty years, showing characteristic deformity, including ulnar deflection of fingers, in advanced arthritis deformans.

especially in the swelling and pain. While, as intimated, one or two joints only are affected at the start, gradually those of the feet, arms, legs, and trunk are invaded symmetrically, until, in the worst cases, every joint is deformed.

The most characteristic symptom is the *deformity*, which manifests itself earliest in the hands. The fingers are generally pointed toward the ulna, rarely toward the radius, and the presence of the osteophytes and the immensely thickened capsular ligaments, together with the retracted muscles, all tend to alter entirely the shape of the joints. The fingers, for example, are flexed and extended upon the hand, and sometimes overlie one another. With the progress of the deformity a partial, and less often a complete, luxation of the joints occurs (see Fig. 32). The joints may become finally either quite fixed, owing to the presence of the periarticular osteophytes, or a limited degree of movement may remain.

Palpation and *auscultation* of the involved joints reveal crepitation during movement. Strangely enough, the thumb remains intact, compensating for the loss of the functional movement of the fingers to a remarkable extent. In addition, the hand is sometimes less affected than the rest of the joints—a fact which enables the patient to perform a great variety of delicate movements, or even to engage in useful and surprisingly skilful handicraft. The adjacent muscles become wasted and are the seat of contractures, causing flexion of the limbs, especially of the thigh upon the abdomen and the leg upon the thigh. Other trophic changes, such as paresthesia and pigmentation or glossy areas of the skin, may be observed. In 3 of my cases onychia was present. In extreme instances the decubitus is lateral and the patient utterly helpless.

The *course* of the disease throughout the more advanced stages is exceedingly variable. Its advance may be arrested and the general health remain unimpaired, and this may take place after implication of but a few joints, so that the entire affection may be confined to a comparatively small part of the body, either in the upper or lower extremities. In progressive cases more or less gastro-intestinal disorder arises; the symptoms of indigestion with subacidity appear, the appetite is impaired, and anemia develops. The patient's sufferings make him irritable. Hypochondriasis may be a concomitant. In established cases the pulse is persistently rapid and the skin inclined to perspiration.

Clinical Varieties.—(1) Of the **chronic form** there are certain subvarieties. The disease may be limited to a single joint (*monarticular*), this form most commonly affecting the hip-joint, when it is known as *morbus coxæ senilis*. It is seen generally in old men, and often follows an injury. Its features—pathologic and clinical—including the muscular wasting, are the same in kind as those of the *polyarticular* variety. Monarticular arthritis deformans may also be confined to the shoulder-joint or the knee, and, as in the preceding form, men who have passed the middle period of life are mainly affected.

A special variety, which is generally not monarticular, involves only the vertebræ (*spondylitis deformans*). With this may be combined disease of one or more of the neighboring large joints, forming the *spondylose rhizomélique* of Marie, or the condition may be confined to the cervical spine, as in a recent case of my own, thus preventing flexion of the head.

A fair degree of rotation usually remains, but it sometimes happens that the entire spinal column is involved and held in a perfectly rigid position.

Still another form in which the distal joints of the fingers become knobbed (*Heberden's nodes*) demands separate description. Heberden's nodosities occur chiefly in women between the thirtieth and fortieth years. According to Heberden, who first described them, the nodes have no intimate association with gout, and this view coincides with my observations. At first the affected joints become *swollen*, *tender*, *slightly red*, and *painful*, and then seemingly undergo great improvement. The condition however, is progressive, advancement occurring in the form of fresh exacerbations, which are only rarely traceable to errors in diet, and are separated by periods of remission. The morbid process is the same as in rheumatoid arthritis, and the destructive changes in the joints proceed until distinct hard nodules are formed. These are usually most marked at the sides of the extensor surfaces of the second phalanges. The disease does not spread to any of the larger joints, and, although incurable, it is free from danger to life.

(2) **The Acute Form.**—This is comparatively rare, and occurs commonly between the ages of twenty and thirty. It occurs in children, and is more common in women than in men. Among its common antecedents in women are pregnancy, delivery, excessive lactation, and the menopause. *Multiple arthritis*, affecting both the large and small joints, sets in acutely, and there are pain and either a slight redness or a considerable swelling, due chiefly to an effusion which is intra- rather than periarticular. There are only a slight tendency to migration from joint to joint, and a slight febrile disturbance.

Still described a form of chronic joint-disease in children which he thinks presents differences sufficiently marked to suggest a distinct clinical and pathologic entity, and differing from the rheumatoid arthritis of adults. It is defined as a progressive enlargement of the joints associated with general enlargement of the glands and enlargement of the spleen. He has studied 22 cases, 19 of which came under his personal observation. It occurs before the second dentition. Stiffness, general thickening of the tissues around the joints without redness or tenderness, except in very acute cases, with limitation of movement and more or less rigid flexion of the joints, characterize the arthritic disturbance. The most distinct feature of the disease is the enlargement of the lymphatic glands, those in relation to the involved joint being primarily affected. The glandular swelling is general and constant, and with the enlargement of the spleen, points toward an infectious origin. Cardiac complications are absent. The *course* of the disease is slow.

Differential Diagnosis.—The diagnosis between the chronic form of the disease and *chronic rheumatism* is often extremely difficult. In the latter, however, a few of the larger joints only are involved, while there is an absence of the peculiar deformity and marked fixity of the articulations. On the other hand, cardiac complications are absent in chronic rheumatoid arthritis, and the course is progressive. A *monarticular arthritis* which differs in its morbid process from rheumatoid arthritis sometimes affects the shoulder-joint. It is not uncommon, and is "characterized by pain, thickening of the capsule and of the ligaments, wasting of the shoulder-girdle muscles, and sometimes by neuritis"

(Osler). I have met with 5 instances of this sort, in all of which pain was intense and the course subacute. All ended in recovery.

The frequency of the occurrence of intercurrent acute polyarthritis in arthritis deformans causes the danger of mistaking this for acute rheumatism.[1] Acute rheumatoid arthritis is to be discriminated by the special etiologic factors, the less severe pain, the less marked redness, the slight tendency to migration from joint to joint, the slighter febrile disturbance, and by the practical freedom from cardiac complications. *Gout* will be distinguished in the description of that disease.

Prognosis.—Though incurable, rheumatoid arthritis is not immediately dangerous to life; in some cases improvement, and in a smaller proportion arrested progress of the disease, may be expected.

Treatment.—This especially involves measures that are directed toward the improvement of bodily nutrition—a generous dietary, systematic warm bathing, and an abundance of fresh air, with properly regulated physical exercise. Tonics may be necessary to invigorate the economy, and iron to overcome the anemia. The prolonged use of cod-liver oil has given me excellent results. Of special agents, the most satisfactory in their effects if administered early are iodin and arsenic. The former may be administered in the form of a saturated solution of sodium iodid, of which 10 to 15 drops may be given in milk one hour after food. Sheffer advocates sodium silicate well diluted (dose 1.5 to 3 grams daily). The silicates are useful in overcoming defective metabolism. The prolonged use of extract of thyroid in small doses (gr. ss to j—0.0324 to 0.0648), with occasional intermissions, is favored where insufficiency of thyroid secretion is suspected. Schüller and Hirschberg[2] have had favorable results in the treatment of this disease by limiting the amount of lime in the diet. The patient may be sent to a warm climate in winter and to a cooler one, preferably a mountain-resort, in summer. These patients also do well at certain mineral springs, such as the Sulphur Springs of Virginia, the hot springs of Arkansas or Töplitz, at Baden in Switzerland, and the warm sodium chlorid baths in Wiesbaden. Hot mineral spas should only be resorted to in the early period of the affection. Strümpell has seen excellent results follow the employment of hot sand-baths, which can be used at home. Stewart advocates the Tallerman method of treatment—*i. e.*, of superheated dry air. Immobile contractures may be broken up under anesthesia and tendons lengthened by tendoplasty, followed by plaster-of-Paris dressing until pain and irritation have subsided (Ochsner). Pain may also be relieved by aspirin or tincture gelsemium.

Eliminative Treatment.—Guaiacol carbonate may be given in doses of from 5 to 15 grains (0.3–1.0 gm.) daily and increased. This combines with the toxins, and is eliminated as guaiacol sulphate (Bannatyne).

The *local means* are of value. If the joints be inflamed, cold compresses, covered with oiled silk, to which some narcotic agent may be added, will afford relief. This should be followed by thorough and systematic massage, which is our best measure for the reduction of the swelling (by promoting absorption) and for lessening joint-rigidity, and restoring the atrophied muscles. Swedish movements are useful in maintaining mobility and mechanical or electric vibration may prove helpful. Bier's hyperemia may be used where the affected joints are few.

[1] Thos. McCrae: *Jour. Amer. Med. Assoc.*, Jan. 6, 1904, p. 164.
[2] *Berlin. klin. Woch.*, 1911, xlviii., 2056.

GOUT.

(*Podagra.*)

Definition.—A form of perverted nutrition due to an auto-infection, accompanied by the formation of a variable (usually increased) amount of uric acid, and characterized clinically by attacks of acute arthritis, with or without uratic deposits in and around the joints.

Nature of the Affection.—The numerous theories that prevail at present in regard to the disease are irreconcilable, but it seems certain that there is (*a*) an excessive absorption of nutritive substances, both solid and liquid; (*b*) a disordered metabolism growing out of the effects of imperfect physical development, combined with too little muscular exercise; (*c*) a defective elimination of waste-products, although in some cases a normal elimination of waste-products exists.

There are a number of *uric-acid theories*, some of which may be briefly mentioned: 1. Garrod contends that an acute attack of gout is invariably produced by an excess of uric acid in the blood, due to increased formation and greatly decreased elimination; also, that inflammation is caused by the deposition in the joints of sodium urate. 2. Haig holds that there is a diminished alkalinity of the blood, and that the latter cannot therefore hold the uric acid in solution, and not an excessive production of uric acid. 3. Ebstein thinks it probable that there exist an excessive production and accumulation in the blood of uric acid. The surcharged blood excites local inflammation, followed by necrosis, and uric acid deposits. 4. Sir William Roberts believes that acute attacks of gout are dependent upon the precipitation of the crystalline biurate of sodium; that the urate is transformed into the less soluble biurate in the blood. 5. V. Noorden concludes that the essential process is a tissue-necrosis attributable to the presence of a hypothetic ferment, and that the uric acid, which is without etiologic effect, is deposited at the necrotic focus. Hall[1] affirms that as an etiologic entity, uric acid must be definitely discarded. 6. Klemperer[2] has shown as the result of observations made in cases of gout, that as long as the function of the kidneys is not materially interfered with the presence of considerable amounts of uric acid in the blood must be attributed to increased formation. But the presence of an equivalent of uric acid in the blood in certain affections other than gout (*e. g.*, leukemia) shows that this factor is not the sole cause of gout. 7. Morhorst states that in any alkaline liquid the basic substances combine with uric acid, if this be present, to form a urate. These uratic precipitations are met in non-vascular tissues only, the alkalinity of which is less than that of the blood, and that they are the essential cause of the symptoms. 8. Kolisch maintains that when the kidneys are healthy the alloxuric bodies are, in great part, excreted as uric acid; but when they are diseased the xanthin bases are increased at the expense of the uric acid. Chittenden and others, however, hold that the xanthin bases are practically free from toxic effects. 9. Luff thinks that uric acid is formed in the *kidneys* from a combination of urea and glycocin, an increased amount of the latter substance being formed in the liver. 10.

[1] *The Practitioner*, 1906, lxxvi., p. 361.
[2] *Deutsche medicinische Wochenschrift*, 1895, No. 40, p. 653.

Duckworth insists that gout is essentially of nervous origin. The view that failure of the renal function precedes the development of gouty manifestations, and the older view, that an increased proportion of uric acid is found in the blood, are widely accepted.

Pathology.—The post-mortem history of gout is concerned principally with the arthritic changes, including the deposits of the urate of sodium in the cartilages, the ligaments, and the synovial membranes. These are fluid in their earliest state and contain numerous small crystalline masses; they soon inspissate and later become hard and dry (tophi). The latter excite secondary inflammatory changes that may lead to fibrous overgrowths, distortion, and fixation of the joints. Gouty tophi may be absorbed or they may finally be discharged through the skin in consequence of an ulcerative process. The chalky concretions have been found also in the cartilages of the ears, less frequently of the nose, eyelids, and larynx. They have also been described in the periosteum and along the tendons of the palms of the hands, where they produce a characteristic form of contraction of one or more fingers (Dupuytren's contraction). Charcot has found them in the penis. If death occur in the *acute* attack, hyperemia and swelling of the capsule, ligaments, and synovial membrane are found, together with an inflammatory exudation into the joint.

The *kidneys* are usually involved, the changes being similar in character to those observed in the joints, and innumerable areas of necrosis, followed by uratic deposits, are seen throughout the organs, though chiefly in the papillæ. Osler says that "the presence of these uratic concretions at the apices of the pyramids is not a positive indication of gout." N. S. Davis, Jr., points out that the kidneys are affected in spots, with intermissions in the degenerative changes, which are microscopical in size, until finally large areas are involved. Granular contracted kidney (chronic interstitial nephritis), with or without arteriosclerosis, is sometimes caused by the gouty condition (*vide* Interstitial Nephritis).

The *heart* and *blood-vessels* always present changes. Gout induces arterio-sclerosis, and the latter in turn causes cardiac hypertrophy, particularly of the left ventricle. In chronic cases fatty degeneration of the heart-muscle sometimes occurs, and chronic valvulitis, with deposits of urate of sodium in the valves, has been noted. Chronic bronchitis, asthma, and emphysema are among the more common changes connected with the *respiratory tract*, acute conditions being rare.

Etiology.—The following are the principal contributing causes:

(*a*) *Heredity.*—Garrod's dictum, "that more than one-half of all gouty subjects can distinctly trace their ailment to an hereditary taint," is doubtless correct, heredity from the grandparents, which is of not infrequent occurrence, being included in this estimate. If the better class of society alone be considered, the percentage will probably be still larger. It must not be forgotten, however, that patients out of pride represent other articular affections as gout. (*b*) *Age.*—Primary attacks are most frequent in middle life. They are rare before puberty, though exceptionally seen even in suckling infants; but after the age of puberty they become more frequent. After the fiftieth year they decrease rapidly in frequency, and are very rare in quite advanced life. The cases that develop quite early in life often show a striking heredi-

tary taint. (*c*) *Sex.*—The arthritic form is less frequent in women than in men, while the former are disposed to the irregular type of chronic gout quite as strongly as the latter. (*d*) *Diet.*—Over-indulgence in the pleasures of the table, together with defective muscular exercise, constitutes a potent factor, and this even in persons who are endowed with exceptional powers of digestion. (*e*) *Alcohol*, and particularly the fermented liquors, are among the chief favoring influences. The fact explains the relatively greater frequency of gout in certain countries (*e. g.* England and Germany), in which the heavier beers and ales are freely used, than in America, where lighter fermented drinks are more popular. The cases, however, are on the increase in this country. (*f*) *Social State.*—Most cases occur among the upper class of society, but there is also a well-defined form of "poor-man's gout" due to an excessive use of malt beverages. (*g*) *Lead.*—Workers in lead furnish numerous typical examples of gout. Garrod found that in 30 per cent. of the hospital cases the patients had been painters or workers in lead. He also showed that the administration of lead salts to gouty persons almost invariably determined a gouty paroxysm. Whether lead produces gout by arresting the excretory processes, and by thus inducing a fibroid change in the kidney and liver, as is held by Oliver of New Castle, is not definitely settled. Poore points out that gout produced by lead or chronic kidney trouble is constantly associated with anemia and emaciation, and forms a distinct clinical entity. We may presume the existence of a primary renal gout. (*h*) Cornillon and others detail cases in which injuries were followed by the first appearance of the disease.

Clinical History.—1. Acute Gout.—The earliest manifestations of the disease are apt to take the form of a more or less typical attack of *acute arthritic gout.* The latter is usually preceded by certain *prodromal symptoms*, which vary in different cases, but are almost constantly similar for the paroxysms of individual cases. The patient may complain either of slight muscular cramps and articular pains, or of dyspeptic disorder, or of an asthmatic seizure; or he may exhibit mental disturbance —irritability of disposition, broken, restless sleep, and depression of spirits. In a small percentage of instances, just prior to the attack the patient feels better than ordinarily. It has been observed that immediately before and also during the early part of a paroxysm the daily amount of uric and phosphoric acids found in the urine is diminished; but Klemperer has shown that no relation exists between the amount of uric acid present in the urine and the character of the disease.

The attack generally develops in the very early morning hours. The patient awakens suffering from pains in the metatarso-phalangeal joint of the great toe, that soon become excruciating, while the joint feels as if it were tightly compressed in a vise. The *local signs* of inflammation—heat, redness, swelling, and excessive sensitiveness—quickly supervene. The skin pits on pressure and becomes shiny. The *body-temperature* rises to 102° or 103° F. (39.4° C.), and the patient manifests intense irritability.

At the end of an hour or two the sufferings abate, the fever often declines, with free perspiration, and the patient may be able to pursue his avocation. During the next day some degree of enlargement and inflammatory edema remains, and on the following night the symptoms are usually repeated in all their violence. The condition usually pro-

gresses in this manner from four to seven or eight days, though after a few days the intensity of the paroxysms is apt to lessen. After the attack the swelling subsides and there is a slight desquamation of the skin, which resumes its normal color, and the general health is often unusually good. These so-called fits of gout usually recur from time to time, the duration of the intervals depending largely upon the patient's habits or routine of life. On the whole, the first interval is apt to be the longest, while later the intermissions may not exceed two or three months. With subsequent attacks the affection is apt to spread to other articulations. There is no tendency to suppuration.

2. Retrocedent Gout.—This term implies the sudden transmission of the arthritic process to some internal organ. During a paroxysm the joint-inflammation may quickly disappear with an equally sudden development of intense pain in the region of the stomach, vomiting, diarrhea, faintness, and a rapid, feeble pulse. Suppressive gout may attack the heart and produce precordial pain, dyspnea, cardiac palpitation, and much anxiety of mind. It may also excite pericarditis with a fatal result. Transmission to the head, with the development of intense cerebral symptoms (maniacal excitement, coma, and apoplexy), also occurs. Nervous phenomena, however, are more commonly due to uremic poison.

3. Symptoms of Chronic Gout.—Chronic gout follows the acute variety. The transition is gradual, the intervals between attacks shorter, while the attacks themselves grow milder and longer. At last the local inflammation does not appear. The condition extends to other joints: first, to the corresponding joint on the opposite side, then to the other toes and the ankles. Later, the fingers and wrists may be invaded, but almost never the largest joints. With the progress of the affection the chalk deposits slowly increase until the characteristic deformity is produced. The skin covering the tophi may ulcerate, exposing the chalk-stones, an unmistakable picture. When the fingers are affected we note a deflection at the second or third joint, constituting a peculiar habitus.

Among important *associated conditions* are chronic gastric catarrh, arterio-sclerosis, cardiac hypertrophy with considerable functional disturbance of the heart, and "contracted kidney," forming a much complicated yet easily recognized clinical picture. If in cases of this sort the *urine* of a gouty person is carefully examined, and is found to contain a small percentage of albumin and tube-casts, the whole train of events becomes easy of interpretation. The cases may be divided into two classes: (*a*) those in which the complexien is florid and the general health vigorous; (*b*) those with pale, sallow facies, emaciation, and enfeeblement. These groups are chiefly dependent upon the differences in the etiologic factors. Gouty subjects often manifest unusual mental vigor.

The *course* of chronic gout is liable to be interrupted by acute exacerbations with fever, during which dangerous complications may arise—*e. g.* uremia, pericarditis, pleurisy, pneumonia.

4. Irregular Gout.—Says Sir Dyce Duckworth: "Gout manifesting itself anywhere but in a joint is to be considered irregular or incomplete." Such cases are confined chiefly to persons of gouty heritage, though I feel confident that the diathesis may be also acquired. But though the etiologic factors that produce lithemia also in time produce

gout, these two conditions should be discriminated; for, while in both we usually note an excess of uric acid in the blood, in lithemia there are no tophi present, and hence no necrotic foci in the joints or elsewhere. Irregular gout, then, rarely occurs in persons who have had previous typical attacks, but should any of the conditions described below be associated, or should they alternate, with acute gout, they may be properly ascribed to the latter. On the other hand, when these conditions occur in persons who are free from hereditary taint, and who are not addicted to the intemperate use of alcoholic beverages, or excessive indulgence in the pleasures of the table, and are not possessed of luxury- and rest-loving temperament, the diagnosis of irregular gout is to be made with extreme caution. It is justifiable to apply a therapeutic test when other means of diagnosis fail.

The features of irregular gout are exceedingly diversified; the following are the more important:

(*a*) *Joint- and Muscle-pains.*—The muscular pains may be anywhere, and "flying" in nature, but the muscles of the back of the neck, the lumbar region, the abductors of the thigh, and the gastrocnemii are especially liable (Tyson). These pains are most severe in the early morning hours and subside as the day grows. Articular pains attended with some degree of swelling and deformity of the joints (the latter, however, not due to uratic deposits) may be of gouty origin; and, according to Paget and Garrod, Heberden's nodosities (previously described under Rheumatoid Arthritis) may present vesicular eminences due to gout.

(*b*) *Gastro-intestinal Disturbances.*—The symptoms referable to the intestines are identical with those presented by lithemia. In one of my cases intestinal colic followed by diarrhea put in an appearance at long intervals. Tonsillitis, pharyngitis, and parotitis may be manifestations.

(*c*) *Cardio-vascular Symptoms.*—Just as in pure lithemia, so in atypical gout, the increased amount of uric acid usually present in the blood, by increasing the blood-tension, excites arterio-sclerosis and chronic interstitial nephritis—affections which are fully described in appropriate sections of this work. Occasionally pericarditis is a manifestation.

(*d*) *Nervous Manifestations.*—The different varieties of headache, including migraine, are common. Sciatica and other forms of neuralgia, tingling, itching, burning sensations, and even pain in the palms of the hands and soles of the feet, are of frequent occurrence. Hot and itching eyeballs are, according to Hutchinson, among frequent manifestations; apoplexy may arise, secondary to atheroma induced by gout; and rarely meningitis (basilar) is among the gouty morbid states. The latter also include certain psychopathia—insomnia, irritability of temper, and melancholia. The possibility of gouty neuritis is to be remembered.

(*e*) *Urinary Symptoms.*—The urine is highly colored, of high specific gravity, often scanty, and the standing specimen deposits lithic acid. This is not peculiar to gout, however. In many cases uric acid is in excess only at intervals, giving rise to so-called uric-acid showers, while at other times it is diminished in quantity. Futcher[1] reports an investigation of the excretion of uric acid and phosphoric acid in a number of gout cases, and concludes that there is a close parallel relationship between the two. He believes that both are products of nuclein disintegration. Gouty persons are liable to gravel: I agree with Tyson, how-

[1] *The Practitioner*, August, 1903, p. 181.

ever, in thinking that the two conditions more frequently alternate than coexist. Intermittent glycosuria is also common in gouty subjects, and may lead to true diabetes mellitus; this glycosuria may alternate with uric-acid showers. With these affections—intermittent glycosuria and gout—obesity is not uncommonly associated. Oxaluria has been noted. Grandmaison believes the association of albuminuria with gout to be a frequent one, and that the early albuminuria is often intermittent. Zuelzer[1] has observed an increased elimination of uric acid in gouty subjects after the use of atophan, and Weintraud has shown that uric acid injected intravenously into gouty subjects is retained for a long time unless atophan be given—a diagnostic sign. Among grave *secondary affections* chronic interstitial nephritis, with its characteristic features (slight albuminuria and later casts), very commonly develops, sooner or later, and cystitis (with gouty hemorrhage into the bladder), urethritis, prostatitis, and orchitis all may be dependent upon gout.

(*f*) *Pulmonary Disturbances.*—Chronic bronchitis, to which asthma and emphysema may be secondary, is often due to podagra.

(*g*) *Cutaneous Eruptions.*—Eczema is frequently associated with the gouty diathesis, and I have often observed eczematous eruptions alternating with the symptoms of bronchitis or gastric catarrh.

(*h*) *Ocular Disorders.*—The chief eye-symptoms are conjunctivitis and keratitis (with tophi in the cornea and eyelids), iritis, hemorrhagic retinitis, and glaucoma. Gouty involvement of the ear (external canal and the auricle particularly) occurs oftenest late in life, though hereditary gout may rarely cause ear symptoms shortly after birth.

Differential Diagnosis.—The distinction between typical acute gout and *acute articular rheumatism* is a simple matter. But when, as is rarely the case, the former manifests itself as a polyarthritis, the discrimination may be difficult. W. H. Thompson has pointed out that in gouty polyarthritis, when the knees, elbows, and phalangeal finger-joints are affected, the points of greatest tenderness on transverse pressure are over the condyles. On the other hand, in acute rheumatism the points of maximum tenderness correspond with the tendons anterior and posterior to the joints. Moreover, gout distinguishes itself by its previous history (heredity, alcoholism, gluttony), by the tophi, which may be first detected in the ears or conjunctivæ, by the development of contracted kidneys, and the less marked fever. After repeated attacks deformities of the joints ensue. In a doubtful case the blood-serum may respond to Garrod's uric-acid test, as follows: Add 5 to 6 minims (0.399) of acetic acid to 2 drams (8.0) of blood-serum in a watch-glass; then place a linen thread in the solution, and after twelve to twenty-four hours this will be encrusted with crystals of uric acid. The same result is obtained in leukemia and pneumonia,—affections from which gout is easily distinguishable.

Chronic rheumatism is distinguished from gout by the fact that the latter disease involves chiefly the small, and chronic rheumatism chiefly the large, joints. Moreover, chronic interstitial nephritis and arteriosclerosis, with their varied and often serious consequences, are frequently attendant upon gout, but not upon chronic rheumatism.

To differentiate chronic gout and *rheumatoid arthritis* may be difficult, but the following table will indicate the main points of difference:

[1] *Berlin. klin. Woch.*, 1911, xlvii., 2101.

Gout.	Arthritis Deformans.
Frequently hereditary.	Not so.
Causes are chiefly dietetic.	Causes chiefly nervous.
Affects males and the better classes most frequently.	Affects females and lower classes most frequently.
Begins in the big toe and extends to other toes; it is unilateral.	Begins in the fingers, which point to the ulnar side; develops in symmetric order.
Attacks are periodic.	More steadily progressive.
Deformity due to tophaceous deposits.	Deformity due to exostosis and ankylosis, and more marked.
Uric acid in excess in the blood.	Not so.
Complications (nephritis, arterio-sclerosis).	Very rare.

Treatment.—(1) **Prophylaxis.**—In order to prevent the development of gout, especially in persons who have inherited or acquired a strong predisposition to the disease, temperate and even rigid habits of living should be adopted. Alcohol, particularly the heavier wines (Madeira, port, sherry, champagne, etc.) and heavier malt liquors, must be eschewed, and the patient must eat sparingly of concentrated meat (particularly red meat). A residence in the country with active out-of-door exercise is of paramount importance, but straining efforts, both mental and physical, are to be avoided. The climate should be temperate and moderately dry. The sleeping apartments should be capacious, well ventilated, and free from draught, and the action of the skin is to be favored by cleanliness, and if the patient be strong by a cold bath in the morning with friction. For the robust, Turkish baths at intervals of two or three weeks constitute an excellent measure. In debilitated patients warm baths on retiring are preferable, and the chilling of the skin-surface is to be carefully gua ded against. The patient should wear flannels.

(2) **Active Treatment.**—(*a*) *Dietetic.*—"There is no diet for gout, but there is a diet for the patient" (H. C. Wood). The amount of food must be lessened as a rule, and taken at regular intervals. On the other hand, spare gouty subjects are met with, and in such I have found a generous diet, including fat-producing foods, of great service. During an attack we should attempt to overcome the perverted metabolism of the liver and gastro-intestinal tract, and to minimize the production of the purin bodies. Broadly speaking, the dietary should be constituted as follows: *succulent vegetables* (cabbage, salads, string-beans); *fruits* (except bananas, tomatoes, and strawberries); *farinacea*, as rice, hominy, and the like (oatmeal to be avoided); *meats* should be restricted; beef and mutton in moderation may be allowed except in well-marked cases of gout; oysters and fish (except those that contain too much protein, salmon, smoked herring, canned sardines, mackerel, halibut, salt codfish, flounder), and fowl, particularly the white meat of chicken, are permissible; *fats* in the form of good butter may be taken freely—from $2\frac{1}{2}$ to $3\frac{1}{2}$ ounces (70.0–100.0) per diem, according to Ebstein; *milk* is entirely unobjectionable, and should be used in large quantities. If whole milk does not agree, it may be mixed with an equal part of Vichy. According to Kolisch, eggs are not objectionable, as the nucleins contained do not form alloxins. *Stale* breads may be used. Occasionally patients do best on albuminoids, while, on the other hand, with about equal frequency they improve on a vegetable diet; but a mixed diet is best adapted to the vast majority of the

cases. Among articles to be avoided are pastry, tea and coffee, hot bread and cakes, sweet puddings, cheese, dried meats, and all highly seasoned dishes.

Beverages.—Alcohol is ordinarily to be interdicted. Rarely it becomes necessary to administer it, particularly in cases of suppressed gout, and when needed, whiskey is to be preferred. Champagne, Tokay, Port, and malted liquors are injurious in their effects, but clarets, Rhine, and Moselle wines can be often taken without unfavorable results.

Mineral waters, particularly the alkaline, are highly advantageous, and sometimes are even curative. Their value, like that of the warm baths and systematic exercise, is dependent upon their power to increase renal elimination. Whether they promote solubility of the uric acid in the blood is questionable; moreover, according to the observations of Klemperer, this is not a rational indication. The carbonate and citrate of lithium are efficient diuretics, but have no other claim to virtue in this disease. Among natural waters of special value abroad are Vichy, Carlsbad, Homburg, Ems, Kissingen, Aix, Buxton, and Bath, and in this country Saratoga and Bedford. These waters are to be taken in large quantities and when the stomach is empty. It is highly probable that the environment, rigid system of hygiene, including exercise and an appropriately modified dietary, play the principal rôle in producing the favorable results obtained at these noted springs.

(*b*) *Medicinal Treatment.*—During an acute attack the pain, if excruciating, is to be relieved by a hypodermic injection of morphin, which is to be followed by a purgative dose of some mercurial. Colchicum is the specific remedy, and must be administered, in the form either of the wine or the tincture, in doses of ♏ xx–xxx (1.333–1.999) every four hours. It alleviates the inflammation and promptly relieves the pain, but its effects during the attack should be carefully noted. The salicylates may also be given to relieve pain. After the paroxysm it should be continued, though in small doses, combined with the citrate or bicarbonate of potassium or lithium. Gudzent reports success with radium emanations (by inhalation in a closed room) two hours daily; this agent causes the disappearance of uric acid from the blood. The limb should be raised and the affected joint or joints wrapped in flannel or cotton-wool. Warm alkaline solutions or hot fomentations often afford relief in the worst cases, and anodynes may be tried locally. The diet should consist chiefly of milk, animal broths, and egg-white during the attack, later rice, eggs, fish, and other light forms of meat may be added, the more liberal dietary previously indicated being slowly resumed.

In the *intervals* between the acute attacks the prophylactic and dietetic measures previously mentioned are to be resorted to, in order that recurring paroxysms may be prevented, and in addition the alkaline diuretics and saline laxatives, together with warm bathing, will be found of value. Hepatic stimulants yield good results.

In *chronic* and *irregular forms* of gout medicines are of subsidiary importance, and are in no wise comparable in their beneficial effects to the previous recommendations. Piperazin has been warmly advocated in all forms of gout for its supposed effect as a solvent of uric acid, and clinicians are almost unanimous in reporting its favorable results. Its beneficial effects are probably due to its diuretic action. The dose is gr. v–x (0.324–0.648) thrice daily, freely diluted with water. The late Sir William Roberts recommended potassium bicarbonate (ʒss—2.0, in a tumbler of water at bedtime) to stem the nightly acid tide. Some authors

highly recommend the salicylates for acute attacks of gout, both primary and intercurrent, in the course of the chronic form. In my own experience they have been less effective in this disease than colchicum. Benzoic acid and colchicin have a double action in that they stimulate the hepatic circulation and also "bind the glycocol, thus preventing it from precipitating the uric acid in the organism."[1] C. von Noorden and L. Schliep[2] have shown that there is a certain tolerance for nucleins in gout; and in every case its exact degree should be determined by allowing a definite number of grams of meat per diem and extracting the uric acid, the diet being restricted accordingly. Luff has demonstrated by experimentation the negative value of the alkalies and salicylates in the treatment of gout. If nephritis or a failure of compensation be present, even the former remedies should be administered with extreme caution.

For *chronic gout* potassium has been much used, though with slight advantage to the patient, I think. Fenner lauds a sterilized solution of thyminic acid (gr. ij—0.13) by intramuscular injection, in subacute and chronic forms of podagra. A small dose (gr. iv—0.26, daily) after meals for three months and then every alternate week, tends to avert the onset of acute symptoms. The bitter tonics, combined with a vegetable salt of iron, as well as change of climate, should be resorted to in the anemic, debilitated class of gouty patients.

LITHEMIA.

Definition.—A condition due to a disturbed cellular metabolism. It is characterized chemically by an excess of uric acid in the blood, and clinically by various digestive, circulatory, genito-urinary, and nervous phenomena. My purpose in describing lithemia is that the common error may be avoided of attributing its symptoms to other causes.

Pathogenesis and Etiology.—Lithemia is comparatively a latent condition. There is an *excess of uric acid*, which may be for a time eliminated through the natural channels (kidneys, lungs, skin, etc.) without the occurrence of symptoms. On the other hand, when, as the result of too little exercise, impaired elimination, luxurious living, the use of sweet wines, uric acid is allowed to collect in different parts of the organism, marked disturbances—nervous, gastro-hepatic, etc.—follow. DaCosta defines lithemia as a condition in which "the income of nutriment is in excess of the output of waste." C. G. Stockton holds that lithemia is a gastro-intestinal auto-intoxication. Among agencies that *predispose* are alcoholism, heredity, climate (temperate or cold climates favor diminished action of the skin), and the male sex.

Symptoms.—The nervous, circulatory, respiratory, integumentary, and genito-urinary symptoms are practically the same as those described under Irregular Gout; but I would here emphasize the broad clinical fact that the urethral and genital mucous membranes often become inflamed on slight provocation, producing urethritis, cystitis, orchitis, epididymitis, vaginitis, endometritis. These conditions resist treatment.

Gastro-intestinal Symptoms.—The appetite is variable, sometimes voracious, and at other times it is impaired or perverted. The tongue is coated, and a metallic taste is often complained of, while various forms of indigestion attend. There may be a delay in the conversion of the

[1] H. Kionka, *Deutsch. med. Woch.*, July 20, 1905. [2] *Berlin. klin. Woch.*, October 9, 1905.

albuminoids, causing *pyrosis*, *gastric oppression*, *fulness*, and sometimes nausea and vomiting soon after food. These symptoms, together with *marked flatulence*, are manifested at a later period after meals if there be failure in the digestion of the carbohydrates. The bowels work irregularly, and there may be diarrhea attended by colicky pain, with frothy and ill-smelling discharges. Hemorrhoids are usual, and melena may occur independently of the hemorrhoids. The liver is somewhat enlarged and often tender. A few prominent **cardio-vascular symptoms** should be mentioned, such as *palpitation*, particularly after eating, or lying abed. *Increased arterial tension* may develop early, and is due probably to the action of toxins in the blood upon the vaso-motor nerves, exciting universal contraction of the arteries. This condition often leads to arterio-sclerosis and sooner or later the well-known clinical group—chronic gout, arterio-sclerosis, and granular kidney—will be presented.

Treatment.—(1) **Prophylaxis.**—The patient should be taught the lesson of thorough mastication, and robust, plethoric persons should exercise with method in the open air, with a view to consuming the fats in the body. For this purpose cycling, horseback-riding, rowing, and walking are all excellent. Nervous persons, however, demand rest (Gray). The constant use of lithia-water, more particularly in the spring of the year, is warmly advocated by Wilcox.

(2) **Diet.**—As in gout, so in the preliminary stages of lithemia, no single dietary suits all cases, though I agree with those who contend that a diet consisting chiefly of albuminoids is proper in most cases. The lighter forms of albuminous articles of diet are to be preferred, and, if well borne, fruits and green vegetables may be added. Assuming that certain cases are dependent upon an auto-intoxication from the gastro-intestinal tract, the object should be to limit fermentation by the use of a nitrogenous diet; and I have found large amounts of water very beneficial in such instances. There are cases in which the gastric digestion is feeble, and in such the carbohydrates are better borne than the albuminoids. Cream and good butter are the only forms of fat to be allowed. Alcohol should be interdicted.

(3) **Medicinal Treatment.**—If the patient be robust, it is well to begin with a saline laxative, such as Carlsbad Sprüdel salt (℥j–ij—4.0–8.0). moderately diluted and taken before breakfast. If necessary, the hepatic function may be stimulated still further by a mild mercurial or by podophyllin. On the other hand, the neurasthenic, delicate sufferer must use a milder form of laxative, such as Rochelle salt in the same dose, or sodium phosphate in the morning, or a rhubarb pill at night. This class of lithemics also requires nerve-sedatives (sodium bromid, etc), and diuretics to aid in the excretion of uric acid. If it be true, as some claim, that the sodium phosphate is for the greater part excreted by the urine, and that it holds in solution more uric acid than any other salt, it is one of the foremost remedies in the treatment of the affection. Personally, I have found it to be a most useful agent. To reduce acidemia and to stimulate gently hepatic activity the salts of lithium, highly diluted, may also be tried. To aid in the digestion of the albuminoids hydrochloric acid may be needful, and if the appetite be impaired it may be combined with a simple bitter or with nux vomica (♏x–xv—0.666–9.999) thrice daily.

RACHITIS.

(*Rickets.*)

Definition.—A constitutional disorder of childhood, exhibiting developmental anomalies, chiefly in the bones and cartilages, causing deformities.

Pathology.—A mere summary of the anatomic characters can be given here. There is a derangement of the nutritive processes which retards and otherwise modifies the growth of the bony skeleton, particularly of the skull, the ends of the ribs and of the long bones. The latter soften or remain unduly flexible as the result either of the absorption of ossified structures or of the greatly diminished deposition of lime-salts. Longitudinal section of the long bones shows the seat of the chief changes to be at the junction of the epiphysis with the shaft. In health we note at this point two thin layers, an outer (next to the epiphyseal cartilage) proliferative zone, and an inner layer (of ossification). In rachitis both zones, though more particularly the proliferative, are greatly thickened, much softened, and their margins irregularly notched. The periosteum is thickened and easily separable from the shaft.

A *microscopic* examination shows an increased rate of proliferation of the cartilage-cells with a scanty, fibroid matrix, while the ossific layer presents disseminated and imperfectly calcified areas. Similarly, the osteoblastic layer of the periosteum is thickened, and remains spongoid. It is highly probable that absorption of true bone-tissue rarely occurs, and that the most characteristic pathogenic change is a lack of development of the normal structures. The morbid changes may arise from the presence of hyperemia of the cartilage, marrow, and periosteum—a process that interferes with the deposition of lime salts.

The cranial bones present areas of the so-called craniotabes, and yield to the pressing finger in consequence of delayed ossification. This may lead to a disappearance of the cranium in certain areas, causing depressions, while flattened protuberances may develop over the arterio-lateral regions. When cases terminate in recovery the bones become hard and ossify, although the deformities persist. The chemist has shown us that rachitic bones may contain less than half the normal percentage of lime-salts. The liver and spleen are moderately enlarged, and rarely the mesenteric glands are increased in size.

Etiology.—(1) Rachitis may occur in the *new-born*. Schwartz states that among 500 new-born children in Vienna, 75.8 per cent. show distinct signs of rachitis. Doubtless this estimate is too high, and entirely at variance with the experience of clinicians in general; but I believe that congenital rickets is by no means a rare condition. Many of the cases are still-born, and those that outlive childhood become peculiarly dwarfed (*micromania*). (2) *Heredity.*—The instances in which rachitis develops at an early period of life, due to ante-partum causes, are not rare, but it must not be forgotten that it is extremely hard to estimate the influence of heredity where both parent and child are exposed to similar unfavorable hygienic and dietetic conditions. Ill-health, malnutrition, close confinement, lactation, and syphilis may all act as predisposing factors during pregnancy. Setting aside syphilis, and perhaps phthisis, the state of

the health of the father has little if any effect in the causation of rachitis in his offspring. (3) *Geographical Distribution.*—The disease is more common by far in large cities than in rural districts, and in European countries—Russia, Germany, Great Britain, and Italy more especially—the disease prevails more extensively than in America. It is rare in tropical countries and during the hot season in temperate climates. (4) *Race.*—The colored race furnishes a preponderance of rachitic subjects. The reason for this may be a racial need of warmth that is not supplied by the temperature of more northerly latitudes, their native habitat being in a more southerly climate. The Italian race also suffers inordinately. (5) *Station.*—It is especially among the ranks of the poor children, whose environment is highly unfavorable, in large cities that rachitis is seen. Joukownski, from personal observations in over 3000 poor children in St. Petersburg examined for rachitis, found that from the working-classes come the greatest number of cases. Like scurvy, rickets may be found in the families of the wealthy under perfect hygienic conditions (Osler). The quarters of the cities in which the poorer classes live are densely crowded, the dwellings are insufficiently ventilated, and there is a great lack of sunlight. (6) *Diet.*—The disease is dependent largely upon unsuitable or insufficient food; and among hand-fed children, especially if the milk is sterilized, the disease is much more common than among those at the breast. It also occurs in breast-fed infants when the mother's milk is poor in quality as the result of previous ill-health or too long-continued lactation. The view was at one time widely held that rickets was produced by a farinaceous diet, and that the active agent was lactic acid, produced by the fermentative processes set up by the starch. Granting that the lactic acid forms a soluble salt by union with the lime of the bone, thus removing it from the system, this does not explain the productive lesions described under Pathology. According to another view, which is supported by experimental proof, rachitis is apt to develop when the amount of both proteids and fats is low. Certain forms of diet predispose to rickets, principally for the reason that they do not supply certain necessary articles in adequate proportion. (7) *Age.*—Of 903 cases, more than 75 per cent. occurred before the end of the second year, but of these only 99 commenced during the first half-year (Bruennische, Von Rittershain, Ritsche). It may occur as late as the tenth year. (8) *Sex* is without effect. (9) *Syphilis.*—Divers views are entertained regarding the rôle played by syphilis as a cause of this disease. It cannot be denied that syphilis brings about a marked impairment of nutrition, so that the disease may engender a predisposition to rickets.

(10) Findlay[1] attributes rickets to lack of exercise and confinement.

Bacteriology.—Mircoli contends that it is produced by the action of ordinary pyogenic organisms upon the osseous and nervous systems.

Symptoms.—The onset is *slow*, and the symptoms of gastro-intestinal catarrh, with their usual effect upon the general nutrition, may precede or accompany the true rachitis symptoms. At the beginning the infant is restless, irritable, and sleeps poorly, and slight fever is present in some cases. About the head and neck the child perspires freely, especially when asleep, wetting his pillow while the rest of the bed is dry. It is also annoyed by the bed-clothes, which it continually throws off,

[1] *Boston Medical Journal*, July 4, 1908.

lying exposed even in a cool temperature. Among the *earlier symptoms* is a tenderness both over the bony surfaces and the soft parts, so that the patient wishes to keep still and dreads to be handled. The child is languid and disinclined to move his limbs or to walk or play, even if he have done so previously.

The symptoms are progressive in their development, rachitis being ordinarily a chronic disease, so that after many months more pronounced features, including various bone-deformities, appear. Owing to the impairment of nutrition of the muscles the use of the limbs may become impossible, and these cases have been spoken of by writers as "rachitic paralysis;" this, however, is a misnomer. Cases have been reported by Berg and others that resembled spastic paralysis, pseudo-hypertrophic paralysis. Urinary phenomena are neither constant nor characteristic. Secondary anemia of mild grade supervenes, the hemoglobin often being comparatively low, and there may be a leukocytosis.

The first rachitic *osteal changes* are presented by the cranial bones, the ribs, the radius, and the ulna. The cranium appears enlarged, though this enlargement is more apparent than real, being due to the diminished size of the facial bones. The sutures remain open, the fontanels are large, and their closure is delayed, sometimes until the fifth or even the eighth year. *Craniotabes* is most frequently seen in infants under one year of age. This soft, thin condition of the bones is due to pressure both from within and without; it occurs on the surfaces on which the head of the child rests while lying. To detect the presence of craniotabes light pressure with the fingers is to be made in a direction away from the sutures. It is to be recollected that craniotabes is often a syphilitic manifestation. *Per contra*, increased hardness of certain bones may be observed (cranio-sclerosis). A *rachitic head* generally approaches a square in outline, or it may present marked angularities, with an increase in the antero-posterior diameter and a flattened top. Hyperostosis may cause prominence of the parietal and frontal eminences, giving the forehead a square, broad outline. A short, round head (brachycephaly) may rarely be met (Bonnifay). The veins of the scalp are enlarged, and the hairy growth is usually scanty, being often removed from the back of the head by rubbing. Drs. Whitney and Fisher first called attention to the fact that the ear placed over the anterior fontanel often detects a systolic murmur. A considerable patency of the anterior fontanel both in health and disease allows of detection of this murmur, however, and hence its diagnostic value is slight. A prominent feature of the disease is delayed teething, the teeth that appear being deficient in enamel, ill-shaped, although not prone to decay.

The *ribs* early become beaded. Anteriorly, where they join the costal cartilages, swellings occur, causing the "rachitic rosary." This is composed of nodules corresponding with the costo-chondral articulations, and these can generally be seen and always felt under the skin. They rarely outlast the fourth or fifth year. The ribs present two short curves —one at the junction of the dorsal and lateral parts of the thorax, and the other in front, where they turn sharply inward toward the sternum. This deformity is the result of the atmospheric pressure upon the softened bones, a shallow groove usually being produced in the line of the costo-chondral articulations or obliquely from the second or third rib downward

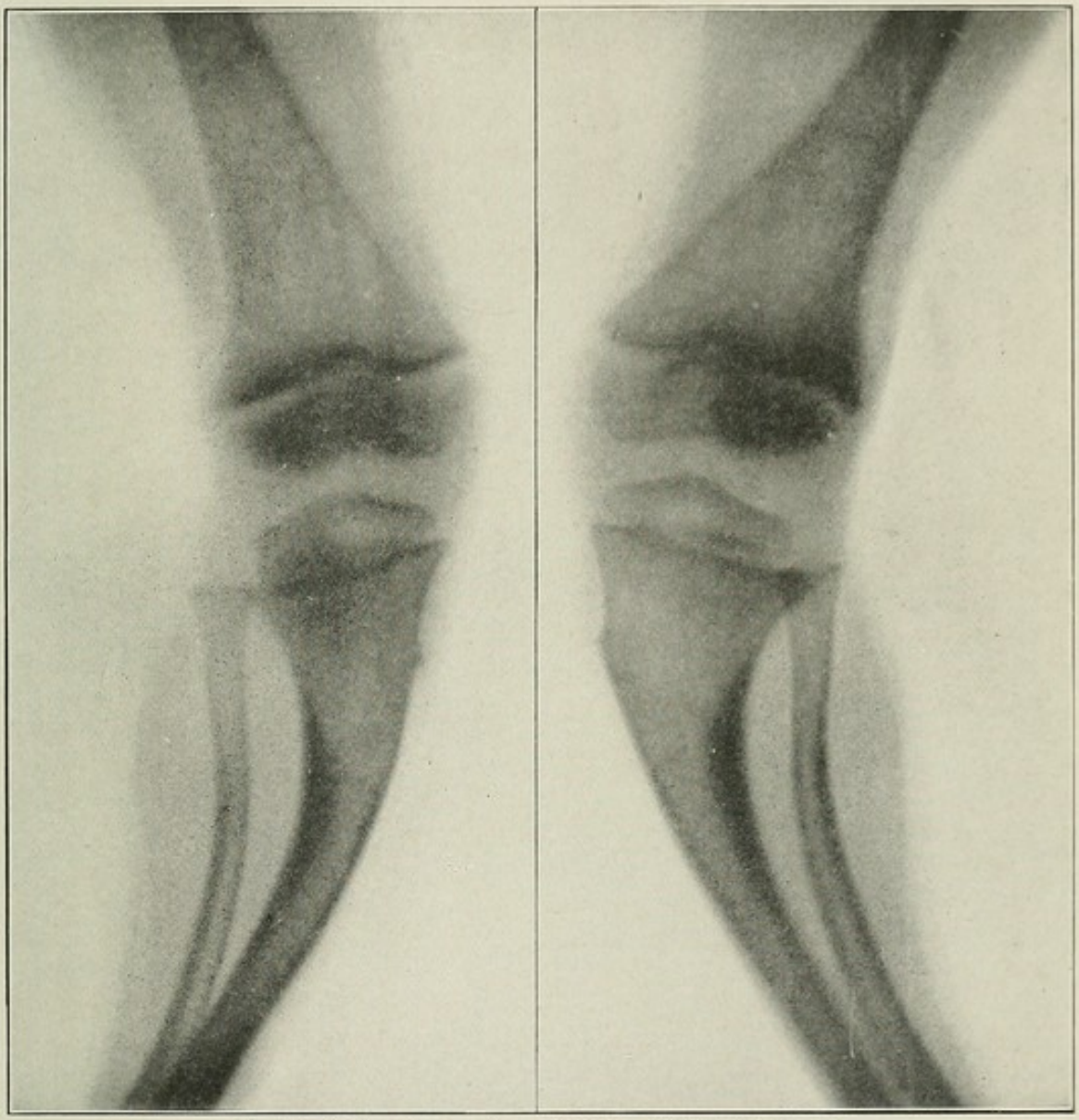


FIG. 33.—Outward curvature of tibia and fibula (Willard).

and outward. These changes lessen the transverse diameter of the thorax in front and interfere with the lung-expansion in the antero-lateral portions of the chest. They also produce bulging of the sternum, resulting in the so-called *pigeon* or *chicken breast.* On both sides, from a point corresponding to the anterior end of the eighth or ninth rib, there passes outward toward the axilla a furrow (Harrison's groove) which is caused by an eversion of the lower part of the thorax, and is heightened by atmospheric pressure, particularly during inspiration. This thoracic deformity is not peculiar to rickets, but is met with in all cases in which there is moderate obstruction to the ingress of air into the lungs.

Among the first indications of rickets is an enlargement of the lower end (junction of the shaft and epiphysis) of the *radius.* The radius and ulna are sometimes twisted and deflected outward, owing to the fact that some of the body-weight is supported by the hands when sitting or crawling. The clavicle may be thickened and curved near either end, and occasionally the scapulæ may be enlarged, but deformities of the upper extremities are rare as compared with those of the lower. Occasionally the vertebræ and intervening cartilages soften, with a resulting spinal curvature, usually antero-posterior.

Pelvic deformities are not uncommon, and are of no little importance in female children as bearing upon the questions of marriage and subsequent labor. The femora may be curved, often forward and more rarely outward; swelling of the lower end of the tibia is, however, the first change to be observed in the lower extremities. In some well-advanced cases the heads of the bones forming the knee-joints are also enlarged, and outward curvature of the femora and tibia is common, especially under the age of one year (see Fig. 33). After the child begins to walk a forward bowing of these bones, due to the weight of the body and to muscular action, occurs. Knock-knee is sometimes observed. Those who have suffered from rickets in infancy usually fall short of the average stature on reaching adolescence, giving rise to disproportion between head and height.

These skeletal changes sustain a causal relation to many, and some serious, affections, chiefly *nervous.* Thus, craniotabes is supposed to induce laryngismus stridulus, though this condition may also arise in the rachitic without cranial softening. Rickets also predisposes to tetany, which affects most commonly the upper extremities. Convulsions are prone to occur in this disease. The reflex nervous excitability is unquestionably exaggerated in rickets, and another cause for the eclampsia often met with is the associated gastro-intestinal catarrh. The abdomen becomes greatly enlarged, chiefly by flatulence, though to a less extent also by the swelling of the *liver* and *spleen* (passive congestion). Wm. Ewart[1] has recently called attention to the importance of abdominal atony and distention in rickets; it interferes with circulation and respiration. *Chest-complications* due primarily to interference with the cardio-pulmonary circulation, and the respiration, are common. Among these are atelectasis, bronchial catarrh, broncho-pneumonia, and emphysema. Anemia, when present, may be accounted for by some complication. *Green-stick fracture* of the bones often occurs in the rachitic subject.

Diagnosis.—Says Holt: "The most important early symptoms for diagnosis are sweating of the head, craniotabes, great restlessness at night, delayed dentition, and enlarged fontanel. All these, taken

[1] *Brit. Med. Jour.*, Oct. 13, 1906.

separately, may mean something else, but collectively they can mean nothing but rickets." At a later period the beading of the ribs and other characteristic deformities are usually present.

Prognosis.—The evolution of rickets is a long process; hence most patients become weak, anemic, and emaciated. The so-called "fat rickets" is not rare. Innately, the disease tends to spontaneous cure, which is attained from the end of the second to the fifth year; but its course may be abridged to a few months by appropriate treatment. When death occurs, it is usually occasioned by one or other of the complications before mentioned (laryngismus stridulus, pneumonia).

Treatment.—Prophylaxis.—Simple means directed to the antepartum causal factors in the mother may be preventive of rickets. Prophylaxis also embraces appropriate feeding and other agencies that tend to maintain the normal nutrition of infants.

Hygienic Management.—*Proper feeding* is an important factor, and if the child cannot be satisfactorily nursed by its mother and if it is under the age of six months, a wet-nurse should be procured. Should this not be practicable, it must be hand-fed, and the best artificial food is cows' milk, if properly prepared. It is diluted to suit the age, and I have found that barley-water, when made in the manner recommended by J. Lewis Smith, may be added to milk, replacing the water most advantageously. A heaping teaspoonful of barley-flour is poured into 25 teaspoonsful (℥iij—96.0) of water, and when the mixture is lukewarm 10 or 15 drops of diastase (Forbes) are added to it, the gruel in a few minutes becoming much thinner from the digestion of the starch. The physician must regulate with much precision the frequency of the feeding, and the amount of food taken according to the age of the child. The stools are also to be inspected. If they are green or if curds appear, either digestion is imperfect or the child is being over-fed. Older children may be given the lighter meats, freely, green vegetables, and fruits, but these must be carefully selected.

Other *hygienic details* are of little less importance than a proper diet. The decubitus of the child must be changed frequently, so as to prevent bony deformities; moreover, the rickety child should not be allowed to walk, and to prevent his doing so splints extending beyond the feet have been recommended. A tepid bath, warm clothing, and a prolonged daily stay in the open air are measures that should not be neglected.

Of *medicines*, those that rank highest are phosphorus, the hypophosphites, iron, and cod-liver oil. The officinal oleum phosphoratum (gr. $\frac{1}{150}$—0.0021) is used by Jacobi. Phosphorus is highly spoken of by many writers. It may be given either pure (gr. $\frac{1}{200}$ to $\frac{1}{100}$—0.0003 to 0.0006) or preferably in the form of an emulsion with sweet oil or cod-liver oil:

℞. Phosphori, gr. $\frac{1}{10}$ (0.00648);
Olei olivæ, ℥ij (64.0);
M. et ft. emulsio.

Sig. ℥j three times a day, after meals; for a child under the age of one year.

Kissel states there is no evidence in favor of the use of phosphorus in rickets. Baginsky, Leray, Weiss, and others have found from its

extensive employment that it is of doubtful value in the majority of cases. Kassowitz, Swetchen, and others, however, observed cases with cure, hence the remedy deserves a trial.

When it is desired to administer cod-liver oil and it is not tolerated by the stomach, it may be rubbed gently into the skin of the thighs and trunk. Arsenic in small doses has proved to be a capital remedy in selected cases; and iron, particularly in combination with arsenic, is indicated if anemia be pronounced. Klotz recommends pituitary extract, supplementing it with calcium carbonate to supply material for bone growth.

The numerous *complications* to which rachitic subjects are liable present special indications which are to be met by the same measures as when they arise under other circumstances. The condition of the digestive organs must be kept constantly in mind; and no remedy, however promising, that is designed to assist the general condition should be continued if it tends to aggravate the digestive disturbance. Ewart advises massage for the abdominal atony and also the use of an elastic belt which gives support to the abdominal parietes and improves circulation and respiration. The treatment of the rachitic deformities belongs to the domain of the orthopedic surgeon and should be undertaken early.

SCORBUTUS.

(*Scurvy.*)

Definition.—A constitutional disorder, dependent upon dietetic errors, and characterized by anemia, excessive weakness, spongy gums, a tendency to muco-cutaneous hemorrhages, and a brawny induration affecting chiefly the muscles of the calves and the flexor muscles of the thighs. Scorbutus and rickets, distinct affections, often coexist.

Pathology.—We know nothing concerning the pathogenesis of scurvy. Evidences of profound anemia are found upon microscopic examination of the blood, which is thin and dark, but there is no leukocytosis. The skin may show spots of subcutaneous hemorrhage (ecchymoses), but the most characteristic hemorrhage is that under the periosteum of the femora. Bleedings into the articulations and muscles may also at times be noted, and occasionally the serous membranes are the seat of hemorrhages, as well as the internal organs. Submucous hemorrhages are extremely common. The intestinal mucosa may also present ulcers. The gums are swollen, spongy, dark in color, and sometimes ulcerated, and the teeth may be loose or missing. The epiphyses, particularly of the lower end of the femora, may be congested, and even detached. The spleen is soft and swollen. The heart, liver, and kidneys sometimes show fatty and often parenchymatous degeneration.

Etiology.—Incidence.—In former times scurvy was very prevalent among sailors at sea and soldiers in the field, and epidemics were common. Doubtless, however, it has declined in importance as a disease incident both to sea-life and to armies; but, as pointed out by Wise, it would seem that changing physiologic and economic conditions may cause it to be dreaded on the land as it has hitherto been on the sea. Osler states that the disease is not infrequent among Hungarians, Bohemians, and Italian miners in Pennsylvania. It is rarely *epidemic* at the present day. F. A. McGrew records an epidemic

(with a total of 42 cases) in Chicago, in 1894. *Endemic* appearances of scurvy are still common, particularly in portions of Russia (Hoffman) and elsewhere also, sweeping through prisons, barracks, almshouses, and other institutions of like kind. While the majority of cases met with are sporadic, the above facts point to the infectious character of scurvy. Russell contends that adult scurvy is caused by lime starvation.

Bacteriology.—Testi and Beri have isolated a micro-organism which has been cultivated and inoculated into guinea-pigs and rabbits, producing in the latter pathologic lesions and symptoms simulating closely those of scurvy. The microbe is perfectly round and is a diplococcus. These experiments require confirmation.

Predisposing Causes.—The chief factor is an unsuitable dietary long continued. The absence of the organic (potassium) salts present in fresh vegetables disturbs normal histogenesis. Albertoni has recently shown that in scurvy of a protracted course free hydrochloric acid is absent from the gastric juice, and that the total acidity is much reduced, but this is so neither in every case nor at all stages of the disease. He found no deficiency of chlorids in the body. Peptonization is feeble. It may be a true toxemia.

Debilitating influences, as unhygienic surroundings, excessive muscular exercise, humidity, and cold, often play no mean role in causing scurvy. Mental anxiety and depression seem to have etiologic significance. The old are very susceptible, and all ages are liable to the disease. *Sex* has no special influence upon scorbutus. Starvation does not predispose to the disease.

Symptoms.—Scurvy has a *slow* onset. The earliest symptoms are generally a swelling around the eyes, over which the skin has the color of a bruise, and a pale face, which looks bloated and wears an apathetic expression. There is noticeable almost from the start a gradually increasing debility, emaciation, an inability to perform mental or physical labor, and despondency. The patient experiences arthritic and muscular rheumatoid pains and dyspnea on slight exertion.

With rare exceptions the *gums* swell, sometimes enormously, and become spongy, bleeding most readily. They may become ulcerated, and may be, though rarely, fungoid in appearance. The *teeth* often become loose, and in rare cases drop out. The *breath* emits an offensive odor, that is sometimes due to necrosis of the jaw. The *tongue* swells, though it is usually clean and often pale. In the mouth may be observed submucous hemorrhages in many cases. There is loss of appetite, but the digestion is usually good; there may, however, be constipation or diarrhea, more frequently the former. *Scorbutic dysentery* has been described by certain writers. The *skin* is dry and of a muddy color, blended occasionally with a greenish or greenish-yellow tinge. At the end of a week or ten days *petechiæ* and *ecchymoses* appear upon the legs, arranging themselves about the hair-follicles. These may also come out later on the trunk and upper extremities. Submucous hemorrhages may give rise to circumscribed swellings, and *subperiosteal hemorrhages* may occur and engender node-like protuberances. There may be frequently noticed a peculiar brawny induration, due to extensive hemorrhagic infiltration of the muscles and subcutaneous tissues, most marked in the hams and calves. The condition is not without considerable *pain*, particularly

if the parts be touched, and in severe cases bullæ and vibices may be seen, as in a recent case of my own. Hemorrhages from the mucous channels of the body occur, and epistaxis is frequent. In bad cases hematuria, also melena and rarely hematemesis, may be observed. Blood may be effused into the serous membranes, accompanied sometimes by inflammatory changes in the latter; also into the lungs, which are rarely the seat of secondary pneumonia. Pulmonary infarction occurs, but is a rare event. Hemoptysis may be a symptom of the lung-complications or may occur as an independent phenomenon.

The *heart* may present symptoms, such as palpitations, feeble impulse, arrhythmia, and sometimes a basic blood-murmur, but these are without diagnostic importance. The pulse is soft, small, and on exertion much accelerated. The *temperature* is sometimes subnormal, and the presence of fever is a certain indication of the existence of some complication.

The *nervous symptoms*, aside from the profound mental depression, are not prominent. Insomnia may be a distressing symptom. Delirium (late) is sometimes witnessed. Meningeal hemorrhage may supervene. Both night-blindness and day-blindness are among the rarer and extraordinary ocular features.

The *urinary symptoms* vary in different cases. Albuminuria is common. The specific gravity of the urine is increased, the color high, and solid constituents diminished, except the phosphates, which are abundant. Albertoni found the proportion of chlorids less than the normal, while other investigators claim that the percentage is high. *Nephritis* may occur as a complication. The bones in long-standing cases may be congested and sometimes necrotic, and the epiphyses may separate from the shafts. In one of my cases an old cicatrix reopened.

Diagnosis.—This rests upon the following points: the history, the peculiar facies, the spongy and swollen gums, the gingival and deep-seated cutaneous hemorrhages, the progressive loss of strength and energy, great mental depression, and the speedy recovery after an appropriate regimen. Scurvy will be distinguished from *purpura* under the description of the latter disease.

Prognosis.—Unless far advanced, the prognosis generally becomes good upon the institution of correct dietetic principles. If the disease have made extensive inroads, the danger to life is considerable. The gravity of the internal symptoms (particularly pulmonary) is far greater than of the external, and, indeed, the presence of the latter is a favorable omen. Certain complications augur a serious termination, such as pneumonia, hemorrhagic infarctions of the lung, pleurisy with bloody effusion, dysentery, acute nephritis, etc.

Treatment.—**Prophylaxis.**—By carrying out the known means of prevention the disease has been diminished more than 90 per cent. among mariners and soldiers. This change has been brought about by the enforcement of governmental regulations which demand that an adequate supply of antiscorbutic articles of food must be provided for military campaigns and for long sea-voyages. Fresh fruits and vegetables can be readily transported in hermetically sealed jars or cans.

Treatment of the Attack.—The chief indication is to be met by the use of fruits and fresh vegetables. Of the former, two or three

lemons daily or oranges and other fruits suffice to work a surprising degree of improvement in a short space of time. Baumann and Howard's experiments show that the loss of various food constituents through the feces is lessened when fruit juices are added to the diet. Antiscorbutic vegetables (potatoes, water-cresses, raw cabbage, lettuce, saur-kraut) in liberal quantity should also be given. Meats, eggs, milk, and farinaceous dishes are not to be prohibited, since the patients require all forms of food; but if the digestive power be feeble it is advisable to begin with the juice of oranges or lemons, conjoined with meat-juice, egg-white, milk, and light farinaceous articles, adding the stronger forms of animal food and fresh vegetables when improvement is noted. We may assist the digestive function by the use of simple bitters, strychnin, and hydrochloric acid; hematinics are sometimes indicated.

Special symptoms may call for appropriate measures. Constipation requires simply an enema. On the other hand, diarrhea presents an indication for intestinal antiseptic and astringent remedies. The oral condition varies, hence the measures to relieve it vary also; but if ulcers be present, the solution of potassium chlorate is best. For swelling of the gums the application by means of a cotton swab of tannic acid (2 per cent.) or a solution of silver nitrate (2–5 per cent.) is serviceable. A combination of boric and carbolic acids in a solution of suitable strength may be used as a mouth-wash. If copious hemorrhages occur, hemostatics are eminently useful. The various complications must be met by the usual measures, according to their nature.

Infantile Scorbutus.

(*Barlow's Disease.*)

Definition.—A constitutional disease, characterized by the same symptoms as scurvy in adults, except that in many instances undoubted evidences of rachitis are associated.

Pathology.—The bones are thickened and excessively sensitive, owing to a marked subperiosteal hemorrhage, with more or less maceration, and want of firmness between the epiphysis and shaft. The muscles may be the seat of effusion. The lesions of rickets are often associated.

The *nature* of the affection is unsettled. Originally looked upon by most observers as acute rickets, it was subsequently described by Cheadle (from the clinical side) and Barlow (from the anatomo-pathologic side) as infantile scurvy. On the other hand, Ashby of Manchester, Fürst and other German writers, are inclined to the view that the affection should be considered a hemorrhagic form of rachitis. The belief that rickets predisposes to scurvy, but that the two diseases have not the same pathogenesis, is probably the correct one.

Etiology.—Scurvy is largely confined to *hand-fed infants*, especially those reared upon the numerous infant-foods which have been foisted upon the market, including condensed milk, etc. Louis Starr, Jacobi, and others have shown that it sometimes follows the prolonged use of sterilized milk, although the etiologic importance of the latter food has been over-emphasized. Concetti[1] concludes that infantile scurvy is caused by alimentary intoxication. An investigation by a committee of the American Pediatric Society[2] showed that of 379 cases the majority

[1] *Archiv f. Kinderheilk*, April 17, 1909. [2] *Medical Record*, July 2, 1898.

occurred between the ages of 7 and 14 months, inclusive, and that the disease has a greater tendency to occur among the rich or well-to-do. "The farther a food is removed in character from the natural food of a child, the more likely its use is to be followed by the development of scurvy."

Symptoms.—The *skin* presents the muddy color peculiar to the disease in adults. The patient may be well nourished, but more often there is a tendency to *wasting*, and other symptoms of impaired nutrition appear, particularly irritability and disinclination to exertion. The more characteristic features appear after one or two months, and the child cries when handled, especially on touching the lower limbs. About the same time there is an irregularly cylindrical swelling of one of the thighs, due to subperiosteal effusion. Soon the other limb is similarly involved, though not always to a like degree. At first the legs are flexed, but later they become straightened and slightly everted on account of the progressive hemorrhage or separation of the epiphyses. The bones in other portions of the body may be involved secondarily in more or less rapid succession, but the swellings are less marked than in the lower limbs. Later, if teeth be present, the gums may swell and become spongy. Ecchymoses in the form of petechiæ appear upon the skin-surface, and particularly about the eyes. Barlow describes a remarkable ocular phenomenon: "There develops a rather sudden swelling of one eyebrow, with puffiness and very slight staining of the upper lid. Within a day or two the other lid presents similar appearances, though often of less severity. The ocular conjuntivæ may show a little ecchymosis or may be quite free. Hemorrhages from the mucous surfaces may finally put in an appearance.

Diagnosis.—To distinguish *rickets* from infantile scurvy Barlow's brief though clear aggregation of the characteristics of the latter disease may be quoted: "(1) Predominance of lower-limb affection, in which there is immobility going on to pseudo-paralysis; excessive tenderness; general swelling of the lower limbs; skin shiny and tense, but seldom pitting, and not characterized by undue local heat; on subsidence revealing a deep thickening of the shafts, also liability to fracture near the epiphysis. (2) Swelling of the gums about erupted teeth only, varying from definite sponginess to a minute, transient ecchymosis."

In incipient and anomalous cases there is danger of diagnosticating rheumatism when scurvy is really the condition present (Griffith).

Prognosis.—Favorable, even in well-established instances, if brought under the proper regimen.

Treatment.—An antiscorbutic dietary—mother's milk or fresh cows' milk, meat-juice, and orange- or lemon-juice—successfully meets the main indication. If there be systemic exhaustion—a condition that is not infrequent—gentle stimulation with brandy (highly diluted) and an abundance of fresh air are pre-eminent among the measures to be employed. Iron, arsenic, and cod-liver oil may be needful to complete the cure, but usually the simple means already mentioned will prove effective. The limbs, especially the lower, may claim attention. Local treatment, however, is rarely necessary, except there be separation of the epiphyses, when suitable splints are to be applied.

PURPURA.

Two main groups are to be distinguished: (1) **Secondary purpura**, which occurs from a great variety of causes and in numerous affections, in which its clinical significance has been pointed out in appropriate sections of this work. It seems pertinent, however, to enumerate the chief among the diseases and conditions under which it may arise, as follows: (*a*) scurvy; (*b*) acute infectious diseases (typhus fever, ulcerative endocarditis, cerebrospinal meningitis, variola, measles, septicemia and scarlatina, and typhoid rarely; (*c*) hemophilia; (*d*) numerous chronic affections, as nephritis, leukemia, pernicious anemia, jaundice, Hodgkin's disease, tuberculosis, syphilis, chronic alcoholism, and heart disease; (*e*) malignant sarcomata; (*f*) nervous affections, as locomotor ataxia, acute and transverse myelitis, and hysteria; (*g*) mechanical causes, straining efforts, paroxysms of whooping-cough, and violent convulsions; (*h*) certain drugs may produce a petechial eruption—the iodids, quinin, copaiba, belladonna, ergot, mercury, chloral, antipyrin, and turpentine; (*i*) snake-poisons produce rapid and extensive hemorrhagic extravasation (S. Weir Mitchell); (*j*) senile purpura (Bateman), situated chiefly on backs of the hands and along the forearms.

(2) **Primary** or **idiopathic purpura** forms the second group. It is divisible into (*a*) simple purpura (*purpura simplex*); (*b*) arthritic purpura, of which two varieties may be recognized: (1) *peliosis rheumatica*, and (2) *Henoch's purpura;* (*c*) hemorrhagic purpura (*purpura hæmorrhagica*).

(*a*) **Simple Purpura.**—The cause is unknown. Among predisposing influences, however, is *age*, the condition being most common in children about the time of puberty. It may be a sequel of the acute, infectious diseases, and in not a few cases develops in seemingly healthy subjects.

Symptoms.—This is the mildest variety of primary purpura. The *hemorrhages* into the skin take the form of petechiæ, vibices, or ecchymoses. The first are extravasations of blood in the form of minute points, that appear, as a rule, in the hair-follicles, and, unlike the erythemas, do not disappear upon pressure. The vibices receive their name from the fact that the hemorrhages occur as streaks, while the ecchymoses are larger, but similar in nature and behavior to the petechiæ. They may exceed in size that of a split pea, and their hue ranges from a deep red to a bluish tint. As they fade away they assume at first a yellowish-brown, then a yellow color, and finally disappear. The eruption appears in a series of crops, and its seat of election, often favored by the erect posture, is the legs (*orthostatic purpura*). Bloody serum may be effused into bullæ or large blebs. Shepherd and others have reported cases in which the purpuric eruption ended in gangrene, though in Shepherd's case the gangrene was due to the use of sodium salicylate.

(*b*) **Arthritic Purpura.**—(1) *Peliosis Rheumatica* (*Schönlein's Disease*). —The *cause* of this remarkable disease is unknown. Formerly many writers inclined to the view that it is of rheumatic origin, and since endocarditis and pericarditis are occasionally observed in association with peliosis rheumatica, considerable coloring is given to this belief. On the other hand, the fact that the cardiac complications are rare in arthritic purpura shows that not all cases of the latter disease are genuinely rheu-

matic. It occurs chiefly in males from the twentieth to the thirtieth year of age, Among the *prodromata* are angina, slight articular pains, headache, loss of appetite, and fever ranging from 100° to 102° F. (37.7°–38.8° C.). The affection is especially characterized, however, by *polyarthritis*, the joints being swollen, painful, and very tender; also by purpura, associated or not with urticarial wheals or erythema exudativum; and by subcutaneous edema. The purpuric *eruption* is the only symptom that has pathognomonic significance, and in this affection it shows a strong preference, as regards distribution, for the affected joints and the legs. The eruption, as already intimated, does not display constant characteristics. It may not differ from that of simple purpura, and the rash consists of petechiæ, ecchymoses, streaks, and rarely of bullæ (*pemphigoid purpura*); or it may be made up of wheals of urticaria, attended with intense itching; and, finally, it may be identical with erythema nodosum. These forms of eruptions may be variously combined.

Hemorrhages from the mucous surfaces rarely occur, though epistaxis is the most common. The extent of the *edema* varies greatly, in rare cases being quite extensive and overshadowing all other symptoms (febrile purpuric edema). *Albuminuria* may be noted, and accompanying the purpuric eruption there will be a mild febrile movement. Convalescence is usually protracted.

The *diagnosis* is made from the presence of three characteristic symptoms—polyarthritis, a purpuric rash, and edema. The combination of purpura and urticaria is one of the chief distinguishing features. It is not always possible to eliminate *rheumatism*, but the non-rheumatic character of some of the cases may be clearly shown by the therapeutic test, as happened in one of my own patients.

Prognosis.—This type of the disease is generally benign, death being very rare. Complications, however, may prove serious, especially the cardiac. The throat-condition may outlast the attack, and terminate in gangrene of the uvula or tonsils.

(2) *Henoch's Purpura*—Henoch and Couty have described a form of rheumatic purpura occurring chiefly in children, and characterized by painful and sometimes swollen joints; by a purpuric eruption, plus erythema multiforme; by vomiting, diarrhea, and intestinal pain; by localized edema of the skin; and by hemorrhages from the mucous membranes and sometimes into the kidneys.

The *diagnosis* is difficult in proportion to the scanty development of the purpuric symptoms, some of which are often wanting. *Intussusception* usually occurs earlier—in babes.

The *prognosis* is favorable, though complications of more or less serious import may arise. One of Osler's cases proved fatal with the symptoms of acute hemorrhagic Bright's disease.

(3) *Factitious Purpura.*—Bruce and Galloway[1] report a case in which any irritation of the skin, such as might be caused by drawing the blunt end of a pencil over it, produced a white line, which presently became pink and then intensely purpuric. In this way letters, figures, and the like could be shown as hemorrhagic outlines.

(*c*) **Purpura Hæmorrhagica** (*Morbus Werlhofii*).—This is the severest form of purpura, and its apparent etiologic connection with certain infectious diseases, particularly rheumatism, malaria, etc., is interesting, but

[1] *British Jour. of Dermatology*, Jan., 1898.

not well understood. The disease is perhaps most common in young females, particularly if they have fallen into general ill health; but all persons are liable, and post-mortem anatomo-pathologic pictures of the disease leave little room for doubt that it is an infectious complaint. The hemorrhages are probably due to an endotheliolysin produced by the action of bacteria (streptococci, staphylococci, pneumococci, etc.) under certain circumstances not perfectly known. Duke[1] believes that purpura hemorrhagica is caused by an agent which reduces the platelets of the blood to a sufficient degree.

Symptoms.—Prodromal symptoms, (malaise, headache, depression, anorexia) may appear, and last one or two days. The invasion is moderately abrupt, with fever, and soon cutaneous ecchymoses appear upon the skin, quickly increasing in size and numbers. Slight *hemorrhages* from the mucous membranes into the internal organs occur. Epistaxis generally comes first; it tends to persist and to recur, and the same peculiarities pertain to bleedings from other points. *Prostration* now becomes rather marked, the patient complaining of pains in the limbs, loins, abdomen, and chest, and the latter often presage a fresh hemorrhage. There is moderate *fever*, as a rule, the temperature during the height of the attack ranging from 101° to 103° F. (38.3°–39.4° C.), or it may reach 104° to 105° F. (40.5° C.), though rarely. The *pulse* is accelerated (120 to 130 per minute), but full and regular, though in the worst cases it becomes small and very rapid. The mind is usually clear. The face may be pale and anxious. *Hematuria* followed by *nephritis* may occur.

There is secondary *anemia*, varying in intensity with the extent of the hemorrhage. It is more pronounced, owing to a greater loss of blood, in this form than in the preceding varieties of purpura. Occasionally the red count falls very low, while there is often a slight increase in the number of leukocytes, although the different varieties are present in normal proportions. "In stained specimens of purpuric blood the number of blood-platelets is found greatly diminished in severe cases" (Pratt). The *course* is run in from seven to ten days in mild cases, while the severer attacks pursue a longer course. The malignant form (*purpura fulminans*) has, however, a speedily fatal termination.

The *diagnosis* of purpura hæmorrhagica rarely presents any difficulty. *Scurvy* may simulate it in some particulars, but is distinguished by its chief etiologic factor—a diet deficient in fresh vegetables and fruits—by the spongy, swollen condition of the gums, the loosened teeth, and brawny induration of the limbs. Moreover, in purpura hæmorrhagica the hair-follicles do not occupy the centers of the ecchymotic spots, and the hemorrhages from the mucous membranes are more copious than in scurvy. Malignant types of the eruptive fevers distinguish themselves by the history of the prevailing epidemic, by the characteristic prodromes and invasion, and by the high temperature, although variola purpura often pursues an afebrile course. A blood examination, which should always be made in purpura, will exclude *leukemia*.

Prognosis.—Grave, except in mild cases. In the malignant type death may come before hemorrhages from the mucosa appear. Certain complications may prove fatal—cerebral hemorrhage, inundation of the lungs with blood, Bright's disease, and shock from rapid, profuse bleedings. Death may also be the result of exhaustion due to protracted bleedings.

[1] *Archives of Internal Med.*, Nov. 15, 1912.

Chronic Purpura.—Two forms have been described—one in which the stage of convalescence of the acute form is interrupted by recurrences of the characteristic manifestations, the other in which the symptoms are continuous, lasting over months or years.

Treatment.—(*a*) The management of *secondary purpura* is embraced, in other portions of this volume, in connection with the treatment of the diseases and conditions which it accompanies.

(*b*) *Simple purpura* demands arsenic, first in moderate doses, and then increased until slight toxic effects are noticeable. Legroux speaks in warm terms of the iron compounds, and especially of iron perchlorid in doses of ʒss–j (2.0–4.0) daily, and if the child is somewhat anemic, the inhalation of oxygen will promote hematosis. The disease also requires fresh air in abundance and a generous diet.

(*c*) *In peliosis rheumatica*, in addition to the measures recommended in purpura simplex, the salicylates should be tried.

(*d*) *Purpura Hæmorrhagica.*—In all kinds of purpura the patient should be confined to bed. An abundance of nourishment, by supporting the patient's power, is of the greatest service. Internally, ergot, turpentine, tincture of the chlorid of iron, acetate of lead, and dilute sulphuric acid enjoy the widest reputation. Calcium chlorid, suggested by Wright, should be tried, as should also adrenalin chloride. A 2 per cent. solution of gelatin in normal salt solution given subcutaneously is a remedy of great value. The following combination, recommended by Hardaway, I have found useful:

℞. Fluidext. ergotæ,
Tr. ferri chlorid., āā fʒij (64.0).—M.
Sig. Three to ten drops in water, t. i. d.

HEMOPHILIA.

(*Bleeder's Disease.*)

Definition.—An hereditary affection, transmitted by females who are themselves not affected (Nasse's law). It is characterized by frequent uncontrollable hemorrhages that are either spontaneous or due to slight traumatism.

Pathology.—The constitutional changes on which the disease depends are to be found in the blood-vessels rather than in the blood itself (Henry); microscopic changes have been found in the arterioles, the middle muscular tunic being either absent or much atrophied. Vasomotor influences also play an important part in causing an attack. The blood presents slight changes, although Addis holds that the cause of hemophilia is an inherited peculiarity in the constitution of the prothrombin whereby its activation into thrombin is retarded. Synovitis with hemorrhages into the joints may sometimes be observed.

Etiology.—Hemophilia is more distinctly hereditary than any other known disease, but Nasse's law is not of such universal application as is supposed. R. Kolster found that of 50 hemophilic families, 18 cases followed this law, 16 others with some exceptions to its provisions, and 12 without any regard to it. The law embraces the following points:

The daughter (not herself affected) of a bleeder transmits the tendency to her sons, who become bleeders; her daughters do not suffer, but in turn transmit the disease to their sons. Females, however, may be bleeders, and, according to Virchow, one woman is affected to every seven men. The disease has been traced for centuries in a few families.

It is observed in all classes of society, and is most frequent in families whose members are large, vigorous, and have delicate complexions, the complaint usually manifesting itself before the end of the second year of life, though exceptionally as late as puberty. An acquired hemorrhagic diathesis is seen occasionally in connection with certain acute infections and more commonly in the graver anemias (leukemia, pernicious anemia). Weil refers the hereditary form to the presence in the blood of anticoagulants, and the sporadic to the absence of substances essential to coagulation.

Symptoms.—The occurrence of profuse and persistent bleedings that are either spontaneous or the result of slight injury characterizes hemophilia. The character of the injuries that lead to dangerous bleedings is often exceedingly trivial; thus a slight scratch, cut, blow, the extraction of a tooth, and other minor surgical operations (*e. g.* circumcision) may be followed by severe hemorrhage.

If we include spontaneous hemorrhages, bleedings take place most frequently from the nose. Legg has made three clinical groups, based on the intensity of the symptoms, as follows: (1) Seen most frequently in men, and characterized by external and internal bleedings of all kinds and by joint-affections; (2) most frequent in women, and distinguished by spontaneous hemorrhages from mucous membranes only; and (3) characterized simply by ecchymoses.

The capillaries ooze blood—a process that may vary in duration from a few hours to as many weeks. A fatal result may occur in a few hours, while, on the other hand, recovery may follow a slow oozing of blood that has continued for many days. In the latter instances profound anemia follows, the blood, however, being rapidly replaced. Extensive blood-extravasations (hematomata) usually follow contusions. Petechiæ, when they occur, are apt to be spontaneous. The coagulation time of the blood is delayed, due to insufficient formation of the fibrin ferment factors (Morowitz and Lossen). Fussell, in 2 cases, found the leukocytes increased (24,000 and 15,000 per c.mm.), while the red cells were moderately diminished.

Arthritic symptoms are common, the larger joints, and especially the knees, being most frequently affected and showing swelling that is due chiefly to hemorrhages into the joints. In other instances febrile synovitis may be present, resembling rheumatism. The joint-symptoms may either announce an approaching hemorrhage or pain alone may be experienced. The attacks are liable to recur, especially in cold, damp weather, and may result in stiffened, deformed joints (Musser).

Diagnosis.—When persistent capillary oozing occurs in a person with a clear, hereditary disposition the diagnosis is clear. Without an inherited tendency we cannot be certain of the diagnosis unless protracted hemorrhages from insufficient causes are repeatedly manifested. The presence of joint-involvement is very helpful.

Differential Diagnosis.—*Peliosis rheumatica* is an affection which, as Osler remarks, touches hemophilia very closely, particularly in the relation of the joint-swelling. It is true that the former may also show

itself in several members of a family, but the presence in this affection of more or less edema, and often of wheals of urticaria, accompanied by intense itching, aids greatly in its elimination.

Prognosis.—In undeveloped forms the outlook is not particularly grave, since in these the tendency may either lessen or become altogether arrested after childhood. In the majority of well-marked cases the children do not survive this period. On the other hand, those who live to become full-grown show a diminished, and in a small class of cases an absolute, disappearance of the tendency. The first hemorrhage rarely proves fatal. Boys suffer from a more serious form than girls. Moreover, menstruation, though sometimes very copious, does not to any great extent endanger the life of a hemophilic woman. Of 130 cases of pregnancy and labor, the death of the mother occurred in only 3, and abortion in 16 cases (Kolster).

Treatment.—The physician can do most in the direction of prophylaxis. All surgical operations that are not absolutely necessary must be avoided; neither should the teeth be erupted nor the operation of circumcision be permitted. Leeches are not permissible. Females who belong to bleeder families, as well as males who have had hemophilia, should not marry.

During the attack absolute rest—mental and bodily—must be enjoined, and light compression, and if this fail strong pressure or styptics, should be tried. In epistaxis ice, tannin, and turpentine should be tried before using nasal plugs; and if the latter prove indispensable, the lightest only should be employed. J. Greig Smith regards lint saturated with spirit of turpentine as the best local application in epistaxis. Adrenalin chloride and gelatin in 5 per cent. solution are of great value locally. Schloessmann suggests the systematic use locally of tissue extract, *e. g.*, fresh goitre tissue. Blood-serum may also be applied to the bleeding part. Internal medicines are of secondary importance, but opium is unquestionably of value, since it tends to favor repose. The remedies that have been given are various. Delafield, Fürth, and others have used successfully the fluidextract of hydrastis canadensis, the dose being from 20 to 40 drops daily; among other hemostatics, gallic acid, turpentine, and iron perchlorid produce the best results. The dose of the latter should be ʒss (2.0) every two hours, with a purge of sulphate of soda (Legg). The use of calcium salts has produced good results in some cases and merits a trial, as does adrenalin chloride. The subcutaneous injection of the gelatin solution already mentioned has undoubted value. Gelatin seems also to have hemostatic power when given by mouth or rectum. The most successful method (first advocated by Weil) is fresh normal blood, either from the horse, or rabbit, or human blood (Lappe). The dose of the fresh serum if given subcutaneously is 20 to 40 c.c. The coagulability of the blood is greatly increased thereby. Sahli advises repeated injections of fresh human serum and repeated small bleedings to stimulate the physiological reactive thrombokinase formation. Kneading the tissues immediately surrounding the bleeding-point liberates thrombokinase and thus tends to arrest hemorrhage. Thyroid extract and inhalations of oxygen have also been advocated. During convalescence, arsenic, iron, the bitter tonics, and a liberal dietary will aid full recovery.

HEMORRHAGIC DISEASES OF THE NEW-BORN.

(*a*) **Epidemic Hemoglobinuria** (*Winckel's Disease*).—This affection, which is *septic* in nature, is occasionally met with in lying-in hospitals, and occurs in children from one to ten days after birth. The infants refuse the breast and show hematogenous (?) icterus; gastro-enteric catarrh is an attendant of the disease. The stools are meconic; the urine is scanty, dark colored (from methemoglobin), often albuminous, and may contain casts. Hemorrhages occur into organs other than the kidney and into the mucous membranes, there also being mild fever, rapid emaciation, and often mild convulsions. It is a very fatal disease. Bacteriologic experiments have shown that the disease may be produced by the growth of the colon bacillus in the buccal epithelium of infants. Kilham and Mercelis[1] report an epidemic of 10 cases occurring in the New York Infirmary; complete bacteriologic studies were made in all, and the organism discovered suggested the diplococcus of pneumonia or the pneumococcus group. There is, however, great confusion in regard to the possible specific micro-organism of this disease.

(*b*) **Acute Fatty Degeneration of the New-born** (*Buhl's Disease*).—This disease may be similar to Winckel's in nature. It was first described by Hecker and Buhl as an infectious disease of the new-born, characterized by cyanosis, jaundice, and copious visceral hemorrhages. The chief *pathologic change* is an acute fatty degeneration of the internal organs.

(*c*) **Syphilis Hæmorrhagica Neonatorum.**—Either at birth or soon thereafter bleedings take place into the skin (ecchymoses) and from the mucous surfaces and the navel. Jaundice may be associated. The viscera are found upon post-mortem examination to be the seat of syphilitic lesions.

(*d*) **Morbus Maculosus Neonatorum.**—Hemorrhage from the gastro-intestinal mucosa of the new-born (melæna neonatorum) occurs, and may be due to intracranial lesions during birth; it may also take place independently of the latter. Preuschen has collected the reports of 37 cases, in 5 of which the brain was examined, and all of these showed cerebral hemorrhages. The latter may occur in spontaneous births and give rise to melæna neonatorum. Gärtner believes the disease to be an infectious one, and claims that in 2 cases he was able to identify a bacillus for which the navel is believed to be the entrance-point. The blood may also come from the mouth, nose, navel, etc. Townsend found morbus maculosus neonatorum in 45 cases in 6700 deliveries, and in most of these instances the bleeding was general. The hemorrhage usually sets in during the first week, rarely later, and the duration of the disease is between one and seven days, the mortality being a little over 50 per cent. Vomiting of the blood which the child has drawn from the breast must not be confounded with true melena. The *treatment* is by gallic acid and ergotin, the latter hypodermically. Gelatin subcutaneously has apparently saved life.[2] Stimulants may also be required, and warmth to the extremities if the peripheral circulation be sluggish.

[1] *Archives of Pediatrics*, March, 1899.
[2] *Münch. med. Wochen.*, Sept. 2, 1902.

PART IV.

DISEASES OF THE BLOOD AND THE DUCTLESS GLANDS.

ANEMIA.

Definition.—A pathologic condition, characterized either by a diminution in the quantity of blood or by a deficiency in one or more of its constituents. Anemias may be subdivided into—I. Primary or Essential (chlorotic and pernicious); II. Secondary (symptomatic); III. Leukemia (myeloid and lymphoid).

Pathology.—Anemia, in its different forms, is characteristic of diseases of the blood or of the blood-making organs. It may be manifest, on examination, as a diminution of the total quantity or body of the blood (*oligemia*); of the number of red corpuscles (*oligocythemia*); of the hemoglobin (*oligochromemia*); and of other constituents, as fluid (*anhydremia*). The diminution of hemoglobin gives rise to the most obvious sign of anemia or impoverished blood—namely, the pallor of the cutaneous surface—but it is important to point out here that the quantity of hemoglobin in the blood is not necessarily proportionate to the number of red corpuscles. Thus the percentage of hemoglobin contained by the red corpuscles may vary in disease, so that a reduction in its amount does not necessarily involve a corresponding decrease in the number of red corpuscles. Conversely, a diminution in the number of the latter may not be accompanied by a proportionate diminution in the amount of hemoglobin, the corpuscular richness in coloring-matter being quite normal. As a matter of fact it frequently happens that oligochromemia is associated with a certain degree of oligocythemia, and *vice versa*, though where they coexist the degrees of reduction may neither be relatively nor proportionately equal.

Anemia can be positively ascertained only by an adequate examination of the blood. It may be inferred from the presence of pallor, languor, dyspnea, palpitation, etc.; but it should be borne in mind that not every pale person has anemia, since pallor of the face may be hereditary, and, at the same time, perfectly consistent with good health, a normal number of corpuscles, and a normal percentage of hemoglobin. Conversely, a person with marked vascularity of the face, and a rosy complexion even, may have anemia.

The anemias embrace those conditions, also, in which there are changes in the shape of the red corpuscles (*poikilocytosis*), and in their size (*micro-*, *macro-*, or *megalocytosis*).

I. THE PRIMARY OR ESSENTIAL ANEMIAS.

Primary anemias constitute those forms in which, so far as our present knowledge of their etiology and pathology goes, no other tissues or organs than the blood and the blood-making organs are either at fault or are directly affected. Future investigations of the life-history of the blood may reveal the exact causation of what are now regarded as primary or essential anemias, and thus permit of a clearer discrimination and a more accurate classification.

CHLOROSIS.

(*Green Sickness.*)

Definition.—A blood-disease, occurring chiefly in adolescent females, dependent upon defective hemogenesis, and characterized principally by a deficiency of hemoglobin in the red corpuscles. Chlorosis is steadily diminishing in frequency of occurrence.

Pathology.—It is so seldom that death occurs in cases of chlorosis that autopsies of this disease have not been frequent enough to determine definitely the nature of the findings. There is no loss of fat in the body, but signs of physical degeneration and disorders of development are quite common, hypoplasia of the vascular system and of the genital organs seeming to be the most prominent. Incurable cases of chlorosis are nearly always characterized by anomalies of the blood-vessels and genitalia (Rokitansky). Virchow has also shown that congenital arrest of development of the aorta and larger arteries, as indicated by their small size, their soft and elastic walls, is quite constant in chlorotics. The uterus (especially) and adnexa manifest the hypoplasia, and yellowish spots and streaks of fatty degeneration are sometimes seen in the intima of the arteries. The cardiac muscle is softened, the whole heart is dilated, and the left ventricle is usually somewhat hypertrophied.

Etiology.—Chlorosis occurs most frequently in girls at or near puberty, and also may appear between that period and twenty or twenty-five years of age. It usually happens that the condition dates from a scanty menstruation, beginning late in the "teens," but it should be recollected that amenorrhea is not, as formerly supposed, a cause, being rather an effect of the underlying blood-disorder. Blondes are oftener affected than brunettes. In males the disease is rare.

The influence of *heredity* in the causation of chlorosis is undoubted in those cases described by Virchow, in which congenital hypoplasia of the blood-vessels and genitalia is found to exist. Other cases also bear the stamp of heredity, in that their mothers have been, and their sisters are, chlorotic. A *family tuberculous taint* may predispose to chlorosis (Jolly); it is probable, however, that constitutional predisposition implies merely delicacy of organization. Such *unhygienic conditions* as bad air, dimly lighted rooms, a lack of nutritious food and out-door exercise, a sedentary occupation, excessive tea- and coffee-drinking; bodily fatigue, as from stair-climbing and standing in constrained positions without intervals of rest—all these predispose to the disease. And yet girls living amid the most luxurious and favorable surroundings have had chlorosis. Sir Andrew Clarke believed that *copremia*—the absorption of the toxic

ptomains and leukomains from the colon in constipation—is often the cause of chlorosis, though physiologic chemists fail to find in the urine the evidences of intestinal putrefaction. Sometimes a previously existing simple constitutional anemia appears to be an underlying cause. I believe that occult gastro-intestinal bleeding due to gastric or duodenal ulcer may be a cause of chlorosis.

Sudden emotional excitement and prolonged mental overexertion operate as causative agencies. Shock from bad news, such as loss of relatives, homesickness, disappointment in love, rankling grievances, and perhaps ungratified sexual desires, may contribute to the "neuropathic" origin of chlorosis. A change of climate seems to operate as a cause, and is manifested especially in the case of girls emigrating from rural Ireland to enter domestic service here (Townsend). A *late chlorosis* has also been described, but its existence must be rare.

Symptoms.—A brief outline of the more frequent and prominent general manifestations of chlorosis—or "green sickness"—may be narrated at the outset. The gradual onset is usually marked by languor, indisposition to either physical or mental exertion, motor weakness, irritability or inertia of mind, and a more or less constant fatigue. *Palpitation of the heart* and *dyspnea* on slight exertion are much complained of in most cases; *headache* is also an early symptom, and may be accompanied by vertigo in some cases; and *dyspepsia* and *constipation* occur in 65 per cent. of cases (Townsend). Probably in one-half of all cases cessation of, or scanty and irregular, menses may form the burden of complaint. A slight fever is present in many instances.

Gastro-intestinal Symptoms.—The appetite is either poor or perverted and a capricious desire for such innutritious substances as chalk, slate-pencils, and even bits of earth (*pica*), or for sour, highly spiced, and unwholesome articles of food (*malacia*), is not uncommon. An abnormal craving for alkalies has been ascribed to an overacid stomach. Morning vomiting or regurgitation of food and eructations occur, in some cases pain after eating may be noticed, and dilatation of the stomach and high position of the diaphragm are found in many instances. The gastric contents show a hyperacidity in most cases. The tongue is pale, flabby, often dry, and the edges show indentations.

Constipation is usually present, though sometimes *diarrhea*, lasting for a day or two, may alternate, as after the ingestion of some unwholesome article that has been eaten to satisfy the perverted appetite.

General Appearance.—The subcutaneous fat is not only well retained, but in many cases is even increased, and the rotundity of the body and members preserved. The peculiar *greenish-yellow tint* of the complexion is, however, the most striking manifestation to the eye. It differs thus from the muddy pallor of cancerous anemia, from the lemon-yellow tint of pernicious anemia, from the saffron hue of jaundice, and from the blanched pallor after severe hemorrhages. The *scleræ* are often pearly- or bluish-white ("cerulean hue"), and, though this is considered by many the earliest positive indication of anemia, when the skin-tint is not characteristic, yet, according to Townsend's analysis of 87 cases of chlorosis, it is not the most constant. The *nails* showed pallor in 95 per cent. of the cases; the cheeks, tongue, and lips were paled in 89, 84, and 76 per cent. respectively, while the scleræ were pale in but

64 per cent. On exertion the cheeks and lips may become quite ruddy in cases of moderate anemia (*chlorosis rubra*).

Circulatory symptoms are breathlessness, palpitation, and the tendency to vertigo and syncope complained of in the majority of cases; other circulatory disturbances may occur. The *skin* and the extremities are frequently cold, owing to sluggish heart-action. The *pulse* is usually full and easily compressible, and, owing to its excitability, it may be accelerated for the time being by various external influences (see Fig. 34). Visible undulating pulsations of the carotid vessels are frequent, and a pulsation at the base of the heart and in the peripheral veins is also observed at times. Physical examination shows the *heart* to be slightly dilated. Systolic murmurs, soft and "whiffing" in character, are heard at the base, though in severe cases they may be heard at the apex of the heart also. *Systolic blowing murmurs* of hemic origin are not infrequently heard over the carotid arteries. More common and characteristic, however, is the *venous hum* or *bruit de diable*—the soft continuous murmur heard over the large cervical veins. Thrombosis of the larger veins or of a cranial sinus may occur, and is always ominous.

Of the *nervous manifestations* that are often present, neuralgias of the head, mental depression, hyperesthesia of the skin, particularly of the abdomen, gastralgic attacks, and hysteria, are most frequently met with. Tinnitus aurium and anemic amaurosis have been known to occur.

Edema of the ankles is found in perhaps one-third of the cases. The *urine* is generally pale, free in quantity, and its specific gravity is somewhat lowered; and according to recent studies there is a diminished

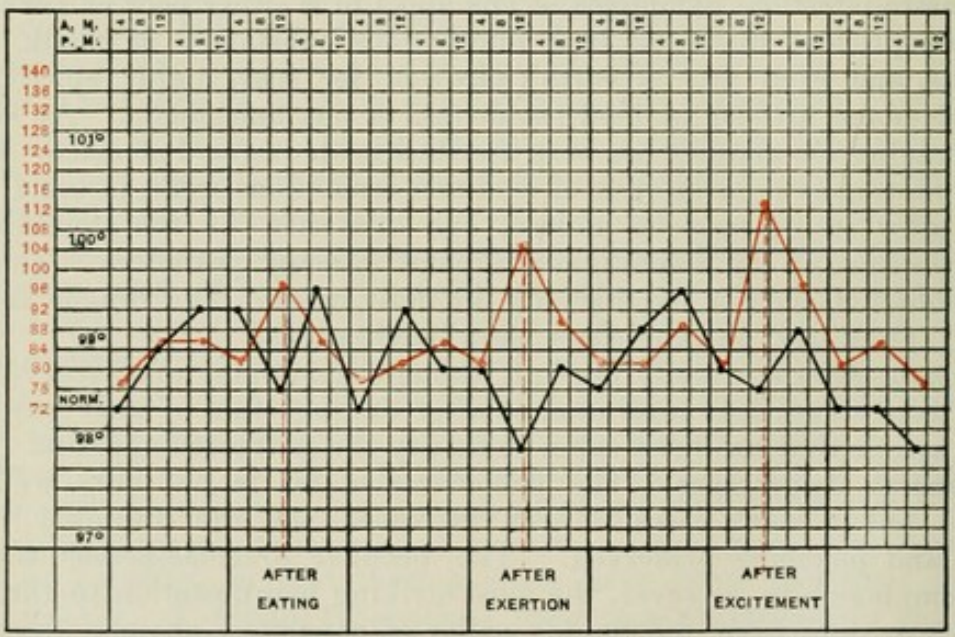


FIG. 34.—Pulse- and temperature-chart of a case of chlorosis, showing the effect exerted upon the pulse by eating, exertion, and excitement.

excretion of urea, despite the abnormal destruction of albuminoids. Movable kidney is often associated.

Blood-examination.—The blood flowing from a punctured finger-pulp or ear-lobule is pale, though seldom thin or hydremic, and the paleness is due to a qualitative rather than a quantitative change. There is a disproportionate reduction of the *hemoglobin* as compared with the

number of the red cells. The hemoglobin may range from 50 per cent. to as low as 16 or 17 per cent. in severe cases, the average quantity being about 38 or 40 per cent. On the other hand, the number of red corpuscles is not greatly reduced, and may even be normal. The moderate *oligocythemia* and *marked oligochromemia* are almost distinctive of chlorosis: these features, however, may be closely simulated by the chloroanemia of syphilis or early tuberculosis. Morawitz [1] calls attention to "masked chlorosis," in which Seiler found that the hemoglobin percentage was only 10 or 15 below normal. The average number of red corpuscles is from 3,700,000 to 4,100,000 per cubic millimeter of blood, but the count in very severe cases may be as low as 1,900,000. Approximately, the number of red corpuscles is from 70 to 85 per cent. of the normal, while the leukocytes are only slightly increased in number (8000 to 8500 per c.mm.). *Microscopically*, the red cells are seen to be paler than normal, and somewhat altered in size and shape. Some are distinctively larger than is usual (macrocytes), but the majority are slightly undersized (microcytes). Irregularity in shape (poikilocytosis) is seen in quite a number of the red cells in the severe cases, and an occasional normoblast (small nucleated red corpuscle) may be noted. There is usually a relative lymphocytosis, especially in severe cases. The eosinophiles are occasionally increased (Cabot). There is a marked increase in the amount of blood plasma (polyplasmia).

Diagnosis.—When the greenish pallor of the face is marked this can often be correctly made at a glance. The blood-examination must be made, however, to completely establish the diagnosis, even when distinctive symptoms are present, such as the shortness of breath, palpitation, weakness and languor, faintness, amenorrhea, capricious appetite, together with a well-nourished appearance of the body. The bluish-white scleræ and pallid nails are confirmatory when observed, and search should be made for the physical signs.

Differential Diagnosis.—The primary character of the anemia may be determined in doubtful cases, or in those in which *incipient tuberculosis* ("chloro-anemia"), or *syphilis*, or *Bright's disease* may be suspected, by exclusion. Here the physical examination of the chest, the history, and urinalysis should supplement the blood-examination. In the chloroanemia of chronic phthisis fever and progressive emaciation are also observed. *Organic disease of the heart* may be simulated by the breathlessness, palpitation, vertigo, and edema.

Prognosis.—This is always favorable, except in those cases in which congenital or developmental anomalies of the vascular system are associated. The discontinuance of proper treatment before a substantial cure is effected is often followed by a relapse, and even after apparent cure one or more recurrences may be witnessed before the age of thirty. The average duration of a case of chlorosis is from two to three months. In cases of very severe type, in which the dividing-line between this disease and pernicious anemia may not be marked clearly, the prognosis should be made with due reserve.

Treatment.—While the treatment of chlorosis by the administration of iron is wellnigh specific, the *hygienic measures* are also important, and particularly in order that relapses may be avoided.

Hygienic.—Pure air, wholesome food, and plenty of rest and sleep,

[1] *Münchener medizinische Wöchenschrift*, July 5, 1910.

with regular habits, are prime requisites. Sometimes a change of occupation, even temporary, where confinement may be replaced by an outdoor life and sunshine, as in the case of store-girls and mill-operatives, is of value in bringing about a rapid improvement. Patients in better circumstances may be sent to rural districts, the mountains, or sea-shore. In cases marked by much palpitation, dizziness, and dyspnea, rest in bed for a week or so is often imperative at the outset. As improvement goes on, however, light and then moderate exercise may be permitted, and the increasing appetite should be gratified by a generous, easily assimilable diet (milk, meat, eggs, fish, purées of green vegetables, stewed fruit). Fats and carbohydrates should generlly be avoided. Coffee, tea, and alcoholics do harm. Hot baths have been recommended.

Medicinal.—The one remedy, *par excellence*, on both rational and empirical grounds, is a good preparation of *iron*. This should be given methodically and persistently until the percentage of hemo-



FIG. 35.—Chart of a case of chlorosis, showing the improvement following the administration of iron. Convalescence almost complete; relapse. Black, red corpuscles; red, hemoglobin; blue, white corpuscles.

globin is 90, and then maintained there by continuing the administration of the iron for several weeks to prevent a recurrence (Fig. 35). Exactly how the iron acts in curing chlorosis has not been definitely proved, but its almost specific action is indubitable. Not all preparations of iron are equally well borne by the stomach, however, and several changes may be necessary during the course of a given case. Probably the best form for general use is the dried sulphate, usually given together with potassium carbonate in the well-known Blaud's pills—2

grains (0.129) of each to the pill. Starting with one pill thrice daily for a week or ten days, the daily dosage is increased until nine pills daily are administered in the third week, and continued for several weeks or as long as the case may require. It is very important, meanwhile, that the bowels should be kept soluble by the use of cascara sagrada, salines, and the like. A preliminary course of intestinal antiseptics for a week or so is strongly advised by some authorities, and is worthy of recommendation. Beta-naphtol, thymol, guaiacol, and salol are used for this purpose. The hematinic effect of the iron seems to be produced earlier and better when this plan is followed; and this fact seems to give corroborative evidence to Bunge's theory of the absorption of the iron in chlorosis—in a certain class of cases at least. Other iron preparations of value in this disease are the citrate, protoxalate, lactate, carbonate, the succinate, and the reduced iron. The albuminates of iron, so much vaunted for a time, are practically worthless. In severe cases Quincke uses at first a 5 per cent. solution of the ferric citrate hypodermically (♏viiss–℥iiss—0.5–10.0, daily). Bitter tonics and dilute hydrochloric acid are indicated in a certain number of cases in which indigestion is troublesome. The acid tincture of iron chlorid is sometimes used in such cases. Mild cases often yield to the simple use of remedies for the cure of gastro-intestinal derangement. Adjuvants in the treatment of chlorosis that may be of use are arsenic, manganese, mercuric chlorid, and arsenite of copper in minute does. Kottman thinks that rarely venesection is required in obstinate cases to whip up the torpid blood-producing apparatus.

PROGRESSIVE PERNICIOUS ANEMIA.

(*Idiopathic Anemia; Biermier's Anemia.*)

Definition.—A grave blood-disease characterized by a great destruction of red corpuscles, and a persistent tendency from a bad to a worse condition. It usually ends in death, and seldom exhibits causal lesions other than those of the blood or blood-making organs.

The term "idiopathic anemia" applied to this disease by Addison, whose first clear description of its clinical history has become classical, is applicable to a proportionately smaller number of cases to-day than during his time. This is owing to the later discovery (*post-mortem*) of adequate causes for the pernicious anemia that during life could not be found. Thus, while Biermier's anemia is usually considered a special disease-entity, for descriptive purposes it will be convenient to classify both groups under the title of *progressive pernicious anemia* in order to describe the invariable tendency of both. Under Diagnosis (*vide infra*), however, will be found differential clinical features.

Pathology.—The subcutaneous fat is rarely diminished, so that emaciation is exceptional. The skin is pale and of a lemon-yellow tint, and most of the tissues and organs are anemic, except the muscles, which are often decidedly red in color. The fat is usually pale and yellowish, and fatty degeneration is one of the most striking changes in this affection. The heart is usually large and flabby, and on section of the ventricular walls there is a marked pallor, as well as a friability, and a fatty change shown by the yellow tint. Micro-

scopically, the fibers or columns of heart-muscle are seen to be distinctly fatty. The heart-cavities contain little light-colored blood. Other organs showing the fatty degeneration (of the epithelium) are the liver, kidneys, gastric and intestinal walls, and the intima of many of the smaller blood-vessels (in patches). This general fatty change is probably due to the deficient oxygenation of the tissues and to the anemic blood-supply.

Owing to the above degenerative change in the vessel-walls, small extravasations of blood are found in different parts. Most frequently these punctiform hemorrhages are seen in the retina and on the principal serous membranes. Ecchymoses are also observed occasionally on the mucous membranes and on the skin. More or less general edema and dropsical accumulations in the serous cavities are not uncommon. The spleen and liver are seldom and only slightly enlarged. The lymph-glands are often somewhat swollen and intensely red in color, owing to the unusual number of red corpuscles.

In a series of 8 cases, Warthin [1] has found changes in the hemolymph glands consisting of "dilatation of the blood-sinuses and evidences of increased hemolysis, as shown by the increased number of phagocytes containing disintegrating red cells and blood pigment."

A marked and important pathologic feature of pernicious anemia is the presence of abundant deposits of iron-pigment, especially in the liver, but also in the spleen, kidneys, pancreas, and other organs. The fact that the abnormal quantity of iron in the liver is peculiarly distributed about the periphery and middle zone of the lobules is particularly noteworthy, and quite characteristic of pernicious anemia. The origin of this iron is doubtless the enormous destruction of red corpuscles, and that the pigment in the hepatic lobules is ferruginous may be determined by a micro-chemic test with ammonium sulphid, granules of black sulphid of iron being formed.

Of special interest are the lesions found in the bone-marrow on account of its hematopoietic function. This is virtually hypertrophied, and is in many cases deep-red instead of yellow, and more like the hemoblastic marrow of childhood (H. C. Wood). While formerly held to be causative, this change is now regarded as being secondary to the severe anemia. Cellular hyperplasia may be seen microscopically in the great number of large and small granular medullary cells, and also in the nucleated red cells.

An atrophied condition of the gastric and duodenal mucosa is noticed in some cases. The sympathetic ganglion cells may also show changes. More constant, however, is the sclerosis of the posterior columns and, to some extent, of the lateral columns of the spinal cord: this is especially marked, according to Burr, in the cervical swelling. Patveu examined 9 cases; in 4 he found hyaline degeneration of the vessels of the white substance, and in 5 small hemorrhages. These changes are probably due to a toxic agent.

Etiology.—There are three etiologic categories into which cases of pernicious anemia may be grouped: (1) those cases in which no discoverable cause for the hemolysis (blood-destruction) is ascertained, either during life or after death—*i. e.* the idiopathic variety of Addison; (2)

[1] *Amer. Jour. Med. Sciences,* October, 1902.

those in which an adequate cause is found *post-mortem* only; (3) those that are apparently traceable, *ante mortem*, to some primary causal condition acting directly or indirectly.

(1) As regards the *obscure* (*genuine*) *cases* of idiopathic anemia, the essential cause of the symptomatic condition is evidently an actively increased *hemolysis*. The blood-generation (hemogenesis) may be normal in power, or there may be a congenital or acquired underlying deficiency in hemogenic power. Grawitz and Stengel believe that the hemolysis originates in the gastro-intestinal capillaries and depends upon poisons generated or absorbed from that tract—an auto-intoxication. Von Jaksch holds that the similarity of pernicious anemia to Kala-azar suggests a protozoon infection. William Hunter[1] concludes that the disease is of infectious (streptococcal) nature, dependent primarily upon caries of the teeth. Goullard and Goodall[2] hold that a hemolytic toxin (not necessarily from the intestines) acts on the bone-marrow.

(2) *Apparently causeless* cases of a pernicious type of anemia may be found *post-mortem* to have been caused by (*a*) obscure malignant disease; (*b*) parasites, especially the *Anchylostoma duodenalis*, and rarely by the *Bothriocephalus*. Not infrequently, by a careful study of the anamnesis of a patient, aided by modern methods of examination, the cause of pernicious anemia may be detected during life. It is held that atrophy of ventricular and intestinal glands is an effect rather than the cause, as formerly believed of the anemia (Grawitz).

(3) Exhausting causes, operating directly or indirectly, may precede this affection, as severe or prolonged hemorrhages or diarrhea, fevers, mental shock, profound chlorosis, pregnancy, and parturition.

Predisposing Causes.—Unfavorable hygienic surroundings and insufficient nourishment, habitually kept up, may favor the development of the disease. Males are more frequently affected than females after the thirty-fifth year and it occurs mostly during middle life. Griffith has collected several cases occurring under twelve years of age. The disease is widely distributed, and it may behave endemically at times, as in Switzerland and Leipsic. Changes left in the tissues after syphilis may be the pathologic basis, and osteosarcoma may act similarly.

Symptoms.—Idiopathic pernicious anemia develops so slowly and insidiously that it is hardly ever possible to fix upon any precise date as the commencement of the disease. The transition from health to progressive pernicious anemia, particularly in persons previously feeble and pale, is usually too gradual to be demonstrable; though a rapid and acute onset is rare, it may occur in pregnant or puerperal women.

Pallor is soon noticed and gradually increases, or when there has been a previous pallor, this becomes more marked. *Shortness of breath* and *palpitation of the heart*, especially on exertion, are complained of; the patient is also easily fatigued, and becomes quite languid. Soreness of the tongue is among the initial symptoms in many cases (Schauman). Occasional nausea may come on early in those cases in which a previous gastro-intestinal disturbance has been noted, and headache, vertigo, tin-

[1] *Lancet*, January 27, 1900.
[2] *Jour. Path. and Bact.*, January, 1905.

nitus aurium, and anorexia ensue and grow progressively worse. General weakness increases, and occasional attacks of faintness and vomiting supervene. Meanwhile, the skin takes on a bloodless, waxy appearance, and soon the characteristic *lemon-yellow tint* appears. The mucous membranes are pale and colorless. *Prostration in bed* gradually becomes almost absolute as the feebleness and flabbiness of the tissue increase. *Malleolar edema* is sometimes noticeable, and ecchymoses—mucous and cutaneous—are seen in profound cases of anemia. Although the intellect is not impaired, except that mental exertion becomes irksome, the tone and manner of speech are feeble. As the debility becomes severe the mind wanders, and, to use Addison's words, the patient "falls into a prostrate and half-torpid state, and at length expires."

Emaciation is rare, the fat being preserved and sometimes increased in quantity. Pulsation in the large arteries is abnormally visible, and a diffuse, exaggerated cardiac impulse is felt. The *pulse* early in the case may be strong, and generally it is rapid (100–120), soft, and compressible, and as full and quick, often, as the water-hammer pulse of aortic regurgitation. Auscultation reveals the characteristic *hemic murmurs*, best heard at the base, and the *bruit de diable* in the veins of the neck. There may be visible pulsations in the latter.

Gastro-intestinal symptoms may be the most prominent signs where gastritis polyposa and gastritis atrophica are present. Diarrhea, dyspepsia, nausea, and vomiting are then seen throughout the course; otherwise, constipation, eructations, and simple anorexia are most common. Friedenwald has analyzed 58 cases with reference to the gastro-intestinal symptoms; he found anorexia in 38 cases, nausea in 27, vomiting in 19, indigestion in 33, pyorrhea in 20, and constipation in 27. Enteroptosis was present in 21 cases.

An ophthalmoscopic examination shows the cause of the *anemic amaurosis*, in the profound cases of anemia, to be one or more retinal hemorrhages. The whites of the eyes become pearly, the conjunctivæ pale. The liver and spleen are rarely palpable. The bones, and especially the sternum, are sometimes sensitive to pressure.

Respiratory Symptoms.—The breathing is accelerated, and the *anemic* dyspnea may become pronounced and stertorous, accompanied by a sense of thoracic oppression and a "hunger for air." Near the end pleural serous effusions and pulmonary edema may appear.

The *urine* is of low specific gravity, and, on account of its pigmentation with pathologic urobilin, dark in color. The urobilin is detected both by chemic and spectroscopic examination. In the former the addition of a few drops of an alcoholic solution of zinc chloride to the urine gives a green fluorescence. The presence of *indican* points to intestinal putrefaction. Albumin and glucose are absent, but uric acid and urea are both increased in amount, the former occasionally and the latter usually. *Fever* of a moderate degree is commonly, though not invariably, present, the evening temperature sometimes reaching 102° F. (38.8° C.). Previous to death the temperature may be subnormal.

Nervous Symptoms.—Paresthesia, spastic paralysis of the limbs, and a loss of control of the sphincters indicate the paralytic tendency of those cases in which sclerosis of the cord occurs. Tabetic symptoms are sometimes marked.

Blood-examination.—The blood is usually pale, though sometimes dark and watery, and the oligocythemia is distinctive of pernicious anemia. The number of red corpuscles may be reduced to less than 200,000 per c.mm., and is seldom more than 1,000,000. The percentage of hemoglobin may be approximately proportionate to the number of red corpuscles in the earlier stages, but as the disease progresses the index rises, so that the individual corpuscles are rich in hemoglobin. In other words, although there is a reduction in the total amount of hemoglobin, it is usually not so great as the reduction in the number of erythrocytes; therefore, the color index is nearly always relatively higher than that of the red globules (see Fig. 36), a condition in marked contrast with chlorosis. Macrocytes (which cause the relatively higher percentage of hemoglobin), microcytes, poikilocytes, and polychromatophilia are constantly present, and the former abundant. The presence of nucleated red corpuscles is also a striking characteristic of pernicious anemia. When normal in size they are known as *normoblasts;* when very large, as *megaloblasts.* In the former, according to Ehrlich, the eccentrically placed nuclei stain deeply; in the latter the large nuclei stain faintly. The former are typical of those nucleated red globules found in the hematopoietic organ of adults; the latter of those found in the blood-development of embryonic life. Megaloblasts may be found in non-idiopathic anemias. There are other forms of degeneration of the red

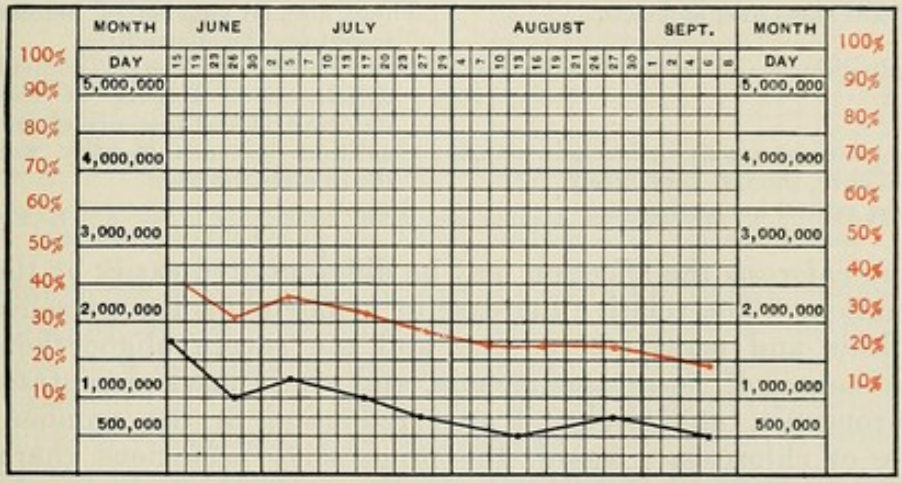


FIG. 36.—Blood-chart of a case of progressive pernicious anemia. Black, red corpuscles; red, hemoglobin.

cells, and Grawitz regards the microcytes of importance as showing erythrocytic degeneration. There is usually a relative increase in the small lymphocytes at the expense of the polynuclear cells; and, according to Cabot, there was always a marked leukopena in a series of 110 cases.[1] Myelocytes are almost constantly present, though usually in low percentage. The average is about 2 per cent. The blood-plates are generally fewer than normal. The blood-plasma is markedly decreased (Adami). Cabot[2] found that a blood examination made for the first time during the period of remission may resemble that in chlorosis, and in 9 of 14 cases ring-like bodies were seen in the red cells. There is a decrease in rouleaux-forming power, due to some change in the corpuscles.

[1] Cabot, *Medical News*, May 5, 1900. [2] *Amer. Jour. Med. Sciences*, Aug., 1900.

Diagnosis and Differential Diagnosis.—The clinical characteristics of the affection, particularly their steady progression with remissions, are quite as important as microscopic study of the blood. An important blood feature of the disease is a high color index. The possibility of hidden carcinoma, gastric atrophy, the anchylostoma or other parasite, and incipient tuberculosis should be borne in mind also. *Intestinal parasites* are recognized from the microscopic examination of the feces after a brisk purge when their eggs or the parasites themselves may be found. *Atrophic gastritis* may be discriminated by examining the viscus and gastric juice by modern methods. The following table will permit the elimination of obscure gastric carcinoma *as a rule:*

Progressive Pernicious Anemia.	Obscure Gastric Carcinoma.
The blood shows characteristic changes, and the red corpuscle count falls to or below 1,000,000 per c.mm.	Blood shows characteristics of secondary anemia, and the count does not fall to 1,000,000, as a rule.
Color index relatively high.	Color index low.
Leukopenia and relative lymphocytosis common.	There may be leukocytosis or a relative increase in the polynuclear cells.
Found earlier in life.	Occurs after middle life.
Gastric symptoms not so prominent.	Gastric symptoms more suggestive.
Lemon-tinted skin common.	Skin of a pale, muddy color, or only slightly jaundiced (saffron-yellow).
Adipose tissue fairly well preserved.	Progressive emaciation.
No glandular enlargements palpable.	Supraclavicular or inguinal glands may be palpable.
No physical signs over stomach.	There may be an area of increased resistance over the stomach.
Free hydrochloric and lactic acids usually absent.	Examination of gastric contents shows deficiency or absence of free hydrochloric acid and presence of lactic acid.
Some improvement may be brought about—even cure, though very rarely.	Condition becomes steadily worse until death ends the case.
May show retinal hemorrhages.	Absent.

From *chlorosis* the affection may be differentiated easily by the blood-examination. The relative increase in hemoglobin, the presence of gigantoblasts and many macrocytes, and the severe oligocythemia are pathognomonic of pernicious anemia, and are in marked contrast to the oligochromemia, and slight, if any, reduction in the number of red globules of chlorosis. Again, the progressive pernicious character of the former and the tendency to hemorrhage should be remembered, as well as the contrasting factors of age and sex in the two affections. Talley[1] states that anemia secondary to portal cirrhosis without hemorrhage occasionally resembles progressive pernicious anemia. *Tabes dorsalis* may be simulated, but the blood examination will show characteristic indications of pernicious anemia.

Prognosis.—The disease, as a rule, terminates fatally. The course of pernicious anemia is usually slow and gradual, and may be interrupted by improvement or apparent recovery. Recurrences, however, invariably occur. Idiopathic anemia is therefore almost hopeless, although a few apparently substantial recoveries have been reported. The duration of the disease is seldom more than a year, and may not be more than two or three months. The nucleated red corpuscles usually become much

[1] *Jour. of the Amer. Med. Assoc.*, October 3, 1908.

more numerous shortly before death (Billings). Death may be caused either by syncope, cerebral hemorrhage, or by slow asthenia.

Treatment.—**Hygienic measures** must be regarded as of signal importance, and rest in bed, together with light nutritious food given at short regular intervals, is indicated first of all. Klemperer advises a fatty diet—one liter of cream and 200 grams of butter per diem. Salt-water baths and gentle and systematic massage are useful adjuvants. Fresh, open air is advisable when it can be taken.

The value of *arsenic* in this disease is, I think, analogous to that of iron in chlorosis. The best action of the drug will be obtained by the administration of gradually ascending doses of Fowler's solution. Beginning with four or five drops of the former, three times daily during the first week, and thereafter adding one drop to the dose every day or two up to the point of tolerance, as much as twenty or thirty drops, well diluted,

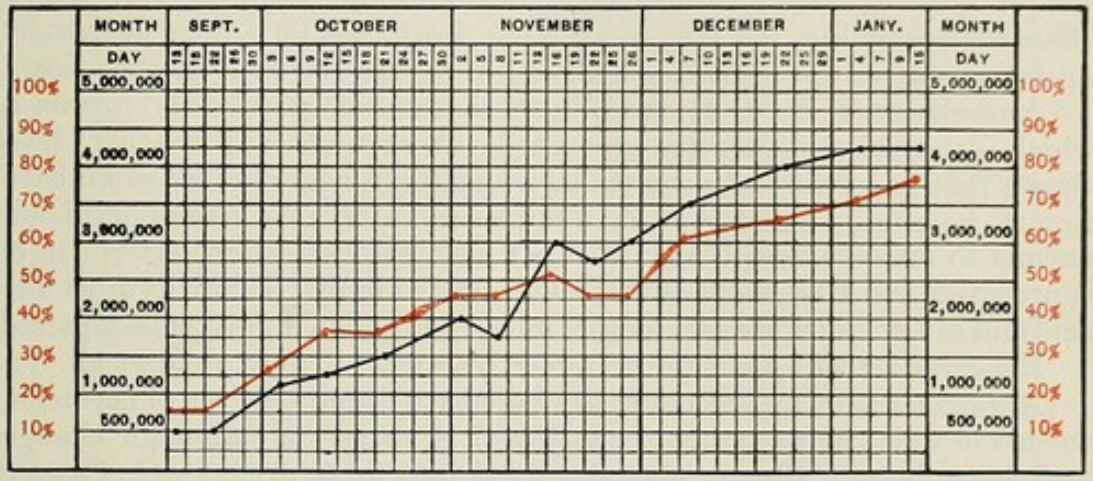


FIG. 37.—Chart of a case of progressive pernicious anemia, showing the improvement following the administration of arsenic. Black, red corpuscles; red, hemoglobin.

may be taken (see Fig. 37). Evidences of gastro-intestinal irritation should be watched for, and the arsenic discontinued temporarily should they appear. Sometimes it is advisable to use the remedy hypodermically. Arsenous acid is given in pill form, commencing with $\frac{1}{30}$ or $\frac{1}{20}$ gr. (0.0021–0.0032). Bramwell is of the opinion that salvarsan is a more effective remedy than arsenic given by the mouth. Dawes[1] has treated 14 cases of undoubted pernicious anemia with sodium cacodylate, administered hypodermically, with a remarkable degree of success. Atoxyl may be used in the same manner.

The introduction by Fraser of Edinburgh of bone-marrow in the treatment of pernicious anemia has been followed by various results: some cases have been reported in Great Britain and in the United States in which it has seemed to do good, while in others it was found to be useless. While the glycerin extract is the preparation generally used, it is not so reliable as the raw red bone-marrow, or that freshly prepared each day by mixing with it an equal quantity of glycerin; an ounce or two may be administered daily. Vetlesen has used glycerin—half an ounce with the juice of half a lemon, thrice daily—with success. Brieger advises pancreatin with a view to reducing the antitrypsin content of the blood-serum. Hunter suggests the use of antistreptococcus serum coupled

[1] *Monthly Cyclopedia and Medical Bulletin*, June, 1911, p. 321.

with the antiseptic care of the mouth and gastro-intestinal antisepsis. Iron is unnecessary, as there are enormous quantities of it in the liver (Gulland).

Near the end of the disease the danger often greatly increases, owing to the marked reduction in the quantity of the blood (*oligemia*). This may be combated by the injection of warm water or a weak saline solution into the colon, and also into the subcutaneous tissue. Both the former procedure and gastric lavage are of value in ameliorating the gastro-intestinal disturbance from fermentation. *Intestinal antiseptics* (salol and beta-naphtol) should be given by the mouth. Grawitz [1] outlines a causal treatment; he eliminates animal albumin from the diet, administering fruit juices to supply the lack of hydrochloric acid. A daily lavage with a 1 or 2 per cent. solution of sodium chlorid and a daily colon irrigation are to be given. Bovaird [2] reports favorable results from transfusion of blood in cases in which the hemoglobin falls below 20 per cent.; it offers the possibility, not only of averting death, but for a time, at least, of initiating one of the periods of quiescence so characteristic of the disease.

Anthelmintics must be used in those cases of pernicious anemia in which intestinal parasites are associated. Morgenroth and Reicher [3] have shown that experimental anemias in animals are benefited by administration of cholesterin, which they have employed in the treatment of pernicious anemia to prevent hemolysis. A 3 per cent. solution of cholesterin in oil is prepared and 100 grams of this administered daily. Dilute hydrochloric acid and bitter tonics are serviceable in cases in which digestion is impaired.

Recurrences will yield to the same treatment, if they yield at all.

Aplastic Anemia.—Senator [4] has called attention to a form of pernicious anemia to which he gave the term *aplastic*. A variety of pathologic changes have been found. Blumer classifies these into three groups: (1) Cases in which the lesions are those of progressive pernicious anemia; (2) those cases in which the bone-marrow shows primary aplasia; and (3) cases in which there is a hyperplasia of the mononuclear elements of the bone-marrow.

The *symptoms* and *physical signs* are those of pernicious anemia, but the cases run a more rapid course—six to nine months. The blood findings are unlike those of pernicious anemia, nucleated red blood-cells being absent and the number of leukocytes greatly diminished. A differential count of the leukocytes shows the small mononuclear forms to be relatively increased 70 to 90 per cent., while the eosinophiles are few.

II. THE SECONDARY ANEMIAS.

The secondary anemias are symptomatic of abnormal processes or of existing disease, whether acute or chronic, and their causes are numerous and various. Several possible causes may exist in a given case of symptomatic anemia, and it may be quite difficult to discover which of these is the active factor in the condition. In certain secondary anemias, also, the associated impairment of the blood-making organs is so evident that the anemia may assume almost a primary importance.

The Blood.—There is *oligocythemia*, usually of a moderate degree, about 3,000,000 red corpuscles per cubic millimeter being noted, although

[1] *New York Medical Journal*, October 15, 1910, p. 777.
[2] *Medical Record*, February 11, 1911.
[3] *Berlin. Klin. Woch.*, Oct. 12, 1908. [4] *Zeit. f. klin. Med.*, vol. liv., Nos. 1 and 2.

in cases following hemorrhage the reduction may be as great for a time as in pernicious anemia. There is also a relative decrease in the amount of *hemoglobin*, and sometimes the percentage may be relatively lower even than is compatible with the decrease of the red corpuscles. Early evidences of secondary anemia are alterations in the viscosity (stickiness) of the red cells and failure to form rouleaux and an unequal distribution of the hemoglobin, certain cells being overcharged while others are inadequately supplied. Next in the process of degeneration of the red cell is irregularity in size and shape (microcytes, macrocytes, poikilocytes), and third is abnormal staining reactions (polychromatophilia, punctate basophilia). Lastly there appears abnormally large nucleated red cells (megaloblasts). Normoblasts are also present in severe cases. There is a relative, and often an absolute, increase in the number of *leukocytes*.

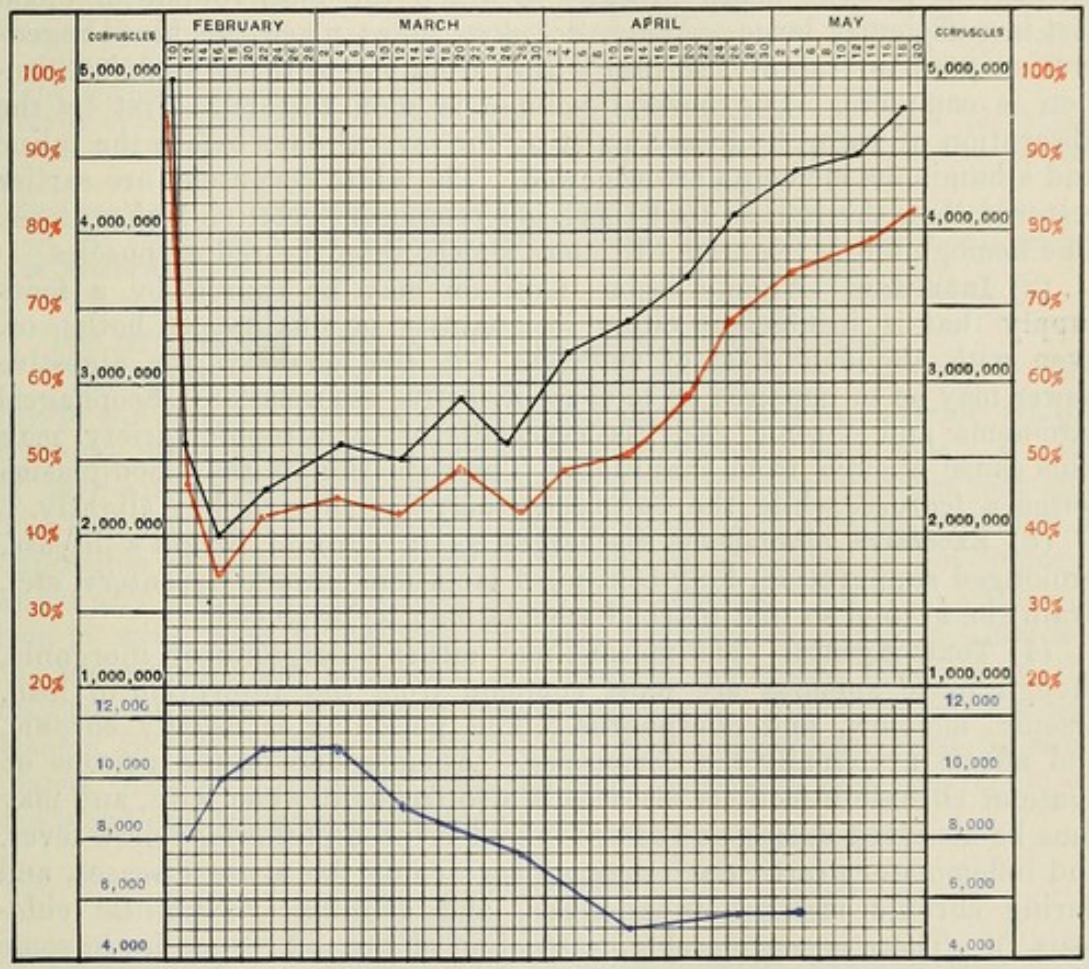


FIG. 38.—Blood-chart of a case of symptomatic anemia. Black, red corpuscles; red, hemoglobin; blue, white corpuscles.

The most important etiologic groups of secondary anemias are as follows: (1) **Hemorrhage.**—Hemorrhages occur under a great variety of circumstances, and if copious, result in an acute secondary anemia. Thus there may be the rupture of an aneurysm, menorrhagia, postpartum hemorrhage, hemoptysis, gastrorrhagia, enterorrhagia, etc., all of which produce the same general effect upon the system. Repeated small hemorrhages may finally produce the same result as a single large one, and spontaneous hemorrhages or epistaxis, such as occur in persons of a hemorrhagic diathesis (hemophilia) or in purpura and scurvy, may

cause profound secondary anemia. Females are most tolerant of losses of blood, but infants of both sexes bear depletion very badly. The total mass of blood may be much diminished (oligemia), and the sudden loss of a great volume of blood may prove fatal in a few moments; but it is often surprising how recovery can take place, and often does, after the rapid loss of several pounds of blood—*e. g.* in hemoptysis, hematemesis, or menorrhagia. Sometimes the source of bleeding is obscure, as in cases of intestinal parasites, hepatic cirrhosis, or duodenal ulcer; or it may be intentionally kept *sub rosa* by females having uterine disorder or bleeding hemorrhoids. The quick blanching of the countenance, the weakness, the coldness of the skin, faintness, dimness of vision, tinnitus aurium, sighing respiration, and feeble, rapid pulse are characteristic symptoms of *acute anemia*. *Unconsciousness* and *epileptiform convulsions* precede death in cases in which the total volume of blood lost is sufficiently large. When *recovery* takes place the blood-regeneration goes on rapidly, so that within from one to three weeks restitution is complete. The normal volume is soon restored—first by the absorption of water, hydremia existing for several days before the saline and albuminous elements are renewed. The white corpuscles are earlier restored than the red, so that there is a temporary relative leukocytosis. The hemoglobin is restored still more slowly than the red corpuscles.

(2) **Inanition.**—Anemia from inanition may be caused by a food-supply that is insufficient either in quantity or quality, or both; or, even with abundant food of sufficient nutritive qualities the digestive power may be so impaired as to cause defective assimilation. Esophageal carcinoma and chronic gastritis, especially of the atrophic variety, may thus cause anemia from inanition. The reduction of the blood-plasma forms a feature, while the corpuscles may be affected but slightly.

(3) **Excessive albuminous discharges,** as in chronic Bright's disease, prolonged suppuration, long-continued lactation, chronic dysentery, etc., drain the system so that marked anemia may be produced.

(4) **Toxic Agents.**—The poisons may either be organic or inorganic, though toxic anemias are most common from the absorption of lead, arsenic, mercury, and phosphorus. The poisoning is usually chronic, and affects principally the corpuscles. Anemia due to the poisons of acute or chronic infectious diseases is also frequently met with, and may thus be observed during and after typhoid fever, diphtheria, yellow fever, and inflammatory (articular) rheumatism among the acute diseases, and during chronic malaria, tuberculosis, and syphilis ("syphilitic chlorosis"). There is considerable destruction of the red corpuscles in some of these diseases, either directly or indirectly, and the greater the pyrexia the greater the action upon the blood or blood-making organs.

Symptoms.—The *common indications* of secondary anemia are the pallor of the face and mucosæ, muscular and mental weakness, loss of nerve-function, neuralgias, coolness of the skin, dyspnea on exertion, cardiac palpitation, impaired appetite and digestion, and a weak pulse. The physical signs are those of the primary or essential anemias.

Diagnosis.—Here may be advantageously contrasted the distinguishing features of symptomatic and essential anemias, respectively:

Symptomatic or Secondary Anemia.	Idiopathic or Essential Anemia.
A symptomatic blood-condition secondary to a detectable disease elsewhere.	A primary disease of the blood and blood-making organs.
Occurs at any age.	Occurs principally during adolescence and early middle life.
Previous or associated history of traumatic or spontaneous hemorrhage, chronic suppuration, prolonged lactation, chronic Bright's disease, carcinoma, chronic lead-poisoning, chronic malaria, heart, liver, or gastric trouble.	Previous history negative in its bearings upon the disease.
Blood-changes not so marked and more variable; steadily progressive in malignant disease.	Distinctive blood-characteristics, and often profound changes, both as to blood-cells and hemoglobin.
Moderate reduction in both, merely the relative proportion being maintained.	Marked reduction in both the hemoglobin percentage and in the number of red corpuscles, but the proportionate ratio is lost.
General symptoms and signs usually subordinate in manifestation to those of the primary disease or lesion.	General symptoms and signs also more characteristic of the respective form of anemia in the case.
Gravity of anemia depends on that of the primary disease.	Gravity depends on type of blood-changes and progressiveness of disease.
Often responds to treatment, depending on the cause; in a few instances, as in hemorrhage, it is short in duration.	One variety (chlorotic) quite curable, the other (progressive pernicious) relapsing, and finally fatal.

The **prognosis** depends upon the cause of the anemia.

Treatment.—Symptomatic anemia is amenable to treatment according to the cause. The traumatic acute variety does well under simple hygienic measures after the urgent indications have been met. Plenty of pure air, wholesome food, and graduated rest and exercise may suffice, and drugs not be needed. Cases in which it is difficult or wellnigh impossible to remove the cause of the anemia of course do not improve under any treatment, as a rule, for obvious reasons. Nutritious aliment, iron in some form, a judicious hygienic regimen calculated to increase the assimilation, and stomachic and general tonics are required in the majority of cases. In severe forms, hypodermic injections of iron and arsenic, in combination, act favorably. Toxic substances must be eliminated, their re-introduction into the body prevented, and the repair of the blood and tissue actively promoted.

LEUKEMIA.

(*Leukocythemia.*)

Definition.—A blood-disease, usually chronic, characterized by a peculiarly marked and persistent increase in the number of leukocytes, associated with lesions occurring either respectively or unitedly in the bone-marrow and lymphatic glands.

Pathology.—Bodily emaciation and pallor are pronounced, and edema, with dropsical effusions in the serous cavities, is by no means uncommon. The cardiac chambers and principal veins are distended with large blood-clots of a greenish-yellow or, in extreme cases, yellowish-white, purulent appearance. Subserous ecchymoses of the pericardium and endocardium are frequent, and the myocardium is often found to have undergone a moderate degree of fatty degeneration. Various

abnormal substances have been found in leukemic blood—leucin, tyrosin, acetic, formic, and lactic acids, and certain albuminous substances (deutero-albumose and nucleo-albumin)—resulting probably from the destruction of blood-corpuscles. The alkalinity of the blood is diminished. The minute, octahedral (Charcot's) crystals are found most abundantly in settled leukemic blood, and have also been detected in the spleen, bone-marrow, and liver, as well as in other affections.

Although the bone-marrow or the lymph-glands may alone show the pronounced pathologic changes of leukemia, it is usual to find both more or less affected. It is customary to speak of two principal groups: (1) *myeloid* leukemia, the more frequent variety; and (2) *lymphoid* leukemia.

There is nearly always some splenic enlargement, and in many cases the enlargement is considerable. Leukemic spleens sometimes weigh as much as from two to eighteen pounds, and their lengths may vary from six to twelve inches. The enlargement is generally uniform, and the notches upon the anterior border may be much exaggerated. White patches of perisplenitis and a thickened capsule adhering to the surrounding organs and the abdominal wall may also be noticed. The consistence of the spleen is firm and resistant to the knife, though in the earlier stages it may be quite soft and pulpy. The cut surface is either of a uniformly brown color or mottled by the presence of grayish- or yellow-white circumscribed lymphoid tumors, or by deep-red or brownish-yellow hemorrhagic infarcts. The Malpighian bodies may or may not be visible. The blood-vessels at the hilum are enlarged. *Microscopic examination* shows hyperplasia of the organ. The cells of the pulp sometimes show granular and fatty degeneration, and in advanced cases the trabeculæ may be thickened by connective tissue. Ewing believes that the splenic enlargement is due to the mechanical sifting of the red and white cells from the circulation with subsequent inflammatory changes.

In the majority of cases the bone-marrow is the primary seat of the disease in the myeloid variety. The medullary substance, instead of being fatty, is rich in lymphoid and blood-cells in various stages of development, and is either reddish-brown or greenish-yellow in color. Neuman regarded the marrow change as an essential lesion of leukemia, and called the former transformation "lymph-adenoid" and the latter "pyoid." The pus-like marrow and the dark red may exist side by side, although the former is more common.

A fine reticulum may be seen between the cells, especially in the dark-red variety, and small hemorrhagic infarcts may also be noted occasionally. *Microscopically*, the medulla contains an abundance of lymphoid cells and nucleated red corpuscles. Eosinophilic, mononuclear, and polynuclear leukocytes are also present, the first-named being quite numerous, as are also certain myelo-plaques and cells showing karyo-kinetic figures. The *lymphatic glands* are more or less enlarged in the myeloid form of leukemia.

In the **lymphoid variety**, especially when acute, an early and marked hyperplasia of all the glands takes place, and may form distinct, soft, and movable tumors, their color being a reddish-gray.

The histologic examination shows an increase in the cellular elements. A similar hyperplasia occurs in those glandular tissues that are allied to the lymphatic glands, such as the tonsils, lymph-follicles, the tongue, mouth and pharynx, thymus gland, the solitary and Peyer's agminated intestinal glands, and the Malpighian bodies in the spleen.

Proliferation of the bone-marrow cells, which are carried to other tissues and there multiply, is the essence of the disease. Available space for the production of red cells is encroached upon by lymphocytic proliferation, hence the anemia.

The *liver* may be greatly enlarged; indeed, some of the instances of greatest enlargement of this organ have been those due to leukemia, the weight being as much as fourteen pounds. The enlargement is uniform and due to a diffuse leukemic infiltration. The capillaries and interlobular tissue are distended with leukocytes, and disseminated whitish or grayish nodules, usually quite small, consisting of lymphoid cells undergoing indirect division of their nuclei, are frequently found. Sometimes these leukemic nodules appear as definite growths, with an adenoid reticulum between the cells (lymph-adenomata).

Similar changes are observed in the *kidneys*, enlargement, paleness, and diffuse and circumscribed leukemic infiltration of the capillaries and interlobular tissue all being noted. Leukemic nodules may also be found in other parts of the body, such as the retina, brain, serous membranes, lungs, testicles, and skin. Karyokinetic figures are numerous in the cells accompanying these leukemic growths.

Etiology.—The primary cause of leukemia is unknown; that it directly affects the blood-forming organs, however, is most probable, though with differences of selection and co-ordination and with different degrees of intensity. The combination of lesions in the spleen, lymph-glands, and bone-marrow, along with the histologic similarity of the leukemic growths to the infectious granulomata, and the clinical history of cases of acute leukemia, would seem to point strongly to the *microbic origin* of the disease. Moreover, various cocci and bacilli have been found, but not one of them has been definitely proved to be the specific cause of the disease. *Auto-intoxication* by toxic albuminoids from the digestive tract is believed by Vehsemeyer,[1] who analyzed 600 cases, to be the important point of departure of the disease. It is likely that the direct cause of the leukocythemia is a simple increase of the cytogenic function of one or more of the hematopoietic organs. Kottnitz held leukocythemia to be a reactive condition following auto-intoxication with peptones, and consequently a leukolysis, the over-action of the hematopoietic organs leading to hypertrophy. Whether the reduction of the erythrocytes is due to diminished production or to increased destruction is not positively known, although the former factor is more probably operative.

The disease has often been preceded by an *injury* or *a blow* in the splenic region, but its direct traumatic origin is hypothetic only. *Intestinal ulceration* has been a frequent feature prior to leukemia, and undoubtedly affords a source of possible infection from the tract. *Stomatitis* also may furnish a means of entrance for the infectious agent. The causal relation of *pseudo-leukemia* and *true leukemia* is uncertain, although a few cases of the one have been observed to pass into the other.

In a considerable proportion of cases leukemic patients have had *malaria* of some form. Syphilis may be associated with the disease, but it is not probable that it acts in a causative manner.

Hereditary influences undoubtedly play a part; a "lymphogenous diathesis" may thus be transmitted, and several generations may be affected by the disease. *Adverse hygienic* and *social conditions* may also

[1] *International klin. Rundsch.*, Vienna, Nov. 25, 1894.

predispose to leukemia. It may also develop after *pregnancy*, or more commonly at the *climacteric*. Anxiety, worry, and mental depression have been mentioned as predisposing causes, with doubtful justification.

Leukemia occurs most frequently in males during the middle period of life, and is apt to attack young persons. It has occurred during infancy, and as late also as the seventieth year, but the average age ranges from twenty-five to forty-five years. Sometimes the previous condition was one of apparently perfect health.

Symptoms.—Acute leukemia, comparatively rare, usually occurs in an adolescent who may have enjoyed previous good health. Fussel and Taylor collected 56 cases from the literature. Hamman tabulated 111 cases, and in several affections of the mouth or throat were observed at the commencement. Its onset is sudden, and usually begins with prostration, hemorrhage of the mucous membranes, and high fever. Acute splenic tumor rapidly develops; the lymphatic glands may enlarge; and palpitation, dyspnea, and gastro-intestinal symptoms of a severe type appear. The skin becomes anemic, and edema of the feet is common. The blood shows a marked increase in the number of leukocytes, the ratio to the red corpuscles being 1 to 30 or 1 to 50, instead of the normal 1 to 350 or 1 to 600. In *acute lymphoid leukemia* the lymphocytes are very numerous. In *acute myeloid leukemia* there is an increase in the blood of a cell originating from the myelocytic cells of the bone-marrow, representing the ancestor of the myelocytes and granular leukocytes, which are numerous in the blood picture. The case grows progressively worse; hematemesis, cerebral or retinal hemorrhages, and petechiæ supervene perhaps, and the clinical features may then resemble an infectious disease with hemorrhagic and purpuric manifestations. Early in life the hemorrhages are less common and the increase in lymphocytes is apt to concern the small variety of cells.

In **chronic leukemia** the *onset* is generally slow and insidious and for many months the earlier symptoms may not differ from those of simple anemia. Languor, a deranged appetite, dizziness, noises in the ears, faintness, breathlessness on exertion, and palpitation may all appear. Sometimes, however, not even these symptoms are present, common as they are to most anemic cases, and the patient may first consult the physician, because of a swelling or distress in the left side of the abdomen—the *enlarged spleen*. Early manifestations may be *hemorrhagic* (epistaxis, hematemesis, enterorrhagia), with nausea, vomiting, and diarrhea; or *increasing pallor* of the countenance, yet at times a patient may appear to be plethoric; or troublesome priapism may appear. As the disease progresses the anemia becomes more marked, *edema* of the dependent portions of the body may appear, and *fever*, though slight at first (99.5° F.—37.5° C.), may gradually rise to 102° or 103° F. (39.4° C.), either remaining constant or alternating with periods of apyrexia.

The *pulse-rate* is increased; in quality it is soft and compressible, though sometimes full in volume. The *dyspnea* may be aggravated by the hydrothorax in advanced cases, or by the upward displacement of the diaphragm owing to the increasing splenic and hepatic enlargement. *Epistaxis* may become obstinate. Retinal hemorrhage is common, and there may be aggregations of leukocytes (leukemic growths). Hemorrhages from mucous membranes are common, and localized gangrene may occur, with the symptoms of infection. Hemic murmurs are quite constant.

Ulcerative processes in the bowels may give rise to severe *dysenteric*

diarrhea. *Ascites* is usually present in advanced cases on account of the splenic tumor, or owing to pressure upon the portal vein by enlarged glands. *Jaundice* is an occasional event. *Leukemic peritonitis* may occur from the presence of lymphomatous growths in the membrane.

Nervous symptoms, such as headache, vertigo, and syncopal attacks, recur as the anemia and prostration increase and the liability to hemorrhage becomes more frequent. Sudden coma and hemiplegia following upon the rupture of a cerebral vessel (apoplexy) may be the immediate cause of death. Minute brain-hemorrhages may account for deafness. Priapism may be troublesome. Peripheral paralysis of several cranial nerves, due to hemorrhages into their sheaths, has been reported.

Cutaneous ecchymoses are sometimes observed, and sometimes there is a troublesome pruritus. The *urine* contains an excess of uric acid, but albuminuria does not occur, except as a complication.

Along with the anemia and debility are the signs of splenic and lymphatic involvement, and rarely of the bone-marrow. The liver may also become enlarged.

Leading Symptoms in Detail.—*The Spleen.*—This organ is generally enlarged in all forms of leukemia, but especially in the spleno-medullary, the most frequent form. It is a prominent feature, both on account of its being the first subject of complaint, and because of the huge size it frequently attains. The enlargement is gradual, and there may be neither pain nor tenderness over it. The tumor may cause a visible projection below the ribs, and in marked cases great abdominal distention may be produced, pushing up the diaphragm and thoracic organs, and extending to the navel in the median line and to the pelvis below, in which case the cardiac pulsation is seen at the second or third interspace. The edge and notch or notches may be felt easily in such instances, while the surface is smooth and the consistence firm. A friction-fremitus is felt sometimes during respiratory movement. The tumor may vary in size, and after severe hemorrhage or diarrhea it may become swollen. Gastric distress after eating and obstructive constipation are usually complained of in cases of great splenic enlargement. Jaundice may also be present. Pulsation has been noted and a systolic murmur—"splenic souffle"—has been heard at times over the tumor. The percussion-note is dull over the tumor, and areas of movable dulness, due to fluid occupying the peritoneal cavity, are not infrequent. A wave of fluctuation may be detected over the abdomen. The liver is often enlarged.

Lymphatic Glands.—In the splenic-lymphatic variety, which is less common than the splenic-myelogenous, and in the still rarer purely lymphatic leukemia, the superficial lymph-glands may be both visibly and palpably enlarged, though not in bunches as in Hodgkin's disease. They are soft, resilient, and movable.

The Bones.—Purely myelogenous leukemia is very rare, and local bone-symptoms are scarcely ever manifested. There may be some tenderness on immediate percussion over the sternum or some of the long bones, and slight swelling, irregularity, or deformity of the ribs, the sternum, or other bones may result from leukemic hyperplasia.

The Blood.—It is by the blood-examination alone that the pathognomonic features of leukemia are determined. The blood is paler than normal, and sometimes has a brownish-red or chocolate color. Upon a microscopic examination of the blood in the myeloid form of the affection

the striking increase in the number of leukocytes is observed at once. The count shows usually from 85,000 to 500,000 white corpuscles per cubic millimeter, and the ratio of the white to the red cells may thus vary from 1 to 150 down to 1 to 10 or 1 to 5 in the average case, instead of the normal, 1 to 500 (see Fig. 40). In extreme cases the number of leukocytes may be equal to, or even slightly greater than, that of the erythrocytes, and such an instance has been recorded by Sörensen, in which the proportion of whites to reds was 3 to 2.

Stained specimens of the blood enable us to recognize the variety of leukemia (Fig. 39, Pl. V.). Thus, in the ordinary myeloid form the characteristic change is the presence of the abnormal *myelocytes*—large, mononuclear leukocytes with the protoplasm filled with fine neutrophilic granules. These may make up 25 per cent. of the white cells, whereas they do not occur in normal blood, and very rarely, and only

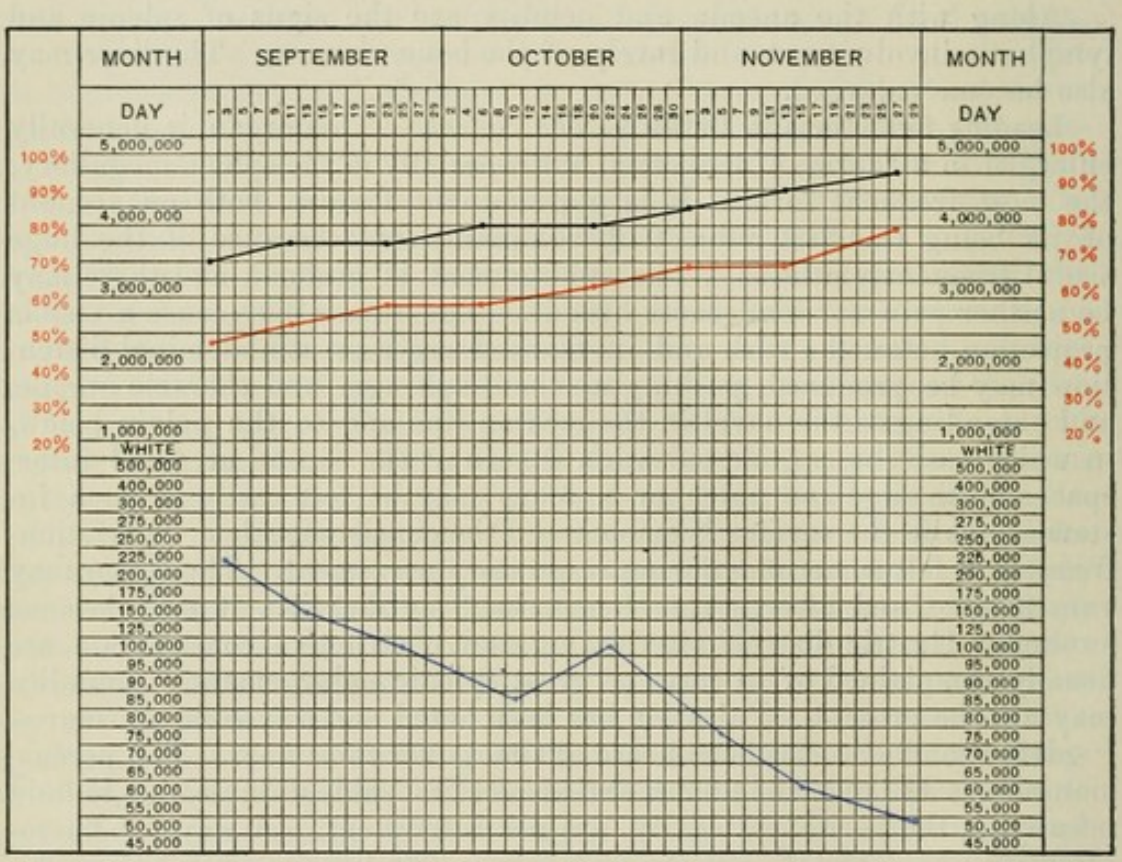


FIG. 40.—Blood-tracing of a case of leukemia. Black, red corpuscles; red, hemoglobin; blue, white corpuscles.

in small numbers, in leukocytosis. They probably correspond to the cells found in the bone-marrow, the large, oval, and eccentrically placed nuclei of both blood- and marrow-cells showing karyokinetic figures. The polymorpho-nuclear leukocytes may be normal in number, but usually they are relatively diminished to about 65 per cent. instead of 75 per cent., as in normal blood. The polymorpho-nuclear cells showing coarse basophilic granules are increased, and may equal in number the eosinophiles. When Ehrlich's triacid stain is used these cells appear as non-granular polynuclear bodies. The lymphocytes are also relatively less in number, making up but 1 or 2 per cent., instead of the normal 15–30 per cent. The bright, acid-stained eosinophiles, though absolutely increased, are not always relatively so. They possess but little diagnostic value, being common to many other conditions.

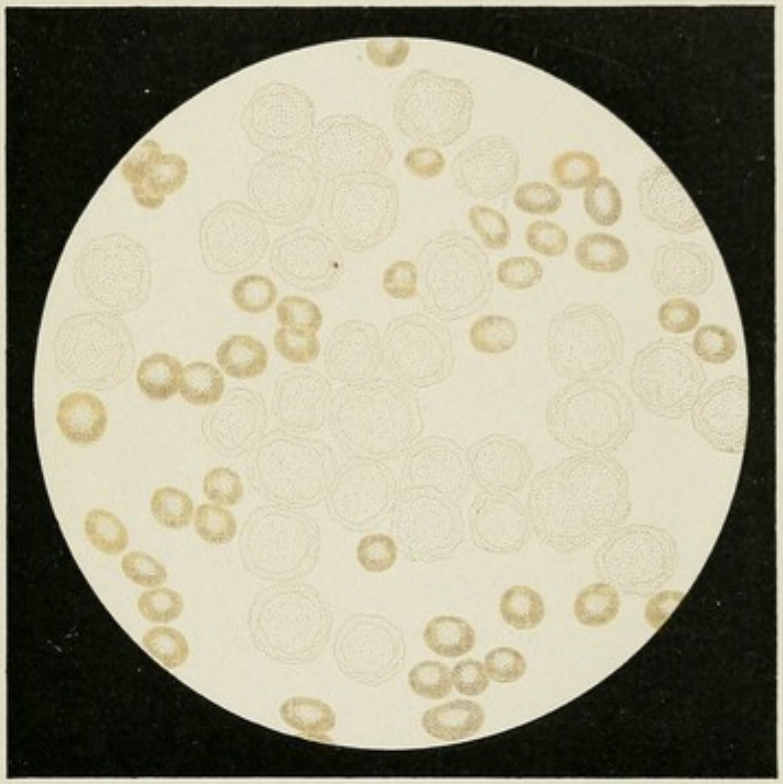


FIG. 39.--Fresh preparation from the blood of a case of leukemia (× 550); large mononuclear leukocytes of immature form.

[Grawitz.]

Moderate *oligocythemia* is noted in the later stages, the reduction being seldom lower than to 2,000,000 per c.mm. The percentage of hemoglobin may also be reduced relatively or in slightly greater proportion. Nucleated red corpuscles, chiefly normoblasts, are invariably found. Cells with large, pale nuclei are occasionally found, and cells with fragmented nuclei are common. Gigantoblasts may be present. Blood of the type of pernicious anemia may subsequently develop a true leukemia. In a majority of cases the blood-plates are considerably increased.

In *lymphatic leukemia*, which is rarer and more quickly fatal than the preceding variety, the *lymphocytes* are increased, all other leukocytes being relatively much diminished in number. Instead of the normal percentage (15 to 30 per cent.), the lymphocytes may number from 90 to 97 per cent. of all the leukocytes. The excess of leukocytes, however, is less than in the myeloid form. This increase affects the small forms in most cases. Cabot has shown that in some instances this increase affects the larger lymphocytes. Nucleated red corpuscles, chiefly normoblasts, are present in small numbers. Myelocytes are not numerous, but quite constant. The erythrocytes show changes in size, shape, and staining out of proportion to the degree of anemia present. Eosinophiles are relatively diminished. *Mixed forms* of leukemia are not at all uncommon, so that the proportions of the various types of normal and abnormal cells are quite variable.

The *blood-plates* may be quite abundant in many leukemic cases, and Charcot's octahedral crystals appear in specimens of the blood on standing. An unusually dense and thick fibrous network is also often found.

Complications.—Fatal hemorrhages may occur at any time, pleuritis, pneumonia, septico-pyemia, renal disease, severe diarrhea, toxemic jaundice, and edema may complicate leukemia and cause death.

Dock[1] has shown that chronic tuberculosis does not distinctly influence the course of leukemia. Acute miliary tuberculosis, however, may follow and also cause a reduction of the leukocytes.

Diagnosis.—This can be made accurately by the blood-examination alone, the distinguishing characteristics of the blood having been enumerated above, both as to the existence of leukemia and the differentiation of its several varieties. Stained specimens of the blood should be studied, since the excess of leukocytes alone is not proof of leukemia, and also because the disease may exist without an excess, owing either to previous medicinal treatment or to temporary improvement.

Differential Diagnosis.—Leukemia is differentiated from a marked *leukocytosis* by the fact that in the latter there is usually a more moderate increase in the number of leukocytes, affecting, as a rule, principally the polynuclear neutrophiles; in addition myelocytes are absent.

Hodgkin's disease may be simulated by the purely lymphatic leukemia on account of the enlarged glands; but in leukemia the lymph-glands are not found in such large bunches, and the blood-examination will show the characteristic changes of lymphatic leukemia if that disease be present. Simply a leukocytosis is present in pseudo-leukemia.

Malignant growths of the spleen and lymphatic glands, and also a malarial and passively congested spleen with anemia, may simulate leukemia. Here again the blood-examination will exclude leukemia.

Prognosis.—Many cases are mild in their progress; children, how-

[1] *Jour. Amer. Med. Sci.*, April, 1904.

ever, when affected, succumb more rapidly than do adults. Lymphatic leukemia is always fatal earlier than the spleno-medullary variety, and in severe acute cases the larger lymphocytes are found. Although recovery does occur occasionally, most cases of leukemia, of whatever form, prove fatal certainly within five years, generally in two or three years, and sometimes in seven or eight months or even less (from two weeks to two or more months) in acute leukemia. In an advanced case the prognosis is hopeless. Grave symptoms heralding an early termination are profound debility, anemia, emaciation or edema, severe and obstinate hemorrhages, cerebral apoplexy, persistent diarrhea, and high fever. Intercurrent affections not infrequently cause death, while, on the other hand, cases are recorded in which the appearance of intercurrent infectious diseases (erysipelas, entero-colitis, pleuritis) has favorably affected the course of leukemia. Remissions may rarely occur.

Treatment.—At present no remedies are known to have any permanent curative effect. The application of the Röntgen ray over the spleen, sternum, and extremities of the long bones (viscera 10-minute exposures, joints 5-minute exposures), at first suggested by N. Senn,[1] is followed by: disappearance of the fever; decided decrease in the volume of the spleen; increase in the number of red cells and in the hemoglobin. The leukocytes are at first reduced and may fall to near the normal number, but later the blood again becomes leukemic. Oettinger, Fiessinger, and Sauphar[2] claim that radiotherapy produces a leukolytic ferment, which by disintegrating the corpuscles, may transform chronic myeloid leukemia into an acute phase, or pernicious anemia. Warthin[3] believes that the Röntgen-ray treatment of leukemia finds a pathologic basis in the selective action which the rays have for cells of the lymphocytic and myelocytic types. Kiralyfi[4] reports constant benefit in seven cases from the use of benzol (gr. viiss—0.5, in equal quantity of olive oil in capsule, q. d., gradually increased to gr. lxxv—5.0 daily). Larrabee[5] has used the mixed toxins of Coley in 18 cases with results encouraging enough to warrant further trials.

The environment should be made as favorable as possible—physically, mentally, socially, and morally. Out-of-door life in a mild, dry climate, an abundance of nutritious and easily digestible and assimilable food, calm and moderate exercise of mind (depending upon the endurance of the patient), should all be advised and encouraged. On the other hand, traumatism, irregular habits of body, worry, excitement, and passionate emotions and appetites should be regulated and avoided.

Arsenic gives the best results in most cases, and should be pushed to the limit of tolerance, as in pernicious anemia. It should be given continuously, regardless of apparent improvement under its use, as the latter may be only the natural remission—a not uncommon incident in the disease. Bone-marrow, either raw and spread upon bread, or in the form of a glycerin extract, may be tried when arsenic fails. Oxygen-inhalations and blood-transfusion have been suggested. The so-called "splenic remedies," whether systemic or local, have no controlling influences upon the disease. Complications may be relieved by appropriate treatment.

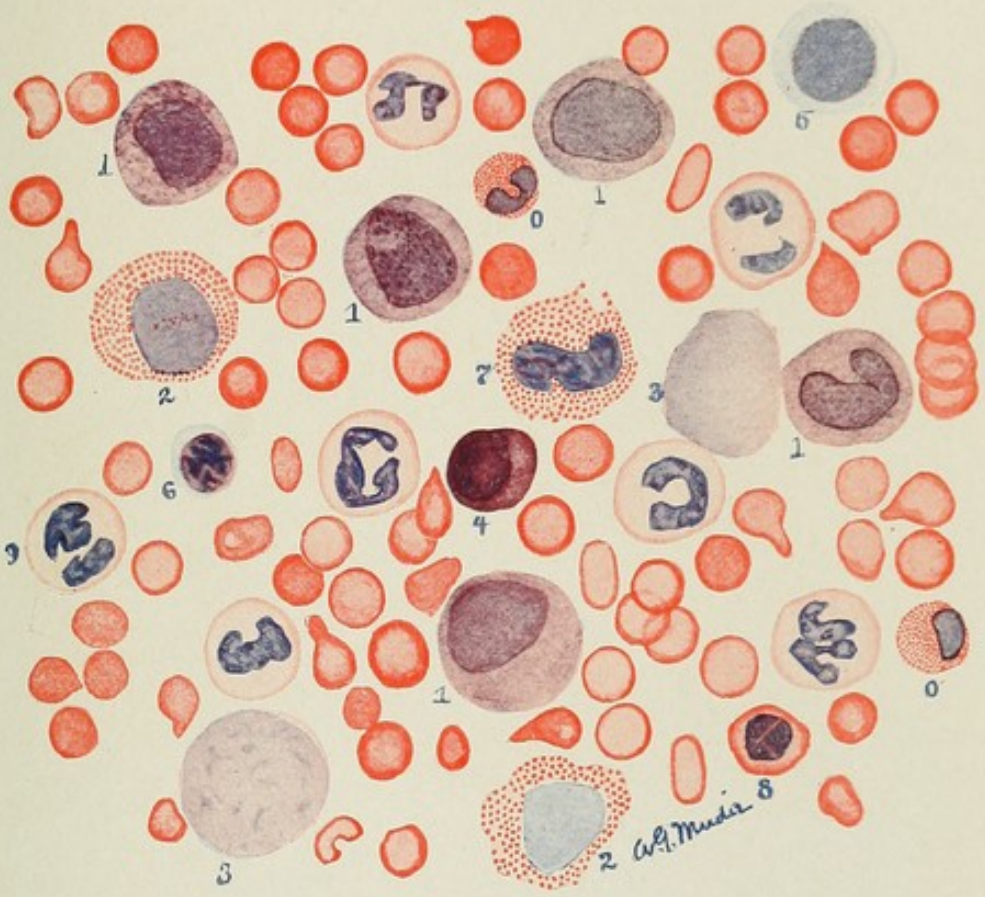
[1] *N. Y. Med. Jour.*, Aug. 22, 1903.

[2] *Archives des Maladies du Cœur*, etc., Paris, May, 1910; *Jour. Amer. Med. Assoc.*, June 11, 1910, 2006.

[3] *Internat. Clin.*, vol. iv., Fifteenth Series, 1906.

[4] *Wien. klin. Woch.*, 1912, xxv., 1311.

[5] *N. Y. Med. Jour.*, Feb. 15, 1908.



BLOOD OF SPLENOMEDULLARY LEUKEMIA.

1, Myelocytes; 2, eosinophilic myelocyte; 3, leukocytic shadows; 4, polychromatophilic megaloblast; 5, large mononuclear leukocyte; 6, small lymphocyte; 7, eosinophile; 8, megaloblast; 9, polymorphonuclear leukocyte; 10, small eosinophiles (stained with eosin and hematoxylin. Obj. B. and L. one-twelfth oil-immersion).

[L. Napoleon Boston.]

LEUKANEMIA.

So-called "leukanemia" (Leube) is most probably either leukemia with terminal anemia, or pernicious anemia with lymphoid or myeloid marrow (Cabot).

CHLOROMA.

Pathologically, it consists of a sarcomatous growth, the primary seat of which is in the periosteum and bone in and about the orbit. Threadgold holds that the bone-marrow is primarily affected in chloroma. The growth shows a pea-green pigmentation. Secondary growths may be widespread. Gulland and Goodall claim that there is no histologic difference between chloroma and lymphoid leukemia. Myeloid chloroma also occurs, 10 cases having been recorded by Jacobaeus.[1]

Symptoms.—Pain in the orbital region, exophthalmos, and deafness are noted early. The principal diagnostic features are gangrenous stomatitis and often a high-grade anemia, usually associated with a hemorrhagic diathesis. There is some enlargement of the lymphatic glands and spleen. In the lymphoid form there occur the tumor-like infiltrations of the orbit and other parts of the skull, and it is seen in children. The blood-picture is that of lymphoid leukemia. The myeloid form is characterized by the presence of neutrophilic myelocytes, making up from 50 to 95 per cent. of the cells, and a marked leukocytosis. It seldom shows tumor growths. In those cases in which the tumor formation is not prominent, the clinical picture may resemble pernicious anemia, acute sepsis, scorbutus, or a septic diphtheria (Bierring).

The *course* of the disease—spoken of by French writers as "green cancer"—is rapid, and death usually comes on within a few months. The Röntgen rays have given marked improvement in the *treatment* in some cases, but not in others.

PSEUDO-LEUKEMIA.

(*Hodgkin's Disease; General Lymphadenoma.*)

Definition.—An anemia characterized by the anatomic peculiarities resembling those of lymphatic leukemia—viz., progressive hyperplasia of the lymph-glands, occasional secondary lymphoid growths of other organs (liver, spleen); and by the absence of the destructive blood-changes of true leukemia.

Varieties.—Although the disease that bears his name was first described by Hodgkin of Guy's Hospital in 1832 as an affection of the lymphatic glands and spleen, two varieties are included under the title of pseudo-leukemia (or Hodgkin's disease), as follows: (1) that which presents simply an enlarged spleen (the less frequent one); and (2) that in which the lymphatic glands are chiefly involved.

Pathology.—The lymph-glands show different degrees of hyperplastic enlargement and consistency. In the earlier stages they are small, isolated, and movable, while in advanced and well-developed cases of the disease they are larger, fused together into great bunches, and more or less fixed by fibrous investment. As a rule, the glands are soft and elastic, though sometimes they are hard and dense, and masses as large as an orange or pineapple may be seen. Single glands may be as large

[1] *Deutsch. Archiv f. klin. Med.*, 1909, Band xcviii., Heft 1 and 2.

as a hen's egg, and the gland-capsules may show connective-tissue proliferation and a thickening periadenitis. Extension of the lymphatic growth into the surrounding tissues by perforation of the capsule may occur. As a rule, the overlying skin is freely movable, though it may rarely be adherent. On section the tumors display a smooth white or reddish-gray surface in the case of the soft and almost fluctuating glands, and a grayish or a yellowish-white color if they are firm. The fusion of the swollen glands into nodular masses is also seen, and when ulceration through the skin has taken place suppuration of the glands may be revealed. In the harder tumors areas of necrosis having the appearance of caseation, and shining masses of fibroid tissue may be visible.

Microscopically, there is a typical hyperplasia of the lymph-cells often obscuring completely the reticulum of the gland, except in the

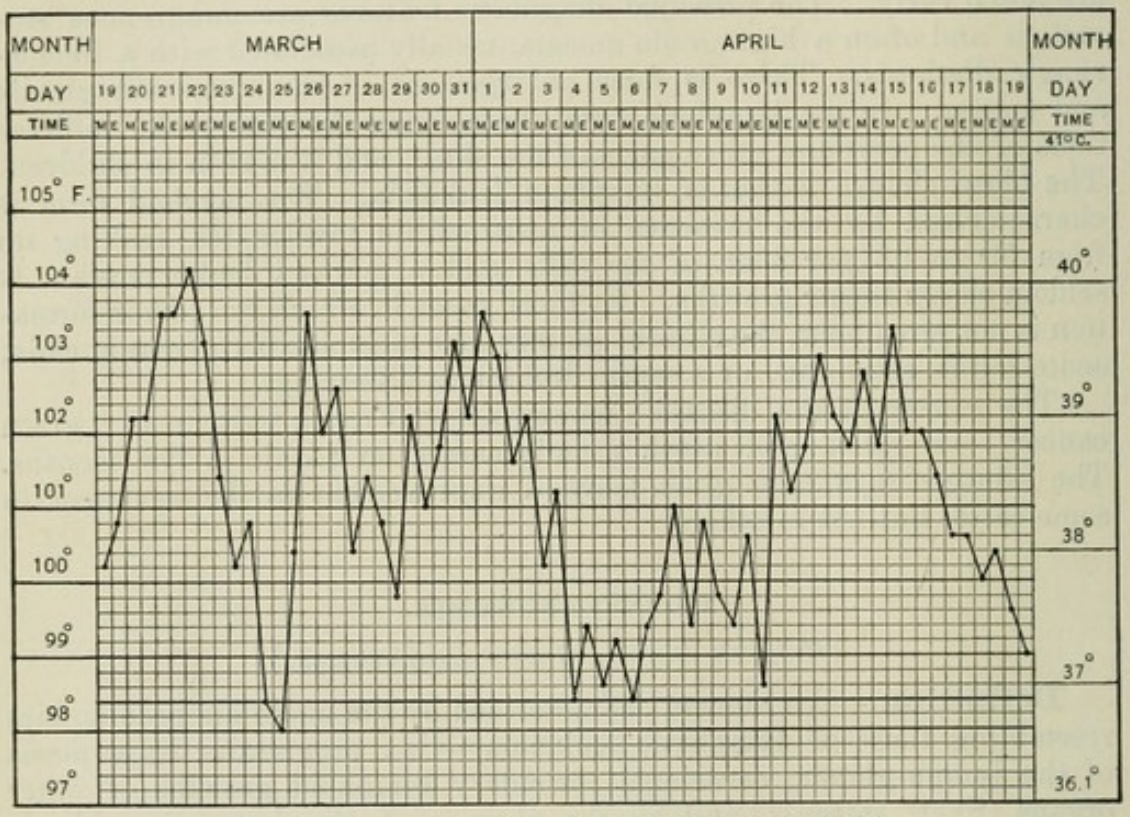


FIG. 41.—Temperature-chart of a case of pseudo-leukemia.

harder enlargements, where the fibrous proliferation shows a very distinct network. The change is a lymphadenoma of the lymphatic glands. Reed [1] states that the true form of the disease has a specific histology, showing large numbers of eosinophiles and a peculiar variety of giant-cell, different from that of tuberculosis. Longcope, Ruffin,[2] and others also believe that the disease shows histologic changes peculiar to itself. Gibbons,[3] on the contrary, is strongly inclined to the malignant theory.

The cervical glands are most prominently involved. The superficial chains of glands—axillary, mediastinal, scapular, and pectoral—especially along the great vessels, are often found connected, and the inguinal, bronchial, and lumbar glands are also affected, though less frequently. The retroperitoneal glands are more frequently affected than the mesenteric; they have occasionally projected externally by perforation through the sternum.

The *spleen* is enlarged in about four-fifths of the cases, but only slightly.

[1] *Johns Hopkins Review*, vol. x., p. 133. [2] *Amer. Jour. Med. Sci.*, April, 1906.
[3] *Amer. Jour. Med. Sci.*, November, 1906.

The *spleen* is enlarged in about four-fifths of the cases, but only slightly. In the majority of cases there are disseminated throughout the organ whitish, lymphomatous growths or nodules from the size of a pea to that of a nut. Their histologic structure is like that of the lymph-glands (lymphadenoma). Occasionally the spleen alone is hyperplastic.

Lymphomata may also develop in the tonsils, lingual follicles, intestinal lymphatics, liver, kidneys, lungs, brain, spinal cord, heart, testicles, retina, and skin. The bone-marrow often appears the same as in pernicious anemia.

Etiology.—There are no well-established *predisposing conditions* to which Hodgkin's disease is referable. In 75 per cent. of cases males are affected, and young and middle-aged persons—between the ages of ten and forty years. In an analysis of 100 cases 30 were under twenty years, 34 between twenty and forty, and 36 after forty (Gowers). Heredity may possibly be a cause. The disease would seem to belong to the group of infectious granulomata, but the *exciting cause* is not known. Flexner thinks that certain protoplasmic foreign bodies (found in the larger nodules of two cases) may possibly have a causal relation. Malaria, syphilis, chronic skin-diseases, and various irritative conditions, especially of the mouth, giving rise to local glandular swellings, have also been assigned as causes. In undoubted instances of Hodgkin's disease the lymphatic glands frequently harbor tubercle bacilli; hence it has been thought that the latter exercise a distinct causative influence. It must be remembered, however, that some of these may be examples of secondary accidental infection; others of primary diffuse lymphatic tuberculosis, indistinguishable from or mistaken for Hodgkin's disease. Musser[1] thinks "the disease is not improbably an expression of lymphatic tuberculosis." Sailer[2] arrives at much the same conclusion. Löffelmann contends that the disease is caused by the tubercle bacillus, having found the latter in six out of seven cases by means of the antiformin method. Reed[3] believes true Hodgkin's disease is not due to the tubercle bacillus, but that it is of infectious origin. It is not uncommon to find pseudo-leukemia developing in a person who immediately preceding the beginning of the disease was apparently in perfect health.

Symptoms.—Usually the first thing to attract attention is the enlargement of the submaxillary and cervical glands, often on one side of the neck alone. These grow gradually until they may finally appear on both sides as large as a fist, and produce considerable disfigurement. Sometimes several years may elapse before other glandular groups are affected, but, as a rule, it is a matter of months only before the axillary, then the inguinal, and perhaps the internal, glands are invaded. The changes vary greatly in rapidity and extent.

At first the *general health* may be but slightly affected. A little constitutional disturbance and some pallor may be complained of, though seldom before the glandular swellings are noticed. Then as the disease progresses the paleness increases and all the symptoms of a marked anemia appear—languor, failure of physical strength, beginning emaciation, gastro-intestinal derangement, headache, giddiness, palpitation, dyspnea, and edema of the legs. Later, the serous cavities contain effusion and there is a tendency to *hemorrhages*. Epistaxis and metrorrhagia may occur, and petechial spots, especially on the lower extremities, are not infrequent. The *physical signs* of anemia—hemic murmurs

[1] *Amer. Med.*, Jan. 4, 1902. [2] *Phila. Med. Jour.*, April 5, 1902. [3] *Loc. cit.*

—are also present. An irregular slight or moderate pyrexia is common to most cases. Fever of a peculiar intermittent type has been observed, the intermissions and paroxysms each lasting for several days or weeks (see Fig. 41), and the term "chronic relapsing fever" has been applied in consequence. When these pyrexial exacerbations occur the cases generally run a more acute course. Ague-like paroxysms may persist for even months, as described by Pel, *of Amsterdam.*

The symptoms due to *mechanical compression* by the lymphomata are varied and numerous, depending upon the number, size, and distribution of the tumors. Hundreds of tumors may be present throughout the body, but, unless they press upon the adjacent nerves, the glands are not usually painful. Enlargement of the tracheal and bronchial glands may cause dysphagia, dyspnea, thoracic pain, disturbed phonation, and venous congestion, by pressure respectively upon the esophagus, trachea, bronchi, thoracic nerves, recurrent laryngeal nerves, superior vena cava, and the jugular veins. The obstruction to respiration may become so great as to produce death by suffocation.

Circulatory Symptoms.—*Congestion* of the head and upper extremities may be quite marked, and in such cases compensatory dilatation of the superficial veins is observed. *Edema* of the hand and arm may result from venous obstruction due to the pressure of very large axillary glands. The *heart's action* may be disturbed by pressure on the pneumogastric, and the heart itself may be dislocated by great gland-tumors within the chest. Under such circumstances the latter may be detected by dulness on percussion over the anterior mediastinal space.

Edema of the feet and legs may be an early indication of enlarged abdominal glands pressing upon the femoral veins. Albuminuria is not uncommon; ascites and hydrothorax are late conditions. *Jaundice* is sometimes attributed to pressure upon the bile-duct. *Gastro-intestinal disturbances* may be troublesome, and are usually symptomatic of lymphoid growths in the stomach and bowels. In thin individuals gland-masses may be palpable over the abdomen. Deafness may be caused by growths in the pharynx.

Nervous Symptoms.—Inequality of the pupils and unilateral sweating of the face, owing to glandular pressure upon the cervical sympathetic, may be noticed in some cases. Sharp lancinating pains along the nerves may also be felt. *Pressure-paraplegia* and *neuralgic pains* variously distributed throughout the body should also be mentioned among the nervous manifestations.

Cutaneous Symptoms.—It has been suggested that the bronzing of the skin sometimes seen in Hodgkin's disease may be due to the pressure of enlarged glands upon the suprarenal capsules. An intense pruritus has been complained of, and the skin may be erythematous. Occasionally the thyroid and thymus glands are involved.

Spleen.—The slightly or moderately enlarged spleen can usually be felt just below the ribs, projecting toward the navel. Tenderness over the spleen and bones may be elicited. The characteristic feature in splenic pseudo-leukemia is the decided enlargement of the spleen without involvement of the lymphatics.

The *blood* shows a moderate diminution in the number of red corpuscles, and a corresponding diminution in the hemoglobin, the former in most instances numbering from 2,000,000 to 4,000,000 per cubic millimeter. There may be more or less leukocytosis, and sometimes the lymphocytes may preponderate relatively; if the latter be present

in great numbers, the blood may also show similarity to that of lymphatic leukemia. An occasional normoblast may be seen. Blood-platelets are abundant (Bunting).

Diagnosis.—Pseudo-leukemia is more readily confused with *tuberculous adenitis* than any other disease, particularly at the outset. Although an acute tuberculous adenitis may very closely simulate Hodgkin's disease and render a diagnosis almost impossible, more often the glands of tuberculous adenitis are slower in enlarging and extending than in this disease. In fact, extension of the lymphatic enlargements of tuberculosis is rarely seen as compared with pseudo-leukemia. Again, tuberculous adenitis is most common in the young, is unilateral rather than circumferential in the neck, and attacks the submaxillary glands oftener than the cervical chains along the sterno-cleido-mastoid. Again, periadenitis, adhesion, and suppuration of the glands occur in tuberculosis. Tuberculous foci in other organs may also be found. Intermittent attacks of pyrexia are an indication favoring Hodgkin's disease. In doubtful cases a gland may be removed for microscopic examination. The tuberculin test will exclude glandular tuberculosis.

The blood should be examined in order to differentiate from *leukemia.*

Syphilis must be carefully excluded by the history, symptoms, and therapeutic test. *Neoplasms* of the lymph-glands may sometimes be difficult to distinguish from pseudo-leukemia.

The diagnosis of *splenic pseudo-leukemia* is to be made on the decided splenic enlargement without involvement of the lymphatics. The following conditions, however, must be distinguished: (*a*) Pernicious anemia with enlargement of the spleen; this is readily done by a blood-examination; (*b*) Cirrhosis of the liver, in which there is splenic enlargement; (*c*) The splenic tumor of chronic malarial poisoning. Here the blood should be repeatedly examined for the organism of Laveran, if the patient resides in a malarial region; (*d*) Idiopathic enlargement of the spleen without any anemia.

Prognosis.—This affection runs an almost invariably fatal course. The remissions and exacerbations of the disease are, however, notable. In some cases the termination may occur in a few months, but usually death ensues after the lapse of two or three years. It should be remembered that some instances of Hodgkin's disease seem to merge into a true lymphatic leukemia.

Grave indications are the rapid extension of the glandular enlargements, great debility, anemia, emaciation, steadily increasing and continuous pyrexia, thoracic pressure-symptoms, hemorrhages, and marked anasarca. Sometimes the tumors diminish greatly before death. In certain cases general streptococcus infection, intercurrent diseases, or such complications as empyema or nephritis, may be the immediate cause of death.

Treatment.—Surgical treatment is of no avail. It is claimed that exposure of the enlarged glands to the Röntgen ray is followed by a decrease in glandular enlargements and an improvement in all symptoms.[1] Hygienic measures and the use of all possible agencies to support the strength of the patient should be resorted to, and the administration of arsenic in gradually ascending doses, as for pernicious anemia and leukemia. The value of Fowler's solution is undoubted in many cases. Phosphorus has also been recommended, and the galvanic current may be applied topically. Tonics, nutrients, and red bone-marrow are of service.

[1] Steinwald, *Jour. Amer. Med. Assoc.*, March 26, 1904, p. 828.

ANÆMIA INFANTUM PSEUDO-LEUKÆMICA.

Definition.—The above title was given by von Jaksch to a form of anemia occurring in childhood that bears certain similarities to leukemia, but it is without the tendency to a fatal end.

Pathology.—Splenic enlargement is the most striking lesion. The organ is hard and dark red. The histologic examination shows a uniform hyperplasia, such as is found in splenic hypertrophy due to various conditions. The liver is usually enlarged; slight enlargement of the lymphatic glands may occur, though never lymphomatous tumors.

Etiology.—Children under the age of four, and particularly during the second half year of life, are especially prone to this condition. It is equally common in the two sexes, and most often seen in rachitic infants, 16 to 20 cases collected by Monti and Berggrün having exhibited this etiologic factor. Hereditary syphilis, intestinal disturbances, and other diseases doubtless play a part in the etiology. The disease is rare.

Symptoms.—The *onset* is gradual. The child becomes pale, weak, and often emaciated, and *enlargement of the spleen*, until it reaches an enormous size, is the most striking feature. Hepatic enlargement is frequently present, but does not correspond to that of the spleen, and the lower border of the organ is sharp instead of rounded, as is the case in leukemia. Gastro-intestinal disturbances may occur. Death may result from increasing weakness, peritonitis, or pneumonia.

Blood.—An examination of the blood will in many cases show an inordinate reduction in the number of red corpuscles. Nearly always the number is below 3,000,000. Degeneration of the red corpuscles, polychromatophilia, and poikilocytosis are seen. Large numbers of nucleated erythrocytes, especially normoblasts, may be found. The reduction in hemoglobin is often relatively greater than that in the number of erythrocytes. A marked increase in the number of leukocytes is one of the characteristics, the number ranging from 40,000 to over 100,000, and the proportion of the red to the white at times being as low as 12 to 1. Von Jacksch insisted that the different forms of leukocytes occur in their usual relative proportions.

Diagnosis.—The lesser grade of hepatic enlargement, and the character of the leukocytosis distinguish these cases from true leukemia. The evidence is in favor of its being a type of secondary anemia.

Prognosis.—Under treatment most cases terminate favorably.

Treatment.—Hygienic measures together with the administration of remedies directed to the anemia constitute the treatment.

SPLENIC ANEMIA.

Definition.—A condition described by H. C. Wood in 1871; it was looked upon by many authors as a splenic form of Hodgkin's disease. The present tendency is to regard it as a distinct variety of anemia.

Pathology.—Among pathologic characters, splenic enlargement is to be specially noted. It is an idiopathic enlargement of the spleen with anemia and without lymphatic involvement. Microscopically, there is hyperplasia of the spleen with atrophy of the pulp and hyaline degeneration of the Malpighian bodies. In other cases replacement of the normal

structure by fibrous tissue and large endothelial cells occurs, with clear protoplasm containing several nuclei and giant cells among them.[1]

Etiology.—So-called splenic anemia is not rare in persons who have resided in malarial districts for a long period of time (although cases are recorded in which no previous history of malaria existed) and in rachitic persons. It is probably a process of chronic toxic nature, *e. g.*, syphilis.

Symptoms.—The affection is characterized by three stages:[2] (1) The initial stage, which shows extreme anemia, with marked loss of power and muscular wasting, emaciation, however, being usually slight, notwithstanding.

(2) The second stage is characterized by progressive enlargement of, and pain in the region of, the spleen. Anemia is now profound, loss of strength extreme, and hematemesis common. Hemorrhages from other mucous membranes and, less often, of the skin, are also noted. The fever is apt to be hectic in type (ranging from 100° to 102° F.—37.6°–38.0° C.—and rarely even higher).

(3) The condition is that of progressive asthenia, ending in death.

In the last stage there may be cirrhosis of the liver, jaundice, and ascites (Banti's disease).

The Blood Characters.—The anemia is of the chlorotic type. The red cells are usually near the normal count (3,000,000 to 4,000,000), while the hemoglobin is greatly reduced (50 per cent. or lower). Poikilocytosis may exist. A leukopenia is usually present. The *treatment* is that of the graver forms of anemia. Splenectomy is indicated by recurring hematemesis. Harris and Hertzog[3] report 14 recoveries in 19 cases, with 1 result not stated.

POLYCYTHEMIA WITH SPLENIC TUMOR.

This is a special complaint of obscure pathogenesis. The spleen is quite hard and usually enlarged. A cheesy tuberculosis has been noted, but this is not constant.

Etiology.—Certain writers regard changes in the bone-marrow as primary, leading to increased erythroblastic activity. Defective venous tone may play a rôle in the pathogenesis. The Hebrew race is markedly predisposed. A majority of the cases occur during middle life, and "the cases are about equally divided between the sexes" (Engelbach and Brown). "I regard it as probable that there is a form of primary polycythemia of unknown etiology, characterized principally by marked polyglobulism and other hemic features, cyanosis, headache, vertigo, and splenic enlargement, but it must be of rare occurrence."[4] On the other hand, the majority of cases which have been reported have had a different etiologic pathology, circulatory stasis resulting from pressure of malignant tumors, valvular heart disease, and the like.

Symptoms.—According to Reckzeh,[5] the first symptoms are vertigo, headache, mental apprehension, general weakness, and gastro-intestinal disturbance. In fully-developed cases marked cyanosis of the skin and mucous membranes, with dilatation of the veins and sometimes hemorrhages, are prominent features. The spleen may extend downward to the level of the umbilicus. The characteristic blood-findings are an enormous increase of the hemoglobin, rarely to 150 per cent., and a high erythrocyte

[1] Osler, *Amer. Jour. Med. Sci.*, November, 1902.
[2] S. West, in Allbutt's *System of Medicine.*
[3] *Annals of Surgery*, July, 1901.
[4] *Amer. Jour. Med. Sci.*, June, 1907, by the writer.
[5] *Zeit. f. klin. Med.*, vol. lvii., Nos. 3 and 4.

count, the estimation ranging from 7,000,000 to 12,000,000 in some cases. The leukocytes vary as to number, but often about normal figures are found. The total volume of the blood is decidedly increased.

Geisböck describes a second form, or *polycythemia hypertonica* (erythrocytosis), in which the blood-pressure is, as a rule, quite high. The majority of cases are associated with arteriosclerosis and nephritis, but not all. Neither is viscosity of the blood responsible for the rise of arterial pressure. The changes in the bone-marrow, in one case at least, were identical with those of Vaquez's disease. In this variety the spleen is not enlarged, but otherwise the symptomatology and treatment are the same as in polycythemia with splenic enlargement.

The *course* is exceedingly chronic, and the *prognosis* unfavorable.

The *treatment* is by arsenic, quinin, and an iron-free vegetable diet and a carefully regulated mode of life. The iodids have been advised in order to decrease the viscosity of the blood. The cerebral symptoms have been benefited by the nitrites. Venesection has proved of service in some cases. Splenectomy may be indicated.

DISEASES OF THE DUCTLESS GLANDS.

DISEASE OF THE SUPRARENAL CAPSULES.

ADDISON'S DISEASE.

Definition.—A constitutional disease, characterized by a degeneration of the suprarenal capsules or semilunar ganglia, a bronzed or pigmented skin, great bodily and mental asthenia, feeble circulation, and gastro-intestinal irritability.

This affection is named in honor of its discoverer, Thomas Addison of Guy's Hospital, London, who first described it in a monograph published in 1855, entitled "The Constitutional and Local Effects of Disease of the Suprarenal Capsules."

Pathology.—Addison emphasized the fact that while the suprarenal bodies were affected with a fibro-caseous alteration in many cases, the anatomic changes were by no means always the same. Both suprarenal capsules are usually diseased at the same time. Tuberculosis is the commonest condition, and is often associated with tuberculous lesions in other parts of the body, as in the lungs, bones, and other glands. Rarely, it seems to be primary, no other evidences of tuberculous infiltration being found. The capsules are enlarged, firm in places, and nodulated on the surface, owing to the caseous masses surrounded by fibrous tissue. Sometimes there is marked cicatricial contraction of the adrenals, and the adjacent structures may be found matted together with the capsules. *Microscopic examination* shows a reticulum of connective tissue surrounding a soft, cheesy, granular, and fatty detritus, lymphoid cells, and some giant-cells. Other morbid, non-tuberculous processes in the adrenals are atrophy of one or both glands from interstitial cirrhosis, carcinoma or sarcoma, and chronic inflammation.

Especial attention has recently been given to the condition of the *solar plexus* and *semilunar ganglia* of the abdominal sympathetic, and implication of these nervous structures by compression, cicatricial contraction, entangled in the cicatricial tissue surrounding the suprarenal bodies, or by chronic inflammation, is not infrequently discovered, together with a degeneration and deep pigmentation of the semilunar ganglion-cells.

Enlargement of the solitary and agminated follicles of the intestine,

and slight enlargement and some softening of the spleen are noted at times; parenchymatous or fatty degeneration of the heart, liver, and kidneys has also been noted in some instances. The thymus gland may be found to have remained normal, or even to have enlarged, perhaps. The deposition of pigment is in the same anatomic elements as in the negro—in the lower layers of the rete Malpighii.

The pathologic connection between the symptomatic phenomena of Addison's disease and the anatomic lesions has not been made out. The experimental evidence regarding the functions of the adrenals is imperfect; but it seems quite probable that some essential "internal secretion," influencing the normal metabolism of the skin and muscles, is diminished or absent in Addison's disease. On the contrary, cases exhibiting the clinical phenomena of this affection have occurred in which no suprarenal morbid processes could be found *post mortem.* Again, marked changes have been observed in these glands, while during life no symptoms of the disease had been noted. Hence, it is maintained by some that the abdominal sympathetic nerves and ganglia are directly concerned in producing the clinical manifestations, either by an independent morbid process or by extension from some adjacent organ. Others hold that both the adrenals and the sympathetic ganglia are the seat of pathologic changes. The data are not sufficient, however, to determine whether the principal involvement is nervous or secretory.

Etiology.—This is obscure. It has been held that some infection of the blood from without precedes the suprarenal and nervous lesions of Addison's disease. A *tuberculous* diathesis or infection has also been emphasized by some investigators, and Fleming and Miller[1] have reported a family with probable Addison's disease. A history of *injury* to the trunk has been noted in several cases. The disease is more common in Europe than in America, though it is rare everywhere. Analysis of 183 cases showed 119 males and 64 females (Greenhow). While the disease may affect all ages (it may even be congenital), it is usually found in early or middle life—between fifteen and forty years of age. That Addison's disease is due either to a general neurosis or to disturbed hematopoiesis is merely hypothetic.

Symptoms.—While it does happen not seldom that tuberculosis or carcinoma affects the adrenals, the purest and most typical symptoms of Addison's disease are apparently primary in their development, and not those that usually attend the course of the former diseases.

Cutaneous Symptoms.—The gradual pigmentation of the *skin* of various parts of the body may be one of the first evidences of the affection. This pigmentation may have either a dusky-yellow, bronze or yellowish-brown, olive, deep or greenish-brown, or even black color. Although sometimes diffuse, the discoloration is not uniform over all parts of the body, but commences earlier, and becomes deeper especially on the exposed parts and where the normal pigmentation is marked, as the face, neck, backs of the hands, the axillæ, abdomen, groins, genital regions, and the areolæ of the nipples. Pigment-spots, often somewhat bluish in color, are also found on the *mucous membranes* of the mouth, lips, conjunctiva, and vagina. On the lips the discoloration takes the form of a dark streak, running lengthwise, near the junction of the skin and mucous membrane; or brownish patches or streaks cor-

[1] *Brit. Med. Jour.*, April 28, 1900.

responding to the points of pressure by the teeth may be noticed. Irregular stains with ill-defined borders may also be shown on the skin, corresponding to the lines of pressure exerted by garments, strings, suspenders, garters, etc. (Greenhow). White patches of *leukoderma* may be seen here and there, in marked contrast to the pigment-deposits. The "white line," produced by drawing the finger lightly over the skin of the abdomen, is characteristic.

General Symptoms.—The constitutional symptoms may exist in a slight degree before the pigmentation first attracts the patient's attention. There is gradual and progressive *asthenia* without apparent cause, great lassitude and loss of physical and mental energy, breathlessness, headache, dizziness, tinnitus aurium, sighing, and fatigue. The *blood-examination* shows a moderate reduction of the erythrocytes and hemoglobin, rarely becoming marked. There is no leukocytosis and often a leukopenia. The fat, particularly of the abdomen, may be well preserved.

Circulatory Symptoms.—The *heart's action* is weak and the pulse small and feeble; attacks of faintness and palpitation on exertion are common, as are functional murmurs and coldness and clamminess of the extremities. The blood pressure is greatly reduced before death (Turner).

Gastro-intestinal symptoms are usually prominent. There is a loss of appetite, and nausea and vomiting may occur early and either be paroxysmal or persistent. The *tongue* may be clean, and the gastric disturbances do not seem to follow errors in diet. *Diarrhea* may be troublesome in the latter stage, and is often associated with intractable vomiting. *Neuralgic attacks* of either sharp or dull, aching pain are referred to the epigastric, hypochondriac, and lumbar regions in about one-third of the cases. The *mind* is usually clear until near the last, but mental weariness is constant, and, as the latter stages of the disease come on, the patient often lies in a somnolent, semi-comatose state. The physiognomy expresses fatigue, dejection, and apathy; the speech becomes slow and incoherent, and in many cases the patient passes into delirium. *Prostration* is profound, the weakness being disproportionate to the general condition.

Renal Symptoms.—Polyuria is sometimes evident, but albumin is seldom present. The amount of indican is increased, as it is in the urine of all of the cachectic diseases associated with destruction of albuminoids. There is usually a diminished excretion of urea, but urobilin and uromelanin may be present in abnormal quantity. Tubercle bacilli may be found in the sputum.

Diagnosis.—The principal error in diagnosis is in the assumption that the case is one of Addison's disease, simply from the presence of patches of pigmented skin. Other conditions in which the discoloration may simulate that of Addison's disease are the following: (1) Carcinomatous and tuberculous disease, particularly when seated in the abdomen and when involving the peritoneum; (2) Hepatic disease, such as the cirrhosis of diabetes, protracted jaundice, chronic congestion, and lithemia ("liver-spots"); (3) Pregnancy, and uterine disease, in which the patchy discolorations (chloasmata) appear principally upon the face; (4) Irritation of lice and dirt and exposure, as in the case of tramps and vagrants ("vagabond's disease"); (5) Tinea versicolor; (6) Melanotic sarcoma; (7) Exophthalmic goiter; (8) Post-eruptive staining of

syphilitic eruptions; (9) The administration of silver nitrate for a long time (argyria); (10) Marked brunette complexions and racial admixture.

When the pigmentation is scanty, of course the diagnosis is more difficult; but in all cases of pigmentation in which other causes may be excluded the progressive asthenia, unaccountable vomiting and diarrhea, easily compressible pulse, great bodily weakness, mental hebetude, and lumbar and epigastric pain render the diagnosis of morbus Addisonii, or *malasma suprarenale*, justifiable. The bronzing of the skin may precede as well as follow the constitutional symptoms.

In the negro the diagnosis of this affection is extremely difficult, both on account of the naturally dark skin and because of the dark discolorations of the oral mucous membrane, found even in health.

Prognosis.—The course of Addison's disease is almost always chronic, though cases have been reported occasionally in which the onset has been sudden, with febrile phenomena and a comparatively acute course of a few months, or weeks even. Usually the disease lasts about one year, although some cases may continue over five or even ten years. Temporary remissions may be observed, but death is inevitable in by far the majority of instances. The termination is gradual, and by profound asthenia, or sometimes by coma, delirium, or convulsions (epileptiform).

Treatment.—The hygienic and medicinal treatment must have the same objects in view as in the other grave cachectic diseases, and is both sustentative and symptomatic. As quiet a life as possible should be strictly enjoined, owing to the dangers of sudden and fatal syncope. Rest in bed is necessary in moderate and advanced cases during a part of the day for the former and constantly for the latter. The diet should be restricted to light nutritive, concentrated, and easily assimilable food. Carbohydrates in the diet have been found to diminish the adynamia. An absolute milk diet may be necessary in some cases.

Iron and arsenic may be administered in the anemic cases, and strychnin, guaiacol carbonate, phosphorus, and the nuclein preparations may also be given, along with bitter tonics. Bismuth and salol may be of great service in controlling the diarrhea that often occurs. The nausea and vomiting may be relieved by unfermented grape-juice, albumin-water, champagne, cracked ice, cerium oxalate, creasote, and the like. Electricity is often a valuable adjunct in the treatment of the muscular weakness and nervous exhaustion, and even in reducing the pigmentation.

It seems quite probable that the administration of the extract of suprarenal capsules will prove to be of considerable value in causing marked improvement, if not a permanent cure, in a certain percentage of cases. In one instance mentioned by Osler, in which a glycerin extract of a pig's suprarenal was given at first in doses of half a glass three times a day, improvement was noted in the temperature, pulse, weight, and physical and mental vigor from the first week of the treatment, which was continued for three months and a half. Eight months after the treatment was begun the patient appeared to be well and strong, and attended to business; the pigmentation, however, was not removed. In a recent case of my own this remedy produced like results. Robin mentions a case treated by the administration of suprarenal gland that has shown persistent good health for three years. For the present, however, too positive a value should not be attributed

to the suprarenal extract, and results contrary to the above are to be found in the literature. Grafting of the gland, inserting only small fragments. has been proposed.

DISEASES OF THE THYMUS GLAND.

Physiologic Pathology.—Nothing is known definitely concerning the functions of the thymus gland. At the end of the second year of life it attains its greatest development, and thereafter slowly and gradually atrophies, especially after puberty. While there is no evidence that the thymus produces an internal secretion, a certain imperfectly understood "relationship exists between the thymus and organs that do produce an internal secretion, such as the thyroid (in Grave's disease), parathyroids, pituitary body (in acromegaly), the ovaries, testicles (atrophying as the sexual organs develop), etc.; and extracts of the thymus are said to cause a fall in blood-pressure and rapid action of the heart" (A. O. J. Kelly).

ENLARGEMENT OF THE THYMUS.

The thymus may become enlarged from various causes (tumor formations, cyst formations, Grave's disease, acromegaly, Addison's disease, Hodgkin's disease, leukemia), and is secondarily affected in tuberculosis, syphilis, and pyogenic infections. On the other hand, atrophy of the thymus has been observed (*e. g.*, infantile marasmus).

STATUS THYMICO-LYMPHATICUS.

(*Status Lymphaticus; Thymic Asthma; Hyperplasia.*)

Definition.—Hyperplasia of the thymus and lymphatic tissues, associated with corpulency and hypoplasia of the heart and blood-vessels, and characterized clinically by stridor and sudden death.

Etiology.—The condition is met with in young children with persistent thymus, although in most cases reported hyperplasia of the entire lymphatic system has been noted,—the *lymphatic constitution* of Paltauf. The cause of the *constitutio lymphatica*, however, is unknown. Says Warthin, "Status lymphaticus may be the consequence of a number of primary morbid processes, such as syphilis, rachitis, some latent infection, auto-intoxication, etc., that are characterized by excessive demands upon the lymphoid and myeloid tissues." Olmacher[1] noted in 18 cases of epilepsy a large thymus gland with hyperplasia of the lymphatic glands throughout the body, and of the lymph follicles of the mucous surfaces.

Symptoms.—These indicate stenosis of the air tract. There is inspiratory and expiratory stridor, as a more or less audible whistling respiration (Schwinn). There are cases in which death occurs without previous stridorous dyspnea, while in others the stridor is congenital or develops soon after birth, and is subject to exacerbations on slight provocation, as screaming or crying or as a result of an acute infection (Kopp's asthma, Millar's asthma). In other instances, the symptoms of suffocation precede the fatal termination. Potts reports 4 cases where children died in this manner after the insertion of a tongue depressor.

Physical Signs.—Among *physical signs* are—inspiratory dilatation of the nostrils, cyanosis, and marked retraction of the supraclavicular, infraclavicular, and intercostal spaces. The fontanelles are sometimes

[1] *Philadelphia Medical Journal*, January 1, 1898; Saunder's *Year-Book*, for 1899.

taut—this in the absence of meningeal retraction is a sign of some value. D'Oelsnitz[1] emphasizes the unusual extent and intensity of the dulness over the manubrium and toward the left. Schridde[2] observed enlargement of the lingual follicles. For diagnostic purposes, a radiographic examination gives trustworthy results.

Prognosis.—The prognosis is highly unfavorable. The sudden death is due to thymic enlargement with secondary laryngeal spasm.

Treatment.—Any recognizable causative factors, *e. g.*, rachitis, anemia, auto-intoxication, etc., should be treated on accepted therapeutic principles. The *x*-rays have been recommended. Operation with a view of removing the offending organ should be undertaken, although there are cases in which the child expires before medical aid can be procured. Siegel in one case, a boy of two and a half years, elevated and stitched the thymus to the fascia over the sternum, with the result that the threatening dyspnea disappeared and the child eventually made a good recovery.

DISEASES OF THE THYROID GLAND.

THYROIDITIS.

Definition.—Acute inflammation of the thyroid gland. The gland may either have been previously healthy or the seat of a goitrous enlargement; when inflammation attacks previously diseased or enlarged thyroid tissue the term *strumitis* is often used.

Pathology.—The gland is swollen, boggy, and, generally, the seat of abscesses; the numerous blood-vessels are engorged; and hemorrhages, thrombi, and areas of tissue-necrosis are found.

Etiology.—Thyroiditis is seldom primary in origin. It may be caused by traumatism, but usually it is secondary to one of the infectious diseases (small-pox, typhus, typhoid fever, malaria). Rheumatism has also been given as a cause. Hemorrhages into the substance of a goiter, whether apoplectic or traumatic, may predispose to a strumitis that may be excited by the introduction of streptococci by an unclean needle, etc. Repeated congestions of the thyroid or a simple acute congestion may dispose to thyroiditis.

Symptoms.—There are *fever*, *pain*, *swelling*, and *suppuration* in one or the other lobe of the gland. *Venous obstruction* may be serious and gives rise to vertigo, headache, cyanosis, and epistaxis; and compression of the windpipe by the great swelling may cause death before the abscess bursts. Resolution occurs infrequently, especially in the "strumous" cases. Indeed, the symptoms of a strumitis are usually more severe, owing to the greater size of the thyroid, a tendency to metastasis, and to the burrowing of pus into adjacent tissues leading to perforation and rupture of the abscess into the trachea or esophagus.

Diagnosis.—Thyroiditis must be differentiated from the *laryngeal perichondritis* that is also seen in the course of infectious diseases, as typhoid fever and small-pox. *Simple congestion*, especially in women from emotional or menstrual disturbances and interference with the circulation from tight collars and the like, must be excluded.

Sclerotic Thyroiditis.—Riedel has described a form of thyroiditis of rapid development involving the entire gland, with fibrous connective-tissue formation and adhesions to surrounding structures. Serious symp-

[1] *Bull. de la Soc. de Pediat.*, Paris, Dec., 1911.
[2] *Muench. Med. Woch.*, Nov. 26, 1912.

toms may result from compression of the trachea and recurrent laryngeal nerves.

Prognosis.—The outcome is usually favorable in all cases in which spontaneous rupture occurs externally or when evacuation of the pus is effected. Myxedema may result from destruction of the gland.

Treatment.—This is antiphlogistic and surgical. The pus must be evacuated, and tracheotomy or thyroidectomy may become necessary.

GOITER.

(*Simple Goiter; Struma; Bronchocele.*)

Definition.—A chronic hypertrophy and hyperplasia of a portion or the whole of the thyroid gland. It is of obscure origin, and is subject to various degenerative changes.

Pathology.—Several different varieties are described. In the *simple hypertrophic* or *parenchymatous* form there is a hyperplasia of all the original tissue-elements. The *follicular* form shows an increase of the true glandular elements alone.

Fibrous goiter is that variety in which the interstitial tissue or stroma is increased out of all proportion to the hyperplasia of the follicles. This variety of goiter may have an inflammatory origin (thyroiditis). In old cases marked sclerosis may be assumed. There is a *vascular* variety, in which the blood-vessels are enormously dilated. More commonly the veins are affected, but in the aneurysmal variety the arteries are chiefly involved. The intense venous variety of vascular goiter has been denominated "cancerous tumor of the thyroid," and the whole gland may in such cases be quite elastic and like spongy erectile tissue. Follicular hyperplasia is often associated with vascular enlargement.

The special varieties of goiter due to degenerative changes are the *cystic*, *amyloid*, *colloid*, and *calcareous*, and of these the first named is the most common. It consists in the development in a large goiter of one or more large or small cysts filled with different kinds of fluid of varying consistency. Sometimes the liquid is colloid or mucinous in nature, and contains the residue of hemorrhages (cholesterin and fatty products). Amyloid changes affect principally the vessels; colloid changes are also frequent, while calcareous infiltration is seen in old fibrous goiters. Inflammation and suppuration of the goitrous gland may ensue.

Etiology.—Goiter may occur anywhere sporadically. Endemically and in its worst forms it occurs in the mountainous districts of Europe, Asia, Mexico, and South America, particularly in the Alps, Pyrenees, and Andes. It has also appeared in certain limestone regions, such as New England and Ontario, Canada, where the *habitual use of limestone-water* for drinking purposes seems to induce the disease. *Heredity* undoubtedly plays a part in its causation, certain children having been born with goiter. Occasionally it has become epidemic in certain sections of the goitrous districts in Europe where military garrisons have been stationed, thus indicating the possibility of some infectious influence. Women are more liable to goiter than men, and it is more common to find it after ten or twenty years of age. It has been alleged that pregnancy also influences the development of this condition.

Symptoms.—The enlarged thyroid is readily *recognized* and *felt*,

though the patient may complain of nothing but the disfigurement, except when the tumor is of sufficient size to cause symptoms of compression. The goiter develops very gradually, and may vary in dimensions from the merest perceptible enlargement to a growth that overhangs the chest and greatly hinders the movements of the head. It may or may not be uniform in its development, and is often more enlarged on the right side and in front than on the left side. It is not infrequently observed to increase in size with each succeeding pregnancy and during or after each menstrual flux.

The tumor is *painless*, is not adherent to the overlying skin or to any of the neighboring bones, and rises and falls during the act of swallowing, moving with the larynx. The *veins* covering it are swollen and prominent. It interferes with respiration oftener than with deglutition, causing dyspnea; alteration or loss of the voice may also ensue. Displacement and distortion of the trachea, the vessels, and other cervical tissues may be produced. Large *pendulous growths* usually cause less serious discomfort than the small encircling tumors that extend downward into the thorax. Headache, somnolence, and marked cerebral symptoms, such as tetany and convulsions, have been described.

The *general health* or nutrition seldom fails unless inflammation and suppuration (strumitis) attack the goiter during the course of some infectious disease, as not infrequently happens, or in cases in which the thyroid function is abolished, leading to the profound nutritional and cerebral disorders of cretinism in children or myxedema in adults.

Dettrich and Osler have each reported an instance of a goitrous growth affecting aberrant portions of thyroid found in the upper region of the pleural cavity, one on the right and one on the left side.

Sudden death may ensue in a few cases, either from pressure on the vagi, or from a severe hemorrhage.

Auscultation often reveals a loud blowing murmur, especially marked in the vascular bronchoceles. *Palpation* over the tumor often shows the bossellated surface present in cystic goiter; fluctuation may also be detected in such cases, as well as over the abscess of a strumitis.

Diagnosis.—Goiter is easily differentiated from other enlargements. The constant location and the character and course of growth of the bronchocele are distinctive. If both lobes of the thyroid are affected, making a symmetric swelling, the diagnosis is almost assured. Bronchocele is not easily confounded with other cervical tumors, such as *lymphadenoma*, *glandular tuberculosis*, *carcinoma* or *abscess* of the *thyroid*, or sebaceous cysts. A characteristic feature of tumors of the thyroid is their vertical movement during the act of deglutition.

Prognosis.—This is guardedly favorable as to life, but unfavorable as to cure. The course is chronic.

Treatment.—Prophylaxis should be practised in goitrous districts by the drinking of boiled water only, and removal to a non-goitrous region is advisable. The majority of drugs recommended for internal and external use have been proved valueless, though in the parenchymatous and follicular forms potassium iodid by the mouth and the vigorous and methodic use of iodin over the tumor have been much lauded. Mercurial ointment—the red or biniodid especially—has also been recommended for local application. Ergot or belladonna in progressively increasing doses may do good in vascular goiters. The younger and

softer goiters may also be benefited by electrolysis, needles attached to the negative pole being inserted into the substance of the tumor while a large sponge or clay positive electrode is placed in the vicinity.

In the older, fibrous, and degenerated goiters surgical treatment alone may be of service. Injections of iodin, tapping of cysts, incisions of the isthmus, and ligature of the thyroid arteries have been practised among the lesser operations. Thyroidectomy, or a partial extirpation of the thyroid, is the radical and final operation. Recently, the fresh, chopped thyroid gland of the sheep, spread on bread, was given in 20 cases of follicular and parenchymatous goiter with gratifying results. Complete recovery, in an anatomical sense, however, was realized in two cases only. The administration of thyroid has transformed several cases of simple goiter into those of the exophthalmic type. McCarrison[1] has treated 33 cases by means of a composite vaccine, with marked success.

EXOPHTHALMIC GOITER.

(*Hyperthyroidism; Graves' Disease; Basedow's Disease.*)

Definition and Nature.—Although the view cannot be unreservedly accepted, exophthalmic goiter is probably of thyroid origin and is dependent upon an abnormal action (or over-action) of the thyroid gland; it is characterized clinically by tachycardia, tremors, enlarged thyroid, and exophthalmos. Among other leading theories the following may be briefly stated: (1) that it is due to disturbed innervation (Buschan); (2) that the seat of the disease resides in the medulla oblongata; (3) that it is an affection of the sympathetic nerves; and (4) that it is a disease of the central nervous system associated with a chronic intoxication.

The theory held by Möbius, that exophthalmic goiter is attributable primarily to a disturbance of the function of the thyroid ("hyperthyroidation"), a condition directly opposed to the lack of thyroid function, as in myxedema, is amply supported by clinical evidence, the complex symptom-group of the former being directly antagonistic to that of the latter disease. Thyroid-feeding, moreover, while it sometimes causes parenchymatous goiters to disappear rapidly, usually aggravates the symptoms of Basedow's disease. Regarding the *pathologic changes* in the thyroid little is known. Brissaud[2] found in 25 cases changes in the thyroid and, although the glands showed no changes peculiar to this disease, yet quantitatively the lesions were always such as to make "hyperthyroidation" possible. A parenchyma increase, in direct proportion to the intensity of the symptoms, occurs (L. B. Wilson). Persistence, and sometimes hypertrophy, of the thymus gland may be causally related. Hector Mac-Kenzie[3] believes that atrophy of the parathyroids may be the cause of some of the more serious symptoms. Muscular changes, probably resulting from toxemia, explain the profound muscular weakness (Askanazy). Jaunin[4] and Gautier[5] contend that chronic iodism and exophthalmic goiter are practically the same condition. Minor[6] affirms that the disease may be due to gastro-intestinal auto-intoxication.

Etiology.—It is more common in women than in men. A table of 200 cases showed 161 females and 39 males (Eshner); and, although it has been met with at both extremes of life, it is seen usually in adults. The influence of heredity is undoubted, and several members of a family

[1] *Lancet*, Lond., Feb. 10, 1912.
[2] *Mercredi méd.*, No. 34, 1895.
[3] *Brit. Med. Jour.*, Oct. 28, 1905.
[4] *Rev. méd. de la Suisse rom.*, No. 5, p. 301, 1899.
[5] *Med. Rec.*, Dec. 2, 1899.
[6] *Ibid.*

may suffer, persons that possess a sensitive nervous organization being especially prone to the disease. Exophthalmic goiter may develop after a trauma.

Among direct causes are emotional disturbance, worry, severe acute disease (noted in two of my cases), and prolonged mental or physical strain.

The disease may also occur as a secondary complication in the course of simple goiter, affections of the nose, and pregnancy; this variety, however, is to be distinguished from the primary or essential form.

Symptoms.—The development of the characteristic symptoms is generally *gradual*, though it may rarely be *rapid*. In the so-called abortive form the symptoms arise somewhat rapidly, but early subside.

In *acute* Basedow's disease the symptoms consist of an excessively rapid action of the heart, incessant vomiting, purging, and marked exophthalmos, with or without pronounced cerebral symptoms. J. H. Lloyd's case proved fatal after an illness of three days. Schlesinger insists that acute exophthalmic goiter should be suspected in every case of rapid loss of weight.

In the *chronic* form heart-hurry is almost constantly a conspicuous early symptom, and not seldom have I found that it precedes for a long period of time the appearance of the remaining characteristic features (enlargement of the thyroid, exophthalmos, and tremor). The pulse remains at or over 100 beats per minute, and upon unusual exertion or excitement the heart's action becomes violent and irregular, the pulse even reaching 160 or over. Palpitation, often with breathlessness, is a distressing symptom.

Cardiac Physical Signs.—*Inspection* reveals a forcible impulse that is not displaced, though late in the affection it may be much extended in superficial area. The carotids and the abdominal aorta beat violently, and the capillaries and veins of the hands may also pulsate visibly. *Palpation* detects an increased force of the cardiac impulse. The area of *percussion-dulness* may be somewhat increased, as hypertrophy and secondary dilatation supervene. On *auscultation*, blowing murmurs over the heart and great vessels, as well as an increased accentuation of the valvular sounds, may be audible for some distance from the patient. Distinct *bruits* may be heard over the base and manubrium.

Exophthalmos.—Protrusion of the eyeballs is usually present. The degree of exophthalmos varies greatly from time to time in the same case—a fact that points to an increased amount of blood or lymph in the orbit as its cause. In advanced cases permanent prominence of the balls may be attributable to augmentation of the orbital adipose tissue. On closing the eyes a rim of white is visible above and below the cornea; this and Graefe's sign, immobility of the upper lid when the eye is turned downward, are two symptoms of great diagnostic importance. Möbius has called attention to the inability to converge the eyes upon near objects; and Stellwag, to an apparent separation of the eyelids, due to spasm or retraction of the upper lid. The pupils and the vision are unaffected, while the patient winks less often than in health. Slight momentary retraction of the upper eyelids occurs on gazing at some object if the latter be moved rapidly up and down (Kocher). Abnormalities are rarely presented by the optic nerves, and ulceration of the cornea may supervene. The retinal arteries pulsate.

Thyroid enlargement may either accompany or follow the exophthalmos, and has for its cause the great dilatation of the vessels, particularly of the arteries. The enlargement is usually moderate, and may be general or partial, the size of the gland exhibiting sudden variations, since it is dependent upon the circulatory disturbance. *Inspection* may also show visible pulsation; *palpation* feels a thrill, and Kocher states that an important sign is tenderness of the thyroid. *Auscultation* renders audible a double systolic murmur. The latter sign is probably present in most instances, though not constantly.

Nervous Symptoms.—*Muscular tremors* form an early symptom; they are involuntary, and fine in character, numbering about eight to the second (Osler). The characteristic features of *neurasthenia* appear and gradually increase in intensity. *Mental disturbances*, particularly marked depression or great excitability, are common, and even mania (which may prove speedily fatal) or melancholia may be observed.

Cutaneous Symptoms.—The *temperature* may at intervals be moderately elevated, and this symptom may be associated with profuse sweatings. Among other cutaneous phenomena, though these are for the greater part occasional, are *pigmentation* (which, in the case of a physician whom I recently saw suffering from Basedow's disease, was as pronounced as in typical Addison's disease), scleroderma, urticaria, pruritus, and circumscribed solid edema. In the advanced stage malleolar *edema* sets in and may become general. A marked diminution in the cutaneous resistance to the electric current has been noted by Charcot. The forehead is not wrinkled as in health.

General Symptoms.—Muscular weakness, either local or general, is pronounced; the patient becomes anemic and is at last extremely emaciated. An early sign is leukopenia, the neutrophiles being much reduced, while the lymphocytes are twice the normal figure. *Vomiting* and *purging* may appear at different times and gastric achylia is commonly present, and in some cases *hemorrhages* (epistaxis, hemoptysis, hematemesis) tend to supervene. Fatty stools have been observed (Bittorf, Falta). *Albuminuria* and an increased amount of urine, with glycosuria, are among the commoner complications. Louise Bryson has maintained that diminution in the chest-expansion is a characteristic sign of exophthalmic goiter; and Patrick,[1] who examined 40 cases, found that there was an average diminution, but believed it to be proportionate to the amount of muscular weakness. Rarely a *myxedematous* condition is associated; probably the disease is also remotely related to scleroderma.

Diagnosis.—The diagnosis of Graves's disease may be made when tachycardia or delirium cordis and fine, general muscular tremors are present. Exophthalmos and enlargement of the thyroid are often late-appearing symptoms, and are as often temporarily lacking even in fully-developed cases. Rarely, either or both of these signs may be permanently absent. On the other hand, in a few cases exophthalmos is the sole characteristic feature for a long time, though it is eventually followed by an unmistakable symptom-group. Dernini emphasizes temporary increase in the clinical diameters of the heart after exertion, as a diagnostic feature. Parenchymatous goiter presents a non-pulsating tumor.

Course and Prognosis.—The chronic form of the disease endures, as a rule, for a few years. A gradual subsidence of the cardinal symp-

[1] *Deutsche med. Woch.*, Dec. 20, 1894.

toms for a long period has been noted, and in such cases complete recovery may be claimed. In fully-developed cases the prognosis formerly was almost hopeless, but since the introduction of the operative treatment many cases have been greatly benefited, and others cured. The disease assumes a more aggravated form in males than in females. Kocher found that the coagulation process is retarded—the more so, the graver the infection.

Treatment.—This is (*a*) *Hygienic*, (*b*) *Medicinal*, and (*c*) *Operative*.

(*a*) **Hygienic.**—The environment, both physical and mental, should be made as favorable as possible. A change of climate, and especially moderate elevation, in cases not too far advanced, bring about beneficial results. Such elevation (3250 feet) produces a sedative effect upon the nervous state that reacts most favorably upon the circulatory organs, while the purity and tonic quality of the air have a general strengthening and restorative effect (Yeo). Among other promising measures may be mentioned the wet-pack, methodical hydrotherapy with massage, and a continuous galvanic current. The electric treatment should be given a thorough trial over three or four months (Osler). The local use of an ice-bag to the precordium has acted admirably in reducing the heart-hurry in a few cases of my own. Rest in bed for a few weeks at a time, at intervals, is often followed by improvement. The diet should be carefully supervised, according to the indications of special cases.

(*b*) **Medicinal Treatment.**—This is probably secondary to the hygenic and operative measures. In two cases of my own, however, recovery followed the persistent use, for about six months, of the following prescription:

℞. Extr. digitalis, gr. iv (0.259);
Extr. ergotæ (Squibb), ʒss (2.0);
Strychninæ sulph., gr. ss (0.032);
Ferri arseniatis, gr. ij (0.129).
M. et ft. capsulæ No. xxiv.
Sig. One t. i. d. after meals.

In 2 other cases (one, a trained nurse) the use of sodium salicylate (gr. x-0.648—four times a day) was followed by almost total relief. L. Webster Fox, Waller, and others also warmly advocate the latter remedy in this affection. Trachewsky, in Kocher's clinic, found that sodium glycerophosphate (gr. xx—1.296—three or four times a day), had the effect of diminishing the size of the enlarged thyroid glands, and Starr[1] has also found this remedy of great service in several cases. Other therapeutic agents that have been extensively employed, but with doubtful advantage, are aconite, veratrum viride, and belladonna. From all of the clinical testimony at hand I feel convinced that thyroid-feeding is contraindicated in the treatment of Basedow's disease, unless a myxedematous condition be associated, when it may prove efficient. S. Solis-Cohen and others have used extract of suprarenal gland with good results. W. G. Thompson holds that the exacerbations, which are of a toxemic character, may be completely checked by the cytotoxic serum of Rogers, prepared from the diseased human glands through animal inoculation. Krumholz[2] holds that serum of thyroidectomized animals is the most valuable drug yet offered. Antithyroid preparations, such as thyroidotoxin, give promise of good

[1] *Medical News*, April 18, 1896. [2] *Illinois Medical Journal*, March, 1910.

results. Möbius's thyroidin (a preparation of the blood of sheep in which the thyroid gland has been removed some time previously) has given good results. The dose is from 2 to 12 drops daily, given for a period of twenty days. Shattuck advises neutral bromid of quinin, and F. Billings, the hydrobromid (gr. v—.324 four times daily), for its vasoconstricting effects. Lecithin is found useful when digestion is undisturbed, but "it fails without the assistance of a milk diet" (Berkley).

(*c*) **Operative Treatment.**—Starr[1] has collected 190 cases in which some form of operation was performed. Of these, 74 are reported as completely cured, many of them having been watched two to four years before the result was published; 45 of the cases were improved, and 23 died immediately after operation. The symptoms preceding the fatal result are sudden hyperpyrexia, with rapid pulse, nervous distress, sweating, cardiac failure, and collapse. The statistics of Kinnicutt and of Abram[2] (particularly the latter) show less encouraging results from operation. It is to be remembered that under the most favorable circumstances a complete cure will not be attained immediately. In cases unimproved by non-operative treatment in a reasonable time, partial thyroidectomy may also be advised. Bilateral resection of the sympathetic nerve has been done by Schwartz and others with marked benefit. Rehu[3] presents a statistical report of 32 resections of the sympathetic: 31.1 per cent. were cured; 50 per cent. improved, 12.5 per cent. were unimproved, and 9.5 per cent. proved fatal. F. Hartley[4] states that, compared with sympathectomy, partial thyroidectomy yields better results, both as regards mortality and cures. Crile claims benefits from ligation of the thyroid artery, which breaks the nerve supply between the brain and the thyroid gland. Both in acute exophthalmic goiter and during an acute exacerbation of the chronic form operative intervention is contraindicated. Kuh[5] employed the serum-treatment in 11 cases with marked improvement in the subjective condition of the patients. Pfahler noted decided improvement from the *x*-ray treatment in about 75 per cent. of 51 cases. Berger and Schwab also contend that this agent is fully equal to any therapeutic measure.

MYXEDEMA.

(*Hypothyroidism; Sporadic Cretinism.*)

Definition.—A nutritional disorder, consequent upon atrophy and loss of function of the thyroid gland, characterized by a myxedematous infiltration of the subcutaneous tissue and a cretinoid cachexia.

Three varieties occur, as follows: (1) True myxedema; (2) Cretinism (the absence of thyroid function—congenital, or lost during childhood); (3) Operative myxedema, due to total removal of the glands for surgical reasons or in experiments upon lower animals.

Nature of Myxedema Proper of Adults.—Charcot, who gave the name of *cachexie pachydermique* to this disease, believed it to be of trophoneurotic origin. Atrophy of the thyroid is pretty constantly present, and the gland may either be converted into a small fibrous mass or be entirely absent, so that the causal relation between myxedema and functional and structural alterations of the thyroid seems to be conclu-

[1] *Loc. cit.* [2] *American Year-Book of Medicine and Surgery*, 1897.
[3] *Soc. Rep., Münch. med. Woch.*, No. 41, p. 1357, 1899.
[4] *Annals of Surgery*, July, 1905. [5] *Medicine*, September, 1905.

sive. Moreover, the therapeutic test of improvement under the administration of thyroid extract sustains this view. It is probable that the active thyroid supplies some essential secretion which maintains normal metabolism, though this product has not been isolated. Its existence being inferred, however, it has been called *iodothyrin*. Ponfick has pointed out that the hypophysis sometimes shows changes resembling those in the thyroid gland. The fact that in a good many cases of myxedema a considerable portion of the thyroid gland is unaltered and partly capable of functionating arouses a suspicion that the hypophysis may share in the production of this disease. The thymus has been found to be enlarged in myxedema.

Etiology.—The thyroid was destroyed by *actinomycosis* in a case of myxedema reported recently. Myxedema may also be secondary to *exophthalmic goiter*, but it is then, as in the case of a simple acute goiter, only a transient condition. Women are much more frequently affected than men, and a neurotic condition may precede some cases. The disease may affect several members of a family, and hereditary transmission through the mother has been observed. Sisters may suffer, one from myxedema and the other from exophthalmic goiter. Döderlein [1] reports the case of a child born with typical myxedema. Pregnancy may cause a disappearance of the myxedematous symptoms (Osler). The symptoms may reappear after delivery.

Symptoms.—The myxedematous condition is most plainly noted in the *face*, the skin being swollen, but inelastic, rough, dry, and firm. The lines of facial expression are obliterated, and the features are broad, coarse, immobile, and bulky. The *physiognomy* is stupid, dull, and phlegmatic, and simulates imbecility. The *hair* falls out, owing to deficient nutrition; and the general bulk of the body is markedly increased. Pressure does not produce *pitting*, as in true edema. According to Ord, the local tumefaction of the skin and subcutaneous tissue is most frequently prominent in the supraclavicular regions. The *mucous membranes* are also infiltrated, and the teeth may become loosened. The tongue, lips, and nose are thickened, and the voice is monotonous, slow, and has a "leathery tone," "with curious nasal explosions at short intervals during speaking." Bodily movements are slow, and the gait is uncertain on account of disturbed coördination.

Nervous Symptoms.—There is obvious retardation of psycomotor action. Mental perception and thought are also slow, and the memory, while retentive, is slow to respond. Not infrequently there may be considerable irritability, or hebetude alternating with sudden excitability. The patient may become suspicious, and later is subject to delusions and hallucinations; or the apathy may pass into a melancholia, ending at last in dementia. Ord mentions "the aggravation of all symptoms during low climatic temperatures;" and "among the minor or accessory signs may be quoted abnormal subjective sensations, belonging particularly to taste and smell; occipital headache; marked alterations of temper; and a curious persistence of thought and action, overriding all attempts at interruption by friends or observers."

The *temperature* in myxedema is usually more or less subnormal. Albumin and sugar are occasionally found in the urine, but the quantity

[1] *Norsk Magazin for Lægevidenskaben*, Christiana, July 4, 1910.

of nitrogen excreted is small, owing to the diminished metabolism of proteids. *Hemorrhages* from the nose, gums, and bowels may occur. *Ascites* may be present in some cases, and may simulate ovarian tumor. The thyroid is not palpable, partly because of its atrophy, and partly because of the thickened myxedematous tissues of the neck.

The **diagnosis** is not difficult if one bears in mind the characteristic manifestations described above. Myxedema could hardly be mistaken for acute or chronic nephritis in the absence of pitting, etc., as some have supposed. Chapman[1] mentions a solid appearance of the conjunctiva as an early sign of diagnostic value.

The **prognosis** is guardedly favorable in a majority of the cases since the introduction in the treatment of thyroid-feeding. The course of the disease is slow, however, often lasting from five to fifteen years, and death from intercurrent disease is not uncommon. The coagulation time is shortened; markedly so in grave cases.

Treatment.—Until the advent of thyroid-feeding the treatment of myxedema was palliative, and usually unsuccessful.

A warm and equable climate is very desirable, owing to the subnormal temperature from which the patients frequently suffer. The various warm baths—as the Turkish, Russian, and electric—should be employed for the same reason. Pilocarpin has been recommended, and strychnin and arsenic have been administered for their tonic effect.

Since the brilliant results obtained by Murray, however, the internal use of the thyroid gland of sheep or calves has come into a well-deserved favor in the treatment of all cases of myxedema, whether of the so-called true form, of sporadic cretinism, or of the cachexia strumipriva. The gland may be given raw or cooked, in the form of the glycerin extract, or in the powdered extract; the last named is sometimes put into tabloid form. If cooked, the gland should be only partially "done." The fresh thyroid is minced and often spread on bread, and from one-quarter to one-half a gland may be taken daily.

If used for hypodermic injection, to a dram (4.0) of the glycerin extract is added half a dram (2.0) of a 1 per cent. solution of carbolic acid in distilled water, of which mixture from 10 to 15 minims (0.66–1.0) may be injected three or four times a week.[2]

It is safest—for reasons that will be pointed out below—to begin with quite small doses, and gradually increase, especially if there is much gastric irritation. Not more than 5 minims (0.333) of the glycerin extract should be given at the start. This dose may be increased gradually until 15 or 20 minims (1.0–1.33) are taken three times daily. From 3 to 5 grains (0.194–0.324) of the powdered gland or tabloid form will be a safe commencing dose in adult myxedema: a caution, however, is necessary regarding the various manufactured preparations of the thyroid gland, some of which are impure and even dangerous.

The toleration of thyroid-feeding does not depend upon the volume, but upon the functional activity, of the gland, and this fact, together with the evidences of toxic action reported in some instances of the administration of thyroids to a maximum degree, make it important to urge again—as intimated above—the necessity of small dosage at the beginning of treatment, and the most careful and judicious increase in

[1] *Lancet*, Sept. 30, 1899. [2] Osler, in the *Amer. Text-Book of Therapeutics*, pp. 926, 927.

the quantity given. The additional fact of an occasional cumulative action should also be emphasized. Should vomiting, renal pain, tachycardia, suffusion of the face, syncope, vertigo, or marked headache supervene, the remedy should be stopped at once. The treatment may be resumed again cautiously, alternating with intervals of cessation. I have observed that by combining arsenic with any of the preparations of thyroid the toxic effects of the latter can be largely obviated. Good results are obtained usually within a month, though it is probable that even after all the symptoms have subsided the treatment may have to be continued at intervals. The activity of the thyroid is enhanced by the presence of iodin (Hunt and Seidell).

Cretinism, Sporadic and Endemic.—Here there is a congenital atrophy or absence of the thyroid gland, or an enlargement by the growth of fibrous tissue at the expense of the glandular elements. Cretinism may also develop in early infancy. The patients are often the children of parents having various neuroses and goiter, and syphilis has also been supposed to have a causative influence. Congenital myxedema is quite common only in regions where goiter is endemic. A marked sporadic case has been in the Philadelphia Hospital for many years.

Symptoms.—Cretins are dwarfs with large heads and faces, thick lips, thick protruding tongues, broad bodies and members, and prominent abdomens. The subcutaneous tissues are myxedematous. Umbilical hernia is often present. The mental condition is that of idiocy, and physical growth is retarded and slow. Speech is unintelligible or nearly so, and the voice harsh. Walking may never be accomplished, and is always slowly developed. There is anemia, the blood being of a fetal type. Rheumatic symptoms sometimes occur.

Prognosis.—The disease is progressive until about the fifteenth year in those cases developing during early childhood. Congenital cases usually die shortly after birth. At the twentieth or thirtieth year "the mental and physical characters are those of childhood."

Treatment.—Thyroid-feeding has been followed by beneficial results, the checked growth having recommenced and the cretinic aspect having been largely lost. S. Küh [1] has employed iodothyrin in one case with quite as satisfactory results as those from the dried thyroid. Pahr [2] has implanted a portion of the thyroid gland of the mother into the spleen with manifest amelioration of the mental state of the child.

Operative Myxedema, or Cachexia Strumipriva.—Extirpation of the thyroid for surgical reasons has given rise to the gradual production of symptoms and conditions identical either with true myxedema or with the cretinoid state. Partial removal of the gland is not followed by cachexia strumipriva, nor is complete thyroidectomy when accessory glands are present elsewhere.

The administration of raw or broiled thyroids, or of their various extracts or preparations, must also be employed in this form of myxedema, and should be continued throughout the rest of the patient's life, perhaps with intervals of withdrawal of the feeding until the improvement gained begins to lapse.

[1] *Philadelphia Medical Journal*, April 8, 1899. [2] *La Bulletin Medical*, June 13, 1906.

DISEASES OF THE PARATHYROID GLANDS.

The parathyroid bodies consists, as a rule, of two pairs, upper and lower, on either side of the lateral lobes (posterior inner edge) of the thyroid gland. These are small ovoid structures from 1 to 3 mm. in diameter and 6 to 8 mm. in length. Recent studies by Halsted and others show that their removal in animals gives rise to symptoms simulating tetany (*vide* p. 1221), which disappear as a result of parathyroid feeding or transplantation. MacCallum has shown that the parathyroid bodies control calcium metabolism; they also, to some extent, influence carbohydrate metabolism. While parathyroid preparations have been used with success in tetany, they have no therapeutic effect in diseases of the thyroid gland, convulsive disorders, and the like.

PART V.

DISEASES OF THE RESPIRATORY SYSTEM.

I. DISEASES OF THE NOSE.

ACUTE RHINITIS.

(*Acute Nasal Catarrh; Acute Coryza.*)

Definition.—An acute catarrh of the Schneiderian membrane, sometimes tending to involve the adjacent sinuses and passages. It is known to the laity as "cold in the head."

Etiology.—Its most conspicuous cause is exposure to draughts of air and to the influence of the atmospheric vicissitudes that are especially prevalent during the winter and spring seasons. It often results from the inhalation of irritants (physical, chemical, or biological). It may also display epidemic behavior, and this fact points strongly to its microbic origin. Hence local disturbances of the circulation due to exposure are to be regarded as the accidental means of preparing the soil for bacterial invasion. Acute rhinitis may be also secondary to, or propagated from, inflammations of the faucial mucosa by contiguity.

Symptoms.—Sensations of *chilliness*, succeeded by *feverishness* (the temperature reaching 100° to 101° F.; 37.7°–38.3° C.), frequent *sneezing*, *headache*, and a feeling of general ill-health are among the prominent features that attend the development of coryza. Pains in the extremities and back tend to appear only in severe cases. The pulse is frequent, the skin dry and unduly warm, thirst is increased, while the appetite is impaired, and constipation often attends. The *nasal mucosa* is swollen, and thus interferes both with the nasal respiration and the senses of smell and taste; its color is deepened, its surface covered at first with opaque mucus, and later with a muco-purulent secretion. Among early symptoms is the discharge of a watery, irritating secretion from the nares and a maceration of the epidermis, with resulting abrasions. On account of the swelling of the mucosa of the lacrymal ducts the tears flow down over the cheeks. Adjacent mucous surfaces may become involved, giving rise to conjunctivitis, catarrhal pharyngitis, laryngitis, and finally, in the severer types, bronchitis. Naso-labial herpes is not uncommon. As the affection progresses the secretion becomes more abundant and turbid and more or less pyoid. The symptoms due to extension of the catarrhal inflammation vary with the organs or structures involved. The disease runs its *course* within five or six days, but

the nasal discharge, which gradually diminishes, usually persists for a few days longer.

Diagnosis.—In the presence of the above-mentioned symptoms the disease is readily recognized. In well-marked cases the possibility that an infectious disease may be developing, the beginning of which is characterized by nasal catarrh (measles), is to be recollected.

Prognosis.—Except in neglected cases, which result in bronchitis, and occur at one or other extreme of life, the disease is free from danger. The nursing infant may have to be fed with a spoon temporarily.

Treatment.—At the outset a purge, consisting of calomel (gr. ij—0.129), or a pill of blue mass (gr. v—0.324) at night, followed by a Seidlitz powder in the morning, is advisable. To children a dose of castor oil may be given. The early administration of a diaphoretic, such as Dover's powder (gr. v–x—0.324–0.648) at night may arrest the complaint, and quinin in a large dose (gr. xij–xv—0.777–0.972) at night, or hexamethylenamine (gr. v–0.324, q. d.), may cut short the course of the disease. When the above-mentioned abortive measures fail, the following tablet produces good results:

℞. Quinin. sulphat., gr. ijss (0.162);
Fluidext. belladonnæ, ♏jss (0.099);
Sodii salicylatis, gr. xxx (1.944);
Camphoræ, gr. ijss (0.162).
M. et ft. tablet No. x.
Sig. One tablet every hour or two.

For the fever aconite may be employed, and, if the throat be involved, bryonia may be given in conjunction.

Local Treatment.—This aims at soothing as well as at reducing the swelling of the Schneiderian membrane. The compound tincture of benzoin forms a soothing inhalation (℥ij to a pint—8.0 per half liter—of water) when raised nearly to the boiling-point; the vapor is inhaled for ten or fifteen minutes at a time. With a view to reducing the swelling a solution of cocain (strength 2 to 4 per cent.) may be temporarily used; Mackenzie recommends this admirable combination:

Menthol, gr. v (0.324);
Pinol ♏v (0.324);
Benzoinol, f℥j (32.0).

In severe cases the patient should be kept in-doors and in an atmosphere of even temperature.

CHRONIC RHINITIS.

(*Chronic Nasal Catarrh.*)

Two forms are recognized, the hypertrophic and atrophic, and these, though, as a rule, occurring separately, may be found in combination.

Pathology.—The morbid changes in hypertrophic rhinitis consist in an enlargement of the lower turbinated processes, together with redness and swelling of the nasal mucosa that may be general or limited

either to the anterior or posterior nares. As the disease progresses the thickening of the membrane increases, until it finally encroaches upon the nasal chambers at every point. In addition to the nasal obstruction there is a hypersecretion of mucus. Opposite changes occur in atrophic rhinitis, such as thinning or atrophy of all the structures, with enlargement of the nasal cavities. The nasal mucosa is coated with thick, yellowish-green, decomposing crusts, which emit a characteristically fetid odor, and the frontal, ethmoid, or other accessory sinuses may, by an extension of the inflammation from the nasal chambers, be invaded by mucopurulent inflammation. The atrophic process does not affect the glandular structures of the upper third of the nose, and this fact explains the most unpleasant feature of the affection—namely, the horrible secretion.

Etiology.—Frequently occurring attacks of acute rhinitis may produce the chronic form, and syphilis and, less commonly, tuberculosis are also among its causes. Abel[1] regards atrophic rhinitis as infectious, claiming that the cause is the *bacillus mucosis ozenæ*, which resembles closely the pneumobacillus, but is distinguishable from it.

Symptoms.—(*a*) In the *hypertrophic* form nasal respiration is impeded, owing to the hypertrophy of the turbinated bodies. The sense of smell is impaired, and there is a discharge of secretion from the nares, particularly the posterior, inducing "hawking." The diagnosis is set at rest by a rhinoscopic inspection of the parts. While this is a common affection everywhere, it is wellnigh universal in this country.

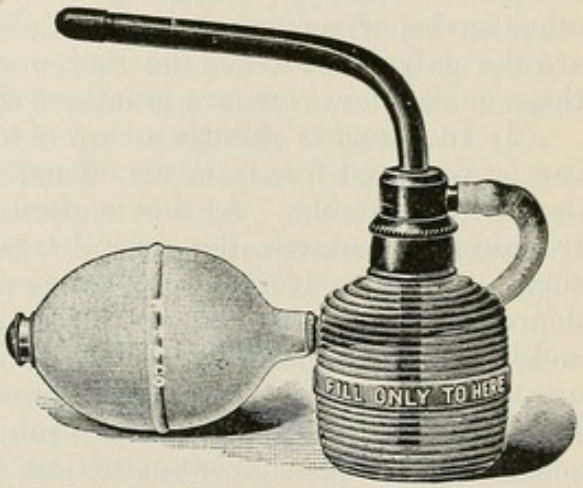


FIG. 42.—Apparatus for cleansing the nasal passages in chronic rhinitis.

(*b*) In chronic *atrophic* catarrh there is some degree of nasal obstruction, occasioned by the presence of the thick crust, but the most conspicuous symptom is the disgusting odor, which makes the patient repellent in society. The sense of smell is lacking. After cleansing the membrane the rhinoscope will show the nasal chambers to be unduly capacious.

Treatment.—(1) **Chronic Hypertrophic Rhinitis.**—The treatment is divisible into *general* and *local*. The physician should procure an environment for his charge most favorable for promoting the general nutrition, which is often below the health-standard. The selection of a suitable climate, then, forms an important part of the management, and a residence in a locality that possesses a mild, equable, comparatively dry and pure atmosphere is to be advised and encouraged. Various tonics may then be demanded by the general condition of the patient, and strychnin and electricity are useful in restoring the loss of power in the contractile elements of the intercellular walls.

Local measures are employed to facilitate thorough cleanliness and

[1] *Zeit. f. Hyg. u. Infektionskrank.*, Bd. xxi. H. 1.

disinfection of the affected parts, though in incipient and mild cases energetic treatment is scarcely needful. The best method of cleansing the nasal passages is by means of the coarse spray (Fig. 42). The apparatus of Lefferts is also to be employed when the secretion is inspissated or tightly adherent. An excellent combination for use in this manner is the following:

℞. Sodii biborat.,
Sodii bicarb., *āā.* ʒj (4.0);
Phenolis, gr. viij (0.518);
Listerin., ℥j (32.0);
Aquæ destillat., q. s. ad ℥iv (128.0).—M.
Sig. Use as a spray three times daily.

It is often desirable to use warm or even hot liquids, in which case the application is made by the use of the anterior and posterior nasal syringe. Powders are harmful, and, as the nasal douche is dangerous in unskilled hands, these should both be abandoned.

In hypertrophic rhinitis the obstruction to nasal breathing is to be removed, and to accomplish this caustics (chromic, glacial acetic, and nitric acids) are used, of which the most efficacious is chromic acid. This should be applied by means of a pointed glass rod, the application being followed by a sloughing away of the diseased tissues. Among other modes of removing the nasal obstruction that may be mentioned are the galvano-cautery, the thermo-cautery, and the cold-wire snare; these modes, however, are practised chiefly by the specialist.

(2) In **atrophic rhinitis** a cure is to be despaired of, but the patient can be rendered free from the offensive discharge, and hence to a great degree comfortable. As this is often but an advanced stage of hypertrophic nasal catarrh, the general treatment is similarly directed: it is therefore well to overcome, as far as possible, by a mental stimulus, the depressed mental state due to the fetor. If the diathesis be tuberculous, cod-liver oil, iron, arsenic, and strychnin, together with a generous diet, are to be advised. If syphilis is associated, appropriate measures must be instituted. Moreover, since a subject of atrophic rhinitis is a fertile source of atmospheric contamination, his living and sleeping apartments must be highly ventilated.

Local Measures.—An antiseptic spray of Seiler's or Dobell's solution, and oiling the nasal cavities, are measures to be first tried. If they prove non-efficacious, the crusts may then be removed with a cotton applicator coated with a solution of hydrogen peroxid. We may then use a spray of liquid albolene and menthol; this serves not only to lubricate, but to supply moisture, both of which are important therapeutic indications. Small ulcerations occur in this affection and induce oft-repeated epistaxis; consequently, an attempt should be made to heal the latter and to obtain an even, moist surface. To accomplish this the method of Clarence C. Rice may be followed—*i. e.* to rub the ulcerations thoroughly by means of a cotton-carrier with a small hard pledget of cotton moistened with listerin or borolyptol for a few seconds at a time. These antiseptic frictions are made at intervals of two or three days for two or three weeks.

AUTUMNAL CATARRH.

(Hay Asthma; Hay Fever.)

By this term is meant a form of asthma that occurs exclusively during the warm season (spring and late summer, particularly).

Etiology.—The direct causes are the odorous principles given off from certain plants (the pollen of the *Anthoxanthum odoratum*, of the rose, etc.), and inorganic dusts of various sorts. In some instances it appears to arise without obvious exposure to a special irritant—for example, it may be excited by strong emotional disturbance. Kyle[1] has advanced the theory, based on experimental studies, that the disease is caused by chemic changes of the mouth secretions.

Predisposing Factors.—The male *sex* suffers more frequently than the female. *Age* has a slight though decisive influence, more than 33 per cent. of the cases occurring before the twentieth year. Often some *abnormality of the nasal passages* (*e. g.* a polypoid tumor, hypertrophy of the mucosa, a deflected septum) acts as a source of reflex irritation. *Heredity* is among the potent contributing causes. The inhabitants of cities are more liable than those in rural districts, though the air of agricultural regions intensifies the condition. Perfect *immunity* is enjoyed by the dwellers in certain climates—chiefly mountainous and marine. Perfect health probably confers immunity.

Symptoms.—The symptoms are (*a*) local and (*b*) general.

(*a*) **Local.**—Hay fever has an *abrupt onset*, and the attacks return annually at or about the same time. The *invasion* is marked by pronounced coryzal symptoms, with much sneezing, stoppage of the nasal passages, copious rhinorrhea, the discharge being thin and watery as a rule, and rarely mucopurulent. Suffusion of the eyes, with itching of the lids and free lacrymation are constant features; the decided itching sensation of the palate and pharynx is also at times a very distressing symptom. The sense of smell may be lost, and taste and hearing are often impaired.

The *course* as regards the local symptoms is marked by alternate amelioration and aggravation of the symptoms, the exacerbations being due to exposure to the open air, especially in changeable weather. Later the catarrhal process invades the bronchi, and cough and asthmatic seizures appear, these often becoming very distressing.

(*b*) **General disturbances** comprise subjective sensations—anorexia, insomnia, lassitude, and chilliness alternating with slight feverishness.

The *course* is from four to six weeks, and cases that develop in August are terminated by the occurrence of a decided frost. Wyman also describes the "rose cold," which comes on in the spring.

Diagnosis.—The recognition of hay asthma is unattended with difficulty, provided that such facts as the time of its occurrence (June) and its annual periodicity are carefully noted. The sudden onset of severe coryza-like symptoms in a neurotic person, particularly in the month of August, should direct attention to autumnal catarrh.

Prognosis.—This is favorable both as to life and length of days, though a permanent cure is a rare event unless permanent removal from the influence of the specific causes can be effected.

Treatment.—Whenever possible the patient should travel till he

[1] *Jour. Amer. Med. Assoc.*, October 1, 1904.

finds a locality in which he ceases to suffer, and subsequently he should there spend the period of annual attack, and by these means escape the exciting causes. The Adirondacks and White Mountains usually bestow immunity. If the patient cannot make the necessary change, the general nutrition is to be improved by hygienic means and the use of such measures as phosphorus, strychnin, quinin, and arsenic.

The local symptoms demand the topical application of various agents to the nasal chambers, such as cocain hydrochlorate solution (1 per cent.); if applied on cotton with a probe, followed by a 4 per cent. solution of antipyrin, the palliative effect is prolonged (Gleason). Beaudoux recommends destruction of the nerve supply to sensitive areas. Hollopeter has had good success by daily *sterilizing* the nasal chambers by means of Dobell's solution used first with an atomizer; then swabbed thoroughly over the naso-pharynx. The membrane is then dried and the nose loosely plugged with cotton saturated with a mild solution of menthol in albolene. Of 1240 patients who used serum-therapy (Pollantin) correctly, 56.1 per cent. either remained free from attacks, or could abort attacks already started (Dunbar). Atropin allays the irritability of the mucous membrane involved and diminishes the rhinorrhea, thus mitigating the constitutional disturbances and sometimes relieving the asthmatic paroxysms. When given internally the dose should not exceed gr. $\frac{1}{300}$ (0.0002), to be repeated every hour till dryness of the throat appears.

My best results have been derived from the hypodermic use of this drug (gr. $\frac{1}{200}$—0.0003) at intervals of three to four hours till the desired effect is produced. Gleason obtained satisfactory results from nitro-muriatic acid freshly prepared (dose ♏v t. i. d. after meals in a tumbler half full of water). Recently the internal use of 5-grain tabloids of suprarenal extract has met with success in the hands of S. Solis-Cohen and others. Adrenalin chlorid applied locally has given good results.

EPISTAXIS.

(*Nose-bleed.*)

Etiology.—The causes of nose-bleed are various, and a convenient grouping is the following: (*a*) Affections of the nasal mucosa (*e. g.* ulcer, polypi, intense hyperemia). (*b*) Injuries, either external, as from a blow, or internal, as from plugging with a foreign body, nose-picking, etc. In this category may also be included epistaxis due to fracture at the base of the skull. (*c*) Acute infectious fevers, particularly typhoid (at the onset) and influenza. (*d*) Chronic affections, such as pernicious anemia, leukemia, and hemophilia. (*e*) Vicarious menstruation. (*f*) Rarefaction of the air. (*g*) Plethora; here may be mentioned cerebro-congestion with intense headache. (*h*) Severe over-exertion. (*i*) Frequent epistaxis may be caused by arterio-sclerosis even in the earlier stage while yet amenable to treatment. (*j*) Chronic interstitial nephritis; mitral disease.

Symptoms.—Except when due to traumatism the blood usually drops slowly from one and occasionally from both nostrils. Rarely, the blood may flow as a continuous stream or the nares may present a projecting coagulum. The blood may also gravitate into the pharynx and

be coughed up, or it may be swallowed and vomited. A rhinoscopic examination often reveals the source in cases in which a previous diagnosis of hemoptysis or hematemesis has been made.

The immediate results of nose-bleed are weakness and a moderate anemia, but these are not prolonged, as a rule. Cases arising from fracture at the base of the skull will generally prove fatal.

Treatment.—A careful search for a local cause is especially demanded in cases in which there are frequently recurring attacks. In most cases a spontaneous arrest occurs, but if not, a resort to simple household measures, such as the application of ice to the nose or to the back of the neck, holding the hands up, or the injection of very cold or very hot water into the nares, are to be encouraged. Various astringents (tannic acid, acetate of lead, alum, zinc) may be employed, and a saturated solution of antipyrin is also highly praised. Adrenalin chlorid is valuable. When an ulcerated bleeding point can be reached, there may be applied to it a solution of chromic acid or it may be cauterized by solid silver nitrate. Prolonged pressure applied upon the facial artery as it passes over the inferior maxilla may be efficacious. A solution of gelatin may be injected into the nostril. I have little confidence in internal astringent remedies. The oil of erigeron, administered in large doses, has seemed to do good in a few of my own cases, but in obstinate cases the posterior nares should be plugged, preferably with gauze lubricated with oil or petrolatum, to avoid recurrence when packing is removed. Tincture of aconite or nitroglycerin may be used in arterio-sclerotic conditions.

II. DISEASES OF THE LARYNX.

ACUTE CATARRHAL LARYNGITIS.

(*Acute Endolaryngitis.*)

Definition.—An acute catarrhal inflammation of the larynx, characterized by cough, hoarseness, and painful deglutition.

Pathology.—The anatomic changes present during life are all lacking *post mortem.*

Etiology.—Acute laryngitis may be a primary affection—and particularly *laryngitis sicca* (Molinie)—but oftener it is associated with and secondary to catarrh of the nose and nasopharynx. Wright attributes laryngitis sicca to the coccus of Löwenburg.

Catarrhal laryngitis has for its chief direct causes traumatism, exposure to cold and dampness, the inhalation of irritating vapors or gases, rheumatism (rarely), and the corrosive effect of certain poisons and hot fluids. A certain degree of predisposition is engendered by immoderate smoking, particularly by the cigaret-habit, and by the use of concentrated alcoholic drinks. These agencies induce hyperemia of the laryngeal mucosa, which is easily converted into active inflammation. Acute laryngitis is often associated with acute infectious diseases.

Symptoms.—There are two conspicuous symptoms—*alteration in the voice* (hoarseness) and *cough.* At first there is merely a huskiness

of the voice, but later there may be pronounced hoarseness or even complete aphonia. The cough is dry and characteristically painful until secretion is free. In the early stages the patient complains of sensations of tickling or the presence of some small object in the larynx, causing a frequent desire to clear the throat. In severe instances deglutition is painful. Edema of the larynx may tend to supervene and cause intense dyspnea, with a feeling of distressing oppression. There is, as a rule, a slightly elevated temperature.

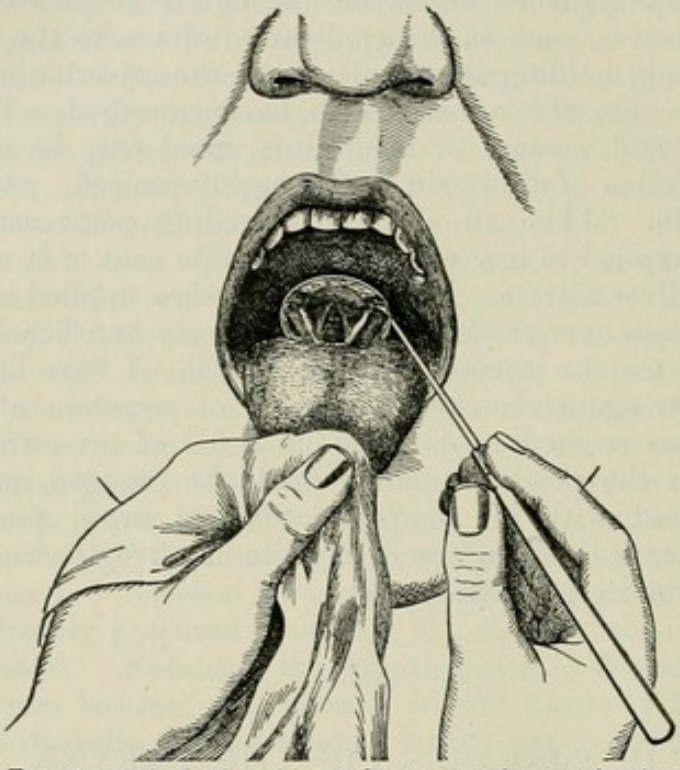


FIG. 43.—Method of making a laryngoscopic examination.

The patient is placed in front of the operator, on an arm-chair, with the back of the chair high enough to afford his head a comfortable rest, and with the source of the light over the right shoulder. The operator then adjusts the head-mirror (the fixed apparatus), warms the throat-mirror over a light sufficiently to prevent the moisture of the breath from being deposited upon it, and touches the hand with the mirror before passing it into the mouth, so as not to use it too hot. The patient's tongue is then protruded, and by means of a napkin is seized between the thumb and the fore-finger and drawn well forward to lay the fauces open to observation. The throat-mirror is then held in the right hand in the same way as one holds a pen. "Finally, it is introduced into the mouth, its handle being inclined downward and outward, its base being parallel with the dorsum of the tongue; it is then passed backward without altering this relation until the edge of the mirror nearly touches the soft palate, the shaft of the mirror in this movement striking the angle of the mouth as a resting-place and fulcrum. The subsequent movement consists in turning the mirror by twisting its shaft between the fingers until it is inclined at an angle of 45 degrees to the line of vision: then it is carried backward and downward until the uvula rests upon its posterior surface, when it is lifted boldly upward and backward until its lower edge comes entirely into view again and rests firmly against the posterior wall of the pharynx. The patient should then be directed to sound in a somewhat high key '*a*,' which lifts the larynx and at the same time the epiglottis, and exposes and brings into view the laryngeal cavity" (Bosworth).

It is important that the mirror itself should be kept in the median line, with its plane always at right angles with the field of vision, as shown in the illustration. In making a laryngoscopic examination we note any abnormalities of color-appearance (the natural being a rose-pinkish tint), of the outline of the different parts, and the deviations from the symmetrical movements of cords, if any, etc.

The laryngeal mirror brings to view a characteristic picture—a swollen, tumefied, and reddened mucosa. These changes affect the vocal cords (whose pearly-white appearance is now lacking) and the ary-epiglottidean folds. It is usual to note also redness and swelling of the epiglottis above and of the trachea below. After secretion has occurred a mucoid covering in streaks or patches is noticeable.

Diagnosis.—This is easy in the presence of marked hoarseness, dry cough, and the image afforded by the laryngeal mirror (Fig. 43). In very early life the larynx cannot be successfully examined; still, *laryngismus stridulus* (owing to the absence of fever, coryza, etc.) could hardly be mistaken, as has been supposed, for acute catarrhal laryngitis. The same is true of *membranous laryngitis*, if we bear in mind the characteristic local features and the more intense constitutional disturbances of the affection.

Treatment.—The physician must enjoin against the use of the voice. The very young and the aged should, in severe or even moderate cases, be kept in bed, and should occupy a single apartment in which the atmosphere is uniformly moist and warm, the temperature ranging from 75° to 80° F. (23.8°–26.6° C.). Inhalations of moist air or steam are of great service, and I have long been in the habit of recommending the following simple apparatus and method of carrying out this mode of treatment: An ordinary tin cup, small pitcher, or other vessel is filled with boiling water to which 1 or 2 drams (4.0–8.0) of the compound tincture of benzoin have been added; the steam is then collected by inverting over the vessel an ordinary funnel. The patient is allowed to inhale the steam by placing the mouth over the narrow neck of the funnel above, or a piece of rubber tubing may be attached to the end of the funnel that is uppermost.

Steam atomizers admirably meet the necessities of the case; and in the case of children the vapor of benzoin, eucalyptol, and other equally sedative and stimulating substances may be diffused in the air of the sick-room. Concentrated solutions or insufflations of powders are not without harmful influence, and neither the cotton-carrier nor the mop should be allowed to enter the larynx in this affection. The external application of the ice-bag or cold compress tends to mitigate the inflammatory process and to obviate spasm.

The *general* treatment differs with the special stages of the complaint. If the case is seen early, a full dose of quinin (gr. xij–xvj—0.777–1.036) may serve to successfully abort the attack, and, in conjunction Dover's powder (gr. v–x—0.324–0.648) may be prescribed. Codein sulphate may be given at prolonged intervals during the attack, and frequently at night, to allay cough; this remedy may be combined with ipecac, aconite, and liquor ammonii acetatis to facilitate secretion and render the cough humid. If we except the abortive measures, the constitutional is wholly inferior to the topical treatment of this variety, though the existence of any particular diathesis may require special internal remedies.

CHRONIC LARYNGITIS.

(*Chronic Endolaryngitis.*)

Pathology.—The laryngeal mucosa is thickened and somewhat reddened, and erosions amounting to superficial ulcerations are rarely seen. A prominence of the mucous glands, especially of the ventricles and epiglottis, is noticeable. Fine villous projections from, and nod-

ular swellings in, the vocal cords are among the rarer morbid changes. Minute vesicles may arise upon the surface (*herpetic laryngitis*).

Etiology.—Oft-repeated acute attacks frequently cause chronic laryngitis, and the long-continued use of the voice (as in public speaking or singing), the inhalation of an atmosphere laden with mildly irritating impurities (tobacco smoke, etc.), and an immoderate indulgence in alcoholic stimulants, respectively or unitedly, predispose to, if they do not excite, the disorder.

Symptoms.—As in the acute form, *hoarseness* and *cough* are the two especially prominent symptoms. The former may be so slight as to present merely a rough tone, or it may involve an almost total loss of voice. The cough shows similar variations in severity, sometimes consisting of a short hack, and again occurring in spasmodic and ringing paroxysms, due to a sense of tickling in the larynx. There may be a small amount of mucous or muco-purulent expectoration, but for prolonged periods the cough may be dry and ineffectual. *Local pain* and discomfort sometimes supervene, and are excited generally by attempts at speaking or singing—events that aggravate all the other symptoms. To complete the diagnosis, the laryngeal mirror is required to show a swollen and slightly red membrane, with a distention of the mucous glands in the immediate vicinity of the epiglottis and ventricles, and occasionally superficial erosions.

Prognosis.—This is unpromising as to complete recovery, although it presents no grave dangers. It is incurable in those instances in which the causal influences cannot be removed, and in all cases in which the patient fails to lend hearty co-operation.

Treatment.—This is (*a*) *hygienic* and (*b*) *medicinal*. (*a*) The sanitary measures embrace preventives that are directed to the removal of all the etiologic factors, whether merely predisposing or exciting. The voice demands rest and the prohibition of smoking and the use of alcoholics in excess, and the patient must also avoid the close, contaminated air of the crowded hall, theatre, and like places. In addition, a tonic regimen, with a view to energizing the nutritive processes, is to be encouraged. In many instances the environment is best arranged with reference to the commonly associated conditions—especially the morbid processes in the nasal and naso-pharyngeal cavities. "A sea-voyage or residence at the sea-shore is, in the large majority of instances, productive of good, and the effects of surf-bathing are often magic" (Mackenzie). My own practice has been to send subjects of chronic laryngitis to pine-forest resorts at low elevations that afford a pure, equable, and somewhat stimulating atmosphere, and I have found that in many cases the selection of a proper climate constitutes the most important part of the treatment. (*b*) The *medicinal* treatment is both local and general. The latter should include creasote, cod-liver oil, and other tonics. Expectorants are of little if any value. The *local* measures, however, are important. Moderate exposure of the neck and daily ablution with cold water are to be advised, and attention to the nose and naso-pharyngeal cavity is of prime importance.[1]

A long list of applications to the larynx from within, including local astringents, disinfectants, and alcoholics, might be enumerated. Of

[1] J. C. Wilson's *American Text-book of Applied Therapeutics*, p. 791.

astringent solutions, however, the best are tannic acid (1–2 per cent.) or alum (.5–1 per cent.) and zinc sulphate (3–5 per cent.). These may be sprayed into the larynx by means of a compressed-air machine with spraying-tubes, although all of the different kinds of inhaling apparatus more commonly used will answer the purpose. If the ordinary hand-atomizer be used, the patient should be taught to draw the vapor into the larynx by gentle and frequent acts of respiration. Disinfectants, such as creasote, potassium chlorate (the latter if ulcerations be present) in solutions of suitable strength, may be used in like manner. I can confidently advise as useful alteratives both iodin and silver nitrate, commencing with a weak solution of the latter (gr. v–ʒj—0.324–4.0), and the strength being gradually increased until the maximum strength that can be endured without distress is reached (gr. xx–ʒij—1.296–8.0). These topical applications should be made directly with a cotton-carrier or brush at intervals of three or four days, preceded by the use of a cleansing spray. The many astringent and sedative lozenges found in the market are only slightly palliative in their effects, and their prolonged use tends to excite gastric disturbance. I am unalterably opposed to the insufflation of powders, believing that they are capable of augmenting the laryngeal irritation and of adding fresh irritation in adjacent parts, particularly in the tracheo-bronchial tract.

SPASMODIC LARYNGITIS.

(*Laryngismus Stridulus; False Croup.*)

Definition.—An affection peculiar to children, chiefly of nervous origin, though also, according to Strümpell and others, often associated with acute catarrhal laryngitis.

Etiology.—The affection is almost solely limited to children between six months and five or more years of age. It is sometimes excited by strong passion or emotion, and it may be associated with tetany. Rachitic subjects are peculiarly liable. The causes of spasmodic croup are in great part those of acute laryngitis.

The mode of action of the direct causes is unknown, but the spasm of the adductors that causes the urgent dyspnea is probably reflex and due to peripheral irritation.

Symptoms.—Two clinical varieties are to be distinguished: (1) That in which the larynx is free from catarrhal inflammation, or the *purely nervous type.* This is especially characterized by sudden brief attacks of dyspnea, either by day or night (often on awakening), that terminate in a high-pitched crowing inspiration ("child-crowing"). The face during the spasm is cyanotic. General convulsions have been noted, but there is neither cough, fever, nor hoarseness. The attacks may be frequently repeated within a single day.

(2) Spasm of the larynx, *associated with mild catarrhal laryngitis.* The attacks generally begin suddenly, about midnight or toward morning on awakening from a sound sleep. Positive evidence of the affection is afforded by the croupy, ringing cough, combined with the hard, stridulous breathing. An approaching spasm may be announced by a harsh

cough and slightly stridulous breathing in the sleeping child. During the attack the countenance may be cyanotic and the breathing most distressing, but these and the above-mentioned severer symptoms generally cease abruptly in an hour or two, and the child resumes its slumber. In my experience the attacks have been repeated for two or three nights in succession, and rarely oftener except in the severest cases. Not infrequently the child manifests the symptoms of mild catarrhal laryngitis between the attacks. A brassy, croupy cough may also attend.

Diagnosis.—*Membranous laryngitis* may be mistaken for spasmodic croup. The development of the dyspnea, however, is more gradual, is without intermission, and without relation to the period of the day. Albuminuria and a false membrane in the throat or nares are usually present in *laryngeal diphtheria*.

Prognosis.—Although the appearance of a paroxysm is alarming, the disease is practically free from danger.

Treatment.—1. The treatment of laryngismus stridulus is quite similar to that of infantile convulsions. A warm bath at a temperature

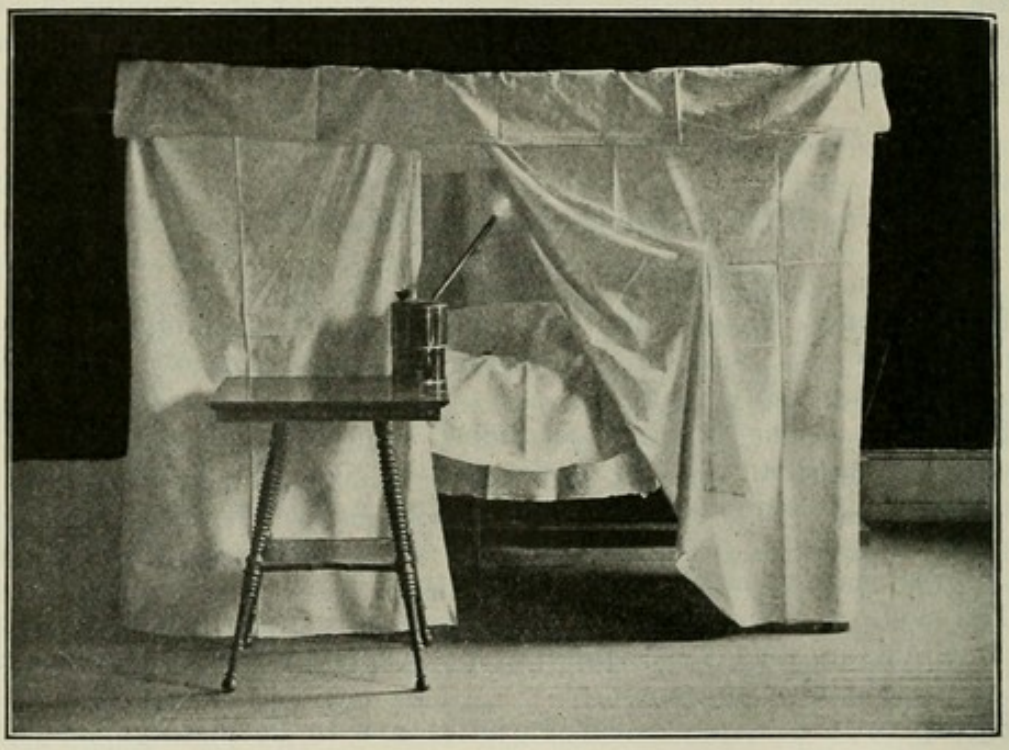


FIG. 44.—Croup-kettle in use.

Four upright rods (5.7 inches in length) are fastened to the legs of the bedstead by a wire or string. Two side-rods are tied on the uprights, and two end-rods (length dependent on width of bed) rest upon the side-rods, These rods form a complete framework for the sheets to hang upon. Four sheets are required (11–4 size)—three to cover the ends and sides, and one to be placed on top. One side should be completely closed, while the opposite is to be left open for ventilation or to be adjusted according to circumstances.

of 98° to 105° F. (36.4°–40.5° C.) is the best means of breaking up the spasm. While in the bath cold sponging of the back and chest is serviceable. The finger may be passed into the fauces, and should the epiglottis "become wedged in the chink of the glottis, it must be released by the finger." After the attack active treatment should be directed at the discoverable causes, and I have been in the habit of giving

small doses of the bromids thrice daily, together with warm cod-liver oil inunctions, with striking effect.

2. In spasmodic croup an emetic is to be given at once, the best being a mixture of alum and syrup of ipecac, of which the dose is ʒj (4.0), to be followed by irritation of the fauces with the finger in order to facilitate emesis. In severe paroxysms a hot bath may be given to aid the emetic. In case the dyspnea is not checked by the above measures, chloral hydrate may be exhibited by enema (gr. ij–v; 0.129–0.324) or a whiff of chloroform may be given. The local application of cold (ice-collar, ice-water cloths) is useful, and sinapisms placed around the throat and over the chest also tend to arrest the spasm. The use of steam-inhalations from the so-called croup-kettle (Fig. 44) is of signal service, and should be more widely employed, particularly when it is inconvenient to use the hot bath.

Between the paroxysms the patient should receive a mild laxative, such as calomel or castor oil, and, in addition, the treatment appropriate in acute catarrhal laryngitis. To prevent recurrences an environment calculated to increase the nervous tone of the child is to be procured, and it is especially advisable to accustom him to the outer air, though protected by suitable dress and without undue exposure to draughts.

EDEMATOUS LARYNGITIS.

Definition.—An infiltration of the mucous membrane of the larynx with serum. In most cases it is a true inflammatory edema.

Etiology.—Two chief classes of causes are operative: (1) Those that excite inflammation. The condition may complicate acute laryngitis, though oftener it appears in chronic affections of the larynx, and particularly if ulceration be associated (*e. g.* tuberculosis, syphilis); it may also appear in connection with certain infectious diseases (erysipelas, diphtheria, typhoid fever). The inflammation inducing the edema may extend from adjacent parts, as the neck, pharynx, and other organs. (2) Factors that tend to excite dropsical effusion. These may be general, as Bright's disease, heart-affections, etc.; or they may be local. Among the latter are enlargements of the cervical and mediastinal lymphatics, aneurysm of the arch of the aorta, thyroid tumors, etc.—*i. e.* conditions that exercise pressure upon the jugular veins. Rice, who studied 41 cases, thinks it doubtful whether edema of the larynx ever occurs from simple catarrhal inflammation.

Symptoms.—In acute cases the initial disturbance is both sudden and severe. There is *dyspnea* that tends to increase rapidly, accompanied by a husky, suppressed voice, with augmenting obstruction. The *respirations* may become stridulous, but there is no cough. The laryngoscope reveals marked swelling of the epiglottis and of the ary-epiglottic folds. Rarely the swelling occurs in or even wholly below the vocal cords. The inserted finger may detect the swollen epiglottis, which may also be seen if the tongue-depressor be used.

Diagnosis.—This can be made with ease from the rapidly developing dyspnea soon reaching the climax, the absence of cough and hoarseness, and by the use of the laryngoscope. In cases in which the epiglottis can be felt or seen a laryngoscopic examination is superfluous.

The **prognosis** is decidedly unfavorable except in the event of early operative interference.

Treatment.—If of inflammatory origin, the ice-bag should be applied to the larynx, and ice should be allowed to constantly dissolve in the patient's mouth. Local depletion, preferably by leeching the front of the neck, is also to be tried, and Levy and Laurens[1] record a case in which a cure followed this measure. If intense dyspnea tends to persist, scarification of the edematous parts with a curved bistoury, the point of which is covered with adhesive plaster, must be promptly instituted, and, if asphyxia threatens, tracheotomy must immediately be performed. Dropsical edema demands scarification and, if relief does not follow, intubation or tracheotomy.

TUMORS OF THE LARYNX.

THESE may be either benign (fibroma, myxoma, lipoma, chondroma, adenoma, angioma, cyst) or malignant (sarcoma, carcinoma). Of these, papillomata or papillomatous fibromata occur most frequently, especially in infancy. Navratil[2] records 42 cases of multiple laryngeal papillomata in children whose larynges were extensively filled. These growths may also occur in chronic laryngitis, and, like other tumors of the larynx, they commonly spring from the vocal cords. Their shape, size, and tendency to pedunculation do not differ from their characteristics when noted elsewhere in the body.

Symptoms.—Small tumors may occupy the larynx without producing symptoms. The first feature then noted is *hoarseness*, which gradually grows worse and may end in complete aphonia. If situated in the upper larynx, *cough* is common, and when the tumor causes obstruction of the larynx *dyspnea* supervenes and tends to increase in severity. A mobile growth may cause sudden occlusion of the glottis, exciting orthopnea and threatening asphyxiation. To confirm the diagnosis a laryngoscopic examination is required.

The **prognosis** is favorable in the benign, but unfavorable in the malignant forms.

Treatment.—This is altogether surgical, though Delavan states that 3 cases of papilloma have been cured by frequently repeated sprays of absolute alcohol. Curetting is often followed by a recurrence, while laryngo-fissure and thorough removal of the growths restore speech and prevent recurrence.

III. DISEASES OF THE BRONCHI.

CATARRHAL BRONCHITIS.

(*Tracheo-bronchitis.*)

Definition.—A catarrhal inflammation of a part or the whole of the mucous membrane of the bronchial tubes. The mucosa of the

[1] *Arch. gén. de. Méd.*, Dec., 1895.
[2] *Berl. klin. Woch.*, Mar. 9, 1896.

trachea is also involved to a greater or less extent, and hence the term tracheo-bronchitis is quite appropriate, being descriptive of the seat and character of the disease. Involvement of the bronchioles may also take place, but not without an involvement of the corresponding alveolar structure, the condition being then, with propriety, termed "broncho-pneumonia." Hence the term "capillary bronchitis," still often employed to describe the latter condition, is not pertinent. A certain class of cases is met with, however, in which the catarrhal inflammation, as the result of downward extension, implicates the smaller bronchial tubes without involving the bronchioles; to such the term "capillary bronchitis" might be appropriately given.

The disease may be acute or chronic, both of these forms occurring either as a primary or secondary affection.

ACUTE BRONCHITIS.

Pathology.—The portions of the mucous membrane of the trachea and bronchi that are implicated become reddened and swollen; they are covered with mucus mingled with epithelial cells, and later muco-pus. Some of the smaller bronchial tubes are dilated. The mucous glands are swollen.

The histologic changes may be briefly stated as follows: desquamation of the ciliated epithelium, edema and swelling of the submucosa, and, in the severer grades, infiltration of the latter with leukocytes.

Etiology.—With rare exceptions tracheo-bronchitis is produced by the direct extension of a catarrhal inflammation from the nares, pharynx, and larynx. Rarely the bronchi are the seat of primary acute catarrh, and in the latter instances the upper air-passages may be implicated secondarily, constituting a reversal of the direction of extension.

The immediate causes are mechanical, chemical, and biologic irritants, which act directly upon the tracheo-bronchial mucosa; and that bronchitis is frequently due to infection at a time when the resisting power of the system is low there can be little doubt. Among organisms commonly met with is the so-called *micrococcus catarrhalis*. The circumstances disposing to bronchitis are many, those pertaining to the individual being—(1) Age, the old and very young being most liable; (2) Debility; (3) Occupation, as in certain trades that expose to irritating vapors and sedentary pursuits. Among the external conditions are—(1) Climatic factors, particularly variability of temperature and humidity; (2) Seasons of the year. "Catching cold" often results from exposure during the spring and autumn months. These two conditions depend substantially upon the same factors. (3) Epidemic influence, which may be independent of influenza. (4) Severe contusion of the chest.

Acute tracheo-bronchitis arises as a secondary condition in a great variety of diseases, as, for example, the exanthemata and other acute infectious diseases (typhoid fever, measles, whooping-cough, influenza, etc.). As shown elsewhere, among this class of diseases the bronchitis may be dependent upon the primary infectious process; but in many others it is due either to the inhalation of pathogenic irritants or to the retention of bronchial secretions that are apt to accumulate and decompose with resulting bronchitis. The accidental inhalation of particles

of food and saliva may also lead to secondary bronchitis, or the condition may be secondary to chronic affections—*e. g.*, Pott's disease, gout, Bright's disease. Among the toxic causes the poison of uremia must be embraced.

Symptoms.—Bronchitis of the larger tubes, which extends down to about the second division of the bronchi, is spoken of usually as a "cold." In such cases the onset is marked by recurring sensations of *chilliness*, and by *coryza*, slight *sore throat*, and *hoarseness;* while in young and feeble children *convulsions* may occur early. Mild *febrile symptoms* may appear, the temperature ranging from 101° to 103° F. (38.3° to 39.4° C.), with slight acceleration of the pulse; and there may be languor and aching in the limbs and lumbar region. With the fully-developed attack substernal soreness, sometimes even *pain*, is experienced, especially on coughing, and the pain may be referred to the intercostal muscles and the line of insertion of the diaphragm. The respirations are increased in frequency, but there is no dyspnea. There may be thoracic oppression and discomfort until the bronchial secretions become free, and there is a *cough* which is at first dry and hard. It often manifests itself in longer or shorter paroxysms, particularly on lying down and on rising after a full night's sleep. At the end of one, two, or more days the cough is moist and attended with an expectoration which is at first *mucoid* and scanty, often viscid, then *muco-purulent* and free; later still it is sometimes distinctly *purulent*. With free expectoration comes relief to the patient. Histologically, the sputum consists mainly of pus-corpuscles with large cells, in which may be seen the so-called myelin droplets of Virchow and carbon particles.

Physical Signs.—Upon laryngoscopic examination the mucous membrane of the larynx and trachea may be seen to be reddened and covered by more or less secretion.

Inspection and *palpation* of the chest are negative, except when the finer tubes become implicated or fever is present, in which case the respirations may be observed to be slightly accelerated. In children the increased rapidity of the respirations is more common and reaches a higher degree. Bronchial fremitus may sometimes be felt. *Percussion* yields negative results, save in very rare instances, in which there occurs a decided accumulation of secretion in the tubes, when there may be found impairment of resonance posteriorly below the scapulæ. *Auscultation* usually renders audible a harsh respiratory murmur, and less frequently piping, sibilant, and sonorous râles. In the advanced stage (with relaxation of the mucosa) large and medium-sized mucous râles are present. The râles change in position from time to time, and after coughing may be altogether absent, only to reappear later.

The **diagnosis** is reached without difficulty through the symptoms (slight fever, cough, and expectoration), the acute course, and the physical signs (harsh respiratory murmur, dry followed by moist râles, heard on both sides of the chest). The recognition of the long list of cases that constitute the secondary forms will be made easily possible by noting the circumstances under which they arise.

Differential Diagnosis.—Bronchitis can readily be separated from *pneumonia* and from *pleural effusion* by its history, its lighter course, and especially by the absence of the signs of consolidation and effusion.

When *broncho-pneumonia* develops in the course of bronchitis, dyspnea and fever are increased, cyanosis is present, and the general condition becomes much more grave. There are small patches that yield dulness on percussion, and broncho-vesicular breathing with moist rales can be detected on auscultation.

Bronchitis cannot be separated from the early stage of *whooping-cough*, but when the characteristic cough of the latter is heard all doubt vanishes.

The bronchitis of *measles* before the characteristic eruption appears is distinguished by the red spots ("Koplik's spots") upon the anterior half-arches of the soft palate.

The *acute suffocative catarrh* of Laennec may be confused with the severer forms of bronchitis. Examination of the chest shows nothing beyond coarse rhonchi, the chief distinguishing feature being the acute suffocation. Both *pulmonary tuberculosis* and *influenza* are apt to be confused with bronchitis (*vide* pp. 135, 273).

The **prognosis** varies with the previous constitutional state of the individual. In healthy adults, after a period ranging from a few days to two weeks, the fever subsides, but the cough, though less marked, and the expectoration usually continue for a variable length of time. In old persons and in those of a gouty or tuberculous diathesis the cases pursue a more protracted course. The cases in which streptococci are found in the sputum are severe and in the old may prove fatal. There is in these subjects a tendency on the part of the catarrhal process to extend downward until the finer tubes are implicated, sometimes endangering life. In the old the secretions are imperfectly expectorated; they gravitate to the most dependent parts and induce bronchiectasis. In young children this downward extension of the affection, with resulting broncho-pneumonia and areas of collapse in consequence of dilatation and occlusion of the bronchioles by muco-pus, is a not uncommon and serious event (*e. g.*, in measles, whooping-cough, *vide Broncho-pneumonia*).

Treatment.—There are many instances in which but little treatment is required, apart from the usual household measures and protection against cold and damp. If seen early, while the coryza is present, the attack may often be aborted by the use at bedtime of a Dover's powder in combination with quinin (gr. iv–viij—0.259–0.518); this may be seconded by a glass of hot lemonade, with or without a portion of whisky, and either a hot bath or a mustard foot-bath. The following morning a saline laxative should be taken. To children a mild calomel purge followed by a dose of castor oil may be administered. The patient should be kept in a warm, moist, equable atmosphere—preferably in-doors—and during this period he should take divided doses of quinin for a day or two. Vanderhoof lauds hexamethylenamine given in large doses (gr. x dissolved in a glassful of water four times daily); it shortens the stage of coryza and lessens, or even prevents, the succeeding bronchitis. If the above mode of treatment fail or if the patient does not come under observation early, the main objects of treatment should be (*a*) to render the secretions free, and (*b*) to hasten the expulsion of the sputum after it has been loosened. The first leading indication is to be met by the use of diaphoretics, diuretics, and relaxants. The subjoined formula combines these classes of agents:

℞. Potassii citrat., ʒvj (23.3);
Liq. ammonii acetat., ℥v (148.0);
Spt. æth. nit., ℥j (30.0);
Vini ipecac., ʒij (8.0);
Syr. pruni virg., q. s. ad ℥viij (216.0).—M.
Sig. ℥ss (7.0) in water every two hours until the secretions are loosened.

If the temperature in any given case be maintained at a considerable elevation, such as 102°–103° F. (38.8°–39.4° C.) or over, tincture of aconite (♏xvj—1.065) may be added to the above mixture; and if there be present much tickling with distressing cough, due to irritability of the affected mucosa, codein (gr. ij–iij—0.129–0.194) may be added to the same. For the incessant irritative cough which is present in severe forms of catarrh opium alone is really effective. When the above prescription is not productive of free secretion and troublesome cough continues, I employ the following:

℞. Ammon. chloridi, ʒv (20.0);
Codeinæ, gr. iv (0.259–0.388);
Spt. junip. co., ℥ss (16.0);
Mist. glycyrrh. comp., ℥iiss (80.0);
Syr. pruni virg., q. s. ad ℥iv (120.0).—M.
Sig. ʒj (4.0) every two hours.

Apomorphin is also excellent as a soothing relaxant in doses of gr. $\frac{1}{20}$ to $\frac{1}{10}$ (0.003 to 0.006) every two hours. Mild counter-irritation by means of mustard-paste, followed by the application of iodin once daily, is also helpful. The patient should keep to his room, in which the atmosphere should be kept moist and of even temperature. (*b*) The expulsion of the sputum may demand stimulating expectorants, though rarely. It is to be recollected that when the tracheal secretion becomes copious the period of convalescence is usually reached, and stimulating expectorants are then entirely unnecessary. When, on the other hand, the cough is no longer dry, and on auscultation the râles are found to be moist, and whilst, at the same time, the expectoration is expelled with difficulty, or if the bronchitis tends to become chronic, then such stimulating expectorants as senega, squills, and ammonium chloride are to be employed. In cases in which expectoration continues to be too abundant terebene, tar syrup, and oil of sandal are to be resorted to.

Debility and secondary anemia must be speedily overcome by exhibiting quinin, bitter tonics, iron, and arsenic; and a suitable change of air often yields prompt and excellent results in protracted cases. The treatment of the various forms of secondary bronchitis will be considered in their appropriate connections in this work. In the aged the general strength must be maintained; the patient's position must be changed at short intervals and stimulants are usually needed.

In children, acute bronchitis is in the main to be treated in the same manner as when it occurs in the adult. Opium, however, is to be used very sparingly, and generally in the form of paregoric. If the secretion is abundant and imperfectly raised, it is well to administer an

emetic, such as the wine of ipecac (ʒss–j—2.0–4.0), and repeat in ten minutes if necessary. If dyspnea be urgent and cyanosis be marked in the lips and finger-tips, a prompt emetic is imperative in order to save life. A child suffering from acute bronchitis should be kept in bed until the fever subsides.

The diet during the dry stage should consist of liquid forms of nourishment, which should, for the greater part, be taken hot. After the "cold" has been loosened solid food should be resumed.

CHRONIC BRONCHITIS.

Pathology.—The lesions of chronic bronchitis manifest considerable variety both as regards their nature and extent. The epithelial layer is, in part, missing, and sometimes the mucous membrane is quite thin. In consequence the longitudinal elastic fibers appear unduly prominent. The mucous glands and the muscular coat undergo atrophy in long-standing cases, and the bronchial tubes are dilated (*bronchiectasis*). In another large group of cases the mucosa is irregularly thickened or infiltrated and granular. Small ulcers corresponding to the mucous follicles are common, and almost constantly emphysema develops in consequence of secondary changes in the vesicular structure.

Etiology.—Chronic bronchitis may either be *primary* or *secondary*. The affection is, however, almost always a secondary one, and, though sometimes the result of repeated attacks of acute bronchitis, it is oftener caused by certain chronic complaints and certain diatheses, as chronic alcoholism, rheumatism, gout, syphilis, pulmonary tuberculosis, and pulmonary emphysema. Organic valvular affections, obesity, and chronic Bright's disease cause *hypostatic bronchitis*. The primary form, which is rare, is the result of exposure to wet or cold or to the daily inhalation of some irritant that maintains a low grade of catarrhal inflammation (dust, vapors). When chronic bronchitis follows the acute form we are often able to detect the operation of some favoring cause, as age, climate, or season. It is most common in the aged, and occurs by preference during the cold season, often recurring regularly in the cold and variable weather of autumn and winter, and disappearing in summer. Hoxie and Lamar report two cases of tracheo-bronchitis due to the presence of fungus stalks as the only causative agent.

Symptoms.—The symptoms are similar to those of acute bronchitis, though rather less severe. *Pain* is rarely present, the patient complaining merely of a feeling of substernal constriction. There may be soreness at the base of the chest if the cough be frequent and severe, and occasionally in the epigastrium as a result of traction of the diaphragm on the ribs. *Cough*, while not a constant accompaniment, is *paroxysmal* and varies in severity and frequency. The degree of the violence of the paroxysm depends upon two factors—the character of the bronchial secretion and the seat of the catarrhal inflammation. Thus when the expectoration is tenacious and scanty, and when the small-sized tubes are affected, cough is most violent. It also varies both with the weather and the season, as is evident from the fact that there is often an absence of cough in summer, while it returns unfailingly with each new winter.

The *expectoration* differs widely in different cases. It is sometimes abundant and sero-mucous in character. On the other hand, there are cases of dry cough in which there is little or no expectoration. As a rule, however, it is rather copious, and either *muco-purulent* or distinctly *purulent* in character. *Fever* is usually absent, though rarely a slight rise of temperature occurs at night. The appetite is good as a rule; the bodily weight and nutrition are also well maintained.

Physical Signs.—On *inspection* we usually note undue enlargement of the thorax, with a decrease in expansile movements due to the associated emphysema. Hence *dyspnea* is commonly observed.

Percussion yields a clear or hyperresonant note. Dulness or impaired resonance is sometimes met with, however, during acute exacerbations, especially over the bases, and is due to congestion and edema (Fox). On *auscultation* rhonchi of various forms and moist râles are heard, their number and size being in proportion to the extent of the swelling of the mucous membrane and the amount and fluidity of the secretory products. The respiratory murmur is enfeebled, though roughened, and the expiratory sound is prolonged and wheezy. The right heart may be dilated from increased tension in the pulmonary circuit.

Clinical Varieties.—Special forms, depending largely upon specific causal factors, remain to be described:

1. The commonest variety of chronic bronchitis has been called the "winter cough of the aged," and, as before intimated, is usually accompanied by emphysema and cardiac disease. For this form the gouty diathesis is often responsible. The cough occurs in paroxysms that are most severe at night, and during the early morning hours it is attended with free expectoration of the secretion that has accumulated during the night.

2. *Bronchorrhea.*—In this form there may be an abundant bronchial secretion, composed largely of serum (*bronchorrhœa serosa*). More frequently perhaps the expectoration is purulent and thin, containing greenish or greenish-yellow masses. It may at times be thick and purulent. Dilatation of the tubes and resulting fetid bronchitis may be developed as secondary conditions.

3. *Fetid Bronchitis.*—In this variety the expectoration emits the characteristic odor of decomposing animal substances. The fetor may indicate gangrene of the lungs, abscesses, bronchiectasis, decomposition of matter within phthisical cavities, or empyema with perforation of the lung. Hence these conditions must be carefully excluded before the diagnosis of true fetid bronchitis is made. In the latter disease the expectoration is usually copious, and on standing separates into three layers, of which the uppermost is composed of frothy mucus, the intermediate of a serous liquid, and the lowest of a thick sediment, that presents a granular appearance and is made up chiefly of small yellow masses—the characteristic Dittrich's plugs. Microscopically, the Dittrich's plugs are seen to be composed of micröorganisms, chief among which is the *Leptothrix pulmonalis;* they may also contain pus-corpuscles, fat-granules, and crystals of margarin. Dèmétre found the colon bacillus and ascribes the fetor to its presence.

The condition may be a grave one, and associated with it may be observed ulceration of the bronchial tubes, with dilatation, pneumonia, abscess, gangrene, and rarely metastatic cerebral abscesses. When *putrefactive changes* take place in the bronchial secretion in the course of chronic bronchitis a new group of symptoms, as a rule, immediately appears. This comprises rigors occurring at irregular intervals and associated with high fever and increased prostration. Cough and pain in the chest also become aggravated, but these acute symptoms may shortly subside and the usual course of chronic bronchitis be resumed. Even under the latter conditions fetor of the breath and sputum may persist.

4. *Dry Catarrh.*—The cough is both severe and paroxysmal, and there is little or no expectoration. When expectoration is present the sputum is very tenacious and is expelled with great difficulty. An asthmatic disposition is sometimes noticeable in this variety, and emphysema is commonly associated. The dry condition of the bronchial mucosa is evidenced by sibilant and sonorous râles. This form occurs in old persons, as a rule.

5. Osler has described a form of chronic bronchitis that occurs most frequently in women, and dates its onset from a comparatively early period of life. It does not undermine the general health. The cough is most pronounced in the morning, and is accompanied by a relatively small amount of muco-purulent expectoration. An examination of the chest yields negative results. The condition seems to proceed from a gouty or tuberculous diathesis in some instances. I have had under observation for several years a young woman in whom this form of bronchitis alternated with eczema of the face.

6. Teichmüller has described an eosinophilic bronchitis. The expectoration is mucoid, as a rule, though occasionally muco-purulent. It is characterized particularly by the presence of a considerable number of eosinophile cells in the sputum. It is not dependent upon adenoid disease of the naso-pharynx. Some writers doubt its existence.

Diagnosis.—The diagnosis of chronic bronchitis is rarely difficult. Since it is usually a secondary condition, it is of the utmost importance to determine the nature of the primary affection. An examination of the heart and of the urine should not be overlooked.

Pulmonary tuberculosis is to be discriminated from chronic bronchitis, and the distinctive points are—(1) A clear tuberculous history. In phthisis there are fever and loss of flesh and strength, while in chronic bronchitis fever is absent and the general health is not impaired. (2) In pulmonary tuberculosis the signs of localized consolidation (usually at one or other apex) appear early, while in chronic bronchitis these are absent. (3) In phthisis the sputum, when examined microscopically, shows the presence of the tubercle bacillus.

In *acute pulmonary tuberculosis* the fever, dyspnea, cyanosis, and increased prostration constitute a group of features that should distinguish it from chronic bronchitis. Coexisting *pulmonary emphysema* is to be recognized by the characteristic symptoms and signs of this complaint. *Primary fetid bronchitis* must be differentiated from the various other conditions previously mentioned, giving fetor of the sputum and breath. In *abscess* of the lung the sputum contains shreds of lung-tissue, including elastic fibers, crystals of hematoidin, cholesterin, and

amorphous blood-pigment; usually localized dulness and broncho-cavernous breathing coexist. In *gangrene* there are contained in the sputum shreds of lung-tissue, but separate elastic fibers are often absent, on account of the presence of a ferment that causes a solution of the elastic tissue (v. Jaksch). *Bronchiectasis* is usually unilateral, and gives rise to dulness and other physical signs that are confined to limited areas, while in chronic bronchitis the signs are general.

Prognosis.—Recovery is the exception, though improvement may frequently be observed. The course is exceedingly protracted, and the danger from the late development of certain complications and sequels, such as emphysema or right-sided cardiac disease, must be borne in mind. Since the disease is generally a secondary affection, the prognosis in most instances depends upon the outlook in the primary disease.

Treatment.—The treatment falls naturally under two main heads —(1) Hygienic, and (2) Medicinal.

1. *Hygienic.*—This has reference, frequently, to the removal of various noxious influences. When the patient cannot make a suitable change of air during the cold season, he must keep his room during inclement weather; he should, however, be allowed to spend as much time as possible in the open air during clear and pleasant weather. The vitiated atmosphere of saloons or public halls is to be avoided. The patient should be carefully clad; he should wear flannels next the skin during all seasons of the year, but his outer clothing need not be unusually cumbersome. If the case be of an aggravated type and the circumstances of the patient permit, he should be sent to a warm latitude in the autumn, in order to escape the effects of a severe northern winter. Patients in whom the bronchial secretions are abundant should be sent to a dry, warm climate or to a region whose atmosphere is impregnated with the balsamic vapors of the pine. On the other hand, patients with dry bronchial catarrh are most relieved by an equable, moist, warm climate. Among suitable resorts, those that should be mentioned are the Riviera, Cannes, San Remo, Sicily, and Algiers abroad, and Florida, Southern Georgia, and Southern California at home. Change of air may also become an effective means of prevention.

Prophylaxis also includes the removal of any diseased conditions that are casually related. The coexistence of cardiac disease, the gouty diathesis, obesity, and particularly any renal disturbance, call for the primary treatment of these conditions. Hardening (*Abhärtung*) is an important preventive method, and is accomplished by hydriatic measures—the cold sponge, douche, or plunge—if there be no contraindications.

The *diet* should be generous, but not stimulating, and articles easy of digestion should be selected. Wines and liquors are to be avoided unless particular indications for their use exist. Special conditions, however (*e. g.* albuminuria), may render necessary a special dietary.

2. *Medicinal.*—In this disease medicines are palliative in their effects rather than curative. Relaxing expectorants are to be avoided, owing to their depressing action, and the stimulating expectorants are, in a majority of cases, not only valueless, but hurtful, tending to lessen the appetite and disorder the digestion. When, however, the sputum is muco-purulent and is dislodged with difficulty, expectorants of this class (squills, senega, ammonium chloride) may be tried. I have obtained good results from the use of the following in severe paroxysms of cough:

℞. Ol. eucalypti, ʒjss–ʒiij (6.0–12.0);
Codeinæ, gr. vj (0.388).
M. et ft. capsulæ No. xviij.
Sig. One every four hours, as required.

Occasionally potassium iodid exerts a curative influence, but its use may be limited to cases that are due to the syphilitic, rheumatic, and gouty diatheses. Five or ten grains of the iodid four times daily may be exhibited, and should there be present a syphilitic taint the remedy should be pushed to the limit of tolerance. The balsam of copaiba is sometimes efficacious:

℞. Balsami copaibæ, ʒj–ʒij (4.0–8.0);
Ammon. chloridi, ʒij (8.0);
Extr. glycyrrh. pulv., ʒj (4.0).
Mist. ammoniaci, q. s. ad f℥iij (96.0).—M.
Sig. ʒij (8.0) every four hours.

Other remedies that possess great value in certain cases are creasote (in ascending doses), turpentine, terpine, tar, the balsams of tolu and Peru, and sandal-wood. Box[1] advises the emptying of the cavities by the process of inversion—night and morning.

If the vital powers are poor, bitter tonics, as iron, quinin, and strychnin, and other measures calculated to invigorate the system, are indicated. When the sputum is excessive in amount, astringents (zinc sulphate and oxid) are sometimes useful. In this class, Barnes recommends the internal use of ichthyol (dose, gr. v–0.3, t. i. d.). Astringents may also be used with advantage in the form of a spray when the expectoration is too free. On the other hand, sprays from properly selected solutions (*e. g.*, ammonium chloridi, gr. v–x ad ℥j—0.324–0.648 ad 32.0) are valuable in assisting expectoration. In fetid bronchitis sprays of antiseptic solutions are to be used, and the following will be found serviceable:

℞. Phenolis, gr. ij–iv (0.129–0.259);
Olei eucalypti, ♏ij–iv (0.133–0.266);
Aquæ, ℥j (32.0).
Sig. To be inhaled from a steam- or hand-atomizer.

Pneumato-therapy has given brilliant results in certain instances. After deciding what microörganism is responsible, the corresponding vaccine (autogenous) should be employed. If different organisms be found, the results are unsatisfactory.

BRONCHIECTASIS.

Definition.—The universal or circumscribed dilatation of the bronchial tubes.

Pathology.—Two main forms are recognized—the cylindrical or

[1] *Lancet*, Jan. 5, 1907.

simple, and the saccular, and both of these may be met with in the same lung. It may be general or partial, the former variety being always unilateral, the latter sometimes bilateral. In *universal bronchiectasis* the bronchial tubes, throughout their extent, are the seat of numerous sacculi communicating with one another. These present smooth, shining walls, except in the most dependent parts, where ulcers are sometimes seen. Extreme conditions of dilatation may take the form of huge cysts, which may extend to the periphery of the lung; the lung-tissue lying between the sacculi then becomes cirrhotic as a rule. In *partial dilatation* the bronchial mucous membrane is implicated, with an occasional narrowing of the lumen. Usually these dilatations are cylindrical, though they may be saccular, and rarely fusiform. The partial is the most common variety.

Histology.—When the walls of the larger dilatations are examined microscopically, the cylindrical epithelium is seen to be replaced by a pavement epithelium. The elastic and muscular layers are thin, and the fibers are usually separated. Contained in these dilatations are found secretions that may frequently be fetid.

Etiology.—In the majority of instances the condition doubtless arises from an involvement of the bronchial mucosa that extends to the submucous tissue and leads to muscular, fibrous, and cartilaginous atrophy. These changes render the wall of the tube unable to resist the pressure of the air in violent paroxysms of cough, and, once the process of dilatation is commenced, the accumulated secretions may tend by their weight to distend further the already weakened walls. Thus the elasticity of the latter is impaired, and finally destroyed. The etiologic factors show the affection to be *secondary* as a rule, and are—(1) Chronic bronchitis and emphysema, chronic phthisis (usually when the seat of the dilatation is at the apex), broncho-pneumonia (in children), and compression of a bronchus (*e. g.*, by aneurysm). Heubner believes that bronchiectasis in adults may be sometimes traced to whooping-cough and measles in young children. (2) Great thickening of the pleura, especially when associated with bronchitis or interstitial pneumonia, with contraction of the lung. (3) Rarely it is congenital.

Among predisposing conditions are—(*a*) *Age*, bronchiectasis being most common in adult or middle life; and (*b*) *Sex*, being more common in males than females.

Symptoms.—There is always *cough*, usually in prolonged and severe paroxysms. The attacks take place most generally in the morning when the dilated tubes are full, and may be excited by a change of posture. Accompanying the cough there is *profuse expectoration*, which may amount to a pint or more in twenty-four hours. The sputum is grayish-brown in color and muco-purulent, emitting a sour or, more frequently, a horribly *fetid odor*. On standing, the expectoration separates into three strata—the uppermost, of brownish froth; the middle, of a thin, sero-mucous fluid; and a thick sediment, of cells and granular débris. Examined microscopically, the sediment is seen to be composed chiefly of pus-corpuscles, with which are intermingled Charcot-Leyden and fatty-acid crystals, the latter arranged in bundles; leptothrices, vibrios, and bacteria are also found. Elastic fibers may be observed if ulcers be present.

Dyspnea is noted, but is not a prominent symptom, unless some other chronic affections of the chest coexist or some complication arises. *Hemoptysis* occurs rarely, and may be due to the bronchiectatic lesion. Abscess of the brain may develop, though rarely.

Physical Signs.—These differ in character according to the size, situation, and nature of the dilatation, and also according to the condition of the surrounding lung-tissue.

On *inspection* retraction of the chest-wall may be noted when chronic pleurisy and interstitial pneumonia are associated. The tactile fremitus is usually increased, but may rarely be diminished. The *percussion* resonance is impaired or even flat, and on *auscultation* bronchial breathing is heard, with occasional râles that have a metallic quality. A *saccular dilatation* immediately beneath the pleura may give a tympanitic note, and may also give typical cavernous or amphoric respiration. A tympanitic resonance over a circumscribed area which prior to cough and expectoration presented dulness, is a significant sign (Babcock). These signs are generally discoverable at the base of one or other lung.

Diagnosis.—Simple dilatation of slight degree may exist without appreciable signs, and in other instances the breathing is broncho-vesicular over localized areas, with râles displaying increased metallic quality.

Saccular Bronchiectasis.	Pulmonary Tuberculosis.
History of chronic bronchitis, chronic pleurisy, and interstitial pneumonia, or of foreign body.	History of cough, hemoptysis, with progressive loss of flesh and strength. Family history.
Cough is paroxysmal, and sputum characteristic and copious.	Cough less paroxysmal. Sputum nummular in the stage of cavity.
Tubercle bacillus absent.	Tubercle bacillus present.
Course longer, with little impairment of the general health.	Course relatively shorter, powers of the system progressively undermined.
Physical Signs.	
The condition is persistent, but non-progressive. Usually located at base.	More apt to be progressive, commonly at one or other apex.

Circumscribed empyema with a fistulous connection with the lung may simulate bronchiectasis. There is often in such cases a clear history of an acute illness with a sudden onset, the symptoms pointing to pleural inflammation. The patient suddenly expectorates, at irregular intervals, large quantities of purulent matter. *Actinomycosis* may also cause conditions that simulate bronchiectasis. The diagnosis may be made by finding granular particles containing the actinomyces in the sputum.

Prognosis.—Apart from certain remote dangers (*e. g.* abscess, gangrene, fatal hemorrhage from an aneurysm in the wall of the cavity), these cases pursue a favorable but exceedingly long course.

Treatment.—The lesion being a permanent one, there is no known remedy that will either abridge or influence the course of the affection. Again, since the cough is protracted and attended with profuse expectoration, sedatives and ordinary expectorants are contraindicated. For the fetor, antiseptics are to be employed both topically and internally, and a solution of carbolic acid (1–3 per cent.) or thymol (1 : 1000) is to be used by inhalation. Internally, terebene (♏v–x—0.333–0.666) in capsules every four hours is valuable; also creasote in increasing

doses (♏j—0.066, increasing by ♏j each day, till ♏vj—0.399—are taken three times daily) is to be persistently employed. Intrathoracic injections are often resorted to with gratifying results.

If the dilatation is situated superficially and not amenable to therapeutic measures, it may be freely opened and thoroughly drained. By raising the foot of the bed or lowering the head of the patient in other ways we favor the discharge of the accumulated secretions from the sacculations. Hoppe-Seyler recommends the continuous nocturnal postural method in cases in which no contraindications exist.

BRONCHIAL STENOSIS.

Definition.—Narrowing of the bronchus, due either to constriction or to compression.

Pathology and Etiology.—(*a*) *Stenosis due to Constriction.*—This form is most frequently occasioned by the presence of foreign bodies; by new growths (polypoid) within the bronchi, or the cicatrices of healed ulcers, and in the smaller bronchi by swelling of the mucosa. The bronchial walls also sometimes become thickened by inflammatory exudates in certain acute and chronic affections, such as syphilis, tuberculosis, and glanders.

(*b*) *Stenosis Due to Compression.*—Compression of one or more bronchi may be met in enlargements involving the thoracic organs, *e. g.*, aneurysm, echinococcus cyst, solid tumors, enlarged glands, mediastinal and pulmonary abscesses, and pleural effusion.

Symptoms.—The symptoms depend upon the size of the bronchus affected and the degree of stenosis. *Dyspnea* is the most conspicuous symptom, but the proper filling of the lungs with air is not accomplished. Under these circumstances the air in the lungs becomes rarefied, and instead of normal expansion everywhere the lower part of the sternum and the lower ribs are *retracted* on inspiration. Obstruction of the primary bronchus on either side of the chest would naturally be followed by inspiratory retraction of the inferior part of the chest-wall and intercostal spaces upon the affected side. The movements of the larynx are slight in bronchial stenosis, while they are marked in laryngeal obstruction. *Cough*, *expectoration*, and *fever* are sometimes present.

Physical Signs.—*Inspection* shows defective respiratory movement upon the side involved. The local tactile fremitus is diminished or absent upon the affected side. The *percussion-note* remains unaltered, though less influenced by forced respiration and, particularly, expiration than in health. Pulmonary atelectasis may occur as a secondary event, and is shown by dulness on percussion. The *auscultatory* signs consist of a greatly diminished vesicular murmur on inspiration, due to the diminished amount of air entering the air-cells, and the presence of râles, sibilant and sonorous, at the seat of obstruction. Obstruction of a small

bronchus may, however, be present without appreciable physical signs, owing to collateral emphysema.

Diagnosis.—The nature and site of the affection may be determined by auscultation, and sibilant and sonorous râles will be conspicuous at the point of constriction. A clear history, together with a careful investigation of antecedent affections of the thoracic organs leading up to the stenosis, are factors that must furnish the etiological data in individual cases after the exclusion of foreign bodies as the possible cause. Tracheal or laryngeal stenosis may be eliminated by careful laryngoscopic examination.

Prognosis.—The duration is indefinite, though usually protracted, and most cases yield an unfavorable prognosis. In those instances, however, in which the narrowing is due to foreign bodies the latter may rarely be dislodged and fortunately ejected.

Treatment.—The treatment must be addressed to the cause in individual cases. Obviously, the question of the removal of foreign bodies from the bronchi falls within the domain of surgery, though the administration of an emetic has been followed by complete success in certain instances. Obstruction due to stenosis of a main bronchus may be treated by dilatation with bougies, the treatment of course being carried out by a specialist.

ASTHMA.

(*Bronchial Asthma.*)

Definition.—A chronic affection, characterized by paroxysmal dyspnea, due to contraction of the muscles of the bronchioles. The paroxysmal dyspnea caused by arterial contraction is also termed asthma.

Pathology.—In many cases there is hyperemia of the bronchial mucosa, due to pneumogastric or vasomotor functional disturbances, and also a characteristic exudate of mucin. In others there may be no lesions whatsoever, and the condition is a pure neurosis, often of reflex origin. Von Leyden considers asthma to be a reflex neurosis, the *primum movens* of which may be situated almost anywhere in the body. The morbid changes peculiar to chronic bronchitis, pulmonary emphysema, and right ventricular hypertrophy with dilatation are found at autopsy.

Etiology.—There is present either a constitutional peculiarity or a singular susceptibility of the local muscular fibers to spasmodic contraction, both of which are of unknown nature. The exciting factors are very various, but may be grouped under five heads:

(1) **Acute Bronchitis.**—It must not be forgotten, however, that a bronchitis may be set up by the paroxysms. Curschmann has observed also a local croupous inflammation of the smaller bronchioles in some of his cases, which he describes as *bronchiolitis exfoliativa*, and which seems to have given rise to the seizures in grave cases. Roentgenoscopy revealed swollen lymph-nodes encircling the bronchi (Chelmonski).

(2) The inhalation of numerous and widely various *irritants*, as

chemical vapors, smoke, fog, dust, and emanations from plants or certain animals. A person may be immune in the city and suffer greatly on going into certain rural districts.

(3) *Reflex Causes.*—The causal connection between obstructive affections of the nose and asthma is a subject that is appreciated by the specialist. Gastric disturbances, intestinal irritation, pregnancy, and uterine disorders may cause this complaint. In dyspeptic asthma Boas found the diaphragm elevated above its normal position.

(4) Asthma may be secondary to, and most probably excited by, cardiac disease ("cardiac asthma," due to an acute rise in blood-pressure —Petreu[1]), emphysema, gout, rheumatism, syphilis, Bright's disease, emotional excitement, and irritating lesions in the region of the medulla. Possibly, some of the latter affections merely constitute predisposing factors. In this connection it is to be pointed out that individual liability to the disease depends upon the special etiologic factor.

(5) The spasm may be due to some chemical substance or toxin acting on the respiratory center.

Predisposing Causes.—*Heredity* takes first place, and is noted in about 50 per cent. of all cases. The complaint is about twice as frequent in *males* as in *females*, and, if we except hay asthma, it is more prevalent in winter and early spring than during the warm season.

Clinical History.—Hyde Salter's collective statistics show that prodromal symptoms appeared in about one-half the instances (in 111 out of 226 cases). They differ, but are chiefly *nervous* in most cases, and appear as irritability of temper, either depression or unusual buoyancy of spirits, headache, drowsiness, and vertigo. Abundant diuresis and digestive disturbances may be seen.

The *paroxysm* usually comes on in the night during sleep, and at a definite time. It may develop, however, while awake or, rarely, during the day. The onset may be sudden, but perhaps more frequently the patient first experiences a moderate grade of dyspnea and thoracic constriction. This augments with unwonted rapidity, and often attains to an inordinate degree, until the patient feels smothered, sits up, grasps his knees with his hands, or places the palms upon the bed so as to raise the shoulders and thus reinforce the accessory muscles of respiration. When the attack is severe, he rushes to an open window if able to leave his bed, or sits on a chair and places his arms on the back of another chair, so as to fix the shoulders and thus give purchase to the auxiliary muscles of respiration while frantically endeavoring to maintain the act of breathing. The *face* is pale, anxious, and soon bedewed with cold perspiration, while the lips, eyelids, and finger-tips are livid, owing to defective oxygenation of the blood. The *temperature* is subnormal and the *pulse* feeble and rapid. The clinical picture wears an alarming aspect.

Physical Signs.—*Inspection* shows enlargement of the chest, which in the advanced stage becomes barrel-shaped. The reason for this is the presence of an increased amount of air in the thorax with a total inability to expel it. The respirations are diminished in frequency to 12 or 10 per minute. The natural rhythm is also greatly disturbed, and inspiration is seen to be short and gasping, and followed immediately by greatly prolonged expiration. The expansile movement of the chest is

[1] *Berliner klinische Wochenschrift*, Dec. 27, 1909, xlvi., 52.

very limited, and in inverse ratio to the patient's efforts at breathing. There is lowering of the diaphragm. *Palpation* is negative in its practical results. *Percussion* yields a hyper-resonance; in advanced cases with associated emphysema semi-tympanic resonance is common. On *auscultation* inspiration is found to be short and feeble, and expiration much prolonged and accompanied by a low-toned wheezing sound that may also be audible to onlookers. A great variety of dry râles are heard, chiefly high-pitched, sibilant, and sonorous, that are more marked on expiration than inspiration. They also change their character and situation frequently. At the close of the attack moist râles may be heard, and occasionally, when bronchitis complicates asthma, the moist râles may be combined throughout the paroxysms.

The *duration* of the attack is various, ranging from a few minutes to several hours, though rarely it may endure a week or two, with spontaneous remissions during the day (*e. g.* when chronic bronchitis coexists). Usually it subsides *abruptly*, with the expectoration of rounded gelatinous masses and, later still, of muco-purulent material. The former, when floated in water, are found to be composed of the so-called *Curschmann's spirals* (mucous moulds of the smaller tubes), and the spiral character of these small, ball-like pellets may even be detectable with the naked eye. When examined microscopically their spiral structure is evident. Two forms are recognized: (1) Composed of *mucin*, arranged spirally; in its meshes may be observed alveolar cells, many of which have undergone fatty degeneration. (2) A perfectly clear and translucent filament that is most probably composed of transformed mucin and occupies the center of the coiled spiral of mucin. In the early stage of the attack Curschmann's spirals (Fig. 45) are invariably

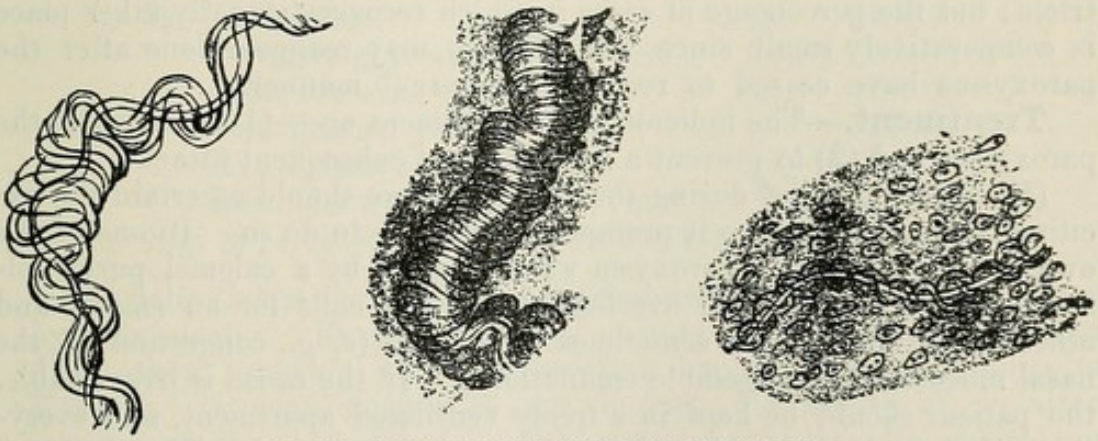


FIG. 45.—Curschmann's spirals (bronchial spirals).

present in the expectoration, and in many instances Charcot-Leyden octahedral crystals are also visible. The latter are a product of the eosinophile leukocytes and are found also in the semen, in leukemia, and in the stools of patients suffering with intestinal parasites. Müller, Fink, Leyden, and others have demonstrated extremely large numbers of *eosinophile leukocytes* in the sputum. Fink and Gabritchewski have found a large excess (ranging from 15 to 35 per cent.) of eosinophile leukocytes in the blood. V. Noorden and Swerchewski found the same increase, but only at the times of the attacks.

Diagnosis.—A clear history, together with the physical signs and a microscopic examination of the sputum, should lead to correct results. The history alone is inadequate to put the physician upon the right track. *Laryngeal affections*, which give rise to spasm of the glottis and dyspnea, are excluded by the hoarseness and aphonia which are usually present, while the characteristic physical signs of asthma are absent. Again, the dyspnea is inspiratory, not expiratory as in asthma. If tuberculosis be suspected, the Röntgen rays should be employed.

Emphysema may be confounded with asthma. The presence of recognized causes, of typical physical signs, and the paroxysmal dyspnea in asthma are the chief points of distinction from emphysema. The sputum should be examined microscopically if doubt remain. The so-called *cardiac asthma* is distinguishable by the presence of indications of chronic nephritis with oncoming failure of the left ventricle.

Course and Prognosis.—In mild cases of asthma there may be but one or two nocturnal paroxysms, with entire freedom from cough and dyspnea during the following day, while in severe ones there is a repetition of the paroxysms from three to five or six nights. Under these circumstances in the intervals (usually corresponding to the period of day) there are slight wheezing and some cough. In long-standing cases asthma leads constantly to the development of chronic bronchitis and emphysema, hence these affections are often combined. The paroxysmal character of the affection is often partly or wholly lost, the patient rarely being entirely free from asthmatic dyspnea, combined with cough and mucopurulent expectoration. The periodicity of the attacks varies; in some it recurs monthly or at shorter intervals, and in others only annually.

There is rarely any danger to life, except when the secondary affection is emphysema, and its remote consequence is dilatation of the right ventricle; but the percentage of cases in which recovery actually takes place is comparatively small, since the affection may reappear long after the paroxysms have ceased to recur in the usual manner.

Treatment.—The indications for treatment are—(1) to cut short the paroxysms and (2) to prevent a recurrence of subsequent attacks.

(1) To bring relief during the paroxysms we should ascertain the exciting cause, and remove it promptly if possible to do so. In one of my own cases a prolonged paroxysm was cut short by a calomel purge followed by an enema. An overloaded stomach calls for an emetic, and other causal factors are sometimes removable (*e. g.*, congestion of the nasal mucosa, dust, vegetable emanations). If the cause is irremovable, the patient should be kept in a freely ventilated apartment, and everything that tends to impede respiration must be removed. The choice of posture as affording the greatest relief may usually be left to the patient.

To cut short the paroxysms: The particular mode of treatment that will afford most speedy relief differs widely in different cases, and not infrequently the patient, as the result of experience, is aware of the most efficacious remedies. As a rule, however, sedative antispasmodics, relaxants, and stimulants are the classes of medicinal agents from which a careful selection is to be made; and while a great variety of these have been employed, I shall content myself by adducing here only the most valuable. In the hands of some observers a few whiffs of chloroform have proved highly efficacious, but in my own they have produced only momentary good effects; ether is the safer remedy and may be tried in like manner.

In a certain proportion of the cases from four to six drops of amyl nitrite thrown upon cotton-wool or a handkerchief, and inhaled, bring speedy and permanent relief. Of stimulants, coffee is the best: immediately upon the appearance of the paroxysm about one pint of strong coffee is to be taken hot (without cream or sugar), and in this way the seizure may sometimes be arrested. Alcohol when given hot and in sufficiently large doses to induce mild intoxication may be found very useful; and with "hot toddy," spirits of chloroform may be combined.

The inhalation of the fumes of niter-paper[1] often gives quick, temporary, and, less frequently, permanent relief. When employed, the atmosphere of the room occupied by the patient must be well filled with the fumes.

Among depressant antispasmodics are belladonna, hyoscyamus, stramonium, and lobelia, and these seem to be of most value when used in the form of cigarets. The leaves of the plant employed are first steeped in a concentrated solution of potassium nitrate or chlorate, and a trial should be made of different sorts of cigarets or pastilles (which are similarly prepared), since all cases are not benefited by the same brand. The inhalation of tobacco-smoke is equally beneficial in rare instances.

A large number of cases, despite the use of the measures above indicated, exhibit an obstinate tendency, and for their treatment no remedy bears favorable comparison with morphin, administered hypodermically, It has occasionally led to the establishment of the morphin-habit; hence it must not be used indiscriminately. It is best given in full doses (gr. $\frac{1}{3}$–$\frac{1}{2}$—0.0216–0.0324), and may be combined with atropin. V. Noorden uses atropin in ascending doses (gr. $\frac{1}{120}$ increased to $\frac{1}{16}$ daily), then gradually diminishes the dose; this treatment is repeated every few months, though lessening the dose and shortening each course of treatment. The following formula has proved efficient in my hands:

℞. Tr. lobeliæ, ʒj (4.0);
Tr. nitro-glycerini (1 per cent.), ♏xvj (1.06);
Sodii bromid., ʒv (20.0);
Vini ipecac., ʒv (20.0);
Ext. hyoscyami, gr. viij (0.518);
Elix. simplicis, q. s. ad ℥iv (128.0).—M.
Sig. ʒj (4.0) every one or two hours in water.

S. Solis-Cohen lauds hyoscin hydrobromate (gr. $\frac{1}{200}$) administered hypodermically. Ergot and adrenal extract are among promising remedies; they probably act by promoting vascular tone. Bullowa and Kapman,[2] in conformity with the angioparetic theory of an attack, recommend the hypodermic administration of adrenalin chlorid, and claim that it cuts short an attack in from two to ten minutes. The dose is 3 to 6 minims of the 1 : 1000 solution. Gunzel reports striking results from the high-frequency interrupted current to the vagus, accessorius, phrenic, and sympathetic nerves. In the protracted cases, associated with chronic bronchitis and emphysema, the above mixture may also be employed, though sodium iodid (gr. v—0.324) should be substituted for the bromid, and given at intervals of three or four hours.

(2) In order to prevent subsequent attacks: During the intervals

[1] Niter-paper is prepared by dipping bibulous paper (filter- or blotting-paper) in a solution of saltpeter.

[2] *Medical News*, October 24, 1903.

the physician must ascertain whether any of the numerous causes (bronchitis, gastric disorders, dust, emanations from plants) are discoverable; if so, efforts to remove them should be instituted. A methodical interrogation of the various organs of the body and their functions must be carried out, and the therapeutic or hygienic indications presented by them, if any, must be met judiciously. The nasal passages should be examined by a specialist, and any causal conditions found therein promptly removed. Rochester[1] emphasizes the importance of "so regulating the intake of food and stimulating the eliminating of waste products, that proper metabolic balance may be maintained." The patient should dine early, so that digestion may be completed before he retires. Bowel regulation is highly important. If the affection be a pure neurosis or due to bronchitis, a suitable climate may often be found in which the patient will enjoy complete immunity from asthma. The choice of the locality cannot, however, be determined by any known rules. To those who cannot adopt this plan, potassium iodid offers the best hope of relief, though its use must be long continued (gr. x–xx—0.648–1.296 three times daily). The systematic use of compressed air in the pneumatic cabinet and also the inhalation of oxygen are worthy of trial. In the belief that asthma is a special form of neurasthenia, Treupel aims to regulate the breathing, training the patient to breathe deeply with a slow inspiration, at the same time raising the arms over the head, and then during expiration applying the hands to the front and sides of the chest, squeezing the walls together to aid in expelling the last traces of air. Calcium chlorid (dose, gr. x—0.65, every two hours) is lauded by Kayser for its prophylactic and therapeutic effects. The presence of any condition of ill-health calls for appropriate treatment.

There are also certain means of prophylaxis for impending attacks. Thus, if there be premonitory symptoms, the use of such measures as strong coffee or the "hot toddy" above mentioned, Hoffman's anodyne, stramonium or belladonna cigarets, the inhalation of the fumes of niter-paper or of a few drops of amyl nitrite, may suffice to ward off the attack.

FIBRINOUS BRONCHITIS.

(*Plastic Bronchitis; Croupous Bronchitis: Mucous Bronchitis.*)

Definition.—A rare acute or chronic catarrhal affection of the bronchial mucosa, attended with the production of fibrinous casts (?) that are expectorated in severe paroxysms of cough and dyspnea. These casts, when unfolded, are found to be molds of the bronchial tubes from which they come, being shaped like the branches of a tree, and thus proving that a bronchial tube and its subdivisions had been blocked.

Pathology.—The pathology is but little understood. When examined microscopically they are seen to consist of a fibrillated base, a few scattered leukocytes and mucous corpuscles, and, rarely, gland- and blood-cells. Curschmann's spirals are often found, and within these or associated with them the Leyden crystals. First, Beschorner and later Grandy have shown the casts to be composed of mucin. In other cases,

[1] *Jour. Amer. Med. Assoc.*, vol. xlvii., Dec. 15, 1906.

however, similar studies show fibrin. In one of my own cases I found the composition of these casts to be identical with that of croupous exudates met with elsewhere, though more dense, perhaps, than the latter. Croupous bronchitis is attended with loss of epithelium in the implicated bronchi, as is the case in croupous inflammation wherever it occurs; but the answers to the questions, "Why should the affection be limited to a definite portion of the bronchial tree?" and "Why does it recur from time to time?" are obscure indeed. In fatal cases the lesions of associated or antecedent complaints, such as chronic pleurisy, pneumonia, and pulmonary tuberculosis, have been found.

Etiology.—What the irritant is that causes the condition is unknown, though streptococci and pneumococci have been found in the molds and in the mucosa. Some of the *predisposing causes* recognized are—(1) *Sex:* it being about twice as frequent in males as in females. (2) *Age:* though met with at all periods of life, it is relatively more frequent from the twentieth to the fortieth year. (3) *Season:* the seizures are most common in the spring months. (4) *Epidemic influences:* Pichini has described a group of instances that occurred in individuals in the same locality. (5) *Hereditary influence* has been traceable in a few cases. (6) *Infective diseases*, such as tuberculosis (quite frequently), pneumonia, influenza, erysipelas, scarlatina, etc., and certain skin affections, as herpes, impetigo, and pemphigus, form antecedent and associated conditions.

Symptoms.—(*a*) *The acute form* is rare. It begins with rigors and fever, soon followed by urgent dyspnea and severe paroxysms of cough, which are usually attended, soon or late, by the expulsion of bronchial casts, and sometimes rather profuse hemorrhage. Abundant expectoration usually causes amelioration of the severer symptoms. On the other hand, urgent dyspnea, oppressiveness, and severe cough, with little expectoration, are grave symptoms, often leading to fatal asphyxia.

(*b*) *The Chronic Form.*—The attacks are less severe than in the acute form and recur at irregular intervals, the interim varying from one week to a year or more. In a case observed by myself the patient has experienced a recurrence once annually (on or about May 1st). The paroxysms may occur at regular though much briefer intervals. The cases usually manifest ordinary bronchitic symptoms, with or without fever at the outset. The cough soon becomes troublesome and is paroxysmal. There is expectoration of rounded masses, which, when unravelled, are found to be true moulds of the affected tubes exhibiting a laminated structure. The larger casts (which are of the size of a goose-quill or even larger) may be hollow. They are of whitish or grayish-white color. Hemorrhage may occur.

Physical Signs.—Owing to the obstruction offered by the casts, there is a diminished amount of air entering the corresponding part of the lung. Hence the tactile fremitus, local expansion, and respiratory murmur are diminished over the affected area. The percussion note over the uninvolved portions of the lung is clear or hyper-resonant. The portions of the lung supplied by the affected tubes give impaired percussion-resonance, and if they collapse, there is a dulness on percussion. Dislodgement of the casts is followed by a normal respiratory murmur.

Diagnosis.—The presence of mucous or of fibrinous casts of the finer bronchial tubules serves to distinguish this condition. The fibrinous moulds met with in diphtheria and pseudo-membranous croup, with

extension into the bronchi, must also be eliminated. In doubtful cases a bacteriologic examination of the membranous casts should be made. If the Klebs-Löffler bacilli are then found, its diphtheritic nature is proven. In truly diphtheritic cases the membrane does not present the laminated structure.

Prognosis.—The prognosis in the acute form is quite grave; the chronic variety, though pursuing an exceedingly long course that ranges from five to fifteen years, rarely terminates fatally.

Treatment.—This is to be conducted on the same principles as those in simple acute bronchitis. In the acute form an attempt should be made to soften and separate the casts by the topical application of steam, by inhalation, and alkaline sprays (*e. g.* lime-water). Pilocarpin was employed in one instance under my own observation with apparent good results; it tends to excite free bronchial secretion. Emetics should be resorted to without delay when the signs of cyanosis show themselves.

In the chronic form nothing can be accomplished by treatment, during the intervals between the acute exacerbations, that will tend to obviate a recurrence of the attacks or to mitigate their severity.

IV. DISEASES OF THE LUNGS.

CIRCULATORY DISTURBANCES IN THE LUNGS.

CONGESTION OF THE LUNGS.

(*Hyperemia of the Lungs.*)

Definition.—The surcharge of the pulmonary vessels with blood. Two forms are recognized: (1) Active hyperemia, and (2) Passive hyperemia.

ACTIVE HYPEREMIA.

Pathology.—The blood-vessels in the bronchial mucosa often appear intensely injected, and the capillaries in the alveolar walls are prominent, while on section a scarlet-colored, frothy liquid flows. The alveolar epithelium may become swollen and granular.

Etiology.—Active hyperemia is usually a symptomatic condition, though rarely it may arise as a distinct primary affection. Active congestion of the lungs exists as an associated condition in many pulmonary affections, as pneumonia, pleurisy, bronchitis, and tuberculosis. On the other hand, active congestion of the lungs may be engendered as an independent affection by the inhalation of hot air, highly irritative substances, as well as by violent physical exercise, the ingestion of large amounts of alcohol, and strong mental emotion. Collateral hyperemia may arise from anemia of the opposite lung.

Symptoms.—The capacity of the air-cells is diminished; hence the oxygenation of the blood is markedly interfered with. There is a degree of *dyspnea* proportionate to the extent and intensity of the congestion. There is some fever (101° F.), *cough*, accompanied by *frothy, bloody expectoration.*

The **physical signs** are bilateral, as a rule, and are generally confined

to the bases. *Palpation* shows increased tactile fremitus. The *percussion-note* is impaired or, rarely, dull, and it is generally exceedingly difficult to determine the pitch of the note, owing to the fact that both sides are usually involved. When the condition is unilateral and not associated with diseases of the opposite side, the impairment is readily appreciated. The breath-sounds are broncho-vesicular in character; less frequently bronchial.

Diagnosis.—In the presence of the etiologic factors, the sudden development of *dyspnea*, *cough*, and a *frothy, bloody expectoration*, with the *physical signs* before enumerated render the diagnosis easy. When fever is present it is of a mild grade and short duration.

Prognosis.—Active hyperemia is frequently followed by collateral edema. Its course is brief, and terminates either fatally in a few hours, in perfect recovery in a few days, or in pneumonia. The condition is therefore ominous.

Treatment.—Prompt measures must be instituted in order to arrest the active fluxion. The special causative factors must be actively treated; dry and wet cups over the entire seat of congestion must be tried; and in the worst cases venesection is demanded. Following the application of the cups, turpentine stupes, sinapisms, and linseed poultices may be employed. I have observed excellent results from the use of veratrum viride combined with saline purgatives. Other cardiac sedatives may also be employed, including nitroglycerin in full doses.

PASSIVE HYPEREMIA.

Passive, unlike active, hyperemia is always a secondary condition, and is quite common. Two forms are distinguishable: (*a*) Mechanical, and (*b*) Hypostatic.

(*a*) **Mechanical Hyperemia** (*Brown Induration*).—**Pathology.**—The pulmonary vessels are distended, the lungs as a whole enlarged, and the air-cells crepitate but little, owing in great part to the encroachment upon the air-spaces by the dark venous blood. The lungs are of a reddish-brown color and afford increased resistance to efforts at cutting or tearing. On section the reddish-brown tint rapidly changes to a vivid red, from oxidation of the hemoglobin when exposed to the atmosphere. The process commences at the extreme base, extends upward, and may finally become general. The interstitial connective tissue is increased, and is often edematous, while the alveolar cells contain altered blood-pigment, usually in the form of hemosiderin and responding to the usual tests for iron.

Etiology.—Mechanical hyperemia results from the obstruction of the return of blood to the left heart, and among special causative conditions are mitral constriction, mitral regurgitation, dilatation of the right ventricle, and certain cerebral injuries and diseases. It may also be a symptom of asphyxia, and rarely it arises from pressure of tumors.

Symptoms.—The most marked feature is *dyspnea*, particularly when secondary to organic cardiac diseases with failure of the right ventricle. *Cough* is common, and an expectoration of *frothy serum* or *blood* (hemoptysis) containing pigmented alveolar epithelial cells, is the most characteristic clinical feature.

Diagnosis.—With a clear history, in addition to the dyspnea, cough,

and the characteristic expectoration, the recognition of passive hyperemia of the lungs is a simple matter. The prognosis and treatment will be considered in connection with the causative affections.

(*b*) **Hypostatic Hyperemia.**—**Pathology.**—The parts of the lung that are affected are dark in color and the vesicles distended with a transudate of blood and serum. In this way the air-cells may become emptied of air (*splenization*, *hypostatic pneumonia*), and the resulting condition is in most instances to be regarded as a mild grade of lobular pneumonia.

Etiology.—Feeble cardiac action, as in long-continued fevers, debilitating chronic affections in old persons, combines with a prolonged dorsal position of the body (gravitation thus favoring its development) in producing the condition. This explains why the condition is found usually at the bases of the lungs, and is most marked posteriorly. It is common for the same reason in carcinoma, tuberculosis, chronic rheumatism, typhoid fever, etc. Hypostatic congestion has followed morphin-poisoning, and is particularly apt to occur in persons suffering from brain lesions, notably those which induce paralysis or coma (Hare).

Symptoms.—The symptoms are wholly indefinite; indeed, none may be present. Priory has pointed out that old persons in the incipiency of the disease begin to sleep with the mouth open, so as to effect the entrance of more air. Commencing *cyanosis* may indicate the development of hypostasis, and a careful *physical examination* of the lower lobes of the lungs will show increased fremitus, slight dulness, diminished vesicular murmur, and, in the higher grades, bronchial breathing, with liquid bubbling râles.

The **prognosis** is based upon the character of the underlying affection.

Treatment.—This is an affection in which the treatment of causes alone will suffice, save in instances secondary to organic heart-affections, in which prompt bleedings are to be advocated. From a pint to a quart of blood should be taken, and I have seen happy results from the employment of this measure in extreme cases. Tapping the right auricle when the blood refuses to flow from an arm vein has been successfully accomplished by competent surgeons. The patient's posture must be changed from the dorsal to the lateral, and even ventral, and as soon as possible he should be gotten out of bed.

PULMONARY EDEMA.

(*Edema of the Lungs.*)

Definition.—An effusion of serous fluid into the air-vesicles and interstitial lung-tissue. Pulmonary edema is scarcely to be regarded as an independent affection, but as a secondary condition, being in most instances associated with pulmonary congestion.

Pathology.—It consists of a transudation of serum into the alveolar walls, interstitial connective tissue, and air-cells. Rarely the process is limited to the interstitial tissue. Two forms may, for the sake of convenience, be recognized:

(*a*) **Collateral Edema** (*Inflammatory Edema*).—This is usually local in character, circumscribing an area of the lung affected by pneumonia, abscess, or pulmonary infarction, and is the result of a mild inflammatory process affecting the vessel-walls. When the condition follows

hypostatic congestion the terms "hypostatic edema" and "splenization" have been applied.

(*b*) **General Pulmonary Edema.**—If congestion be not associated, the portions of the lungs involved look pale; when pulmonary congestion or pigmentation of the tissue is present, the lung appears darker than the normal and the serum is blood-tinged. The weight of the lung-tissue, owing to the more or less airless condition of the alveoli, is increased, and yet, though heavier than the normal lung, the affected tissue does not sink in water. To the feel it is boggy, and pits on pressure, while on section a serous or sero-sanguinolent (if congestion be present) fluid of low specific gravity, and poorer in albumin than plasma, flows from the cut surface. Edema is most frequently observed at the bases of the lungs, though it may become general. Hydrothorax may be present.

The *mode of production* of pulmonary edema is not definitely known. Increased fluidity of the blood on the one hand, and increased tension in the pulmonary vessels on the other, seem to be influential factors in many cases. The heightened blood-pressure may be in great part due to a failure of cardiac power, and particularly to failure of the left ventricle (Welch). When weakness of the left is out of proportion to the weakness (paralysis) of the right ventricle, the tension in the pulmonary capillaries is apt to be greatly increased, at least until transudation of serum is induced. Edema also occurs as a result of weakness of the right ventricle alone. Obstruction to the outflow, such as occurs in weakening of the left ventricle, or even obstruction in the aorta, leads to heightened tension and, secondarily, to paralysis of the right ventricle. The third factor entering into the production of pulmonary edema is the increased permeability of the vessel-walls, due to impairment of their nutrition and "disturbance of the cardiopulmonic innervation" (Huchard). This usually arises in connection with toxic and infectious diseases, when the blood also exhibits more or less change, as in cachectic states, uremia, general septicemia, and the like. Instances are met with in which pulmonary edema, due to vasomotor relaxation from toxic states, develops suddenly.

Etiology.—Pulmonary edema is secondary to pneumonia and acute and chronic affections, but not with any degree of constancy; nor is it especially liable to be associated with congestion or with low grades of inflammation of the lungs. Among the diseases of which it forms a terminal condition are—valvular affections of the heart, fatal forms of anemia, acute and chronic Bright's disease, cerebral lesions (hemorrhage, traumatism), and acute infectious fevers with failure of cardiac power. Edema may follow thoracentesis and, rarely, the intravenous injections of saline solutions.

Symptoms.—In edema of the lungs the air-space is lessened in direct proportion to the amount of serum occupying the alveoli; hence *dyspnea* is always present and is often a conspicuous symptom. There are *cough* and *bronchorrhea*. The *sputum* is usually abundant and frothy, and is expectorated with difficulty. At times, and especially in the acute forms, it is tenacious and may give rise to alarming laryngeal obstruction. It is blood-stained if congestion be combined. The condition does not give rise to elevation of temperature, except in the inflammatory type, in which fever is constantly present. The pulse is accelerated and feeble, and cyanosis, particularly in cases of collateral edema, usually appears. The extremities are cool and often livid.

Physical Signs.—The reasons adduced to explain the dyspnea likewise render intelligible the physical signs encountered. There is dulness, though rarely complete, over the areas involved; the vesicular murmur is feeble or absent or there may be broncho-vesicular breathing. Since the bronchioles contain serum, small bubbling râles are audible with inspiration and at the beginning of expiration over the seat of the edema.

A *recurrent variety* has been studied by Crummer, Riesman, and others, coming on without any apparent exciting cause and often proving fatal. "The chief symptoms are agonizing dyspnea, cyanosis, cough, expectoration of frothy, albuminous fluid, and profound prostration" (Riesman). Recovery from an attack is frequent and sudden.

The **diagnosis,** with a clear history, is based upon the incomplete dulness that is usually bilateral and most marked at the bases, upon the bubbling râles heard over the corresponding area, and upon the absence of any febrile movement, except the latter be due to some underlying affection. *Hydrothorax* bears some points of resemblance to edema of the lungs, but in this condition, unlike edema, the upper level of dulness is movable on change of position of the patient. Moist râles, audible in pulmonary edema, are absent in hydrothorax. *Broncho-pneumonia* may be mistaken for pulmonary edema, though it has a different mode of onset. It is also accompanied by fever, glairy, tenacious expectoration, and more sharply localized areas of dulness than appear in edema.

The **prognosis** is governed by the pre-existing condition to which the edema is due. Thus, if secondary to a general dropsy due to renal or cardiac disease, it often destroys life with great rapidity. Inflammatory edema, following lobar pneumonia, is also grave in the extreme.

Treatment.—The treatment is that of the associated or causative affections. These must be sedulously treated, and the limitation of the transudation and the direct removal of the serous effusion from the lungs is of great importance. We should frequently change the position of the patient's body, so as to prevent the gravitation of blood to the dependent portions of the lungs. I have witnessed excellent results from the use of dry cups placed over the thorax, particularly over its posterior and lateral aspects, and renewed at intervals of six to eight hours. The number applied should range from one and a half dozen to three dozen. In aggravated forms that develop quickly prompt venesection is imperatively demanded. This is a measure which, if resorted to at the proper moment, will often rescue the patient from imminent danger. The condition of the heart and kidneys must receive attention. Nitroglycerin and atropin, particularly the latter in full dosage (gr. $\frac{1}{50}$, to be repeated in one hour if required), are often serviceable. Stengel advocates morphin in small doses in the recurrent variety. Tincture of strophanthus (♏iij every three hours) is effective in pulmonary edema in children.

HEMOPTYSIS.

(*Broncho-pulmonary Hemorrhage.*)

Definition.—An expectoration of blood. Its source may be the bronchial mucous membrane (usually the small bronchi), and less frequently eroded vessels in lung-cavities or their walls; rarely the larynx, trachea, and larger bronchi. When from the bronchial tubes, the term *bronchorrhagia* should be applied. The source of the hemorrhage, how-

ever, is not always easily demonstrable, even when it has resulted fatally and the lungs are minutely examined.

Pathology.—The lesions are often microscopic, and consist for the most part of ruptured capillary blood-vessels, though larger vessels may also become the seat of erosion or rupture. After death the bronchial mucosa is sometimes found to be swollen, bleeds easily, and is of a dark-red color—soon becoming decidedly pale. The lung-tissue proper may look paler than in the sound lung. In advanced pulmonary tuberculosis the lung-cavity may contain a ruptured aneurysm, or mere ulceration of an exposed vessel may be observed. I have witnessed small, dark-red dense masses in the air-sacs scattered throughout the lung whence came the hemorrhage. Doubtless these are blood-coagula, which result from the clotting of the blood after the latter has been carried into the alveoli. Various associated lesions may be observed.

Etiology.—(1) **Pulmonary Affections.**—(*a*) *Pulmonary congestion* from whatever source may result in hemoptysis, usually of small amount. There are many causes that excite congestion of the lungs, some of which reside in adjacent organs, it being common in organic disease of the heart, and particularly in disease of the mitral segments. That form of pulmonary congestion which is associated with other affections of the lungs, as well as primary active congestion due to inhalation of hot air, irritating substances, and violent physical exercise, may also result in hemorrhage. (*b*) *Hemorrhagic infarction* may lead to slight hemorrhage (*vide* Pulmonary Embolism). (*c*) *Croupous Pneumonia.*—In this disease hemorrhage is caused by rupture of the capillaries, and the blood, when expectorated, has undergone a change and become rusty-colored. (*d*) *Pulmonary Tuberculosis.*—This is pre-eminently the most common cause. Of 5302 cases analyzed by the writer, hemoptysis was found in 1950, or 36.6 per cent. It is to be recollected that hemorrhages due to tuberculosis are less apt to take place in higher altitudes than at sea-level; but, as pointed out by Bonney,[1] they are decidedly more severe and associated with more shock when they occur. Hemorrhage may take place early when it originates from a sharply limited and minute tuberculous focus, and it may also be attributable to congestion. Undoubtedly its exact source is the mucosa of the small bronchi; later it is the direct consequence of the ulceration of an artery or of the rupture of an aneurysmal sac that has its seat in a branch of the pulmonary artery. After the tuberculous cavities have healed, calcareous masses are, from time to time, expectorated, together with more or less blood. (*e*) *Ulcers of the Larynx, Trachea, or Bronchi.*—Rarely, ulcers in adjacent structures erode the larger branches of the pulmonary artery and cause copious and speedily fatal hemorrhages. Osler observed a fatal hemorrhage in a case of chronic bronchitis with emphysema. (*f*) *Fibrinous bronchitis* induces hemoptysis by rupturing the capillaries in the bronchial mucosa at the time of separation of the bronchial casts. (*g*) *Carcinoma of the lung* produces frequent expectoration of blood. (*h*) *Gangrene* and *abscess of the lung.* (*i*) *Parasites* (Paragonimus Westermanii). (*j*) *Injuries* to the thorax.

(2) **Diseases of Other Organs than the Lung.**—(*a*) *Affections of the heart* act as a cause, and especially advanced mitral disease with pulmonary congestion. It not infrequently develops during the stage of adequate compensation. In a preponderating proportion of the latter

[1] *Pulmonary Tuberculosis and its Complications*, p. 130.

instances the hemorrhage is slight, but it may be profuse and recur at intervals for many years. (*b*) *Aneurysm* of the branches of the *pulmonary* artery and of the arch of the aorta (usually with rupture of its coats) is a rare cause of hemoptysis.

(3) **Certain diseases,** such as *purpura hæmorrhagica*, *scurvy*, *anemia*, *hemophilia*, and *malignant forms of certain acute infectious diseases* (*e. g.* yellow fever), cause hemoptysis. In this class of cases the hemorrhages are due either to a diseased condition of the vessel-walls or to blood-changes.

(4) **Vicarious hemoptysis** is not uncommon during menstruation or when amenorrhea is present. Unless occurring at the time of the regular menses it is not to be regarded lightly, and is of the same significance as when taking place in the male.

(5) **Arthritic (Gouty) Endarteritis.**—According to Sir Andrew Clarke this is a common cause of recurring hemorrhages in aged persons.

Symptoms.—Hemoptysis is so commonly a symptom of that most frequent and dread disease, phthisis, as to raise suspicions of the latter in the minds of the laity and physicians as soon as it occurs. It is appropriate, therefore, to note, first, the features of hemoptysis when dependent upon pulmonary tuberculosis, and then to point out its clinical peculiarities when due to other conditions.

In **incipient pulmonary tuberculosis** hemoptysis develops suddenly as a rule, a *warm*, *saline taste*, lasting but a few moments, generally preceding the expectoration of blood. The blood is *coughed* up, and the bleeding may last only a few minutes or may continue for days, the sputum being apt to remain blood-stained for a longer period. The immediate effect of the hemorrhage, however slight, is to alarm the patient, inducing, besides mental agitation, cardiac palpitation and other nervous concomitants. A small hemorrhage is not attended with any other results, but large ones give rise to the symptoms of *shock*, combined with those of *symptomatic anemia*. When the hemorrhage is large, blood to the amount of a mouthful may be ejected with each cough, and in these instances the effect of the profuse bleeding is evidenced by such symptoms as vertigo, syncope, cold extremities, excessive pallor, perspiration, and a rapid, small, feeble pulse. This is followed, if the attack does not prove speedily fatal, by considerable restlessness, and later not infrequently by mild delirium and more or less fever.

In comparatively rare instances the same patient has a single hemorrhage; more frequently he has several at shorter or longer intervals. Large or small bleedings may precede by weeks, months, or even years any rational symptoms or physical signs of pulmonary tuberculosis. In such instances the pre-existence of latent foci of disease may be assumed.

In *quantity* the hemorrhage varies greatly, ranging from less than an ounce to a pint or more. In advanced cases in which cavities have formed large vessels may become eroded, followed by copious and dangerous hemorrhage. Fatal hemorrhage may take place into a cavity without the occurrence of hemoptysis, as in a case dissected by Osler at the Philadelphia Hospital. The distinctive characters of the blood discharged are mainly as follows: *bright color*, very *frothy* (being mixed with air), and *not clotted*. In the case of hemorrhage proceeding from a large cavity, the blood may, rarely, pour forth in a free, dark stream.

Physical Signs.—These are, for the most part, negative. Quite commonly moist bronchial râles are audible on auscultation; palpation and percussion should not be practised either during or immediately after the hemoptysis.

Hemoptysis Not Due to Pulmonary Tuberculosis.—(*a*) In *affections* of the *mitral* and *aortic valves*, especially in mitral stenosis, hemorrhage from the bronchi is not uncommon, and the way in which these lesions lead to pulmonary congestion (*vide* discussion of Organic Affections of the Heart). During the progress of these cases, hemorrhages often occur at considerable intervals; they may either be slight, lasting only a few minutes, or quite free, extending over periods of a few days or a week.

(*b*) As a rule, in the beginning small hemorrhages occur for several weeks from pressure of an *aneurysmal dilatation* upon the bronchial mucosa, or there may be weeping of blood through the exposed layers of fibrin composing the walls of the sac. The bleeding point can be discovered with the laryngscope, when an aneurysm of the innominate or of the aorta impinges upon the trachea. A large and often quickly fatal hemorrhage occurs from rupture into the respiratory tract.

(*c*) "*Arthritic hemoptysis*" is undoubtedly associated with gouty, degenerative changes in the terminal blood-vessels of the lung, though no coarse pulmonary lesions are induced by the recurring hemorrhages. Although the hemorrhages may occur at intervals for years, as a rule they finally become arrested, and only rarely lead to a fatal issue. I have never observed this form of hemoptysis occurring independently of *chronic bronchitis*. In *emphysema* and *chronic bronchitis* small hemorrhages may occur, and occasionally coagula in the form of casts are formed in the bronchi and afterward ejected. It is probable that the source of the large bleedings is an *ulcer* in the bronchial mucosa.

(*d*) The hemoptysis that is connected with the *menstrual function* is of frequent occurrence. I saw recently a patient in whom free bleeding has occurred at intervals of four weeks for a couple of years, with an absence of the menses. In another instance, a patient of Dr. Byers, recurring hemorrhages of the lungs took place instead of the regular menstrual discharge for three successive months, and a comparatively rapid and fatal form of phthisis was developed. This case typifies a large class that is especially prone to develop pulmonary tuberculosis.

(*e*) The preceding group is to be distinguished from those cases in which *trivial bronchial hemorrhages* sometimes occur, and in delicate, hysterical females. Although these bleedings are accompanied by cough, it is not uncommon to find, upon careful examination, that the blood comes from the upper air-passages.

(*f*) Hemoptysis may result from *severe injuries* inflicted upon the thorax, and last for days.

(*g*) A person may have a single or many recurring attacks of hemoptysis *without assignable cause*, if we except severe muscular strain or intense mental excitement. Although pulmonary tuberculosis does not supervene in instances of this sort, yet not a few may be excited by a permanently limited tuberculous focus which may be indeterminable by the usual methods of examination. I have more than once seen a cure result from an active course of treatment with creosote and appropriate hygienic measures. In well-marked instances of the kind a complete arrest of the trouble resulted from a change of climate

(*h*) *Parasitic hemoptysis* due to paragonimus Westermanii. The sputum resembles that of lobar pneumonia with intermittent hemoptysis.

Differential Diagnosis.—A reliable diagnosis necessitates the exclusion of hemorrhage from the higher air-passages, pharynx, esophagus, and stomach. In *epistaxis* the blood may directly enter the nasopharynx, excite cough, and be discharged as in hemoptysis. An examination of the nasal chambers should be made when epistaxis is suspected. Bleeding may take place from the gums, from chinks in the pharynx, or from varicose veins. If the seat of the bleeding be the *pharynx*, the hemorrhage is not free, the blood being commingled with a preponderating proportion of mucus; if from the gums, it may be more copious (as in ptyalism or scurvy). An inspection of the mouth will decide the question. Strümpell distinguishes hysteric hemoptysis by the smaller bleedings, the absence of pus-elements, and the large amount of squamous epithelium, leptothrix, and the food-remnants present.

Hemoptysis must be distinguished from *hematemesis* (*q. v.*).

Prognosis.—The gravest apprehensions are constantly entertained by sufferers from hemoptysis, but immediately fatal results are of rare occurrence; and of this fact the patient should be repeatedly assured by his physician. In case, however, of thoracic aneurysm the consequences of hemoptysis are fatal. With reference to the effect of hemoptysis upon tuberculous pulmonary disease, opinions differ widely. Prior to the existence of cavities it often exerts a favorable influence upon the disease. On the other hand, if cavities exist, an opposite effect is observed. Some blood finds its way into the bronchi below the point of bleeding and into the air-cells, causing at times irritation and even lobular inflammation. Thus hemorrhages may aid in rendering the tissues susceptible to tuberculous infection. In cases of profuse hemorrhage, due to the erosion of large branches of the pulmonary artery in phthisical cavities, death may be suddenly induced, and is caused largely by inundation of the lung and the consequent impossibility of respiration. Fatal hemorrhages are less common in the female than in the male sex.[1]

Treatment.—Since the hemorrhage is ascribable to (1) congestion of the bronchial mucosa, (2) erosion of the vascular walls, and (3) blood-changes, obviously the treatment of individual cases must be modified according to the character of the causative condition.

In many instances of *hemoptysis due to congestion of the bronchial mucosa* the hemorrhages are, comparatively speaking, slight; hence, apart from keeping the patient at absolute rest for from twenty-four to forty-eight hours after the cessation of the bleeding, little treatment is required. If free, the physician's aim should be to decrease the force of the heart's contraction, and to accomplish this end the patient should be placed in bed, and not allowed to change his position nor to speak above a whisper. The patient should lie on the diseased side (if this be known). The affected side of the chest may be strapped with adhesive plaster. The diet should be light, nutritious, and non-stimulating, all hot drinks and alcoholics being prohibited. Among cardiac sedatives employed with a view to reducing the rapidity of the heart's action and lowering the blood-pressure are the ice-bag to the precordia, and aconite and other arterial sedatives internally.

[1] *Trans. Amer. Climat. Assoc.*, 1909, xxv., 27, by the writer.

Arthur Foxwell[1] recommends venesection in cases in which *venous congestion* is present, and also lays stress upon measures that confine the blood to the systemic circulation—*i. e.*, nutritious food, large doses of the nitrites, hot foot-baths, leeches to the anus, and ligatures applied to the thighs and arms. Brown, Otis, and others advise that the blood-pressure be frequently observed, and if found to be high, nitrite of sodium or nitroglycerin should be employed. The pulmonary capillaries may also be effectually depleted by the use of salines. I have found dry cupping over the chest of the greatest service in cases dependent upon congestion. Eating ice and partaking freely of iced drinks are also useful measures. If the attack tends to become prolonged and exhausting, we may increase the coagulability of the blood by the use of gallic acid, acetate of lead, sodium chlorid, or calcium chlorid. Hemoptysis is usually accompanied by *cough* that constantly disturbs the vascular serenity and excites fresh bleeding; it demands opium or morphin (hypodermically). In blood-spitting due to the gouty diathesis Mays recommends the salicylate of sodium. Wilkinson claims that tuberculin has an extraordinary effect in preventing and arresting hemorrhage. Amyl nitrite in doses of from 3 to 9 minims often promptly arrests the bleeding by causing an immediate fall in blood-pressure at the bleeding points, "thus giving time for clotting to take place" (Calvert). Fish[2] reports excellent results from the use of chloroform in 19 cases; he gives from 2 to 4 c.c. by inhalation. Subsequently the inhalation of 15 to 20 drops every hour is continued for a few days.

When hemoptysis is associated with *organic disease of the heart*, the main indication is to strengthen that organ by bodily rest and quiet and by the use of cardiac tonics, especially digitalis. I have had under observation for several years a young physician who has been suffering from frequent, marked hemoptysis, due to mitral regurgitation, and in whose case the bleedings are readily controlled by the free use of digitalis.

When in *thoracic aneurysm* or *advanced pulmonary tuberculosis* the blood is ejected in mouthfuls, we may safely infer that erosion of a vessel or rupture of the aneurysm has taken place. Here the object is to bring about the formation of a thrombus that will arrest the hemorrhage. Perfect quiet in the horizontal position tends to allay the vascular excitement, and the induction of fainting by venesection is a measure worthy of a trial. Opium is contra-indicated in these cases, since if cough be checked inundation of the bronchial system with the blood (the chief danger) will be favored. R. H. Babcock gives an immediate injection of atropin sulphate (gr. $\frac{1}{50}$—$\frac{1}{25}$) when hemorrhage occurs from a cavity. Finzi reports good results from the induction of artificial pneumo-thorax.

In all instances of hemoptysis treatment should not end with cessation of the hemorrhage. A tendency to recurrence is manifested in many cases, and for small, repeated hemorrhages, turpentine and aromatic sulphuric acid may prove of service. The patient should not be allowed to indulge in a stimulating diet; he should eschew tobacco and alcoholic stimulants, and avoid all physical and mental strain. Every source of bronchial irritation should be carefully avoided, and attacks of bronchitis, however mild, should receive the most careful attention. A climate far removed from the seaside is best. Moderate exercise is serviceable, as well as a liberal amount of nutritious food.

[1] *Brit. Med. Jour.*, 1894, p. 194. [2] *Jour. Amer. Med. Assoc.*, 1909, lii., 1918.

PNEUMORRHAGIA.

(*Pulmonary Apoplexy.*)

Definition.—An escape of blood into the air-cells and interstitial tissue, with or without laceration of the pulmonary parenchyma.

Pathology.—It may be, though rarely, (*a*) diffuse, when the lung-tissue is lacerated, as in cerebral apoplexy; or (*b*) circumscribed, as when the blood is effused into the air-cells and the interstitial tissue, without rupture of the parenchyma. (See Pulmonary Infarction, *infra.*)

Etiology.—*Diffuse pulmonary apoplexy* is caused by the rupture of a thoracic aneurysm that has become adherent to the surface of the lung. Its most common cause is traumatism, especially penetrating wounds, but adult life and the male sex are to be regarded as predisposing factors. Septico-pyemia and cerebral disease are causes.

Symptoms.—These are ill-defined. *Profuse hemoptysis, urgent dyspnea*, and *cyanosis*, followed by increasing evidences of *collapse*, together with a clear history, should raise suspicions of the existence of diffuse pneumorrhagia.

The *physical signs* are indicative of extensive consolidation arising suddenly, but not of the nature of the lesion.

The **prognosis** is practically hopeless, and abscess or gangrene may result if these cases recover from the immediate effects of the hemorrhage.

Treatment.—Absolute rest of the body in the horizontal position is the one measure that offers a slight prospect of alleviation, for thus the formation of a clot, followed by arrest of the hemorrhage, is encouraged. It is unwise to use opium to allay the cough, since the action involved assists in ejecting the extravasated blood, which will, in consequence of gravitation and the effect of respiration, submerge speedily so much of the lung-tissue as to hasten the fatal termination. Ergot is contra-indicated, but the internal and external use of cold has been highly recommended. With the onset of collapse cardiac stimulants become absolutely necessary, though many cases are so rapidly progressive as to reach a moribund state before remedial agents can be applied by the physician.

PULMONARY EMBOLISM.

(*Hemorrhagic Infarction; Embolism of the Lungs.*)

Pathology.—Embolic infarctions are firm, airless, brown or black, wedge-shaped masses, with their bases usually at the pleura, which soon becomes lustreless and covered with fibrin. The infarctions may be single or multiple, and may occupy the greater portion of the lobe; in most cases, however, their size equals that of a walnut. Their most frequent seat is at the back of the lower lobe. The microscope shows the presence of leukocytes and red blood-corpuscles in the air-cells and in the alveolar septa. Collateral congestion and edema are frequent concomitants, and, rarely, pneumonic consolidation.

Etiology.—The condition is produced by the blocking of the pulmonary arteries by an embolus or thrombus. When the circulation in the pulmonary capillaries is feeble, hemorrhagic infarction may be the result of stasis, and this is probably the most frequent form. It is met in diseases of the lungs and also in mitral affections. The plug that

occludes the blood-vessel may be composed of leukocytes, as in leukemia, and the chief sources of the emboli are the thrombi in the right heart, in consequence of dilatation, and in the systemic veins. Infectious emboli, resulting in abscesses, occur (*vide* Abscess of the Lungs). An embolism of placental cells in cases of eclampsia has been described. Occlusion of a branch of the pulmonary artery cuts off completely the circulation to the territory supplied by that branch, and hemorrhagic infarction occurs—venous extravasation, with expression of air.

Symptoms.—Not all infarctions give rise to symptoms; on the contrary, occlusion of a main branch of the pulmonary artery usually terminates life speedily. The latter accident occurs not infrequently in connection with organic disease of the heart, and if death be not the immediate result or if a narrower branch be occluded, alarming symptoms ensue, such as *syncope*, *dyspnea*, *pain in the side*, and *convulsions* with *unconsciousness*. The first and most distressing symptom is dyspnea, attended by frantic efforts at breathing and by great mental anxiety. Occasionally *hemoptysis* is an early symptom, and of primary significance if it occur in a patient suffering from mitral disease. *Cough* usually supervenes, accompanied by the expectoration of dark, gelatinous, mucoid masses. Large lymph-cells containing blood-corpuscles are found in the sputum, most commonly in instances of organic cardiac affections.

The **physical signs** may either be negative—as, for example, when the infarctions are small or deeply located—or they may give information as to the seat and extent of the affected part. When present they are those of sharply-localized consolidation (increased fremitus, percussion-dulness, moist râles, bronchial or broncho-vesicular breathing). It is not improbable that in many cases the physical signs are due, in great part, to associated conditions, such as bronchitis, edema, or collateral consolidation. The appearance of the friction-sound in the course of suspected cases is a great aid in diagnosis. The *heart's action* becomes enfeebled, the *pulse* is small and frequent, and the surface of the body is cool and frequently bedewed with cold sweat. *Fever* may either be present at the onset or absent throughout. The signs of embolic abscesses in the lungs will be elsewhere detailed (*vide* Pulmonary Abscess).

Diagnosis.—To establish the diagnosis of pulmonary embolism there must be a clear history of some etiologic condition, and the sudden appearance of such symptoms as dyspnea, cough, bloody expectoration (in particular), chest-pain, loss of consciousness, and convulsions, corroborated by the physical signs of a sharply-defined spot or spots of consolidation.

Prognosis.—The prognosis differs with the character of the primary condition. On the whole, it is exceedingly grave, though the absorption of an embolism, followed by the disappearance of the urgent symptoms, is not impossible. In case death does not occur soon, infarcts may give rise to abscess or gangrene, due either to bacteria in an original embolus or to their entrance through the air-passages. In other cases an infarct may undergo fibroid change and contraction, and may even calcify.

Treatment.—Beyond procuring absolute rest of the body and a relief from the distressing symptoms, the treatment should be aimed at the affections on which this form of embolism depends. Dyspnea and pain may require the hypodermic use of atropin and morphin, preferably in combination. Heroin relieved the dyspnea in one of my cases.

CHRONIC INTERSTITIAL PNEUMONIA.

(*Fibroid Induration; Cirrhosis of the Lung.*)

Definition.—A chronic inflammation of the lungs, characterized by the formation of fibrous or connective tissue. It may occur as a primary or as a secondary affection.

Pathology.—Two leading forms may be recognized: (*a*) *Local*, and (*b*) *Diffuse*, though these do not demand separate description. It is a unilateral affection, and the lung of the side involved is much shrunken, its dimensions in some cases being incredibly small. It lies tightly against the spine, and has frequently been overlooked. The heart occupies the affected side, being drawn in that direction during the progress of the disease, and it is enlarged, principally owing to hypertrophy of the right ventricle. The pulmonary artery is the seat of atheromatous change. The other lung is overdistended (*compensatory emphysema*) and may encroach upon the mediastinum. Intrapleural and pleuro-pericardial adhesions may be exceedingly firm and thick or only moderately so, and rarely the pleuræ are intact. The cut surface of the affected lung is hard, dry, airless, shiny, and usually light gray in color (rarely reddish-yellow), and the lung-tissue cuts with great resistance. The blood-vessels and bronchi may be observed gaping in the cut section. Cavities may be due to bronchiectasis or to the superaddition of a tuberculous process. Phthisical cavities may often be discriminated by their usual situation at the extreme apex. The lung that is unaffected by the fibroid process is also quite often the seat of tuberculous change.

Etiology.—The disease is almost invariably secondary, and very generally accompanies prolonged inflammatory and chiefly local changes in the lungs. It may also follow acute inflammatory processes. Examples of **localized interstitial pneumonia** are seen in connection with pulmonary tuberculosis, emphysema, syphilis, hydatids, and fibroid induration secondary to thickening of the pleura.

Diffuse interstitial pneumonia has a variety of causes: (*a*) It may follow *acute lobar pneumonia* in cases in which resolution is delayed, and here the fibrinous exudate filling the air-cells becomes organized into connective tissue. Fibrous tissue is also substituted for the alveolar walls. The condition is exceedingly rare.

(*b*) Pneumonia, appearing as a complication in influenza, is very liable to produce chronic interstitial pneumonia.

(*c*) The disease may also result from atelectasis due to compression, as by aneurysms or neoplasms.

(*d*) It most frequently, however, follows *broncho-pneumonia* of either acute or subacute form (Charcot). The process starts in the bronchi and extends to the surrounding lung-tissue, till finally an entire lobe, or even an entire lung, may become involved. Tuberculous broncho-pneumonia also leads to the production of new fibrous tissue, but here the process is a conservative one (*vide* Pulmonary Tuberculosis), and hence is not to be classed with chronic interstitial pneumonia.

(*e*) The initial lesions may be located in the adherent *pleura*, with secondary involvement of the lung, connective-tissue bands ex-

tending into its substance. The bronchi are inflamed and sometimes dilated.

Chronic interstitial pneumonia may, however, exist without implication of the pleura, and in view of this fact the primacy of pleural thickenings cannot be granted without reserve when they form a part of the lesions of fibroid induration.

The various forms of the disease thus far described arise *secondarily*. It may also occasionally originate as a *primary* affection (1) from the inhalation of different forms of dust (*vide* Pneumonokoniosis). (2) Delafield describes "a special form of lobar pneumonia." He contends that lobar pneumonia terminates only in resolution or in death, and that this special disease, with its production of newly-formed connective tissue, is a distinct form of inflammation. The variety described by Delafield runs a subacute or even chronic course, and terminates by crisis. It is an exudative inflammation, with the formation of new tissue from the onset. The consolidated areas are not so large as in ordinary pneumonia, and sections lack the granular character of the latter.

Symptoms.—The patient suffers from *cough*, which increases in intensity with the progress of the affection. There is a mucous, sero-mucous, or rarely bloody expectoration; *dyspnea* occurs early, and frequently is present only on ascending heights; uneasiness, or even *pain*, over the side of the chest involved may be experienced. In cases in which the bronchi become dilated the characteristic symptoms of bronchiectasis are superinduced. The *general symptoms* consist merely in a loss of flesh and of strength. *Fever* is altogether absent.

Physical Signs.—*Inspection.*—The chest-wall of the affected side is retracted, while the healthy lung is enlarged (*compensatory emphysema*). The spinal column is curved laterally. The affected side is fixed during respiration, and the heart is displaced by traction toward the affected side. If the left lung be involved, the apex-beat will be displaced to the left and slightly upward; if the right, the apex-beat will be observed to the right of its normal position. The ribs approximate, thus obliterating the interspaces, and the shoulder droops over the shrunken chest-wall.

Palpation.—The tactile fremitus is usually increased; if the pleura be much implicated or thickened, however, fremitus may be decreased. Palpation discovers no expansile motion.

Percussion.—The percussion-note varies. Dulness is common, owing to consolidation of the lung, but flatness is sometimes met with, and a tympanitic or amphoric note is occasionally elicited over a dilated bronchus.

Auscultation.—The breathing is bronchial or more or less sonorous as a rule, and over bronchiectatic cavities it is cavernous or, rarely, amphoric. Near the base it is frequently feeble, distant, or even altogether suppressed. Subcrepitant, sonorous, sibilant, or gurgling râles may be audible, and dry, creaking, or leathery friction-sounds may also be heard.

Prognosis.—The course of the complaint is exceedingly chronic, lasting over many years. Death may result from an intercurrent attack of acute pneumonia affecting the other lung. The disease always shortens life, and may be the direct cause of death. Rarely a fatal issue is due to dilatation of the right heart, followed by tricuspid regurgitation.

Treatment.—The condition is incurable. The patient should, however, be placed under the best sanitary conditions, and if practicable he

should make a permanent change of climate. A suitable resort should be selected in accordance with the rules indicated in the treatment of Pulmonary Tuberculosis, and every effort should be put forth to improve the general nutrition of the patient. Due attention should be given to the associated bronchitis, as well as to any symptoms that may arise during acute exacerbations.

BRONCHO-PNEUMONIA.

(*Capillary Bronchitis; Catarrhal Pneumonia.*)

Definition.—An inflammation of the minute bronchi and air-vesicles, due either to the extension of inflammation from the capillary bronchi to the air-vesicles or to an inflammatory process set up in atelectatic lobules.

Pathology.—*Macroscopically*, the lungs present decided variations in persons who have died of broncho-pneumonia. On the pleural surface may be noticed purplish or slaty patches, often sunken (atelectasis), intermingled with the more elevated patches of healthy lung and grayish consolidation, and smoother and more moist than croupous pneumonia. Similar appearances are presented by the cut surface. On pressure fluid exudes—edematous from the healthier areas, and grayish and puriform from the consolidated areas. The mucosa of the large bronchi may look natural, though frequently it is congested, while the small bronchi usually contain more or less muco-purulent material. Their walls are greatly thickened, and on section the cut surface presents a nodular appearance. Dilatation of the smaller bronchi may be observed, and minute consolidated areas, varying in size from that of a pin's head to that of a pea, may be seen surrounding the thickened walls of the bronchi. When, as frequently happens, they become confluent, large areas—an entire lobe and even an entire lung—of lung-tissue may become consolidated. The solidified zones are firm to the touch, being destitute of air, and at first they contain blood; hence their color is a dark red, later turning to a grayish hue. The condition is usually bilateral. As a rule the bronchial glands are swollen and inflamed. In the non-consolidated portions of the lung the air-cells are considerably dilated.

The essential lesion is a productive inflammation of the bronchi and of the immediately surrounding air-spaces. The inflammation is from the first not exudative, but productive; that is, with the formation of new tissue (Delafield). This form of inflammation may merge into sclerosis of the lung or chronic thickening of the pleura. *Microscopically*, the walls of the bronchioles and alveolar passages are seen swollen and infiltrated with cells; they likewise contain plugs of mucous exudate, most marked near the centre of the process. The air-cells toward the periphery show much less exudate. The latter consists of serum, some mucus, and many swollen cells from the alveoli

(soon showing fatty degeneration), leukocytes, and also red blood-cells in small numbers. Fibrin is seen in small quantity if at all.

In deglutition- and aspiration-pneumonia the leukocytes are present in much larger numbers, and the exudate tends to suppuration, while in the hemorrhagic forms the red blood-cells are relatively increased.

Among the associated lesions to be mentioned are—(*a*) Catarrhal inflammation of the mucous membrane of the bronchi; and (*b*) Exudative inflammation of the air-cells, which become filled with epithelium, fibrin, and pus, with resulting consolidation of the lung. The epithelial cells lining the air-sacs, since they are more numerous in young children than in adults, form a larger part of the inflammatory exudate in the former than in the latter. (*c*) The pulmonary pleura is often coated with fibrin, but less regularly than in croupous pneumonia.

Etiology.—(1) A marked predisposing influence is *age*, the disease being most prevalent amongst young children. In them it may appear in association with measles, whooping-cough, scarlet fever, and diphtheria, but not infrequently it is entirely independent of these diseases. Infants are especially susceptible to the affection, most instances of pneumonia at this period of life being of the lobular form. Other conditions that act as predisposing factors in children are improper exposure to cold, unsanitary surroundings (especially impure air), rickets, and chronic diarrhea. Broncho-pneumonia is also frequent in the aged, often being occasioned by certain debilitating causes and chronic diseases that are common to advancing years (emphysema, gout, chronic valvulitis).

(2) *Season.*—The affection prevails especially in the winter and spring months; particularly is this the case in those forms that are unassociated with the acute infectious group of diseases.

(3) It also supervenes as a complication in such acute infectious diseases as influenza, typhoid fever, erysipelas, and small-pox, and is of serious import. According to my own observations, it is more commonly met with in the diseases above mentioned than is lobar pneumonia.

(4) The *inhalation of food-particles and other substances* often serves to convey the agents of inflammation to the lobules of the lungs. A long-continued recumbent posture predisposes the patient to broncho-pneumonia. It is, however, in conditions in which the larynx and bronchi have totally or in part lost their sensitiveness—as in coma due to apoplexy, uremia, and allied cerebral states—that retention of bronchial secretions occurs, and that, owing to gravitation, these secretions reach the minute bronchi. Pneumonia is similarly produced when we cut the vagus nerves, the paralyzed structures permitting irritants to be carried to the lung by inspiration. *Inhalation pneumonia* may follow operations upon the nose, mouth, larynx (tracheotomy particularly), and is often secondary to carcinoma of the larynx and esophagus. It is also the pneumonia of new-born children.

(5) It must not be forgotten that quite commonly broncho-pneumonia is caused by the *tubercle bacillus* (*vide* Pulmonary Tuberculosis).

Bacteriology.—Weichselbaum has shown the presence of streptococci with the greatest frequency in the usual, secondary form. The pneumococcus is often found, and in a goodly number of cases the staphylococcus aureus (Neumann), while in influenza the specific organism may itself cause broncho-pneumonia (Pfeiffer). Numerous other

organisms have been found (typhoid bacillus, bacillus coli communis). Mixed infection with the Diplococcus pneumoniæ is almost the rule when lobular pneumonia is secondary to the acute infections.

Symptoms.—Two clinical forms may be distinguished:

(*a*) **Primary broncho-pneumonia**, which occurs in 30 to 35 per cent. of all cases, is met with generally in children, and presents, in great part, the symptoms of an acute bronchitis of severe grade (*cough*, *dyspnea*, *pain*, *fever*). When occurring in weakly subjects the onset may be gradual. The cough is attended with *expectoration* (glairy and tenacious), that may be blood-tinged, in the form of droplets or points. The fever is moderate, the temperature ranging from 101° to 104° F. (38.3°–40° C.), and is of irregular type; in severe cases, however, continued high temperature may occur. Physical examination gives the same result as in the secondary form. The *duration* is from two to four weeks, the fever terminating by *lysis*. West holds that primary broncho-pneumonia in children is of pneumococcic origin.

(*b*) **Secondary broncho-pneumonia** is the variety usually met with. The symptoms are frequently veiled by those of the primary affection, and, indeed, a moderate grade of lobular pneumonia is frequently unsuspected during life when arising in the course of other grave diseases.

It is usually preceded by bronchitis affecting the larger bronchi, and in this common event the first symptom that directs attention to the disease is the *sudden increase* in the frequency of the *respirations*, which rise as high as 60 or even 80 per minute. An initial chill is rare. *Fever* develops suddenly, or, if previously present, increases rapidly. An early symptom is the *cough*, which is usually hard, harassing, frequently painful, and accompanied by *expectoration*. The *pulse* is frequent, and in the later stages may be quite rapid, feeble, and irregular. The type of the fever is similar to that of the primary form.

Blood.—There is usually marked leukocytosis of the polynuclear type. Absence of leukocytic increase is of serious meaning, implying lack of resistance. On the other hand, a high leukocyte count does not necessarily indicate a favorable prognosis, but a good reaction.

Physical Signs.—At the beginning of the attack the only sign is the presence of subcrepitant and sibilant râles, pointing to general bronchitis. Shortly larger or smaller areas of consolidation become manifest. At first rapid breathing, and soon cyanosis, affecting first the lips and conjunctivæ, may be observed; later, the face becomes dusky and the finger-tips blue. *Palpation* shows defective expansion and increased tactile fremitus over the consolidated areas. The *percussion-note* is dull or, less frequently, hyperresonant if the area be small. *Auscultation* reveals numerous fine, subcrepitant râles, corresponding to the consolidated portions. The respiratory murmur may be bronchial, though more often broncho-vesicular. The signs are usually noted in both lungs. Confluence of the numerous, small, consolidated areas may result in large fields of dulness, and true bronchial breathing. In cases of extensive consolidation, there may be inspiratory retraction of the lower sternum and lower ribs, indicative of deficient lung expansion (Butler).

Duration.—(1) In children this varies in different cases. Rarely do fatal instances last more than two or three weeks, while they may be as brief as two or three days. On the other hand cases in which recovery

ensues frequently last from six to eight weeks, though rarely from one to three weeks only. Two special forms demand brief description:

(*a*) The *cerebral*, in which restlessness, convulsions, and delirium become so marked as to overshadow entirely the pulmonary symptoms. Not infrequently the onset is characterized by convulsions, high fever, prostration, and alternating stupor and delirium. After the lapse of from two to five days, pulmonary symptoms appear, while the cerebral decline.

(*b*) Other cases may manifest a *subacute onset*, in which there is anorexia and occasional vomiting, with the nervous symptoms before noted.

(2) The *protracted forms* are those in which (*a*) the symptoms of

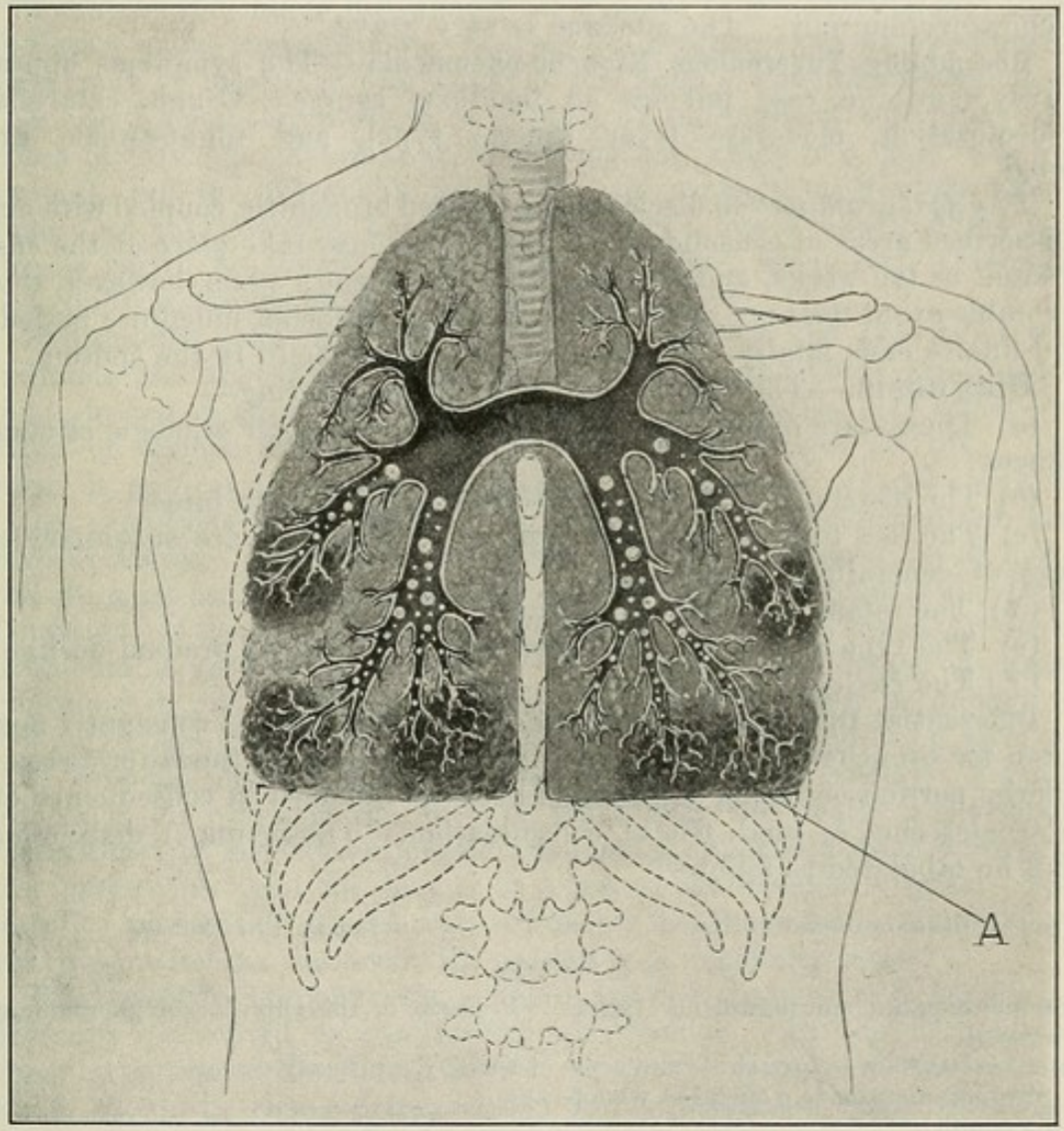


FIG. 46.—Illustrating broncho-pneumonia. The dark spots represent the consolidated areas; the white dots indicate râles: *A*, coalescence of two areas of consolidation.

acute broncho-pneumonia give place to those of a similar though chronic state. The general disturbances may not be marked in some instances, but usually there are cough, loss of appetite, or inability to gain in flesh and strength, and the signs of consolidation persist. (*b*) Those presenting fever of an irregular type, together with decided prostration, in addition to the symptoms of the preceding variety. In many cases belonging to this form the lesions are tuberculous.

In adolescence the cerebral symptoms are not as well marked as in children. Two anomalous varieties are met with in practice:

General Broncho-pneumonia.—The attack develops suddenly and is severe. There are chills, high fever, marked prostration, headache, chest and loin pains, a rapid pulse (soon becoming feeble), rapid and labored respirations, cyanosis, restlessness, delirium, and cough that is at first dry, and followed by muco-purulent, blood-tinged sputum.

The physical signs are defective expansion and an increased tactile fremitus. The percussion-note may be either normal, tympanitic, or dull; the auscultatory signs are large moist, subcrepitant, crepitant, sibilant, and sonorous râles over both lungs, and a harsh or broncho-vesicular respiratory murmur. The affection is very grave.

Resembling Tuberculous Broncho-pneumonia.—The symptoms appear slowly, and the case pursues an insidious course. Cough, catarrhal expectoration, moderate fever (hectic type), and night-sweats are noted.

Physical examination discloses generalized bronchitis, coupled with circumscribed areas of consolidation. Resolution may take place at the end of eight or ten weeks, and complete recovery ensue; when, however, this favorable event does not occur, the case drags on for an indefinite period, and finally ends fatally. There are no tubercle bacilli in the sputum.

Diagnosis.—This can be arrived at by considering—

(*a*) The nature of the antecedent affections and their etiologic circumstances;

(*b*) The distribution of the consolidated areas in both lungs;

(*c*) The fact that the physical signs of consolidation are subsidiary to those of generalized bronchitis;

(*d*) The intense dyspnea and cyanosis;

(*e*) The type of the fever, irregular as a rule, and its gradual decline;

(*f*) The frequent long duration.

Differential Diagnosis.—Doubtless, *lobar pneumonia* is constantly mistaken for broncho-pneumonia, and particularly when, in the latter disease, a large portion of one or both lungs becomes inflamed in consequence of the coalescence of small foci of consolidation. The points of distinction may be tabulated as follows:

Broncho-pneumonia.	Lobar Pneumonia.
Etiology.	
Presence of pathogenic organisms[1] (streptococci).	Presence of the Diplococcus pneumoniæ.
Usually secondary to bronchitis and acute infectious diseases (*e. g.* measles, whooping-cough).	Usually a primary disease.
Clinical History.	
Onset gradual, without rigor.	Onset abrupt, with rigor; previous health generally good.
Fever is, in proportion to the extent of inflammation, of irregular type, and declines by lysis after a variable duration.	Fever is high, of continued type, and falls between the fifth and ninth days by crisis.

[1] The diagnostic value of the discovery of streptococci is not pronounced. Numerous other organisms have been found in broncho-pneumonia, and a similar organism (*Streptococcus pneumoniæ*, Weichselbaum) in cases of croupous pneumonia.

Sputum glairy, tenacious, and in adults may be blood-tinged.	Sputum characteristic (rusty, or prune-juice).
Dyspnea and evidence of carbon-dioxid poisoning prominent.	Dyspnea and cyanosis relatively less marked; anxious countenance.
Herpes labialis absent.	Herpes labialis commonly present.
Physical signs of generalized bronchitis always marked, and usually preponderating over those of consolidation.	Signs of bronchitis generally absent, those of lobar consolidation always preponderating.
Consolidation commonly bilateral.	Commonly unilateral.
Duration indefinite, often extending over many weeks.	Duration definite as a rule, convalescence following crisis.
Consolidated areas liable to become the seat of tuberculous infection.	Far less likely to become the seat of tuberculous infection.

It may be difficult to distinguish *tuberculous broncho-pneumonia* from the disease under consideration. Indeed, a non-tuberculous broncho-pneumonia may be located at the apex of the lung. The differentiation is to be based upon the presence or absence of the signs of softening, and upon a microscopic examination of the sputum (which in a child may be vomited), and the tuberculin test. The softening in tuberculous pneumonia does not, however, begin very promptly; but if elastic fibers and tubercle bacilli be found, the diagnosis is at once set at rest. Hemoptysis confirms the diagnosis of the tuberculous form.

Prognosis.—In broncho-pneumonia the severity and gravity of the symptoms and the extent of the involvement of lung-tissue are proportionate to one another; hence it follows that the disease may either be devoid of serious import or fraught with great danger to life. Its course is subject to decided fluctuations, the periods of exacerbation in the symptoms often marking the time of the development of the gravest features. Apart from the extent of the lung-tissue involved, however, we must consider especially the condition of the patient at the time of invasion. If the constitution have been previously undermined, broncho-pneumonia is very apt to be fatal. The disease is less dangerous when it develops in the course of, or follows, measles than when secondary to whooping-cough, influenza, or diphtheria. Wiry, thin children seem to stand broncho-pneumonia better than fat, flabby ones (Osler). During childhood, the younger the subject, the higher the death rate (Hare). *Deglutition* and *inspiration* lobular pneumonia, especially when occurring after operations upon the larynx or trachea, are frequently fatal. The mortality rate in this disease varies from 25 to 50 per cent. In primary broncho-pneumonia, however, the mortality is decidedly lower.

Treatment.—Prophylaxis.—There are few diseases that can be so effectually prevented as can broncho-pneumonia. In the first place, proper attention to the mouth as well as to the position of the patient (which should be changed frequently) during attacks of acute infectious diseases will prevent its development in a great proportion of this large class of cases. Adequate protection against exposure to cold during convalescence from measles, whooping-cough, etc. is also a potent factor in preventing the disease, as is the timely handling of catarrhal affections of the nose, pharynx, larynx, and larger bronchi.

Treatment of the Attack.—Certain *sanitary arrangements* are of the utmost practical importance. The sick-room should be well ventilated and its atmosphere kept at a uniform temperature—68° to 70° F. (20°–21.1° C.). The air of the room should also be well laden with moisture, which may be generated from a croup-kettle or other suitable vessel.

Local Measures.—In young children the chest should be enveloped in a jacket-poultice of linseed meal, which should be covered with a layer of oiled silk or waxed paper to prevent its growing cool. This should be renewed at intervals of about six hours. After the more active symptoms have subsided the linseed jacket-poultice may be replaced by one of absorbent cotton, which should also be covered with oiled silk or wax paper. In older subjects the application of iced poultices to the chest exercises a most favorable influence, not only upon the local inflammation, but also upon the fever and the nervous symptoms.

General Measures.—High fever calls for tub-baths, the temperature of the water at first being set at 95° F. (35° C.), and then gradually cooled to 75° or 80° F. (26.6° C.). The gradually cooled bath or the cold pack may be used two or three times daily. The effects are to reduce temperature, to promote refreshing sleep, and to improve the character of the respiration. This mode of treatment is especially effective in cases that begin abruptly. In such the tincture of aconite or veratrum viride may be employed temporarily. In cases presenting moderate pyrexia cold spongings, combined with the use of the ice-bag to the head, may suffice. The following fever-mixture may be employed, though it is not to be regarded as a substitute for hydrotherapy, but is merely supplemental to the latter:

℞. Potassii citrat., ʒijss (10.0);
Spts. ammon. aromat., fʒij (8.0);
Spts. æther. nitrosi, fʒss (16.0);
Liq. ammon. acetat., fʒiij (96.0);
Glycerini, q. s. ad fʒiv (128.0).—M.
Sig. ʒj (4.0) every two hours for a child of five years.

In *children* a mild mercurial purge at the outset is advantageous, and subsequently by the use of salines or glycerin suppositories a daily evacuation of the bowels is to be secured.

The Diet.—The bodily strength is to be maintained by careful, methodical feeding, milk, eggs, albumin, and broths being the best forms of food. The milk should be predigested if there be marked pyrexia, and egg-white may be given in cold water or as egg-lemonade. The cough is often wellnigh constant and very distressing. Frequently the use of remedies that promote secretion, combined with a small dose of opium, will, under these circumstances, afford relief. A useful formula is the following:

℞. Vini antimonii, ʒj (4.0);
Spts. æth. nit., ʒijss (10.0);
Tr. opii camph., ʒijss (10.0);
Liq. ammon. acetat., q. s. ad ʒij (64.0).—M.
Sig. ʒj (4.0) every two hours, diluted, for a child of from three to five years.

Dover's powder is also of value in relieving the cough. When the expulsion of the sputum is attended with great difficulty the preparations of ammonium often meet the indications. Of these the muriate is the most effective, but, unfortunately, this is often objected to, and we must

then rely upon the carbonate or the aromatic spirits. The bronchi may contain an abundance of secretion that cannot be expelled, despite the use of the above measures. Under these circumstances an emetic may be given, composed of the wine of ipecac (ʒj—4.0), combined with alum (gr. xx to xxx—1.296–1.944), and administered to a child every ten or fifteen minutes until emesis occurs. Oxygen by inhalation is to be used early and persistently to overcome the cyanosis.

Cardiac stimulants (alcohol, strychnin, Hoffman's anodyne) are required if the pulse fails, with increasing cyanosis. The preparations of ammonium owe much of their reputation in this disease to their stimulating properties. These agents when boldly used may suffice to re-establish the cardio-pulmonary circulation. Sudden heart exhaustion may occur, associated with mucous râles in the larger bronchi and rapidly developing cyanosis; atropin (dose, gr. $\frac{1}{500}$ to $\frac{1}{200}$—0.0001–0.0012 gm. every second or third hour) tends to arrest this mucous secretion. Alternating douching with hot and cold water and electricity should be given a trial. Injections of salt solution increase arterial tension and act as a "whip" to all emunctories, aiding in the elimination of toxins. They should be used in serious cases. In streptococcic broncho-pneumonia antistreptococcus serum may be tried.

PULMONARY ATELECTASIS.

(*Collapse of the Lungs; Compression of the Lungs.*)

Definition.—Atelectasis of the lungs is a condition occasioned by the removal of the air from the air-cells—a state directly the opposite of emphysema. The air disappears largely in consequence of the process of absorption.

Pathology.—The affected lung-spots sink in water, being non-crepitant. They present through the pleura a bluish-red tint, and on cross-section a brownish-red color. The surface of the affected areas is smooth and depressed. The bronchi supplying the collapsed parts may be occluded by inflammatory products, but, as shown by Legendre and Bailly, the air-cells involved may be inflated by means of a blowpipe.

Apart from more or less capillary distention, there are no *histologic* changes in the atelectatic areas, though they are of firm consistence (splenization, carnification). There can be no longer any doubt as to the entire propriety of the pathologic distinction between lobular pneumonia and atelectasis.

Etiology.—The condition occurs most frequently in the new-born, and is then due to defective respiration. Thus in children dying soon after birth the lower lobes may be found to be atelectatic. When acquired, however, there are three modes of production: (1) The first step consists in a more or less complete plugging of the smaller bronchi with muco-pus and other products of bronchial inflammation. If complete, air can no longer enter on inspiration, and as the contained air gradually becomes absorbed atelectasis is the natural result. This condition is very commonly associated with broncho-pneumonia, especially in children. New growths may occlude the smaller bronchi and produce a

similar result. (2) A frequent mode of origin is through compression of the lungs, resulting from positive intrathoracic pressure, after the normal contractility of the lung has been overcome. Instances of this may be produced by pleural effusion, hydrothorax, pneumothorax, pericardial effusion, great cardiac hypertrophy, a solid tumor, or an aneurysm of the arch. Not infrequently abdominal tumors, excessive meteorism, and ascites make sufficient upward pressure against the diaphragm to cause compression of the lower lobes of the lungs. (3) Conditions that weaken and obstruct the inspiration may produce this disease, such as certain brain-affections, paralysis of the pneumogastric, and paralysis of the chest-walls. Thoracic deformities may produce pulmonary atelectasis, and in extreme grades of kyphoscoliosis the lung occupying the side corresponding to the convexity of the spinal column is small. Whilst the lung-expansion and the growth of the organ are greatly interfered with, true atelectasis rarely occurs from this cause, particularly if the condition arises in youth, owing to the natural retractility of the lung. Among conditions arising from deformities of the chest is the so-called aplasia of the lungs.

Symptoms.—Atelectasis is a secondary condition, and its symptoms are very generally veiled by those of the primary disease. It arises frequently in the course of broncho-pneumonia, but passes unnoticed unless it becomes very extensive. *Respiration* is carried on by the upper and anterior portions of the lungs, is increased in frequency, and is laborious. The *pulse* is small, rapid, and feeble; the *skin-surface*, especially that of the extremities, is cool.

The form presenting the most typical symptoms is that occurring in the new-born. It is evidenced by *shallow, rapid breathing, lividity, cold extremities*, a *faint whining cry, drowsiness*, and sometimes by evidences of *motor irritation*, such as muscular twitching and convulsions. Congenital anomalies of the circulatory organs are associated.

Physical Signs.—When it involves a goodly portion of the lower lobes posteriorly, as frequently happens, there is marked retraction during inspiration over the lower portion of the thorax, due partly to external atmospheric pressure, and partly to the contractile efforts of the diaphragm. Dulness on percussion is only revealed when the atelectasis is extensive, and the tactile fremitus, though very various, is generally decreased or even absent. Localized compensatory emphysema may present semitympanitic resonance over small areas of collapse.

Auscultation shows a greatly diminished or absent vesicular murmur, and, if the area of collapse be large, bronchial breathing. Among associated sounds is the subcrepitant râle, due to broncho-pneumonia, and, indeed, capillary bronchitis and atelectasis are often combined, there being, moreover, no reliable signs that will separate them clinically.

The *aplasia* of the lung that is produced by spinal curvature (*kyphoscoliosis*) richly deserves brief separate description, owing to its clinical importance. In many instances the chest is more or less twisted on its own axis, shortened in the vertical diameter, and thoroughly fixed. Under these circumstances lung-expansion is impossible, and hence respiration is purely diaphragmatic. In many other patients life may be prolonged for an indefinite period, nothing more being observed than

slightly labored breathing. Such persons, however, upon great physical exertion suffer from urgent dyspnea, and the development of an ordinary bronchitis may lead to similar results, and even to speedy death.

The **physical signs** are those of localized emphysema, combined with those of more or less compression of the lungs. There is an extension of the cardiac dulness to the right, and other evidence of right ventricular enlargement, to which may succeed dilatation with its usual clinical events. Death is not rarely due to this failure of compensation.

Autopsies have shown the lungs to be small and more or less compressed, some portions being almost airless. Areas of emphysema are often associated. The right ventricle may be hypertrophied merely, or dilatation may also have taken place. Congenital atelectasis, by keeping up high pulmonary pressure, may lead to persistence of the ductus Botalli and of the foramen ovale.

Diagnosis.—Atelectasis may be distinguished from *lobar pneumonia* by the absence of an initial rigor, fever, crepitant râles, and the pain of the latter disease, and by the characteristic inspiratory retraction of the lower portions of the chest and the smaller areas of dulness.

Pleuritic effusion gives a flat percussion-note, the upper level of which varies with a change in the position of the patient—a sign that is wanting in atelectasis.

Prognosis.—When the condition is limited to small areas it is rarely serious, but equally seldom does extensive atelectasis lead to recovery. The outlook depends to some extent upon the nature of the associated affections ; thus, when secondary to whooping-cough and widespread broncho-pneumonia, it is very fatal. Other diseases that may complicate and increase the gravity of the atelectasis are pleurisy and pulmonary tuberculosis. On the other hand, compensating emphysema often coexists, and is to be regarded as salutary in its effects. When due to *compression* by pyo-pneumothorax, tumors, and the like, the prognosis is especially gloomy.

Treatment.—The treatment corresponds with that of the primary disease. *Capillary bronchitis*, which is so apt to be followed by collapse of the lobules, must receive active treatment, and prophylactic measures are of the utmost practical importance. The patient should be instructed to practise full inspiration at regular intervals ; he should not be allowed to lie continuously in the dorsal decubitus, but should change his position frequently. Another useful preventive measure is the use of cold shower-baths (*i. e.*, a stream of cold water poured over the region of the neck), and this can sometimes be depended upon as a curative agency when the condition already exists. Tonics and the judicious use of stimulants, together with a nourishing diet, are invariably required. I have also seen good results follow the inhalation of compressed air and of oxygen.

In *kyphoscoliosis* tepid baths are indicated. The heart-condition demands careful attention, and cardiac stimulants are to be resorted to at the first loss of compensation or when compensation fails to become established.

EMPHYSEMA.

Definition.—In general this term implies the presence of air in the interstitial alveolar tissue. As applied to the lungs, however, two forms are recognized: (1) Interlobular; and (2) Vesicular, an abnormal dilatation of the alveoli.

INTERLOBULAR EMPHYSEMA.

This is produced by the rupture of the air-cells, the air contained in the lung escaping into the interlobular connective tissue. Among its causes are—(*a*) Injuries of the lung (usually by a fractured rib) and penetrating wounds of the chest; (*b*) Violent paroxysms of coughing, as in whooping-cough; and rarely defecation, parturition, and hysterical convulsions. When arising in this way its favorite situation is the anterior margin of the upper lobe.

Pathology.—In the interlobular septa immediately beneath the pleura air-bubbles are sometimes seen to be arranged in well-defined rows. The pulmonary pleura may become detached, and the air-tumors may then become as large as an English walnut or even of greater size. Unlike the condition in vesicular emphysema, these sacs are freely movable, and the air may find its way from the root of the lung into the mediastinal connective tissue, and thence into the subcutaneous tissue of the neck and the wall of the thorax. Rarely these air-sacs perforate the pleura, setting up pneumothorax, with or without pleuritis.

Interlobular emphysema is sometimes associated with advanced vesicular emphysema.

VESICULAR EMPHYSEMA.

(*Alveolar Ectasis.*)

Definition.—Dilatation or enlargement of the alveoli and infundibular passages.

Varieties.—The cases are classified into—(1) Compensating, (2) Hypertrophic, and (3) Atrophic forms.

COMPENSATING EMPHYSEMA.

This variety is limited to certain parts of the lung, and arises in consequence of pathologic changes in other parts of the same organ that prevent full expansion of the lung on inspiration. Hence a vicarious increase in the volume of the air-cells is observed in circumscribed morbid processes such as occur in pulmonary tuberculosis, lobular pneumonia, cirrhosis, and pleurisy with adhesions (particularly when the latter is situated at the inferior border of the lung). An entire lung, unaffected by the primary disease, may be the seat of compensating emphysema when the causal disease invades the whole or a greater portion of the other lung, as in cirrhosis, extensive pleurisy with effusion, lobar pneumonia, and pyo-pneumothorax. When, however, the latter conditions are confined to a portion of one lung, the remainder of the same organ becomes distended also. The term *acute emphysema* is applicable to many of the cases.

As a rule, this pulmonary change is physiologic and beneficial: only rarely secondary atrophy of the walls of the air-cells develops.

Symptoms are not presented by the lungs in consequence of the changes met with in compensating emphysema. The condition is sometimes recognizable by means of the usual physical signs, but even these are not always to be relied upon. Fortunately, its existence may be safely inferred when there is conclusive evidence of the presence of the local causative diseases (broncho-pneumonia, pulmonary tuberculosis, pleurisy, lobar pneumonia).

HYPERTROPHIC EMPHYSEMA.

Nature of Emphysema.—The symptoms are dependent upon a loss of elasticity in the lungs, and, the latter condition being the result of overstretching, the contractile energy of the lungs is in great part destroyed; hence they become permanently enlarged. We may in some cases account for the loss of elasticity in the lungs by the operation of causes that produce an abnormal degree of stretching, either temporarily or constantly; but under these circumstances emphysema would be developed despite the pre-existence of normal contractility of the lung. In true emphysema, however, which develops at a comparatively early period in life, we may safely assume that the retractile energy is defective (probably a congenital condition), and hence in such cases the action of the usual causal factors will speedily engender over-distention, or emphysema may develop even in the absence of causative influences. In these instances there is probably a quantitative as well as a qualitative defect in the elastic-tissue element of the lungs.

Pathology.—The thorax is enlarged (barrel-shaped), and upon removing the sternum the lungs are found completely to fill the mediastinum, and do not retract as in health. They present a pale, anemic appearance, although pigmented patches and streaks may be noted. To the touch they appear soft and feathery, though dry. They readily pit on pressure (a leading characteristic).

Immediately beneath the pleura enlarged air-cells can be distinguished macroscopically, and air-sacs as large as a walnut may project above the lung surface. Occasionally they may be pedunculated. At the anterior borders a series of air-blebs, resembling a frog's lung, may be observed. Here, and near the root of the lung, distention is usually more marked, owing to the direction taken by the distending force. The pleura is pale, and in patches the pigment may be absent (*Virchow's albinism*).

Upon microscopic examination it is observed that the dilatation starts in the infundibular and alveolar passages. The septa are partially obliterated, the alveolar walls thinned and, lastly, perforated, while in consequence of these changes the air-cells communicate with one another, forming larger or smaller air-sacs. The process is an atrophic one, the smaller elastic fibers disappearing, while the larger become less prominent and often ruptured. After the latter changes have begun the capillaries likewise disappear, and the epithelium of the air-cells undergoes fatty degeneration, though in the larger bullæ a pavement-layer is retained. The smooth muscular element may also occasionally be found hypertrophied

(Rindfleisch). The clinical phenomena probably arise from the loss of the capillary blood-vessel system and collateral hyperemia of the larger bronchial vessels.

The bronchial mucous membrane is usually the seat of chronic inflammation. It may be roughened and thickened, or the submucous elastic tissue may present prominent longitudinal lines, while the bronchial mucosa is covered with muco-pus. The smaller tubes may be dilated (*bronchiectasis*), and hyperplasia of the peribronchial connective tissue may be associated. The diaphragm is lowered and the subjacent viscera correspondingly depressed.

Physiologic Pathology.—The heart is pushed downward and somewhat backward. The right side shows well-marked changes; the cavities are dilated and hypertrophied, due to obstruction in the pulmonary circulation; and in long-standing cases hypertrophy of the left chambers may also develop. The pulmonary artery and its branches are enlarged and the seat of atheromatous degeneration. The liver, kidneys, and other viscera present the changes that belong to chronic venous engorgement.

Etiology.—The affection is often secondary to, and develops in consequence of, other affections of the lung—notably *whooping-cough* and *chronic bronchitis*, particularly the dry form. The disease is attributable to the mechanical influences to which the alveolar walls are subjected during respiration. This abnormal strain attends inspiration to some extent, but mainly expiration, owing to the obstruction to the egress of the air in the smaller bronchi, with increased *intra-alveolar air-pressure*. The increased tension in the air-cells may be accounted for partly by the severe and persistent cough, the air being thus driven into the apices of the lungs, forcibly expanding them and causing emphysema. Syphilis and alcoholism are among the recognized causes.

Bronchial asthma, on account of the obstruction of the exit of the air from the lungs, produces during the attacks an acute emphysema that may result finally in a condition of permanent overdistention. *Certain occupations*, such as blowing wind-instruments, or those that entail severe muscular strain (*e. g.* blacksmithing), act as predisposing causes, and hence, emphysema is of common occurrence among the working classes, and is more common in males than females. Edsall's studies, however, show that glass-blowers and players on wind instruments are not especially liable. The constant straining in certain pelvic disorders may induce emphysema. The disease is often *hereditary*. During *advanced years* the lung-elasticity often diminishes, and as a consequence a disposition to emphysema is engendered. On the other hand, emphysema is not infrequently met with in children, and in such there may be a temporary respite, with a recurrence at a later period. An emphysematous tendency also results from congestion of the lungs associated with mitral valvular disease.

Clinical History.—In nearly all cases the disease develops insidiously, the symptoms being gradually added to those of the primary affections (chronic bronchitis, asthma, etc.). When due to occupation its development is also slow, and not infrequently its origin dates back to childhood or beyond the recollection of the patient. Rarely it may exhibit a more acute development, *e. g.*, after whooping-cough.

The first symptom is a variable degree of *dyspnea*, and to this may

be added temporary *cyanosis* and *cough*. The severity of the dyspnea varies with the degree of distention of the air-cells, even though additionally aggravated by the coexistence of the primary disease. In moderate emphysema the dyspnea is only apparent on going up stairs, running, walking rapidly, or after a hearty meal; on the other hand, in advanced grades of the affection it is constant, and is intensified by the slightest exertion, even to orthopnea. Speech is interfered with, the patient's utterances taking the form of fragmentary sentences or syllables. The labored breathing is shown particularly in expiration, and, as in asthma, in which the alveolar spaces are acutely distended, so in emphysema the rhythm of the respiration is changed. The inspiration is shortened, and the expiration is greatly prolonged and accompanied by wheezing when chronic bronchitis coexists.

In the later stages cyanosis becomes more marked, and is noticeable in proportion to the loss of compensation and interference with the cardio-pulmonary circulation. It often attains to an extreme degree, and the patient's alarming appearance may be in striking contrast with his apparent degree of comfort. In mild forms the cyanotic tint is confined to the lips, lobes of the ears, and the extremities. Any increase in the degree of dyspnea after exertion results in an increased blueness of the surface.

The *cough* is dependent upon the presence of chronic bronchitis, which frequently co-exists, particularly during the winter. The expectoration is identical with that of chronic bronchitis, and when this disease reaches an advanced stage the cough persists throughout the year (*vide* Chronic Bronchitis). Intercurrent acute attacks of bronchitis are often followed by temporary attacks of asthma; and since chronic bronchitis in its highest grades is met with at an advanced period of life, so, as would be expected, the cases of advanced emphysema are also met with at the same period. Osler has described a group of cases occurring in patients "from twenty-five to forty years of age who, winter after winter, have had attacks of intense cyanosis in consequence of an aggravated bronchial catarrh." These patients are short-breathed from infancy, and their condition is attributed to a primary defect of structure in the lung-tissue.

General Symptoms.—There is no fever, the temperature being generally subnormal, and the pulse, though sometimes feeble, is not increased in frequency. There is a very gradual loss of flesh and strength, and the patient is stoop-shouldered, presenting a peculiar cachectic appearance —in strong contrast with the dusky appearance of the face, the swollen neck, and the enlarged chest.

Finally, other symptoms may be mentioned that are for the most part secondary to hypertrophy, followed by dilatation, of the right ventricle. This hypertrophy is the result of pulmonary congestion and obliteration of the pulmonary capillaries induced by the emphysema. Under these circumstances severe attacks of cough occur, attended with extreme dyspnea and lividity, and later the conditions that usually succeed a moderate grade of tricuspid insufficiency supervene, such as congestion of various viscera and edema of the feet. Anasarca is rare.

Physical Signs.—The shape of the chest is characteristic: owing

to the increased antero-posterior diameter, it becomes barrel-shaped (Fig. 47), and the sternum bulges, as do also the costal cartilages. The infraclavicular and mammary regions are also prominent, and give the thorax an abnormally rounded appearance. The episternal notch is deeper than the normal, the clavicles and muscles of the neck are unduly prominent, and the neck itself appears to be shortened, owing to the elevated position of the clavicles and the sternum. There is an antero-posterior curvature of the spine and a winged condition of the scapulæ. Below, the thorax appears contracted. The intercostal spaces are widened and depressed, and a network of dilated venules frequently extends laterally above the inferior costal border.

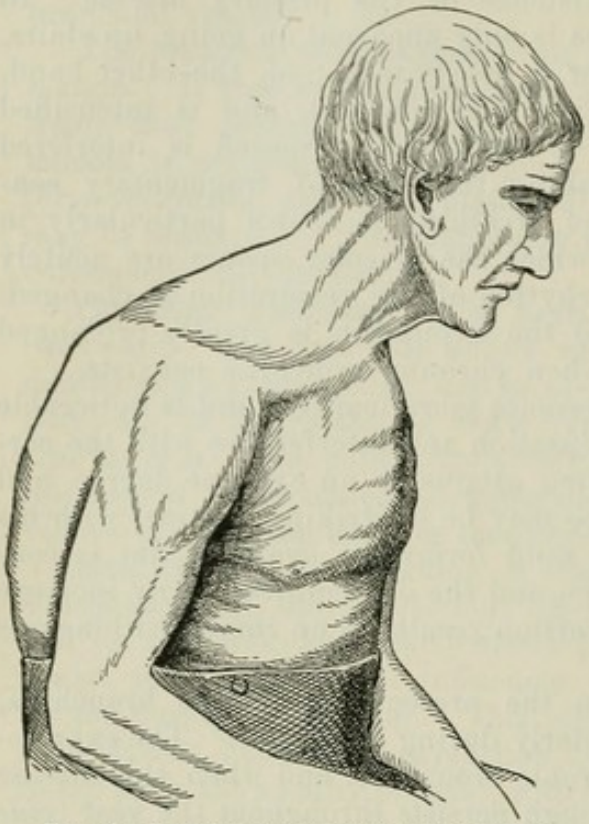


FIG. 47.—Barrel-shaped chest in emphysema.

The movements of the chest are vertical rather than expansile, and the lungs are constantly in a state of extreme expansion; in the lower thoracic and upper abdominal regions there may be observed retraction rather than expansion during the act of inspiration. The respiratory acts, as a whole, are labored, and the diaphragm and abdominal muscles are seen working with considerable violence. The heart's apex-beat is invisible, but marked epigastric pulsation is frequently noticeable. Venous pulsation may be seen in the neck after failure of the right ventricle has occurred.

On *palpation* the character and direction of the chest-movements may be accurately appreciated. The tactile fremitus is decreased, but not absent. In the early stages the apex-beat is feeble, while in advanced cases it cannot be felt. Owing to displacement of the heart and engorgement of the right ventricle there is a distinct systolic shock over the ensiform cartilage, and also a pulsation in the epigastrium.

Percussion yields a characteristic hyper-resonance. This may be distinctly "Skodaic" or semi-tympanitic, and in extreme instances the tone may be woodeny. The area of percussion-hyper-resonance extends higher above the clavicles than naturally. The area of cardiac dulness is lessened and finally obliterated by the distended lungs; while the upper limit of liver-dulness, both anteriorly and posteriorly, is found to be one or two interspaces lower than normal, owing to the fact that the diaphragm is depressed. The upper level of splenic dulness is also lowered.

On *auscultation* the inspiration is short and feeble, while the expiration is greatly lengthened, the normal ratio of these sounds being reversed. Their pitch is somewhat lowered, particularly that of expiration; and

when râles are present the respiratory murmur (particularly the inspiratory) may be scarcely audible. In well-marked instances of emphysema inspiration and expiration may rarely be of equal length. It is a fact worthy of emphasis that the parts of the lungs less markedly emphysematous than others give a harsh, exaggerated vesicular murmur, owing to the great efforts of breathing. Râles of various sorts are frequently audible, due to the accompanying bronchitis usually present; less frequently the auscultatory signs of asthma, pleuritis, and phthisis are encountered. Rarely, rubbing sounds, attributed to the friction of enlarged air-cells against the pleura, are audible, and when the interlobular variety supervenes upon vesicular emphysema a *crumpling* sound is heard. The so-called "*Laennec's râle*," which resembles somewhat the subcrepitant râle, is not infrequently present. The vocal resonance varies from an almost total absence to a greatly increased intensity. The tricuspid insufficiency that develops late in this affection is betrayed by its characteristic murmur.

Diagnosis.—A positive diagnosis may be arrived at from a consideration of the history, including such points as heredity, occupation, the long duration of the condition, coupled with the most characteristic symptoms (dyspnea, cyanosis, signs of chronic bronchitis), and from the physical signs. In a case of beginning emphysema, particularly among children, a certain diagnosis is not to be attempted.

Differential Diagnosis.—*Pneumothorax* is the disease most apt to be confounded with emphysema. It develops suddenly, however, while emphysema is of slow development, and the rational symptoms of pneumothorax are more constant and urgently distressing than those of emphysema. Pneumo-thorax is unilateral, and gives a purely tympanitic percussion-note, while hypertrophic emphysema is bilateral and its percussion-note is hyper-resonant. Amphoric breathing, metallic tinkling, the characteristic succussion splash, and an absence of the vesicular murmur, usually present in pneumo-thorax, are absent in emphysema.

Another affection giving rise to dyspnea, cough, and cyanosis is *pleurisy with effusion*, but the slow course, the absence of fever, and the universal hyper-resonance that characterize emphysema do not belong to pleurisy. The latter affection yields a flat percussion-note.

Prognosis.—Hypertrophic emphysema of acute form (*e. g.*, resulting from whooping-cough) is often curable; but the usual slowly generated variety gives an unfavorable prognosis as to recovery. In many cases, however, life is not materially shortened. Temporary improvement is possible when the lesion consists merely of a distention of the air-cells, and is shown by a corresponding improvement in the physical signs. If the vital capacity, as shown by the spirometer, is reduced one-half or more, the prospect is unfavorable. Recurring attacks of bronchitis intensify the symptoms of a disease that is innately progressive. Intercurrent affections, such as pneumonia (lobar and lobular) and pulmonary tuberculosis, may prove fatal. Dropsy, following broken compensation, is a dangerous complication; other late accidents are hemoptysis and sudden dilatation of the right heart.

Individual circumstances, such as the patient's social condition, the stage of the affection in which he comes under proper treatment, and the degree of care he is willing to exercise, greatly influence the outcome of the case.

Treatment.—The treatment is to be directed toward the removal of the causes of emphysema, and chiefly of the chronic bronchitis. From personal observation I am firmly convinced that the progress of the disease can be arrested, and that the condition is sometimes improved, by relieving the chronic bronchitis. The iodids (potassium, sodium, and ammonium) at times produce effects that are truly remarkable. If not well borne by the stomach, the syrup of hydriodic acid may be employed. If the occupation of the patient tends to aggravate the disease, it must be forsaken for a less harmful one. Violent paroxysms of cough and intercurrent attacks of asthma contribute to the production of alveolar distention, and hence must be alleviated promptly by appropriate therapeutic measures. Attacks of acute bronchitis are to be prevented by suitable clothing, by avoidance of exposure to inclement weather, dust, and the vitiated atmosphere of overcrowded halls, churches, and the like; whenever practicable the result can be most successfully obtained by a residence in an equable climate. Since a severe bronchitis is apt to increase the severity of the emphysematous symptoms, it must be relieved as speedily as possible. Passive congestion, flatulence, and constipation, with other gastro-intestinal symptoms, demand careful regulation of the diet and especially a restriction in the use of carbohydrates. The bowels must also be moved regularly with the same end in view.

The heart needs to be carefully watched, and as soon as signs of broken compensation appear digitalis and strychnin will be found highly useful. Diuretics and cathartics may also become necessary. The sudden development of urgent dyspnea and extreme lividity, especially if associated with weak cardiac action and a rapid, feeble, irregular pulse, calls for free bleedings. In my hospital practice I have seen the lives of patients suffering from emphysema saved by timely venesection.

To assist the patient in expiration Gerhardt has suggested systematic *mechanical compression* of the thorax during expiration. Pressure is made by an attendant, who places his hands flat on the lower lateral portions of the thorax, and the manipulation is to be continued for from ten to fifteen minutes daily. The results obtained by certain German authors have been encouraging, but in my own hands the method has failed, except in two instances occurring in young adults with yielding chest-walls, in whom it was of the greatest service. The aim should be to strengthen the muscles of the diaphragm by prolonged expiration, supplemented by drawing in the abdominal walls as the act of expiration draws to a close —not before. The *pneumatic treatment*, comprising the inhalation of compressed air and the breathing into rarefied air, richly deserves further trial,[1] its use having been productive of permanent improvement in a number of cases, as shown by physical examination (including mensuration). Oxygen by inhalation has proved serviceable.

SENILE EMPHYSEMA.

This variety is a senile atrophy of the lungs, and has been appropriately termed "small-lunged emphysema" by Sir Wm. Jenner. In consequence of the complete atrophy of the alveolar walls, coalition of the air-cells takes place, with the production of large air-sacs. The lungs

[1] Waldenberg's portable apparatus is not convenient for use.

contain less than the normal volume of air, instead of an abnormal quantity as in hypertrophic emphysema, hence occupy less space in the chest-cavity than do healthy lungs. The pulmonary tissue is deeply pigmented. The condition does not produce right ventricular hypertrophy.

The **symptoms** are negative, although subjects in whom senile emphysema develops may have previously had chronic bronchitis with more or less dyspnea. They quite frequently present a withered appearance, and the chest on inspection is seen to be contracted, owing to the fact that the ribs approximate more closely and take a more oblique direction than in health.

Treatment is unavailing.

GANGRENE OF THE LUNGS.

Pathology.—The affection presents itself in two forms—as a (*a*) diffuse, and a (*b*) circumscribed process.

(*a*) The **diffuse** variety is rare. It may, however, be met with in lobar pneumonia, and very rarely in consequence of occlusion of the large branch of the pulmonary artery; it may also be secondary to the circumscribed form. The greater part of the lobe, or even an entire lung, may be involved, the pulmonary parenchyma degenerating into a putrid, greenish-black, pulpy mass, with no obvious line of demarcation.

(*b*) The **circumscribed** form may involve either one or both lungs, though the right is affected somewhat oftener than the left. To this category belongs the so-called *embolic gangrene*, the nodules of which have their favorite seat in close proximity to the pulmonary pleura. All etiologic varieties of the circumscribed form more frequently implicate the lower than the upper lobe of the lung, occurring in sharply defined areas, which may either be single or multiple. The affected area first presents a greenish-brown appearance; its central portion soon undergoes softening, and a cavity is thus formed whose walls are ragged and irregular and contain a foul-smelling, dark, greenish liquid. The surrounding lung is inflamed, and the air-sacs contain inflammatory products (fibrin, epithelium, pus), while the highly-irritating and putrid material sets up an intense bronchitis. These gangrenous foci may increase in size by a peripheral extension, and thus the adjacent veins may become plugged with infectious thrombi or the vessels may become eroded. Emboli may then be detached from the infectious thrombi, and, entering the circulation, may set up foci of septic inflammation in remote organs. A truly remarkable connection exists between circumscribed gangrene of the lung and cerebral abscess. When the gangrenous spot is situated near the pleura, simple or gangrenous pleurisy may arise as a complication, or the pulmonary pleura may be perforated and pyo-pneumothorax result. When recovery ensues the cavities formed as the result of the conversion of lung-tissue present a limiting wall of dense connective tissue. Such cavities may remain permanently or may slowly become contracted.

Etiology.—Gangrene of the lungs is caused by the bacteria of putrefaction (probably the staphylococcus albus or aureus). The disease is

rare. It is only when the lung-tissue has become impaired or peculiarly altered that the specific bacteria are capable of producing gangrene. It may occur in several ways:

(1) Secondary to lobar pneumonia, hemorrhagic infarctions, cavities in the lungs, bronchiectasis, wounds of the lung, contusions of the thorax, carcinoma of the esophagus, or to compression or embolism of the pulmonary artery or of the bronchial vessels.

(2) By lodgement of an embolus, derived from a gangrenous area in distant parts; this form is common, especially in children. The embolus is often the result of otitis media, mastoiditis, or thrombosis of the lateral sinus. There is a post-operative gangrene of the lungs.

(3) Pressure from a thoracic aneurysm may give rise to gangrene.

(4) The most important causal factor, however, is the entrance of foreign bodies, especially bits of food, into the bronchi and lungs. Whether or not the specific bacteria of putrefaction enter the lungs with the foreign bodies, the latter render the tissue-soil receptive to the former, and once the process has been initiated it is apt to extend itself. There are several ways in which these foreign particles gain entrance into the bronchi and lungs: (*a*) By a faulty swallowing of the food; (*b*) by inhalation; (*c*) by a carcinomatous perforation of the esophagus into the bronchus or into the lung.

(5) In debilitated states of the system, as during convalescence from protracted fever (rarely), and in diabetes mellitus (frequently).

Symptoms.—These are *local* and *general.*

Local Symptoms.—There is severe *cough*, which is accompanied by an exceedingly *fetid expectoration* that is usually quite profuse. When abundant, and when expectorated into a conical glass and allowed to stand for a time, it separates into three layers: (*a*) the uppermost, being frothy, opaque, and of a grayish-yellow color; (*b*) the middle, clear and watery; and (*c*) the lowest, appearing as a greenish-brown sedimentary layer containing shreds of lung-tissue and sometimes blood. The microscope shows it to consist of numerous elastic fibers, bacteria, fat-crystals, muco-pus, granular matter, and leptothrices. Small quantities of blood in the sputum are very common. Kannenburg and Streng have also described ciliated monads as occurring in the sputum. The patient's breath is, as a rule, intensely fetid, even though there be no expectoration, but this fetor of breath may be absent, as in a case of my own (which came to autopsy), in which the localized gangrenous process had no fistulous connection with the bronchus. If any of the large branches of the pulmonary artery be eroded, free and even fatal hemoptysis will result. *Pain* in the chest is complained of when the lesions are superficially situated.

Physical Signs.—The physical signs are sometimes obscure, as when the areas involved are smaller and deeply situated, and in such instances signs of bronchitis only may be detectable. When large and favorably situated, however, the affected spots usually give signs of consolidation, rapidly followed by those of cavity. In addition bronchial râles—usually moist—and coarse cavernous râles are usually audible. It is obvious that when the pleura is implicated the signs of pleurisy are added, and if pneumothorax be present those belonging to the latter condition also.

The chief **general symptoms** are irregular fever, emaciation, and

profound prostration. Leukocytosis is found. A septic condition of the system is commonly developed, and the patient sinks from exhaustion. The serious general features may overshadow the local in the lungs. Rarely there may be an almost total absence of constitutional disturbances, and such instances terminate in recovery.

Diagnosis.—The distinctive feature is fetidity, both of the sputum and the breath. The physical signs may readily determine the existence of the pulmonary lesion, but it is difficult to eliminate *abscess* and *fetid bronchitis* associated with bronchiectasis. The results of a careful examination of the sputum, together with the less horribly fetid odor of the breath, in *abscess* will usually suffice to eliminate the latter affection. In *fetid bronchitis* the fetor of the breath and sputum is less marked, while its course is slower and more favorable than in gangrene.

Prognosis.—The prognosis is always grave, though rarely recovery in circumscribed gangrene of the lungs ensues. The chief dangers are exhaustion and hemorrhage. Improved methods of surgical treatment, however, have saved life in a few instances, and promise to reduce still further the mortality-rate of this serious affection.

Treatment.—The leading indications are—

(*a*) The disinfection of the gangrenous focus or foci in the lungs. This may be accomplished by the internal administration of creasote or carbolic acid or by the use of an antiseptic spray.

(*b*) The patient's nutrition must be maintained, if possible, by a concentrated liquid diet, administered in fixed quantities and at regular intervals; also by the judicious cultivation of the digestive functions, together with the use of stimulants and tonics. Morphin is indispensable for the cough, which would otherwise rapidly induce exhaustion. Traube suggests lead acetate when the sputum contains blood. For a description of the surgical treatment of gangrenous cavities of the lungs the reader is referred to special works on surgery. It is the physician's duty, however, to determine whether or not the patient's general condition will admit of surgical intervention, and also to localize as nearly as may be the affected zones for the surgeon's guidance.

ABSCESS OF THE LUNGS.

(*Suppurative Pneumonitis.*)

Pathology.—This affection is characterized by the formation of pus and the degeneration of lung-tissue. It may be (*a*) a mere infiltration of the blood-vessels, bronchi, or interstitial tissue, but more frequently is seen as (*b*) an ordinary abscess. In size the abscesses range from that of a walnut to an apple, and I have observed in one case inflammation of the whole of the middle lobe of the right lung. The abscess-walls are irregular and decidedly ragged; and in the case of old lesions there is a dense fibrous wall; the contents are purulent and rarely necrotic. The most common seat ("80 per cent.") is in the lower lobes. If the contour of an abscess touches the pleura, empyema is the result. Rupture of the abscess into the pleura may also occur.

Etiology.—Streptococci are found, though they are not the only direct causes of abscess of the lung. The diplococcus pneumoniæ and Friedländer's bacillus have been found, as well as certain other organisms. *Predisposition* is noted in certain conditions, as (1) during or following the occurrence of inflammation, as in lobar and lobular pneumonia. Suppurative infiltration, however, more frequently arises under these circumstances than abscess, and in the rare instances in which the latter occurs it is apt to be comparatively small and multiple. In all forms of inhalation and deglutition broncho-pneumonia, however, abscess of the lung is a frequent sequela.

(2) Perforation of the lung from without or from adjacent organs, *e. g.*, esophageal carcinoma, hepatic abscess, or suppurating hydatid cyst.

(3) Infectious emboli, found in connection with septico-pyemia, frequently cause metastatic abscesses in the lungs. In a mechanical manner they may produce hemorrhagic infarctions, followed by suppuration, or the latter process may occur independently of the former. The abscesses are usually situated close to the pleura, and are frequently wedge-shaped; they vary in number from one to several hundred, and in size from a pin's head to an orange.

(4) Inward extension of a purulent pleurisy.

(5) As elsewhere stated (*vide* Pulmonary Tuberculosis), suppuration is quite generally associated with chronic pulmonary tuberculosis.

Symptoms and Diagnosis.—The examination of the *sputum* is of the greatest value in the diagnosis of this disease, since, being purulent, it usually presents a yellow, or less frequently a greenish- or brownish-yellow, color. It emits a fetor that is less pronounced than that of either gangrene or putrid bronchitis. Particles of lung-tissue may be visible in the pus, and on microscopic examination of the latter, elastic fibers, the presence of which is of the utmost importance in the diagnosis, may be found in profusion. The *physical signs* of cavity are of the greatest assistance in distinguishing abscess of the lung; these, however, are wanting unless the abscess is of a considerable size. The signs of cavitation, together with the characteristic sputum, leave no room for doubt. Chills and suppurative fever often attend. Leukocytosis is present. The history is of considerable importance, as confirming the more characteristic features. Thus antecedent pneumonia or septicopyemia would be strongly corroborative. *Tuberculosis* distinguishes itself by the history, the diminished amount of pus present, and the sputum test.

Prognosis.—The prognosis is often hopeless, as, for example, when the disease is associated with pyemic processes in other parts of the body. On the other hand, those rare instances in which it is secondary to pneumonia give a comparatively favorable outlook.

Treatment.—The chief aim in the therapeusis should be to support the system by the administration of tonics, stimulants, and antiseptics, as well as by energetic feeding with light forms of nourishment. Inhalation of antiseptic sprays (phenol, creosote, thymol) should be tried. When the abscess is situated near the periphery of the lung, surgical interference is to be advised as soon as the first indications of increasing weakness appear. Pulmonary abscess occurring as a sequel of pneumonia with free expectoration should receive an expectant treatment, unless it tend to become progressive, when it calls for operative intervention. For the details of the operation of pneumonotomy for pulmonary abscess

the reader is referred to works on surgery. The statistics of Eisendrath,[1] relating to abscess following pneumonia, may, however, be mentioned, as follows: of 25 cases of acute simple abscess, 24 recovered and 1 was improved; in chronic abscess the results were much less favorable.

PNEUMONOKONIOSIS.

(*Anthracosis, Chalicosis, etc.*)

Definition.—A form of chronic interstitial pneumonia that arises from the inhalation of dust-like particles. Different terms have been applied to the condition according to the nature of the dusts inhaled, the chief among these being—(1) Anthracosis (coal-miners' disease), due to the inhalation of coal-dust; (2) Chalicosis (stone-cutters' phthisis), caused by the inhalation of mineral dusts; and (3) Siderosis, caused by inhaling metallic particles, particularly iron oxid.

(1) **Anthracosis.**—Among dwellers in cities a moderate degree of pigmentation of the lung-tissue with coal-dust is the rule, while in those residing in rural districts the condition is decidedly less common. True anthracosis, however, has reference to such an accumulation of the carbon particles as can be due only to the inhalation of a well-laden atmosphere, or under other circumstances *e. g.*, when the mucous membrane is unhealthy or without perfect ciliary action. Under such conditions the normal scavengers of the respiratory organs—the mucous corpuscles lining the trachea, the bronchi, and the alveolar cells—fail to deal successfully with the numerous dust-particles that gain entrance along with the inspired air; hence some of the latter pierce the mucosa and reach the lymph-spaces and lymph-vessels. Here they are taken up by the leukocytes and are conveyed to a more remote destination. Arnold shows that after the particles enter the lymph-system they are carried "(*a*) to the lymph-nodules surrounding the bronchi and blood-vessels; (*b*) to the interlobular septa beneath the pleura, where they lodge in and between the tissue-elements; and (*c*) along the larger lymph-channels to the substernal, bronchial, and tracheal glands, in which the stroma-cells in the follicular cord dispose of them permanently," with resulting indurative enlargement of these structures. Rarely the carbon particles may find their way into the general circulation; this may occur, as shown by Weigert, when the pigmented bronchial glands become adherent to the pulmonary veins. Petit[2] claims that anthracosis may be of intestinal origin in cases in which the mesenteric barrier has already been broken down by tuberculosis.

Anthracosis leads, primarily, to chronic bronchitis, to be soon followed by emphysema; but extensive anthracosis may be present without any other changes in the lung than the presence of carbon particles stored in the protoplasmic cells. The lung-tissue presents great variations in its degree of susceptibility to these foreign particles. Sooner or later there is usually produced, as the result of their irritant action,[3] a

[1] *Phila. Med. Jour.*, Nov. 9, 1901.

[2] *La Presse Médicale.*

[3] Cohnheim contends that coal particles do not produce irritative changes in the lung, and that the latter are due to irritating substances inhaled with the particles of coal.

proliferation of the connective-tissue elements—*i. e.*, a chronic interstitial inflammation. This fibroid change usually starts in the peribronchial lymph-structures, though the bronchial and tracheal glands are, as a rule, similarly involved at a comparatively early period. The affected lung-tissue is frequently coal-black, dense, and airless. The pneumonokoniotic areas vary greatly in size and numbers, and not infrequently coalesce, in which case large portions of the lung-tissue may become the seat of fibroid change. The alveolar walls are much thickened in some instances, and firm pleuritic adhesions exist. Bronchiectatic cavities may be present, and later necrotic softening of the indurated areas occurs, leading to the formation of small cavities that contain a dark fluid. When the latter communicate with the bronchi their walls are prone to ulcerate. I have noticed that the process almost invariably terminates in pulmonary tuberculosis, and particularly is this true of cases that follow the inhalation of mineral and vegetable dusts (*vide infra*).

(2) **Chalicosis.**—Changes similar to those previously described are induced in the pulmonary connective tissue by the inhalation of stone-dust by those who follow such occupations as stone-cutting, knife- and axe-grinding, and millstone-making. The irritating properties of this form of dust are proved by the great disposition in this subvariety of pneumonokoniosis to the formation of fibrous nodules and diffuse areas of sclerosis in the lungs. The nodules have a gray center and a darker periphery; they are exceedingly dense, and sections are made with much difficulty.

(3) **Siderosis.**—This term implies a collection of iron oxid in the lungs, also due to the pursuit of certain occupations (dyeing, iron-smithing, etc.). Cases of much the same nature are caused by the inhalation of vegetable dusts by grain-shovellers, cotton-spinners, cigar-makers, etc. The *pathologic* changes are identical with those in anthracosis, though the color-appearance is red instead of black.

Symptoms.—Rarely the *onset* is marked by the symptoms of acute, followed by those of chronic, bronchitis; but in a vast majority of instances chronic bronchitis gradually develops after long exposure to the action of the exciting cause. The symptoms of emphysema are soon superadded, the patient now suffering from dyspnea, and less frequently from asthma. The *sputum* is diagnostic in anthracosis, being quite dark; in chalicosis a microscopic examination is essential to show the particles of silica; while in siderosis the expectoration presents a reddish color. Apart from the foreign particles, the sputum is for a long period of years muco-purulent in character, and later it often contains the tubercle bacillus.

The *physical signs* are not distinctive, being identical with those met with in chronic bronchitis associated with emphysema, and followed by those of interstitial pneumonia, and sometimes by those of cavity.

The **diagnosis** is to be made both from the history and from a gross or microscopic examination of the sputum. It may be confirmed by the invariable presence of the signs of bronchitis and emphysema, as well as by the effect of removal to an atmosphere free from dust. In the later stages the detection of infallible evidences of phthisis only serves to corroborate the earlier diagnosis of pneumonokoniosis.

An *acute pneumonokoniosis*, due to the inhalation of Thomas phosphate meal, has been described. This dust causes a diffuse pneumonic

inflammation affecting principally the lower lobes. The symptoms and progress of the cases are like those of lobar pneumonia.

The **prognosis** is favorable in hygienic surroundings until the more advanced stage is reached. The condition favors the invasion of new growths (lympho-sarcoma, or cobalt-miners' disease; *vide infra*).

Treatment.—A change of occupation or several hours of exercise in the open air daily for those who are exposed to dust in work-rooms should be advocated. Dusty work-rooms must be properly ventilated.

The active treatment is the same as for chronic bronchitis and emphysema from other causes, and is to be appropriately modified when pulmonary tuberculosis develops.

NEW GROWTHS OF THE LUNGS.

CARCINOMA OF THE LUNG.

ALL varieties of carcinoma have been met with in the lung, but, with rare exceptions, carcinoma of this organ is of secondary origin. Ordinarily the primary new growth involves a vein or lymph-channel, and the latter carries the germ of the disease to the lung. It is also to be recollected that it may result from extension, or by contiguity from neighboring organs (as the esophagus, mamma, pleura, or mediastinum).

Etiology.—The causes of primary carcinoma of the lung must be, in the main, identical with those of carcinoma in general, and are as yet unknown. Most cases occur in middle-aged persons, and, while sex has an influence upon the appearance of the primary form of the disease, it occurring much oftener in males, especially those exposed to the vapors of arsenic, the secondary form is more frequent in the female than in the male. In the female, secondary carcinoma of the lung is often preceded by carcinoma of the breast. We may also regard hereditary influence as a potent predisposing factor. Secondary carcinoma of the lung is most commonly consecutive to primary carcinoma of the bones, and of the digestive and urinary tracts.

Pathology.—The pathologic varieties of the primary form are scirrhous, encephaloid, and epithelioma, and of these the latter is the most common. *Primary carcinoma* is usually unilateral, the tumors attaining to a massive size and frequently involving the greater part of one lung. Their favorite seat is in the upper part of the right lung. Henrici claims that most cases have their origin in the bronchial epithelium. Extension to the pleura occurs quite often. Less frequently there is pleurisy with sero-fibrinous exudate, which may be hemorrhagic. Carcinomatous involvement of the cervical, bronchial, and tracheal lymph-glands is quite usual, and rarely even the inguinal glands become implicated. *Secondary carcinomata* are, as a rule, multiple, and may be miliary in size. They are disseminated widely throughout both lungs, though in the rarest instances they may be unilateral. In the softer varieties the central portion of the tumor-mass may undergo fatty degeneration, with subsequent discharge through adjacent bronchi.

Symptoms.—The symptoms vary according to the location and extent of the disease. Among the most marked symptoms belongs *pain*, particularly when the pleura is implicated. As a rule, for a considerable period of time the symptoms of *bronchitis* obtain, and later the breathing-

space is diminished sufficiently to excite dyspnea and cyanosis. With the increase in size of the new growth compression of the heart, aorta, and large veins may result, whereupon *disturbances of the circulation* will arise. The new growth may exert pressure on the esophagus, causing *dysphagia;* or upon the recurrent laryngeal nerve, causing *aphonia* and *hoarseness;* or on the trachea or a main bronchus, followed by the symptoms of *stenosis* of those organs. There are *cough* and *expectoration*, the latter frequently containing blood-corpuscles with mucus, and resembling in appearance currant-jelly; the sputa may also rarely exhibit a grass-green color, due to transformation of the blood-pigment. In carcinomatous lungs putrefactive changes sometimes take place, and if so the expectoration and breath emit an offensive odor, while a microscopic examination of the sputum frequently discloses the presence of carcinomatous elements. A leukocytosis, usually of moderate degree, may be present. The well-known cancerous cachexia invariably develops.

Physical Signs.—These will naturally depend upon the extent and location of the new growth. *Inspection.*—If the lung-tissue be extensively involved, the walls of the thorax become unduly prominent and fixed over the seat of the tumor. Indeed, the tumor may, though rarely, protrude between the ribs. The intercostal spaces are widened, and the superficial veins, in view of the fact that they cannot empty themselves into the internal veins, appear engorged; from the same cause edema affecting the thorax, neck, face, and arms may be noted. Swelling of the lymph-glands in the neck or axilla is an important sign. On *palpation* the tactile fremitus may be diminished or absent. The *percussion-note* will be flat, since the air-vesicles and smaller bronchi are replaced by the solid growth. On *auscultation* friction-sounds are the rule. The respiratory sounds may be greatly enfeebled or absent; but if the carcinomatous tumor communicates with a wide-mouthed bronchus, bronchial breathing may be audible, and the physical signs of lung-cavity may be developed. The signs of general bronchitis are present in most instances, especially in the disseminated form of the disease; in the latter the lung may shrink, with retraction of the chest-walls on the affected side. If secondary pleurisy with effusion occurs, the detection of the characteristic cancer-cells in the contents of the pleural cavity will show the nature of the thoracic affection.

Diagnosis.—The following symptom-group will pretty well establish a diagnosis: A peculiarly shaped dull area (as when it extends under the sternum), perhaps a marked prominence over the site of the tumor, enlarged and hard lymphatic glands in the vicinage, and certain of the compression-symptoms — circulatory, nervous, bronchial, or tracheal. Rarely the diagnosis may be made by the occurrence of metastasis to the chest-wall. Again, the discovery of cancer tissue in masses accidentally detached gives reliable indication of the disease. An exact diagnosis can often be made from an examination of the particles obtained on aspiration of the tumor and pleural effusion.

The **differential diagnosis** between pulmonary carcinoma and *pulmonary tuberculosis* can be made with positiveness only by a careful microscopic examination of the sputum. From *fibroid induration* of the lung it is easily discriminated, owing to the history and slower course of the latter affection.

Prognosis.—This is bad, as death may occur suddenly from abundant hemorrhage or more frequently from either exhaustion or asphyxia. The duration of the affection varies from six months to a year or more.

Treatment.—The treatment must be addressed chiefly to the relief of pain and other subjective symptoms, though the effect of the *x*-ray should be tried.

SARCOMA OF THE LUNG.

Primary sarcoma of the lung is rare, but in instances of generalized sarcomatosis the lungs show larger or smaller nodules "in almost every case" (Birch-Hirschfeld), occurring in connection with osteo-sarcoma of other organs or in lympho-sarcoma of the cervical glands.

Secondary sarcoma of the root of the lung by sarcomatous disease of the post-bronchial glands, is more common than secondary carcinoma. The *diagnosis* is reached as in carcinoma (*vide* p. 578).

Neoplasms occurring among the cobalt-miners of Schneeberg were described by Hesse and Tragner as lympho-sarcomata—slowly growing masses that attained to a large size and gave metastasis to lymph-glands, pleura, liver, and spleen. In most cases there was an associated pneumonokoniosis, which had probably predisposed to the new growth.

HYDATID CYST OF THE LUNG.

Hydatids in the lungs may either be primary or secondary, the former variety being exceedingly rare and the latter somewhat less so. Almost invariably the echinococci are developed in other organs—the liver in particular—and find their way to the lungs, either by direct perforation through the diaphragm or by entering through the blood-current. The lungs are involved in about 12 per cent. of hydatid disease.

For **etiology** and **pathology** see Hydatid Cysts of the Liver.

Symptoms.—The clinical manifestations are quite varied, even though the cyst may entirely conceal itself. It is important to recollect that similar involvement of the liver usually coexists; and in addition to the symptoms of the latter affection there may be *pain* in the chest, *dyspnea*, considerable *cough*, and, rarely, blood-stained *expectoration*. General weakness and emaciation may attend the more advanced stages.

The **physical signs**, when present, are as follows: Diminished vocal fremitus, defective expansion, dulness on percussion with an absence of the respiratory murmur—all signs pointing to pleural effusion. The cysts are more common in the right lung and frequently cause marked bulging over the base. Later signs of cavity-formation may appear. In other cases, the signs of consolidation may preponderate.

A positive **diagnosis** of hydatid cyst of the lung can be made only when the scolices, pieces of membrane, or the hooklets of the echinococcus are demonstrable either in the sputum or the aspirated fluid. Besides being evacuated into the bronchi, the cysts may rupture into the adjacent serous sacs (pleura, pericardium), or externally, the latter being the most favorable termination. Unless they are discharged by ulceration into the bronchi or externally, they are apt to excite inflammation of the adjacent lung-tissue and tubes, accompanied by an active febrile movement and an aggravation of the aforementioned symptoms: these

complications (pneumonia, gangrene) may assume a dangerous form, or the patient may, if the growth becomes large, become asphyxiated. From *gangrene*, *pleurisy*, and *phthisis* echinococci are distinguished by the sputum-test or by an examination of the aspirated fluid.

Prognosis.—The affection is always attended with great danger, and is of more serious import when secondary to involvement of the liver than when primary.

Treatment.—When it can be shown that the growths are situated at the periphery of the lung operation should be carefully considered. The physician stands powerless to do more than to relieve urgent symptoms in special cases and to support the vital functions.

V. DISEASES OF THE PLEURA.

PLEURISY.

(*Pleuritis.*)

Definition.—An inflammation, either local or general, of one or both pleural membranes. The disease, as shown by postmortem examinations, is of great frequency.

Varieties.—Pleurisy has been variously classified. *Etiologically*, the distinction between primary and secondary forms of the disease should be made, as well as a division into tuberculous, carcinomatous, septic, etc. *Pathologically*, all cases may be summarized under the following heads: Localized and generalized and dry (plastic) pleurisy and pleurisy with effusion (sero-fibrinous, purulent, hemorrhagic). They may also be classified according to their duration into acute, subacute, and chronic pleurisies. I shall describe the following forms, which are based partly upon their etiology and clinical course, though mainly upon their pathologic manifestations—viz. (*a*) acute plastic pleurisy; (*b*) sero-fibrinous pleurisy; (*c*) purulent pleurisy (empyema); and (*d*) chronic adhesive pleurisy.

Bacteriology.—In all forms of the disease the *direct* causes are various micro-organisms or their irritating chemical products. Conspicuous among these is the *bacillus of tuberculosis*. Inoculation of guinea-pigs with the latter by Eichhorst gave positive results in 15 out of 23 cases, and by La Damany in 47 out of 55 cases. By taking a large amount of exudate either for cultures or inoculation of animals, the bacillus tuberculosis can be found, as a rule. Netter, Prudden, and others have found in the exudation of **fibrino-serous pleurisy** the *streptococcus pyogenes*, the *staphylococcus*, the *typhoid bacillus*, and the *diplococcus* of *pneumonia*. The micro-organisms most commonly present in **empyema** are the *micrococcus lanceolatus* and the *streptococcus*, the former especially in the pleurisy associated with pneumonia (in two-thirds of the cases occurring in children—Levy), and the latter in those independent of pneumonia, particularly in adults. Among other bacteria that have been found rarely in the effusion are the *colon bacillus*, the *proteus*

vulgaris, the *gonococcus*, the *ameba coli*, *Friedländer's bacillus*, *anthrax bacillus*, *influenza bacillus*, and various saprophytic bacteria. Except in the case of the pleuritic exudation in pneumonia, in which the diplococcus is alone present in about one-half of the cases, the afore-mentioned micro-organisms are generally found in association.

ACUTE PLASTIC PLEURISY.

(*Dry, Fibrinous Pleurisy.*)

Pathology.—The lesions are usually circumscribed, the part inflamed being intensely injected. It has lost its natural lustre, and instead has a dull, non-glistening surface "like a tarnished mirror," due to a slight fibrinous exudate. Minute ecchymoses are seen. Later the exudate may become more copious, when the pleura presents a rough, shaggy appearance. On account of the friction between the two pleural membranes in high grades of dry plastic pleurisy, the exudate may be very thick, and its color-appearance is then yellowish- or reddish-gray. This sheeting of fibrinous exudate entangles in its meshes numerous embryonic round cells, out of which blood-vessels and connective tissue are developed. The opposing surfaces of the pleura adhere. Occasionally, in the lighter grades, the disease does not advance to firm adhesion, and in such instances the products of the exudate undergo fatty degeneration and are absorbed.

Etiology.—The affection may be (*a*) primary or (*b*) secondary. (*a*) By the *primary form* is meant an inflammation of the pleura occurring in previously healthy persons. It is exceedingly rare, and doubtless many instances of true secondary pleurisy are regarded as belonging to this category. Aschoff's studies of 200 cases of pleurisy showed 41 to be idiopathic. Of great etiologic prominence is exposure to cold and wet, and next to this stands mechanical injury. It is more common in men than in women, and especially during the time of active life, on account of the greater liability to exposure of the former sex. In almost all instances a careful search will disclose the existence of some diathesis (tuberculous, gouty, rheumatic) that may be properly regarded as the favoring cause. The changeable weather of the winter and spring augments the proportion of cases during these seasons as compared with summer and autumn.

(*b*) The *secondary form* of dry plastic pleurisy arises from extension of acute and chronic inflammatory affections of the lungs and other neighboring organs. Hence it frequently follows croupous pneumonia, somewhat less frequently broncho-pneumonia, and more rarely still hemorrhagic infarct, abscesses, and pulmonary carcinoma and gangrene. When pleurisy occurs on the right side it must be recollected that it may have originated in hepatitis. Plastic pleurisy sometimes arises in acute articular rheumatism, to which it may essentially belong. It is an almost constant accompaniment of chronic pulmonary tuberculosis, and may, though rarely, even constitute the primary lesion (primary tuberculous pleurisy). The disease may appear as a complication in chronic alcoholism and in chronic Bright's disease. Finally, inflammation of other serous membranes, as of the pericardium and peritoneum, by direct extension through the lymphatics may invade the pleura.

Symptoms.—The affection may vary in intensity between the extremes of mildness and great severity, though, as a rule, well-marked local symptoms attend the onset. Among the latter a *sharp "stitch" in the side*, that is usually referred to the nipple, is the most prominent. The pleural *pain* is increased by inspiration as well as by voluntary motion of the affected side, and hence the patient assumes a fixed position in which he favors the affected side by leaning toward it. There is a dry, distressing *cough* that is restrained for obvious reasons, and the *respiration* is somewhat hurried, painful, and jerking in character until the exudation is poured out, when relief from the latter symptom ensues.

The general symptoms are not pronounced, and, save in comparatively rare instances, do not correspond with the local signs. The temperature is not typical, rarely exceeding 103° F. (39.4° C.), and more often it is below 101° F. (38.3° C.). The pulse is usually small and tense or soft in character, registering from 90 to 120 beats per minute. Not infrequently the cases are so mild as to be attended by few, if any, subjective symptoms. The patient may complain of ill-defined, uneasy sensations in the affected side, but does not discontinue his usual occupation. On the other hand, the worst cases of acute plastic pleurisy—which, fortunately, are rare—manifest violent symptoms: there is a distinct chill, a speedy development of high fever (104° F.—40° C.), and profound prostration, and the general and local symptoms are proportionately aggravated. The illness then is often a fatal one.

Physical Signs.—On *inspection* the movements of the chest-wall on the affected side are observed to be much restricted, particularly during the first day of the affection. *Palpation* confirms the results of inspection, while *percussion* yields a normal note. *Auscultation* renders audible a grazing friction-sound, most intense at the end of inspiration. These signs are not uncommonly situated at the apices.

With the occurrence of fibrinous exudation *palpation* detects over the corresponding area a diminution of the tactile fremitus. On *percussion* there is, as a rule, a slight though variable degree of dulness; and on *auscultation* rubbing friction-sounds or a rustling sound due to fine râles are heard both on inspiration and expiration, being intensified by deep breathing. These sounds frequently persist for a day or two after the other symptoms have disappeared. Rarely the plastic exudation may be so extensive as to cause compression of the lung, in which instance the breath-sounds may become bronchial in character; and such cases have been mistaken for lobar pneumonia.

Diagnosis.—By exercising ordinary care the clinician can scarcely mistake other thoracic affections for dry pleurisy, the latter being diagnosticated to a certainty by the presence of the characteristic friction-murmur. *Intercostal neuralgia* may present features not unlike those of acute pleurisy. In both affections there is frequently a history of exposure, followed by severe chest-pains that are excited by coughing and deep breathing. In neuralgia, however, there are painful pressure-points, and the friction-sound does not occur. Schepelmann points out that bending the trunk to the side affected increases the pain of intercostal neuralgia, while in pleurisy this symptom is aggravated by bending the trunk to the sound side. *Pleurodynia* may also give a history very similar to that of acute pleurisy, but the characteristic physical signs of pleurisy are absent.

Prognosis.—The duration of the affection varies from a few days to three weeks, and the immediate outcome is favorable as a rule. Undoubtedly, however, a primary attack predisposes to subsequent attacks, and thus, as a result of repeated seizures, pleural thickening and intrapleural adhesions often arise. Lung-expansion may in this manner be restricted, with the gradual development of interstitial pneumonia as a consequence. Acute plastic pleurisy is not infrequently a terminal condition in serious forms of illness (*e. g.*, septicopyemia and chronic nephritis).

Treatment.—The first object in the treatment is to relieve the pain, and this can best be accomplished by the hypodermic use of morphin. The inflammatory process is best controlled by absolute *rest* in the recumbent posture, or fixation of the affected side by means of adhesive plaster. I am also in the habit of administering moderate-sized doses of quinin (gr. iv—0.259—three times daily). After the exudation has appeared, the iodids of iron and potassium, in combination, may be employed. *Locally*, nothing is so effective as cold in the form of the ice-water bag or Leiter's coil, preceded, in robust patients, by the local abstraction of blood (℥iij to vj—96.0–192.0) by leeches. At the end of one week the morphin may usually be discontinued. During *convalescence* the patient should be instructed to take deep inspirations several times in succession, not less than a dozen times each day, with a view to obviating as far as possible pleural adhesions and other unfavorable consequences. Symptomatic anemia may be present at this time, and should be met by iron given internally. At this time iodin may be used locally with great benefit; I have not, however, seen any favorable results from blisters. For the pain which continues in the side after all detectable physical signs have disappeared the use of the constant current over the seat of the pleurisy for twenty minutes at a time gives almost instantaneous relief (Loomis).

SERO-FIBRINOUS PLEURISY (PLEURISY WITH EFFUSION, SUBACUTE PLEURISY).

Pathology.—During the first stage of sero-fibrinous pleurisy the changes are the same in character as those met with in dry pleurisy, though of severer grade, and usually involving the greater portion of the pleura on the side affected. There is an abundant exudation of serum, and usually the entire pleura becomes coated with a fibrinous exudate, that varies greatly in thickness and arrangement. The exudate is thin and smooth in some instances, though more frequently it forms a thick layer, presenting a shaggy surface on the one hand or an irregular, honeycombed surface on the other. Lymph in the form of flocculi is rather abundant in the serous effusion. The interlobular pleural surfaces are also invaded as a rule, in consequence of which they become adherent. The fluid exudate varies greatly in quantity (½ to 8 pints—4 liters), is often of a citron color, and is, in the majority of instances, clear or slightly turbid. Rarely it is of a dark-brown color.

Unless adhesions between the pleural surfaces have previously existed the effusion gravitates to the most dependent portion of the pleural cav-

ity. Microscopically, there are found leukocytes, red blood-corpuscles, endothelial cells, threads of fibrin, and, rarely, crystals of cholesterin and uric acid. The composition of the fluid is almost identical with that of blood-serum, and on boiling it is found to be rich in albumin. Spontaneous coagulation may take place on standing.

Changes in the Neighboring Organs.—So long as the normal retractility of the lung is not overcome by the fluid that collects in the pleural cavity, the latter does not produce positive intrathoracic pressure, and hence does not produce displacement of adjacent organs. It may be assumed that until the pleural sac is at least one-half filled with sero-fibrinous exudate the natural contractility of the lung is not destroyed. At this period there may be a slight displacement of the mediastinum toward the opposite side, due to traction exerted by the normal retractility of the sound lung. Obviously, large effusions must in a mechanical manner displace the pleural membranes, thus causing compression of the pulmonary structures lying above the effusion. A very copious effusion may push the lung up and back against the vertebral column and convert it into a small, flat, bloodless, and airless mass (atelectasis). While a total absence of air in the collapsed lung is due chiefly to compression by the fluid, to some extent, however, the air may be absorbed by the vessels or even by the effusion (Strümpell).

Together with compression of the lung by the effusion, pressure is also exerted by the latter against the mediastinum, causing displacement of the heart. The mediastinum also loses the normal traction-force of the lung upon the affected side, and hence the lung on the sound side draws the mediastinum toward itself by its own retractile energy. Osler shows that even in the most extensive left-sided effusion the heart's apex is not rotated, but that the normal relative position of the apex and base obtain, though the apex is in some instances lifted, and in others the heart lies more transversely. The right chambers of the heart occupy most of the anterior part of the organ, showing that the displacement of the mediastinum with the pericardium and its contents to the right involves no appreciable twisting of the heart itself.

Downward displacement of the diaphragm takes place in extensive effusion on the right side, depressing the liver to a variable distance below the inferior costal border; on the left side large effusions produce pressure-displacement of the stomach and the transverse colon, and, to a slighter extent, of the spleen. Pre-existing adhesions may prevent displacement of the adjacent organs.

Etiology.—The causative factors are identical in nature with those producing dry plastic pleurisy. It is highly probable that the degree of severity is dependent upon the previous condition of the patient, whether he be suffering from some other affection or not, and upon the amount of specific poison gaining access to the pleura.

The affection may be *primary*, but is much more often *secondary*, and this fact may be explained by reference to any of the specific micro-organisms producing the affection.

Direct Causes.—Many of the cases follow quickly upon exposure to cold or wet or an injury to the thorax. I thoroughly agree with those authors who contend that about three-fourths of the cases of sero-fibrinous pleurisy are of tubercular origin. The tuberculous process may invade

the pleura primarily, but more often it is secondary to tuberculosis of the lungs; less frequently, though oftener than is generally supposed, it is secondary to tuberculous peritonitis. In these instances the tubercle bacilli probably find their way from the peritoneum to the pleura by traversing the lymphatics in the diaphragm. A large percentage of apparently primary cases of tuberculous pleurisy have their origin in a circumscribed and more or less latent tuberculous focus in the lungs. It is not improbable also that tuberculous processes in other viscera may furnish the tubercle bacilli for secondary pleural infection. Moreover, the fact that many cases of sero-fibrinous pleurisy recover does not disprove their tuberculous nature.

The affection is not infrequently secondary to acute articular rheumatism, which is itself most probably a microbic affection. It also arises as a complicating condition in the course of various acute and chronic affections of the chest, as pericarditis and catarrhal pneumonia, and may develop in acute infectious diseases, as typhoid fever or lobar pneumonia. The typhoid bacillus of Eberth has also been known to provoke pleurisy (Bozzolo, Fernet, and others).[1] It may occur as a complication in the chronic affections of various viscera (chronic nephritis, cirrhosis and carcinoma of the liver). The *predisposing causes* are the same as for the dry plastic form.

Symptoms.—The description here refers particularly to primary sero-fibrinous pleurisy, and it is important to recollect that when secondary to other acute and chronic affections characterized by great bodily weakness the pleuritic symptoms may be in abeyance.

With few exceptions the onset is *insidious*, the symptoms being quite mild; but rarely there is a *sudden onset* with active symptoms (rigor, high fever). In the majority of instances the patient first complains of a *stitch-like pain* in the side; this is rarely pronounced, but is aggravated upon deep breathing and upon any muscular exertion. *Dyspnea* soon arises and gradually increases in intensity. *Cough* may be present or absent, and in some instances is attended by a scanty mucoid expectoration that may rarely be blood-streaked.

The *constitutional symptoms* are of correspondingly slow and gradual development. From the commencement of the attack a moderate febrile movement at night may be observed, and the pulse will be found to be frequent, small, and compressible, or, more rarely, tense. At the time of the patient's first visit to his physician he may give a history of having gradually lost flesh and strength for a period of weeks together, though he may not have been obliged to abandon his vocation. He looks pale, his countenance wears an anxious expression, and he is without appetite. These cases frequently drag on from two to four weeks before consulting a physician, the local symptoms going unnoticed.

Sometimes the period of invasion develops acutely and after lasting a few days the symptoms exhibit a decided remission; subsequently there may be a sudden recurrence of the local and general phenomena, and particularly of the dyspnea. The pleural cavity, which may have been one-half or two-thirds full, now becomes completely filled.

Special Symptoms.—*Pain.*—Chest-pain is an almost constant but not highly characteristic symptom, and, though usually among the earliest symptoms, it may not be present until a few hours or a day after the

[1] *Annual of the Universal Medical Sciences*, vol. ii., p. 12.

commencement of the affection. It may be described as a sharp, shooting pain, and is popularly termed a "stitch in the side." It may, however, be tearing or dragging in character. Its intensity is not a safe indication of the severity of the disease. It is usually referred to a small spot below the nipple or to the mid-axillary region; exceptionally, however, it is more diffuse, and in my experience it has not infrequently been retrosternal or referred to limited areas below the inferior costal border. When absent it may be excited by coughing, sneezing, deep inspiration, and stooping. With the appearance of the effusion the pain diminishes, and, as a rule, soon disappears.

Dyspnea.—The breathing is shallow, "catching," inspiration being made up of a series of gasps, and it is hurried in consequence of the severe pleural pain; in copious effusions, that render one lung functionless, the dyspnea may become intense, even attaining to orthopnea. It reaches its most pronounced form in previously robust subjects, and in those in whom the effusion has developed rapidly. On the other hand, when the pleural sac fills slowly dyspnea may be absent except on exertion. Following marked disturbances in the respiration, *cyanosis* appears and may become quite marked.

Cough and Expectoration.—Little need be added to what has already been stated. When there is present much expectoration it is not uncommonly due to associated bronchitis or to pulmonary tuberculosis; there may, however, be a total absence of expectoration, and in such instances the exciting cause of the cough is probably the pleuritis. Both the cough and expectoration are apt to be increased during the process of resorption of the exudate as the result of a catarrhal bronchitis that is prone to develop in the re-expanding lung.

Fever.—The rise of temperature is not rapid as a rule, nor does it reach a high point (101.5° to 103° F.—38.6°–39.4° C.). At the end of a variable period—usually one to three weeks—the temperature falls by lysis, and soon touches the normal. The temperature may be of the continued type in many acute cases. In subacute forms the temperature rarely rises above 101° F. (38.3° C.), or the fever may, finally, become hectic. The surface-temperature of the affected side is from one-half to two degrees (0.4°–1.6° C.) higher than that of the normal side.

Pulse.—The pulse is quickened, beating 100 or more per minute, and its volume and tension are diminished. Irregularity both of the volume and rhythm of the pulse may also be observed. These pulse-characteristics are to be attributed to the pressure of the effusion upon the heart and great vessels. There is a leukocytosis in non-tuberculous pleuritis, although the increase in the white cells is moderate.

Gastro-intestinal Symptoms.—Loss of appetite is commonly present, and more rarely nausea and occasional vomiting may arise at the outset. Constipation is the rule. *Sweating* is a common symptom in the more protracted cases.

Renal Symptoms.—The amount of urine is diminished both during exudation and while the exudate remains at its maximum level. The daily quantity may not exceed eight or ten ounces, but the specific gravity is increased, ranging from 1018 to 1028. Rarely, the quantity is increased with existing effusion. An increase in the daily amount of urine excreted is frequently the first sign of commencing absorption of the exudate, and the rapid resorption of the copious effusion may greatly

augment the flow of urine to 80 or 100 ounces (2.5 to 3 liters) daily (Strümpell). The cause of the diminished secretion of urine is, in the main, diminished arterial pressure.

Physical Signs.—The physical signs of sero-fibrinous pleurisy differ with the stage of the affection: those of the first stage are identical with the signs pointed out in connection with dry plastic pleurisy, and need not be restated here. We will note the physical signs (1) during the stage of effusion, as well as (2) those presented when absorption of the effusion has taken place.

(1) *Stage of Effusion.*—When the pleural sac is only partly filled there is noted, on *inspection*, but little change in the thoracic contour. The respiratory movements are, however, restricted, owing to mechanical hindrance to the lung-expansion. In the majority of instances the effusion increases until positive intrathoracic pressure and noticeable bulging in the middle and lower third of the chest-wall on the affected side take place; the intercostal spaces below are shallow, widened, and sometimes even effaced. The apex-beat of the heart is displaced, being visible in right-sided pleurisy to the left of the vertical mammary line in the fourth and fifth interspaces, and in left-sided pleurisy to the right of the right mammary line in the third and fourth interspaces. The apex of the heart may take a position behind the sternum, when no impulse will be visible. In moderate effusions rhythmic lateral displacement of the heart (which approaches the affected side during inspiration and moves outward in expiration) occurs (C. L. Greene). Litten's phenomenon, or the shadow of the diaphragm, is absent in this disease.

Palpation.—The limited range of expansion is readily appreciated on palpation, and in large effusions the chest-wall is practically fixed. The separation of the ribs and the obliteration of the intercostal spaces are easily made out in the same manner. Edema of the chest-wall is rarely present, and fluctuation almost never. An important and early physical sign is the diminished tactile fremitus, which is soon abolished, except in infants, in whom it may be excited on crying. This is a less valuable sign in women than in men, owing to the differences in the vocal vibrations in the two sexes. In copious effusions tactile fremitus may sometimes be obtained when bands of adhesion, which serve as a medium for the transmission of vocal fremitus, connect the pulmonary with the costal pleura. The apical impulse can also be readily located by palpation. The displaced spleen or liver can be felt through the abdominal wall, and must not be mistaken for an actual enlargement of these organs.

Mensuration.—In right-handed adults the right side is, normally, slightly larger than the left; and it is only after the effusion is considerable that the cyrtometer shows any alteration in the thoracic contour. The tape, however, exhibits the difference in expansive motion of the two sides early. At the end of expiration the circumference of the affected side will be found to be one or two inches greater than that of the unaffected side, while at the end of inspiration the difference will be but slight. The cyrtometric tracing also shows a discrepancy between the horizontal outlines of the two sides.

Percussion.—At first the percussion-note is impaired, either posteriorly or in the infra-axillary region, and a little later there is dulness, tending toward flatness (deadness), the upper level of which rises

from day to day with increasing effusion. Over the exudate the note has a wooden quality (flat) and there is great resistance. When the effusion rises to the fourth rib anteriorly there is dulness over the fluid above and absolute flatness below. Since both the flatness and dulness are due to the free fluid, it is obvious that the line of demarcation must change with the posture of the patient; hence the limit of dulness will be higher in the sitting than in the recumbent position. When the pleural sac is filled or when the effusion is confined by adhesions, movable dulness is not obtainable. When the exudate rises to the lower border of the third rib, the percussion-note above the line of dulness is tympanitic or vesiculo-tympanitic (*Skoda's resonance*); this holds also in more moderate effusions, and is attributable to mediate relaxation of the lung. In copious exudations the cracked-pot sound may be elicited immediately below the clavicle, and "*Williams's tracheal tone*" may sometimes be obtained. This may also be obtained at a point corresponding to the seat of the compressed lung. When the patient is sitting or in the erect posture the upper limit of dulness in large effusions is not a horizontal line, but is highest at the spine and falls as we proceed to the front, which is its lowest point. The upper line of dulness in moderate effusions begins "relatively low down in the back, passes upward from the vertebral column, and soon turns upward and proceeds obliquely across the back to the axillary region, where it reaches its highest point; thence it advances in a straight line, but with a slight descent, to the sternum" (Ellis). This curved line resembles the italic letter *S* (Garland). Grocco's sign (a triangular area of dulness over the back on the opposite side in unilateral pleurisy, which dulness disappears when the patient lies on the side of the effusion) is confirmatory, although it is not invariably present. On the right side the flatness is continuous with that of the displaced liver; on the left it passes into and may obliterate Traube's semilunar space.

Auscultation—The signs of the first stage have already been described (*vide* Plastic Pleurisy). With the appearance of the effusion the breath-sounds become weak, distant, and have a bronchial quality. Soon the respiratory sounds over the affected side will be entirely absent, except near the upper level of the fluid posteriorly, where distant bronchial breathing is audible. The latter sounds may exhibit a metallic or amphoric quality, and may be accompanied by râles (pseudo-cavernous signs). The latter are more frequently met in children than in adults, and often give rise to a false diagnosis. Above the level of the fluid there is broncho-vesicular breathing, and on the opposite side intensified breath-sounds may usually be noted. In pneumonia with pleural effusion there may be loud and persistent bronchial respiration over the exudate. The vocal resonance is diminished and may manifest a nasal quality, simulating somewhat the bleating of a goat (*Laennec's egophony*). This is best obtained near the upper level of the fluid in large effusions, and at or above the angle of the scapula when the effusion is moderate.

(2) *Stage of Resorption.*—With resorption of the fluid there is a decrease in the size of the affected side, together with a return of the normal appearance of the intercostal spaces and the respiratory movements. In many instances there is positive retraction, leading to thoracic deformity with displacement of neighboring organs toward the affected side; and this retraction may be either general or circumscribed. The infe-

rior intercostal spaces are more or less narrowed; the shoulder droops; the nipple approaches the median line; the spine may be curved, the convexity being directed toward the sound side (quite rarely toward the affected side); and the scapula projects from the chest-wall on the affected side. In children, and even in adults, the lungs and thorax gradually expand in order to overcome this chronic deformity.

Palpation.—The tactile fremitus closely follows the fluid as it subsides from above downward without any extreme degree of thickening of the pleural membranes, though cohesion of their surfaces may prevent its return over the lower segment. The inspiratory movement of the chest-wall gradually returns, but not to its former limit.

Mensuration shows a steady diminution in the size of the side involved, which finally becomes smaller than its fellow.

Percussion.—The dull or flat note gives way to normal percussion-resonance, proceeding from above downward in a gradual manner; but the latter is not renewed over the lower portion of the pleural cavity for a long period after the exudation has disappeared. The abnormal areas of flatness due to displacement of organs (liver, spleen, heart) also disappear.

Auscultation discloses most important signs during the stage of absorption. The breath-sounds reappear at first above, and then lower down, until the base is reached. With commencing subsidence of the fluid the respiratory sounds are feeble and distant, but later they resume their natural distinctness; and partly as a result of the revival of the natural muscular tonicity, and partly in consequence of the disappearance of the fluid, the two roughened pleural surfaces come in contact and play upon one another, giving rise to a rubbing, creaking friction-sound on auscultation. These friction-murmurs may persist for months after the effusion has been absorbed. Occasionally the lower portion of the compressed lung remains permanently inexpansile; the upper portion of the lung is now the seat of compensatory emphysema. The heart-sounds return to their normal position.

X-rays.—Williams[1] states when the effusion is large no more rays pass through it than through the liver, and the outlines of the diaphragm, ribs, and heart are obliterated on the side of the effusion. The fluoroscope also shows the direction and extent of cardiac displacements due to pleural effusions. Williams affirms that displacement of the heart to the right may not be recognized by percussion, even when it has been pushed much beyond its normal place. Displacement of the pleuritic fluid when the patient's position is changed and also with the movements of the diaphragm has been noted with the fluoroscope (Bergoné and Carrière).

Special Clinical Forms of Acute Sero-fibrinous Pleurisy.—(1) **Tuberculous Pleurisy.**—This is, in the majority of instances, secondary to pulmonary tuberculosis. On the other hand, the primary lesions may be situated in the pleural sac and give rise to (*a*) *Acute sero-fibrinous pleurisy* (with the usual course); (*b*) *Subacute pleurisy* (with insidious course), leading to tuberculous invasion of the lungs; and (*c*) *Chronic adhesive pleurisy*, in which the course and physical signs correspond with those to be depicted in a special section on Chronic Pleurisy.

The morbid lesions are similar to those met with in other forms, plus the specific tubercles, which may be exceedingly numerous (miliary

[1] *Philadelphia Medical Journal*, January 6, 1900.

tubercles) on the one hand, or confined to a few circumscribed areas on the other. This variety has no special etiologic connection with empyema, and the effusion is usually sero-fibrinous and often blood-stained.

It should be pointed out that tuberculous pleurisy is sometimes followed by tuberculous pericarditis or peritonitis, or both. The two latter affections have been considered elsewhere (*vide* p. 279). We must grant that tuberculous pleurisy may proceed favorably with apparent recovery, though too often, after a variable interval of time, tuberculous symptoms are manifested. R. C. Cabot obtained the subsequent histories in 221 cases of pleural effusion in the Massachusetts Hospital; he followed them five years until phthisis or death took place in 117; at the end of five years 96 had recovered. It is found that about 30 per cent. become tuberculous.

(2) **Diaphragmatic Pleurisy.**—This term is applied to those instances in which the diaphragmatic portion of the pleura is involved, either alone or in part. There occurs an exudate that may be either plastic or sero-fibrinous, though rarely large in amount. The *symptoms* are acute, and the pain, which is lancinating in character and situated in the epigastric region, is the most prominent feature. Geuneau de Mussy[1] holds that pain along the tenth rib, extending from the anterior extremity to the sternum and xiphoid cartilage, is pathognomonic. It is increased by deep inspiration and by pressure over the insertion of the diaphragm at the tenth rib, and often abates when effusion takes place. Dyspnea is a marked symptom in most cases, and the patient may be forced to assume a stooping or sitting posture, the respirations being superficial, purely thoracic, and "catching." Cough, nausea, and even vomiting, may occur. In a case under my own care vomiting, due most probably to associated peritonitis, was a troublesome symptom.

The *constitutional features* are quite pronounced, particularly the fever, which exceeds that met with in other forms of pleurisy. The patient's anxiety is extreme. The effusion may be purulent, and if so bulging of the lower intercostal spaces, followed by edema, may occur.

The physical signs are for the most part negative.

(3) **Encysted Pleurisy.**—This term has reference to effusions that are circumscribed in consequence of adhesions between the pleural membranes. There may be two or more pouches, with or without communication. This so-called encapsulated pleurisy may occupy any part of the chest, and is exceedingly variable in extent. The symptoms and physical signs are rarely trustworthy for diagnosis, but should usually afford ground for suspicion, and lead to an exploratory puncture.

(4) **Interlobar Pleurisy.**—This variety is usually secondary to, or associated with, the ordinary type of acute sero-fibrinous pleurisy. The serous surfaces between the lobes are involved in the inflammatory process, and the fluid becomes encapsulated in this position in consequence of interlobar pleural adhesions. It is more frequent on the right than on the left side, and its favorite seat is near the root of the lung, between the upper and middle lobes. Osler[2] met with a case following pneumonia in which there was between the lower and upper and middle lobes of the right side an enormous purulent collection that looked at first like a large abscess of the lung. Fistulous connection with a bronchus often occurs, and the purulent expectoration that follows may be the first

[1] *Arch. gén de Méd.*, 1853, vol. xi., quoted by Fox. [2] *Practice of Medicine*, p. 567.

symptom to attract attention to the process of suppuration in the thorax. Prior to the occurrence of this accident the patient presents indefinite symptoms. The patient may or may not give a clear history of antecedent pleurisy. These cysts contain, as a rule, but a small amount of fluid, and cause little bulging of the intercostal spaces. Indeed in a case of my own at the Philadelphia Hospital there was actual retraction, though the aspirating needle showed the presence of effusion.[1]

(5) **Hemorrhagic Pleurisy.**—By this term is meant an admixture of

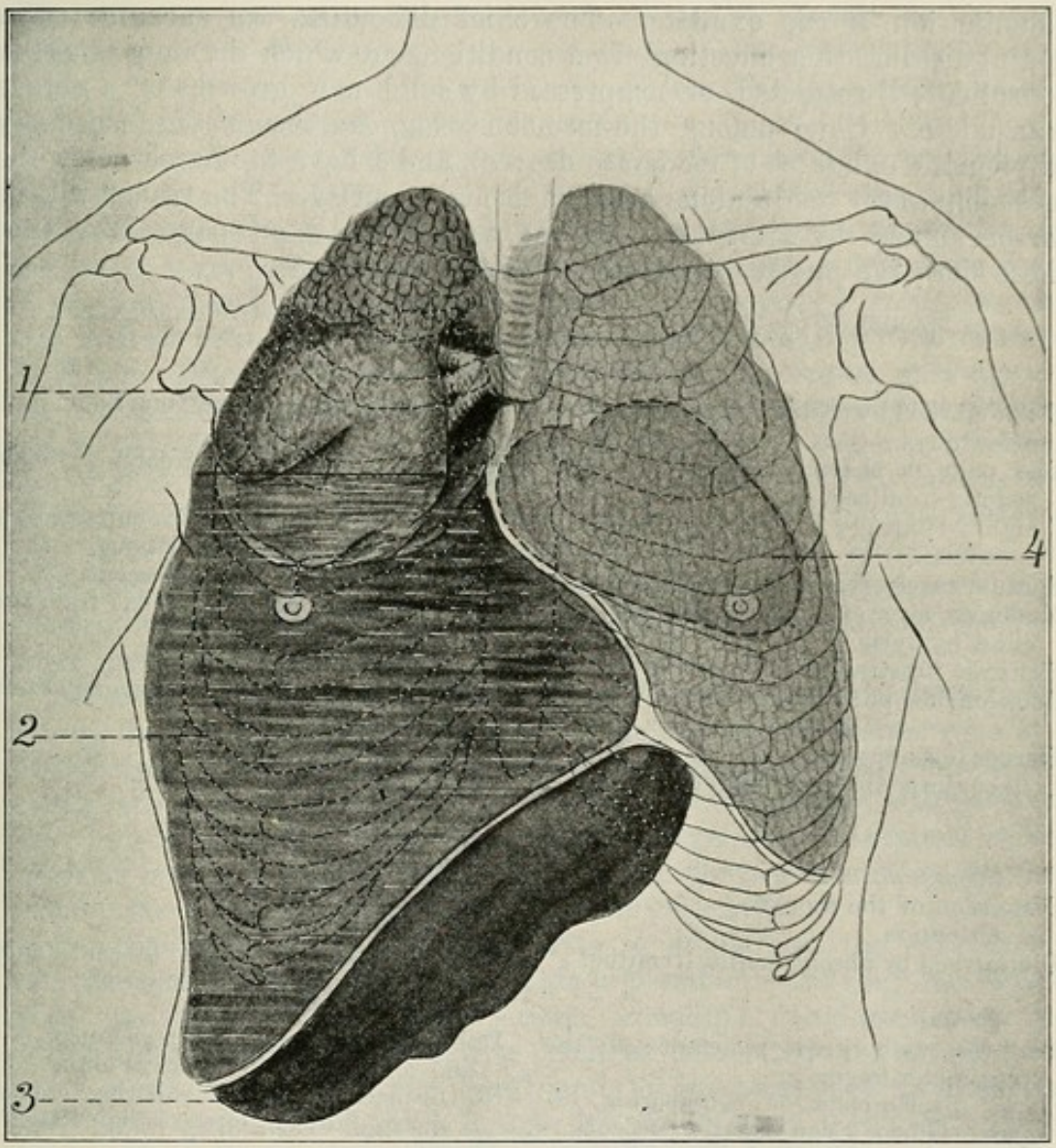


FIG. 48.—Illustrating pleurisy with effusion: 1, compressed lung-tissue, giving dull tympany on percussion; 2, fluid exudation obliterating intercostal spaces; 3, depressed liver; 4, displaced heart.

blood with the exudate in acute sero-fibrinous pleurisy, in quantities sufficient to be detectable by the unaided eye. The condition must be separated from *hemothorax*. The *causes* of hemorrhagic pleurisy are—(1) Tuberculous infection, either of the miliary or the chronic (circumscribed) form, following tuberculous disease of the lung; (2) Carcinoma of the pleura; (3) Bright's disease and cirrhosis of the liver; (4) Adynamic states

[1] *International Clinics* (1894), vol. i., p. 39.

of the system associated with malignant forms of acute infectious diseases (pneumonia). (5) Advanced age and alcoholism.

The fact that it may be engendered by an accidental wound of the lung during thoracentesis must be remembered.

Diagnosis.—In diagnosticating pleurisy our attention must be directed chiefly to the physcial signs. Unfortunately, the rational symptoms are often too slight to call attention to the chest. The diagnosis should embrace the particular etiologic variety (*e. g.*, tuberculous pleurisy, streptococcus, or pneumococcus pleurisy), by a bacteriologic examination of the exudate. The chief difficulties are encountered in distinguishing this affection from conditions in which the lung is either consolidated, retracted, or compressed by solid new growths or a serous transudate. Chief among the former is *croupous pneumonia* (especially in pleuritic exudates of moderate degree), and I have tabulated below the most important distinctions between it and pleurisy. The reader will be further aided by comparing Fig. 48 with Fig. 12, on page 122, since these show the physical conditions in the two diseases.

Pleurisy with Effusion.	Primary Lobar Pneumonia.
Rational Symptoms.	
Onset marked by chilliness, persisting for a few days.	*Onset* acute, rigor, lasting one hour.
The pain is sharp, "stitch-like," and strictly localized.	Acute pain (similar), but soreness more diffused.
Cough irritating; no expectoration, or catarrhal.	Cough more marked and accompanied by rusty or bloody expectoration.
Sputum rarely shows tubercle bacillus.	Shows presence of pneumococcus.
Moderate fever of continuous type; decline by lysis.	Intense fever; decline by crisis from the fifth to the ninth day.
Systemic prostration (moderate).	Prostration marked.
Countenance pale and anxious.	Countenance congested; mahogany flush on the cheeks.
Herpes does not appear.	Herpes quite common.
Leukocytosis absent or slight.	Leukocytosis usually marked.
Physical Signs.	
Inspection.	
Distention of the thorax.	None.
Palpation.	
Diminished or absent tactile fremitus.	Marked tactile fremitus (absent only when a bronchus is plugged).
Percussion.	
Flatness, with great resistance to the plexímeter-finger.	Dulness less wooden, less resistance, and sometimes a tympanitic note.
Shows displacement of neighboring organs. Grocco's sign usually present.	No displacement of neighboring organs, if uncomplicated. Grocco's sign absent.
If the sac be partly filled, line of flatness changes on varying the position.	Absent.
Auscultation.	
Diminished or absent breath-sounds, bronchial breathing frequent, but diffused and distant and unaccompanied by râles, as a rule.	Harsh bronchial breathing and presence of râles in first and third stages, unless a bronchus be plugged.
Vocal resonance diminished or absent; egophony.	Bronchophony (loud), unless a bronchus be blocked.
Friction-sound in early and late stages.	No friction-sound, except crepitant râles in the first stage.
Aspiration.	
Yields serum.	Yields a few drops of thick blood.

Consolidation of the lung, due to tuberculous infection, may be differentiated by means of the physical signs contrasted in the foregoing table, the history of the case, and by the discovery of the tubercle bacillus in the sputum.

Hydrothorax presents physical signs that simulate strongly those of pleural effusion. Hydrothorax, however, gives the history of cardiac or renal disease, is oftener bilateral, and is unassociated with a rise in temperature or with the pain or friction-sounds peculiar to pleurisy. In hydrothorax the withdrawn fluid has a specific gravity below 1015, while that of the pleural exudate is above 1017. Iodine or its salts, administered by the mouth, are recoverable in large quantities, and within a short time in the transudate; whereas in the exudate only a trace is found (Rosenbach and Pohl's test).

Tumors and *cysts* of the thorax will give complete dulness, will displace the heart, and compress the lung on the affected side, thus causing an absence of the respiratory murmur, etc. But the history of the case, the situation of the dulness (usually over the upper or middle parts of the lung), and the absence of uniform distention extending to the base, will serve to distinguish these affections from pleurisy with effusion.

Echinococcus cyst of the liver, or *abscess* of this organ, pushing upward, will cause retraction or even compression of the lung, and produce most of the physical signs of pleurisy with effusion. The former affections can be discriminated only by a correct appreciation of the history, by the presence not infrequently of a friction-sound on auscultation, of Litten's sign, and by an immovably fixed upper convex boundary of dulness. If doubt remains, an exploratory puncture should be made, and the fluid withdrawn should be subjected to a chemical, microscopic, and bacteriologic investigation.

An *enormous pericardial effusion* may be mistaken for a pleural effusion on the left side. In the former, however, there is commonly a history of rheumatism, and dyspnea is an urgent symptom, while the heart-sounds are greatly enfeebled; moreover, the heart is not displaced to the right as in pleural effusion. Again, flat tympany is obtained in the posterior portion of the axilla and good pulmonary resonance at the base in the postero-lateral region of the chest in pericarditis.

For practical purposes it is desirable to distinguish the *tuberculous* from the rarer forms of pleurisy. This is possible by paying due regard to the previous history of the patient, including hereditary taint, by noting certain clinical peculiarities (such as associated disease of other serous membranes and of the lung), and by the results of an examination of the exudate. A high proportion (65–95 per cent.) of lymphocytes in the cells found in the effusion is indicative of tuberculous origin. Leukocytosis is absent in tuberculous pleuritis. The tuberculin reaction may be elicited in cases presenting but little fever. Inoscopy—*i. e.*, the digestion and centrifugalization of the previously coagulated exudate, often shows tubercle bacilli. In a dubious case the guinea-pig should be inoculated with the exudate, and if the patient be tuberculous positive results may be confidently expected.

Duration and Prognosis.—This depends largely upon the cause. The course of acute sero-fibrinous pleurisy is not definite, but is made up of two parts—the febrile followed by the non-febrile stage. The fever lasts from one to three weeks; it corresponds to the period when the effusion

occurs, and the appearance of a non-febrile period indicates the subsidence of the inflammation. The exudate may be poured out rapidly, and may be absorbed not less rapidly; more commonly, however, the effusion takes place rather gradually, and the same is true of resolution. The continued absence of bacteria in the pus speaks for tuberculosis. In individual cases the prognosis depends chiefly upon the bacteriologic cause (the outlook being especially bad in streptococcic pleuritis) or the gravity of the basal disease. Simple serofibrinous pleurisy, including the hemorrhagic variety, unless it appears as a complication in the later stages of some other grave disease, has a comparatively favorable prognosis. Death rarely ensues suddenly without adequate lesions to explain its occurrence. Moreover, the appearance of empyema renders the prognosis far less hopeful. Again, the crippling influence upon the lung-tissue of previous attacks, owing to resulting adhesions, must be borne in mind, since chronic bronchitis, emphysema, fibroid induration, and phthisis often supervene. Contrast between the temperature and physical signs is an unfavorable sign.

Treatment.—In the first stage the treatment is the same as for dry or plastic pleurisy. During the second stage, that of effusion, the objects of treatment are threefold: (1) To limit the extent and intensity of the inflammatory process; (2) to accomplish the removal of the effusion; and (3) to support the strength of the patient.

(1) **To Limit the Extent and Intensity of the Inflammatory Process.**—To this end two classes of agents are employed—namely, (*a*) *Internal*, and (*b*) *External*.

Among the latter are counter-irritants, as sinapisms and iodin, by means of which constant counter-irritation is to be maintained. Another agent of great worth is cold, applied by means of the ice-bag or ice-water bag, and if the temperature rises to 102° F. (38.8° C.) cool spongings of the surface of the body, together with the use of the ice-cap, are useful. Roberts recommended keeping the affected structures at complete rest to relieve the pain by mechanical fixation of the side affected. For this purpose strips of adhesive plaster must be firmly and evenly applied to the chest; they should be removed during the stage of effusion.

The *internal* remedies embrace quinin, the salicylates, and opium. Opium and quinin are potent in controlling inflammation of serous membranes; the former being given preferably either in the form of suppositories or hypodermically, and the latter in divided doses, in capsule, followed by a few drops of mineral acid, administering gr. xvj to xx (1.036–1.296) daily.[1] I have observed good results from the salicylates (ʒj–ij—4.0–8.0, daily), which have been warmly advocated by Fiedler, Koester,[2] and others, as valuable in mitigating or even aborting the inflammation of the pleuræ, and thus in limiting the amount of effusion. It must not be forgotten that the effusion is due to an inflammation, and not to a simple transudation. The use of mild diaphoretics and diuretics, coupled with repeated small doses of salines, also aids in reducing the inflammation in the pleura. With a subsidence of the inflammatory process the temperature falls, and then our efforts should be directed toward the fulfilment of the second leading indication, (2) the **removal of the effusion.**

Little is to be accomplished by local means, though iodin, persistently employed, sometimes does good. The following ointment may also be tried:

[1] *Internat. Clin.* (1892), vol. i., 2d series. [2] *Ann. of Univ. Med. Sci.* (1893), vol. i. (A–31).

℞. Ung. ichthyol. (12 per cent.),
Ung. iodi comp., *āā.* ʒvj (24.0);
Ung. belladonnæ, q. s. ad ℥ij (64.0).—M.
Sig. Apply twice daily.

Blisters are not admissible.

Mild hydragogue cathartics, and especially the salines, after the Matthew Hay method (*i. e.* ʒij to ℥ss—8.0–16.0, in the smallest possible amount of water, on rising in the morning), stimulate absorption from the pleural cavities by draining the blood of a certain amount of serum. Unirritating diuretics may also be employed, but I have found no appreciable advantage from their use. Free diaphoresis (from the use of pilocarpin) sometimes assists in the absorption of the exudate, but it should not be employed in the presence of feeble heart-action or marked displacement of the organ. Among measures to promote absorption, the best, in my own experience, is the following combination:

℞. Potassii iodidi, ʒj (4.0);
Syr. ferri iodidi, ʒij (8.0);
Syr. sarsap. comp., ℥j (32.0);
Ess. pepsini, q. s. ad ℥ij (64.0).—M.
Sig. ʒj (4.0) every four hours, diluted; the dose to be doubled at the end of four days if well borne by the stomach.[1]

Diuretin is sometimes of service in causing absorption of rheumatic effusions.

The patient should be put upon a dry *diet* in order to increase the plasticity of the blood, which is thus induced to absorb the liquid exudate from the pleural cavity. The *modus operandi* of this treatment is different, but the effect aimed at is the same as when saline purgatives are given. Gilbert and Fede advocate autoserotherapy to stimulate absorption, the method consisting in removing 1 c.c. of the exudate and reinjecting it into the subcutaneous tissue. The exudation, however, defies all efforts at removal in about 33 per cent. of the cases, and in such the withdrawal of the liquid by aspiration (thoracentesis) must be practised. The indications for thoracentesis arise at two different periods in the course of pleurisy with effusion:

(1) During the febrile stage, in order to avert imminent danger to life, and not merely to remove the fluid. The conditions demanding immediate thoracentesis are—(*a*) when one pleural sac is completely filled or when Skoda's resonance extends from the clavicle downward no farther than the second interspace; (*b*) in double pleurisies, when both sides are half filled, since death may occur from rapid filling of one or the other side; (*c*) in cases of copious effusions, upon the first signs of involvement of the unaffected side, such as moist râles, broncho-vesicular breathing, and impaired resonance; (*d*) the appearance of serious symptoms, such as orthopnea or syncopal attacks with cyanosis; (*e*) marked displacement of the heart, especially if one or more murmurs develop in the organ.

(2) The indications for aspiration during the second or afebrile period, when the main object is to remove the exudate, are—(*a*) if no

[1] The author has employed this formula in more than 60 cases with very good results.

diminution in the quantity of liquid effusion has taken place one week after the temperature has reached the normal; (*b*) in subacute cases, in which there is little, if any, temperature from the beginning; aspiration should not then be withheld longer than two weeks.

The operation is free from danger if carried out under antiseptic precautions and if a modern aspirator is employed. The instrument should always be tested before it is used. The patient rests in bed in the semi-recumbent posture, the arm of the affected side being brought forward with the hand placed on the opposite shoulder, so as to separate the ribs from one another. The point of puncture is in the sixth interspace on the right side and the seventh interspace on the left, in the mid-axilla, or just below the outer angle of the scapula in the seventh right and eighth left interspaces, respectively. An assistant draws up the skin from the interspace, while the operator uses the forefinger of his free hand as a director. The needle should be introduced with a quick thrust, hugging the rib below the interspace, but endeavoring to avoid striking its periosteal covering. The fluid may not be obtained at the first operation, and the reasons for this failure are several. The costal pleura may be excessively thickened, or we may meet with a much-thickened fibrous band. Again, the fluid may be encapsulated; and, lastly, the needle may become blocked. Under these circumstances repeated trials should be made. Inaspirable effusion, or *blocked pleurisy* (Mosny and Stern), is ascribed to abnormal rigidity of the sac containing the fluid. Two needles may now be introduced, one of which is the means of injecting sterilized air.

The amount of fluid withdrawn at one time should never be large (℥xij to xxiv—384.0–768.0), though a relatively larger quantity may be taken during the febrile stage than during the afebrile, since in the latter instance the lung has been compressed for a longer period of time. The fluid is allowed to drain away slowly, a small needle being used, so as to invite the lung to expand in a gradual manner. If this precaution be not taken, the paretic pulmonary capillaries are apt to become the seat of sudden fresh congestion, followed by edema, and often by a speedily fatal termination. Thoracentesis is to be repeated at intervals of several days if nature does not take up the work of absorption, following the first operations. If during the operation incessant cough, dyspnea, a tendency to syncope, marked thoracic constriction, or sudden intense pain be developed, the needle must be withdrawn instantly.

Thoracentesis should not be resorted to in cases in which croupous pneumonia is associated, and never in very aged and excessively feeble persons. In tuberculous and cancerous pleurisy, Achard and others advise insufflation of unfiltered air as a harmless means of allowing a pleural effusion to be evacuated. Achard uses an ordinary bicycle or other vacuum pump for this purpose.

Holmgren[1] recommends blowing out, instead of aspirating, pleural effusions. Air is pumped in at an opening above to take the place of the effusion as it is forced out below by the pressure of the instreaming air. Davies[2] has used oxygen instead of air for the replacement of the fluid because of the fact that it is absorbed more quickly than the latter.

(3) **To Support the Strength of the Patient.**—The powers of the system are to be maintained by a nutritious diet, bodily rest, and other

[1] *Mitteilungen aus den Grenzgebieten der Med. und Chir.*, Jena, xxii., No. 2, p. 173.
[2] *Lancet*, London, December 28, 1912.

hygienic measures. The lighter forms of solid food may be allowed whenever they are found to agree, and it is important to promote the digestive power, if weak, by the administration of suitable remedies. During the stage of *convalescence*, therefore, tonics (strychnin, quinin, and arsenic) are to be administered. The dietary should be liberal, though composed of wholesome articles. Gentle exercise in the open air is to be encouraged, and massage of the muscles of the affected side tends to re-establish their usual vigor. To bring about the best possible chest-expansion nothing is so good as light gymnastic exercises, together with the methodical practice of deep inspirations for a minute or two at intervals of three or four hours. The management of the third stage, or that of convalescence, is similar to that of tuberculosis.

EMPYEMA (PURULENT PLEURITIS).

Definition.—A suppurative inflammation of the pleura.

Pathology.—On opening the pleural sac after death we may find a thick, creamy pus, though oftener it is seropurulent and separated into two layers—an upper, clear, greenish-yellow serous, and a lower, thick, purulent layer. In a smaller proportion of cases the exudate is fibrino-purulent. The odor emitted from the purulent collection is either sweetish or fetid (*e. g.*, when due to wounds), and, when the condition is associated with gangrene of the lung or pleura, horribly offensive. *Microscopically* the inflammatory products are identical with those of purulent inflammation in general. The pleural membranes are the seat of a more intense inflammation than in acute serofibrinous pleurisy, and are greatly thickened (1 to 2 mm.). They present a granular suppurating surface, and both visceral and costal pleuræ may exhibit perforations, and the latter, often erosions.

Histologically, the altered membranes consist of new connective tissue, new blood-vessels, and numerous leukocytes.

Etiology.—The following are the chief circumstances under which empyema arises: (1) As a sequel of the acute, sero-fibrinous variety. However clear the effusion may be, it always contains corpuscular elements, which in the further progress of certain cases undergo coincident increase in numbers until the effusion presents a milky aspect, when it is said to be purulent. Thoracentesis may be responsible for this change, though never if performed under rigid aseptic precautions.

(2) In children the effusion early becomes purulent in many instances.

(3) Secondary to the acute and chronic infectious diseases—*blood metastasis*—(pyemia, scarlatina, pneumonia, tuberculosis, and dysentery most frequently; typhoid fever, measles, whooping-cough rarely).

(4) Secondary to malignant affections of contiguous organs (lungs, esophagus), or tuberculous cavities which perforate the pleura. Rarely, carious ribs and vertebræ may cause empyema.

(5) Lymphatic metastasis is probably an important means by which bacteria reach the pleura from neighboring but not contiguous tissues (McFarland).

(6) Injuries to the chest may set up empyema (fracture of the ribs, stab or other penetrating wounds).

Bacteriologic investigation has shown that the organisms most frequently present are the micrococcus lanceolatus (*meta-pneumonia*), streptococcus, staphylococcus, and tubercle bacillus. The cases due to pneu-

mococci usually pursue a favorable course. The leptothrix pulmonalis is often found in putrid effusions.

Clinical History.—The symptoms vary with the cause. The *onset* may be characterized by acute symptoms (*e. g.*, Streptococcus Empyema), such as rigor, followed by high temperature and signal prostration, and in the affected side there may be severe pains, aggravated by deep breathing and bodily movements.

If the exudate becomes gangrenous, a *typhoid state* develops early, and the case is apt to prove fatal in the course of a few weeks. It is quite a common event for the acute symptoms that characterize the invasion to be replaced at the end of a week or more by the more obscure rational symptoms of chronic empyema. The latter, however, may develop very insidiously as a secondary affection. The rational symptoms in a well-marked case should always excite a suspicion of the presence of the affection, but cannot settle the diagnosis. The *local symptoms* (pain, cough, and expectoration) are of a mild character; dyspnea may be more or less intense. I have on more than one occasion found an utter absence of these symptoms. The *general symptoms* are those of septic infection—diurnal chills occurring at irregular intervals, followed by intense paroxysms of fever and profuse sweating—and such patients lose flesh and grow pale and weak. The temperature is higher than in pleurisy with effusion and is intermittently, though irregularly, elevated.

Peptonuria is a symptom of purulent pleurisy that is not without diagnostic value. It, however, also occurs in suppuration associated with the third stage of pulmonary tuberculosis, and in suppuration due to other causes. While not indicative of empyema, it nevertheless serves sometimes to eliminate sero-fibrinous pleurisy. The urine also contains *indican* in excess in the various suppurations, at least from time to time, if not constantly. *Blood examination* invariably shows leukocytosis, often of high degree.

If the pus is not removed artificially, it frequently breaks into the lung, penetrates it, and finally discharges through a bronchus. Pneumothorax now tends to supervene. Traube contends that necrosis of the pulmonary pleura may allow of the soaking of the pus through the spongy lung-tissue into the bronchi, without the establishment of a fistulous connection between the latter and the pleural sac, hence without the formation of pneumothorax. E. Smith's figures give 28 cases of empyema, of which 3, or 10.7 per cent., showed pleural vomicæ, while James, of Edinburgh, found them present in 44.18 per cent. In these, perforation into a bronchus has occurred with expectoration of pus. Lord states that it is a complication often overlooked. Besides rupture into the lung and external rupture, empyema may perforate neighboring organs (esophagus, pericardium, stomach, peritoneum). In rare instances the pus burrows along the spine behind the peritoneum and the psoas muscle, reaching, finally, the iliac fossa and simulating psoas or lumbar abscess.

Physical Signs.—These are, for the greater part, identical with those of pleurisy with effusion. Attention will therefore be called only to such as are more or less distinctive of the affection. Slight *edema* of the chest-wall over the seat of effusion, especially in children, is often present, and if the pleural sac be not aspirated, the abscess may point

externally and evacuate itself spontaneously. In the latter event a *protrusion* between the ribs shows itself: this may be the seat of fluctuation, and present an inflammatory appearance prior to its rupture, with subsequent discharge of its contents. The opening is usually found in the fifth interspace in front, and less frequently in the third and fourth interspaces or below the angle of the scapula behind. The upper level of the fluid does not change so readily on varying the posture of the patient, requiring a longer period of time than in serous effusion.

Baccelli's sign, or the transmission through a serous exudate of the whispered voice, is sometimes an aid in the discrimination of pleurisy with effusion from empyema. According to my own observation, though it is not invariably propagated by large serous exudations of the pleura, it is yet detectable in a large majority of instances, while I have never observed it in empyema.

Certain writers have recently emphasized the importance of recognizing small collections of pus in the pleural cavity, either as complications or sequelæ of pneumonia, scarlatina, typhoid fever, and other infections. Invasion is accompanied by a rigor only in cases in which the infecting organism is the streptococcus. The temperature is irregularly elevated or distinctly septic in character. The leukocyte curve rises promptly as a rule. Of local symptoms, circumscribed tenderness "elicited by pressure of the finger, and at first deeply seated, suggests both the fact of abscess and its location" (Musser). The physical signs of circumscribed effusion are to be sought along the fissures of the lobes and at the bases. A friction-rub is usually audible in the earlier stages.

Pulsating Pleurisy.—Pulsation synchronous with the cardiac beat in pleural effusion has received various designations (*pulsating empyema*, *empyema necessitatis*, *pulsating pleurisy*). The latter term is the most appropriate one, in view of the fact that it occurs not only in empyema necessitatis but also in empyema, which manifests no tendency to point externally and rarely in sero-fibrinous pleurisy.

Its *etiology* is imperfectly known. The principal causes, however, seem to be—(1) a copious effusion; (2) paresis of the intercostal muscles, inducing relaxation of the thoracic wall; (3) a somewhat forcible heart-beat (Henry). The rational *symptoms* of empyema are present. The *physical signs* are also identical with those of the latter affection, with the pulsation superadded. There are instances in which palpation alone detects the systolic pulse in the pleural effusion. With rare exceptions the effusion occupies the left pleural sac. The pulsation may be limited to two or three interspaces, or it may be visible over the entire antero-lateral aspect of the chest; pulsation at the back, however, is rare.

Differential Diagnosis.—An absolute distinction between empyema and *pleurisy with effusion* rests solely upon the results of an aseptic exploratory puncture. For this purpose the needle attached to the ordinary hypodermic syringe, or, preferably, the surgeon's exploring-needle, may be employed, withdrawing but a very small quantity of the fluid, which, if purulent in character, should be examined bacteriologically.

Pulsating pleural effusion simulates closely *aneurysm of the thoracic aorta*. When pulsation occurs in empyema, however, it is seen to be to the left of the normal course of the aorta: the rational symptoms and usual physical signs of purulent pleural effusion are usually present also,

while the vascular symptoms and signs of aneurysm of the aorta (thrill, bruit) are absent.

Prognosis.—Empyema is a serious disease, but, obviously, the outlook will be modified by the special etiology. Spontaneous absorption may occur, though it is extremely rare. Rupture into the bronchial tubes is a comparatively favorable event, some cases in which this occurs recovering, while in others death follows in consequence of the sudden inundation of the bronchi. An empyema may, in rarer cases, empty itself externally with favorable issue (*empyema necessitatis*). Evacuation of the pleural cavity is often followed by a continuous discharge of pus for an indefinite period. As a result of the long-continued suppurative process, death may take place by slow asthenia. It must not be forgotten, however, that an unfavorable termination may be, in part at least, ascribable to certain associated affections (phthisis, pericarditis). Double empyema, fortunately a rare condition, is exceedingly grave.

Among children the outlook is much more favorable than among adults. The prognosis has been rendered less serious by the application of surgical principles in the treatment of the disease. In all cases of recovery there is a progressive obliteration of the pleural cavity, owing to adhesions, which finally become universal and lead to marked retraction of the affected side (*pleuritis retrahens*).

Treatment.—The treatment of empyema is chiefly surgical. In a child, especially in empyema following pneumonia, recovery may follow one or more tappings. In the vast majority of cases, however, free drainage should be provided at the earliest possible moment. The pleural sac should be opened in the fifth or sixth interspace in the mid-axillary line, the incision being from 2 to 3 cm. in length, and if this affords good drainage, nothing more is needed. Resection of a rib (Estlander's operation) may be employed in long-standing cases or in those in which close approximation of the ribs prevents free drainage. Opinions are divided as regards the value of irrigation of the pleural cavity. When the pus emits an offensive odor irrigation with a disinfecting solution is imperative. Carbolic acid should, however, not be used. In rare instances accidents arise during irrigation (sudden collapse, convulsions), and I have observed a dangerous, and in one instance a fatal, collapse as the result of irrigation in a child. For further details in the operative treatment of empyema the reader is referred to text-books on surgery. Every effort should be made to favor obliteration of the cavity during post-operative treatment. The indication is to bring about the best possible degree of re-expansion of the compressed lung, and in order to accomplish this the method advised by Ralston James has been practised with great success in the surgical wards of the Johns Hopkins Hospital. The patient daily for a certain length of time, increasing gradually with the increase of his strength, transfers water by air-pressure from one bottle to another. The bottles should be large, holding at least a gallon each, and by an arrangement of tubes, as in the Wolff bottle, an expiratory effort of the patient forces the water from one bottle into the other. In this way expansion of the compressed lung is systematically practised. The abscess-cavity is gradually closed, partly by the falling in of the chest-wall and partly by the expansion of the lung.[1] In long-standing cases, in which the lung cannot

[1] Osler's *Text-book of Medicine*, p. 605.

expand on account of thick bands of adhesion, the pleural layers cannot be brought into juxtaposition without more or less sinking in of the chest-wall. De Lorme's operation (stripping the pseudo-membrane from the compressed lung) may be advisable. This retraction of the thorax is probably hastened by timely resection of one or more ribs, the amount of bone to be removed depending upon the "expansive power of the lung and elasticity of the thorax." The small collections (pleural or interlobar) described above demand prompt drainage.

The duration of empyema is longer than in pleurisy with effusion, and the former affection tends to exhaust to a greater degree the powers of the system than the latter; hence the physician's attention should be directed to the support of the vital forces by all possible agencies, modified to some extent by the special etiology of the case.

CHRONIC PLEURISY (ADHESIVE PLEURISY).

Definition.—Chronic inflammation of the pleural layers —(*a*) with effusion, and (*b*) without effusion.

(*a*) **Chronic Pleurisy with Effusion.**—This subvariety may follow acute sero-fibrinous pleurisy, and less frequently it has an insidious development. The morbid lesions, including the character of the exudate, may also be identical with those of the acute or subacute forms of the affection. Fibrin and serum are present in varying relative proportions, the latter, however, in nearly all of the cases preponderating when compared with the composition of the exudate in acute pleurisy. The secondary consequences of copious acute effusions also are met with—*i. e.* displacement of adjacent organs (liver, spleen, heart) and unilateral dilatation of the chest. When the fluid is either absorbed or removed and the case ends in recovery, marked contraction of the affected side results, since the lung, which is covered by thick, organized bands of adhesion, cannot re-expand. *Symptoms.*—But for slight dyspnea upon muscular exercise the subjective symptoms are frequently wanting. The pulse is compressible and accelerated, as a rule, and there is a trifling rise of temperature in the evening hours. If the effusion becomes purulent, hectic fever develops, leading to asthenia, and the latter condition eventually terminates life. Death may also be due to secondary suppurations (abscess of brain, etc.). In most cases occurring in children the effusion early changes to pus. The physical signs do not differ from those in acute sero-fibrinous pleurisy. The *duration* of the cases varies from three months to several years, or intercurrent pulmonary tuberculosis may shorten the course of the affection.

(*b*) **Chronic Dry or Adhesive Pleurisy.**—(1) This may succeed to the acute or chronic sero-fibrinous pleurisy. If the liquid portion of the exudate is absorbed, the pleural membranes come into more or less close apposition, being separated only by fibrinous elements that become organized into a layer of firm connective tissue. Hence the two layers of the pleura, that are greatly thickened, cannot be separated, owing to the firmness of the adhesions. In most cases the autopsy shows the latter condition to be most pronounced at the base, while the lung is found to be compressed and the seat of fibroid change. If it follows the acute form, the extent of retraction is slight, since there are no

dense fibrous bands to prevent a fair degree of lung-expansion; if it succeed the chronic form, however, or empyema, the extent of retraction and flattening will be quite marked. The exudate may undergo calcareous degeneration, and occasionally little pouches of fluid may be found between the false bands.

There is a large class of cases that are dry from the onset (*idiopathic dry chronic pleurisy*), and this variety may either be a sequel of acute plastic pleurisy or primarily tuberculous. The condition is very commonly met with at autopsy in subjects who during life had never presented symptoms of pleurisy with effusion. The plastic exudate, however slight, invariably tends to become organized, with resulting fibrinous adhesion of the two layers of the pleura. Most generally the adhesions are circumscribed, and if tuberculous in origin are most frequently apical and often bilateral. Under these circumstances small caseous masses and little tubercles may be found embodied in the somewhat thickened pleura. General synechia is, however, not rare, particularly unilateral.

Symptoms.—Definite rational symptoms are rarely present, and the physical signs lack uniformity or may be entirely negative. In other cases of a mild grade the main characteristics are restrained mobility of the affected side and feebleness of the respiratory murmur. In rarer cases the weakness of the breath-sounds is out of all proportion to the expansive motion of the chest. In still another category—composed of a considerable number of instances—certain physical signs are quite pronounced. Inspection reveals decided contraction, with immobility of the affected side and a compensatory distention of the healthy side. The heart is displaced, and the apex-beat may be missing (*e. g.* when the heart is drawn or pushed behind the sternum, or overlapped by the emphysematous lung). The spinal column is curved, the scapula dislocated, the shoulder ill-shapen and drooping, and the lower part of the thorax shrunken, while the ribs are obliquely placed and closely approximated, or even overlap one another. The tactile fremitus is decreased or absent over the lower portion of the chest, and there is impaired percussion-resonance or dulness over this area. The breath-sounds on auscultation are exceedingly feeble, and in some cases an occasional dry, leathery, or creaking friction-sound is audible.

Rarely, and particularly if the case be tuberculous, vasomotor symptoms arise in chronic pleurisy, such as unilateral flushing or sweating of the face, or dilatation of the pupil.

Doubtless some instances of chronic pleurisy merge into the pleurogenous type of cirrhosis of the lung, and fatal complicating conditions may arise in connection with the general circulation. Thus, I have observed in one instance enlargement followed by dilatation of the right ventricle, and in turn by general dropsy, with fatal result.

Treatment.—In the treatment of this affection two objects must receive especial attention: (1) the removal of any effusion that may be present; and (2) the improvement of the nutrition of the patient. The first indication is presented only by a limited number of the cases, and the rules for meeting it have been stated in the treatment of sero-fibrinous pleurisy and empyema; the second indication is presented by all cases. Careful regulation of the diet is of the utmost importance: it

must be generous, with modifications to suit special diatheses (as the gouty or tuberculous), if they be present. Lung-gymnastics are most useful if methodically pursued. The method of Ralston James (previously described) richly deserves a trial in suitable cases. It is to be borne in mind, however, that in old cases efforts at overcoming the lung-pressure will be unsuccessful. Climato-therapy is advantageous, particularly if a tendency toward tuberculosis exists; and in my own experience low, mountainous elevations combined with purity of atmosphere have given the best results. Of medicines little need be said. It is especially important to promote the digestive power of the patient to the greatest possible extent. In cases in which the digestive function has been feeble I have observed excellent results from a brief stay at any well-regulated seaside resort or in the country. We may also use, with a probability that the effect will be beneficial, small doses (℥j—4.0) of cod-liver oil, three times daily after food, or the following formula:

℞. Acidi hydrochlorici dil., ℥ijss (10.0);
Pepsini pur., ℥ij (8.0);
Tinct. nucis vom., ℥iss (6.0);
Glycerini, ℥iss (46.0);
Aquæ, q. s. ad ℥ij (64.0).—M.
Sig. ℥j (4.0), well diluted, ten minutes after each meal.

Intercurrent catarrh of the stomach may sooner or later become a troublesome feature, and in combating it lavage is frequently our most effective measure.

PNEUMOTHORAX.

(*Sero-pneumothorax; Pyo-pneumothorax.*)

Definition.—A collection of air in the pleural cavity. Since the latter, as a rule, contains at the same time serum or pus, the terms sero- and pyo-pneumothorax are frequently employed to describe the same condition. It is an uncommon condition.

Pathology.—When the pleural sac is punctured air usually escapes, accompanied sometimes by an audible hissing sound. The pleural sac in pure pneumothorax is greatly distended, and the lung is impacted against the spinal column. Other organs (spleen, heart) are also displaced, owing to positive intrathoracic pressure. The heart is not rotated, however, and the relation of its parts is maintained much as in the normal condition (Osler). The air may occupy but a portion of the pleural cavity, on account of previous firm adhesions (*circumscribed pneumothorax*). The point of perforation, as a rule, can be easily found, and frequently corresponds to the seat of rupture of the tuberculous cavity or superficial caseous mass. In other instances the cause of pneumothorax cannot be discovered. Inflation of the lung under water may reveal the aperture, which is usually small, by the escape of air-bubbles at the seat of puncture. Occasionally a fistulous connection between the pleural sac and the bronchi can be traced.

Simple pneumothorax is, however, of rare occurrence. The air that gains admission into the pleural sac is laden with micro-organisms (*vide* Bacteriology, p. 580), which set up various forms of inflammation, accompanied by equally various exudations. Hence the cavity is usually filled, in part, with an effusion that is purulent or sero-purulent, as a rule, and rarely serous or sero-fibrinous. The *gas* in cases of pneumothorax may be of bacterial origin; this contains substances not found in air, such as H, H_2S, or marsh-gas.

Etiology.—The *predisposing* influences are—(*a*) *age*—the condition occurring in adults, as a rule, though instances are also observed in young children; (*b*) *sex*—males suffer more than females; (*c*) the left side is affected nearly twice as often as the right; (*d*) *emphysema*, in which the superficial air-sacs are dilated and atrophied, and so rendered liable to rupture from excessive muscular exertion.

The **exciting** causes are—(1) *Perforation of the lung and pulmonary pleura* (the most frequent cause), arising in one or other of three ways: (*a*) From the rupture of a tuberculous cavity into the pleural cavity. This accident rarely occurs at the apex of the lung, but commonly near the upper border of the lower or middle lobe; less frequently near the lower border of the upper lobe. A caseous focus immediately beneath the pleura may also, during the process of softening, puncture the pleural sac and invite the entrance of air during the early stages. It cannot occur, however, except in cases in which previous adhesions have failed to form at the point of perforation. At least 70 per cent. of the cases of pneumothorax are tubercular (Morse).[1] (*b*) As the result of necrotic processes, in connection with certain other lung-affections, as gangrene, broncho-pneumonia, suppurating bronchial glands, abscess, and echinococcus cysts. (*c*) From rupture of the normal air-sacs in consequence of severe muscular effort (S. West, DeH. Hall). This accident is sometimes ascribable to the violent paroxysms of cough in pertussis.

(2) Some cases of *empyema*, by perforating the visceral pleura, the lungs, and bronchi.

(3) Perforations of the pleura in *malignant disease* and *abscess of the esophagus*.

(4) A *peripheral bronchiectasis* may open the pleural space.

(5) Pyo-pneumothorax may be of *subdiaphragmatic origin*, consecutive to *perforation by malignant disease* or *ulcer of the stomach* or *colon*.

(6) Pneumothorax may be occasioned by *gases* resulting from the action of a gas-forming bacterium on the pleural exudate.

(7) *Wounds* causing direct or indirect perforative lesions of the lungs. Fractures of the ribs may produce laceration of the visceral pleura, and allow the air to enter the pleural sac.

Symptoms.—The earliest symptoms vary according to the cause or causes that produce the condition. When it develops, as it does so often, in the course of pulmonary tuberculosis, the *onset* is sudden, marked by agonizing *pain* in the side, by intense *dyspnea*, and frequently *cyanosis*. The dyspnea is often accompanied by a sense of impending

[1] *American Journal of Medical Sciences*, May, 1900.

suffocation. The severity of the pain and the degree of oppression depend largely, however, upon the amount of air that gains entrance into the pleural sac or is formed from the exudate, the rapidity with which it enters, and the presence or absence of previous pleuritic adhesions. If the orifice be large and valvular, the air cannot escape, but rapidly accumulates and forces all the air out of the lung by compression; the patient then sinks rapidly into collapse from shock, and sudden death ensues. Fortunately, the *open* form is commoner, especially in non-

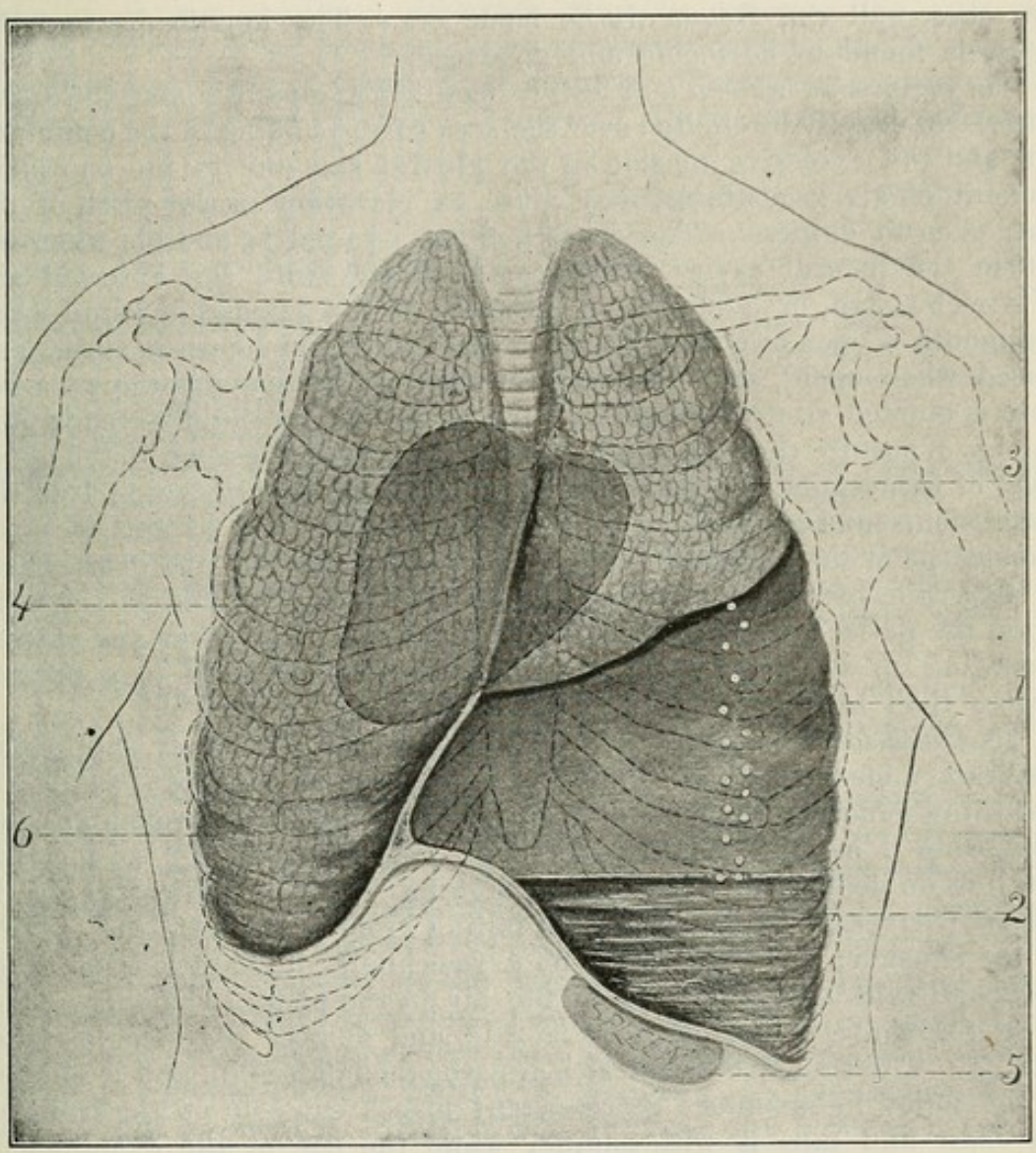


FIG. 49.—1. Air in the pleural sac; 2, fluid exudate at base of pleural sac; 3, compressed portion of lung; 4, displaced heart; 5, depressed spleen; 6, mediastinum pushed toward the right.

tuberculous pulmonary affections. The *respirations* are frequent; the *pulse* is also frequent and feeble, sometimes reduced to a thread; and cold sweats are not uncommon. The *temperature* at first is apt to fall one or two degrees below the normal, owing to sudden collapse; *fever*, however, follows almost invariably, and frequently is of the *hectic* type. Its cause is pleuritis, often purulent, and if this be the case, the dyspnea may be due in part to the increasing effusion. The patient now also suffers from the grave symptoms of empyema above described. Edema of the hand of the affected side is sometimes present

as an early manifestation; it rapidly disappears (Weil). When pneumothorax develops in the last stages of phthisis acute symptoms may be entirely absent.

Physical Signs.—These are marked (see Fig. 49), although rarely they may be in abeyance for several days (masked pneumothorax). *Inspection* shows marked distention and immobility of the affected side; also some degree of distention with unnatural mobility of the healthy side.

Palpation shows the tactile fremitus to be diminished above and greatly diminished or wholly absent over the effusion below. Edema of the chest-wall can frequently be made out. The impulse-beat of the heart is found to be feeble and displaced.

On *percussion* a deep and full, or modified tympanitic note (*bell-tympany*) can usually be elicited over the area corresponding to the contained air, and the excessive tension in the pleural sac, due to the enormous amount of air it contains, may cause an elevation in the pitch of the note even to dulness. The "cracked-pot" sound is audible when the air in the pleural cavity freely communicates with the external air. Wintrich's sign, or a change in the pitch of the percussion-sound when the mouth is open or closed (being lowered when the mouth is closed and raised when open), may also be observed. In pyo-pneumothorax a flat note is elicited from the base upward as far as the fluid extends, and change of posture causes a more marked temporary variation in the upper level of flatness than occurs in pleurisy. Modifications in the pitch of the percussion-sound result from an alteration in the form as well as in the dimensions of the air-space. Owing to displacement of the heart, there is, as a rule, resonance over the normal cardiac region, and particularly when the patient assumes a recumbent posture. The liver and spleen, according to the side affected, are displaced downward to a greater degree than in simple pleuritic exudates.

Auscultation discloses a greatly weakened or altogether suppressed respiratory murmur when collapse of the lung is incomplete. Amphoric breathing is audible in cases of *open* pneumothorax, and bronchial râles possessing a metallic quality are sometimes heard, as well as metallic tinkling on deep inspiration or on coughing. The metallic tinkling is caused frequently by drops of fluid falling from above upon the surface of the effusion; less frequently by a re-echoing of vibrations of moist bronchial râles communicated to the air in the pleural chamber. The vocal resonance is enfeebled, as a rule, and evinces the same metallic quality. The so-called coin-test is a pathognomonic sign, and is elicited in the following manner: An assistant places one coin on the front of the chest and taps it with another while the ear of the examiner is placed on the thorax posteriorly, where will be heard the intensified echo of the coin-sound thus produced. Another most characteristic sign is the so-called Hippocratic succussion, which is elicited by placing one ear upon the patient's chest while the latter's body is shaken, when a distinct splashing sound is heard.

Diagnosis.—When the attack is of ordinary severity, pneumothorax is diagnosticated by the history of one or other of the causal factors, together with certain physical signs that do not belong to any other affection (*coin-sound, succussion-splash*). The sputum test and also bacteriologic study of the aspirated purulent exudate with a view to deter-

mining the special etiologic variety present in a given case is of the greatest importance. It is only when the air and fluid in the pleural sac are encapsulated that it may become difficult to eliminate (*a*) a large pulmonary cavity; (*b*) excessive gaseous distention of the stomach; (*c*) an abscess below the diaphragm into which air has entered (*pyopneumothorax subphrenicus*); (*d*) a diaphragmatic hernia; (*e*) emphysema; and (*f*) pleurisy with effusion.

(*a*) *A Large Pulmonary Cavity.*—The "cracked-pot sound" and Wintrich's sign are more frequent in cavity than in pneumothorax, and the former condition does not tend to dislocate the adjacent organs. There is an absence of the succussion-splash, and, except in rare instances of the coin test, these signs are often present, even in circumscribed pyopneumothorax. Tabulated, these points of difference are—

Pyo-pneumothorax.	Large Pulmonary Cavity.
Immobility and bulging of the interspaces. The apex-beat is usually displaced.	Immobility, flattening of the chest, and depression of the interspaces. Apex-beat not displaced.
Diminished vocal fremitus.	Fremitus usually increased.
Percussion-note deep and full. The effusion sinks to the base, and yields flatness, the outline of which changes with the posture of the patient.	Percussion gives tympany or a "cracked-pot sound," and Wintrich's change of sound as a rule.
Respiratory murmur and vocal resonance usually absent. Amphoric breathing may be heard if the opening in the lung is patulous. The coin-sound and Hippocratic succussion-splash are noted.	Bronchial breathing is heard, and the vocal resonance is increased. Crackling, gurgling râles, cavernous or amphoric breathing, and pectoriloquy may be present. Absence of bell-tympany (generally) and succussion-splash.

(*b*) *Excessive gaseous distention of the stomach* is to be eliminated by the history of the case and by the results of the application of the therapeutic test, evacuation of the stomach and bowels.

(*c*) *Subphrenic Abscess Containing Air.*—This is exceedingly rare, and occurs relatively oftener on the right than on the left side (Leyden). Its leading causes are ulcers of the stomach or duodenum, followed by circumscribed peritonitis, perforation, and abscess, the latter occupying a position immediately beneath the diaphragm and above the liver. The gases that gain admission to the abscess-sac from the intestines force the diaphragm upward, and thus cause retraction or even compression of the lung. The symptoms are now identical with those of circumscribed pyo-pneumothorax, limited to the base. A knowledge of the steps in the production of subphrenic abscess; the absence of cough and expectoration, and of marked displacement of the heart; and the presence of bulging of the hypochondrium, of striking depression of the liver, and of Pfuhl's sign (*q. v.*), are indications favoring subphrenic abscess.

(*d*) *Diaphragmatic Hernia.*—This either results from a severe injury or is congenital, and the most valuable point of difference between hernia of the diaphragm and pneumothorax is the peculiar cause of the former. The next most valuable point is the fact that the hernial protrusion may return suddenly to its normal position, whereupon the patient will be relieved; the condition may then reappear not less suddenly. The third

distinctive feature is the presence of rumbling sounds in the protruded bowel. All other signs and symptoms of one affection may have their counterparts in those of the other.

(*e*) Pneumothorax may be confounded with *emphysema* by the careless observer; but the latter affection is slow in onset, free from serious shock, is bilateral as a rule, and does not exhibit the distinctive physical signs of pneumothorax (metallic tinkling, coin-sound, succussion-splash). In *pleurisy with effusion* hyper-resonance may be noted above the fluid, but it lacks the bell-like tympany of pneumothorax. Over the same area there is diffuse, distant, bronchial breathing (at times slightly amphoric), whilst the metallic tinkling, coin-sound, and succussion-splash are totally wanting.

Prognosis.—This depends largely upon the cause. The cases attributed to advanced phthisis usually reach a fatal issue in the course of one, two, or more weeks, and rarely they run a very rapid and fatal course. On the other hand, the pulmonary condition is at times favorably influenced by its occurrence. Following empyema, or when due to trauma or abscess of lung, pneumothorax sometimes takes a favorable course. It is fraught with especial danger when it is the resultant condition of some acute lung disease (gangrene, broncho-pneumonia). The prognosis is worse in right-sided pneumothorax.

Treatment.—The leading indication is the alleviation of the patient's sufferings by a prompt resort to morphin, and it often becomes necessary to administer it hypodermically. If the patient's previous strength has been moderately good, the question of operative intervention should be seriously considered, the nature of the surgical procedure then depending upon the character of the effusion. If this be serofibrinous, aspiration, as in simple pleurisy, must be performed to relieve the urgent dyspnea; if purulent, permanent drainage should be procured for the same indication. A costal resection may be advisable. When pneumothorax develops late in phthisis radical measures are not to be thought of, and the physician must rely upon aspiration (when necessary) to relieve urgent symptoms. We may also tap the air-chamber above the fluid with a fine needle, with a view to lessening the excessive tension. Unverricht has recently reported good results from a somewhat novel mode of treatment. When there is a pulmonary fistula present, he inserts a tube into the pleural sac. This allows free entrance of air, the lung collapses completely, and the fistula has a chance to heal. For the dyspnea, atropin administered hypodermically is valuable; for the feeble cardiac action, alcoholic stimulants, aromatic spirits of ammonia, strychnin, ether, and other cardiac stimulants should be employed. Locally, cutaneous irritants may be applied (turpentine stupes, mustard pastes).

HYDROTHORAX.

(*Dropsy of the Pleura; Thoracic Dropsy.*)

Definition.—A collection of transuded serum in the pleural cavity.

Pathology.—Hydrothorax is generally a bilateral condition. The transudate is a clear, amber-colored liquid that is free from fibrin, but may contain cholesterin and a few endothelial cells. It has an alkaline

reaction, a comparatively low specific gravity (1009 to 1012), and is non-inflammatory. The pleural surfaces are usually smooth, though sometimes decidedly pale and edematous. The mechanical effects of hydrothorax upon the lungs and other thoracic and abdominal viscera are similar to those of the exudates that accompany inflammation of the pleura, though they are rarely so marked as in sero-fibrinous pleurisy.

Etiology.—Hydrothorax is a secondary affection, and is usually connected with one or other of the various forms of general dropsy (hemic, renal, cardiac). The cases that are due to blood-impoverishment are more numerous than is generally indicated by writers upon the subject, and not infrequently is hydrothorax secondary to either chronic dysentery, chronic diarrhea, leukemia, pernicious anemia, carcinoma, malaria, syphilis, or scurvy. Strictly local causes may also induce it, as carcinoma of the pleura, or the compression of the superior vena cava or of the thoracic duct by a tumor. Fetterolf and Landis have demonstrated that the fluid comes from the visceral, and not the parietal, pleura, including the azygos veins.

Symptoms.—The *subjective symptoms* are attributed to the mechanical effects of the fluid, and may be quite in common with those of the causal affection; these are *dyspnea* (often culminating in orthopnea), *cyanosis*, *asthmatic seizures*, *irritative cough*, and a *feeble circulation*. The *general symptoms* arise from the primary affection.

Physical Signs.—The physical signs are much the same as in pleurisy with effusion—with this difference, that they are more often bilateral. Hydrothorax is often unilateral, however, and an enlarged right auricle may be the cause of this condition in some instances. The right side is the one usually affected. I have also observed that quite frequently the two sides of the chest exhibit great variations as to the relative amount of fluid contained.[1]

Prognosis.—This depends upon the nature of the primary disorder that causes the dropsical transudation.

Treatment.—The treatment of hydrothorax has intimate relations with the indications presented by the underlying affection. If the measures directed toward the removal of the general dropsy (*anasarca*), of which hydrothorax is a part, are unsuccessful, and the amount of transudation in the pleural sac interferes with the functions of the heart and lungs, then aspiration must not be too long delayed, and must be repeated as often as occasion demands.

NEW GROWTHS OF THE PLEURA.

Almost all instances of new growths developing in the pleura are secondary to primary carcinoma of the lung, the pleura being invaded by the direct extension of the neoplasm. It may also arise by metastasis from carcinoma of the lung, mammary glands, etc. The pleura presents circumscribed areas of thickening, or the growth takes the form of papular projections from its surface, becoming pedunculated as they enlarge. Their size varies from that of a pea to that of an orange. The adjacent pleura is inflamed, often adherent, and much thickened, and an effusion into the pleural cavity is often observed.

[1] For the differential diagnosis between pleurisy and hydrothorax, see Pleurisy, p. 593.

Primary carcinoma of the pleura is very rare indeed, and E. Wagner, who first described it, called it endothelial carcinoma. Most pathologists of to-day, however, look upon endothelioma as a variety of *sarcoma*. It owes its orgin to a proliferation of the endothelial cells of the connective tissue and the lymph-apparatus of the pleura. This invariably assumes the diffuse form, and by metastasis we have involvement of the other organs (lungs, lymphatics, liver).

Spindle-cell sarcoma of the pleura, as well as the round-cell variety, is occasionally met with.

Symptoms.—The subjective symptoms are slight in cases in which there is a single circumscribed carcinomatous mass in the pleura; but they are quite severe in the diffuse form, particularly when, as commonly occurs, it is of a secondary nature. The symptoms are now those of plastic or sero-fibrinous pleurisy, in addition to those of primary carcinoma of the lung, and the former may oftentimes more or less completely overshadow the latter.

Diagnosis.—The circumstances under which the condition arises often throw the strongest light upon its nature. The symptoms of slowly developing pleurisy, either plastic or sero-fibrinous, following carcinoma of the lung or the breast, and accompanied by the cancerous cachexia, would point strongly to the existence of *carcinoma of the pleura*. Characteristic cancerous elements may also be found by microscopic examination of the usually hemorrhagic fluid obtained on aspiration. The exudate contains fatty endothelial cells. Mitotic figures in cells of serous exudates are of confirmatory diagnostic value.

The difficulties surrounding the diagnosis of primary carcinoma of the pleura are great and usually insurmountable. The cases are very similar in their clinical manifestations to *chronic pleurisy with or without effusion*. *Pain* is always a more prominent symptom, however, than in simple chronic pleurisy, and this fact, when combined with evidences of a cancerous cachexia, should excite strong suspicions.

The prognosis is wholly unfavorable, and the treatment merely palliative.

DISEASES OF THE MEDIASTINUM.

The affections of the mediastinum may be divided into three classes: (*a*) Inflammation, (*b*) Tumors, and (*c*) Mediastinal hemorrhage.

(*a*) **Inflammation.**—This may affect (1) the glands or (2) the connective tissue. **Lymphadenitis** of moderate grade is found in association with broncho-pneumonia and the various forms of bronchitis. The condition appears in its most pronounced form in the bronchitis of measles, influenza, and whooping-cough, and De Mussy held that enlargement of the glands in the posterior mediastinum is potent in exciting paroxysms of whooping-cough. According to De Mussy and Guitéras, these glands when greatly enlarged give rise to dulness in the upper part of the interscapular region or down to the fourth dorsal vertebra in cases of influenza and whooping-cough. I have, moreover, been able to confirm this dictum in cases of influenza, though aware of the fact that

many authorities consider it questionable. Tuberculous lymphadenitis is elsewhere described (*vide* Tuberculosis, page 243). The mediastinal lymph-glands may undergo suppuration in consequence of local specific infection, and though not recognizable during life, the condition may lead to perforation into either the esophagus or a bronchus, with serious results. In other instances spontaneous absorption occurs, leaving behind inspissated contents that undergo calcareous change.

Abscess of the Mediastinum.—This is of rare occurrence, its most frequent seat being the anterior mediastinum. Of the commoner causes may be mentioned traumatism and the infectious diseases—erysipelas, rheumatism, measles, and small-pox in particular. It may also be the result of an extension of a suppurative process from neighboring structures. Pulmonary tuberculosis is the most potent factor in producing chronic abscess in this situation.

Symptoms.—*Acute Abscess.*—Pain and tenderness in the sternum are the most prominent features, the pain being acute and often of a throbbing character. Cough and dyspnea are usually present. The general features are fever, frequently accompanied by rigors, profuse sweats, and prostration. The chief physical sign is dulness upon *percussion*, usually found anteriorly and increasing gradually with the development of the abscess. Later, the tumor may reach the surface of the body, and rarely the sternum is eroded. *Palpation* now detects pulsation and fluctuation. The abscess may either find its way downward into the abdomen, or it may perforate the trachea or the esophagus.

In *chronic abscess* the symptoms bear a closer similarity to those of solid tumors than do those in the acute form. Fortunately, chronic abscess quite often results in spontaneous cure, in which case it is in part absorbed, and the remainder of its contents become inspissated.

Diagnosis.—*Acute* abscess must be differentiated from solid mediastinal tumors and aneurysm. The more acute onset and general symptoms of the suppurative process (hectic type of fever, chills, sweats) and the more rapid course will serve to distinguish abscess from *aneurysm* on the one hand, and *solid tumors* on the other. Further, the absence of strong expansile pulsation, diastolic shock, and the aneurysmal bruit aid materially in eliminating *aneurysm of the arch*. In obscure cases an exploratory puncture with a small needle may be safely practised, and, as a rule, with definite results.

The **treatment** is mainly surgical.

(*b*) **Tumors of the Mediastinum.**—Two forms only demand practical consideration—carcinoma and sarcoma. Hare's analysis of 520 cases gave the following ratio: of carcinoma, 134; sarcoma, 98; lymphoma, 21; fibroma, 7; dermoid cyst, 11; hydatid cyst, 8; and fewer cases of ecchondroma, lipoma, and gumma. In 48 of the cases of carcinoma and in 33 of sarcoma the tumor occupied only the anterior mediastinum. It is quite probable, however, that sarcoma, and not carcinoma, is the commoner neoplasm of this region. The clinical term "cancer" was formerly used promiscuously by many authors, and the pathologic diagnosis was then difficult, so that statistics are scarcely trustworthy. Upon investigating 25 of the older reports of "cancer," Pepper and Stengel found

in 13 unquestionable evidence that the growth was sarcoma, while in the remaining 12 they could not, for the greater part, decide to which form the disease belonged. Primary sarcoma may spring from the remnant of the thymus gland, from the lymphatic glands, the pleura, or lungs, or from the fibrous tissues of the mediastinum. Primary carcinoma may originate in the esophagus, bronchi, lungs, or rarely in the thymus gland. Secondary mediastinal tumors are most apt to have their seat in the lymphatic glands. Carcinoma is less frequently primary than sarcoma. Among *predisposing causes* are sex and age—males being more prone to the affection than females, and the period of chief liability is between the thirtieth and fortieth years.

Symptoms.—The earlier symptoms are vague (slight substernal pains, dyspnea, general languor). Later, pressure-symptoms gradually supervene.

The *pain* may or may not be severe, but is invariably accompanied by a feeling of oppression. Its chief seat is in the upper sternal region, but it may radiate to the sides of the chest and even down the arms (in which case it is due to pressure on the brachial plexus). *Dyspnea* appears early, is constant, and may become intense. It is caused by pressure either upon the trachea, upon a primary bronchus, or upon a recurrent laryngeal nerve. *Asthmatic seizures* may occur before there is constant dyspnea and before the tumor has reached notable size. There is *cough*, which may be paroxysmal and of a brazen character. Aphonia may be present. There may be *dysphagia* from pressure upon the esophagus, though this is rare. If there is an inflammation of the vagus or sympathetic nerve, the rate of the pulse may be either slowed or markedly quickened. Owing to implication of the sympathetic there may be local hyperemias and pupillary inequalities.

Compression of the superior vena cava or of the subclavian vein may be followed by cyanosis and edema of the parts drained by these vessels, and the early occurrence of venous occlusion and marked dilatation of the superficial veins is quite characteristic. Collateral circulation may be rarely established. Less frequently the inferior cava may also be compressed.

Physical Signs.—*Inspection.*—In advanced cases a swelling, usually somewhat irregular and often diffuse, appears in the sternal region. The tumor may cause erosion of the sternum, and a little later occupy a position immediately beneath the skin, Osler[1] being of the opinion that the rapidly-growing lymphoid tumors, more commonly than others, perforate the chest-wall. I saw a case in which the perforation occurred at the right edge of the sternum, precisely at the point at which aneurysms of the ascending arch most frequently appear. In the early stages, however, this prominence is not present. *Palpation.*—When a tumor is present it may pulsate distinctly, and the heart's apical impulse may be detected in various abnormal positions. Tactile fremitus is feeble or absent over the seat of the growth.

On *percussion* dulness is noted, and this is true even in many instances that do not present a visible swelling. The dull area varies in outline

[1] *Practice of Medicine*, p. 579.

with the size and position of the tumor. *Auscultation* usually reveals no sounds over the dull area, except a bruit in rare instances. The heart-sounds are inaudible over the tumor-site as a rule, and the breath-sounds and vocal resonance are feeble or absent. To the above physical signs are frequently added those of pleural effusion.

The **diagnosis** of mediastinal growths is made, if at all, principally by exclusion.

Aneurysm is differentiated from solid mediastinal tumors with only slight success in many instances. It is most valuable to note carefully the length of time the condition has lasted, since aneurysm runs a longer course, on the average, than mediastinal tumor. The tumor when due to aneurysm communicates a strong, heaving, expansile pulsation—a characteristic that is absent or only feebly manifested in the case of solid mediastinal growths. The severe diastolic shock, noted on both palpation and auscultation in cases of aneurysm, is also absent in solid tumor. Kassabian has shown that new growths can be early recognized by an *x*-ray examination. On the other hand, shadows situated in the anterior portion of the chest and to the right of the median line are generally produced by aneurysms.

The *duration* of the disease varies from six to eighteen months.

The **prognosis** is absolutely hopeless, except in the case of benign tumors, which may be removed in some instances.

The **treatment** is directed toward the relief of the most urgent symptoms. Anodynes are required sooner or later, and should not be withheld if indicated. As a routine the preparations of iodin and mercury are employed; but, as these are useless, they are unwarranted. Arsenic has sometimes seemed to influence sarcomatous and lymphadenomatous growths favorably, though only temporarily.

(*c*) **Mediastinal Hemorrhage.**—This term signifies hemorrhage into the mediastinal connective tissue. It oftenest results from the rupture of aneurysms of the arch or of the large vessels within the thorax. It may be of traumatic origin (wounds, fractures).

PART VI.

DISEASES OF THE CIRCULATORY SYSTEM.

I. DISEASES OF THE PERICARDIUM.

PERICARDITIS.

Definition.—An inflammation of the serous covering of the heart.

Varieties.—(*a*) Plastic, or fibrinous; (*b*) sero-fibrinous, or subacute; (*c*) purulent; (*d*) hemorrhagic; (*e*) adhesive. There is also a tuberculous pericarditis which has been described (*vide* Tuberculosis, page 279).

Bacteriology.—Rudini's experiments have shown that the staphylococcus aureus may be a cause of pericarditis; but they have not conclusively demonstrated that it is the specific cause, as is evidenced by the fact that the disease is sometimes caused by other organisms. Moreover, staphylococci have not been encountered without demonstrable cause. Among other organisms, the pneumococcus, streptococcus, the bacillus coli, the tubercle bacillus, and probably also a variety of the bacillus pyocyaneus and the gonococcus may be named. Microorganisms are not always found in pericarditic exudates.

ACUTE PLASTIC OR FIBRINOUS PERICARDITIS.

Pathology.—The morbid changes are frequently localized, and less frequently are general. At the onset the membrane is smooth, swollen, and injected, and punctate ecchymotic spots may be visible; soon it presents a grayish, roughened appearance from the deposit of a thin layer of fibrin. In the severer types the fibrinous deposit increases in thickness for a time, and the natural movements of the pericardial surfaces upon one another sometimes cause the exudate to assume a honeycombed appearance. Most examples that I have seen, however, have resembled the roughened surfaces produced by separating two slices of thickly-buttered bread; the surfaces are grayish-yellow in color. In the later stages the exudation becomes partly organized, and, as the result of friction produced between the opposed surfaces by the incessant action of the heart, may present a villous appearance; hence the term "hairy heart" of the ancient authors. For like reasons we may see the exudate arranged in the form of little ridges, forming a "tripe-like membrane."

Though invariably present, the amount of serous effusion, as the term would indicate, is never large in dry or plastic pericarditis. Myocarditis may frequently be found as an associated condition.

Etiology.—In each variety of pericarditis there are special contributing factors, so that it is desirable to give its etiology separately, except in the sero-fibrinous and acute plastic types, which have practically the same etiology. The two latter are the more common forms of the disease. Acute plastic pericarditis most frequently occurs in young and middle-aged males. It may be *primary* or *secondary*. It often occurs in acute articular rheumatism (in more than one-half the cases), chorea, lobar pneumonia, chronic nephritis, and, rarely, in other acute infectious diseases. In this form the infective agents are transmitted to the pericardium by means of the circulation. It may be caused also by direct extension of inflammation from adjacent structures (secondary pericarditis), as in simple pleurisy; more commonly the extension occurs from a pneumonia or tuberculous pleurisy, or the condition may complicate new growths and inflammatory conditions affecting the esophagus and bronchial glands. It may also be secondary to chronic disease of the aortic valve, the pericardium becoming involved by extension through the walls of the aorta. Finally, it may be the result of traumatism, and this may cause any of the other forms of pericarditis.

Clinical History.—Owing to the fact that acute plastic pericarditis is usually a secondary affection, the symptoms that enable one to recognize it are obscured by those of the primary disease. This is particularly true of that large class of cases that develop in acute articular rheumatism, in which subjective symptoms are often entirely wanting. Only in the severest types of this sort are the symptoms referable to the heart well enough marked to arrest the attention. There may be a feeling of *distress* or *constriction* with or without slight *pain* in the precordium. During the first stage or prior to the pouring out of the effusion the pain is most marked, extending sometimes into the left arm or the back, and at others to the ensiform cartilage or even to the abdomen. This pain is, rarely, increased by pressure over the precordia. *Palpitation* and *dyspnea* may be present, and the *pulse* is increased in frequency and strength, as a rule, except in the later period, when it may be weak and slightly irregular, particularly if the muscular tissue of the heart be involved. There is some *fever*, but the degree of elevation of temperature perhaps never exceeds 102° F. (38.8° C.). In this class of cases the *urinary features* depend largely upon the character of the leading etiologic factors; though in many instances the urine is scanty, high-colored, and acid in reaction.

Physical Signs.—*Inspection* discloses increased vigor of the apex-beat. Friction-fremitus (due to rubbing of the altered pericardial layers upon one another) may sometimes be felt during the earlier and later courses of the disease or when the membrane is comparatively dry, and is usually most intense near the base, just to the left of the sternum. *Percussion* gives negative results. *Auscultation* usually reveals a double friction-sound, sometimes quadruple (locomotive murmur)—the most important sign for a positive diagnosis. The friction-rub is caused partly by the exudate and partly by the dry state of the membrane. Its usual seat of maximum intensity is in the fourth and fifth interspaces and the

adjacent portions of the sternum—*i. e.*, where the pericardial surfaces can be but slightly separated from one another. Another favorite point is the cardio-aortic junction. It is usual to hear the rub over *small* areas, though occasionally it is audible over the whole precordia, and its distinguishing feature is its superficiality, seeming closer to the ear than endocardial murmurs. Pressure with the stethoscope, which approximates the layers, increases its intensity; though, if too much force be exerted, the murmur may disappear entirely. In like manner the friction-sound is influenced by respiration, losing in distinctness on deep inspiration. The quality of the sounds, like their position, exhibits great variability. They are sometimes soft; but quite commonly they are grating or rubbing, and in the later stages I have noticed that they may have a loud creaking quality. Though with few exceptions they are double, and are primarily produced by the rhythmic movements of the heart, they do not always occur synchronously with the heart-sounds, and usually exceed the latter in duration—facts that go to show that the quality, location, or superficial area of a given murmur does not indicate the extent of the lesion. When the exudate is soft and the heart's action weak, the characteristic murmur may be absent.

Complications.—There may be an extension of the inflammatory process to the external surface of the pericardium, either from the deeper pericardial structures or from the pleura, particularly the left. This is a complicating condition termed "*external pleural pericarditis*" or "*mediastino-pericarditis*," in which the mediastinal connective tissue is also, as a rule, involved. It is most frequently secondary to tuberculous pleurisy (*tuberculo-mediastino-pericarditis*), sometimes also to pleuro-pneumonia, and rarely to simple pleurisy or plastic pericarditis. The recognition of these combined lesions rests chiefly upon the detection of a friction-murmur that is partly dependent upon the cardiac and partly upon the respiratory movements. These sounds are most distinctly heard along the left edge of the heart. Momentary arrest of breathing suppresses the pleuritic friction-sound, there remaining merely the sounds produced by the rhythmic cardiac action, and even these may be absent. On the other hand, during forced respiration nothing is audible, as a rule, except the strong pleural rub. In normal respiration the inspiratory movements decrease while expiratory movements increase the intensity of the sounds. During inspiration the *pulse* may become small and slow, owing to the partial occlusion of the aorta, brought about by the traction of fibrous bands of adhesions which pass over the vessel, being at the same time connected with the pleura. When these bands pass from the exterior of the heart-muscle or pleura, they may cause, as first pointed out by Riegel, an absence of the apex-beat during expiration. Instances of this sort are not uncommon.

Diagnosis.—Although the presence of a to-and-fro friction-sound is, as a rule, indicative of plastic pericarditis, it is an error to regard it as an infallible sign, since complete calcification of the coronary arteries, as well as excessive dryness of the pericardial surfaces, may rarely produce friction-murmurs. The etiologic factors are important diagnostically.

Differential Diagnosis.—The harsh double murmurs due to chronic *valvular lesions* can be eliminated if it be recollected that they are more constant, more distant, and that each has an area of transmission beyond

the limits of the precordia. The sitting posture, leaning forward, or moderate pressure with the stethoscope, all fail to produce or to increase *endocardial murmurs*, whether acute or chronic. A double aortic murmur is associated with cardiac hypertrophy, the Corrigan pulse, and systolic flushing of the capillaries.

Prognosis.—The termination is always favorable as to life. Complete resolution does not often occur, but the exudate becomes connective tissue, and agglutinates the two layers of the pericardial sac. The acute may merge into the chronic form, and dry, plastic pericarditis often constitutes the first stage of sero-fibrinous and purulent pericarditis.

Treatment.—Absolute quiet in the recumbent position should be enjoined. The diet should be composed chiefly of light, easily digested solids, and allowing little drink, thus endeavoring to avoid an overfilling of the vessels. With the same object in view, if the patient's strength be good, a half-dozen leeches should be applied over the heart, followed by the use of the ice-bag; the bowels are to be kept soluble by using stewed fruits or saline laxatives. Calomel in doses ranging from gr. $\frac{1}{4}$ to $\frac{1}{2}$ (0.016–0.032) every hour or two, combined with a little opium to prevent purgation, is serviceable. At the beginning veratrum viride may also be cautiously administered, with a view to dilating the arterioles throughout the rest of the body, and thus virtually "bleeding the patient into his own vessels." The salicylates are indicated in cases of rheumatic origin. Later, the iodids of potassium and iron should be substituted for the purpose of absorbing the effused material. Tonics and a change of air may be required during convalescence.

SERO-FIBRINOUS PERICARDITIS.

Pathology.—The anatomic changes may be grouped into three stages—the *first* being characterized by a plastic exudation (corresponding with the lesions in dry, plastic pericarditis, though more pronounced); the *second stage*, by a variable amount of effusion composed largely of serum. The exudation usually begins about the origin of the great vessels at base of the heart, and ultimately forms a thick covering of fibrin, especially on the visceral layer. The quantity of serous effusion may be from 2 to 10 ounces (64.0–320.0), but occasionally it is as much as 3 pints ($1\frac{1}{2}$ liters). The admixture of a small number of red blood-corpuscles or leukocytes sometimes occurs in this form of the complaint. The *third* is the stage of absorption in the most favorable cases. Perfect resolution rarely takes place, but, instead, the liquid effusion is alone absorbed, and the lymph causes firm adhesions of the visceral and parietal membranes. If, as sometimes happens, the serum remains, the acute passes into a chronic condition. The myocardium may become involved by an extension of inflammation from the visceral layer; it is always the seat of more or less collateral edema. The grade of the myocardial inflammation will depend much upon the extent and duration of the pericarditis, though usually it is moderate in the fibrino-serous variety.

Etiology.—The disease is most frequently observed to be associated with acute rheumatism, Bright's disease, and pulmonary tuberculosis. Sears collected 100 cases of pericarditis, of which 51 were due to acute

rheumatism; and, according to Baumgarten, the former disease arises as a complication of the latter in about one-third of the cases. I believe that exceptionally both serofibrinous and plastic pericarditis may occur in the course of rheumatic dyscrasia without the slightest evidence of arthritis. The disease also occurs in the course of the eruptive fevers and lobar pneumonia, and from extension of inflammation from neighboring parts. Of 66 instances of pericarditis in children, 24 were caused by rheumatism. Next in frequency were tuberculosis and pleuro-pneumonia (Baginsky). (See also Bacteriology, p. 615).

Clinical History.—When, as rarely occurs, a *primary* pericarditis develops, the initial symptoms common to inflammation of other serous membranes manifest themselves, as *anorexia*, sometimes *nausea and vomiting*, *chills*, *fever*, *increased respiration* and *pulse-rate*, together with local *pain*. The pain is usually of a dull, aching character, and less frequently merely a slight soreness, or it may be absent altogether. Acute pain is experienced only when the pleura is implicated.

When pericarditis is *secondary* there are, in many cases, no subjective symptoms to indicate its presence. In other instances there may be *precordial oppression* with or without slight pain or a feeling of soreness. Hence in affections in which pericarditis is likely to arise physical examinations of the heart should be systematically conducted. Important symptoms are due to the intrapericardial pressure of the exudate.

Dyspnea comes on simultaneously with the appearance of the effusion and may lead to actual orthopnea. Pressure is exerted upon the left lung if the exudate be large—a fact that explains in part the presence of dyspnea. The cardiac muscle, especially the right ventricle, is also pressed upon by the effusion, thus impeding the cardiac diastole. Under these circumstances the veins fail to empty themselves into the heart, the arterial system is incompletely filled and the blood-pressure falls as the result. Prior to the occurrence of the effusion the circulation is too actively carried on, the *pulse* being full and strong. It is clear from the above explanation that during the second stage the pulse is small, feeble, and irregular. When the exudate is small, the heart-action may be apparently feeble, while the pulse remains strong—a valuable rational sign. On the other hand, an excessive amount of fluid may cause the radial pulse to disappear during inspiration (the *pulsus paradoxus*). *Fever* is present, as a rule; the temperature is irregularly elevated, ranging from 101° to 103° F. (38.3°–39.4° C.). In favorable cases defervescence takes place by lysis. *Nervous symptoms*, as headache and mild delirium, often appear, and sometimes give place to stupor or even coma. Acute mania is rarely observed. The urine is decreased in amount, and occasionally general dropsy occurs.

Physical Signs.—*Inspection.*—The skin-surface and mucous membranes are observed to be pale and more or less cyanotic. The neck-veins are prominent, and sometimes exhibit undulatory movements or pulsations. The expression is anxious; the respirations are increased, labored, and at times irregular. The decubitus is dorsal; the head and shoulders are elevated, and the patient may be forced to assume the sitting posture. In young subjects precordial prominence, with effacement or even bulging of the intercostal spaces, may result from the pres-

ence of a moderate effusion. In adults, however, a large collection is indispensable for the production of this effect. If the lung be shrunken or if there are pleuritic adhesions, expansion of the pericardium and, hence, also bulging will be prevented. The distended pericardium may depress the diaphragm. Elevation of the left nipple in consequence of marked anterior expansive bulging has been observed. In the first stage the apical beat is exaggerated, but as the exudate increases (forcing the heart backward and upward) it is displaced in an upward and outward direction, at the same time becoming weaker as well as more diffused, since with expansion of the sac comes greater mobility of the organ. When the pericardial sac becomes filled the impulse-beat disappears, the fluid now completely surrounding the heart.

Palpation confirms the results of inspection. The apical beat is diffused and feeble or lost. When detectable it is found to be displaced upward and to the left. Altering the patient's posture changes the seat of the apex-beat (Oppolzer), and if the shock has been lost, turning the patient on his left side or bending his body forward may cause its return. The cardiac impulse disappears earlier when, on account of myocarditis, the systole is greatly enfeebled. On the other hand, old adhesions and marked hypertrophy of the heart may retain the apex-beat in contact with the chest-wall, despite the presence of a large accumulation. A friction-rub can be felt occasionally over the base of the heart even in the stage of effusion, and, if absorption takes place, the friction fremitus becomes more marked. Fluctuation is rarely detected. In large effusions the liver is *depressed and easily palpable.*

Percussion.—The area of cardiac dulness is increased, and assumes a characteristic triangular outline with the base downward and the apex extending up to the third or even second interspace to the left of, though near, the sternum. The lateral border-lines of dulness obviously diverge from above downward, the right passing to a point corresponding with the right edge of the sternum, along which it runs to the hepatic flatness; the other to the left, finally intersecting the base-line, and extending to splenic flatness, or the lower limit of pulmonary resonance. Flatness may be met in the axillary region, even obliterating Traube's semilunar space. Rotch points out that even in moderate effusions there is flatness in the fifth interspace to the right of the sternum (*cardio-hepatic triangle* —Ebstein). Broadbent, however, has found several instances in which dulness in this area was present, but at necropsy, dilatation without effusion was found. The margins of the lungs surrounding the heart may be retracted and the heart carried forward or dilated; the dull space will then appear larger than is justified by the amount of fluid. Retraction or moderate compression of the lung may give rise to a modified tympanitic resonance to the left of the flat area. Occasionally the lung is attached anteriorly, and the heart is crowded backward by the effusion, while the area of flatness on percussion is relatively diminished. The triangular shape of the flat space, noted when the patient is in the sitting posture, is lost and its area diminished when he lies down, the effusion obeying the laws of gravitation. Sibson's notch, or narrowness of the dull area at the third costal cartilage in the transverse diameter, with reflection of the dulness to the left below this level, thus forming an

obtuse angle, obtains in medium-sized effusions. The feeble impulse can be at times felt within the dull area and not at its boundary.

Auscultation.—The characteristic friction-rub of the first stage has already been described. It may, however, also be audible over the base during the stage of effusion, and always returns, after absorption of the fluid, for a brief period. The heart-sounds grow more and more distant, faint, and muffled, though the second sound, as heard over the extreme base of the organ, may remain clear. Over the area of dull tympany corresponding to the lower antero-lateral portion of the left lung (which is more or less compressed) may be heard broncho-vesicular breathing.

Course and Duration.—It will appear obvious that the course must vary in individual cases with the cause and severity of the infection. Observation has shown that in one class of cases the three stages are passed through in rapid succession, while in another class each stage is proportionately lengthened. The latter form has been termed "chronic" by some and "subacute" by others. The *acute* may be followed by the *chronic* variety. Usually sero-fibrinous effusions complicating rheumatism are absorbed with rapidity once the process has begun, seldom requiring more than two weeks. When recovery is about to occur, the temperature falls by *lysis;* the dyspnea gradually disappears, and with it the effusion is gradually absorbed. Convalescence is further indicated by a return of the appteite, normal heat of the skin, and a more infrequent, full, and regular pulse. In cases that tend to a fatal termination either the fever continues or there is suddenly developed *hyperpyrexia*, as may happen when pericarditis occurs in the course of acute rheumatism; in such cases the dyspnea is urgent and cyanosis is often marked, with signs of failing circulation. *Nervous symptoms*, as extreme restlessness, insomnia, and active delirium, may be present. Under these circumstances death usually ensues at the end of a week or ten days. In a fatal case of *acute articular rheumatism* that I saw, complicated by pericarditis, with hyperpyrexia, death occurred on the sixth day.

Complications.—Copious effusion may, by causing pressure upon the recurrent laryngeal nerve, produce paralysis of the vocal apparatus, or, it may press upon the esophagus, causing dysphagia. Rarely *acute pleuritis* is a complication; it lengthens the course of the pericarditis and renders the outcome uncertain. When there coexists *extensive myocarditis* syncopal attacks often endanger the life of the patient. Associated endocarditis and a complicating pneumonia may be observed.

Prognosis.—In sero-fibrinous pericarditis recovery is the rule under favorable conditions. The outlook, however, becomes gloomy when the above-mentioned complications arise, and particularly when there is hyperpyrexia in connection with acute rheumatism. Occurring as a secondary event in serious acute diseases, as pneumonia, or in chronic diseases, as Bright's, or organic affections of the heart, the pericarditis often precipitates a fatal termination. The strong possibility that these cases may only partially recover or assume a chronic form must be recollected.

Diagnosis.—The disease is often overlooked, because unsuspected. Ordinarily the recognition of pericarditis by the characteristic triangular area of percussion-dulness and by the friction-sound is not difficult. The causative factors, and the symptoms dependent on the mechanical pressure of the exudate, are of considerable diagnostic importance. Atypical

cases or those first seen during the stage of effusion can only be correctly diagnosticated by exclusion.

Differential Diagnosis.—*Acute pleurisy* of the left side may simulate pericarditis with copious effusion, and, as before stated, these diseases may coexist. Acute pain, however, belongs to pleurisy alone. In pericarditis the characteristic physical signs are elicited over the precordia: in pleurisy they are apt to occupy not only the anterior but also the axillary and posterior aspects of the chest; hence the percussion-flatness in pleurisy extends to the left, far beyond the boundary-line of the percussion-flatness in pericarditis. The pericardial friction-sound has a different situation usually from the pleuritic, and the latter is heard synchronously with the respiratory movements, while the former is intimately related to the time of the cardiac movements. The friction-murmur of pleurisy ceases if the breathing be momentarily suspended. *Encapsulated pleural effusions* limited to the antero-lateral portion of the chest are exceedingly difficult of elimination, and especially in the absence of pleuritic friction. In the latter complaint, however, the heart-sounds are clear and the apex-beat often pushed to the right; on the other hand, in pericarditis the general disturbance is usually greater, while a friction-rub may be detectable over the base. The heart-sounds are distant and muffled. The diagnosis is often aided by the bearing of any facts in the previous history upon the known etiology of these affections. We encounter formidable difficulties in attempting to exclude *cardiac dilatation*, though the following brief table will render assistance:

Pericarditis with Effusion.	Cardiac Dilatation.
(*Clinical History.*)	
Recent history of gout, acute rheumatism, acute infectious or septic disease, scurvy, chronic nephritis, or tuberculosis.	Usual history of chronic valvular disease of the heart.
Fever and slight pain often associated.	No fever or pain, as a rule.
Nervous symptoms are often present.	Absent.
(*Physical Signs.*)	
Inspection often reveals bulging (more marked in the young). Apex-beat pushed up, is feeble, and later absent.	Apex-beat usually visible, wavy, and diffused.
Heart's impulse usually absent or occupies center of dull area. Friction-fremitus may be present over the base.	Though feeble, the impulse is palpable.
Percussion shows a triangular flat area, and the boundary-line above changes on altering the posture. There is dull tympany in the axillary region. Ebstein's angle obtuse.	Dull area varies with chambers dilated; it is coextensive with a wavy impulse, does not extend so high (except in mitral stenosis), and does not vary with change of position. No dull tympany.
Auscultation shows the first sound distant and muffled; a double friction-rub is often present over the base.	First sound clear, short, and sharp. No friction-murmur present, but an endocardial murmur may appear.
X-ray shows triangular, movable shadow.	Upper level of shadow (quadrangular) fixed.
Resistance gymnastics negative in their effects.	Resistance gymnastics decrease dull area (Schott).
Digitalis has slight influence.	Digitalis diminishes the field of dulness.

Treatment.—The management of the first (or dry) stage is identical with that detailed in discussing the plastic variety. During the stage of effusion the patient should be kept at absolute rest in the recumbent pos-

ture, and mental excitants should be prohibited with a view to minimizing the labor of the heart. The *diet* is to consist mainly of easily digested albuminous articles: fluids are not to be given in large amounts, since this tends to increase the arterial tension and delays absorption.

Local Measures.—Flannel should be kept over the precordia, so as to avoid exposure and undue chilling. The ice-bag or Leiter's coils (to be used in the first stage) should be cautiously employed during the second stage, until the temperature has defervesced considerably, thus indicating a subsidence of inflammation in the pericardium.[1] Subsequently, if absorption does not proceed, blisters may be applied over the precordia; but should the patient's general condition be bad, an absorbifacient containing iodine, lanolin, and ichthyol may be substituted with advantage.

The *therapeutic measures* must be chosen with sole reference to the primary disease, which the physician must continue to treat while he attempts by other means to relieve certain symptoms and promote absorption. For example, if the pericarditis be due to rheumatism, the use of the salicylates must be persevered in, and opium may be added to quiet restlessness and procure relief from pain. In my own experience absorption has been best promoted by the use of the double iodid of potassium and iron, or of iron and manganese. These agents are seldom contraindicated unless they are badly borne by the stomach. Diuretics and saline purgatives are not without value, but do good only in the later stages. Depressing measures of whatever sort are not to be resorted to unless the circulation be good. If the pulse be small, weak, and rapid, with marked cyanosis, stimulants are indicated and are to be given in moderate quantity. Strychnin and the salts of ammonium are useful. Digitalis and strophanthus are not to be thought of when myocarditis is associated; at other times they often improve the peripheral circulation and increase the urinary secretion. When the breathing becomes greatly embarrassed and the circulation fails, as shown by the feeble, broken, rapid pulse and cyanosis, cardiocentesis is indicated, and has, in recent years, given good results if not too long delayed. A preliminary puncture with a hypodermic needle should be made. In cases where the apex cannot be localized, the sixth space at about the mamillary line is the point of greatest advantage for paracentesis. "If it be definitely determined that the dilated heart extends beyond the mamillary line, one would then seek a point a little outside of the supposed position of the apex" (Thayer). In a case of extreme dilatation of the heart and marked excitement of the patient, Curschmann punctures through the eighth interspace from the rear. The operation must be performed with the strictest asepsis, and the amount of liquid withdrawn at any one time should not exceed 6 ounces. Of 60 cases of paracentesis for pericarditis of different varieties, collected by Roberts, 24 terminated in recovery.

PURULENT PERICARDITIS.

(*Empyema of the Pericardium.*)

Pathology and Etiology.—The condition may, rarely, follow the sero-fibrinous form. Septic and tuberculous processes involving the peri-

[1] If the pericarditis be secondary to an acute febrile disease, this fact must modify the method here recommended accordingly.

cardium are apt to cause purulent effusion from the start, and many of the cases that arise in the course of the acute infectious diseases belong to this category. The pneumococcus has been found in the pus (Shattuck and Porter). The membrane is much thickened and presents a gray, granular surface, and the myocardium underlying the visceral layer is softened, fragile, and pale looking (fatty).

Clinical History.—The local subjective symptoms and physical signs are the same in kind as in the former variety, but the amount of exudation is frequently less. At the onset *rigors* often occur, and may be repeated at varying intervals. The *temperature-curve* is of the suppurative type; the *pulse* is small, rapid, and irregular; and *physical prostration* is pronounced. Purulent pericarditis runs a comparatively rapid and an almost uniformly unfavorable course.

Diagnosis.—The chief clinical features are often referable to the primary or causal disease; hence in every instance in which purulent pericarditis is apt to arise a physical exploration of the chest is imperative. The purulent character of the effusion cannot readily be ascertained, as a rule; but the history of an affection having etiologic importance, the observance of rigors, a leukocytosis showing relative increase in the polynuclear forms, and the presence of the fever-curve peculiar to suppuration, would all point strongly to purulent effusion, and should lead to aspiration with the hypodermic needle—a harmless procedure if carefully performed, and one that almost constantly gives reliable results.

Treatment.—It is within the physician's province to treat the primary disease assiduously, but not pericardial empyema. Incision (after preliminary resection of a rib—Brentano) and drainage of the sac are advisable and feasible measures.

HEMORRHAGIC PERICARDITIS.

In purulent pericarditis the effusion may be hemorrhagic, and particularly when it is of tuberculous origin. In non-purulent tuberculous pericarditis also the exudation is apt to be hemorrhagic. In the non-purulent instances that are due to chronic Bright's disease or that occur in the aged the effusion is sometimes blood-stained; and future observation may show that this variety is of more frequent occurrence than has hitherto been supposed. In ordinary serous pericarditis there is apt to be present more blood than in serous pleuritis. M. T. Ferrier has found 5 examples in 9 collections. Sears found a pure growth of pneumococci in the exudate from a case of hemorrhagic pericarditis. This etiologic variety scarcely calls for separate clinical consideration.

ADHESIVE PERICARDITIS.

(*Chronic Pericarditis.*)

Pathology and Etiology.—Chronic pericarditis follows the acute forms, and, as in the case of the latter, it may be partial or general. The effusion may rarely remain as a permanent condition, but not infrequently a clear history of the preceding acute attack is wanting. In most instances the opposed surfaces of the membrane are either universally or over a limited area firmly adherent. The amount of new connective tissue present or the degree of thickening of the layers varies greatly, and is dependent upon the type of the primary acute attack.

If the latter is of mild grade—*e. g.*, the sero-fibrinous rheumatic form, not much thickening is encountered in the resulting chronic form.

Chronic tuberculous pericarditis is not uncommon, and is usually secondary. The disease may be chronic from the time of onset. The layers become enormously thickened, with obliteration of the sac.

In the dense exudate that remains after complete absorption of a pericardial effusion calcareous depositions occur, forming a bony casing, which either partially or totally encircles the organ. The external surface of the pericardium may become united with adjacent tissues (spinal column, anterior thoracic wall, aorta, sinus pleuræ). The myocardium is the seat of atrophic and degenerative changes.

Etiology.—The principal etiologic factors are tuberculosis and rheumatism. Pilt[1] analyzed 400 cases of acute pericarditis, of which 70 per cent. were due to rheumatism, about 50 or 60 per cent. died, and from 30 to 40 per cent. left the hospital with an adherent pericardium.

Symptoms.—Autopsies frequently discover an unsuspected adhesive pericarditis. Hypertrophic dilatation of the chambers usually develops sooner or later, and is due to adhesions that interfere with the free action of the organ as well as with its systole. When present the subjective symptoms point to a giving way of the right ventricle, as shown by the presence of *venous stasis* and *dropsy*. The *pulse* is rapid, of low tension, and irregular, and, though not diagnostic, the *pulsus paradoxus* is noted.

Pericarditis Callosa (Galvagni[1]).—A form of chronic fibrous pericarditis which comes on insidiously during childhood and is exceedingly difficult of diagnosis (*vide infra*). Pericarditis callosa is characterized principally by facial cyanosis, slight edema, full and tortuous jugular veins without pulsation. The typical physical signs of pericarditis are wanting also. On the other hand, a congestive cirrhosis of the liver may supervene and lead to ascites.

Physical Signs.—*Inspection.*—Depression or pitting of the intercostal space, in place of the apex-beat, may be noticed. Synchronous with the systole there is also a retraction of the chest-wall in the apical area, and less frequently over the whole precordia, the latter being an unerring sign of universal adhesions. The degree of systolic recession is slightly influenced by the respiration, inspiration increasing it, except adhesions exist between the pericardium and the adjoining pleura. It is best appreciated on palpation. When the apex-beat is not palpable, the systolic pitting over its site may be due to atmospheric pressure. During the diastole the heart forcibly rebounds, causing the so-called diastolic shock, which is of great diagnostic importance when associated with marked systolic retraction. Though not always visible, it can be readily felt on *palpation*. Friedreich's sign (the sudden collapse of the jugulars during diastole) may frequently be observed. Prior to the onset of dilatation the apex-beat may be forcible and visible over an increased area, indicating hypertrophy; but after the myocardium is weakened (from interference with its nutrition) and dilatation comes on, the impulse-beat is faint or wanting, and in marked systolic retraction may be vibratory. The fixed position of the apex-beat when the patient is turned over upon his left side is a strong confirmatory sign.

[1] *Practitioner* (London), Aug., 1912.

Percussion.—The area of cardiac dulness is increased, especially upward and to the left, owing to the associated hypertrophy and pleuropericardial adhesions, and, since the latter do not allow the lungs to overlap the heart during inspiration, the upper and left lines of dulness remain fixed (C. J. B. Williams). The most trustworthy symptom is the unchanging shape of the area of precordial dulness during inspiration and expiration (Davis).

Auscultation.—In many cases no murmurs are detectable. Loud murmurs, quite independent of any value as regards cardiac lesions, however, may be audible; these may be due to the vortiginous movements in the endocardial blood-current occasioned by the jogging cardiac action. The murmur of tricuspid regurgitation, from a breakdown of the right ventricle without apparent exciting cause, is most significant.

Differential Diagnosis.—The condition is apt to be confounded with *chronic myocarditis* and *simple hypertrophic dilatation.*

As before stated, chronic pericarditis may be associated with effusion, and it is important to distinguish such instances from the adhesive form, if we would institute a proper treatment. In chronic pericarditis with moderate effusion the seat of the apex-beat is higher and less undulatory, and when the amount of effusion is large the impulse is absent and there is bulging. Adhesive pericarditis with hypertrophy causes bulging in young subjects, but the apical beat is retained. There is no forward elevation of the chest during inspiration (Wenckebach). In pericarditis with effusion the upper and left limits of dulness are not stationary, and there is an absence of systolic retraction and diastolic concussion.

Course and Prognosis.—The hypertrophy that comes on early in consequence of the obstruction offered to cardiac action is compensatory, and this harmonious balance may be maintained for a long period of time with apparent comfort. After myocardial degeneration, followed by atrophy or dilatation, has occurred, the condition becomes quite serious, and death usually ensues amid signs of extreme cardiac dilatation.

The **treatment** must be ordered chiefly with reference to the nutrition of the heart-muscle, following the principles noted in dealing with the management of valvular affections of the heart. Precordial thoracectomy is advocated by Brauer and others.[1] If chronic effusion be present, early operative measures are to be warmly advocated.

HYDROPERICARDIUM.

(*Dropsy of the Pericardium.*)

Definition.—A condition in which the pericardium contains a serous transudation, but shows no signs of inflammation.

Etiology.—(*a*) Hydropericardium is usually associated with general cardiac or renal dropsy, of which it forms a component part. Under these circumstances it develops late, and frequently follows hydrothorax, on account of which condition it is liable to be overlooked. It may also occur suddenly in chronic nephritis, and particularly in the scarlatinal variety. (*b*) It may arise from local mechanical causes, as the pressure of mediastinal tumors, aneurysm, or thrombosis of the cardiac veins.

[1] *Semaine Médicale*, Sept. 7, 1910.

Symptoms.—No subjective symptoms are present, save perhaps dyspnea, and the diagnosis rests upon the history and the physical signs. None of the latter, however, are distinctive. They point to the presence of fluid in the pericardial sac, and the area of percussion-dulness assumes the same form and exhibits even greater change, with alteration of the patient's posture, than in pericarditis. No friction-murmurs are heard on auscultation and no bulging of the pericardium is observed. Again, there is neither a history of infectious disease nor inflammation of adjacent organs, as in pericarditis. It is rare indeed to see an excessive amount of serum in the pericardium at the *post-mortem* The symptoms and signs of hydrothorax generally precede and accompany hydropericardium, and the latter condition tends to intensify the effect of the former. Osler remarks: "Naturally there are in the pericardial sac a few cubic centimeters of clear, citron-colored fluid, which probably represents a post-mortem transudate." In rare instances the transudate has a milky appearance (*chylo-pericardium*).

The **treatment** suitable for cases of general dropsy, as a rule, affords relief. In large serous accumulations aspiration should be practised.

HEMOPERICARDIUM.

By the term "hemopericardium" is meant hemorrhage into the pericardial pouch—a rare event. Among the causes are—(*a*) perforation by aneurysms of the aorta and the coronary arteries into the sac; (*b*) rupture of the heart, due to injuries or cardiac aneurysms and fibrous formations from myocarditis; (*c*) direct injuries, especially stab- and bullet-wounds. *The symptoms and course* depend greatly upon the nature of the exciting cause. The most frequent factor, rupture of an aneurysm, proves quickly fatal from overcrowding of the heart. In rupture of the heart-muscle there is sometimes a slow outpouring of blood, with a correspondingly slow course, varying from a few hours to a couple of days in duration. The physical signs of pericardial effusion come on with dyspnea and failing circulation, which lead to cardiac exhaustion and death. The blood-stained effusions, occurring in certain forms of pericarditis (*vide supra*), are not to be regarded as instances of hemopericardium. Unconsciousness appears early, to be quickly relieved when the pressure is removed.

PNEUMOPERICARDIUM.

(*Air in the Pericardium.*)

In this complaint, besides air or gas, there is usually present pus, and less frequently blood; hence an appropriate term in most instances would be *pyo-pneumopericardium.* *The causes* are the following: (*a*) wounds; (*b*) a fistulous connection between the adjacent air-containing organs and the pericardium as the result of diseased processes, such as pulmonary tuberculosis or empyema; (*c*) rarely decomposition of liquid pericardial effusions, or the development of gas-producing bacteria. *The symptoms*

are equivocal. In the main they do not differ from those of pericarditis with effusion, excepting that dyspnea is more intense than in the latter affection. The physical signs, however, are different. In pneumopericardium there is tympanitic percussion-resonance over the precordia, though the fluid, when present, gives rise to a boundary-line of dulness. The change of the patient's posture decidedly alters the area of the tympanitic note. On auscultation may be heard loud, rasping, friction-sounds having a metallic quality, intermingled with churning, splashing noises, or the so-called "water-wheel sounds." *Pneumothorax* when encysted in close proximity to the heart, displacing the latter organ, must be eliminated. The latter complaint gives cardiac dulness in an abnormal position and a metallic auscultatory sound synchronous with the respiratory movements—two diagnostic points of pneumothorax that are absent in pneumopericardium. The *prognosis* is grave, death coming on most commonly in a day or two. The admission of air alone might result in a spontaneous cure, as occurs rarely in pneumothorax. The *treatment* is the same as has been recommended for purulent pericarditis.

II. DISEASES OF THE HEART.

ENDOCARDITIS.

Definition.—Inflammation of the lining membrane of the heart. The process is usually confined to the valves, though the cardiac layer may also be affected.

Varieties.—(*a*) Simple acute endocarditis; (*b*) ulcerative endocarditis; (*c*) chronic endocarditis. The pathologic processes involved in the first two, the acute forms, are identical in nature, though they differ in severity. I have met with two instances that apparently occupied a middle ground.

SIMPLE ACUTE ENDOCARDITIS.

(*Endocarditis Verrucosa.*)

Pathology.—The disease is characterized by the formation of small vegetations on the segments, varying in size from excrescences that are scarcely visible to those the size of a pea. They are found chiefly on surfaces that are opposed to the blood-current, near the margin of the valve, and "forming a row of bead-like outgrowths." Their seat corresponds to the point of maximum contact (Sibson), but the mitral valve is much more commonly affected than the aortic. With the segments the chordæ tendineæ are sometimes affected, and very rarely the latter are alone involved. The left side of the heart is much more frequently the seat of acute endocarditis than the right, except during fetal life, when the right side is almost exclusively involved. To account for the greater frequency of occurrence on the left side after birth, it has been suggested that freshly oxygenated blood affords the most favorable condition for the multiplication of the micro-organisms principally concerned in the

inflammatory process. Corroborating this view is the fact that during fetal life the blood in the right chamber is the more completely oxygenated. It has also been pointed out that before birth the right side, and after birth the left side, is the more active, and that the active side is apt to suffer on account of higher pressure. Obviously, the vegetations form an obstruction to the current of the circulation as it flows through the valvular opening. In the early stage the membrane in the vicinity of these excrescences shows a bright-red color, which has usually disappeared in fatal cases before they come to autopsy. The *histologic changes* consist in a proliferation of the subendothelial tissue (small-celled infiltration), which forms the principal component part of the vegetation. On this basal mass of granulation tissue there is deposited fibrin from the blood, the latter being separable from the former in acute forms of the complaint. Micro-organisms have repeatedly been found in the fibrinous depositions, but the specific causal irritant has not as yet been discovered. In favorable cases either the vegetation is ultimately absorbed or there remains a small indurated mass. When the vegetations are of considerable size emboli may become detached by the force of the blood-current, and be carried to the vessels of the extremities and to the various viscera, particularly the brain, spleen, and kidneys, giving rise to embolic infarcts. The latter event is frequently observed in cases in which acute endocarditis is engrafted upon chronic valvulitis.

Simple acute endocarditis may end in the more serious or ulcerative variety (*vide infra*). More commonly, however, does the simple form terminate in chronic valvulitis with deformity.

Etiology.—The most frequent cause of acute endocarditis is *acute articular rheumatism*, which induces the disease in not less than 40 per cent. of the cases. In young rheumatic subjects the liability to the complaint is particularly pronounced. The severity or mildness of the rheumatic attack does not, however, influence the appearance of the cardiac complication. Cases of acute endocarditis of rheumatic origin are met with in which the arthritic phenomena are secondary. It may complicate *tonsillitis* when the latter is due to or associated with rheumatism. In *specific fevers* it is also encountered, and is common in scarlet fever, but rare in typhoid fever, diphtheria, measles, erysipelas, variola, and varicella. It is not uncommon as a complication in *pneumonia*. Osler, as the result of 100 autopsies in cases of pneumonia, found it present in 5 instances. *Tuberculosis* is not infrequently the basal disease. Of 11,000 records of autopsies in cases of tuberculosis, 151 instances of endocarditis were found (G. W. Norris). It has frequently developed in the more serious forms of *chorea*, and intercurrent acute endocarditis may result from chronic diseases attended with emaciation and general weakness or suppuration, such as ulcerative carcinoma, gleet, gout, chronic Bright's disease, and diabetes. Lastly, acute endocarditis may occur as a secondary event in pre-existing sclerotic endocarditis, when it is termed *acute recurrent endocarditis*. In chronic endocarditis the liability to the acute form is greatly increased by the puerperal state, and, to a lesser extent, by pregnancy.

Bacteriology.—All cases of acute endocarditis are microörganismal in character. The disease, however, is the result of various microörgan-

isms or their toxins, whose action is assisted by the friction between the blood current and the surfaces of the valves. Fränkel and Sänger affirm that the staphylococcus pyogenes aureus is the chief specific organism. The diplococcus pneumoniæ, the streptococcus pyogenes, and, less commonly, the Bacillus coli communis, the gonococcus, the Bacillus diphtheriæ, the bacillus of Eberth, that of Pfeiffer, and the micrococcus endocarditis capsulatus and rugatus are found.

Clinical History.—It is only occasionally that definite **subjective symptoms**, as precordial *pain* (sometimes extending down the left arm), *dyspnea*, and *cardiac palpitation*, are complained of by the patient. If fever have been present, as is common, the temperature usually rises rather abruptly. In the vast majority of instances the condition is discovered accidentally. This being true, its frequent occurrence in acute articular rheumatism, and its occurrence in the other diseases mentioned under "Etiology," should be kept in remembrance. The symptoms of *embolism* are rarely observed. F. Billings reports a case with multiple emboli.

The **physical signs** by which acute endocarditis is recognizable are dependent upon the valvular insufficiencies caused by the morbid lesions previously described. In some cases, including those in which the valves are not affected, distinct physical signs are absent.

On *inspection* the area of visible impulse may be seen to be increased, to the left in most cases. The impulse is sometimes forcible and often irregular during the initial period, but later it becomes less distinct and more feeble. *Palpation* confirms the result of inspection. I have found the impulse to vary at each visit, with a general tendency to lessen in intensity in the later period of the disease. A very weak impulse is indicative of associated myocarditis or of the poisonous effect of a severe type of primary infection. In recurrent endocarditis the apical impulse is often heaving, on account of pre-existing compensatory hypertrophy, and its area is exceedingly variable. A systolic thrill is sometimes felt.

On *percussion* the cardiac area of dulness is found to be either normal, or, more commonly, enlarged in the transverse diameter, especially to the left; this results from the increased diastolic tension in the left ventricle. While the right ventricle meets with greater resistance, it rarely dilates, owing to its power of accommodation during the course of acute endocarditis. In recurrent acute endocarditis the area of dulness corresponds to the increased area of the apical beat.

Auscultation.—Acute endocarditis is usually attended with a soft blowing, systolic murmur, which, since the mitral segments are the favored seat of the disease, is heard much more frequently at the apex than at the base. The point of maximum intensity of this murmur is often movable, but its area of transmission is limited. In rheumatic endocarditis this murmur is preceded by a prolongation of the first sound. It is associated with accentuation of the second pulmonic sound. The murmur is sometimes heralded by a dull first sound and delayed radial pulse, with apparent intensification of the second, suggesting ventricular dilatation as the cause of the murmur. The characteristic presystolic murmur, indicating mitral stenosis, may be, in exceptional cases, associated. In acute endocarditis affecting the mitral valves aortic murmurs may coexist, but their true nature is more than doubtful. There is also a short, low-toned, and double systolic murmur over the tricuspid orifice

in a small proportion of the cases; this is due most probably to a relative incompetency. When acute endocarditis arises in connection with chronic valvular disease, the auscultatory signs of the latter are but little changed, and hence an assured diagnosis is not possible.

Complications.—There may be developed by direct extension secondary myocarditis (*vide* p. 690) and pericarditis.

The **diagnosis** is based principally on the physical signs, though these are by no means trustworthy. The points gained by careful inspection and palpation are of especial diagnostic importance, as is also the previous history of the patient. Leube[1] points out that if the dulness is slightly increased to the left and there is fever—in fact, if there is infectious disease present—a diagnosis must be made of acute insufficiency of the ostium mitralis occurring in the course of acute endocarditis. Rosenau states that blood-cultures should be made for the identification and study of the infecting organism as well as for prognostic reasons.

Differential Diagnosis.—The soft bellows murmur is often present in *acute febrile diseases* in which the autopsy fails to reveal the lesions of acute endocarditis. The functional murmurs that arise in the specific fevers, however, are principally heard over the aortic and pulmonary areas, while those occurring in endocarditis are commonly heard over the mitral area. The murmurs present must be called *accidental* (functional) if the area of cardiac dulness is normal, the second pulmonary sound not accentuated, and if the murmur be heard only at the pulmonary cartilage, or at this point and at the apex, and, at any rate, more distinctly at the pulmonary cartilage (Leube[2]). The distinction between simple acute endocarditis and *pericarditis* should be categorical, in view of the manifold differences between their signs. But the fact that these two affections may be associated, more especially when they are of rheumatic origin, must be steadily borne in mind, and also that when combined the pericardial friction-sound and the later effusion obscure the signs belonging to the endocarditis. I have found, however, that, fortunately, endocarditis precedes pericarditis in the majority of the cases. The elimination of *old endocarditis* or *chronic valvular disease*—a matter of importance—may be accomplished by attention to the character of the murmur in acute endocarditis, as well as to its limited area of diffusion, and by the absence of the signs of hypertrophy and of marked accentuation of the second pulmonary sound.

A *relative insufficiency* distinguishes itself by a pure systolic murmur, loud and not invariably uniform, by a weak cardiac impulse, a slight accentuation of the second pulmonary sound, and a comparatively small and often irregular pulse. It is met with in excessive dilatation of the left ventricle, in anemia, "and particularly in certain changes of the valvular muscles, due to myocarditis" (Leube).

Prognosis.—The immediate dangers are few, and depend largely upon the primary disease. In many instances, however, acute endocarditis initiates permanent lesions of the valves.

Treatment.—Prophylaxis.—The prevention of acute endocarditis in rheumatism has been dealt with in discussing the latter disease. No known direct measures can prevent the development of this condition in the course

[1] *Deutsch. Archiv f. klin. Med.*, Nov. 5, 1896. [2] *Loc. cit.*

of the specific fevers, though absolute rest in bed and protection of the body against "cold" may diminish somewhat the tendency to it.

The Attack.—The sick-room should be free from draughts, though well ventilated, and flannel is to be applied to the chest. The diet may be liberal, but should be composed chiefly of milk and other light nutritious substances. Stimulants are required in most instances, and in abundance should the heart be failing. Digitalis is to be employed cautiously if at all. When the myocardium is involved, its use is not without danger; under these circumstances the drug increases the sufferings of the patient by throwing the inflamed and weakened cardiac muscle into firm contractions. The salts of ammonium, particularly the carbonate, should be given continuously with a view to obviating intracardial coagulation of blood; and should the latter accident occur despite all efforts to prevent it, the carbonate, together with strychnin and alcoholic stimulants, should be freely administered. I am convinced that in endocarditis due to acute articular rheumatism it is wise to continue the exhibition of the salicylates, though in moderate doses, provided that the heart is guarded by the use of stimulants. During convalescence from an acute endocarditis the patient should be kept at rest, so as to minimize the strain upon the affected valves; even after he has apparently recovered, and particularly should the murmur still be present, perfect quiet is to be enjoined for a period of several weeks.

ULCERATIVE ENDOCARDITIS.
(*Malignant or Infectious Endocarditis.*)

Malignant endocarditis is variously characterized, though usually either by perforative ulceration, by suppuration of the valves, or by both, giving rise to the physical signs of acute endocarditis. These develop amid the symptoms of a severe primary infectious or septic disease. There is at hand enough clinical evidence to warrant the assumption that ulcerative endocarditis also occurs, though rarely, as a primary affection.

Pathology.—(*a*) *Valvular Endocarditis.*—In its early development the valves are the seat of vegetations (such as are met with in simple acute endocarditis) which later undergo necrosis. The latter process tends to spread, destroying more or less of the endocardium. In the interior of the vegetations suppuration not infrequently takes place, and the abscesses thus formed rupture and produce various lesions according to their size and situation. The vegetations take on a grayish- or yellowish-green appearance. Histologically, they are composed of granulation tissue, veiled by granular and fibrillated fibrin, containing numerous microorganisms. At the base there is usually developed more or less reactionary inflammation. After rupture the blood-current may enter the abscess-cavity, and, if there be no complete perforation, the endocardium will be pouched out, and an aneurysmal dilatation of the valve will result. Ulcerative lesions are most frequently observed. They may be mere erosions of the endocardium, but, as a rule, are penetrating and often result in complete perforation. I have seen repeated instances in which the three classes of lesions above depicted were all present. Osler, in an analysis of 209 cases examined by him with a view to ascertaining approximately the relative frequency with which the different parts of the heart were

affected, obtained this result: Aortic and mitral valves together, 41; aortic valves alone, 53; mitral valves alone, 77; tricuspid in 19, pulmonary valves in 15, and the heart-wall in 33 instances. In 9 instances the right heart alone was involved.[1]

(*b*) *Malignant mural endocarditis* gives the same set of changes as the valvular form; indeed, the two may be combined throughout. It is a comparatively rare condition, as is shown by the foregoing figures of Osler. The ulcerative process may invade the chordæ tendinæ and the valves, and may perforate the septum or even the ventricular wall itself. The vegetations are detached in small or large masses, and are conveyed by the blood to various organs, especially to the spleen and kidneys, less frequently the intestines, meninges of the brain, and the skin. Their site is determined largely by their size, and they may be so large as to plug vessels of the caliber of the external iliac. When found in the lungs they may originate in endocarditis affecting the right heart. These emboli, containing, as they do, the agents of inflammation, form suppurative infarcts that may be either white or red in color. The detached vegetations are sometimes so laden with irritants as to cause rapid softening of the coats of the vessel at the point where they become arrested, with consequent aneurysmal dilatation directly opposite their seat. The number of infarcts varies greatly in different cases; thus there may be only one or two, as in a case in my own knowledge in which the spleen alone contained two small infarcts, or there may be more than a thousand minute abscesses widely scattered throughout the body.

Etiology.—It is to be kept in remembrance that the condition is, with few exceptions, most probably a secondary one. This explains why the lesions peculiar to simple acute endocarditis usually precede and accompany those of the ulcerative form.

Bacteriology.—The specific irritant is usually the *streptococcus pyogenes* (Fränkel and Sänger); hence the diseases in which ulcerative endocarditis occurs as a complication merely furnish the opportunity for the invasion of the streptococcus. The bacillus diphtheriæ, however, as well as the staphylococcus, the bacillus coli, the bacillus anthracis, the pneumococcus, the gonococcus, and other organisms, have been found in some cases in the absence of the streptococcus.

In purely septic diseases the cardiac element serves to facilitate the generation and rapid diffusion of the poison; and, since the latter is prone to attack the valve-segments, the morbid lesions within the heart not rarely constitute the chief pathologic factor in septicopyemia.

Predisposing Affections.—The malignant form occurs, in connection with acute articular rheumatism, in about 10 per cent. of the cases in which acute endocarditis appears. In lobar pneumonia the ulcerative type is common, occurring almost as frequently as the simple variety, and was found by Osler in 11 out of 23 cases. The septic processes that arise from the puerperal state or from gonorrheal infection may also be complicated with ulcerative endocarditis. Among many other diseases that furnish occasional instances of this serious complication are measles, scarlet fever, typhoid fever, erysipelas, small-pox, chorea, tuberculosis, and chronic nephritis.

[1] *Text-book of Medicine*, p. 631.

Clinical History.—That form of ulcerative endocarditis which is a more or less prominent factor in septic diseases has been considered in connection with septicemia. Malignant endocarditis being usually a secondary event, its clinical features must not be confounded with those of the primary affection. It is, however, often impossible clearly to separate the symptoms of the former from those of the intercurrent affection. I shall describe first the common *typhoid form.*

Local symptoms are often entirely wanting, or, when present, consist merely in slight precordial pain and oppression, and are not sufficiently well pronounced to arrest attention. Subjective symptoms are, however, connected with other organs than the heart, and are due to the irritating effects of emboli that occupy the various organs of the body. *Gastro-intestinal disturbance*, as shown by the occurrence of vomiting and diarrhea, is common. *Pain* ascribable to local peritonitis over the spleen, and sometimes also over the liver, is observed. *Hematuria* and *dimness of vision* are also frequent concomitants, and are due to renal and retinal hemorrhages. The *urine* may be scanty and albuminous. The more *general features*, that are the result of the local embolic processes or small abscesses, and, in part, of the valvular lesions, are for the most part typhoid in character. The *onset* is usually signalized by a severe rigor that may be repeated at intervals varying from one to several days, and there is often an irregularly continued fever-curve, often touching a high mark (105° or 106° F.—40.5° or 41.1° C.). I saw a case in which the febrile movement pursued the continued type for seven weeks. The *pulse* is rapid and irregular, though frequently becoming slow within a brief period. The patient rapidly *emaciates*, and from the onset is profoundly prostrated; *nervous symptoms*, as headache, mild delirium, followed by somnolence, and sometimes even coma, appear. Profuse sweating sets in and persists, and as a result the *skin* may be covered by sudamina. An ecchymotic eruption due to cutaneous *emboli* is also common, often associated with a papular or a diffused roseolar rash. At times arthritis may occur. Micro-organisms may be discovered in the blood.

Physical Signs.—These may be negative as regards the heart. In the majority of instances, however, a *systolic murmur* is present, which, when associated with other clinical indications of this affection, is valuable for diagnosis, and especially so if developed while the patient is under treatment for the primary attack. The second sound is sometimes accentuated even when no organic lesions have previously existed. The physical signs of pneumonia and pleuritis (particularly the latter) may not infrequently be noted. Cases occur in which infarcts of the right lung give rise to signs of localized consolidation; the spleen becomes swollen, easily palpable, and is quite tender as a rule; the liver is likewise moderately enlarged and slightly sensitive.

Cerebral Variety.—In a small though decisive percentage of the cases all the clinical features of acute suppurative meningitis are presented, and sometimes to the almost total exclusion of symptoms pointing to the primary disease or to the more typical typhoid form of ulcerative endocarditis. For a description of the symptoms that characterize the cerebral form the reader is referred to the discussion of Purulent Meningitis.

Recurrent Malignant Endocarditis.—By this term is meant an acute ulcerative endocarditis coming on in the course of chronic valvular dis-

ease. As has been pointed out, simple acute recurrent endocarditis is common, though difficult of recognition. The latter condition, as well as the lesions in chronic valvular disease, predisposes to secondary infection by the streptococcus and other organisms. The onset is usually abrupt and marked by a chill. The patient has fever, which may be quite high (104° F.—40° C., or over), and may present either an irregularly intermittent or a truly intermittent curve. The latter is often associated with recurring chills. In either of the above *groups* the course is likely to be acute. In some cases the pre-existing murmur becomes louder and more decidedly blowing; the character of the superadded murmur is changeable; in many other instances, however, there is no appreciable alteration. The condition may arise suddenly, amid the signs of failing compensation, as in a fatal case reported by Dr. H. P. Loomis,[1] in which the patient was semi-conscious, cyanotic, and suffering from intense dyspnea and general dropsy. It was impossible to diagnosticate the cardiac lesions by the murmur present. Occasionally these severe intercurrent febrile attacks end in recovery, and such cases probably belong to the benign form of acute endocarditis. There is a third group of cases that run a subacute or even chronic course, with more moderate elevations of temperature, or, as rarely happens, none at all. Mullin, of Hamilton, has reported a case that lasted more than a year. Here the other clinical phenomena, especially those referable to the heart, are often scanty and indefinite.

In a series of 14 cases of chronic infectious endocarditis reported by F. Billings[2] 5 were implanted on normal valves, while in 2 previous heart lesions were in doubt.

Diagnosis.—It is of paramount importance to consider the previous history and all the circumstances under which individual cases occur. These points, together with the early symptoms, more particularly the severe rigor, early high temperature, and profound prostration, the sweatings, the various embolic phenomena, and the presence of cardiac symptoms, are often adequate for a certain diagnosis. With a clear history and the presence of the more characteristic general symptoms (in particular, the *signs of embolism*), a correct diagnosis is possible, even though cardiac murmurs be absent. Instances in which no data can be found to explain the occurrence of the disease are especially puzzling, and will remain unrecognized if the heart manifests no special symptoms, and embolic phenomena are absent. Here the existence of a chronic valvular affection would afford strong probability of the presence of recurrent malignant endocarditis, especially if an intercurrent fever be present. A blood culture should be undertaken in all cases.

Differential Diagnosis.—The subjoined Table will, I feel, be found valuable as an aid in eliminating enteric fever from the typhoid form of malignant endocarditis:

Ulcerative Endocarditis.	Typhoid Fever.
Previous or associated disease, as acute rheumatism or pneumonia.	Previous health good. History of an epidemic.
Very rarely a primary affection. No prodromes observable.	Always idiopathic, with a prodromal stage.

[1] *Transactions of the New York Pathological Society*, 1890.
[2] *Archives of Internal Medicine*, Chicago, November, 1909.

Ulcerative Endocarditis.	Typhoid Fever.
Ushered in suddenly by a severe rigor, which may recur.	Invasion marked by slight recurring chilly sensations. (Severe chill very rarely.)
The fever rises rapidly.	More gradually, in step-like fashion.
Profound prostration as early as third day.	Profound prostration not earlier than seventh day.
The fever is markedly irregular from time of onset, as a rule.	Less so, especially in the first week.
Embolic symptoms (hemiplegia, etc.) may appear.	Extremely rare.
Cardiac symptoms, especially loud systolic murmur, often present.	Sometimes a soft systolic murmur.
The blood usually shows signs of septic leukocytosis.	The blood shows a leukopenia.
Blood culture may show a micro-organism other than the typhoid bacillus.	Blood culture may show the typhoid bacillus.
Widal reaction and characteristic eruption absent.	Both symptoms usually present and diagnostic.[1]

When no etiologic factors are discoverable, and embolic and cardiac phenomena are absent, we must rely upon the Gruber-Widal reaction, and other laboratory tests, to distinguish typhoid fever from ulcerative endocarditis.

Prognosis.—Most cases that run an acute course terminate in death, and when supposed instances of malignant endocarditis recover they are usually to be regarded as being of benign character. Subacute or chronic varieties, however, such as are most frequently met with in connection with organic heart-disease, sometimes end in recovery.

Treatment.—This is largely supportive. The feeding is to be pushed vigorously, and concentrated forms of liquid food should be given at regular, brief intervals. *Rest* and arterial stimulants in liberal quantities are also demanded, and in addition, quinin, sodium salicylate, and antiseptics may be tried. For the embolic symptoms the salts of ammonium give slight promise of beneficial results, and I prefer the carbonate for this purpose. Potassium iodide in moderate dosage is recommended for its control over vascular metabolism. Antistreptococcic serum has proved efficacious in certain cases in which the streptococcus was the causative agent. Broadbent thinks the vaccine treatment affords a better chance of success than the antistreptococcic serum. Moritz treated a case with antistaphylococcic serum, with a favorable issue.

CHRONIC ENDOCARDITIS.

(*Chronic Interstitial Endocarditis.*)

Two clinical varieties are met with—one following the acute form, the other beginning as a chronic inflammation.

Pathology.—The lesions may be limited to the valvular endocardium (their most common seat), or the mural endocardium may also be involved. In not a few instances the lesions are confined to the edges or bases of the segments, and when seen in the early stages there may frequently be observed merely a slight thickening of the free border of the leaflets; in most cases small prominences appear near their free margins. The endocardium looks opaque and its normal elasticity is lost quite early. When the auriculo-ventricular valves are affected the primary seat of inflammation is the auricular face, but lesions of the semilunar valves

[1] The septic form may simulate malaria in its general course. The points of dissimilarity may be found in the discussion of Septicemia.

begin on the ventricular side and implicate the Aurantian body. Extension of the morbid process to other and all parts of the valvular curtain is common, and it is in cases of this sort that the greatest degree of shrinking and crumpling occurs. The most characteristic lesions consist of inflammation and exudation, which produce cohesion of the segments, roughen the surfaces, and lead to the deposit of fibrin upon them. The *histologic alterations* consist for the most part in a proliferation of the endothelium and a round-cell infiltration of the subendothelial connective tissue. Organization of these products of inflammation into connective tissue, with resulting induration and contraction, is the necessary subsequent pathologic event. In old cases calcification of the diseased structure is frequent. The fibrinous deposits in acute endocarditis may become calcareous "at the same time that the sclerotic processes are taking place in the valve" (Stengel). The shrinking shortens the curtains or curls their free edges, and produces insufficiency in either case, since on dropping into the plane of the valvular orifice they fail to close it perfectly. Valves thus deformed may also obstruct the blood-stream. Cohesion of the invaded segments leading to constriction or stenosis may take place.

Involvement of the semilunar (aortic) segments in the ways previously described opposes an obstruction to the outflowing blood-current on the one hand, and, owing to the inability of the segments to effect perfect closure of the aortic orifice, allows on the other hand a diastolic reflux of blood into the left ventricle. The aortic ring to which the semilunar segments are normally attached becomes sclerosed, and finally the seat of atheromatous changes, either fatty or calcareous. Again, chronic inflammation of the intima of the aorta produces a similarly thickened condition of this layer in spots, followed by atheroma. These changes are most prone to take place in the course of the ascending arch of the aorta or just above the aortic segments. The diseased processes before described may extend to the coronary arteries. Hence sclerotic and atheromatous alterations in the blood-vessels are found frequently in association.

Much less commonly similar lesions are noted at the orifice of the pulmonary artery. A similar involvement of the auriculo-ventricular valves also causes regurgitant and obstructive deformities at the mitral orifice, and in advanced cases the chordæ tendineæ, and even the papillary muscles, are almost invariably invaded by direct extension from the valves. As these structures undergo marked thickening with subsequent contraction, they become shortened and rigid, causing an actual narrowing of the cardiac orifice. In mitral stenosis during the early stages a more or less complete ring of vegetations encircles the mitral orifice on its auricular aspect. The margins of the orifice also become hardened and roughened, with extension to the valvular curtains and the chordæ tendineæ. Under such circumstances the thickened valve could not, during the ventricular diastole, be forced back against the ventricular wall, but would occupy a nearly central position. Owing to cohesion of the free edges of the valvular structures and to contraction of the chordæ tendineæ drawing the leaflets toward the apex of the heart, the transition from this condition to the formation of a hollow cone (*funnel mitral*) is by natural, easy stages. Extensive union of the segments along their free margins may reduce the aperture to a mere button-hole slip (*button-hole mitral*) as viewed from the auricular aspect. The last two forms of lesions are far less commonly met with at the aortic orifice, though they

occur rarely in moderate degree; on the other hand, curling of the valvular edges is far more commonly seen at the aortic than at the mitral orifice, if we except the cases that occur in children. The curtains of the thick, rigid valves may also permanently occupy the plane of the orifice, presenting a small ring-like opening (*annular mitral*).

Fatty degeneration leading to the formation of necrotic (atheromatous) ulcers is common; and calcareous deposits are frequently seen in old cases, either in localized areas or coextensive with the diseased tissue, converting the entire valve into a calcified mass, with loss of the valvular outlines.

Under such conditions of the valves the deposit of fibrin would be greatly favored, and the presence of an ulcerative surface or of a fibrous deposit on the valves affords a ready and satisfactory explanation of the occurrence of embolism in these cases. Emboli may also become detached from cardiac thrombi or from thrombi formed in the peripheral veins. For anatomic reasons the favored seats of embolic processes are, as in acute endocarditis, the spleen, brain, and kidneys, and irritants that cause acute endocarditis, find here a tissue-soil whose capacity for resistance to invasion is greatly lowered. *Chronic mural endocarditis*, which exhibits lesions in the form of grayish-white patches, and chronic myocarditis are, as a rule, due to the extension of the inflammation from the valves, though the ventricular endocardium may be invaded independently of the valvular affection. In one instance of mitral stenosis I observed an enormous calcareous mass partly in the subvalvular tissue and partly in the wall of the ventricle, the segments remaining altogether intact. In advanced stages of most cases of chronic endocarditis myocardial degeneration occurs. It takes the form of fibroid change or fatty degeneration, or both. Aortic-valve involvement, especially when complicated with atheromatous change in the coronary arteries, is most prone to these forms of myocardial disease. Chronic endocarditis may be said to persist until death, although Musser has reported two cases in which the murmur of this lesion disappeared during life.

Sequelæ of Valvular Lesions.—The various valvular defects constantly produce lowering of the blood-pressure in the arterial system and increased pressure in the veins. The variations in arterial and venous pressure causes the blood-current in the capillaries to become slowed, the blood loses more of its oxygen to the tissues, and as the result of this abnormal condition of the circulation, cyanosis and, finally, edema ensue. The effect of valvular deficiencies upon the several cardiac chambers will be most advantageously studied when the individual lesions of the segments are considered .

Etiology.—There can be no doubt that most cases of organic heart-disease occurring in children and young adults are caused by primary *acute rheumatic endocarditis and tonsillitis*, and, although the latter affection cannot in truth be said to terminate invariably in chronic endocarditis, it probably does in most instances. This result, in my opinion, is more frequent in children suffering from acute endocarditis than in adults. On the other hand, not a few cases of chronic endocarditis originate in a very mild grade of acute valvular inflammation, which may be, though itself mute, reinforced by a rheumatic diathesis. Indeed, acute endocarditis may be the sole expression of rheumatic disease. Not less than one-half of all cases of organic valvular disease are caused by rheumatism, and

more than one-half occur between *twenty* and *thirty* years of age. Acute endocarditis complicating scarlatina, measles, chorea, pneumonia, may also be followed by the chronic variety, although probably not so commonly as in the case of acute endocarditis of rheumatic origin.

The *second variety*, in which slow interstitial changes occur from the beginning, is dependent upon—(*a*) biologic irritants (*e. g.* syphilis, malaria, and chronic rheumatism); (*b*) chemical irritants (uric acid, alcohol, lead); and (*c*) mechanical influences. Doubtless the influence of *repeated straining efforts* is the most potent cause of this class of cases. Heavy muscular labor increases constantly the tension in the arterial system, and this acts injuriously upon the valve-segments, setting up a gradual sclerotic change. In like manner, *arterial sclerosis* and *Bright's disease* may cause chronic interstitial endocarditis by maintaining a persistent increase in the vascular tension, though the fact that these affections may in turn result from the action of some of the leading causes of organic heart-disease must also be recollected. *Trauma* has produced in valves previously healthy a sudden, incontestable proof of valvular paresis or laceration that has persisted in a few well-attested cases. This accident is more frequent in cases in which the valves have been already diseased (*e. g.*, ulcerative processes).

The **predisposing causes** of organic valvular disease may be discussed briefly. *Hereditary influence*, as pointed out by Virchow, is especially potent in persons in whom there is hypoplasia of the heart and aorta (*e. g.* in chlorosis). Any malformation of a valve is certain to throw an undue strain upon certain portions, and hence is likely to be followed by interstitial change. Osler, in 17 cases of bicuspid aortic valve, has reported the segments to be uniformly sclerosed. The cases of supposed hereditary transmission are doubtless, however, for the most part, due to the causes mentioned above, and particularly to rheumatism. *Age* exerts a predisposing influence, its effects, however, varying with the valve implicated. During fetal life this is on the right side of the heart in a vast majority of cases; during childhood, adolescence, and early adult life, when the infectious diseases and rheumatism are frequent, it is the mitral valve in most instances; and finally, during middle and especially during advanced life the aortic segments are especially involved. I have, however, found aortic disease to be more common in young adults than most writers are ready to admit, and that it is favored especially by an occupation involving muscular strain (*e. g.* blacksmiths, draymen, soldiers during campaigns). Sex *per se* has little if any effect, though, owing to the greater frequency of certain well-known causes of valvular disease (chorea and rheumatism) in girls and young women, females may be more frequent sufferers than males.

AORTIC INCOMPETENCY.

(*Aortic Insufficiency; Aortic Regurgitation.*)

Definition.—The failure of the aortic valves to prevent a return flow of blood into the ventricle, owing, as a rule, to a diseased condition of the aortic leaflets (sclerosis) that is followed by crumpling and attended with

contraction, shortening, or curling of the edges, and finally calcification.

Pathology.—The aortic orifice may be enlarged (relative insufficiency), and here the normal cusps fail to effect complete closure when they become tense. The cusps of the diseased aortic valves sometimes adhere to the intima of the aorta, and laceration of the semilunar segments, which are the seat of diseased processes (particularly ulceration), is sometimes found *post mortem*, and may be the chief factor in determining the development of the condition. This accident may, though rarely, occur as a result of a severe straining effort in the case of valves previously healthy. Occasionally, also, the principal factor in the production of this valvular lesion is a congenital malformation of the segments whereby they are rendered very prone to chronic endocarditis in consequence of the undue strain to which they are subjected. At times the lesions that give rise to stenosis may coexist with simple aortic incompetency.

Mechanical Influence of the Lesion.—The reflux current passes from the aorta backward through the imperfectly closed semilunar valve into the left ventricle during the diastole of the heart or while the left ventricle is being filled by the normal blood-flow from the auricle. It is clear that over-distention of the left ventricle must result at once from two simultaneous influx currents of blood, with a tendency to an increasing dilatation, especially since the lesion itself is steadily progressive. To expel the increased amount of blood from the left ventricle demands increased cardiac power, and the over-exertion causes a compensatory *hypertrophy*. Dilatation and hypertrophy of the left ventricle develop *pari passu* until this chamber reaches enormous dimensions, forming the *cor bovinum*, which weighs 1000 grams or more (30 to 50 ounces). Under these circumstances the arterial system is overfilled at each ventricular systole. In the very early stage the reflux of blood from the aorta into the ventricle tends to lessen the volume of the circulating medium in the arterial tree, but this depleting influence is successfully counterbalanced by the augmented column of blood thrown from the ventricle during cardiac systole. Hence the requirements for bodily nutrition are, for a longer or shorter time, satisfied. The abnormally large amount of blood that is thrown into the arteries with undue force subjects them to increased tension, and as a result arterio-sclerosis, leading sometimes to atheroma, is commonly developed, and presents its ulterior dangers (aneurysm, apoplexy). The coronary arteries are similarly involved, their caliber being reduced, and particularly at the point of origin. Soon or late the blood-supply to the heart-muscle may become inadequate, and nutritional disturbances now manifest themselves in fatty and fibroid degeneration of the cardiac muscles; these pathologic changes are attended with *secondary dilatation*, which soon predominates over the hypertrophy. The imperfect blood-supply to the ventricular tissue may be accounted for, in great measure, by the narrowed lumen of the coronary vessels, and also in part by the inelasticity of the walls of the latter and by the inefficiency of the aortic recoil. In consequence of the increased tension to which they are constantly subjected the *mitral leaflets* may become the seat of sclerotic endocarditis, and this may lead to mitral insufficiency (usually of mild grade); or there may be a

displacement of the mitral segments in the direction of the auricle, producing a considerable degree of incompetency. There is also in the majority of cases a marked degree of fatty degeneration, with more or less flattening, of the papillary muscles. Again, *secondary dilatation* commonly produces relative insufficiency at the mitral orifice. When incompetency has been established here, impeded pulmonary and general venous circulation, together with the secondary lesions in the left auricle, pulmonary vessels, and right ventricle that are characteristic of mitral incompetency, are the necessary result. The blood-current through the mitral ring may be retarded, owing to the simultaneous influx into the left ventricle from the aorta, thus causing pulmonary congestion without either change in the segment, or overdistention of the orifice.

Special Etiology.—(1) *Acute Endocarditis.*—Incomplete resolution of the acute form of endocarditis leads to progressive chronic valvular disease. In the young it is caused with comparative frequency by rheumatic endocarditis often associated with involvement of the mitral segments. Aortic regurgitation may also arise, though rarely, in the course of acute endocarditis, attended with destructive ulceration. Such instances usually terminate in speedy death.

(2) *Chronic Infectious Irritants.*—I have found syphilis to be a not uncommon factor (though rarely the sole cause), hence the disease is frequent in sailors and soldiers. Syphilis as a causative agency is shown by a positive Wassermann reaction. It is quite probable that chronic rheumatism has a similar influence.

(3) *Chemical Irritants.*—(*a*) *Uric Acid.*—In chronic and irregular forms of gout the irritating qualities of uric acid give rise to interstitial endocarditis and arterial sclerosis. (*b*) *Lead.*—The effects of lead may be primary on the cells of the valvular endocardium, or secondary to chronic degeneration of the kidneys. (*c*) *Alcohol*, by its persistent irritant action, may excite chronic valvulitis.

(4) *Augmented Aortic Tension.*—The excessive functional activity of the heart occasioned by the immoderate use of cardiac stimulants (alcohol) tends to raise the blood-pressure above the normal point, and thus sclerotic endocarditis may be developed very slowly. The effect of *occupation* in causing this disease, by increasing the vascular tension, is more notable than in the case of alcohol, though both of these factors are found not infrequently to be present in the same case. It is undeniably true that strong-bodied men in the middle period of life and engaged in such occupations as entail strain—"not a sudden, forcible strain, but a persistent increase of the normal tension to which the segments are subject during the diastole of the ventricle" (Osler)—are the most frequent sufferers from aortic incompetency.

(5) From personal observation I feel convinced that chronic endocarditis (affecting the aortic valves) may be secondary to *aortic endarteritis* as the result of direct extension. It must be borne in mind, however, that arterio-sclerosis is also often secondary to chronic valvulitis.

(6) Relative insufficiency is caused, in some instances, by pronounced dilatation of the root of the aorta, or by an aneurysm just beyond the aortic orifice. Incompetency of muscular origin (*e. g.*, in marked dilatation of the left ventricle) occurs, though rarely.

Among the more effective predisposing factors are *age* and *sex*. The

disease occurs much more often in males than in females, chiefly on account of greater percentage of the former engaged in occupations that are causally related to the disease. As to age, a preponderating proportion of the cases arise during advanced middle life, and a comparatively smaller number at an earlier period.

Symptoms.—So long as the hypertrophy of the left ventricle successfully overcomes the otherwise injurious consequences of the valvular defect the harmonious balance of forces is maintained, and there is an almost entire absence of symptoms. I have observed, moreover, that compensation does not fail so early in young subjects as in those more advanced in years, or at a period of life when aortic incompetency is often a sequel of atheroma combined with hypertrophy and dilatation of the left ventricle. With the development of marked hypertrophy severe muscular exertion and strong mental excitement will, by exciting over-action of the powerful heart, bring on a train of symptoms as *throbbing headache*, *vertigo*, and *tinnitus aurium*. *Per contra*, the clinical manifestations of arterial *anemia*, particularly of the brain, and also those of general *arterio-sclerosis*, frequently coexist. The patient's countenance exhibits *pallor*, and he complains of *headache*, *flashes* of *light* before the eyes, and *dizziness*. Dilatation of the peripheral vessels often leads to hot flushes and drenching sweats. Cases exhibiting the latter symptoms may be mistaken for *phthisis*. *Dizziness* is often distressing, and is more marked upon rising quickly from the recumbent to the erect posture. *Dyspnea* may come on early, but this rarely happens except upon inordinate exertion or great mental excitement—conditions that cause strong cardiac action and prohibit the discharge of blood from the left auricle into the left ventricle, thus causing *pulmonary congestion*. Oppression in the precordial region and cardiac palpitation are commonly present, as is a *dull aching pain*, but it radiates not infrequently to the shoulders, and thence down the arms, particularly the left. Genuine *angina pectoris* may be a concomitant. I have seen instances of aortic regurgitation in which severe pain simulating rheumatism was located in the left shoulder-joint.

Following immediately upon **failure of compensation** the cardio-pulmonary circulation is retarded, and there is increased *dyspnea*, the latter symptom being greatly intensified by undue exertion and at night. There may be *cough*, and not rarely *hemoptysis*, though less frequently than in simple mitral disease. Later on, *general venous congestion* of a moderate grade follows pulmonary congestion, and the dyspnea now becomes severe. It is nocturnal, and often compels the patient to assume a semi-erect posture in bed. In the later stages the symptoms, particularly those of venous stasis as shown by cyanosis and malleolar dropsy, are due to mitral incompetency, followed by failure of compensation. Marked enlargement of the liver due to passive congestion may now ensue and give rise to the suspicion of a new growth. *Edema* of the feet rarely goes on to general anasarca. In aortic incompetency a higher grade of *symptomatic anemia* is reached than in any other cardiac lesion—a recent blood-count showing 2,800,000 red corpsucles to the c.mm. Hence slight edema of the feet may be due solely or in part to anemia. The intercurrence of acute endocarditis, as evidenced by prostration and irregular fever, is observed, not infrequently as a terminal condition.

The symptoms of cerebral, splenic, and renal *embolism* may arise. Probably *sudden death* ensues, as the result of involvement of the coronary arteries, with greater frequency in this than in all other forms of valvular disease combined; and yet this accident is by no means frequent. Instances of aortic incompetency, in which *nervous phenomena*, as peevishness, irritability, delusions, or melancholia, manifest themselves, are too common to be looked upon as mere coincidences. Many patients are led to commit suicide because of their cardiac lesion when other and erroneous explanations are given to account for their acts.

Physical Signs.—*Inspection* brings to light an enlarged apex-beat; this is displaced downward and outward, being visible in the sixth and seventh spaces, and most marked between the mid-clavicular and anterior axillary lines. The precordial zone may be arched, particularly in young subjects, and the apex-beat is usually markedly heaving in character. The carotids throb forcibly, as do the temporals, brachials, and radials, though less violently. These abnormal pulsations are due chiefly to the strong action of the hypertrophied ventricle, though frequent factors of lesser influence are associated—an arteriosclerosis and a regurgitant blood-stream from the aorta into the left ventricle. The impulse becomes widely diffused and wavy with the progressive enfeeblement of the left ventricle, and venous pulsation due to tricuspid insufficiency may be associated with arteriopulsation later in the affection. Epigastric throbbing may be noticed, and on gently rubbing a spot upon the forehead an alternate paling and blushing appear (*Quincke's capillary pulse*); this may also be noted in the finger-nails. It is not peculiar to aortic insufficiency, however, and may be observed in cases of decided neurasthenia and in anemia. Very rarely the pulse-wave is propagated from the capillaries to the veins of the neck, hand, and back of the foot, giving rise to a visible venous pulsation. L. Webster Fox informs me also that the retinal vessels are often seen to pulsate in this disease. The systolic and diastolic blood-pressures are widely separated.

On *palpation* a forcible heaving impulse is usually felt. When, however, dilatation predominates over hypertrophy, the impulse is weak and undulating. A diastolic thrill is sometimes felt over the base of the heart, and a presystolic thrill is also discoverable, though rarely. The arteries are lengthened and the pulse is characteristic; it is quick, leaping, and full, but, upon striking the finger, recedes abruptly, and is known as the *Corrigan* or *water-hammer pulse*. This sudden collapse of the pulse is most decided when the arm is held in a vertical position. It may lose its distinctive character after compensation is lost. Broadbent has noted a considerable increase in the interval between the apex-beat and the pulse-wave in severe aortic regurgitation. A glance at the sphygmographic tracing will show a sudden rise and fall, with absence or delay of the secondary wave (*vide* Fig. 50). There is marked excess in the arterial pressure in the lower extremity over that of the upper (Hill).

Percussion.—Cardiac dulness is coextensive with the impulse, extending in some cases downward to the eighth rib, and to the left from one to two inches without the mid-clavicular line. Later, enlargement of the left auricle may cause dulness upward and to the left of the sternum. Enlargement of the right ventricle causes an increase of dulness to the right. When the dilatation exceeds the hypertrophy the area of dul-

ness will be much extended transversely and slightly upward, the apex now being more rounded.[1]

On *auscultation* a diastolic murmur is audible with its seat of greatest pronunciation at, or a little below and to the left of, the aortic cartilage and is transmitted down along the left edge of the sternum; this is produced in the left ventricle. From the xiphoid it may be transmitted to



FIG. 50.—Normal pulse-tracing.

the left as far as the spinal column as a mere diastolic whisper. It may be heard, at times, in the vessels of the neck. A. Borgherini affirms that the special direction taken by the regurgitant current determines largely the variable position of the murmur and the variable size of the heart. The *rhythm* of the murmur can be most readily determined by auscultating over the base, for while the pulmonic second sound is usually



FIG. 51.—Pulse-tracing in a case of aortic regurgitation (William Hoffman).

audible at the apex (the murmur appearing to follow it), it is not so when, as sometimes happens, the murmur is quite loud. The first sound is often dull, indefinite, and widely diffused, owing to hypertrophy of the left ventricle. In *quality* this murmur is usually soft, blowing (long-drawn), and frequently musical; sometimes, however, it is somewhat rough and loud. *Associated murmurs.*—In most instances a systolic murmur, brief and harsh in character and transmitted into the vessels of the neck, is also discovered over the aortic region (*double aortic*). The presence of the murmur with the first sound is not diagnostic of actual aortic stenosis. It is more often due to a mere roughening of the semilunar segments or of the intima of the aorta. In advanced cases a soft systolic murmur is commonly heard at the apex; it is readily distinguished from the diastolic murmur by its rhythm, and is occasioned usually by a relative mitral incompetency. Still another murmur, of rare occurrence, is rolling in character, generally presystolic in time, and may be heard at the apex over a limited surface-area. This may be accounted for by the presence of excessive dilatation of the left

[1] A dilated aorta with thickened walls—a condition sometimes associated with aortic regurgitation—may give rise to dulness over and to the right of the manubrium sterni.

ventricle, in consequence of which the mitral leaflets must remain free in the blood-stream during the diastole, and here they set up vortiginous movements that cause the presystolic (Flint) murmur. Duroziez discovered a double murmur in the femoral, but this may be noted occasionally in the absence of aortic regurgitation. Traube has described another arterial phenomenon—a systolic sound in the leg ("pistol-shot"), probably due to sudden systolic distention of vessels that were previously empty.

The **diagnosis** demands the presence of a diastolic murmur, the signs of left ventricular hypertrophy, the peculiar arterial pulsations, and the characteristic water-hammer or Corrigan pulse. The secondary manifestations are usually confirmatory. The diastolic murmur may be absent, in which case a certain diagnosis must not be made. It may be rarely heard with the unaided ear, and not with the stethoscope. In rare cases a diastolic murmur has its origin in the veins adjacent to the heart (*e. g.*, in the anemias). For the *differential diagnosis*, see Aneurysms of the Arch.

AORTIC STENOSIS.

Definition.—A narrowing or stricture of the aortic orifice, due to thickening or adhesion of the valve-segments, and causing an obstruction to the flow of blood into the aorta.

Simple aortic stenosis may be met with, though it is a great rarity. Its development is soon followed by more or less valvular incompetency. It may be secondary to aortic insufficiency; but this is rare, the latter lesion being unfavorable to the development of the former.

Special Etiology.—Rarely rheumatic endocarditis, and still less commonly other forms of acute endocarditis, cause union of the semilunar segments, with resulting stenosis. The most common causative factor is a *slow sclerosis of the aortic valve*, accompanied by calcareous deposits. The more or less immobile, rigid valves narrow the aortic orifice and oppose a barrier to the outflowing blood-current from the left ventricle. The aortic ring may be the seat of changes similar to those just described, resulting in a moderate grade of stenosis with intact leaflets. The lesions are most frequently to be regarded as a part of a general arterial sclerosis, most marked in the region of the thoracic aorta; sometimes, as Peter contends, they are distinctly secondary to sclerotic changes at the root of the aorta. The coronary arteries may be the seat of sclerotic changes. The condition is also rarely congenital. *Males* who have reached *advanced years* are especially prone to aortic stenosis, atheromatous processes belonging to that sex and period of life. Gallavardin [1] has described a rare non-congenital and non-rheumatic form of aortic stenosis occurring in young subjects; it is characterized by extreme latency.

Mechanical Influence of Lesion.—To propel the normal volume of blood through the constricted aortic orifice requires increased strength on the part of the left ventricle, and, as a consequence, the latter hypertrophies. This hypertrophy develops slowly, is uncombined with extensive dilatation unless incompetency be associated, and keeps pace with the progress of the valvular lesions. The undue ventricular tension some-

[1] *Lyon Med.*, January 31, 1909.

times induces more or less sclerotic change in the mitral valves. Hypertrophy of the left ventricle eventually gives way to extreme dilatation, resulting in relative mitral incompetency, with its sequelæ, namely, pulmonary, followed by general venous, stasis.

Symptoms.—The symptoms date from the commencement of failure of compensation often many years after the onset of the disease. Their appearance will be found to follow some unusual muscular effort or the operation of some depressing influence, as the too free use of tobacco or alcohol. They are due to disturbances of circulation arising from a gradual secondary dilatation of the left ventricle, which is now unable to propel the normal quantity of blood into the arterial tree. Hence *anemia*, especially of the brain and peripheral parts of the body, becomes pronounced, and is evidenced by such symptoms as *syncope*, *dizziness*, *headache*, and *pallor*. Since aortic incompetency usually manifests itself secondarily, the clinical features of both affections are sooner or later variously commingled. In cases in which mitral lesions develop they are overcome by compensatory enlargement of the right ventricle: the latter chamber may at a later period become dilated, in which

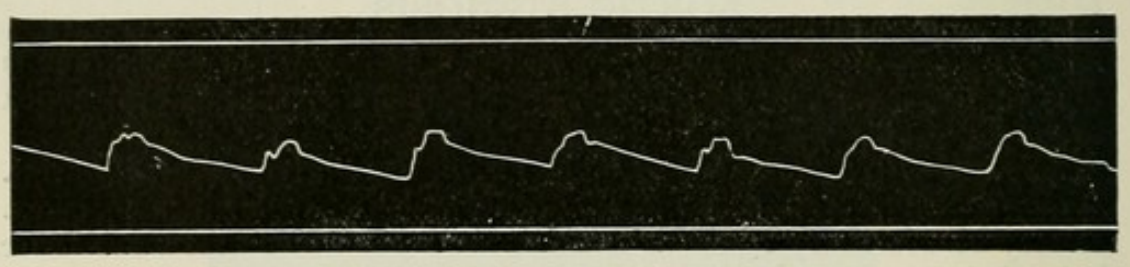


FIG. 52.—Sphygmogram of aortic stenosis, from a man aged sixty years.

event tricuspid regurgitation and the symptoms of general venous engorgement appear. *Slight edema* of the feet is common as a terminal symptom; marked dropsy, however, is uncommon. From the fibrous deposits on the segments, as well as from any small clots behind the valves, *emboli* are apt to become dislodged by the forcible blood-stream and be conveyed to the brain, spleen, kidneys, or other organs.

Physical Signs.—*Inspection.*—The apex-beat is gradually displaced downward and to the left, owing to left ventricular hypertrophy. It is, as a rule, slow, forceful, and heaving, but less frequently may be lacking in strength. It may be enfeebled, diminished in area, or absent, owing to associated emphysema. Absence of the apex-beat may be occasioned by diminished contraction of the myocardium, or during vigorous contraction of the heart, the ventricle emptying itself from the beginning, so that there is "no closing period and with it no apex-beat" (Leube).

Palpation discloses the forcible and heaving impulse-beat, unless obscured or even absent owing to emphysema. A marked systolic thrill, with the seat of greatest intensity in the aortic region, is quite generally present. I have rarely felt this thrill in the apex region. The pulse-wave is small, regular, not compressible, and of normal or slightly lessened frequency (*sluggish*). The estimated blood-pressure is about normal. The sphygmographic tracing shows slowness of the ascending curve and a gradual formation of the descending line (*vide* Fig. 52).

Percussion.—Although hypertrophy of the left ventricle is present,

the area of cardiac dulness is largely dependent upon the degree of emphysema associated. In the absence of this condition the dulness is increased to the left and downward, especially so when insufficiency coexists.

Auscultation.—A systolic murmur, harsh in quality, most audible at the aortic cartilage (the second right), and transmitted into the carotids, is present in typical aortic stenosis. When non-compensation is advanced the murmur is neither so rough nor so loud, and quite late it may be missing altogether. The second sound is faint or inaudible on account of the diminished blood-tension in the aorta and the character of the valvular lesion. As aortic incompetency is commonly associated, a regurgitant or diastolic murmur is also heard, forming a *double* or *see-saw* murmur, the stenotic bruit more or less completely masking the regurgitant. A soft, blowing apical murmur (with the systole) is not infrequent after relative insufficiency of the mitral valves has appeared.

The **diagnosis** demands the concurrence of the following signs: a systolic thrill, most marked at the base; a tense, small, somewhat slow pulse; indications of left ventricle hypertrophy (unless emphysema be present); a rough, loud, systolic murmur at the aortic cartilage, propagated into the vessels of the neck, and a feeble second aortic sound.

Differential Diagnosis.—A calcareous plate lying on the intima of the aorta and a markedly roughened condition of the aortic segments are conditions frequently mistaken for aortic stenosis, since they give rise to a murmur possessing many of the characteristics of the one above described. These murmurs, however, are seldom musical. while the murmur of aortic stenosis is often so; moreover, the second sound is decidedly accentuated, while in aortic stenosis it is faint. In *chronic Bright's disease* with arterial sclerosis and left ventricular hypertrophy a murmur of maximum intensity may be developed at the base; but here the urinary symptoms, together with intensification of the second sound, are sufficient for a discrimination. In *aortic regurgitation* a systolic murmur frequently coexists, but it cannot be reckoned as indicating actual stenosis unless it has a musical quality and a systolic thrill can be felt on palpation. In combined aortic regurgitation the characteristic condition of the pulse of stenosis may be missing. The basic murmurs of *chlorosis* and other forms of anemia are soft and distant, and not harsh; the intense thrill and ventricular hypertrophy are absent. The venous hum may also be heard in the veins of the neck. *Pulmonary stenosis* occurs in young subjects, and while it gives rise to a harsh systolic murmur, is best heard to the left of the sternum, is propagated upward and to the left, and the second pulmonic sound is weak.

MITRAL INCOMPETENCY.

(*Mitral Regurgitation; Mitral Insufficiency.*)

Definition.—Imperfect closure of the mitral valve due to rupture (rare) or contraction of the mitral leaflets. It is also caused by dilatation of the left ventricle and by a diseased condition of the chordæ.

Pathology.—This is the most frequent form of organic disease of

the heart. Thomas G. Ashton,[1] from clinical observation of 1012 cases of heart-affection, comprising all the different varieties, found that 54.4 per cent. were instances of mitral regurgitation. The predominating lesions may be brought under four heads: (*a*) Acute endocarditis, leading to contraction and deformity, particularly curling, of the margins of the valve; (*b*) primary sclerotic form; (*c*) relative insufficiency from excessive dilatation of the left ventricle (the segments being healthy); also from insufficiency of the valvular muscles; and (*d*) adhesion of a segment with the walls of the ventricle, and also contraction and weakening of the chordæ tendineæ.

Mechanical Influence of the Lesion.—The mitral leaflets normally close, and prevent the reflux of the blood from the left ventricle into the left auricle during systole. Hence incomplete closure of the mitral segments allows a portion of the blood to return into the left auricle during the systole. This regurgitant wave meets and offers an obstacle to the normal blood-current coming simultaneously from the pulmonary veins into the left auricle. It is clear that vortiginous movements must result under these circumstances and give rise to a murmur. The double blood-current, entering the left auricle during the systole of the left ventricle, causes over-filling (hence dilatation) of the left auricle, and in a gradual manner induces compensatory hypertrophy of its walls since its labor has been increased. During the next diastole the abnormally large contents of the auricle stream under increased pressure into the left ventricle, producing over-distention (dilatation) of that chamber. This increased volume of blood in the ventricle is not all expelled into the aorta, but a portion of it returns into the left auricle. Thus the left ventricle, in consequence of its increased labor, becomes hypertrophied as well as dilated. Under these circumstances the volume of blood that is poured into the aorta remains about normal, and hence the arterial tension for a longer or shorter period is also normal. Soon the cardio-pulmonary circulation becomes impeded. The blood that returns into the left auricle must, by reason of pressure, offer increased obstruction to the outflow of blood from the pulmonary veins, and the pressure in the latter must, in turn, be similarly increased. The current of the blood through the pulmonary capillaries and branches of the pulmonary artery is thus retarded, owing to the gradual backward accumulation. The walls of the lung-vessels are the seat of a sclerotic process, and present an abnormal obstacle to the passage of the systolic wave from the right ventricle to the distal end of the cardio-pulmonary arc. As a consequence of the lung congestion and vascular changes the right ventricle becomes dilated and hypertrophied. The abnormally increased tension in the pulmonary vessels is shown by the accentuated pulmonic second sound. Thus the right heart compensates the lesion in the left, though to supply an adequate amount of blood to the peripheral arteries the left ventricle must maintain its proper degree of hypertrophy. As soon as this harmonious balance is disturbed, either as the result of increase in the degree of incompetency or of failure of muscular power, the progress of the blood from the right auricle to the right ventricle is hindered. Increased pressure in the right auricle produces dilatation of its chamber, with subsequent general venous congestion as a natural backward effect (*vide* Tricuspid Regurgitation). It is now seen that

[1] *Medical News*, June 30, 1894.

when the right heart fails a lessened amount of blood reaches the left ventricle, and hence an abnormally small amount finds its way into the aorta; this fact explains the presence of the low arterial tension late in the disease. Hypertrophy of the left ventricle in this disease has also been attributed in part to the augmented tension in the general capillary vessels that is occasioned by the venous stasis.

Special Etiology.—(*a*) *Rheumatic endocarditis* is the most frequent cause, though mitral regurgitation also results less frequently from acute endocarditis due to other causes. (*b*) It may be a part of a *general arterio-sclerotic process*, caused, not rarely, by syphilis and alcohol. (*c*) *A diseased condition of the columnæ carneæ* or *chordæ tendineæ*, if it contracts them or weakens their structures so that the free edges of the segments pass beyond the plane of the orifice, produces insufficiency. (*d*) It rarely arises in the course of *aortic valvular disease* (a secondary mitral affection), and is then excited mainly by undue tension of the blood in the left ventricle. Here the lesion is of a mild grade, as a rule. (*e*) It is frequently occasioned by *enlargement of the left auriculo-ventricular ring*, resulting from excessive dilatation of the left ventricle, as in aortic incompetency, aortic stenosis, long-continued fevers (toxic myocarditis), and the graver anemias (relative incompetency). (*f*) *Ulcerative endocarditis*, either by perforating or producing rupture of the valve-curtains or by destroying the chordæ tendineæ, may bring about mitral incompetency. Among *predisposing factors* age and sex are worthy of special mention, the incompetency occurring with greatest relative frequency in young adults (from twenty to thirty years of age, according to Ashton's figures) and somewhat more commonly in males.

Symptoms.—*During Compensation.*—In healthy persons the compensatory forces keep pace with the valvular lesions for an indefinite and usually lengthy period, during which time there may be an entire absence of symptoms. When present they are dependent upon disturbances of the cardio-pulmonary circulation that are occasioned by trivial causes, such as excitement, going up stairs, or other forms of active physical exertion. Under these circumstances the force of the regurgitant current is increased (by the hypertrophied left ventricle), thus producing more or less *pulmonary congestion* that may proceed to edema of the lungs or hemoptysis. The condition is usually a temporary one, and is attended by *dyspnea*, *palpitation of the heart*, a *short, hacking cough*, and *expectoration* of a frothy serum that may be blood-stained. The relation existing between the severity of the dyspnea and the degree of active physical exertion is positive and vital. Shortness of breath may be the sole feature during a long period. The rational symptoms rarely warrant a suspicion of the existence of mitral disease until compensation has failed, but the patient's appearance often indicates heart-disease. The *face* is pale and the features peaked, the eyes, lips, and ears are dusky, and the minute vessels of the cheeks are prominent. Clubbing of the finger-nails is observed most frequently in the young.

After Failure of Compensation.—Failure of compensation implies failure of the right ventricle to cope efficiently with the augmented tension in the pulmonary circulation, with accompanying congestion of the lungs, followed by engorgement of the systemic veins. The latter process begins at the right heart and proceeds toward the periphery, involving

the viscera, mucous membranes, and extremities until it is universal. The *pulmonic symptoms* above detailed are now more marked, particularly the dyspnea (which may be constant), cough (with expectoration of alveolar epithelium containing brown pigment-granules), and cardiac palpitation with arrhythmia. *Pain* is rare unless stenosis coexists. *General venous engorgement* manifests itself by an enlargement of the liver and of the spleen, in the features of gastro-intestinal catarrh, in hemorrhoids, in marked cyanosis of the surface, and in the passage of a scanty albuminous urine containing tube-casts and blood-corpuscles. Dropsy follows, beginning in the feet and progressing upward, until finally the trunk and the serous sacs are involved. By stimulation the heart may be reinforced, and all of the unfavorable symptoms disappear. I have at present under observation a case in which not less than half a dozen instances of broken compensation have occurred at intervals of six to eight months.[1] In all cases, however, there comes a time when compensation cannot be restored, and the end is soon reached.

Physical Signs.—*Inspection.*—The precordia is prominent, particularly in children, and the area of the apex-beat is enlarged, later becoming diffuse and wavy. It is carried to the left and downward, sometimes to the sixth interspace, corresponding with the degree of hypertrophy of the left ventricle. A pulsating epigastrium is in frequent association, particularly after dilatation of the right ventricle appears. With the failure of the right heart also come wavy pulsations in the cervical veins, and occasionally a mild grade of jaundice.

Palpation sometimes discovers a thrill at the seat of the apex-beat, synchronous with the first sound. The impulse during the stage of full compensation is forceful and heaving, but with the beginning of failure of compensation it grows feeble and irregular, and late in the affection is excessively weak and arrhythmic. The pulse bears a definite relation to the apical impulse; it is commonly regular and full during the compensatory period (though at times the tension is slightly lowered), but becomes small, easily compressible, and exceedingly irregular during the period of broken compensation. One meets with cases in which irregularity appears during the period of fair compensation.

Percussion.—The dull area is increased to the left, extending frequently to the anterior axillary line; and also to the right, frequently from $\frac{1}{2}$ to 1 inch (1.2–2.5 cm.) without the right sternal margin. Dilatation of both ventricles exerts a widening influence; hence cardiac dulness is increased more laterally than vertically. The upper arc of cardiac dulness commences usually at the third intercostal space.

Auscultation reveals a systolic murmur, with greatest intensity at the apex (see Fig. 53). It is rarely loudest in the fourth or third space in the vertical nipple line. Balthazar Foster first called attention to the fact that the murmur of mitral regurgitation may be loudest at the base of the heart, and at times audible only in that situation—an occurrence that has since been confirmed. It is sometimes audible in the recumbent posture and inaudible in the erect. From the

[1] Neglect of hygienic precautions and intercurrent complaints of various sorts often determine the occurrence of failure of compensation.

apex it is transmitted to the left as far as the angle of the scapula, with progressively diminishing clearness. It has a blowing quality, and frequently ends in a musical tone. Loudness implies strength of contraction (Broadbent). It is fair to assume that on account of the defect in the closing of the mitral valve, there is often a decreased tone-formation with systole. Over the third left costal cartilage, and frequently at the apex, there is heard the accentuated pulmonic second sound, due to the increased tension in the pulmonary vessels engendered by the hypertrophy of the right ventricle. *Combined murmurs* may be heard, and not infrequently a rough, rolling, or rumbling presystolic murmur is detected. A frequent late occurrence is secondary dilatation of the right ventricle, causing relative tricuspid insufficiency with its characteristic soft, low-pitched, systolic murmur, heard best at the ensiform cartilage. A spurious diastolic murmur may

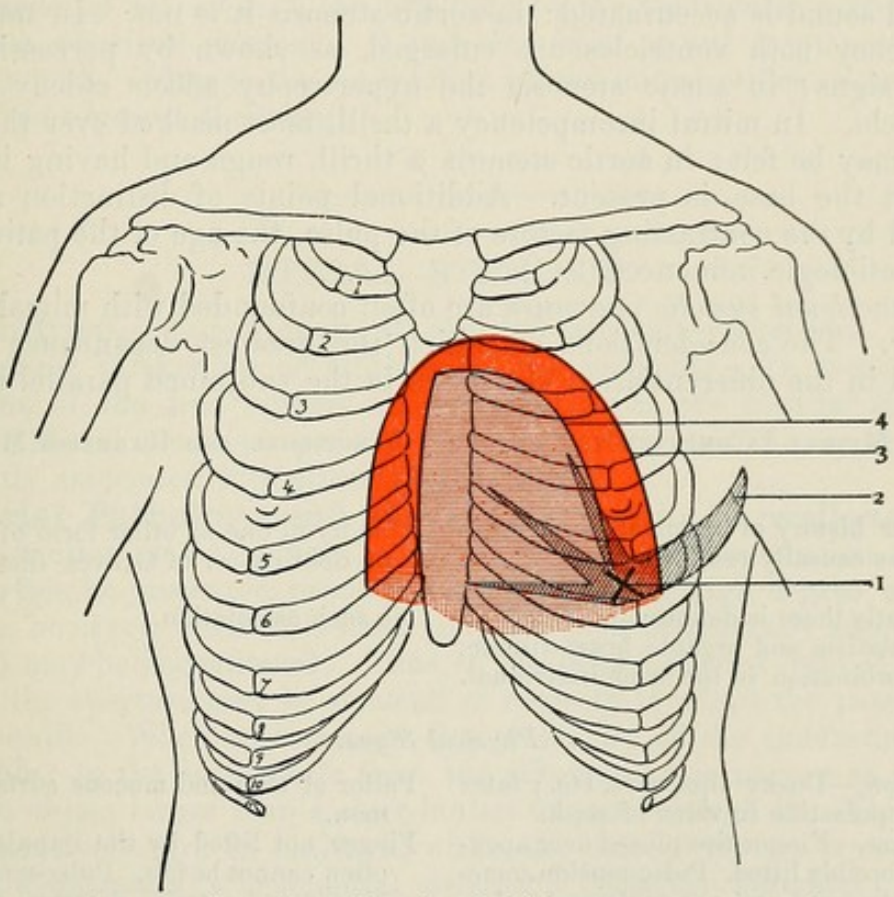


FIG. 53.—1, Seat of greatest intensity; 2, direction of chief transmission; 3, boundary line of relative dulness; 4, boundary-line of absolute dulness (modified from Sahli).

be noted, though rarely, when the sounds are timed with the pulse. This is due to a weak systole that fails to cause a radial pulse.

Diagnosis.—In the presence of the following group of features the diagnosis is set at rest: A marked broadening of the area of cardiac dulness; a systolic, *apical* murmur that is conveyed to the left axilla and may be heard even at the back; and a decided accentuation of the pulmonary sound. Obviously, the latter sound becomes feeble after dilatation of the right ventricle has occurred. A systolic thrill is of the highest diagnostic importance, but is unfortunately absent in

perhaps a majority of the cases. Free regurgitation through the mitral orifice may be safely inferred when the following signs are concurrent: (*a*) An absence of the sound of mitral-valve tension, a murmur replacing the first sound; (*b*) accentuation of the pulmonic second sound; (*c*) an enlarged area of the left cavity; (*d*) an enlarged area of the right cavity (Sansom).

Differential Diagnosis.—There are two organic lesions of the heart that are sometimes mistaken for mitral incompetency, since both are accompanied by a systolic murmur—the one *aortic stenosis*, and the other *tricuspid regurgitation*. How to distinguish mitral from tricuspid incompetency is a question that will receive due attention when the latter disease is considered. *Aortic stenosis* generates a systolic murmur, but it is loudest over the base, and is transmitted through the great vessels of the neck; while the mitral systolic is most intense over the apex and is transmitted far to the left. In mitral incompetency the pulmonary second sound is accentuated; in aortic stenosis it is not. In mitral insufficiency both ventricles are enlarged, as shown by percussion and other signs; in aortic stenosis the hypertrophy affects chiefly the left ventricle. In mitral incompetency a thrill, most marked over the apex-beat, may be felt; in aortic stenosis a thrill, rough and having its chief seat at the base, is present. Additional points of distinction are furnished by the contrasting factors of the pulse, the age of the patient, and other etiologic influences.

Functional systolic murmurs are often confounded with mitral insufficiency. The considerations on which the greatest dependence is to be placed in the differentiation are given in the subjoined parallel tables:

Mitral Incompetency.	Functional and Harmless Murmurs.
History.	
Previous history of rheumatism or other disease causally related.	History of one or other form of anemia, of debility, or of Graves' disease.
Frequently there is definite knowledge of rheumatism and organic heart-disease, in combination in the same individual.	No such association.
Physical Signs.	
Inspection.—Dusky lips, ears, etc.; later wavy pulsation in veins of neck.	Pallor of skin and mucous surfaces common.
Palpation.—Finger-tips placed over apex-beat forcibly lifted. Pulse-tension somewhat lowered and not prolonged. Impulse displaced.	Finger not lifted by the impulse, which often cannot be felt. Pulse-tension prolonged and arterial pressure increased generally. Impulse not displaced.
Percussion.—Evidence of dilatation of both ventricles.	Dilatation of right auricle, but only in about one-half of the cases, giving rise to dulness above or to the right of the right edge of sternum.
Auscultation.—A systolic apex-murmur (often musical), with characteristic area of transmission. This murmur is often heard posteriorly; pulmonary sound accentuated.	Soft systolic murmur at apex (variable in intensity, rarely transmitted to axilla), usually preceded by or associated with a basic systolic murmur and a venous hum in the veins of the neck.

To differentiate *relative* from *organic mitral incompetency* is difficult. It rests upon two points: (*a*) the character of the murmur, which is softer

and shows greater changes in intensity (*e. g.*, being either less pronounced or disappearing if the heart is "whipped up" by digitalis) than that due to valvular lesions; and (*b*) the antecedent history of the patient. Thus, relative insufficiency of the mitral segments probably exists in patients in the middle period of life, in whom the previous history either furnishes such etiologic factors as chronic gout, syphilis, or evidences of myocarditis, fatty heart, or anemic conditions; or in persons who exhibit arteriosclerosis or organic disease of the aortic valve and an apex-systolic murmur. Again, if present in chronic *renal* disease, with concurrent symptoms of high arterial tension and of left ventricular hypertrophy—accentuation of the second aortic sound, a mitral systolic murmur—it is to be ascribed to relative insufficiency. On the other hand, if the signs of mitral regurgitation occur in a younger subject or in one who has been afflicted with acute rheumatism, it is highly probable that the mitral-valve segments are the seat of chronic endocarditis of rheumatic origin. *Compression of the edge of the left lung* by the ventricular systole may produce a spurious murmur. A rare sequel of mitral incompetency is mitral stenosis, owing to the contraction of the mitral orifice or cohesion of the free edges of the cusps.

MITRAL STENOSIS.

Definition.—Constriction of the left auriculo-ventricular orifice, due to either thickening or adhesion of the segments. In most cases, adhesions of the free borders of the valve or of the chordæ tendinæ obtain. Mitral stenosis is generally combined with insufficiency, and also frequently associated with adhesive pericarditis.

Special Pathology and Etiology.—It is to be recollected that the constriction may be almost inappreciable, and yet an uneven, roughened surface be presented, producing a murmur. A high degree of constriction, however, "which is more frequent than is generally supposed" (Elliott), may be encountered. Thus, in the *funnel-shaped* form of mitral stenosis the aperture may be so small as scarcely to admit the passage of a goose-quill. When moderate in degree the tip of the index-finger is admissible; in the *button-hole* form the slit may be so narrow as not to allow an object larger than a shirt-button to pass through it. This form is comparatively rare in children, while the *funnel* variety is common, and is occasionally a congenital condition (possibly hereditary). In adults, however, the funnel-shaped constriction is rare, while the button-hole valve is common; in 62 *post-mortem* examinations only 3 showed funnel-form contraction (Hayden and Fagge). Mitral stenosis is, generally, dependent upon a mild or limited endocarditis of rheumatic origin. It is more common in *young adults* and in *children* after the fifth year than in older persons, and a greater incidence is shown in *females*, for the reason that the affections that are causally related to endocarditis are more frequent in females (rheumatism, chorea, chlorosis). The endocarditis of measles and scarlatina may also lead to narrowing of the mitral orifice, and I quite agree with Osler in the belief that whooping-cough, owing to the great strain that it imposes upon the heart-valves, may be accountable for certain cases. In adults *arteriosclerosis* and *chronic nephritis* may

act as causes. The hemorrhagic cases may at times be the immediate effect of an accident.[1] In not a few cases the etiology is obscure, particularly in adult women. Ball-thrombi have been found in the auricle.

Mechanical Influence of the Lesion.—The task of the left auricle is greater than normal, and as a consequence its walls hypertrophy. They may be found to be one-fourth or even one-half inch (1.2 cm.) in thickness, the normal thickness being only three-twentieths of an inch (3.7 mm.). Dilatation of the auricle comes on early, since this chamber cannot take on much hypertrophy owing to lack of muscular structure, and in the later stages its walls become extremely thin. For a varying period of time the increased power due to hypertrophy of the left auricle and the increased resistance to the circulation that is the result of the mitral lesion are exactly balanced. At a comparatively early period, however, the auricle can no longer maintain this equilibrium; and then, owing to retardation of the current from the pulmonary veins to the auricle, the vascular tension in the lungs and right ventricle is increased. The right ventricle, in seeking to overcome the obstruction, becomes greatly hypertrophied and dilated, and late in the disease tricuspid incompetency supervenes. The hypertrophy of the latter chamber counterbalances the lesion during the period of compensation. For a brief time the left ventricle exhibits no abnormal proportions. Later and at autopsies its cavity is found smaller and its walls thinner than the normal, these conditions being due to its abnormally light labor. The apex of the heart is formed almost exclusively by the enlarged right ventricle. If the left ventricle be hypertrophied, it is owing to co-existence of mitral incompetency.

Symptoms.—The subjective symptoms are scanty. During the period of compensation they may be absent except on going up stairs or on attempting some unusual muscular effort, when *dyspnea* appears. Fragments of fibrinous coagula dislodged from between the musculi pectinati of the auricle or swept from the valves may give rise to the phenomena of *cerebral embolism* (aphasia and hemiplegia). The same conditions may arise, and in the same way, from recurring endocarditis, to which such patients are specially liable. The patient in well-marked cases presents an *anemic* appearance: a *stitch-like pain* in the apex-region is frequently present, and active exertion, by overtaxing the left auricle, induces *cardiac palpitation* and *dyspnea*.

After failure of compensation the symptoms referable to the pulmonary system are almost identical with those manifested in mitral incompetency. Owing to the pulmonary engorgement the *dyspnea* is constant, and is increased by exertion. After severe physical exercise *congestion*, followed by *edema* of the lungs, may supervene, attended by a copious blood-stained, serous *expectoration*. True *hemoptysis* may arise from time to time. The sputum often contains large, mostly oval, nucleated cells showing yellowish-brown pigment ("heart-failure cells"). The increased tension in the pulmonary vessels leads to sclerosis, followed by atheromatous degeneration of their walls, and may result in *pulmonary apoplexy*. Intercurrent *febrile attacks* (due to recurring endocarditis) are common, particularly in the later stages, and are attended with marked aggravation of the circulatory disturbances. Mitral steno-

[1] See also "Trauma and Heart Disease," by J. C. Wilson, *Jour. Amer. Med. Assoc.*, Feb. 10, 1912, p. 405.

sis differs from mitral incompetency in that *general anasarca* is rare, though marked enlargement of the liver and other evidences of portal congestion (including ascites) are commonly present. Boinet, Osler, and others state that paralysis of the left recurrent laryngeal nerve may occur either as the result of compression or traction.

Physical Signs.—*Inspection.*—The apex-beat is diffused, but not displaced downward, unless there be excessive enlargement of the right ventricle or associated hypertrophy of the left. There is usually observed pulsation in the second left intercostal space, and sometimes in the third and fourth, occasioned by increased tension in the pulmonary artery; there is also a diffuse impulse along the right border of the sternum. Epigastric pulsation is common. A prominence over the fifth and sixth left costal cartilages and the lower half of the sternum is observed, particularly in children. After failure of compensation the impulse is feeble and undulating, with engorgement and pulsation of the jugular veins.

Palpation discovers a presystolic thrill in a great proportion of cases. In certain instances active physical exertion may render this appreciable, or when in the recumbent posture on the left side the elevation of the arms may accomplish the same result. It is, however, absent in rare instances before failure of compensation occurs, and more frequently by far after the latter event. This fremitus is best felt over the third and fourth (less frequently the fifth) interspaces, just within the nipple, and during expiration. It commences after the second sound (during the diastole) as a purring fremitus, increasing steadily in volume and intensity, and terminates abruptly with the severe shock of the new impulse. The fremitus and systolic shock are pathognomonic, and may be relied upon in the absence of the murmur. The heart's impulse is most forcible over the lower portion of the sternum and along the right border, being due to the enlarged right ventricle; in a smaller propor-

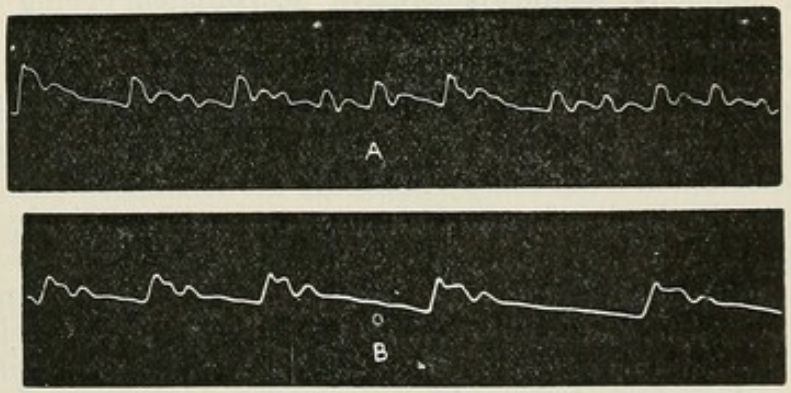


FIG. 54.—Sphygmograms in a case of mitral stenosis treated by extract of convallaria, and subsequently by digitalis: *A*, before treatment, showing the interpolated pulsations; *B*, after treatment (Sansom).

tion of cases, in the third, fourth, and fifth interspaces to the left of the sternum. The radial pulse is small, compressible, and markedly irregular (disorderly) as the propulsive power of the right ventricle diminishes. Arrhythmia often appears early, and is due to failure of contraction of the left auricle. The sphygmographic tracing is notably irregular (*vide* Fig. 54).

Percussion shows an extension of heart-dulness to the right, fre-

quently 5 centimeters (2 inches) beyond the sternal margin, as a result of hypertrophy of the right ventricle, and upward as high as the second rib on either side of the sternum. Increase in the cardiac dulness to the left also occurs not infrequently, and is attributable to excessive enlargement of the right ventricle, though more often of the left ventricle in consequence of associated mitral insufficiency.

Auscultation reveals a rough, presystolic murmur, which may be characterized as churning or rolling, acquiring increased intensity. It occurs synchronously with the thrill. Its point of greatest pronunciation is just above and about one inch within the normal apex-beat. The area of transmission does not exceed a couple of inches in any direction. Griffith, however, has shown that the murmur may be widely transmitted. This murmur sometimes exhibits atypical characters: it may be brief, low-toned, and inconstant. After the right ventricle becomes weak, the murmur may absent itself either temporarily or permanently. In most cases, the clear, accentuated first sound is retained, even though the murmur disappears. Improvement in the muscular power of the heart as the result of judicious treatment may cause the murmur to reappear. For purposes of diagnosis, nothing is so vitally important as the rhythm of the murmur, hence the observer must palpate the heart, and not the radial pulse, while practising auscultation. The finger as well as the ear will thus become sensible of the systolic shock which replaces the cardiac impulse, and it will be noted that the murmur terminates at the same moment. In cases in which the impulse cannot be felt, the finger should be placed over one or other carotid, since here the pulse is practically synchronous with the systole. In most cases the murmur occupies only the latter half of the diastole. In some cases it is purely diastolic, the blood being driven under high pressure in the lesser circulation, from the auricle into the relaxed ventricle, at the beginning of the long pause. Owing to the presence of right ventricle hypertrophy the second pulmonic sound is greatly accentuated, being distinctly audible at the apex, while the second aortic sound is often absent or feeble. Reduplication of the second sound is not rare.

Secondary Murmurs.—While mitral stenosis may rarely follow mitral incompetency or aortic valve disease, in the vast majority of instances it is a primary affection. Secondary murmurs are not uncommon, however. Among these the bruit of *mitral incompetency* is relatively frequent. After compensation is ruptured the murmur of *tricuspid insufficiency* usually becomes audible at the lower end of the sternum and persists until the end. In so-called "*relative mitral stenosis*," associated with primary dilatation of the left ventricle, which holds the orifice open, there occurs also a mitral regurgitant murmur.

Diagnosis.—The distinctive features of mitral stenosis are—(1) A presystolic thrill at the apex. (2) An increase in the precordial dulness upward and to the right. (3) A murmur which (*a*) has its seat above, yet near, the normal apex-beat; (*b*) is usually localized; (*c*) is presystolic in time, terminating abruptly with the systolic shock (sharp impulse); and (*d*) is rough and vibratory in character. (4) A marked accentuation of the second pulmonic sound.

Differential Diagnosis.—When the murmur of mitral stenosis is very brief, it is difficult to eliminate a *mere roughening* without valvulitis.

In the latter condition, however, there is no increase in intensity of the murmurs on exertion or when the arms are uplifted, they are not vibratory in character, and there is no right ventricular hypertrophy. From simple mitral stenosis the lesion of *mitral incompetency* is easily distinguished by its systolic rhythm, greater area of transmission, and by the soft, more blowing character of its murmur. As stated, the majority of the cases of mitral stenosis are associated with mitral incompetency; it is clinically important to recognize the combined presence of these two valvular lesions, and also which lesion predominates in the individual case. The presence of the systolic murmur is distinguishable by its synchronism with the impulse or carotid pulse, and by its area of transmission to the left as far as the axilla. If now the stethoscope be applied just above and to the right of the normal apex, a limited superficial area will be found where a typical presystolic murmur is distinctly heard. Points can also usually be found where a continuous bruit, covering a portion of the period of diastole and the systole, is audible. A rumbling apical sound resembling a presystolic murmur may be heard in pericardial adhesion. Its seat is different and it does not end in sharp systolic shock.

In *aortic regurgitation* the presence of a presystolic thrill and murmur has rarely been recorded, and Fisher, Phear, and others have noted them in simple dilatation ("relative mitral stenosis"). When a purely diastolic murmur is present in the aortic area, indicating aortic regurgitation, the diagnosis of mitral stenosis must be made with due caution.

TRICUSPID INCOMPETENCY.

(*Tricuspid Regurgitation.*)

Definition.—An imperfect closure of the tricuspid valve, due either to a dilatation of the right ventricle that is secondary to mitral or lung-disease, or, less frequently, to an inflammatory shortening of the valves.

Pathology and Etiology.—As a primary disease tricuspid incompetency is rare. It, however, is not uncommonly due to chronic organic changes, though originating in fetal endocarditis. After birth this variety is most common during childhood, and the frequency of occurrence is in inverse ratio to the age. At any period of life, however, chronic affections of the lungs or organic disease of the left side of the heart may, by augmenting the tension in the right ventricle, produce chronic interstitial changes in the tricuspid segments. These are usually of mild grade. In chronic bronchitis associated with emphysema, and in pulmonary tuberculosis, extensive lesions of these valves are seen rarely, owing to the fact that dilatation of the right ventricle is soon followed by relative insufficiency, and thus the strain is in great part removed from the valves themselves. And yet, according to Byron Bramwell, the

tricuspid valve is implicated in 50 per cent. of all cases of acute endocarditis. He suggests that the acute form frequently results in cure because of the relatively diminished right intraventricular tension. In rare instances one of the leaflets has been ruptured by straining. The relative tricuspid insufficiency, produced in a manner analogous to relative mitral insufficiency, is an exceedingly common secondary condition in affections of the lungs and heart that cause hypertrophy and dilatation of the right ventricle (mitral incompetency and stenosis, emphysema, sclerosis of the lung).

Physiologic Pathology.—In tricuspid leakage every systole of the right ventricle is accompanied by a reflux of venous blood through the imperfectly closed tricuspid orifice into the auricle, and thence into the veins. This causes venous stasis and visible pulsation, and in this manner the engorged pulmonary circulation is somewhat relieved. An unfavorable consequence, however, of the reflux current from the right ventricle is the lessened blood-supply to the already engorged pulmonary arteries. The hypertrophied and dilated right heart now undergoes further enlargement in the same manner as in the hypertrophy of the left ventricle following mitral incompetency, though to a less extent. In mitral incompetency the right ventricle compensates the mitral lesion after failure of the left auricle, but there can be no such effective compensatory mechanism after failure of the right auricle in tricuspid incompetency, since the right heart is not reinforced by a fellow as is the left. The blood-stream flowing into the right ventricle during diastole, however, is abnormally large, owing to moderately increased venous tension. When the right ventricle fails to maintain the pulmonary circulation, progressive dilatation of its chamber occurs, with a proportionate thinning of its walls, until its dimensions are enormous.

Symptoms.—In most instances the indications of the primary or causal affection must be noted, though these are often more or less screened by the more characteristic features of the disease under consideration. The symptoms of tricuspid incompetency point to passive congestion of the lungs and engorgement of the systemic veins, and have been described in connection with mitral lesions. *Cardiac dropsy* is common, though present in by no means all cases. It is a prominent feature in the cases that are secondary to mitral disease. Frederick Taylor[1] contends that *ascites* is absent frequently, because the liver acts as a diverticulum to accommodate the excess of venous blood.

Physical Signs.—*Inspection.*—Venous pulsation, caused by the backward blood-wave from the right ventricle and auricle, is a pathognomonic sign. It is confined to the lower portion of the jugular veins so long as the valve that lies above the jugularis remains closed, but soon this yields, and then the veins seem to pulsate through their entire course. This is best seen when the patient is in the semi-recumbent posture, and is most marked in the right side. The venous pulse is presystolic-systolic in time (Leube). The increase in the venous tension, and the slowing of circulation in the capillaries, combine to produce a cyanosis that is more noticeable when the breathing is discontinued temporarily than in ordinary respiration. Tricuspid incompetency may be shown

[1] *Lancet*, Nov. 22, 1890, p. 1126.

by pressing on the vein with the finger rather firmly, commencing just above the clavicle and passing upward, thus emptying it of blood. If, now, the right ventricle be capable of producing a return wave sufficiently powerful to overcome the valve in the external jugular, pulsation is seen in the vessel slowly and increasingly until the vein, as far as the point compressed, becomes filled. The vein fills "by jets synchronous with the heart-beat" (Sansom). If an impulse be communicated to the jugulars from the underlying carotid artery, the light pressure upon the vein below does not arrest the pulsation above, as is the case in tricuspid incompetency. A feeble presystolic venous pulse, due to the weaker contraction of the right auricle as compared with that of the right ventricle (*anadichrotic venous pulse*) may occur. The area and seat of the apex-beat vary with the nature of the primary affection; in mitral incompetency, for example, the beat is displaced to the left and downward, while in uncomplicated mitral stenosis no appreciable displacement occurs. To the right of the sternum an undulatory pulsation is seen, due to contraction of the right auricle and ventricle, but this is not characteristic, since it may take place in simple mitral stenosis without tricuspid regurgitation. Epigastric pulsation is almost invariably observed.

Palpation detects the heaving impulse of the right ventricle in the upper epigastric region. Rhythmic expansile pulsation of the veins of the liver is quite diagnostic and is usually detectable. To obtain this sign the patient should lie on the back with the arms raised, and the examiner should place the palm of his left hand over the right mid-axillary region, and that of the right hand over the upper abdominal region. He will thus be enabled to feel an expansile pulsation of the liver synchronous with the ventricular systole. This is to be carefully distinguished from mere systolic depression of the organ due to the impulse of an enlarged right ventricle, transmitted through the diaphragm and left lobe of the liver to the epigastrium.

Popoff and others have noted an inequality in the radial pulses in tricuspid regurgitation. This is probably due to the pressure of an enlarged auricle. The radial pulse is small, irregular, and often rapid. The blood-pressure in the arterial tree is low.

Percussion.—The extent and form of precordial dulness are variable according to the nature of the causative disease, but a dulness extending far beyond the right edge of the sternum is especially characteristic.

Auscultation.—A systolic murmur having its seat of greatest intensity at the base of the ensiform cartilage (*vide* Fig. 55) is almost constantly audible. The area in which it is best heard varies according to the intensity of the murmur. Lowering the patient's head, while in the recumbent posture, causes murmurs to become evident which were not previously heard (Stern's sign). It is clearly conveyed to the left one inch beyond the left sternal margin, and to the right and upward for an equal distance beyond the limit of cardiac dulness. It is soft in character, short, and often faint. If the heart be weak, it may be absent. Additional murmurs, due to primary lesions, are often heard, and usually at other orifices. The second pulmonic sound is not much accentuated.

Diagnosis.—I believe that the most valuable symptom for diagnosis is the venous pulse, whether observed clearly in the neck or determined positively by bimanual palpation of the liver. The murmur is generally audible. Relative incompetence distinguishes itself from that due

to valvulitis by greater extension of dulness to the right, and by disappearance of the positive venous pulse and murmur, with restoration of compensation. The **differential diagnosis** between mitral and tricuspid regurgitation is easy when either exists alone, if it be remembered that the seat of greatest pronunciation, the area of transmission, and the acoustic character of the respective murmurs are widely different. But it is extremely difficult to discern a faint tricuspid murmur when it

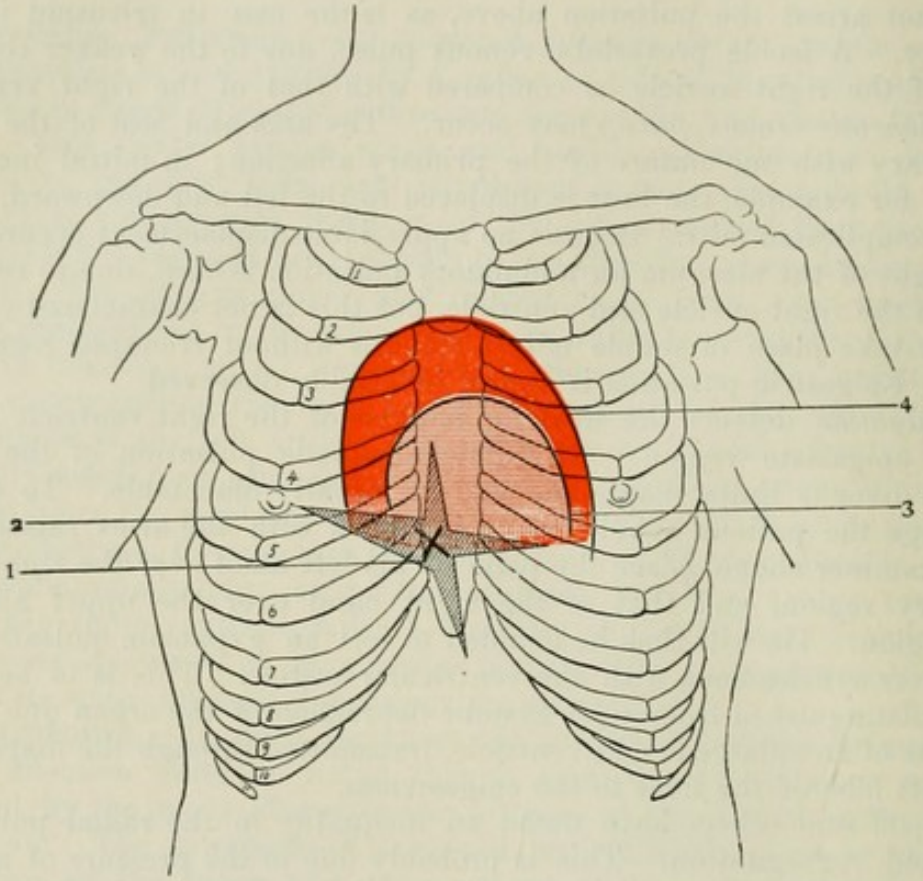


FIG. 55.—1, Seat of greatest pronunciation; 2, chief direction of conveyance; 3, boundary-line of absolute dulness; 4, boundary-line of relative dulness (modified from Sahli).

develops secondarily to the murmur of mitral incompetency. If a careful observation of the murmur fails to establish the diagnosis of tricuspid insufficiency, absolute reliance should, in my opinion, be placed upon the venous pulse when present. On the other hand, with characteristic symptoms of tricuspid insufficiency, the diagnosis of simultaneous mitral insufficiency is assured if the systolic murmur is heard dorsally.

TRICUSPID STENOSIS.

THIS is a rare condition, occurring as a congenital and an acquired disease with about equal frequency. As a primary, independent disease tricuspid stenosis is very rare, being usually seen in association with organic disease of the left side of the heart. The lesions of mitral and tricuspid stenosis are observed to be combined most frequently,

while those of tricuspid stenosis and aortic insufficiency coexist less frequently. The morbid changes are practically identical with those of mitral stenosis, the right auricle becoming dilated, and this being followed by general venous stasis. The right ventricle, however, is usually hypertrophied, owing to the obstruction in the pulmonary circulation that results from the combined valvular deficiencies.

Special Etiology.—The fact that mitral and tricuspid stenosis frequently have a common cause, acting concurrently, can scarcely be doubted. Judson Daland and E. L. McDaniel, who have collected 186 cases of associated mitral and tricuspid stenosis, believe that most of them occur in hearts overdistended as the result of attempts at compensation, after acute endocarditis and simple mitral disease. *Rheumatic antecedents* are furnished by the history in from 30 to 40 per cent. of the cases of tricuspid stenosis. As in mitral stenosis, *sex* is a potent factor, the statistics of Bedford, Fenwick, Herrick, and of Leudet (which embrace a total of 160 cases) showing a ratio of 5 to 1 in favor of the female sex.

Symptoms.—These are those of the combined affections—venous stasis, marked polycythemia, and dropsy, particularly hydrothorax.

Physical Signs.—*Inspection* may reveal a feeble venous pulse in the jugulars, due to right auricular systole, hence presystolic in time. *Palpation* may detect a presystolic thrill over the body of the right ventricle. *Percussion* may reveal the enlarged right auricle. *Auscultation* gives usually a presystolic rolling murmur, which is best heard over the lower sternum and along its right border. The above physical signs are to be relied upon in *uncombined* cases, which are exceedingly rare. On the contrary, it is difficult in the extreme to differentiate the signs of tricuspid stenosis from those of the lesions with which it is almost uniformly associated—viz., mitral stenosis and aortic insufficiency.

PULMONARY INCOMPETENCY.

(*Pulmonary Regurgitation.*)

THIS is an exceedingly rare complaint that results from acute (malignant) or chronic endocarditis after birth; it is also rarely due to a congenital malformation. In the latter form union of two of the segments is often observed; in the former, the usual sclerotic processes, with the occasional adhesion of one or more segments with the pulmonary artery wall, may be noted. The effect of the lesion is to cause hypertrophy and dilatation of the right ventricle. The *physical signs* furnish no diagnostic characteristics. There is developed a diastolic murmur which is most audible in the second left interspace, and is transmitted to the lower sternal region, simulating the murmur of aortic regurgitation. The water-hammer pulse and marked hypertrophic dilatation of the left ventricle are present in the latter complaint, however, and are absent in pulmonary regurgitation. In pulmonary insufficiency, on the other hand, hypertrophy and dilatation of the right ventricle ensue. Preble reports a case of *relative insufficiency* of the pulmonary cusps (the so-called Graham-Steell murmur); at the autopsy aortic and mitral insufficiency were also found.

PULMONARY STENOSIS.

A QUITE frequent form of *congenital malformation* of the heart is the narrowing of the pulmonary orifice. In the rarest cases it is of *post-natal* date, and may result in induration, contraction, and fusion of the segments. In one of Osler's cases the orifice "was only two millimeters in diameter, with vegetations of acute endocarditis on the segments." I saw one case in which the pulmonary artery near the valve was contracted to one-half its normal caliber. *Myocarditis* with resulting contraction of the conus arteriosus may cause pulmonary stenosis, and some of the cases that originate during adolescence and later in life are due to *atheromatous* change, while others possibly are the result of *chronic endocarditis*, direct *violence*, and *ulcerative endocarditis*. The lesion is compensated by an hypertrophy of the right ventricle, following which dilatation and tricuspid incompetency may appear.

Symptoms.—*Cyanosis* and *distention of the systemic veins* are observed.

Physical Signs.—A systolic *thrill* may be felt at times over the base. There is considerable enlargement of the right ventricle, as elicited by *percussion* and *palpation*, and a *systolic murmur* is audible, its greatest distinctness being, as a rule, in the third left space near the sternum. It is *harsh*, superficial, and transmitted a short distance upward and to the left. Occasionally this murmur is heard best at the aortic valve, but it is never conveyed to the vessels of the neck, and hence is easily distinguished from the aortic systolic murmur. Its harsh character and loudness would serve to obviate confusion with *functional* or *anemic murmurs* that are sometimes heard here. The pulmonic second sound is weak, and, not rarely, there is a diastolic murmur of the same character, indicating *pulmonary regurgitation*. Broadbent asserts that a *temporary* systolic murmur due to severe exertion may be observed, and I have noted a systolic murmur in the pulmonary area in young adults of remarkably vigorous build and unusual endurance. A careful review of the literature indicates that stenosis of the pulmonary artery predisposes to lung tuberculosis. The conditions are not favorable to healthy nutritive processes, especially of the lungs.[1]

COMBINED FORMS OF CARDIAC DISEASES.

IT may be asserted safely that in more than one-half of all cases combined lesions or murmurs are exhibited before the fatal termination. As already stated, stenosis of an orifice when due to valvular disease is associated with incompetency of the corresponding valve. Thus aortic stenosis is constantly combined with or followed by aortic incompetency, and in like manner mitral stenosis by mitral incompetency. The association may also have reference to lesions at two or more different valves. In

[1] *The Amer. Jour. of the Med. Sci.*, Jan., 1902, by the writer.

the table of F. J. Smith, the relative frequency of the chief murmurs found in combination is as follows:

Aortic diastolic and systolic and mitral systolic,	16.55 per cent.
Aortic stenosis and mitral stenosis,	6.12 "
Aortic diastolic and mitral systolic (common in children),	5.21 "
Aortic diastolic and systolic and mitral presystolic and systolic,	3.77 "

When two lesions coexist at the same valve, the one may compensate, in part at least, for the other, as, for example, in the case of aortic stenosis in association with aortic regurgitation. Here the stenotic deficiency lessens the reflux current from the aorta into the left ventricle during the diastole; hence the latter receives a correspondingly diminished amount of blood. During the contraction of the ventricle the distending force in the aorta is diminished, both on account of the narrowing at the aortic orifice and the relatively lessened contents of the hypertrophied ventricle. Similarly, in dominating mitral incompetency an associated mitral stenosis by lowering the strength of the regurgitant current renders the conditions more favorable. Relative insufficiency at the mitral valve, following aortic insufficiency, may prove salutary by preventing over-distention of the left ventricle, and also the over-filling of the arterial tree and the possible rupture of a blood-vessel. On the other hand, when mitral incompetency is secondary to aortic stenosis, the latter defect may hasten the unfavorable tendencies in the former.

Relative tricuspid incompetency, secondary to mitral disease, usually results in the development of a serious impediment to the systemic venous circulation, and if it occur in the course of diseases of the aortic cusps, an early fatal termination is reached. In advanced mitral disease a slight leakage at the tricuspid valve may be the means of obviating disastrous consequences to the right ventricle in case of undue strain.

Physical Signs.—These are confusing, but a systematic analysis often leads to the correct inference. That one of the valvular lesions predominates over all others is a fact of paramount importance for the solution of these cases. The chief lesions can usually be determined by noting the *seat*, the area of transmission, and the character of the most pronounced murmur. More important still is the *correct timing* of any murmurs that may be audible. When a murmur occupies both the aortic and mitral areas the student will note two points of maximum intensity, and that each grows weaker as the stethoscope is moved toward the mid-præcordial region. The secondary alterations in the heart frequently coincide with the predominating murmur, and observers should recollect that mitral murmurs are often secondary to aortic, and that tricuspid murmurs point to accompanying mitral disease. In children, however, rheumatic endocarditis often affects both valves on the left side of the heart. Unquestionably, a single observation of these cases, however carefully made, is often profitless.

Complications of Valvular Disease.—Most of these have already been spoken of at sufficient length, but to restate them collectively in this connection may prove useful to the student and physician. They are—(1) acute endocarditis (including the ulcerative form); (2) acute pericarditis; (3) pleurisy; (4) pneumonia; (5) nephritis,

followed by uremia; (6) local or general arterial sclerosis; (7) chronic gastric or intestinal catarrh with intercurrent acute attacks; (8) embolic processes; (9) angina pectoris; (10) edema of the lungs; (11) hysteria, neurasthenia, epilepsy, and insanity; (12) rupture of the skin of the extremities in consequence of excessive edema, with erysipelatous inflammation; (13) febrile paroxysms, accompanied not rarely by synovitis, occur at varying intervals of time, and are due to various causes, as rheumatism, simple, acute, and ulcerative endocarditis, and pericarditis.

Course and Duration.—When valvular disease consists in rupture of a segment the course is brief and usually proves quickly fatal. Apart from these exceptional instances the duration is measured by months, or more often by years or even decades. Statements applicable to all cases cannot be made, however, owing to the wide differences in different cases. Among the circumstances affecting the duration I would mention in particular the patient's mode of life, the hygienic conditions under which he lives, his occupation, mental condition, and the severity of the morbid processes. Every experienced physician has doubtless met with a small class of cases that have terminated fatally in from six months to a year, having developed in that period all of the serious phenomena and complications of the more chronic forms of organic heart-disease. In the preponderating proportion of cases, however, the course is exceedingly slow, and often cases have existed many years before they have finally been recognized. In numerous instances the patient follows his usual vocation, which may even be laborious, for years, and without discomfort. In other cases the symptoms, as dyspnea on exertion, are so slight as not to excite suspicion. Facts such as these render it obvious that while the period of compensation is long, its exact limits are indeterminable.

The *progress after failure of compensation* is more definitely known, since frequent opportunities for observation are afforded. At this time the cases also exhibit wide differences in duration; in my own experience they have varied from two or three months to as many years (rarely even longer), depending much on the patient's mode of living. The course may be shortened by severe external injury, intercurrent acute illness (especially febrile disease), vicious habits, straining efforts, and the like.

Prognosis.—The detection of a cardiac murmur should not alone lead to a gloomy prognosis. Says Osler: "With the apex-beat in the normal situation and regular in rhythm, the auscultatory phenomena may be practically disregarded." Individual cases require separate and careful consideration. It is well not to advance positive assertions until all the circumstances that may influence the prognosis of any given instance have been well weighed. Observation of a case for some weeks and months enables the physician to speak with greater confidence and knowledge concerning the probable outcome. Prior to the occurrence of disturbances of compensation the prognosis is measurably favorable. After this pivotal event the prognosis as to life becomes wholly unfavorable in direct proportion to the extent of the degenerative changes of the myocardium. Disturbances of compensation that are attended with marked arythmia, urgent dyspnea, and general dropsy may admit of complete relief. Later, restoration of the balance of forces becomes only partial, and finally the above-

mentioned symptoms become more pronounced; Cheyne-Stokes' breathing may then develop, and after a prolonged and distressing struggle for breath the patient succumbs. Death may also occur suddenly from cardiac paralysis. Among ominous and yet common *complications* and intercurrent affections may be cited again extensive edema of the lungs, pneumonia, marked arteriosclerosis, embolic processes, ulcerative endocarditis, acute endocarditis, obstinate gastritis, and nephritis. On the contrary, *favorable indications* are sound general health, good external conditions (absence of poverty, hunger, etc.), strong and regular action of the heart, absence of arteriosclerosis, of excessive hypertrophy, of syphilis (unless recognized early), and of rheumatic antecedents, as well as any vices of life. *Age* influences the prognosis to some extent. In children under ten years the lesions are usually somewhat more rapidly progressive than in adults, and the compensatory hypertrophy is developed with corresponding rapidity; hence the period of failing compensation is reached earlier. This may be said to be a broad general rule, and I have found that it is one to which there are many exceptions. Among other reasons for the more gloomy prospect when heart disease occurs in young children are the following: the mitral valve is generally implicated, the liability to rheumatic intercurrences is great, and there is a greater tendency to overtax the reserve cardiac power by violent forms of exercise. After the twelfth year the prognosis becomes more favorable. *Sex* is also a modifying prognostic factor, women bearing valvular lesions better than men, apart from the influence of childbearing, though even this is an influence the significance of which has been greatly magnified by many writers. To explain the more favorable outlook in women we have two main facts—viz., a less laborious as well as a more quiet life, and a diminished liability to arteriosclerosis and involvement of the coronary vessels. The particular valve involved has some influence on the prognosis.

Aortic regurgitation gives a fairly good prognosis in those cases that begin in early adult life, and in which the second sound in the neck is not abolished, granting that the patient regulates wisely his manner of living. A long, loud murmur indicates a strong heart with slight leakage. When the lesion is due to acute endocarditis, the prospect of life is better than when it originates in degenerative changes. A chief danger arises from associated arterio-sclerosis—a frequent occurrence, particularly in advanced life—and from implication of the coronary arteries. Much depends upon the condition of the latter vessels. When their lumen is narrowed, starvation of the heart-muscle quickly ensues, followed by myositic degeneration. Blocking of one of the branches of the coronary artery is the most frequent cause of sudden death in this affection. After failure of compensation, the prognosis is less satisfactory by far in aortic regurgitation than in mitral regurgitation, since restoration of compensation is not as readily accomplished in the former as in the latter variety. Aortic regurgitation stands first among valvular affections in the order of gravity (Broadbent). In *aortic stenosis* favorable predictions are warrantable when the disease is uncomplicated. When the left ventricle gives way, the condition is serious. Osler states that the rheumatic form of early life is more serious than the late sclerotic variety. The size of the radial

artery is proportionate to the size of the blood-stream, hence indicative of the degree of stenosis.

Mitral regurgitation, when a primary lesion, is propitious, except in the very young, and not infrequently the progress of the morbid process is apparently arrested. In a considerable proportion of cases the disease does not materially shorten the life of the sufferer. In a larger percentage, however, there is special liability to a renewal of the causative affections (*e. g.*, rheumatism) and to pulmonary conditions of serious import, producing exacerbations and permanent aggravations of the disease. The gravity of these intercurrent complaints is also increased by the existence of the cardiac lesion. If a good first sound is audible as well as the murmur, it is of good prognostic significance. Failure of compensation at once renders the prognosis decidedly unfavorable. In *mitral stenosis* compensation of the right heart fails somewhat earlier than in mitral insufficiency, and hence the accidents and conditions referable to the lung (diffuse pulmonary apoplexy, edema) are not so long delayed as in the latter disease. In my experience mitral stenosis is better borne by women than by men, and better during adolescence and early adult life than during more advanced years. The congenital forms are comparatively benign. Mitral stenosis causes sudden death more frequently than any other form of organic disease of the heart except aortic regurgitation. *Tricuspid incompetency*, whether secondary to disease of the lung or of the left side of the heart, is grave; it is extremely serious when it arises in the course of aortic incompetency. It is usually indicative of dilatation following hypertrophy of the right ventricle. Compensatory hypertrophy, however, can be re-established repeatedly.

Treatment.—This falls naturally into three subdivisions: (1) Prophylaxis; (2) management during the stage of compensation; (3) treatment of the stage of non-compensation.

(1) **Prophylaxis.**—The statistics of Sibson show that complete rest and protection of the surface during an attack of acute articular rheumatism lessen the average percentage of cases in which acute endocarditis develops. When the latter complication occurs in acute rheumatism the patient should keep to his bed for some time after all rheumatic symptoms have disappeared (two to six weeks) or until the improvement in the cardiac condition has ceased absolutely. This precautionary measure will often lessen the extent of the ensuing chronic endocarditis, and also increase the proportion of perfect recoveries. When the physician is cognizant of hereditary predisposition to organic heart disease, or has to deal with the arthritic diathesis (gouty or rheumatic) or the alcoholic habit, he can frequently, by timely advice and hygienic suggestions, direct his patient to adopt measures that will obviate the occurrence of valvular disease. Systematic treatment of syphilis would greatly lessen the incidence of valve disease. All persons predisposed by heredity or otherwise should be told of the probable effect of muscular strain (*e. g.*, competitive sports), alcohol, and other exciting factors; too often, however, when he first sees his patient the physician is confronted by an incurable malady.

(2) **Management During the Stage of Compensation.**—Three main objects are to be accomplished: (*a*) The avoidance of every agency that tends to aggravate or maintain the lesion or lesions. Under this head

the detection and removal of all causal factors is imperative. Thus, if the patient's vocation entails undue muscular effort, it must be abandoned; violent exercise, as running up flights of stairs, heavy lifting, or straining at stool, is dangerous and must be prohibited. If alcohol has been a factor, it must be discontinued; if syphilis, it must be treated specifically. A rheumatic or gouty taint must be overcome as far as possible by special measures. Fatigue and exposure must be avoided, particularly if the patient be young. Emotional excitement and mental overexertion injuriously affect the cardiac lesion; therefore tranquillity of mind should be insisted upon, though moderate and systematic mental exercise has no risks for the patient. In the case of children at school careful supervision of their studies as well as of their recreative exercises is essential. Fright and sudden emotion must be avoided if possible. The use of tea, coffee, and tobacco should be rigidly prohibited. In mitral disease, bronchitis is to be especially guarded against.

(*b*) The *diet* of the patient demands careful regulation. Only a moderate amount of food, composed for the most part of readily digested albuminous articles (milk, eggs, light forms of meats), green vegetables and stewed fruits, is to be taken, since overloading the stomach will disturb the action of the heart; particularly is this true at night. The carbohydrates may be allowed, but only in limited quantities, since they are apt to decompose and form gases that distend the stomach and intestines. The coarser and more indigestible food-stuffs should also be avoided. The amount of liquids taken should not exceed the actual requirements of the patient, inasmuch as overfilling of the blood-vessel system increases the work of the already overburdened cardiac forces. Alcoholic beverages should not be used, as a rule; but if the patient has been moderate in the use of alcohol, and particularly if he be advanced in years, light wines may be allowed in small quantities to aid digestion.

(*c*) *Carefully regulated exercise* is beneficial, but it must be gentle and should be taken out-of-doors. A good general muscular development is an aid of no mean value to the conservative powers of the heart. Oertel, with a view to assisting the compensatory forces of the heart, has recommended graduated physical exercise; he advises that patients be instructed first to ascend low elevations, and with increased endurance, mountains of a considerable height, the object being to bring about full compensation. This method, however, has been found to be inapplicable to a large percentage of cases. Cardiac distress, palpitation, and dyspnea are complained of by this large group of patients if other than the gentlest forms of exercise be undertaken. With respect to exercise, then, the sensations and experiences of each patient must be consulted before the physician can advise judiciously. Woolens should be worn next to the skin during both the warm and cold seasons. The skin should be kept clean by daily sponge-baths, followed by friction of the surface. Thus the nutrition will be improved and the liability to intercurrent attacks of bronchitis lessened. The bowels should be moved each day, and usually the use of stewed fruits suffices to accomplish this end; if not, salines, as Rochelle or Carlsbad salts, and the bitter waters (Friedrichshall, Hunyadi-Janos) must be brought into requisition. In winter a warm climate may prove advantageous, though long journeys are often

illy borne, owing to the fatigue induced thereby. If the patient be anemic or his nutrition is notably impaired, a suitable change of air,[1] or the use of hematinics, arsenic, small doses of mercury, and cod-liver oil, is to be recommended. Digitalis should not be employed when compensation can be preserved in other ways. We should train the heart up to the amount of work required of it (Brunton).

(3) **Treatment of the Stage of Non-compensation.**—The principal object to be kept in view in this stage is the reinvigoration of the exhausted cardiac muscle, and thus to relieve the impeded circulation. *Sudden death* may, though rarely, occur from the blocking of a branch of the coronary artery or from acute dilatation. Failure of compensation, however, *begins gradually* as a rule, the condition often existing without marked or characteristic symptoms; but its early recognition is important from the stand-point of therapy. Increased dyspnea on exertion, and nocturnal seizures of shortness of breath and irregular action of the heart (*arrhythmia*), are among the earliest clinical features. The latter symptom may have been present before, particularly during active exercise in mitral disease, but is now more marked, and may be constant. The patient's nutrition often suffers, and he is pale and rather feeble. Absolute quiet, liberal feeding with suitable food, and iron may in a little while restore the impaired cardiac tone. If this treatment fails, by the end of a fortnight a small dose of digitalis should be exhibited (5 minims—0.333—of the tincture three times daily); this should be promptly withdrawn upon the disappearance of the symptoms. *Decided indications* of lost compensation are marked dyspnea and arrhythmia; the canter rhythm; an irregular, small, compressible pulse; and cyanosis, with or without the presence of dropsy. The object now is the maintenance of the blood-pressure at an adequate height by the following means: (*a*) *Absolute rest in bed* This diminishes greatly the work of the heart, and thus enables it to regain largely its former vigor. Rest joined with massage, careful yet liberal feeding and attention to the bowels will often restore disturbed compensation in from one to two weeks. In a considerable number of cases treated at the Medico-Chirurgical Hospital this method succeeded admirably.

(*b*) *Cardiac stimulants and tonics.* Of these the most important is digitalis. By stimulating the pneumogastric, by increasing the blood-supply to the heart-muscle, by causing the systole to be more complete and the period of diastole to be lengthened, digitalis becomes an invaluable aid to the nutrition of the cardiac muscles. In addition, the heart contracts more regularly and the blood-pressure is raised. As a result of the use of this drug the tissue calls upon the cardiac forces from the outlying portions of the body are satisfied and the reserve energies of the heart-muscles are maintained.

In *mitral disease* the influence of digitalis is most beneficial, the pulse becoming slower, of better tension and more regular, while the urine increases in amount. In mitral incompetency its good effects are ascrib-

[1] Observation and experience have confirmed my belief that sea-air during the warm season and high altitudes at all times are injurious in their effects in valvular disease of the heart.

able in part to the powerful contractions of the left ventricle, whereby the blood-stream from the ventricle to the aorta is greatly increased. On the contrary, the patient's condition is occasionally aggravated by the drug, because "the leak is increased as much as the normal flow" (Hare). Digitalis exercises its most beneficial influence by rendering the systole of the right ventricle more energetic, the blood-pressure being raised in the pulmonary circuit and left auricle; this fills the left ventricle better during diastole and "resists reflux through the mitral orifice in the systole" (Broadbent). In mitral stenosis digitalis, by lengthening the period of diastole, allows time for the blood to pass from the auricle through the narrowed mitral orifice into the ventricle. Slight toxic effects may sometimes result from digitalis, the pulse becoming thread-like and irregular, and the urine scanty. Under these circumstances the drug should be discontinued.

In *aortic regurgitation* digitalis exercises a beneficial effect in cases dependent on chronic valvulitis: the theoretic view, however, that by prolonging the diastole digitalis causes overfilling of the left ventricle rests on too slender a foundation to be regarded as a valid objection to its use. It may, however, produce excessive hypertrophy, in which case it should be withheld. When atheroma is associated, and especially if relative insufficiency at the mitral orifice has not as yet supervened, digitalis produces harmful effects. After secondary mitral insufficiency is developed, this drug may be employed, but it should be guarded by nitroglycerin when marked arterio-sclerosis coexists, to counteract the musculo-arterial contraction caused by the digitalis, and thus diminish arterial resistance. Digitalis is also powerless and probably harmful in proportion to the extent of fatty and fibroid degeneration of the myocardium. In aortic regurgitation, nausea and vomiting sometimes follow the administration of digitalis; when this is the case it should be stopped. When secondary dilatation comes on in aortic stenosis, digitalis is needed to increase left ventricular power. The dose is to be calculated according to the degree of cardiac exhaustion. When tricuspid incompetency is secondary to mitral disease, striking results are obtained from the use of digitalis (*supra*); but when it exists alone—*e. g.*, following emphysema or cirrhosis of the lung—digitalis often fails. The cardiac contractions, if they have previously been irregular, may become somewhat more regular, but the precordial distress will often be increased, while the circulatory disturbance, as evidenced by the objective signs, will remain unrelieved. If *dropsy be slight* or absent, 2 to 3 drams (8.0–12.0) of the freshly prepared infusion, three or four times daily, will suffice. If symptoms of *decidedly unfavorable import* be present, including marked dropsy, the dose should then be larger (of the infusion, ℥ss—16.0, every two or three hours) for two or three days, when the dose must be diminished or given at longer intervals. Quantitative estimations of the urine should be made during the use of the drug, and if the effect be good, the daily amount will often be greatly increased; if bad, there will be a diminution rather than an increase in the amount. There are not a few patients in whom the symptoms of commencing failure of compensation recur as soon as the drug is discontinued. To such, digitalis may be administered continuously or until toxic symptoms are manifested.

I believe that the solid preparations (powdered leaves and extract) can be taken for longer periods than the liquid forms without exciting untoward symptoms. This suggestion should be followed, particularly in cases that are seen at long and irregular intervals of time. With this exception, it should be a cardinal rule to discontinue the digitalis when the symptoms of disturbed circulation have vanished. When it fails of its effect or is not well borne, and when, as often happens, the arrhythmia is not favorably influenced by it, the physician is compelled to resort to other cardiac stimulants. These are numerous, and, whilst their effects are not comparable to those of digitalis in every respect, some of them seem to meet certain indications that are not met by this drug. Among the more important are nitroglycerin, strophanthus, convallaria, strychnin, cocain, spartein, and caffein. Nitroglycerin in small doses is at the same time a cardiac stimulant and an arterial relaxant, and hence is more often useful in aortic than in mitral valvular disease. In larger doses, when left ventricular hypertrophy is excessive, as may occur when general arteriosclerosis is associated with aortic regurgitation and also (though rarely) aortic stenosis, it is highly useful, widening the blood-paths, and causing less powerful contractions of the heart. Strophanthus should be employed in instances in which digitalis must be interrupted, since the action of these two remedies upon the heart-walls is very similar. The tincture is usually employed, the dose (varying with the indications of each case) being from 4 to 10 minims (0.266–0.666) every three or four hours, and in controlling the irregularity or intermittency of cardiac action it is sometimes better in its influence than digitalis. Many cases of marked arrhythmia will not yield to either when but one is given; and in such I have occasionally obtained good results from digitalis and strophanthus in combination. Another drug which is an excellent stomachic tonic, and one that may be employed as a substitute for digitalis, is convallaria. It should be stated that, rarely, strophanthus, like digitalis, does harm rather than good, being sometimes badly borne by the stomach. Under these circumstances I have employed the following combination:

℞. Caffein. citrat., ʒss (2.0);
Strychninæ sulphat., gr. $\frac{1}{3}$ (0.021);
Spartein. sulphat., gr. iij (0.193).
Ft. capsulæ No. xii.
Sig. One every three or four hours.

The above prescription is not only a good heart stimulant, but also a good diuretic. Caffein citrate is superior as a diuretic. Spartein is a potent diuretic and heart stimulant in doses of gr. $\frac{1}{6}$ to $\frac{1}{2}$ (0.01–0.032) every four to six hours, and is especially serviceable when dropsy as a symptom and nephritis as a complication exist. Strychnin, given hypodermically in full dose, gr. $\frac{1}{30}$ to $\frac{1}{15}$ (0.002–0.004), is the most efficient cardiac stimulant known to medical science. It should be employed in this manner in sudden failure of heart power. Given in doses of average size, its effects in chronic valvular disease are not striking. Atropin may be advantageously combined with it.

When the indications are urgent and the above agents are not available, diffusible stimulants, as ether or ammonium, may be used until more

suitable remedies can take effect. Cocain simulates strychnin in its action. The dose is gr. ¼ (0.016) every four hours, and the drug may be given with digitalis in pill-form. Later, systemic tonics are often demanded by the *anemia* and other constitutional indications, and here iron and quinin should be joined with strychnin. Unquestionably, the value of iron in full doses as an aid in the completion of the work of restoring broken compensation has been and is still scarcely appreciated by the profession at large. When iron disagrees, arsenic may be given instead. In many cases of failure of compensation the restoration of the balance of the cardio-systemic circulation can be greatly assisted by depleting the over-filled venous system. There are two ways of attaining this end:

(*a*) *Venesection.*—When the right heart is over-distended, as shown by its very feeble efforts at contraction, and the whole venous system is intensely engorged, as shown by marked cyanosis and orthopnea, bleeding directly from a vein is not only warrantable, but often imperatively demanded in order to save life. From 16 to 30 ounces (473.0–887.0) may be removed safely, and the heart's action will almost immediately be observed to grow stronger and more regular, and the pulse fuller and of better tension. As before intimated, the form of dilatation of the right ventricle that follows emphysema is disinclined to yield to digitalis. In such instances, following the suggestion of Osler,[1] I have obtained brilliant results from free bleedings.

(*b*) *Depletion by purgation* affords less pronounced relief to the heart, though it is of the greatest value in cases in which a moderate grade of cyanosis and dropsy exist. As in the case of venesection, a feeble, irregular pulse is not a contraindication to the use of purgatives, since the latter remove directly a considerable portion of the heart's burden. The purgative to be used will vary with different cases. I select at the outset Rochelle or Epsom salts, employing them after the method of Matthew Hay—*i. e.* from 1 to 2 ounces (32.0–64.0) of Rochelle or 1 to 1½ ounces (32.0–48.0) of Epsom salts, in concentrated solution, to be given from a half to one hour before breakfast. Watery evacuations (three to six in number daily) usually follow the administration of the saline; but, unfortunately, one meets with many patients in whom it produces symptoms of marked catarrhal irritation. Next to salines, the most satisfactory results have been obtained from the use of elaterium; I often combine this with podophyllin and belladonna. I have never seen good results from the use of mercurials when the object has been to procure venous depletion, but they are of service in dropsy, and particularly in ascites.

Schott of Nauheim has introduced a special treatment by baths and resistance movements that is applicable to most forms of valvular disease, simple dilatation, and nervous affections of the organ. The beneficial effects are principally attributable to the salt and the carbon dioxid, which act as cutaneous stimulants. Greene[2] regards the warmth and moisture as the important features. Twenty-one baths are given in one month, dropping one every fifth, fourth, third, and second days. The water contains sodium chlorid, calcium chlorid, and carbon dioxid, and the temperature ranges from 82°–95° F. (27.7°–35° C.). The first bath lasts seven or eight minutes; the time is then gradually

[1] For illustrative cases from Prof. Osler's wards, see article by Leufler, *Medical News*, July, 1891.

[2] *Jour. Amer. Med. Assoc.*, Oct. 15, 1898.

lengthened, the temperature lowered, and the carbon dioxid increased. After the bath the patient is rubbed and allowed to rest for an hour.

Artificial Nauheim baths are successfully employed in certain American hospitals at the present time. They are prepared as follows: Five pounds of sodium chlorid and eight ounces of calcium chlorid are dissolved in one half bath (30 gals.—114 liters), the temperature of the water being 95° F. (35° C.). In a few days the bath is charged with carbon dioxid by adding sodium bicarbonate (1 lb.—453.6) and HCl (½ lb.—226.8), the latter just before the bath is taken. The effects are to lower the pulse-rate, to decrease the size of the heart, to stimulate the nerves, and, indirectly, the cardiac nutrition. There is also a tendency toward improvement of the nutritive processes and an increase of the urine.

Gentle resistance exercises (consisting of all the more reasonable movements that a person naturally makes, and resisted by an attendant) form an important element of the treatment, since they tend to stimulate the muscles and nerves and propel the blood from the congested veins. The Nauheim treatment is not suitable in aortic regurgitation, aneurysm, advanced arteriosclerosis, marked dropsy, or fatty degeneration of the heart, although the movements alone are beneficial in these conditions and may be employed without the baths.

Individual symptoms frequently demand special treatment.

(1) *Dyspnea and Orthopnea.*—When these phenomena are caused by engorgement of the pulmonary vessels, the cardiac stimulants above detailed usually afford relief. Frequently the patient cannot lie down, in which case a suitable bed-rest often gives immediate comfort and support. For the severe attacks of nocturnal dyspnea (amounting sometimes to orthopnea), particularly when accompanied by cardiac palpitation, the subjoined formula has proved itself of great benefit:

℞. Sodii bromidi, gr. xv (0.972);
Tr. opii deod., ♏ x–xv (0.666–0.999).—M.
Sig. To be taken in one dose at bed-time.

In the late stages of heart-disease morphin, given hypodermically, is to be preferred in combating this symptom, and is entirely free from the usual objections to the habitual use of the remedy. Its influence for good is inestimable. Dyspnea may also be produced by associated bronchitis, edema, emphysema, and hydrothorax—conditions that must be treated according to the customary rules. Frequent physical explorations of the chest should not be omitted. Hydrothorax demands aspiration, and this repeatedly in some instances.

In valvular disease (particularly aortic), owing probably to coronary arterio-sclerosis, paroxysms of severe dyspnea (*cardiac asthma*) are apt to arise. These are best overcome by nitroglycerin in ascending dosage in combination with sodium bromid at bed-time, to be repeated as needful. His lauds the Karell "milk cure," which is a strict diet of 800 to 1000 c.c. of milk per day for a period of five or six days; it should be carried out only in bed patients.

(2) *Cough.*—Cough is common after failure of compensation, and is due to bronchitis resulting from stasis in the pulmonary vessels. In mitral disease it may come on before the rupture occurs. Beyond the treatment directed to the causal condition (the cardiac failure) nothing

is needed to relieve the cough. These subjects, however, are prone to suffer from catarrhal bronchitis due to cold, and this impairs the compensatory mechanism. J. Weiss extols heroin in cases not relieved by the ordinary remedies.

(3) *Hemorrhage* may take place, and generally from the lungs, though it may also proceed from the nose, stomach, bowels, or uterus. In a recent case of double aortic disease and relative mitral insufficiency hemorrhages occurred from the bowel with apparent relief to the patient. The hemoptysis, which is an accompaniment of mitral lesions, is rarely excessive, and is probably always beneficial. I would advise against active treatment unless the hemorrhage is actually copious in amount.

(4) *Palpitation* may be due to different causes, the recognition of which in each case is important. At times undue hypertrophy maintains a constant throbbing and distress in the precordial region, the condition being distinguished by the strength of the impulse and by the full, tense pulse at the wrist. Palpitation is best met by the use of the tincture of aconite, ♏ j–iv (0.066–0.266) every four hours. With the aconite I frequently associate the bromid with excellent effect. An ice-bag to the precordia is worthy of recommendation. Unless the patient's discomfort is significant, however, this symptom does not call for active treatment. The administration of a saline purge not infrequently serves to quiet the heart. The patient may suffer from pure nervous palpitation, in which case the diet and the condition of the stomach must be carefully looked to, while for the throbbing the bromids of ammonium and sodium, together with preparations of valerian, are the most reliable.

(5) *Anginose Pains.*—These are seen in aortic incompetency accompanied by sclerotic vessels, and also in mitral stenosis. When dependent upon rigid blood-vessel walls nitroglycerin should be tried; if the attacks be severe, amyl nitrite by inhalation deserves a trial. this failing, morphin and atropin may be employed hypodermically. Local measures alone are sometimes sufficient when the pain is only moderately intense, and the ice-bag or Leiter's coils may be tried. The sedative effect of a blister (4 by 6 in.—10 by 15 cm.) has more often proved effectual in my experience, though its use should be limited to patients whose general strength is not materially impaired.

(6) *Pain* referred to the stomach, and less frequently to the abdomen also, occasionally assumes prominence and is relieved with great difficulty. It is dependent, in part at least, upon obstinate subacute gastritis, and I have quite recently seen an instance of the sort verified by autopsy. Among many drugs tested in this case, opium alone gave relief. Should this fail, however, carminatives in combination with some antiseptic agent, as salol or guaiacol carbonate, should be used.

(7) *Gastric Symptoms.*—Soon after compensation is broken the appearance of mild symptoms of catarrh of the stomach may be said to be the rule, and these yield to simple measures in addition to the cardiac stimulants and laxatives already indicated. But there are not a few instances in which such symptoms as gastric distress and uneasiness, constant nausea with frequent vomiting, particularly after food, occur, and assume a distressing phase. In such cases digipuratum deserves a

trial. These patients sometimes do well on the capsules before adduced composed of strychnin, spartein, and caffein. When the above remedies cannot be borne I employ hypodermically digitalin and strychnin or caffein citrate, the latter being made soluble by the addition of sodium benzoate in solution. Cases of this class reach an early fatal termination, as a rule. The symptoms may be partly due to gastric catarrh, and partly to uremic intoxication.

(8) *Nervous Symptoms.*—Insomnia and restlessness are almost constantly present at some period in the course of heart disease, and notably in the more advanced stages. The restiveness is rendered more distressing on account of hideous dreams and cardiac palpitation on awaking. For these phenomena stimulation often answers a better purpose than sedation. Hoffman's anodyne (℥j—4.0, well diluted), spirits of chloroform (♏xv—0.999), or ether (℥ss—2.0), taken in whisky (℥j—32.0) are serviceable. The elixir of ammonium valerianate is also of value. Recently, trional (gr. xv.) in combination with sodium bromid (gr. xx) has given satisfaction. Paraldehyd, veronal, medinal, and chloralamid are among the remedies of choice in the treatment of this symptom, but I have had no experience with their employment.

In the later stages there is no objection to the use of morphin hypodermically. Headache due to uremia may frequently be a troublesome symptom in connection with sleeplessness, and in such cases morphin is the remedy *par excellence;* it is to be supplemented by free purgation and cardiac stimulants. Should the right heart be found flagging, venesection may be practised.

(9) *Dropsy.*—As above pointed out, rest with attention to the diet and the judicious use of hydrogogue cathartics will often restore broken compensation. In the severe grades of failure of the balancing forces, complete rest, purgatives, and a cautious employment of morphin hypodermically, often suffice to remove the dropsy. If this method of treatment proves unsuccessful, then the therapeutic indications, so far as the dropsy is concerned, are for the use of cardiac stimulants, diuretics, and purgatives. Diaphoretics, particularly the hot-air and vapor baths, are not to be thought of, since they tend to depress the already weakened heart. While describing the action of digitalis as a cardiac stimulant, incidental allusion was also made to its action as a diuretic. In view of the fact that it raises the blood-pressure in the peripheral vessels and capillaries by contracting their walls, and because of its stimulating effect on the heart, digitalis in large doses becomes a most efficient diuretic in cardiac dropsy. When it fails, I have frequently found that a combination of strychnin, spartein, and caffein (*vide supra*) will excite free diuresis. Acet-theocin sodium may be tried in doses of 0.2 gm. (gr. iij) dissolved in water twice daily, to be increased to three or four times if necessary; it demands a kidney which is tolerably intact.[1] Nitroglycerin may also be prescribed, especially in cases presenting evidences of advanced arteriosclerosis. Diuretin has acted well in recent cases as a diuretic. When digitalis fails, Fraenkel uses strophanthin intravenously, giving as the initial dose 0.5 milligram and repeating only when the favorable effect is no longer apparent. An unirritating yet highly effective diuretic mixture in these cases is the following:

[1] *International Clinics*, vol. ii., 20th Series, p. 9.

℞. Potassii acetatis, ℥j (4.0);
Inf. digitalis, ℥ij (64.0).—M.
Sig. ℥ss (16.0) every three hours.

Purgatives are of the utmost value. Frequently, after a few copious watery evacuations as the result of the action of hydragogue cathartics, a free discharge of urine can be established, when before the latter event it has been impossible. Salines and elaterium, with podophyllin and belladonna, are agents that have been already recommended as purgatives (to deplete the venous system), and these should be first employed in the order named. Compound jalap powder may also be combined with the elaterium. A course of calomel, followed by salines until free catharsis is set up, is valuable from time to time. Mercury is especially applicable when the liver is much enlarged and ascites is a marked feature, or when the history of syphilitic infection is obtainable. It may be combined with cardiac stimulants and other diuretics as follows:

℞. Pulv. digitalis fol.,
Pulv. scillæ, āā gr. xij (0.777);
Mass. hydrargyri, gr. xxiv (1.555);
Ext. belladonnæ, gr. ss (0.0324).
M. et ft. pil. No. xii.
Sig. One every three or four hours.

When efforts at relieving the dropsy by means of medicines fail, then the most dependent parts of the body, or those most swollen, should be scarified under strict aseptic precautions. Fine silver trocars with rubber tubes attached (Southey's tubes) may be inserted and the liquid allowed to drain off in a gradual manner.

Means to Prevent Recurrence of Broken Compensation.—When the compensation has been successfully re-established, the after-treatment must be prosecuted with vigor for at least a year. The cause of the rupture of compensation is most probably fibroid and fatty degeneration of the cardiac muscle, and hence the mere restoration of the compensatory power of the heart does not imply a complete cure of the impaired muscular structure of that organ. Much can be done, however, to overcome the tendency to degeneration by the peristent use of hematinics and other tonics, as cod-liver oil and mercuric chlorid, the latter in small doses. I have obtained excellent results from the use of the following prescription in these cases:

℞. Liq. arsenici chlor., ♏xlviij (3.186);
Tinct. ferri chlor., ℥ss (16.0);
Hydrarg. chloridi cor., gr. ss (0.0324);
Elixir digestivi comp., q. s. ad f℥iij (96.0).—M.
Sig. ℥j (4.0) after each meal, well diluted.

This preparation may be taken indefinitely with occasional brief interruptions. The patient should lead a very quiet life, and follow rigidly all hygienic rules that tend to prevent the production of valvular disease. Appropriate *diet*, it should be emphasized, is not inferior to appropriate medication in its salutary effect. Should the faintest evidence of failure of the right ventricle manifest itself, the patient must be put to bed

and the foregoing treatment carried out. I am inclined to the view that the plan herein advocated not only renders the course of recurring attacks of failing compensation milder, but that, in a considerable proportion of the cases, the much-dreaded recurrence is thus prevented.

CARDIAC THROMBOSIS.

Pathology.—True cardiac thrombi are seen most frequently on the right side of the heart, in the auricular appendices, and, less commonly, in the right ventricle near the apex. They are of firm consistence, and are tightly adherent to the endocardium, considerable force being required to dislodge them. The color, while generally grayish-brown or red, varies with the age of the thrombus, being more colorless as it becomes older. Cardiac thrombi may be pedunculated or sessile, and their contour is, as a rule, more or less rounded. Recklinghausen and others have observed globular masses, the so-called "ball-thrombi," in the auricles, without the slightest endocardial attachment. They vary greatly in size, from a mustard-seed to a hen's egg, and sometimes exhibit calcareous degeneration. Cardiac thrombi may occur singly or in groups of considerable numbers. From the cavity in which they have their primary seat they may project into other chambers of the heart, or from the left ventricle into the aorta for a considerable distance. It is evident that fragments detached by the blood-stream from these cardiac blood-concretions will tend to lodge in various viscera and in the peripheral tissues, and set up embolic processes. The microscope shows degenerated round cells and detritus, but no pus-cells. Secondary degenerative changes, and later softening, may take place in the central portions of a thrombus, and these areas may contain a reddish-brown liquid.

Etiology.—The causes of cardiac thrombosis are to be found chiefly in some previously diseased or injured condition of the endocardium, though sometimes alterations of the blood constitute a factor of considerable importance. The condition may occur in the course of both acute and chronic diseases, in which the intracardiac conditions favor the formation of a blood-clot. Hence it is seen in connection with organic diseases of the heart in which the valvular and often the mural endocardium are roughened, and the obstructive and regurgitant lesions at the various valves cause retardation in the blood-current. Chronic obstruction in the lungs may contribute to the result by slowing the circulation in the heart. Cardiac thrombosis has been observed in many of the acute affections, and almost invariably there is a loss of endocardium, due to inflammatory action (endocarditis) at some point in the cavities of the heart. This becomes the seat of the fibrinous deposit which is subsequently imperfectly organized. Among the most important of these acute primary diseases are *rheumatism*, *diphtheria*, *lobar pneumonia*, and *pyemic* and *puerperal conditions*. It may be questioned whether, given a healthy endocardium, as contended by some writers,

the slowing of the circulation alone suffices to cause true cardiac thrombi.

Symptoms.—These will depend very much upon the rapidity with which the thrombus is formed, as well as upon its seat and dimensions. Thrombi invariably lack definiteness, and, as their effects are largely mechanical, signs of obstruction to the cardiac circulation and failure of the cardiac muscle are developed. The *pulse* becomes weak, rapid, and irregular; *dyspnea*, *vertigo*, and attacks of *syncope* are frequent; and later *cyanosis* may appear. It is probable that at times the liquefied products of a clot may be absorbed, producing blood-poisoning. When the thrombus is formed rapidly the symptoms are suddenly developed and the course is rapid. Rarely a valvular orifice, an efferent vessel, or the coronary artery may become blocked and instant death follow. Since the right heart is the most frequent seat of these thrombi, pulmonary embolism with its usual symptoms is a common event. When portions of a clot are broken off and swept into the systemic circulation, the clinical phenomena of cerebral, splenic, or renal *embolism* are exhibited. A localized gangrene of the foot has been described.

The **physical signs** consist of a feeble impulse with marked arrhythmia; the area of dulness is somewhat increased to the right, and often upward; and the heart-sounds are greatly enfeebled and quite irregular, with marked change in any murmurs that may previously have been audible. A presystolic murmur may be engendered.

Differential Diagnosis.—It is important to distinguish true cardiac thrombi, such as are above described, from the less dense and usually darker clots that are formed either immediately before or after death. The latter may seldom show an attempt at a very low grade of organization, and may present a somewhat decolorized appearance, but they do not adhere firmly to the endocardium. Moreover, *antemortem* and *postmortem* clots, as the latter may be appropriately termed, have a different causation from true thrombi. For instance, they are apt to form in diseases in which the fibrin-factors of the blood are greatly increased, as in pneumonia. Perhaps a more potent causal element is the progressive weakening of the heart-muscle, resulting in partial expulsion of the contents of the right ventricle; the blood that remains in the chamber is merely whipped up, and the deposition of its fibrin must thus be greatly favored. Such heart-clots may be generated if the endocardium be healthy, and cannot be separated positively from true cardiac thrombi by clinical observation.

The **prognosis** is uniformly bad and sudden death may be expected.

Treatment.—Beyond measures calculated to meet the symptomatic indications nothing can be suggested.

HYPERTROPHY OF THE HEART.

(*Hypertrophia Cordis.*)

Definition.—Hypertrophy is an increase in the muscular structure of the heart, evidenced usually by an increased thickness of its walls. It is almost invariably associated with dilatation of the chambers.

Pathology.—When the two processes—hypertrophy and dilatation—coexist, they cause great enlargement of the organ. To this condition the term "*eccentric hypertrophy*" has been given. Hypertrophy without dilatation receives the name "*simple hypertrophy*," and hypertrophy with diminution in the size of the cavities was formerly described as "*concentric hypertrophy*," but this term should now be regarded as obsolete, inasmuch as the condition is due to postmortem contraction of the ventricles.

The increase in size may affect the whole heart, one chamber on either side, one whole side, or but a single cavity (*general* and *partial hypertrophy*). The process may also be limited to a minute division of the heart (*circumscribed hypertrophy*). Owing to its important physiologic function the left ventricle is more frequently enlarged than the right, while the right auricle is more frequently involved than the left. The *weight* of the normal heart in a man of average size is approximately 9 ounces (255.0); in a woman it is 8 ounces (226.0). In bilateral hypertrophy, however, the weight of the heart may be greatly increased; hearts weighing from 15 to 25 ounces (425.0–710.0) are seen in moderate grades of hypertrophy, and those from 40 to 50 ounces (1134.0–1420.0) in extreme cases (*cor bovinum*). Measurements showing the thickness of the walls also indicate the degree of hypertrophy[1] and the exact seat of the enlargement when not general. The normal diameter of the left ventricular wall is from 8 to 12 mm. ($\frac{1}{3}$–$\frac{1}{2}$ in.); that of the right ventricle, from 5 to 7 mm. ($\frac{1}{5}$–$\frac{1}{4}$ in.); that of the left auricle, about 3 ($\frac{1}{8}$ in.), and of the right 2 mm. ($\frac{1}{12}$ in.). In cardiac hypertrophy the normal thickness of the various cavity-walls is usually doubled, not infrequently trebled, and, rarely, even quadrupled. In cases in which there is a concomitant dilatation the walls may appear thinned, while the measurement will show them to be in reality thickened.

The *shape* of the heart is also altered according to the seat and extent of the hypertrophy. If both ventricles are enlarged, the apex is widened and appears flattened; if only the left ventricle is involved, the apex is lengthened and is more or less pear-shaped; and if the right ventricle alone is hypertrophied (as in mitral stenosis), it may form the largest part of the apex, which will be less conical than in health.

The papillary muscles and columnæ carneæ are greatly thickened, and, particularly in the eccentric form of hypertrophy, they are often decidedly flattened. In this form the septum frequently shows increased thickness—a condition that I have never observed in simple hypertrophy. The muscular trabeculæ generally assume greater prominence on the right than on the left side. The muscular structure is usually of a deeper red color and also firmer than normally. The hypertrophied left ventricle can, as a rule, be lacerated readily, while the right, as first pointed out by Rokitansky, may be tough and leathery. As the heart continues to enlarge it sinks lower in the chest-cavity, owing to an increase in weight as well as in size. In hypertrophy of the heart there is a multiplication of muscular fibers, to which alone the enlargement of its walls is attributable.

Etiology.—Hypertrophy of the left ventricle (sometimes termed

[1] Measurements should not be attempted until the *rigor mortis* has been overcome by soaking the organ in water.

general hypertrophy) results from obstructions to the arterial circulation of whatever sort. These may be classified, according to their seat, into—(1) **Lesions of the Heart.**—(*a*) Aortic incompetency and aortic stenosis; (*b*) Mitral insufficiency; (*c*) The fibroid form of myocarditis; (*d*) Pericardial adhesions, particularly in the young. In such cases the adherent pericardium exerts a counter-traction force during the systole, and thus the work is increased beyond the capacity of the normal heart, with consequent hypertrophy. Late in life the heart may become atrophied.

(2) **Abnormal Conditions of the Blood-vessels.**—(*a*) Narrowing of the aorta—*e. g.* congenital stenosis, external pressure, and the development of an aneurysm; (*b*) General arterio-sclerosis, by raising the pressure; (*c*) Increased arterial pressure, due to contraction of the peripheral vessels in consequence of the local action of certain chemical and biologic irritants (lead, Bright's disease, gout, syphilis). Hassenfeld[1] has recently shown that hypertrophy of the left ventricle occurs only when the visceral arteries exhibit an extreme degree of sclerosis, or when the thoracic aorta is sclerotic. In cases of pure contracted kidney all the chambers of the heart are hypertrophied; but when extreme arterio-sclerosis is present also the left ventricle is disproportionately enlarged. In all of these cases, whether the blood-pressure is raised in larger or smaller vessels, increased cardiac action is essential to meet the demands of the system-circulation.

Attention should be called to the causes of the so-called "**primary idiopathic hypertrophy.**" The main causal conditions are—(1) Prolonged physical exertion, as in certain occupations (blacksmiths, locksmiths, draymen, and athletes). Excessive bicycling causes hypertrophy, particularly if arterio-sclerosis exists. (2) Constant over-distention of blood-vessels, as in the case of excessive beer-drinkers (*beer-heart*). Here the direct action of the alcohol upon the heart-muscle must also be taken into account. (3) Functional disturbances (neuroses), constant over-action of the heart, and even paroxysmal tachycardia, tea, coffee, tobacco, and alcohol may give rise to primary and general hypertrophy. Idiopathic hypertrophy of the heart is undoubtedly due to increased activity, which is dependent on a variety of irritating influences acting upon the heart muscle (De Domenicis[2]). *Primary congenital hypertrophy of the heart* is attributable either to circulatory disturbance (Simmonds[3]) or, as Virchow holds, to a diffuse myomatous neoplasia of congenital origin.

Hypertrophy of the right ventricle develops secondarily to any condition that offers obstruction to the pulmonary circulation or to the blood-current through the right ventricle. Among them may be mentioned—(1) mitral incompetency and stenosis; (2) emphysema, chronic bronchitis, collapse of a portion of the lung, contraction of a lung from pleural adhesions, and cirrhosis of the lung; (3) right-sided valvular lesions, particularly obstruction at the pulmonary orifice; (4) it is doubtful whether, on account of the normal situation of the right ventricle, pericardial adhesions induce hypertrophy of this chamber.

Hypertrophy of the Auricles.—Hypertrophy with dominant dilatation

[1] *Deutsch. Arch. f. klin. Med.*, Dec. 9, 1897; *Phila. Med. Jour.*, Jan. 22, 1898.
[2] *Wien. klin. Woch.*, May 22, 1897. [3] *Münchener med. Woch.*, 1899, No. 4, S. 108.

of the left auricle occurs in mitral disease, and especially in mitral stenosis. The right auricle hypertrophies, though not invariably, when the blood-pressure in the pulmonary vessels is pronounced from any cause. Stenosis of the tricuspid orifice is occasionally the sole cause of thickening of the right auricular wall, which also becomes hypertrophied in tricuspid incompetency.

Symptoms.—There is usually an entire absence of subjective symptoms when compensation is efficient. When present, their intensity varies with the degree of the hypertrophy, which is then pronounced, as a rule, and often already attended by incipient dilatation. They may be *local* entirely, though frequently *general* as well. Of the former, *precordial discomfort* and *uneasiness* from the violence of the impulse occur. They are most annoying when the patient is in the recumbent posture on the left side and when the hypertrophy is dependent upon nervous causes. *Pain* and *palpitation* are seldom complained of except by neurasthenics and patients suffering from enlargement due to tobacco or excessive muscular exertion. Decided aggravations of the local manifestations may follow undue mental emotion or excitement, physical exhaustion, active bodily exercise, and gourmandizing.

The *general symptoms*, when present, may fluctuate or even intermit. Those most frequently observed are fulness in the head, often amounting to actual *headache*, *tinnitus aurium*, *carotid pulsations*, *flushing of the face*, *flashing of light before the eyes*, *and often prominent eyeballs*. These symptoms are attributable to the increased vigor of the cerebral circulation.

Remote Effects.—General or total hypertrophy promotes high tension throughout the arterial tree. Endarteritis and arterio-sclerosis are, as a consequence, frequent simultaneous developments in advanced cases, especially when the cause of the enlargement has been increased tension in the peripheral vessels, as in Bright's disease. With a circulation too forcibly carried on, as in hypertrophy, the sclerotic vessels are overstrained, and are apt to rupture. The break often occurs in the brain (*apoplexy*) or in the lung (*pulmomary apoplexy*), and hemorrhage from the lung (*hemoptysis*), due to left ventricular hypertrophy, is more common, I believe, than is supposed. The blood-pressure is high, although the records vary with the dominating cause. Some of the symptoms are due to the cause or causes of the hypertrophy.

Physical Signs in Left-sided Hypertrophy.—*Inspection.*—In females and in children with soft, yielding ribs there is visible arching. The intercostal spaces are much broadened and the apex-beat covers an increased area, the extension being downward and to the left. The whole body of the patient, and even the bed on which he may be lying, may share visibly in the cardiac impulse.

Palpation.—In pronounced grades the impulse may be felt as low down as the seventh interspace and as far to the left as the axilla. In simple hypertrophy it is carried downward to the sixth intercostal space and outward to a point near the anterior axillary line. The impulse is slow, forcible, and heaving, the "thrust" lifting the fingers of the examiner. In eccentric hypertrophy (hypertrophy with dilatation), though heaving and forcible, it is somewhat more abrupt, as in cardiac dilatation. Over the aortic orifice a short diastolic impulse may also be felt occasionally (double impulse). Pressing the fingers into the second and

third right spaces will detect an impulse if the aorta be dilated. The pulse in pure hypertrophy is full, strong, regular, and of normal rate; it is also prolonged, owing to increased tension. In eccentric hypertrophy it is more abrupt, soft, full, and somewhat accelerated.

Percussion.—This defines only approximately the degree of enlargement, as the hypertrophy may take a backward direction or there may be more than the usual overlapping of the heart by the lung. Traced upward, dulness may terminate in the second interspace, whilst to the left it may extend 1 or 2 inches (2.5–5 cm.) beyond the mid-clavicular line. When hypertrophy is of moderate extent the left limit of dulness corresponds with the results of palpation and inspection; but when it is of immoderate extent the extension of dulness does not keep pace with the systolic impulse, which is diffused to points without the limits of contact of the heart with the thoracic wall. If concomitant hypertrophy of the right ventricle be present, dulness will also extend to the right (*vide infra*).

Auscultation.—The sounds vary with the grade of the morbid process and the variety. In simple hypertrophy of marked type a prolongation of the first sound is always appreciable, and usually it is duller than the normal. The second sound (aortic) is intensified, clear, and often ringing. The degree of accentuation depends partly upon the vigor of the left ventricle, though chiefly upon the condition of the blood-vessels. Reduplication of the second sound, due to high tension, is common (*e. g.* in Bright's disease). The first sound may also be duplicated. In dilated hypertrophy the first sound is clearer and more abrupt, while the second is less marked or even faint. Modification of these sounds occurs when hypertrophy is due to chronic valvular disease.

Hypertrophy of the Right Ventricle.—One or more of the causal factors that produce augmented tension in the pulmonary vessels are present, and, if properly appreciated, will throw light upon the condition. There may be an absence of all symptoms if the hypertrophy exactly balances the result of the obstructive forces, and this state may be maintained for a long period of time. Undue exertion, however, soon leads to *temporary dyspnea* in many cases. When secondary to emphysema or cirrhosis of the lung the symptoms occasioned by the latter diseases, such as *cough* and *dyspnea*, may completely veil any symptoms that may be due to the hypertrophy. *Discomfort* in the cardiac region should, however, arouse suspicions of the existence of the latter condition. When *dilatation* of the ventricle supervenes, as is usual, and the clinical evidences of tricuspid incompetency develop, then pulmonary symptoms, due to venous congestion, are prominent; these are bronchial catarrh, shortness of breath, and the like. Later, general cyanosis and edema appear. As pointed out in the discussion of Mitral Stenosis with permanently heightened tension and overgrowth of the right ventricle, the lung-vessels become atheromatous and the lung-tissue the seat of brown induration. Owing to the fact that the sclerotic vessels are easily ruptured, *hemoptysis*—a not uncommon event after sudden great exertion—is to be expected; intense pulmonary congestion and apoplexy may also be met with in hypertrophy with dominant dilatation.

Physical Signs.—These have been in the main detailed in speaking

of affections of the mitral valve. *Inspection* discloses bulging of the sixth and seventh left costal cartilages and of the lower sternum. In the angle between the ensiform cartilage and the seventh rib an epigastric impulse may be visible, but more commonly the impulse is in the sixth interspace, close to the left edge of the sternum. It is also very generally seen to the right of the sternum, in the third and fourth interspaces, and particularly is this the case in eccentric hypertrophy, forming a highly characteristic sign. The apex-beat is therefore diffuse, the radial *pulse* is small, and in dilated hypertrophy it is increased in frequency, and is small, unsustained, and irregular.

Percussion shows the extension of cardiac dulness to a point an inch (2.5 cm.) or more beyond the right sternal border. When there is great increase transversely, dilatation is most probably associated and may predominate over hypertrophy. The *auscultatory* signs are not distinctive unless dilatation also exists, when the first sounds are clear and sharp. In simple hypertrophy the first sound is slightly prolonged and lower than in health. Owing to the high vascular tension throughout the lungs the second sound at the pulmonary valve is accentuated, and reduplication of the second sound may occur for the same reason.

It must be kept in remembrance that when advanced emphysema is present all the physical signs will be greatly modified, and may even be entirely negative, though the heart be of large size. Under these circumstances venous pulsation in the neck would be diagnostic of dilated hypertrophy of the right ventricle.

Hypertrophy of the Left Auricle.—This may be assumed to occur in mitral stenosis and incompetency in order to compensate for these lesions: it cannot, however, be recognized positively by physical signs. When the chamber is at the same time extensively dilated, the dulness may be extended upward to the left of the sternum, passing over the third and even second interspaces. At this point—the second interspace—a presystolic wave may now be noticeable.

Hypertrophy of the right auricle, associated with dilatation, is perhaps more common than its counterpart on the left side. It is secondary to tricuspid incompetency (rarely stenosis) and enlargement of the right ventricle, and hence has the same etiology as the latter conditions.

The *physical signs* are—systolic jugular-pulsation, sometimes a presystolic wavy pulsation over the third and fourth interspaces to the right of the sternum, extension of cardiac dulness to the same interspaces, and other signs of tricuspid regurgitation.

Diagnosis.—The recognition of cardiac hypertrophy is possible only by attention to the physical signs. Next to these, in point of diagnostic value, come the causes, which should therefore be diligently searched for; the rational symptoms are least in value, though usually corroborative. It is difficult to establish a diagnosis, even approximately, when extensive emphysema coexists. The size of the heart can be accurately determined by the use of the *X*-rays, or, at all events, by means of the orthodiagram.

Differential Diagnosis.—Conditions that cause an increase in the precordial area of dulness, except hypertrophy, must be eliminated. (1) *Pericardial Effusion.*—A careful analysis of the physical signs and the history will suffice. (2) *Aneurysm.*—In this affection the enlargement

is altogether upward and to the left or right. This fact, joined with the other evidences of aneurysm, should obviate error. (3) *Mediastinal growths* also enlarge the dull space mainly upward and to the right or left, though the point of cardiac contact may be increased and the heart carried forward. (4) *Displacement of the heart* does not give a heaving impulse nor an increased area of dulness; moreover, it usually furnishes its special cause (pleural effusion). (5) Abnormally narrow-chested persons present a considerably increased superficial zone of dulness, partly owing to the position assumed by the lungs and partly (perhaps chiefly) to their imperfect development. Since there is usually an entire absence of all other physical signs of hypertrophy, ordinary caution will exclude the latter complaint. (6) *Affections of the Lungs and Pleuræ.*—Left-sided pleurisy with retraction may, by exposing a large part of the anterior surface of the heart, give rise to signs of moderate hypertrophy. The presence of the former condition, the lack of lung-expansion on deep inspiration, the displacement of the heart to the left and upward, and an absence of the causes of hypertrophy should lead to a correct conclusion. (7) *Phthisis and cirrhosis of the lung*, with or without pleurisy, may in like manner produce apparent enlargement of the heart. It must also be remembered that cirrhosis of the lung is one of the causes of right-sided hypertrophy, and that the latter condition may therefore be present.

Prognosis and Course.—The course that will be pursued depends largely upon the stage at which the case has arrived and the character of its special cause. I have repeatedly found *postmortem* evidence of a moderate grade of hypertrophy in persons who died of other affections, and with especial relative frequency in those who had constantly followed manual pursuits. Simple cardiac hypertrophy, being compensatory as a rule, exerts in nearly all instances a salutary influence, and if the processes that constitute the causal factors are not steadily progressive, life may not only not be curtailed, but be greatly lengthened by its existence. Even in organic valvular disease of the heart hypertrophy prolongs life by overcoming the ill effects of the valve-lesion and by maintaining the normal circulatory equilibrium. But since in this class of cases the lesion is progressive despite treatment, a limit is reached sooner or later beyond which the increased vigor on the part of the heart cannot be maintained. The nutritive functions become inadequate in obedience to a natural law, and muscular degenerations then occur, followed by disturbances of the circulation due to cardiac weakness and secondary dilatation. It must, however, be recollected that the heart may at no time, in the course of certain cases, fully compensate for the causal condition—*e. g.* as when a valve ruptures with startling suddenness. Failure of the cardiac nutrition at once renders the prognosis unfavorable. The cardiac sounds now give notice that the hypertrophy no longer meets the requirements of the case. The systolic pause grows longer (with abbreviation of the first sound), and the diastolic shorter. Occasionally, as the result of undue muscular exercise, acute dilatation, followed by a speedy termination of life, is observed. I believe that hypertrophy of the left ventricle warrants a more favorable prediction than can be made in hypertrophy of the right, and this for two reasons: first, the increased capacity for work of the

left ventricle; second, the milder character of the many factors that are productive of left ventricular hypertrophy, as compared with those of the right. In special instances, however, the reverse may obtain, as when left-sided hypertrophy is associated with or caused by general arterial degeneration. It may be of advantage to the student and junior physician to recapitulate here a few of the chief points that are prognostically favorable as well as those that are unfavorable: *Favorable Conditions.*—(1) When the hypertrophic development fully compensates the causal lesion; (2) when the causes are removable or more or less amenable to treatment; (3) when the external conditions under which the patient lives, his habits, and general nutrition are good. *Unfavorable.*—(1) When signs of imperfect nutrition of the heart arise; (2) when evidences of advancing cardiac dilatation (dyspnea, rapid, irregular pulse, edema) show themselves; (3) when poverty, poor food, intemperate habits, and an unhygienic environment are all combined; (4) when apparent cardiac vigor suddenly gives place to dilatation and great cardiac weakness.

The **treatment** has for its prime object the prevention of failure of compensation on the one hand and overhypertrophy on the other (*vide* Chronic Valvular Disease).

Over-hypertrophy, as indicated by certain cerebral and thoracic symptoms, may require the employment of measures to reduce the contractile energy of the left ventricle, although direct cardiac depressants (aconite, and the like) are rarely needed. It requires careful dietetic and hygienic management. Briefly, the *diet* should be nutritious, but the more concentrated forms of food should be used very sparingly, and the daily quantity should be slightly less than that required in health. It must be non-stimulating, and tea, coffee, alcohol in all forms, and smoking must be prohibited. The physical exercise should be moderate in amount and of the gentlest sort; and if the patient's occupation tends to stimulate the heart, it must be immediately abandoned. A mild saline purge (℥ij to ℥ss—8.0 to 16.0—of Rochelle salts once daily) is quite beneficial.

For relief of the cerebral symptoms (tinnitus aurium, vertigo, fulness) and the precordial discomfort the physiologic relaxants of the capillaries and the arterioles are of great service, particularly when arterio-sclerosis is a traceable cause. Among them nitroglycerin in full doses and veratrum viride are most useful; the efficacy of both may often be enhanced by the addition of the bromids. In cases of nervous origin the bromids, with preparations of valerian, are the most valuable agents. Nothing, however, is of higher importance than the determination and removal of the cause when possible. After compensation has failed the further treatment is identical with that of cardiac dilatation.

DILATATION OF THE HEART.

Definition.—By dilatation of the heart is meant an enlargement of its various cavities. The walls of the chambers may in consequence be thinner than in health, but much more commonly they are thicker, as in *dilatation with hypertrophy*. Both hypertrophy and dilatation are rela-

tive terms, but the latter has reference to that condition in which the cavities are distended out of proportion to the diameter of their walls.

Varieties.—(1) *Dilatation with Hypertrophy.*—Here there is a progressive increase in the capacity of the chambers until they attain to large dimensions. The cardiac walls continue of abnormal thickness, yet the vigor of the divisions affected may be relatively diminished to a remarkable degree, owing to the weakening influence of the degenerative processes that attack the hypertrophied muscles. In eccentric hypertrophy the heart-cavities are dilated, but the hypertrophied cardiac walls are sufficiently vigorous to meet the demands of the circulation. This condition should not be regarded as identical with *dilatation with hypertrophy*, but frequently merges into the latter, the size of the cavities now being proportionately greater than is the thickness or the functional power of their walls.

(2) *Dilatation with Thinning of the Heart-walls.*—The diminution in the thickness of the cardiac muscles may be slight if the capacity of the chambers involved be only moderately increased. Instances of this sort are sometimes seen to follow prolonged fever (typhoid). On the other hand, the process of attenuation may reach a high grade, the greatly thinned cardiac wall being scarcely capable of holding the weight of the contained blood.

(3) *Dilatation with little or no variation from the normal cardiac wall* has also been described by some authors. It is to be observed, however, that stretching of a cavity whose walls are of normal thickness must be attended with thinning of those walls.

Pathology.—Dilatation with hypertrophy is generally secondary to valve-lesions, and affects more than one cavity as a rule. It may happen, as in advanced aortic regurgitation, that all the divisions are dilated. The right ventricle is somewhat more frequently dilated than the left, however, for reasons previously adduced. The auricles (especially the left) are more frequently expanded than the ventricles; hence of all the chambers the left ventricle is least apt to dilate. The extent of the relative increase in the capacity of the cavities is variable, and often remarkable. As an example of extreme dilatation of a chamber, the left auricle in cases of mitral stenosis may be singled out; I have seen an instance in which this auricle was capable of containing twenty-two ounces of blood. The septum may be seen to bulge when one ventricle only is stretched. Extensive dilatation of the chambers produces a dilated condition of the auriculo-ventricular rings, which in turn gives rise to relative incompetency. Other cardiac orifices are found to be similarly dilated. Dombrowski[1] has drawn attention to the fact, first pointed out by Wolf, that the surface of the mitral leaflets greatly exceeds the orifice, and Kirschner and Garcin contend that the anterior flap alone suffices to close the mitral orifice, "even when the left heart is considerably dilated." Dombrowski believes that functional incompetency is due, in many cases, "to muscular dilatation, producing a separation of the insertions of the papillary muscles, which in systole cannot approach each other near enough to allow the valves to close, the contraction of the papillary muscles only increasing the difficulty."

[1] "Functional Insufficiency of the Valves of the Left Heart," *Revue de Médecine*, Sept. 10, 1893.

Great dilatation of the left auriculo-ventricular ring is, however, probably an important factor in the causation of relative mitral incompetency. The tricuspid valves, being scarcely competent, normally, are unquestionably incompetent when that orifice is considerably dilated.

The *shape* of the heart is altered according to the seat and extent of the dilatation. When all the cavities are dilated the organ assumes a globular form, while dilatation of the ventricles only produces broadening of the apical region.

Condition of the Endocardium and Cardiac Muscle.—The muscular tissue generally exhibits degenerations (fibroid, fatty, or parenchymatous). Important as is the part played by the ganglia in maintaining the nutritive integrity of the heart by supplying nervous force, our knowledge of the alterations that may occur in them in this condition is as yet very imperfect. Ott and others have, however, found them to be degenerated. Opacity and patchy roughening of the endocardium are common. The parietes and endocardium may, however, have a normal color and structure.

Etiology.—Entering into the causation of cardiac dilatation, there are two essential factors: (1) increased endocardial tension; (2) diminished resistance. These often act together. Broadbent contends that the special feature of dilatation is the imperfect emptying of the ventricles.

(1) **Increased Endocardial Tension.**—It is to be premised that a primary and a secondary form occur, the latter being of greater importance clinically than the former. Primary dilatation occurs from a recent obstruction to the circulation of considerable magnitude and at any point throughout the blood-vessel system. A good example is afforded by aortic constriction, in which condition the obstruction of the aortic ring engenders dilatation of the left ventricle by raising the intraventricular pressure; this is quickly overcome by compensatory hypertrophy. In the vast majority of these instances the nutrition of the muscular fibers eventually suffers, with consequent dilatation.

Other causes of augmented endocardial pressure have been considered in the discussion of Hypertrophy and Chronic Valvular Lesions. In eccentric hypertrophy dilatation is a compensatory arrangement, until finally the cardiac nutritive functions fail and dilatation at once predominates (dilatation with hypertrophy). Compensation has now been ruptured. Among the exciting factors that may precipitate this accident are—recurrent endocarditis, intercurrent febrile affections which overstimulate the heart and impair its muscular tissue, general disturbances of nutrition, and physical and mental overstrain.

Acute primary dilatation may be brought about by sudden, great exertion, as in ascending mountainous elevations, excessive bicycling, and the like. Under these circumstances the heart palpitates violently, and there are epigastric pulsation and often pain in the cardiac region—evidences of dilatation of the right ventricle. Although the heart's reserve capacity for work has been exceeded, rest followed by moderate exercise often restores the conditions to the normal. I have seen acute primary dilatation produced by strong emotion; here sudden contraction of the peripheral vessels occurs, attended with arrest of the heart's action; this soon gives place to violent palpitation and, rarely, to dilatation. *Angio-*

spastic dilatation is a condition due to acute transitory spasm of the vessels (Jacob).[1] Sudden fright may act similarly.

The remarkable endurance of the athlete and the gymnast is in part owing to the abnormal amount of physiologic cardiac reserve force which they naturally possess, but it is mainly due to the invigorating effect of training. If, however, the training be not so conducted as symmetrically to develop the entire muscular system, or if the exertion be in excess of the reserve functional power of the heart, then acute dilatation may suddenly arise. From this accident (cardiac fatigue) recovery may take place; sometimes, however, it initiates organic valvular disease, and thus prohibits the further undertaking of unusual feats. Acute dilatation has been made conspicuous by recent contributions, in which bicycling is assigned as the cause.

Apparently idiopathic cases of cardiac dilatation of indeterminate etiology rarely occur.

(2) Diminished Resistance owing to Weakened Cardiac Walls.—The conditions that weaken the cardiac wall are numerous, and not a few lead to acute primary dilatation, such as *myocarditis* due to acute specific fevers (scarlatina, typhoid, malaria, typhus). It is especially prone to occur in *rheumatic endocarditis* and *pericarditis*. B. Robinson[2] calls forcible attention to serious dilatation due to the toxic action upon the heart muscle of the rheumatic poison. The *chronic degenerations* (fatty, fibroid) impair the contractile power of the heart. *Nutritional disturbances* of varied origin, such as digestive disorders, ill-ventilation, lack of open-air exercise, and improper or defective food-supply, may induce enfeeblement of the cardiac muscle. Dilatation is met with also in *diseases of the blood* (chlorosis, anemia, leukemia).

Clinical History.—In *acute* dilatation the *onset* is sudden. It is accompanied by rapidly augmenting dyspnea and cardiac palpitation, a feeling of coldness, and frequently by pain in the precordial region.

The physical signs may be incontestable. They are *venous pulsation* in the neck, a *rapid, feeble apex-beat*, and a *systolic murmur* at the tricuspid valves, all of which declare the presence of tricuspid regurgitation. In *angiospastic dilatation* the pain may begin in the extremities, and the second heart-sound may be louder at the apex than the first. Among signs of subsidiary value are a venous turgescence, a marked epigastric pulsation, and a sudden extension of *dulness* to the right; the *pulse* is small, irregular, and exceedingly rapid.

In the more *chronic form* which arises from slowly-acting causes, or in that which accompanies eccentric hypertrophy or follows simple hypertrophy due to left-sided heart- or lung-trouble, the manifestations in the earlier stages are not striking. They indicate weak heart-walls, and such chambers expel their contents imperfectly during systole. With each subsequent diastole the abnormal amount of blood contained in them is increased. This blood-stasis often extends from the left heart to the pulmonary vessels, from the latter to the right heart, and finally to the general venous system. Increased viscosity of the defibrinated blood is said to be an early sign of cardiac failure. Both in the acute and chronic forms, however, *failure of the right ventricle* more often

[1] *Zeitschr. f. klin. Med.*, February 4, 1899.

[2] *American Journal of the Medical Sciences*, Dec., 1899.

determines rupture of compensation. The symptoms are chiefly those of tricuspid incompetency. Dilatation of the right heart, without tricuspid insufficiency, is a frequent complication of pulmonary tuberculosis (Maisonneuve[1]).

Physical Signs.—*Inspection* in dilatation of the left ventricle shows the apex-beat to be displaced outward and downward, and a diffuse, weak, fluttering, and often distinctly undulating impulse. The apex-beat will show a greatly diminished vigor in its normal area; or there may be no recognizable point of strongest impulse as in health. Distinct pulsation in the second left interspace is not rare. Its feebleness and diffuse character are confirmed by *palpation*. It may be quick and sharp, though always lacking in power. Walsh first made the capital observation—since abundantly corroborated—that the impulse may be visible, yet not palpable. There may be a mere vibration or an utter absence of the apex-beat in advanced cases. The pulse is small (rarely large), short, often rapid, and irregular. Palpation of the pulse should always be combined with the use of the sphygmomanometer, which may show an unexpected exaltation of the blood-pressure, especially when dilatation develops somewhat abruptly. *Percussion* shows a lateral increase in dulness to the left, to or even beyond the mid-clavicular line, upward to the second rib, and downward as far as, though rarely below, the sixth interspace, except perhaps, in rare instances, in dilatation with hypertrophy. In emphysema the lungs unduly overlap the heart.

Dilatation of the right ventricle demands separate consideration so far as the impulse and percussion-dulness are concerned. The normal impulse is largely replaced by the abnormal apex-beat of the right ventricle, which advances to the anterior chest-wall. The chief impulse is now seen and feebly felt, as a rule, below the xiphoid cartilage, or, less commonly, to the right or left of the latter. A wavy pulsation is seen to the left of the sternum, over the fourth, fifth, and sixth interspaces and close to its right edge. If dilatation of the right auricle be associated, as is often the case, a distinct pulsation also occurs in the third right interspace. Dulness reaches to a point 1 inch (2.5 cm.) or more beyond the right sternal border on a level with the fourth interspace.

On *auscultation* variable results are obtained according to the state and diameter of the cardiac walls. When thin and not much disorganized, the first sound is much shorter, sharper, and louder than in health. In advanced cases the systolic sounds may be feeble, though almost always audible in the aortic area (unlike the first sound in hypertrophy). The first closely resembles the second sound, the long pause being shortened, resembling the systolic pause (*fetal heart-sounds*). This form of arrhythmia is a serious indication of failure of the ventricles. The *canter rhythm* is equally common. Irregular and intermittent cardiac action are usual phenomena. Reduplication may occur, but is not frequent.

Pre-existing *organic murmurs* obscure the sounds due to dilatation, and, on the other hand, the dilatation may also alter the murmurs (previously audible), and even cause them to disappear, as, for example, in mitral stenosis. Again, dilatation may induce relative incompetency or superadd a murmur, as in cases of chronic valvular disease at the auriculo-ventricular orifices. It is interesting to recall here that proper treat-

[1] *Gaz. hebdom. de Méd. et de Chir.*, Oct. 30, 1898, No. 45; Année, No. 87.

ment may remove a murmur due to relative insufficiency, and that this treatment may, in turn, reproduce an organic murmur.

Diagnosis.—This is made readily when there is obtainable a clear history, together with the following characteristic features: a weak, irregular heart action (throbbing of the precordium); an extended, wavy impulse; a small, vigorless, and intermittent pulse; often an indistinct apex-beat; an outward, upward increase in the percussion-dulness on one or both sides, causing the outline to resemble a square; and a brief, sharp, yet feeble first sound that resembles the second, which is enfeebled.

Differential Diagnosis.—*Hypertrophy*, like dilatation, gives rise to an extended area of impulse and of percussion-dulness; hence by the careless observer these conditions are sometimes sadly confounded. From dilatations, in which the diagnosis rests upon the points above enumerated, hypertrophy is to be distinguished by symptoms of an opposite nature, such as indicate increased energy on the part of the heart. The latter are—a slow, heaving impulse; a full, sustained, regular pulse; an increase in the area of dulness, chiefly outward and downward; abnormal position of the apex-beat; and the prolonged, dull first and accentuated second sounds. To determine the point at which eccentric hypertrophy ends and dilatation (with hypertrophy) begins is often difficult; and I have already discussed the initial symptoms of dilatation following hypertrophy (chiefly of the right ventricle) in connection with Chronic Valvular Disease. Occurring in left ventricle hypertrophy, dilatation first betrays itself by a change in the position of the visible apex-beat and the palpable impulse. Thus, the maximum point of the apex-beat of hypertrophy very early becomes rounded and indefinite, and later is diffuse and wavy. The strong, heaving thrust of the impulse gives place to the shorter, more sudden shock of commencing dilatation, indicating weakness. These signs, together with a reduction in the strength and an increased frequency or irregularity of the pulse, show the condition to be dilatation with hypertrophy.

The **prognosis** is bad, as a rule, being that of the causative factors.

Treatment.—This in all essential particulars is identical with the treatment of organic heart affections after rupture of compensation. The etiology in many cases differs from that of the organic valvular affections of the heart; and the removal of the remote and near causes of the dilatation is the most important part of the treatment. Individual cases frequently present special indications; but in all the work of the heart is increased and the propulsive power of the organ diminished. The indications are to diminish the heart's labor by bodily and mental rest, light diet, purgation and relaxing the peripheral vessels (T. A. Claytor), and to increase the functional power of the heart by the use of cardiants, baths, and massage. In cases of non-valvular origin digitalis and other heart stimulants may be omitted early, as a rule; though they should be resumed if there be a recurrence of serious indications of dilatation. The best guide in the treatment is the sphygmomanometer, which should be employed at intervals of several days. When the dilatation has been overcome, careful attention is to be bestowed upon all the details of the patient's life and sanitary surroundings in order to force his bodily nutrition to the utmost. Every precautionary measure having for its aim the prevention of a recurrence of the dilatation must also be advised and enjoined.

MYOCARDITIS.

(*Carditis.*)

Definition.—An inflammation of the muscle-substance of the heart. It may be acute or chronic.

ACUTE MYOCARDITIS.

Pathology and Varieties.—(1) **Acute Parenchymatous Myocarditis.**—This is characterized by a granular degeneration of the muscular fibers of the parenchyma of the organ, with a numerical increase in their nuclei. The muscle-structure throughout looks pale, is turbid, and very soft. Many cases of a severe type terminate in fatty degeneration.

(2) **Acute Diffuse Interstitial Myocarditis.**—Here the primary alterations affect the connective tissue of the myocardium; the histologic changes consist in round-cell infiltration.

(3) **Acute Circumscribed Myocarditis.**—In this variety the degenerative processes result in necrosis of the tissues over large or small areas, with abscess-formation. Though usually multiple, these abscesses vary in number, and may rupture either into the various cardiac chambers or into the pericardium. Thus, the purulent contents of the abscess, when there is established a fistulous communication with an endocardial chamber, find their way into the blood-stream, frequently setting up embolic processes of an infectious nature in the various viscera. The blood in turn enters the abscess-cavity, exerting pressure on its walls, and may either produce an acute aneurysmal dilatation of the heart-wall or occasion fatal rupture into the pericardium. More commonly the connective-tissue wall of the abscess yields gradually during the ventricular diastole. Occurring in the vicinity of one of the auriculo-ventricular valves, abscesses may cause mitral or tricuspid incompetency. They may perforate the interventricular septum, thus creating a fistulous connection between the two sides of the heart, and resulting in an intermingling of venous and arterial blood. The abscess may become encysted, then caseous, and finally undergoes a calcareous process. Multiple abscesses usually affect the left ventricle.

Etiology.—*The causes of myocarditis* are—(*a*) endo- and pericarditis in the course of rheumatism: it is probable that rheumatic myocarditis may also exist without involvement of the endo- or pericardium; (*b*) the infectious processes in acute specific fevers (influenza, diphtheria, typhoid); (*c*) infectious emboli, lodging in the branches of the coronary arteries in connection with septicemia, pyemia, and acute ulcerative endocarditis, and commonly terminating in abscesses (circumscribed myocarditis). The first two of these causes give rise to acute diffuse interstitial and acute parenchymatous myocarditis, as a rule, although Freund calls attention to the frequency with which circumscribed myocarditis is associated with rheumatism and diseases of the joints. As compared with the female *sex*, the male suffers much more frequently.

Symptoms and Diagnosis.—The symptoms are practically negative. They point merely to great cardiac enfeeblement. When cardiac weakness, as shown by a rapid, small, compressible, and arrhythmic *pulse*, and by attacks of *cardiac palpitation* and *syncope*, comes on suddenly

in the course of rheumatism, septicemia, or other causal affections, myocarditis may be suspected. Later, signs of *venous stasis* appear. The systolic blood-pressure is commonly low, though fluctuating, varying from 100 to less than 80 mm. Hg. The *mental symptoms* may suggest meningitis or salicylic-acid poisoning. Koplik [1] calls attention to certain symptoms (pallor, faintness, vomiting, irregular, feeble heart-action, disturbed respiration, and pulse-ratio) that should arouse suspicion of myocarditis in the course of an infectious disease in childhood.

The physical signs simulate those of dilatation, and may, indeed, be largely dependent upon the presence of the latter condition. Early the action of the heart is tumultuous; the sounds on auscultation are short, sharp, and finally very feeble. *Murmurs* in myocarditis are not rare, and are not necessarily dependent upon dilatation. Krehl's work shows the dependence of the valves for their complete closure upon a normal state of different portions of the heart-muscles, and thus explains these murmurs. The special conditions rendering the murmurs audible are great dilatation, softening of the papillary muscle, and abscesses near the valves.

The great variability as to the intensity of these murmurs is an important point, especially in attempts to discriminate from murmurs due to *endocardial changes*. The latter usually coexist with a more marked accentuation of the second pulmonary sound. For the recognition of *cardiac aneurysm*, see p. 700. The symptoms of visceral or cutaneous *embolic processes*, combined with a murmur and a septic type of fever, are suspicious of the existence of circumscribed myocarditis. The murmur of relative tricuspid regurgitation and the venous pulse may eventually develop, accompanied by the symptoms of general venous engorgement.

Prognosis.—The diffuse forms are often fatal; the circumscribed form rarely eventuates in recovery. Myocarditis may end life suddenly.

The **treatment** is identical with that indicated for endocarditis and pericarditis—diseases of which myocarditis is often a complication. The effects of digitalis, particularly when myocarditis supervenes upon old heart-lesions, are quite unsatisfactory, but diffusible stimulants—*e. g.*, aromatic spirit of ammonia, brandy, and the like—are useful. When myocarditis is suspected as an independent condition absolute rest must be enjoined, the general nutrition maintained, and the more urgent symptoms relieved.

CHRONIC MYOCARDITIS.

(*Fibrous-Myocarditis.*)

Definition.—A gradually developing inflammation of the cardiac interstitial connective tissue, resulting in induration.

Pathology.—The characteristic changes may be diffuse, though most frequently they are confined to certain portions of the muscular structure, the left ventricular wall, the septum, and the papillary muscles being the three favorite seats of the process. This is sometimes of antenatal development, and then its usual seat is near the apex of the right ventricle. The hardened spots take the form of more or less rounded patches or broad lines. In color they are gray, grayish-white, or grayish-yellow, the latter tint being due to the intermingling of fibers that have undergone fatty degeneration. Their size is exceedingly variable, some being so minute as to elude detection by the unaided eye, while

[1] *Medical News*, March 31, 1900.

others measure 1 or 2 inches (2.5–5 cm.) in diameter. Inflammatory induration (contraction) of the *conus arteriosus* of either ventricle causes narrowing of the pulmonary and aortic orifices, with the usual signs and symptoms. Similar changes, by disturbing the functions of the papillary muscles, produce valvular incompetency. Compensatory hypertrophy of the uninvolved portion of the heart is also observed; the hypertrophic enlargement may frequently be accounted for in part either by an associated chronic endocarditis or general arterial sclerosis. Dilatation of the ventricles follows, with fresh and grave disturbances of the circulation.

Chronic inflammation usually attacks early the intima of the coronaries, and leads to thrombosis, with the formation of anemic infarcts (*vide* p. 695). It is probable that most cases of localized fibrous myocarditis have their origin in an obliterating endarteritis. Pasquier offers proof that myocarditis results from chronic congestion due to stopping of the vessels. The calloused zone may yield to the endocardial blood-tension, and thus produce saccular dilatation (aneurysm). *Microscopically*, the affection is characterized by hyperplasia of the interfibrillar connective tissue with subsequent development of new fibrous tissue. Fatty degeneration and atrophy of the muscle-fibers (due to compression) are also observed. Fragmentation of the muscle-fibers (the *état ségmentaire* of Renant) has also been observed. This occurs as a *postmortem* change.

Etiology.—The disease is most commonly traceable to the action of one or more of the following factors: an *excess in the use of alcohol* or *tobacco*, *lead-poisoning*, *phosphorous*, *gout*, *rheumatism*, *diabetes*, *chronic nephritis*, *malaria*, and *syphilis*. Thus, it may be produced by many infections and chemical irritants, the latter, in most cases, first causing a sclerosis of the coronary arteries, to which the patchy fibroid degeneration is secondary. Some of the causes of acute diffuse interstitial myocarditis may by their more slightly irritant effect lead to the subsequent development of the general chronic form (*e. g.*, rheumatism). Certain irritants that engender localized lesions of chronic myocarditis may affect the entire myocardium (syphilis, alcohol, gout). Certain exhausting diseases, as dysentery, carcinoma, and the anemias, may act as causes. Chronic myocarditis may arise in consequence of a *direct extension* of the inflammatory processes in chronic endo- and pericarditis; it may also follow *injuries* of the anterolateral thoracic region. *Sex* and *age* possess a predisposing effect, the disease being more common in males, and after middle life than before that period. The right ventricle is apt to be the seat of chronic myocarditis during fetal life, if at all.

Symptoms.—Extensive indurated myocarditis has been met with *post mortem* in numerous instances that have been unattended by perceptible symptoms during life. In many of these cases the presence of compensatory hypertrophy accounts for the absence of any symptoms, and it may, therefore, be inferred that mild grades that fail to manifest themselves must frequently exist. The symptoms when present are, almost without exception, untrustworthy for diagnostic purposes, since they bear a striking resemblance to those of the organic valvular diseases, minus their more characteristic physical signs. Among the earliest phenomena that point merely to failing heart-power are *dyspnea*, and sometimes also, on exertion, *palpitation* and a *sense of heaviness or constriction* in the precordia. The patient suffers from marked general debility, and becomes *fatigued* in consequence of the slightest

physical exertion. *Mental inertia* is the rule, and *chronic mania* may come on and last to the close. Later, more positive disturbances of the circulation gradually arise, and when the breathing becomes more difficult (*cardiac asthma*) signs of *venous stasis* affecting the liver, gastro-intestinal tract, and kidneys, and edema finally appear.

Two symptoms that are frequently manifested, and not without some diagnostic import, remain to be mentioned: (1) *Angina pectoris*, which is attributable to the sclerosed condition of the coronary arteries. (*Vide* Angina Pectoris, p. 713). It is often followed by some form of arrhythmia. Recurring paroxysms of angina pectoris, with or without arrhythmia, may be the only phenomena of the disease.

(2) *Cardiac Arrhythmia.*—Brachycardia is associated as a rule, there being a reduction in the pulse-rate to 50 or even 40 beats. With this decreased rate intermittency is often combined, and various other forms of disturbed rhythm are also observed—*e. g.*, the phenomena of the Stokes-Adams syndrome and extrasystoles. Slowing of the pulse does not prohibit the cardiac palpitation that is apt to arise during anginal attacks. Arrhythmia, however, may be entirely absent.

Chronic myocarditis may be the sole cause of the *pseudo-apoplectic seizures* that often terminate life abruptly. Preceding the unexpected attack the patient, usually advanced in life, may have experienced from time to time slight vertigo, syncope, and oppression. These seizures may also be caused by a heavy meal or intense mental or physical exertion, and may consist in a momentary loss of consciousness. At other times they last a number of hours, and are accompanied by paralysis which outlasts the coma, as a rule, by a few hours only. Convulsive twitchings may be present. During the attack cerebral hemorrhage may occur and leave the patient hemiplegic. It is highly characteristic of these pseudo-apoplectic seizures that they tend to recur, sometimes at intervals of a few hours for a day or two, but more commonly at longer intervals during many weeks or months.

Physical Signs.—The impulse may be feebly heaving (sometimes absent); the apex-beat is displaced downward and to the left, while the dull area is enlarged correspondingly in the same direction. The *pulse* is slow, irregular, and the blood-pressure more or less elevated. Should *fatty degeneration* be conjoined, the pulse may be quickened and irregular, and this effect likewise obtains when the patient escapes sudden death and the usual dilatation supervenes. Quite early the heart-sounds may be clear and strong, owing to compensatory hypertrophy of the healthy portion of the myocardium, but subsequently they become weak and muffled.

With the occurrence of dilatation comes an apical, systolic murmur (due to relative incompetency), with a gallop rhythm of the heart. A contraction of the papillary muscles and of the chordæ tendinæ may cause mitral incompetency with its customary murmur during compensation.

Differential Diagnosis.—(1) *Chronic valvular disease* can, as a rule, be eliminated prior to the occurrence of secondary dilatation, in the course of fibrous myocarditis, but not after that, even though chronic endocarditis manifests the greater degree of hypertrophy. During the period of compensation murmurs do not occur in myocarditis unless the valvular adnexa (the chordæ and papillary muscles) are affected. In cases in which these structures are involved, the secondary alterations

in the heart, the symptoms, and whole course of the complaint are the same as in certain chronic valvular lesions.

(2) *Idiopathic Hypertrophy.*—After the occurrence of *dilatation*, following indurated myocarditis, the differential diagnosis between the latter and hypertrophy, with secondary dilatation, so far as the physical signs and accompanying symptoms are concerned, is purely conjectural. A clear history may furnish differential-diagnostic points before failure of compensation occurs; for example, evidences of decided arterio-sclerosis, due to syphilis, would be in favor of chronic myocarditis.

(3) *Fatty overgrowth* must be distinguished from fibrous myocarditis, and is met with chiefly in brewers, publicans, and butlers. The disease is also found to be specially related to obesity, and sometimes to over-eating and drinking, combined with indolent habits. These subjects suffer more frequently from bronchitis, emphysema, and nocturnal asthma than patients having chronic myocarditis alone. Slight vertigo is common, but true syncopal attacks are rare. In fatty overgrowth the heart-sounds are weak and decidedly muffled throughout; the pulse is weak, though regular as a rule and of normal rate.

Prognosis.—Chronic myocarditis is a fatal disease. Its course and duration, however, are subject to great variations. Among unfavorable surroundings are certain causal and associated conditions, particularly arterio-sclerosis, chronic interstitial nephritis, and diabetes mellitus. On the other hand, if syphilis has been the cause, hope for temporary improvement, if not for actual cure, may be reasonably entertained. Sudden death may result from a blocking of a vessel that is the seat of sclerosis.

Treatment.—The treatment should be managed according to the considerations pointed out in the treatment of Organic Valvular Disease. Rest of body and mind is imperative. Next to this come the dietetic and hygienic details. Residence in a mild climate in winter and a change to the country or to a moderate elevation in summer are advisable. Cases caused by syphilis are favorably influenced by the iodids. Those rather frequent cases that present such closely united conditions as arterio-sclerosis, gout, and chronic nephritis sometimes do well while sojourning at certain mineral springs, such as Marienbad, Carlsbad, Kissengen abroad, and Bedford or Saratoga at home These waters must, however, be cautiously used. Bell,[1] after excluding advanced arterial fibrosis, aneurysm, and advanced cardiac insufficiency with dropsy, recommends saline baths administered in a manner similar to the artificial Nauheim baths (*vide* p. 672). The Oertel cure (protein diet, restriction of fluid intake, graduated exercises up hill) may prove serviceable.

When dilatation arises cardiac stimulants are called for, but must be used with an unusual degree of caution. Strychnin has proved itself to be valuable if perseveringly exhibited, and here, as elsewhere, digitalis deserves a trial; its careless administration, however, may give bad results if the pulse be much retarded or arterio-sclerosis coexist. For the angina pectoris morphin, administered hypodermically, is to be preferred. Recurrences of this distressing symptom may be averted by the cautious use of nitroglycerin, the use of which should, however, be limited to cases that seem to be dependent upon arterial degeneration

[1] *Medical News*, New York, May 7, 1904.

with high tension. Attacks of syncope are most successfully met by the hypodermic use of the diffusible stimulants (ammonia, ether), and at the same time by putting the patient at rest with the head lowered.

DISEASES OF THE CORONARY ARTERIES.

It has previously been noted that in pyemia and allied disorders septic emboli may block the branches of the coronary arteries, causing suppurative infarcts (acute circumscribed myocarditis).

It has also been shown that one of the chief effects of sclerosis affecting the coronary arteries is the production of *chronic myocarditis.* Sudden blocking of one coronary artery by an embolus causes instant death. In numerous instances in which death has occurred suddenly either thrombotic or embolic obstruction has been the only discoverable *post-mortem* lesion. In others the pathologic evidences of local or general atheroma have coexisted. Ligation or plugging of the coronary vessels in the lower animals causes arrhythmia or even an abrupt arrest of cardiac action; a partial or even slight reduction in the lumen of the coronary vessels by diminishing the supply of blood to the heart-muscle induces degenerations in the latter. Kronecker found that occlusion of the coronary arteries by injecting paraffin, even when it solidified in only the smaller branches, caused the heart to become irregular, and to stop almost at once. The anatomic peculiarity of the coronary arteries in that they are end-arteries is to be noted, since it affords a ready interpretation of the usual effects following total or partial occlusion. According to F. H. Pratt, however, the vessels of Thebesius, which extend from the auricles and ventricles to the myocardial capillaries and coronary veins, may rarely maintain the nutrition of the heart-muscles even after occlusion of the coronary arteries.

The blocking of the terminal branches by emboli or by the formation of thrombi usually produces the so-called *anemic necrosis* or *white infarct*—a condition that deserves brief description:

Anemic necrosis (*anemic infarct*) is met with most frequently in the left ventricle and septum, which receive their blood from the anterior coronary artery. The involved areas are small and circumscribed, and present irregular margins that project slightly above the surface. Rarely the infarct is wedge-shaped. Its color is grayish-white or grayish-red, while the central portion is often white and firm; less frequently it breaks down into a soft detrital mass (*myomalacia cordis*). When softening does not occur the fibers lose their nuclei, becoming first hyaline and subsequently sclerotic. The histologic changes are of two sorts: (*a*) the striæ of the muscle-fibers are lost, the latter becoming granular and breaking down; and (*b*) the fibers assume a homogeneous hyaline appearance, the nuclei having disappeared.

The *symptomatic* consequences of the lesions are often obscure and unreliable. Sudden death may take place, and rarely this accident may be due to rupture of the heart. Weak and irregular action of the heart, evidences of embarrassed circulation (especially in the cardio-pulmonary circuit, as shown by cough and dyspnea), and finally angina pectoris, are among the principal features observed. Death may ensue in the first attack. The paroxysms are presumed to be due to

sudden occlusion of a branch of the coronary artery; but it should be stated that occasionally in fatal instances of true angina pectoris a total absence of lesions, including emboli, has been noted. I desire to lay stress upon the medico-legal importance of coronary disease; it may be the only lesion found in cases of quick death.

DEGENERATIONS OF THE HEART.

(*a*) **Fatty.**—The term "fatty heart" includes two pathologically distinct affections: (1) Fatty degeneration, in which the cardiac muscle-fibers have been converted into fat; and (2) Fatty overgrowth, in which an abnormal quantity of fat is deposited in and about the heart. According to Leyden, the cases of "fat-heart" (fatty overgrowth) are divisible into two subclasses: (*a*) fatty overgrowth, and (*b*) fatty infiltration.

FATTY DEGENERATION.

Pathology.—The condition may be either general or localized. Its most frequent seat is in the left ventricle, the papillary muscles and trabeculæ, first appearing as yellowish spots or stripes beneath the endocardium. The affected portions are light yellow or yellowish-brown (faded leaf) in color, due to an associated brown atrophy; they are soft and friable, and are easily lacerated. The heart is enlarged and its walls lack firmness. The microscope reveals characteristic changes: the striæ and nuclei begin to fade, oil-drops and granules appear in the fibers, and finally the latter are occupied throughout by minute globules.

Etiology.—Fatty degeneration, as already mentioned, occurs in both the *primary* and *secondary* forms of *cardiac hypertrophy*. It is found in association with fatty change in other organs in severe forms of *primary* and *secondary anemias*. It is most common, however, in the *cachectic states* produced by such chronic diseases as carcinoma and phthisis, and in the course of *acute infectious diseases* of intense type. Warthin finds that focal or diffuse fatty degeneration of the myocardium may be associated with numerous spirochætæ of syphilis without interstitial change. In poisoning by arsenic and phosphorus and in pernicious anemia it advances to a high grade. The various lesions of the *coronary arteries* previously considered bear the most significant causal relation.

Predisposing causes are—(*a*) *age*—it being most common after forty years of age; (*b*) *sex*—it occurs somewhat more frequently in men than in women, notwithstanding the fact that there are predisposing influences at work in the latter that do not obtain in the male sex, such as childbirth and amenorrhea; and, lastly, (*c*) whatever may be its apparent etiology, it is invariably preceded by a defective nutritive supply to the muscle-cells: this may be dependent upon a narrowing of the lumen of the coronary vessels, or upon impairment of the oxygen-carrying power of the blood, as in the anemias. An excessive supply of glucose, glycogen, and nuclein may be a factor.

Symptoms.—The disease may exist in an advanced form without

noticeable symptoms, though the conditions under which it is most liable to occur afford secure ground for suspicion. The evidences of *cardiac enfeeblement* are usually present, but in pernicious anemia and chlorosis the pulse may even be full and regular.

Dilatation is apt to supervene early, owing to the weakened state of the heart; and hence many of the symptoms are due to secondary dilatation. Among these are *palpitation*, *dyspnea*, a *small*, *irregular*, and somewhat *quickened pulse*, and *cool* and *clammy extremities*. The heart-sounds are weak, as a rule, and the action of the heart often irregular. *Dropsy* is rare in uncomplicated cases. Sometimes sudden, great physical exertion produces equally sudden dilatation, whereupon a canter rhythm and an apical systolic murmur develop. In most instances, however, the symptoms of dilatation are more gradually brought to light. *Breathlessness* on exertion is often a striking feature, and syncopal attacks are sometimes troublesome. The *pulse*, in consequence of irritation of the inhibitory center in the medulla, often becomes greatly retarded, dropping from the normal rate to 30 or 40 beats per minute, and, in rare cases, to 10 or 12 beats. The fatty *arcus senilis* is devoid of diagnostic value. There are frequent attacks of *cardiac asthma* in the mornings, and these are apt to be accompanied at intervals by *angina pectoris*. *Disturbance of the intellect*, sometimes taking the form of maniacal delusions, may come on and persist. Syncopal attacks occur. *Pseudo-apoplectic attacks*, such as have been described (*vide* Chronic Myocarditis), may occur. *Cheyne-Stokes breathing* is among the later manifestations. It happens that this symptom and pseudo-apoplectic seizures are found in association; they are more apt to be due to uremic toxemia, perhaps, than to fatty degeneration of the heart. Epileptiform attacks resembling *petit mal* may arise.

The **diagnosis** is sadly obscure. The history, the age of the patient, and the symptoms of cardiac weakness and subsequent dilatation, together with retardation of the pulse, apoplectic attacks, and Cheyne-Stokes breathing, in the absence of precedent hypertrophy merely justify a probable diagnosis. With a clear history and the presence of the more significant symptoms, including the signs of dilatation following hypertrophy, fatty changes may be inferred with some degree of assurance, although a positive opinion should be withheld.

The **prognosis** is as varied as the etiology. Death may come quickly, the process being commonly associated with sclerosis of the coronaries, though oftener the end is reached in a gradual manner, the signs and symptoms of advanced dilatation dominating the closing scene. The more corpulent the subject, the graver the prognosis.

Treatment.—The cause in each individual case should be determined, and when ascertained a bold attempt should be made to remove it. This course often places the patient in the most favorable position for the successful treatment of the cardiac condition. Anemia in one form or other plays an important rôle in the majority of the cases, and the particular variety present in each instance must determine the character of the remedies to be employed. In that large category of cases occurring in certain cachexias (cancerous, tuberculous) hematinics, arsenic, and strychnin are the remedies of choice.

A frequent, irregular pulse and other signs of cardiac failure indicate commencing dilatation, and under these circumstances digitalis should be employed in *small doses*. When found to be serviceable, its use should be continued until the dilatation is overcome; it may be conveniently combined with other cardiants.

I believe that gentle indulgence in physical exercise and light gymnastics is beneficial, since it tends to invigorate the heart-muscle; it is to be increased in proportion to the manifest improvement in the patient's condition. It sometimes happens, however, that even gentle exercise is badly borne, and it should then be discontinued. Kinesitherapy, particularly the milder Swedish method of gymnastic exercises (alternating movements of resistance), increases the contractile power of the heart and at the same time lessens the peripheral resistance, and should be accorded a careful trial. I have been in the habit of advising daily inhalations of oxygen gas in this class of cases with good results. Recourse to massage is also in the line of sound practice, but the sittings should not exceed half an hour in duration at the start. The more *prominent symptoms* may require special measures. The syncopal and anginal attacks are to be handled in the manner indicated for the same symptoms in chronic myocarditis. For the pseudo-apoplectic attacks rest in the recumbent posture, with the head slightly elevated, is useful. Therapeutic agents, as digitalis, ammonia, and ether, may be used hypodermically to stimulate the heart; it is also good practice to withdraw from 12 to 24 ounces (355.0–710.0) of blood directly from a vein. If the arteries be hard and tense, nitroglycerin is of distinct service.

A strictly horizontal posture and the application of ice to the precordial region often quickly terminate the attacks of cardiac asthma, and spartein sulphate, with nitroglycerin, is worthy of a trial. Hot toddy and other diffusible stimulants are valuable adjuvants. Should these remedies fail, hypodermic treatment by morphin is to be adopted.

FATTY OVERGROWTH.

Pathology.—The normal fat, particularly in the auriculo-ventricular furrows, is increased. I have elsewhere suggested the term "subpericardial over-fatness,"[1] to indicate the condition when unaccompanied by fatty infiltration. This over-production of fat may become so excessive as to form a complete enveloping mantle measuring an inch or more in thickness. In these extreme grades the muscular fibers may, from too great pressure, undergo atrophy and thus become weakened.

Etiology.—The principal cause is general corpulency. (For a consideration of the factors predisposing to fat production see OBESITY.) In the cachexias of carcinoma and phthisis, and the general atrophy of old age, fatty overgrowth and fatty degeneration coexist.

Symptoms.—The condition may be unaccompanied by any symptoms. The muscle-fiber is weakened (not degenerated, as a rule), hence extra labor suddenly thrown upon the organ excites the clinical indications of a weak (dilated) heart, as urgent dyspnea, vertigo, syncope, palpitation, and cyanosis. Later recurrences arise on every provocation,

[1] *Amer. Jour. Med. Sci.*, April, 1901.

Distressing attacks of asthma may develop after a full meal or without an apparent exciting cause. A passive form of bronchitis may supervene. The cardiac impulse is feeble and may even be missing. The pulse is, as a rule, regular and moderately tense. Slight intermittence and, in marked heart-weakness, decided arrythmia may be noted. In moderate grades the heart-sounds may be clear; in marked cases with ensuing dilatation a systolic, apical murmur may be audible.

The diagnosis rests upon the combined presence of marked obesity and cardiac enfeeblement. (For the **differential diagnosis**, see p. 694).

Treatment.—I wish to advocate warmly the system of treatment introduced by Oertel, as I have seen excellent results from its employment. It should not be resorted to in chronic valvular disease, in the stage of broken compensation, nor in marked atheroma.

Oertel's method comprises three parts: (1) The reduction of the amount of liquid taken with the meals and during the intervals, the total for each day being 36 ounces (1064.0). Frequent bathing (including the Turkish bath in suitable instances) and pilocarpin are employed to promote free diaphoresis.

(2) The diet is composed largely of proteids, as follows: *Morning.*—A cup of coffee or tea, with a little milk—about 6 ounces (178.0) altogether; bread, 3 ounces (93.0).

Noon.—Three to 4 ounces (90.0–120.0) of soup; 7 to 8 ounces (218.0–248.0) of roast beef, veal, game, or poultry, salad or a light vegetable, a little fish; 1 ounce (32.0) of bread or farinaceous pudding; 3 to 6 ounces (93.0–186.0) of fruit for dessert. No liquids at this meal, as a rule, but in hot weather 6 ounces (178.0) of light wine may be taken.

Afternoon.—Six ounces (178.0) of coffee or tea, with as much water. An ounce of bread as an indulgence.

Evening.—One or two soft-boiled eggs, 1 ounce (32.0) of bread, perhaps a small slice of cheese, salad, and fruit; 6 to 8 ounces (178.0–236.0) of wine, with 4 or 5 ounces (120.0–148.0) of water (Yeo).

(3) Graduated exercise up inclines of various grades. The distance to be undertaken each day is to be carefully specified and frequently, though gradually, increased. A like plan is to be pursued with reference to the degree of inclination. This is the most important part of the system, since it directly invigorates the heart-muscles.

Fatty Infiltration.—This condition may be associated with grave forms of myocardial degeneration, principally fibroid and fatty. In this place the term is limited in application to an infiltration or a dipping of fat between the muscle-fibers even to the endocardium, that is *secondary* to extreme obesity (*e. g.*, the anemic variety). It is clearly a rare condition, if we except the not uncommon instances in which the morbid process is limited to a thin layer of muscle-fibers situated directly beneath the epicardium. I have reported 5, and collected 7 additional cases from the literature.[1] The subjects are extremely obese.

The *symptoms* may develop abruptly, after some unusual muscular exercise or after a profound systemic shock. More commonly, however, the clinical indications, which are not sharply defined as a rule, manifest themselves in a gradual manner. The principal features are *urgent dyspnea* (often an asthmatic form of breathing), and *utter exhaustion*

[1] *Loc. cit.*

upon muscular exercise, *precordial discomfort*, *pain under the sternum*, *cardiac palpitation*, *arrhythmia*, *syncope*, *vertigo*, *cyanosis*, and *angina pectoris*. Marked and constant disturbance of the cardiac rhythm is symptomatic of fatty infiltration. Hydrostatic *bronchitis*, with cough and expectoration, is commonly present. The angina pectoris may be dependent largely upon associated sclerosis of the arterial system. Emotional disturbance and mental apprehension were the chief nervous phenomena in my cases. The *physical signs* are neither constant nor characteristic; they are, in the main, those of cardiac dilatation. The *pulse* may be regular and of good tension, but after dilatation comes on it becomes irregular, frequent, and easily compressible. Moderate hypertrophy probably exists in the majority of cases, but cannot always be demonstrated owing to the extreme subpericardial over-fatness. A basic systolic murmur may be heard; it is not due to valvulitis as a rule. The *prognosis* as to cure is almost hopeless, although marked improvement may follow appropriate treatment. A fatal termination is often due to spontaneous rupture of the heart. The *treatment* must be directed especially to the over-fatness and the cardiac dilatation.

(*b*) **Brown Atrophy.**—A form of degeneration in which accumulations of yellowish-brown pigment-granules occur in the muscular fibers. The color exhibited by the heart-muscle is a reddish-brown, and in pronounced cases a dark-red brown. Brown atrophy is most commonly seen in the hearts of the aged, though also quite often in cases of chronic valvular disease that have reached an advanced stage.

(*c*) **Calcareous Degeneration** (*Calcification*).—Calcareous infiltration of the muscular fibers of the myocardium has been noted, though very rarely. Somewhat more common are the bony callosities that result from myocardial abscesses (*vide* Circumscribed Myocarditis).

(*d*) **Amyloid Degeneration.**—This form of degeneration is rare. It is limited to the blood-vessels and interstitial connective tissue; its causes are the same as those of amyloid degeneration of other viscera.

(*e*) **Hyaline Degeneration.**—This is sometimes seen in association with amyloid change. It also occurs independently in prolonged fevers (*hyaline transformation of Zenker*). The fibers are swollen, translucent, and homogeneous, and their striæ almost entirely disappear.

CARDIAC ANEURYSM.

(*Aneurysm of the Heart.*)

A CARDIAC aneurysm may involve either the whole diameter of the myocardium (aneurysm of the walls),[1] or merely the valves, together with a few myocardial fibers. Aneurysmal dilatation of the coronaries due to sclerosis or embolism is also recognized.

Aneurysm of the Walls.—This is not of frequent occurrence. Its most common seat is the wall of the left ventricle near the apex; it is quite generally a sequel to chronic myocarditis, which occurs oftenest at this point. In size cardiac aneurysms are exceedingly variable, and may either be very small or as large as the average-sized head of an adult. As to form,

[1] Of 87 cases collected by Pelvet, 57 were in this situation, and of 90 collected by Legg, 59.

two types should be recognized: (*a*) an equable dilatation of a part of the ventricular wall, and (*b*) the sacculated form. Layers of fibrin are often found in these aneurysmal dilatations—an indication of Nature's attempt at a cure, and occasionally she is successful. Once an aneurysmal distention has begun, a straining effort may cause sudden increase of its dimensions or rupture it. The structures adjacent to the aneurysm exhibit fibrous overgrowth. This condition may rarely be congenital. Males are more commonly affected (74 per cent.—Hare).

Diagnosis.—Aneurysm of the myocardium has no characteristic features. Usually the *symptoms* and *local signs* of chronic myocarditis or *dilatation* are more or less conspicuous, but the presence of the aneurysm is not even suspected unless certain physical signs develop. These are—a *pulsating prominence* in the apex region that may even perforate the chest-wall, and a coextensive dulness. The abnormal area of dulness, which is peculiarly circumscribed, is best appreciated early by stethoscopic percussion. An aneurysmal dilatation may also be confirmed by the *X*-rays or the orthodiagram. The *course* of these cases is unfavorable, death ensuing (rarely) from rupture of the sac or (more frequently) from gradual cardiac exhaustion.

Valvular aneurysms sometimes arise in acute ulcerative endocarditis, which destroys the segmented endocardium and permits of dilatation as the result of the intracardial blood-pressure. They occur with much greater frequency on the aortic than on the mitral valves. They are spheroid in shape, and project into the left ventricle when found at the aortic segments, and into the left auricle when at the mitral. Rupture of these aneurysms is common, with the subsequent development of valvular incompetency. They cannot be *diagnosticated* during life.

RUPTURE OF THE HEART.

This rare and serious accident may either be *complete* or *partial*. The term partial rupture implies laceration of the trabeculæ ventriculi, whereby the chordæ are liberated. Rarely, the papillary muscles are torn, causing valvular incompetency. Complete rupture consists in a solution of continuity of the total diameter of the myocardium.

Pathology.—The chief seat of rupture is the anterior wall of the left ventricle, though it may also occur in the right ventricle and in the auricles. The rent runs parallel with the muscular fibers, and is to a certain extent the result of laceration, although chiefly of a separation, of the fibers. The fissural communication presents irregular edges, and at autopsy is seen to contain blood-clots; the pericardial sac is also occupied by coagula. If pericardial adhesions have previously obliterated the cavity, the escaped blood-clots may occupy the pleural cavity. Histologic examination of the adjacent muscle-structure shows the characteristic changes of fatty and other forms of degeneration.

Etiology.—Both *predisposing* and *exciting* causes may be at work. The former are the more important and named in the order of their frequency of occurrence are,—disease of the coronary arteries (with anemic

necrosis and abscesses), fatty degeneration,[1] fatty infiltration, chronic myocarditis, parietal tumors, and parasites in the heart-wall.

The influence of *age* is notable; rupture of the heart usually occurs after the sixtieth year has been passed. Males suffer somewhat more frequently than females. The exciting cause is, as a rule, some form of muscular exertion, though it may occur during sleep.

Symptoms.—In the majority of instances rupture of the heart results in *sudden death*. Sometimes, however, the patient survives the accident for several hours or even for as many days. The symptoms are those of *internal bleeding*, and *pain* that may be agonizing and is referred to the heart. The body-temperature falls, the skin surface becomes pale and cool, and it may be covered with cold perspiration, while the *pulse* grows small, very frequent, and finally almost vanishes. Occasionally gastro-intestinal symptoms and syncope tending to convulsions appear in consequence of the irritation of the vagus centers due to cerebral anemia. The *physical signs* of cardiac failure rapidly develop, and, if the leak be not too large, those of pericardial effusion more gradually.

Diagnosis.—Heart-anguish, rapidly progressive cardiac failure, the evidence of internal hemorrhage, and the speedy development of the signs of pericardial effusion should always excite suspicion of rupture, and in many cases suffice for a correct inference.

The **prognosis** is hopeless. When immediately fatal, death is the result of heart-shock; it may result from anemia of the brain or compression of the heart by the effused blood.

Treatment.—*Prophylaxis* is of the utmost importance. The physician should give ample warning of the dangers connected with muscular strain of whatever sort. If rupture has either occurred or is suspected, the patient must be put at complete rest in the horizontal position. Full doses of morphin should be given hypodermically, and the ice-bag locally applied. Warmth to the extremities may be useful. The use of cardiac stimulants will be attended with increased bleeding from the rent, but agents that relax the peripheral arterioles, such as nitroglycerin, may be employed with a view to diminishing the heart's labor without diminishing its power. Should the rupture be partial and the hemorrhage slight, the patient's life may be prolonged, or even saved, by keeping him at absolute rest for a long period.

MINOR AFFECTIONS OF THE HEART.

(*a*) **New Growths.**—Primary carcinoma or sarcoma is rare indeed. Metastatic growths occur, but are very rarely sufficiently large (except perhaps the colloid variety) to be detected by *physical examination*, or to give rise to symptoms. Rarely, large tumors may weaken the heart. The separation of portions of the tumor may block one of the valvular orifices and cause sudden death, or more minute portions, becoming released, may give rise to embolism in distant parts.

(*b*) **Parasites.**—Four forms may invade the heart-muscle—the tænia echinococcus, actinomyces, cysticercus cellulosæ, and the pentastomum denticulatum. The former two only are productive of mischievous re-

[1] According to Quain's statistics, about 75 per cent. of the cases are due to this cause.

sults. The echinococcus growths may attain to considerable dimensions and are often multiple; they are secondary to echinococcus-cysts in other organs. Their effects are produced in a purely mechanical manner unless fragments become detached, when they may excite embolic lesions at different points in remote organs.

(*c*) **Misplacement** (*Transposition of the Heart*).—During intra-uterine life the heart (and rarely all the other thoracic and abdominal viscera) may either be transposed to the right side of the thorax, or the fetal position—in the median line—may be retained. The sternum may be missing in whole or in part, and the heart, which now lies immediately beneath the skin, can be seen and felt as a throbbing tumor. Recently a healthy man of about forty years applied at the Medico-Chirurgical Hospital in whom the lower half of the sternum was absent; his heart occupied a position in the median line directly underneath the skin.

Very exceptionally other anomalous positions are acquired during ante-natal development, and the heart may become displaced upward in the chest-cavity even to the neck or downward into the abdominal cavity.

(*d*) **Floating Heart.**—The structures that serve to maintain the heart in its normal anatomic relations may become weakened or unduly lax, in consequence of which the organ may exhibit increased motility.

III. NEUROSES OF THE HEART.

PALPITATION.

Definition.—A more or less rapid action of the heart that is perceptible to the patient, and usually accompanied by an increased force of the cardiac contractions or a disturbance of the rhythm, including extra-systole (*vide infra*), and often also by precordial distress, anxiety, and dyspnea.

Etiology.—Chronic valve-disease and other organic affections of the heart seldom produce palpitation. Among predisposing causes are—(1) Mental excitement, depression or emotion; (2) Anemia (from the local irritant action of the altered blood-state); (3) The acute infectious diseases, in which the toxins in the blood irritate the cardiac accelerating nerves; (4) Dyspepsia, even in robust-appearing persons (as in the gouty) who wittingly or unwittingly commit dietetic errors. Special articles of diet may excite over-action (*e. g.*, strawberries, shell-fish), the palpitation thus arising from reflex irritation being dependent upon gastric catarrh. (5) The use, and more especially the abuse of tea, coffee, alcohol, and tobacco. (6) The female sex manifests a greater disposition to the complaint than the male, especially about the period of puberty and the menopause. In the male it is most common at or after the middle period of life, a time when the effects of the work and worry of life show themselves. (7) Disturbances of the ovaries and other pelvic organs may induce palpitation reflexly.

Symptomatology.—Cardiac over-action, as a rule, displays a definitely *paroxysmal* character. The *onset* is sudden, and immediately preceding the attack there are often a blanching of the face and a slow-

ing of the cardiac action, symptoms due to the momentary inhibitory effect of the nerve affections that cause the "palpitation." The patient's *perception* of increased force and rapidity of the heart's action is the essential symptom. The patient may complain of *palpitation*, with a normally acting (or, more rarely, abnormally slow) heart, the symptoms being wholly subjective in character. *Mental anxiety* is common, and dyspnea, the latter symptom assuming curious phases.

Physical Signs.—*Inspection* shows the impulse to be somewhat diffuse and forcible. Visible throbbing of the superficial vessels is also common. The *finger-tips* easily appreciate the increased strength of the impulse. At the wrist the pulse, though strong and full, as a rule is rapid, the rate varying from 120 to 160 per minute. *Percussion* does not show the area of cardiac dulness to be enlarged as a rule, while *auscultation* reveals louder sounds than the normal. Anemic murmurs may be present. The attack is usually of brief *duration*—but a few minutes—though sometimes it may last for hours or days.

Attention should here be called to the *irritable heart* described by DaCosta—a form of palpitation common among young soldiers during the late Civil War. It was caused partly by mental excitement and partly by inordinate muscular exertion. A minor part in its production was also played by diarrhea. The leading symptoms were palpitation, a very frequent pulse, dyspnea, and cardiac pains of varying intensity.

Differential Diagnosis.—Nervous palpitation must be distinguished from the comparatively rare cases in which the heart contracts rapidly and irregularly, but does not excite subjective sensations. Some of the latter instances are to be looked upon as physiologic, while others are due to exhaustion and other causes. They do not constitute cases of palpitation, since they are unperceived by the patient.

Palpitation due to *chronic-valve disease* should also be differentiated. Here chief reliance is to be placed upon the presence of a murmur and other physical signs during the intervals between the attacks. The presence of a diastolic murmur would exclude nervous palpitation.

Prognosis.—The condition is free from real danger to life. Most authors, however, are agreed that cardiac hypertrophy may be a sequel.

Treatment.—The chief indications for treatment are—(1) *The arrest of the paroxysm.* The patient must be put at absolute rest in bed in a large, well-ventilated, darkened chamber, and his clothing loosened so that the respiration is unimpeded. Pressure upon the vagus in the neck or upon special points on the abdominal parietes (the ovarian region in particular) sometimes arrests the attack. An ice-bag applied to the precordial region is useful; it should be removed every third hour in protracted cases, and the patient be told to take large draughts of cold water or to swallow bits of ice. On the other hand, I have observed a few instances which were speedily relieved by the ingestion of hot and somewhat stimulating drinks.

Among the many drugs that have been employed, morphin alone has given good results, and particularly when administered hypodermically. However, before employing morphin, other sedatives and narcotics should be tried, such as the bromids (in large doses), hyoscyamus, hyoscin, and camphor monobromate. In hysteric subjects the bromids and the prep-

arations of valerian are highly serviceable. I have found the following capsule of great utility:

℞. Zinci valerianat., gr. x (0.648);
Strychninæ sulph., gr. $\frac{1}{3}$ (0.0216);
Ext. sumbul., gr. x (0.648);
Ext. hyoscyami, gr. v (0.324);
M. et ft. capsulæ No. x.
Sig. One after meal-time.

If a special article of diet or an overloaded state of the stomach is the cause, an emetic may be given and the attack thus speedily controlled. Oxygen-inhalations have been warmly advocated.

(2) *To prevent a recurrence of the paroxysms*, the causal conditions, some of which may long antedate the occurrence of palpitation, must be removed, if this be possible. All exciting factors must also be avoided. When cardiac palpitation occurs in neurasthenia and hysteria, the Weir-Mitchell rest-cure should be advised. If the heart be weak, digitalis may be exhibited. I have observed good effects from the use of baths (carbonated).

TACHYCARDIA.

(*Tachycardia Paroxysmalis; Synchopexia; Rapid Heart.*)

Definition.—A rapid movement of the heart occurring in paroxysms of variable duration, and directly dependent upon either paralysis of the pneumogastric or stimulation of the sympathetic nerves. Martius believes that the condition is attributable to sudden dilatation. Gordon[1] claims that tachycardia may be determined by dilatation of the splanchnic area, diminishing greatly the supply of blood to the left ventricle.

Pathology and Etiology.—It occurs as a physiologic condition in certain individuals; in such cases the pulse may range from 90 to 100 beats per minute or over. Certain persons can increase the pulse-rate by their own volition. The pathologic forms are divisible into—(1) Essential or neurotic tachycardia, and (2) Symptomatic tachycardia.

(1) **Neurotic Tachycardia.**—The causes of this variety are identical with many of those that excite palpitation. Thus, among disposing factors are hysteria, anemia, neurasthenia, chlorosis, and toxic agencies (tea, coffee, tobacco, the poisons of fevers). Violent exercise, intense mental agitation, fright, grief, and other forms of shock are determining influences. Not a few cases are met at or about the menopause.

(2) **Symptomatic Tachycardia.**—The lesions that induce this form are —(*a*) *central* and (*b*) *peripheral*. In the former group are especially to be placed tumors, clots (due to hemorrhage), and softening of the medulla and cord; and in the latter, tumors, aneurysms, enlarged lymph-glands (which paralyze the vagus by exerting pressure upon it either in the neck or thorax), and neuritis, affecting the pneumogastric nerve. The latter lesion may be associated with polyneuritis (alcoholic or infectious). Rapid heart may be due to *reflex irritation* (gastric, intestinal, arterial, uterine, ovarian), or gastro-intestinal intoxication.

[1] *British Medical Journal*, March 12, 1910.

Symptoms.—The clinical picture in most instances of the complaint is made up of recurring paroxysms of heart hurry (*paroxysmal tachycardia*). These attacks may come suddenly without prodromes. If the latter occur, they consist of vertigo, tinnitus, a sense of impending danger, and sometimes a "heart-flop" due to extra systole. The "flop," however, more commonly ends the paroxysm. With the onset of the paroxysms the *cardiac movements* leap to 150, 175, 200, and 250, or even

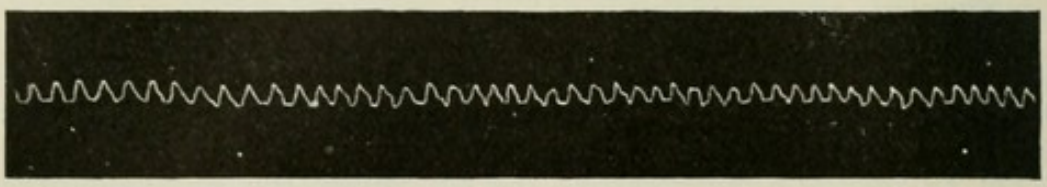


FIG. 56.—Radial pulse during an attack of paroxysmal tachycardia.

to 300 beats per minute. The *pulse* is feeble, small, readily compressible, as a rule, and sometimes irregular (Fig. 56). Rarely it is full, strong, and of good tension. The *respiration* may or may not be increased in frequency, but dyspnea is not common. At first pale, the *skin* soon becomes flushed, and the *countenance* may wear an anxious expression; but unless "palpitation" is associated there are no symptoms present that denote an intense degree of suffering. In many cases the patient is not conscious of palpitation, or there may be a sense of slowing of the heart, when in reality the cardiac contractions may be increased to 200 or more; this is *typical tachycardia*. In a chlorotic girl I found that the pulse-rate increased from the normal rate to 200 beats, and lasted for a few minutes at each visit to my office. H. C. Wood reports a case occurring in a physician in his eighty-seventh year, who has had attacks since his thirty-seventh year, the pulse rising.

Physical Signs.—A diffuse, rapid, and sometimes irregular impulse may be observed on *inspection* and *palpation*, but seldom is there an enlargement of the heart. The *sounds* are slightly modified, the first being accentuated and the second aortic greatly diminished in intensity, owing to the lessened amount of blood thrown into the aorta with each systole; the intensity of the second pulmonic, however, may be increased. An apical *systolic murmur* is occasionally audible. The carotids pulsate, and on auscultating over them a murmur is sometimes heard.

Diagnosis.—I would restate the fact that a high pulse-rate (200 or over a minute) and an absence or only a slight sense of palpitation or rapid heart-action are the distinctive features of true tachycardia. In *palpitation* (previously considered) the pulse-rate is not usually so high, while the associated phenomena of dyspnea precordial constriction, smothering, and painful anxiety are correspondingly more pronounced.

Prognosis.—In the majority of cases no serious impairment of the general health follows, though the course is exceedingly chronic and recoveries are comparatively rare. The *duration* of tachycardia varies from one to two or more decades. When symptomatic tachycardia is due to lesions that are removable, it is often curable, though not invariably so.

The **treatment** is to be conducted on the lines advanced for "Palpitation" (*vide* p. 704). Fairbrother has cut short the paroxysm in his own case by either walking or an exercise like a girl skipping the rope.

An abdominal binder, with a view to emptying the splanchnic vessels, may reduce the excessive rate of the heart. The attacks can be averted by the taking of ice-water or strong coffee.

BRACHYCARDIA.

(*Bradycardia.*)

Definition.—Slowness of the pulse. The condition may be physiologic, the rate of the pulse being sometimes 60 or less, and very rarely as low as 40 per minute during perfect health.

All cases of pathologic brachycardia fall naturally and conveniently into two groups: (1) those that are secondary to other complaints (*symptomatic brachycardia*); and (2) those that are due to a neurosis.

Pathology and Etiology.—Symptomatic Brachycardia.—(*a*) Arising during convalescence from acute infectious diseases, especially *pneumonia*, *typhoid*, *diphtheria*, *influenza*, and *acute rheumatism*. According to Riegel, who analyzed 1047 cases, the *acute fevers* must be awarded the first place among the causal factors. I have met 3 cases of diphtheria in which the pulse fell to 30 a minute. That such instances are, as Traube contends, due to exhaustion is true of some cases, but not of all. The slowing of the pulse that is observed after premature or full-time delivery is similarly produced. (*b*) The second place belongs easily to gastro-intestinal and hepatic disorders (*chronic gastro-intestinal catarrh*, *ulcer*, or *carcinoma of the stomach*). (*c*) Brachycardia occurs in diseases of the circulatory system—in *coronary disease*, fibroid and fatty myocardial change, most frequently; and chronic valvular disease much less frequently, if we except aortic stenosis. (*d*) Pulmonary complaints (emphysema and asthma). (*e*) Toxic agencies, as in jaundice, blood-poisoning, alcoholism, the unwonted use of tea, coffee, tobacco, and a few drugs (*e. g.*, digitalis, strophanthus). (*f*) Constitutional affections (anemia, chlorosis, gout, diabetes). (*g*) Rarely skin diseases and affections of the sexual organs, and commonly myxedema, are associated with brachycardia. (*h*) In various organic nerve affections (apoplexy, meningitis, epilepsy, tumors of the cerebrum and the medulla, injuries, and diseases of the cervical portion of the cord). Brachycardia is produced by direct or reflex irritation of the center or peripheral portion of the vagus, except in cases in which it is brought about by exhaustion of the automatic motor apparatus of the heart. The condition is more common in men.

(2) **Brachycardia associated with a neurosis** may be found to be marked in *epilepsy*; less so in *hysteria*, *melancholia*, *mania*, and *general paresis* of the *insane*. It precedes palpitation.

Symptoms.—The sole characteristic symptom is the *slow action* of the heart, and this may either be temporary or permanent. If *paroxysmal*, both the onset and termination are apt to be sudden. A slow emergence is, however, more common than a slow beginning. Among prodromes are: vertigo, tinnitus, and a sense of impending danger. During the paroxysm the patient may suffer from *syncopal attacks* or become *unconscious* for hours at a time; *physical prostration* may be marked, and especially when secondary to chronic valve-disease. The *pulse* is weak and small, and the beats per minute vary from 50, 40, 30,

20, to 10, or even 8. When the condition arises in the course of organic valve-lesions the *cardiac contractions* may be increased in power, though greatly reduced in frequency. The heart-action must be noted by auscultation, and the rate compared with that of the peripheral pulse. The *impulse* and the *heart-sounds* are feeble.

Diagnosis.—A pulse below 48 beats per minute, with corresponding slowness of the systole, suffices for a certain diagnosis.

The **prognosis** is governed by the cause, being very grave in cerebral and advanced cardiac diseases. When fatal, sudden death is the rule.

Treatment.—Rest in the recumbent posture, particularly if the condition complicates organic heart disease, and such remedies as atropin, strychnin, caffein, nitroglycerin, and ammonia are to be tried. Since atropin paralyzes the vagus terminals in the heart, a marked increase of the heart-rate after the exhibition of atropin points to an extracardial cause of the bradycardia. In the intervals between the attacks the general health must be improved and the causal states eradicated.

ARRHYTHMIA.

(*Irregular Heart- and Pulse-beat.*)

Our knowledge of this subject has been advanced by the investigations of Wenckebach and by J. Mackenzie, Walter B. James, Lewellys F. Barker, and others as the result of observations with the electrocardiogram. James suggests that the cases be classified into rhythmic irregularity and arrhythmic irregularity. Doubtless, the myogenic theory explains the functional rhythmicity of the heart's action. Among recognized functions of the heart muscle are excitability, stimulus production, contractility, conductivity, and tonicity (Gaskell). As shown by Mackenzie and others, not all of the heart muscles are equally endowed with these functions. The clinician must attempt to correlate the symptoms of the different forms of arrhythmia with the condition of the different functions of the cardiac muscle.

(1) **Extra-systole.**—(*a*) Intermittent heart-beat. This signifies a missed or dropped beat. This occurs at irregular intervals in most of the cases, though sometimes a cyclical irregularity is observed —*i. e.*, every second, fourth, sixth, or eighth beat being lost. To explain this variety of arrhythmia we have the so-called "refractory phase" of the heart-muscle, in which it lacks excitability. Again the stimulus may be insufficient to excite a normal degree of contractility. In the absence of the refractory stage (beginning shortly before the systole and continuing a short time after it) the ventricle may again contract during this phase, producing an extra-systole following which there is a long pause. As explained by Engelmann, this long pause is a consequence of an extra-systole, the ventricle being still in the refractory stage when the next physiological stimulus reaches it, and it is not until the following stimulus arrives that contraction can again be produced. It is probable that the various forms of arrhythmia described below are due to the occurrence of these extra-systoles. (*b*) Twin-pulse (*coupled beats, allorrhythmia*). When two beats follow each other quickly (the diastole being shortened), and the next two not so quickly (the diastole being

lengthened), we have produced the *pulsus bigeminus*. The first and second beats may be of equal strength, but often the second is relatively feeble. This is best determined by auscultation of the heart, since the second systolic contraction (of the ventricle) may indeed be so weak as not to give rise to a palpable beat at the wrist. I have frequently observed the *pulsus bigeminus* in mitral disease. With respect to the diastole, the approximated pulsations may be in blocks of three (*pulsus trigeminus*), or even of four (*pulsus quadrigeminus*). (2) **Combined irregularity** of time and volume. (3) The **paradoxical pulse** of Kussmaul also consists in irregularity of volume, strength, and time, though not indicative of so great peril as the preceding. It is dependent upon the act of inspiration—the beats during inspiration being more rapid, though weaker, than during expiration (*vide* adhesive pericarditis, pleuro-pericarditis). (4) **Delirium cordis** is a term very appropriately given to great irregularity and inequality of the pulse-beats. It is seen in extreme dilatation and advanced exophthalmic goiter. (5) **Embryocardia or Fetal Heart-rhythm.**—There is a shortening of the long pause with a striking similarity of the first and second sounds, as in the fetal heart. I have already pointed this out in connection with cardiac dilatation. (6) **Cantering Rhythm** (*bruit de galop*).—The sounds simulate the triple footfall of a horse at a canter. The interpolated sound is due to a re-

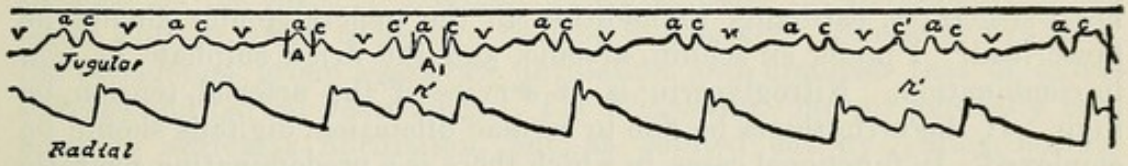


FIG. 57.—Extra-systoles of ventricular type at *c'* and *r'* (Mackenzie).

duplication of the second, though rarely it is the first that is doubled instead. It is developed in the hypertrophy of arterio-sclerosis and Bright's disease, in profound anemias, and in the myocarditis of certain acute infectious diseases.

Etiology.—Baumgarten's classification of the causes of arrhythmia (quoted by Osler) is the best, and is here given:

(1) Those due to central—cerebral—causes, either organic disease, as in hemorrhage or concussion, or more commonly psychical influences.

(2) Reflex influences, such as produce the cardiac irregularity in dyspepsia and diseases of the liver, lungs, and kidneys.

(3) Toxic influences. Tobacco, coffee, and tea are common causes. Various drugs, as digitalis, belladonna, and aconite, may induce it.

(4) Changes in the heart itself. (*a*) In the cardiac ganglia. Fatty, pigmentary, and sclerotic changes have been described in cases of this sort. (*b*) Mural changes are common in conditions of this kind. Simple dilatation, fatty degeneration, and sclerosis are most commonly present. A high blood-pressure may be the cause of extra-systoles.

Symptoms.—Arrhythmia, particularly when functional or of reflex origin, may exist for many years. Symptoms referable to the heart may be absent, although in some instances the extra-systole causes a thud to be experienced.

Diagnosis.—*Palpation* and *auscultation* of the heart while examining the pulse are matters that should never be neglected if reliable results are to be obtained. Sphygmograms will often show the kind and degree of arrhythmia and also distinguish marked dicrotism from irregularity.

It is important to *differentiate* functional arrhythmia or that of reflex origin from arrhythmia due to more or less grave myocardial disease. Important information is supplied by carefully reviewing the varied etiologic factors and close observation of the cardiac symptoms.

The **prognosis** is variable. A gentleman with whom I am acquainted was rejected by a life-insurance company twenty-five years ago on account of occasional slight arrhythmia, though he is still in active business life. When the myocardium becomes involved, as occurs in chronic valvular and coronary disease, the prospect is gloomy; on the other hand, when it is functional or due to extracardial causes, the course pursued is, as a rule, favorable. When the second sound follows closely the first (marked abbreviation of the systolic pause) it is a serious indication.

Treatment.—There are many cases of the more benign form in which no treatment is required apart from methodic, physical training to improve the strength of the heart-muscle and the general systemic development. Removal of the causal forces, as tea, coffee, alcohol, indigestible food-stuffs, conditions acting in a reflex manner, must be executed promptly. When the condition is due to changes in the heart-structures, cardiants in addition to the general tonics should be prescribed. I prefer strychnin, arsenic, and the dried sulphate of iron in combination. Nitroglycerin is of service if the arterial tension be high. If the arrhythmia be due to cardiac dilatation, digitalis should be employed. In functional cases, in which there is a predominating neurotic element, the subjoined formula has been useful in my hands:

℞. Ferri valerianatis,
Zinci valerianatis, *āā*. gr. xxx (1.94);
Strych. sulph., gr. j (0.0648);
Pulv. digitalis, gr. viij (0.518).
Ft. capsulæ No. xxx.
Sig. Take one after meal-time.

AURICULAR FIBRILLATION.

Definition.—A condition due to abnormal impulses arising in different auricular areas, and replacing the normal regular stimulus which is produced by a single area.

The condition is closely connected with extra-systoles, auricular flutter, and heart-block. The systoles of the auricle and ventricle do not follow one another regularly. The ventricle receives, instead of an abnormal stimulus or impulse, as in extra-systole, a variety of impulses, and in endeavoring to respond to these "increases its rate and its contraction becomes entirely irregular" (Talley). There are no constant or definite morbid lesions, and in some of the cases nothing abnormal is noted. Certain microscopic changes in the auricular tissue are most commonly met with—*e. g.*, atrophy of the muscle cells and a diffuse fibrosis.

Etiology.—Lewis' analysis of 126 cases showed an antecedent his-

tory of either rheumatism or chorea in 70 per cent. Among non-rheumatic subjects it was more frequent in males than females, and was commonest in advanced life. In the rheumatic type the vast majority of patients are from twenty to fifty years of age. Of Lewis' series of fibrillating cases, 52 per cent. presented mitral stenosis. Other cardio-vascular conditions with which fibrillation is associated are mitral regurgitation, aortic disease (rare), myocarditis, and interstitial nephritis.

Symptoms and Diagnosis.—In general, the ventricular rate varies from 100 to 160. With normal conduction the rate may reach 200 or over, while impairment of this function may reduce it to 40 or even less. An accurate diagnosis requires the use of the electro-cardiogram. Graphic curves, both arterial and venous, show an irregular succession of strong and weak contractions. The ventricular form of venous pulse is present, but this is also found in tricuspid regurgitation and certain forms of heart-block.

Auricular fibrillation is also often recognizable by clinical observation. The pulse is rapid, usually over 120 per minute, and irregular; this is best appreciated by the stethoscope at the apex. The associated phenomena and cardio-vascular lesions, often grave and indicating cardiac failure, are of great diagnostic import.

From other forms of arrhythmia, such as extra-systoles and partial heart-block, auricular fibrillation is distinguishable by its greater persistence by the effect of exercise, which increases the irregularity of the latter while it tends to remove that of the former. Similarly drugs of the belladonna group aggravate fibrillation and diminish that of extra-systoles and partial heart-block. The presystolic murmur generally vanishes in auricular fibrillation, but, as pointed out by Talley, not invariably.

Prognosis.—This depends upon the associated conditions, the ventricular rate, and the response to treatment. If the patient is willing not to make any unwonted exertion, a guardedly favorable prognosis is permissible in many cases.

Treatment.—Rest is an important item of treatment. Digitalis acts most satisfactorily, more particularly in the rheumatic forms. On the other hand, in cases of myocardial degeneration, this drug may fail. The rate of the pulse serves as a guide to its administration, and when normal again for the individual it should be discontinued. The hygienic and dietetic details are quite similar to those recommended in chronic valvulitis (*vide supra*).

Auricular Flutter.—This term implies extremely rapid action of the auricle, the rate being usually between 200 and 330. The condition is closely related to auricular fibrillation and tachycardia of less rapid rate, and particularly to heart-block. It may be paroxysmal in character. In general, the ventricular rate is either one-half or one-fourth that of the auricle, or the impulse to the ventricle may be quite irregular. Fulton [1] reports a case of the paroxysmal type lasting seven years with progressive improvement. Digitalis should be cautiously employed in the treatment, although in the presence of evidences of heart-block the atropine series deserve a trial.

[1] "Auricular Flutter," read by Dr. Frank T. Fulton before the American Climatological Association, May 6, 1913.

STOKES-ADAMS DISEASE.

(*Heart-block.*)

The syndrome known by the above name was first recognized by Adams (1827), although more accurately described later by Stokes (1846). It is characterized clinically by bradycardia, vertigo, syncope, and auricular impulses in the veins of the neck. The Stokes-Adams syndrome may rarely be absent in fatal cases.

Physiologic Pathology.—Physiologists have conclusively shown that the rhythmic contractions of the heart have as their basis a stimulus conducted not from the nerve-centers of the organ but from the sinus region to the auricle and ventricle. Gaskell's experiment elucidates the pathogenesis of the condition in man; he showed that constriction of the circular layer of muscle at the auriculo-ventricular junction in the heart of tortoises causes a cessation of the rhythmic action of the heart so that the auricles and ventricles become independent in their contractions, the former beating more rapidly than the latter. The impulse in the human subject flows through a bundle of muscular tissue extending from the right side of the interauricular septum to the interventricular septum just below the *pars membranacea*; it is approximately 18 mm. long, 2 mm. broad and 1.5 mm. thick. Now, if this pathway for the impulse is blocked the phenomena of Stokes-Adams disease are produced. Erlanger has been able to gradually compress the bundle of His and bring about varying degrees of heart-block: *e. g.*, at first there occurs an occasional failure of ventricular contraction, then a ratio of auricular to ventricular beats of 2 to 1, 3 to 1, 4 to 1, and finally complete block when the ventricles contract slowly and independently of the auricular rhythm.

Various *pathologic changes* in the bundle of His have been found post-mortem, thus confirming the results of physiologic experiment. For example, Stengel found an atheromatous patch over the bundle of His; Ashton, Norris, and Laveson and others a gummatous involvement of this structure, while Walter James[1] noted recent ulceration. *Temporary and incomplete heart-block* has been noted in certain acute infections (typhoid fever, diphtheria, influenza, pneumonia, and others) and in the fibroid heart. It may also follow the prolonged use of digitalis.

Symptoms.—The important features are: (*a*) Bradycardia, (*b*) cerebral attacks, and (*c*) visible auricular pulsation of the cervical veins.

(*a*) Bradycardia—the slow pulse is a persistent feature in most cases, but it may be paroxysmal. The rate falls to forty, thirty, twenty, or even less beats per minute, and it often bears a definite relation to the normal for the sufferer. The pulse is scarcely influenced by exercise or drugs that quicken the heart action. Arrhythmia of the ventricular contractions may be noted, but is not common.

(*b*) We often observe a 2 to 1 or 3 to 1 rhythm on comparing the auricular impulses as noted in the veins of the neck with the ventricular systole. This is due to the fact that most of the former fail to cross the bundle of His. Feeble auricular sounds may be heard, when the ventricle is in asystole. The blood-pressure is notably increased (De Renzi).

(*c*) The cerebral attacks consist principally of vertigo, which is usually momentary, syncope, rarely convulsive seizures, and pseudo-apoplectiform attacks. The attacks of unconsciousness may prove fatal.

[1] *Amer. Jour. Med. Sciences*, October, 1908.

Renal changes and albuminuria have been observed.

Diagnosis.—*Bradycardia* must be distinguished from Stokes-Adams disease. The former is characterized by a pulse below forty-eight beats per minute, with corresponding slowness of the systole and the auricular impulse, and it has a different etiology (*vide ante*). Doubtless, atypical cases of the Stokes-Adams syndrome occur, due to slight degenerative changes in the bundle of His, and these may exist for years before the clinical picture becomes typical.

Recurring extrasystole, simulating heart-block, may be due to hyperrhythmicity of the atrioventricular bundle and not due to blocking of this structure (*functional variety*). Here there occurs a forcible venous pulsation in the neck "without either a radial pulse or a discoverable pulsation in the innominate artery" (James). Seven cases have been recorded in the literature.[1]

The **prognosis** is grave in cases showing anatomic lesions. There are instances in which the bundle of His shows no pathologic changes (Edes and Councilman). The functional form gives a favorable prognosis.

Treatment.—This consists in rest in bed and in overcoming the feeble condition of the circulation by free stimulation. In cases presumably due to syphilis, the iodids in massive doses should be given. In the functional form, atropin changes the ratio between auricular and ventricular beat, so as to cause the heart-block to disappear.

ANGINA PECTORIS.

(*Stenocardia, Breast-pang.*)

Definition.—A paroxysm of violent precordial pain extending into the neck, back, and left arm, and at times attended by a sense of impending death. It scarcely deserves to be classified as a separate disease, being merely symptomatic of either cardiac or aortic lesions.

Pathology.—It is claimed to be a neurosis affecting the cardiac sensory filaments that are given off chiefly from the pneumogastric, and in many cases the vasomotor apparatus is also involved. Albutt[2] believes that the symptoms owe their origin to disease in the aorta—acute and chronic aortitis. Sudden anemia of the myocardium consequent upon sclerosis of the coronaries, and irritation of the ganglia and sensory nerves explain its origin (Leube). May[3] suggests chemical change in the myocardium as the stimulus to the nerve-endings, while Bramwell believes spasmodic contraction of the muscular fibers of the left ventricle, due to a sudden strain in cases of atheroma of the coronary arteries, to be the cause. Heberden first made the observation that angina is essentially a vascular pain, and showed its extra-cardiac origin.

Etiology.—Cases of angina unassociated with *arterial selerosis*, *hypertrophy*, *aortic regurgitation*, or adherent pericardium, are rarely encountered. With few exceptions sclerosis of the aorta and coronaries is present, and predisposes to the condition. This view receives some degree of color from the fact that angina usually occurs after the *fortieth year*, and principally in the *male* sex. Angina pectoris in the young

[1] See Pepper and Austin, *Amer. Jour. Med. Sci.*, May, 1912, 716.
[2] *Phila. Med. Jour.*, June 30, 1900. [3] *Brit. Med. Jour.*, January 1, 1910.

should always suggest syphilis. It may be a sequel of influenza. Dyspeptic disturbances may be responsible for, or at least aggravate, the disease. The overuse of tobacco may rarely cause it. The *determining factors* of the attack are undue exertion and mental emotion.

Symptoms.—The *paroxysm* begins quite suddenly during the action of one or other exciting cause. There is excruciating pain of a grip-like character, affecting the entire chest and rendering the body motionless. The pain *radiates* most frequently to the left shoulder, though also to the right, and thence to the back, neck, and down the arms to the fingers. Not less agonizing than the pain is the awful sense of *impending death. Coldness* and *numbness* of the fingers or in the precordial area may be present. The *countenance* is frequently pale, and may assume a leaden hue, and is usually bathed in cold perspiration. The *respirations* are shallow or even temporarily arrested, and the patient's anxiety is extreme. The *heart's action* may be regular, and the arterial tension is generally increased. The *duration* of the paroxysm varies from a few seconds to a minute or two (rarely it lasts for hours), and after the attack, which subsides suddenly, gaseous eructations, vomiting, or the discharge of a large amount of clear urine may occur. Cheyne-Stokes breathing has been observed. After the seizure there may be an *absence of signs*, and, though there is weakness, this soon disappears. The attack may *recur* at intervals varying from a few days to as many years.

Varieties.—1. **Angina major,** or severe form, in which arterial disease is uniformly present. Its subvarieties are thus classified by Osler: (*a*) the fulminant or rapid form with one or two attacks only, or sometimes with the condition known as status anginosus; (*b*) the form with a duration of one and a half to two years and a varying number of attacks; (*c*) the chronic form, lasting upward of ten years with a varying number and growing intensity of attacks, that, for instance, of John Hunter, whose first seizure occurred twenty years before his death; and (*d*) lastly, the small group of cases which may last for months, or as long as two years, with attacks of great severity, and yet may completely recover.

2. **Angina minor,** or mild form, usually described as "false or pseudo-angina," with its well-known subgroups, the neurotic, the vasomotor (Nothnagel), and the toxic forms.

In *angina vasomotoria* the pain in the heart-region is preceded for a few minutes by pallor of the face, coldness and stiffness of the limbs, due to spasm of the peripheral vessels.

Angina pectoris without pain (*angina sine dolore*) may occur, the main feature is an indescribable, almost painless sensation or precordial oppression.

In addition, *angina abdominis* is recognized, and simulates the gastric crisis of locomotor ataxia.

Diagnosis.—The characteristic events are a sudden, intense pain in the substernal and left parasternal regions with marked constriction of the chest, the peculiar manner of radiation of the pain, and the fear of death. Less diagnostic, though of considerable value, are the brevity of the attack, its sudden cessation, the age and sex, and the anxious, moistened features. There are light forms, in which one or more of the diagnostic phenomena above described are absent. If they occur between the ages of forty and sixty years in persons in whom either arterial scle-

rosis or aortic regurgitation is present, this disease should be thought of; and after the exclusion of certain complaints in which paroxysmal pain is prominent, such as gastralgia, intercostal neuralgia, and locomotor ataxia, the diagnosis of angina becomes reasonably certain.

The term pseudo-angina pectoris is probably a misnomer in the present state of our knowledge, and apparently typical cases have been observed to merge into true angina pectoris.[1] It must be confessed that one meets with hysterical and neurasthenic females, in whom paroxysms of diffuse pains over the thoracic region, accompanied by restlessness and emotional disturbance, and lasting from one to several hours, occur. These cases, however, have nothing in common with angina pectoris.

The **prognosis** is bad, yet uncertain. When the arteries are sclerosed (particularly the coronaries) life is often suddenly terminated. Occasionally the sufferer dies of syncope. The nature of the causal and associated lesions must be considered, *e. g.*, when it accompanies aortic incompetency, the prospect of life is bad. In the vasomotor angina of Nothnagel the outlook is less grave.

Treatment.—*Prevention* of the attacks in persons who are subject to them is of the utmost importance. The exciting factors are to be avoided. The patient should be instructed to carry constantly such agents as nitroglycerin and amyl nitrite *perles* (strength 3 to 5 drops), and how to use them.

The *treatment of the attack* must be prompt and energetic, though carefully conducted, amyl nitrite being inhaled at once from a handkerchief in doses of 3 to 5 drops according to the severity of the attack. The patient should then be placed in a cool apartment at absolute rest in bed, with restriction of food. *Locally*, the use of the ice-bag may prove efficacious and should be tried at first. Rarely, hot applications (hot cloths or sinapisms) give better results than cold. If the pain is not controlled promptly by this method, the nitrite should be reinforced by the hypodermic injection of morphin (gr. $\frac{1}{3}$—0.0216) combined with atropin (gr. $\frac{1}{120}$—0.0005). This usuully brings speedy relief, and is best suited to those instances in which there is no increase of arterial tension. In cases exhibiting high arterial tension the spirits of nitroglycerin, hypodermically, should be employed (dose ♏j—0.066, to be repeated once in a minute if needful).

During the *intervals between the attacks* the aim should be not only to obviate the action of the exciting causes, but also to overcome any predisposing influences that may exist. Prolonged rest in bed should be enjoined in true organic cases, and in those who cannot walk without pain. Excessive tobacco smoking must be discontinued. Schott[2] prefers baths (effervescent) and passive movements to drugs or other methods. In true angina, passive movements alone should first be performed by an assistant, but later may be safely entrusted to the patient. In cases in which the arterial tension is habitually exalted, nitroglycerin in increasing doses is to be used perseveringly, beginning with ♏j (0.066) and increasing by ♏j (0.066) every five or six days until the physiologic effects are produced. Sodium nitrite may be employed alternately with the nitroglycerin, the dose being gr. j–iij (0.0648–0.184) three or four

[1] "Angina Pectoris, True and False," *Jour. Amer. Med. Assoc.*, Nov. 3, 1906, by the writer.

[2] *Medical Record*, March 11, 1899.

times daily. Marked arterial sclerosis, particularly if there be a syphilitic history, is favorably influenced by a long course of potassium iodid. In cases in which there is quick recurrence, sodium nitrite in 2½-gr. tablets (dose, 1 to 4 tablets) is recommended. Albutt lauds—(*a*) the high-frequency current, and (*b*) the administration of the lactic acid bacillus. When hypertrophy of the left ventricle is excessive, I use the following:

℞. Tr. aconiti rad., ♏xlviij (3.10);
Sodii bromidi, ʒss (16.0);
Elix. simplicis, q. s. ad ʒiij (96.0).—M.
Sig. ʒj (4.0) t. i. d.

It may be omitted at the end of every two weeks for two or three days. The presence of a gouty diathesis would call for special treatment. Dyspeptic troubles should be rectified. For Cheyne-Stokes breathing Albutt advises the inhalation of oxygen and carbon dioxid alternately. Venesection may be employed in high arterial tension.

In the *vasomotor* form amyl nitrite and nitroglycerin are most valuable. Additionally, hot foot-baths are also of the highest utility.

IV. CONGENITAL AFFECTIONS OF THE HEART.

These result from two leading causes: (1) Arrested development, and (2) Fetal endocarditis. Occasionally, both these factors are operative.

(1) **Arrested development** may produce a great variety of anomalies: (*a*) Acardia, absence of the organ. (*b*) *Cor biloculare*, or *reptilian heart*, in which the septum between the auricles and ventricles is absent, thus reducing the number of chambers to two. (*c*) *Absence of the interventricular septum*, the heart consisting of three chambers (*cor triloculare*). (*d*) *Patency, or incomplete closure of the foramen ovale.* Persistence of the foramen is, in the majority of cases, associated with obstruction of the pulmonary valve, though it may be solitary. (*e*) An anomaly known as *ectopia cordis* deserves mention. The sternum is usually divided vertically, and the heart is either entirely exposed or beating just beneath the skin in the cardiac, thoracic, or abdominal region. The most common form of malposition, however, is *dextrocardia* (*vide supra, misplacement*, p. 703). (*f*) *Anomalies of the valves.* There may be either a numerical increase or decrease of the cardiac valves, particularly the segments of the semilunar valves of the aortic and pulmonary orifices. Supernumerary segments are usually rudimentary.

(2) **Fetal Endocarditis.**—The valve-lesions originating during fetal life are most frequently situated on the right side. They may occur at the pulmonic, the aortic, or the auriculo-ventricular orifices. The changes are of the sclerotic form. The leaflets present smooth, thickened, and contracted borders. Union of the mitral segments is common, and the chordæ are often thickened and contracted.

The most frequent congenital valvular lesion is **stenosis of the pulmonary orifice** as the result of chronic endocarditis. Rarely, it is due directly to defective development, and perhaps more rarely still to endocarditis verrucosa. **Pulmonic constriction** of antenatal origin may be an associated lesion in other forms of valvular disease in the young adult. With

stenosis at this orifice, there usually coëxist stenosis of the conus arteriosis of the right ventricle, an open foramen ovale, and a patent ductus arteriosus; according to Peacock, "in 86 per cent. of the patients with congenital heart disease living beyond the twelfth year the lesion is at this orifice." **Atresia of the pulmonary orifice** occurs, though rarely.

At the **tricuspid orifice** there may be stenosis or contraction of the valves, producing either obstruction or regurgitation. Similar lesions of the aortic orifice are infrequent. **Congenital mitral disease** occurs only exceptionally; it is usually associated with tricuspid stenosis. Boys are more liable to congenital affections of the heart than girls.

Symptoms.—There is a constant and striking symptom in congenital heart-disease—*cyanosis*. The *tint* of skin observed is variable, being at one time a general duskiness, at another a deep violet, and rarely almost black. This coloration is noted about the lips and mucous membrane of the mouth, the nostrils, conjunctivæ, the fingers, toes, and lobules of the ears, and, as a rule, is general, though it may be a local condition. The tint may grow less distinct, when the child is in perfect repose or sleeping; excitants or efforts at coughing, however, increase the intensity of the discoloration. The cyanotic hue comes on almost invariably during the first week of life. The *fingers* present a decidedly clubbed appearance, and the *nails* are thickened and claw-like. The *temperature* is subnormal, while the extremities are cool to the feel. *Dyspnea* on exertion and *cough* are usual concomitants. Variot reports two cases, namely, interventricular perforation and narrowing of the pulmonary artery. Cyanosis was absent from one case, and "this disproves the two leading theories with regard to the origin of cyanosis—the mixture of the two bloods and the obstruction to the pulmonary circulation."

Physical Signs.—In the very young the impulse is feeble, the *percussion-dulness* is increased, especially to the right, and a loud *systolic murmur* is audible at the pulmonary orifice. When the auriculo-ventricular valves are the seat of endocarditis, the murmur may be apical. In pure pulmonary stenosis the second sound is feeble.

In older children the area of *dulness* is only slightly extended, particularly to the left, while the *murmurs* heard are loud and often musical.

In rare instances *cerebral abscess* is an associated condition.

Differential Diagnosis.—

Congenital Lesions.	Acquired Lesions.
History of almost constant cyanosis, beginning in the first week after birth.	Not so; history of endocarditis or of rheumatism or other complaints in which endocarditis occurs as a complication.
Slight enlargement of the heart. It is the right ventricle and non-progressive.	Enlargement marked, frequently involving the left ventricle, and progressive.
Loud and musical murmurs present, audible over upper third of sternum, with small area of transmission upward and to the left; second sound weak.	Audible over apex or base; definite large areas of transmission. Second sound frequently accentuated.
Deficient bodily development.	Bodily development good, as a rule.
Mental faculties in abeyance.	Mental faculties normal.

Prognosis.—The prognosis is exceedingly grave. Many succumb within a few days after birth, more than one-half before the expiration of one year, and not less than three-fourths before the end of the third

year. Few survive the first decade of life, and fewer still reach full adolescence. The form giving the most favorable prognosis is pulmonary stenosis with defective septa. There is a disposition to affections of the lungs (phthisis), nerve-complaints (convulsions).

Treatment.—The treatment is, in the main, hygienic. The body must be warmly clad. The diet is to be judiciously arranged, yet liberal. Gentle exercise, when it can be taken, is valuable, as are also daily spongings of the surface followed by friction. Special therapeutic indications may arise, and must be met in accordance with general principles.

V. DISEASES OF THE ARTERIES.

ACUTE AORTITIS.

Pathology.—The morbid changes coincide with those noted in acute endocarditis, including the ulcerative variety.

Etiology.—The causes are not clear, but the condition generally follows the acute infectious diseases (typhoid fever, pneumonia, miliary tuberculosis). Alcoholism and syphilis are among the rarer causes. Various microörganisms have been discovered to be causal irritants. Boinet and Romary have recently shown that in experimentally produced aortitis a point of lessened resistance (either from traumatism or other previous arterial lesion) is necessary.

Symptoms.—The symptoms are *local* and *general*. Of the former, diffuse thoracic *pain*, with substernal *tenderness* under pressure and cardiac *palpitation*, are the chief. The pain may assume the type of true angina pectoris. Among the *general* symptoms a moderate febrile movement is almost constant. In a certain percentage of cases embolism is betrayed by the usual signs, as rigors, accompanied by a steep temperature-curve. These forms are analogous to the malignant variety of endocarditis. A cardiac murmur may be heard over the base.

Diagnosis.—All that the best clinicians can do is to establish a probable diagnosis even in the presence of the most frankly expressed features of the affection. From *acute endocarditis*, aortitis is to be discriminated by its diffuse pain and by the higher seat of its murmur.

The **prognosis** is serious, owing to the liability to infectious emboli and aneurysmal dilatation and the possibility of aortic rupture.

The **treatment** is similar to that of acute endocarditis.

ARTERIAL SCLEROSIS.

(*Arterio-sclerosis; Arterio-capillary Fibrosis; Endarteritis Chronica Deformans; Atheroma.*)

Definition.—An atrophic degeneration of the median arterial coat followed by compensatory thickening (hyaline) of the intima.

Pathology.—The most frequent seat of the sclerotic process is the aorta, and the next most common the coronary arteries. Other vessels implicated are the arteries of the brain, the temporals, radials, brachials, ulnars, femorals, and iliacs. On the other hand, certain arteries, as the

gastric, hepatic, and mesenteric, are rarely affected. Two forms may be recognized: (*a*) the circumscribed (atheroma) and (*b*) the diffuse. There is also a secondary variety due to hypertension, causing dilatation of the vessels, slowing of the current, and compensatory thickening of the intima.

(*a*) **Circumscribed Arterio-sclerosis.**—Naturally, the intima presents a smooth internal surface, but when atheromatous changes occur it shows localized areas of thickening, often hemispheric in outline, yellowish-white in color, and their favorite seats are the orifices of the branches. They increase in depth and superficial area, and on reaching an advanced stage their interior disintegrates into granular material (*atheromatous abscess*).

Histologically, in circumscribed or nodular atheroma, the middle coat is the primary seat of the changes, which consist of localized infiltrations. These lesions weaken the media and then (as shown by Thoma) compensatory processes are set up in the intima and adventitia (Adami), which lead to the formation of the so-called *atheromatous button*. The latter consists in a hyperplasia of the intima with a deposit of round cells, which causes a gradual compensatory thickening. Josue and Pearce and Stanton[1] have confirmed experimentally Thoma's view of the nature and sequence of pathologic events in arterio-sclerosis. When the prominences in the intima undergo softening or liquefaction, rapid dilatation (*aneurysmal*) of the affected vessels may occur; more commonly this accident arises early or before the intima has reinforced the other layers.

(*b*) **Diffuse Arterio-sclerosis.**—The morbid process (histologically similar to that described above) is distributed throughout the greater part of the arterial system; the circumscribed form is generally "but not necessarily" (Councilman) combined with it in the aorta. Dilatation of the aorta and of its branches commonly coëxists. Apart from the yellowish, translucent, elevated areas, the intima may be smooth and the naked-eye appearances almost normal. Klotz's experiment shows that increased intravascular pressure alone may be the cause of the medial degeneration and weakening in the first place; of the giving way of the arterial wall in the second; and of the intimal hypertrophy in the third. The primary cause of arterio-sclerosis is degeneration and exhaustion of the elastica (W. E. Sanders[2]). Sheffer holds that hypertension is the cause, rather than the result, of arterio-sclerosis. *Microscopically*, there is observed an extensive proliferation of the subendothelial connective tissue and a hyaline transformation of the entire media, particularly in the larger vessels. The muscular fibers and elastic tissue have *in advanced cases* almost totally disappeared. Necrotic degeneration of the media, especially in the smaller arteries, is also observed, and calcareous deposits, causing rigidity of the walls, occur. This is particularly true of the so-called *senile arterio-sclerosis*. In this variety the larger arteries are elongated and tortuous, with thin, stiff (calcified) walls. Atheromatous abscesses that burst, forming atheromatous ulcers, are likewise common pathologic events in the aged. There may be associated atrophy of the heart, liver, and kidneys, due to a lack of nutritive supply in consequence of the narrowing of the vessels.

Sclerosis of the pulmonary artery exhibits all the changes observed in connection with atheroma of the systemic arteries, including aneurysmal

[1] *Journal of Experimental Medicine*, 1906, vol. viii.
[2] *Amer. Jour. Med. Sciences*, November, 1911.

dilatation. From the terminal tributaries the process may extend to the capillaries, and even to the pulmonary veins (*angiosclerosis*).

The *effect of arterio-sclerosis* upon the physiologic functions of the vessel-walls are of first importance. The elastic coat is either destroyed or impaired; this predisposes to dilatation of the vessels (aneurysm).

Another result of extensive atheromatous degeneration of the vessels is an increase in the resistance to the blood-current, and a consequent hypertension. As a consequence, the left ventricle generally becomes hypertrophied (especially if the splanchnic area is involved), "provided the general nutrition of the patient is still well maintained" (Strümpell).

The reduction of the lumen of the vessel, owing to the thickening of the intima, must lessen the blood-supply to the various viscera, and thus are explained such secondary affections as fibrous myocarditis, renal cirrhosis, chronic interstitial pancreatitis (Opie), and cerebral softening.

Sclerosis of the veins (*phlebosclerosis*) may accompany arterio-sclerosis. It is often found in association with hepatic cirrhosis and mitral disease (due to increased tension) when the portal system and pulmonary veins are involved. Arterio-sclerosis apart from sclerosis of the peripheral veins may be encountered, though rarely. *Microscopically*, thickening of the intima and atrophic degenerative changes in the media are commonly observed. Calcification and hyaline degeneration of the layers also occur. Moderate dilatation is not exceptional.

Etiology.—The diffuse form has, in part, a special etiology. It may appear in the young, though rarely; I have met with a case in the Medico-Chirurgical Hospital in a man aged twenty-four years. It is, however, most frequent in strongly built, middle-aged men, and in the aged. At an earlier period it occurs as a result of *alcoholism*, *syphilis* (the overshadowing factor), *lead-poisoning*, *gout*, and *chronic nephritis*—agencies that subject the vascular system to undue wear and tear. Fremont-Smith has collected 144 cases in the young. Congenital syphilis may cause either diffuse or localized arteriosclerosis. In old persons atheroma is often *physiologic* and characterizes the natural involution period of life. *Heredity* may play no inconspicuous part in arteriosclerosis dependent upon the age. This fact furnishes the reason why senile changes in the arteries occur at a much earlier period of life in some families than in others. *Negroes* are more liable than *whites*, and *males* than *females*, though it is more frequent in the latter sex than the circumscribed variety. The frequent occurrence of emphysema and diffuse angiosclerosis has been noted (Anderson).

The *general causes* may be thus classified—(1) *Biologic irritants*, as the specific micro-organisms of malaria, rheumatism, and syphilis. Thayer[1] examined 182 patients who had had typhoid from one month to eighteen years previously, and found the blood-pressure in all cases somewhat high, and over 50 per cent. of the cases over twenty years of age showed palpable arteries. Klotz's experiments indicate that diphtheritic toxins lead to medial degeneration, while others—*e. g.*, typhoid toxins—have no effect on this coat, but induce a primary intimal degeneration. (2) *Chemical irritants* (chronic alcoholism, lead-poisoning, uric acid in gout, diabetes, obesity). The above toxic agents, including endogenous toxins (indol, in particular), produce their effects partly by their direct

[1] *Medical News*, New York, Nov. 21, 1903, p. 1004.

irritant action and partly by increasing the resistance in the peripheral vessels and thus raising the arterial pressure. (3) *Bright's Disease.*—There is a class of cases in which arterio-sclerosis is secondary to Bright's disease, but when found in association the former is more frequently the primary disease than the latter. The two diseases may develop independently of one another, and yet simultaneously, in consequence of the action of a common cause. (4) *Constant overfilling of the blood-vessels*, resulting from excesses in eating and drinking, also causes arterio-sclerosis. (5) *Muscular overstrain*, which augments the blood-pressure while at the same time obstructing the peripheral circulation, is a leading factor. (6) The main causes of sclerosis of the pulmonary artery are *mitral disease* and *emphysema*.

Clinical History.—The disease may be latent for years; or it may be discovered at *autopsy*. In many cases the earlier symptoms resemble those of neurasthenia, and these are accompanied by a slowly progressive failure of the general nutrition. The accessible peripheral vessels (radial, temporal, femoral, and brachial) should be carefully felt when the presence of the disease is suspected. In developed cases the *walls* of the affected artery feel *hard*, and the *pulse*, owing to increased tension, is incompressible; as a result of this rigidity of the arterial walls the degree of vascular tension is difficult of estimation. In marked cases the pulse-wave may not be detectable on palpation. Again, the *tension* may be high, and yet sclerosis of the vessel-wall be slight or absent. When doubt arises, the pulse should be palpated by means of two fingers. If now, while compression of the pulse is made with the index-finger, the middle-finger detects a pulse-wave, arteriosclerosis is present. Rarely, however, a recurrent pulse may be felt notwithstanding, but, as Ewart points out, pressure on the ulnar artery at once arrests it. On account of the loss of elasticity of the vascular walls the pulse is retarded, and the *sphygmogram* shows a short sloping ascent, a wide top, and a slow, gradual descent, with almost an effacement of the dicrotic notch. The blood-pressure is high, as a rule, in arterio-sclerosis, but this may also precede the sclerotic process.

The increased resistance to the circulating medium (due to the rigid vessel-walls) calls forth a correspondingly increased cardiac action, and thus *hypertrophy of the left ventricle* is engendered, with its customary symptoms and signs, including the ringing, accentuated second sound. The balance of the cardio-vascular forces may thus be maintained for a long period of time, during which the health of the patient often remains unimpaired. It happens sometimes that hypertrophy preponderates and veils completely the symptoms of arterio-sclerosis. In elderly persons suffering from atheroma the first sound is often surprisingly feeble. *Myocardial degenerations* frequently come on in the later stages, when dilatation of the left ventricle, accompanied by a mitral systolic murmur and marked rapidity of the pulse, may supervene. The *aorta* may be so dilated as to give rise to an abnormal area of dulness in the upper sternal region. *Palpitation*, *dyspnea on exertion*, a feeling of *precordial constriction*, and light *febrile attacks* are not uncommon. *Angina pectoris* is an infreque t symptom except in coronary atheroma. Certain writers have emphasized abdominal pain, flatulence, and other gastro-intestinal features.

It cannot be stated that involvement of the *external arteries* implies

a serious involvement of the aorta and its main branches. On the other hand, the circumscribed variety is not attended with characteristic alteration of the pulse. The *pathologic*, and particularly the *clinical*, events may be more pronounced at one portion of the body than at others, and this fact has given rise to several distinct or *special types*, as follows: (*a*) cerebral, (*b*) pulmonary, (*c*) renal, and (*d*) peripheral types.

(*a*) **Cerebral Type.**—In the milder grades of this type such symptoms as headache, tinnitus, vertigo, syncopal attacks, and local palsies are variously blended as a rule. Especially in the aged, the condition is apt to lead to *thrombosis* or cerebral *embolism*, emboli being detached from the aortic area and conveyed to the brain, with the development subsequently of the symptoms of anemic softening of the latter. The loss of elasticity of the vessel-walls in atheroma renders them more liable to rupture than normal arteries, while the tension is much increased. Under these circumstances the danger from cerebral apoplexy is obvious.

(*b*) **Pulmonary atheroma** is considered in its clinical relations in connection with the diseases of the heart and lungs.

(*c*) The **renal type** includes those instances of kidney-lesion that are associated with or follow general arterio-sclerosis. The condition is essentially an atrophic nephritis, due to the diminution of the blood-supply to the organs in consequence of the narrowed lumen of the renal arteries.

(*d*) In this *type* the peripheral arteries become obliterated and cause starvation of the tissue, with resulting cramps and even gangrene.

Diagnosis.—Hardened arteries, increased arterial tension, left ventricular hypertrophy, and marked accentuation of the aortic second sound form a group of clinical characters that leaves no doubt as to the diagnosis. It may be the occurrence of apoplexy, acute cardiac dilatation, or of some other such accident that leads to the discovery of general arterial sclerosis. Slight albuminuria is generally present. A murmur may be heard over the base. An ophthalmoscopic examination is of the utmost value as a diagnostic aid.

C. Beck[1] and others have found that the *x*-rays are useful in determining the extent of arterio-sclerosis (*e. g.*, whether local or general).

To **differentiate** the murmurs of dilatation of the left ventricle following the hypertrophy of this disease from *organic mitral lesions* is only possible by the history or the results of treatment. In *aortic stenosis* the second sound is weak and the pulse less voluminous than in arterio-sclerosis (*vide* Aortic Stenosis).

Prognosis.—Arteriocapillary fibrosis is an exceedingly chronic, though usually a progressive, disease, and frequently it terminates life. The axiom that a man is as old as his arteries has been borne out by the test of extensive clinical observation. The condition may prove fatal, either with great suddenness, as when it occasions apoplexy, or with unwonted slowness. Very rarely the aorta ruptures, causing instant death.

Treatment.—Though the progress of the disease cannot in most instances be successfully stayed, it can be retarded frequently by correcting aggravating habits and by removing the influence of ascertainable causes. The syphilitic taint, if present, requires the liberal use of the iodids.

The *diet* must be simple and free from stimulating properties; skimmed milk is excellent, particularly if renal symptoms be manifested. The

[1] *N. Y. Med. Jour.*, Jan. 22, 1898.

lactic acid and sour milk treatment may be employed in cases in which auto-intoxication is an etiologic factor. A salt-free diet (green vegetables, fruits, fresh butter, cream, potatoes, rice, sugar, salt-free bread) is useful for a week or two at a time when the blood-pressure rules high. In the earlier stages potassium iodid is serviceable; it should be administered for several years, combined with appropriate physical exercise (*e. g.*, golf, horseback riding, walking) to regulate the bodily function. Recent researches show that small doses of potassium iodid reduce the viscosity of the blood by acting on the corpuscles without diluting it.[1]

For the increased arterial tension, more especially if due to temporary vasoconstriction, nitroglycerin or the other nitrites should be employed, in increasing doses, until an impression has been made upon the blood-pressure, after which this effect should merely be maintained. Guipsine (dose, gr. $\frac{3}{4}$—0.05, every three or four hours, in pill form) acts well in cases due simply to arterial spasm. Electrical discharges in the form of auto-condensation reduce the hypertension to an appreciable extent, and also probably stimulate the metabolic processes. In persistent (chronic) hypertension the tincture of aconite is useful. Venesection has a more lasting effect than have any of the drugs used (Lawrence). The cases in which the blood-pressure is quite elevated in consequence of vaso-constriction due to nervous causes, combined with a mild grade of arterio-sclerosis, need mental and physical rest, arterial relaxants, and liberal feeding.

For the local aortic symptoms (fever, pain) absolute rest, a liquid and unirritating diet, and a small blister are most efficacious, together with internal minute doses of calomel, quinin, and potassium iodid.

ANEURYSM.

Definition.—A true aneurysm is a circumscribed dilatation of an artery, formed of one or more of its coats.

Classified according to their form, aneurysms are—(1) sacculated, (2) cylindric, and (3) fusiform. They are termed *axial* when the complete circumference of the vessel participates in this dilatation, and *peripheral* when a single sac is confined to the side of the vascular duct.

Miliary aneurysms occur along the course of the cerebral vessels. On the other hand, aneurysms may attain the size of the human skull.

By a *false* aneurysm is meant one in which the coats are ruptured.

A *dissecting* aneurysm is one that, owing to laceration of the internal coat, dissects between the layers of the vessel-wall. For its *seat* it usually selects the aorta, and may traverse its entire length.

An *arteriovenous* aneurysm arises from a direct fistulous connection between an artery and a vein (*aneurysmal varix*), or an aneurysmal sac may intervene (*varicose aneurysm*).

Pathology and Pathogenesis.—The wall of the aneurysm is commonly the seat of arteriosclerosis, which Malkoff[2] claims is a compensatory arrangement. Osler states that the origin of aortic aneurysm is to be traced to mesaortitis, so different from chronic aortic degeneration. The common atheromatous disease does not often produce aneurysm. Extreme atrophy of both the intima and media is not uncommon in the

[1] E. Romberg, *Deutsch. med. Woch.*, Aug. 31, 1905. [2] Ziegler's *Beiträge*, 1899, xxv.

later stages, the wall of the sac being formed chiefly by the adventitia. The intima (as in Daland's case of aortic aneurysm, in which there were both an old and a new transverse rent) may become lacerated, and finally the media and adventitia tear; this results in rupture unless the adherent neighboring structures compensate for the natural wall.

The blood in the aneurysmal sac is composed of old and new thrombi. The latter when comparatively recent may be soft, and when old may be firm or even calcified, yellowish in color, and adherent to the wall.

Etiology.—Among recognized *causes* are—(1) **Arterio-sclerosis.**—Some of the conditions that originate the latter at all events also tend to bring about aneurysms. According to Rasch, syphilis was present in 56 per cent. of 25 aneurysms of the aorta discovered in the course of 3165 necropsies at Copenhagen, and Annsperger found it in 48.6 per cent. of 37 cases. (2) **Sudden Great Strain.**—This may be productive of aneurysm, particularly in the early stage of arterio-sclerosis or before compensatory endarteritis occurs. Thus may the fact be accounted for that most instances of aneurysm occur during the period of greatest bodily activity in the male sex. (3) **Embolic plugging of a vessel,** if complete, may cause aneurysmal dilatation on the proximal side of the point of obstruction. The development of aneurysm may be facilitated by the mechanical effects of the embolus, which may be of calcareous hardness, as when it comes from diseased heart-valves. Infectious emboli set up inflammation and softening. (4) **Mycotic Aneurysms.**—That aneurysms sometimes owe their existence to mycotic origin was first pointed out by Osler, who found an abundant growth of micrococci in the aneurysmal sacs. They are met with in ulcerative endocarditis, and are often small and usually multiple. (5) **Traumatism.**—Aneurysms have been produced experimentally by traumatism (Malkoff); hence it is obvious that it may become one of the assignable causes. (6) **Age and Sex.**—Aneurysms are most frequent between the *thirtieth* and *fiftieth* years, this being the period of greatest physical exertion. The *male sex* is more frequently affected than the *female*, owing to differences in occupation.

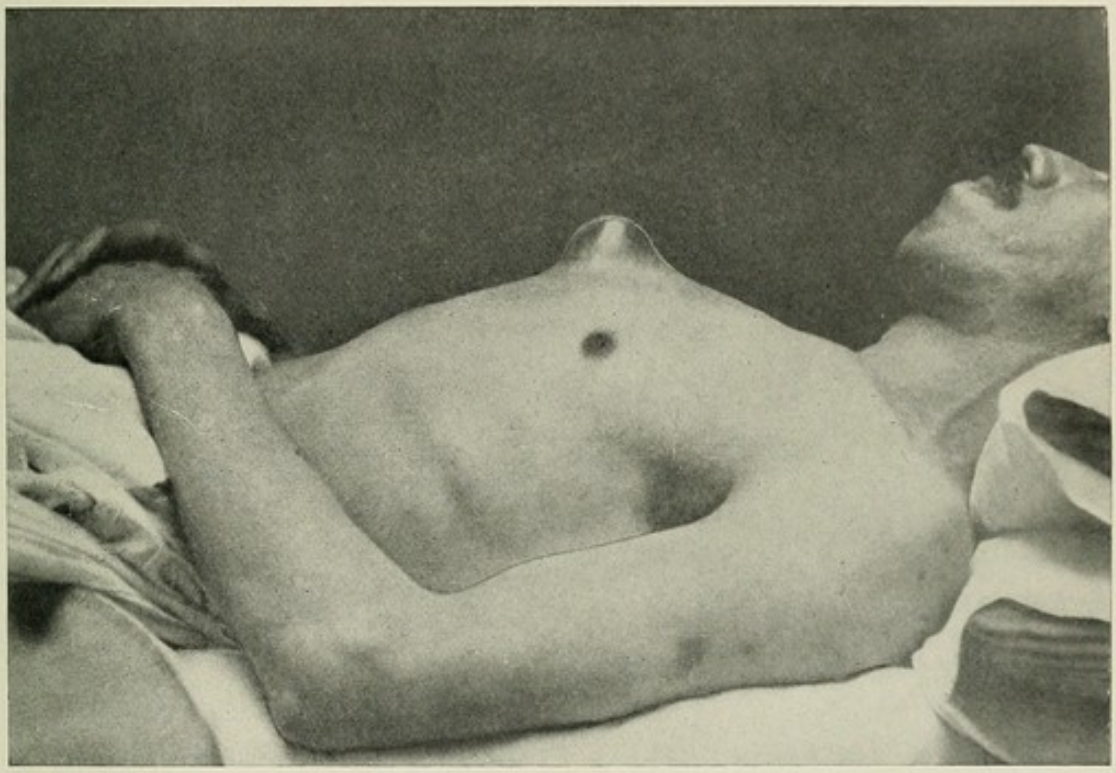
ANEURYSM OF THE THORACIC AORTA.

(*Aneurysma Aortæ.*)

The *thoracic portion* of the aorta is involved in about 75 per cent. of the cases, and the *abdominal aorta* and its branches in 25 per cent. Within the thorax nearly 60 per cent. of the cases originate in the *ascending portion of the aorta* (Lyman). Hare and Holden[1] collected 570 cases of aneurysm of the ascending arch, of which 504 were of the saccular variety.

Symptoms.—Intrathoracic aneurysms may exist, particularly if they are small, without symptoms or noticeable physical signs. When they attain to any considerable dimensions, however, they usually excite characteristic signs and distressing symptoms, the latter being the results of direct pressure, and hence varying with the seat and direction of the progressive enlargement. In a few instances diagnostic symptoms are present in the absence of a detectable tumor or physical signs. Aneurysms of the **ascending portion** of the arch usually *compress* the vena cava, causing *distention of the veins* of the head and arms, though in a pro-

[1] *Amer. Jour. Med. Sci.*, October, 1899.



ANEURYSM OF AORTA.

portionately small number of cases the subclavian may be the only vein compressed, with resulting *enlargement* and *edema* of the right arm. The largest aneurysms may even compress the inferior vena cava, causing edema of the lower extremities. The *heart* is displaced outward toward the left pleura, and usually upward, and rarely causing erosion of the ribs and sternum. The right recurrent laryngeal nerve may be implicated, giving rise to *dyspnea* and *aphonia*. *Pain* is a constant feature.

Aneurysms of the **transverse portion** of the aorta, when they attain any considerable size, cause the most intense symptoms, owing to the relatively shorter antero-posterior diameter of the chest at this point, in consequence of which greater *compression* of the neighboring tissues takes place. By protruding backward they may exert pressure upon the trachea, causing paroxysmal *cough* and *dyspnea*, or on the esophagus, causing *dysphagia*; these are common events. The pressure may fall also upon the bronchus, inducing *dyspnea*, *bronchorrhea*, and *dilatation*, the latter in turn sometimes leading to circumscribed abscess. The left recurrent laryngeal nerve may be implicated, with resulting *aphonia*.

Upward extension of the aneurysmal process, with involvement of the coats of the carotid and subclavian on the left side, or of the innominate and carotid on the right, may occur. The *sympathetic nerves* in the cervical region may be irritated, causing dilatation; or they may be paralyzed, causing contraction of the *pupils*. Compression of the *thoracic duct* may occur, with resulting rapid emaciation. A *tumor* may appear in the jugular fossa.

The aneurysm may grow *forward*, in which event it lies directly behind the manubrium, which from the pressure becomes eroded and may finally disappear in part. In aneurysms involving the transverse portion of the arch, lateral pressure, both toward the right and the left, is also made, causing recession and compression of the lungs.

When the **descending portion** of the arch is affected the pressure is exerted upon the spinal column to the right, and upon the tissues as far as the shoulder-blade to the left. As a consequence of destruction and absorption of the vertebræ, compression of the spinal cord may ensue, and is an intensely painful process. Pressure may be made upon the esophagus, causing *dysphagia*, or upon the left bronchus, causing *bronchiectasis*, with its usual *sequelæ* (bronchorrhea, fetid bronchitis, gangrene of the lung).

The sac may, in consequence of the slow ulcerative process that attends its progress, *rupture* (*vide* Prognosis). Frequently repeated small *hemorrhages*, due to weepings from the thinned walls, may precede the fatal rupture. I saw a case of aneurysm of the transverse portion in which rupture into the esophagus resulted, with instantaneous death.

When the tumor has reached the subcutaneous tissue and bulges externally, the skin covering it becomes tense and shining, and with increased pressure the surface becomes reddened and finally necrotic. The necrosed area is covered with a dry brown scab, which later is thrown off, leaving an oozing surface. Rupture soon follows.

Leading Symptoms in Detail.—Among these *pain* stands primarily, being the first and most constant. It is of two kinds: (*a*) due to direct pressure upon and stretching of the nerves. When aneurysm is developed suddenly, a sharp, excruciating pain is felt in the upper sternal region, accompanied by a feeling of "something giving way." In consequence of the stretching of the nerves a constant pain is expe-

rienced that is subject to exacerbations when the intra-aneurysmal pressure is raised. Pressure against the bony structures causes erosion, and usually produces a continuous boring pain. In a recent case of aneurysm shown in clinic at the Medico-Chirurgical College, however, a tumor of the size of a goose's egg, had given rise to no suffering whatever. In latent aneurysm there is an absence of pain. Anginose attacks sometimes occur when the sac has its seat near to the heart. (*b*) Reflected pains of a neuralgic character may be excited by aneurysm. This is true, in particular, of aneurysms situated in the transverse portion of the aorta, in which instances pain is commonly felt in the region of the neck and occiput and down the left arm. When the growth is situated along the course of the descending aorta, intercostal neuralgia may be excited, due to pressure upon the nerve-trunks.

Cough.—The cough is paroxysmal, and frequently has a peculiar brazen, ringing character that points to its laryngeal seat. Pressure upon the windpipe excites a paroxysmal dry cough. Compression of a bronchus may lead to bronchiectasis, and the cough then occurs only in severe paroxysms which recur at intervals of a day or even longer, and are attended with copious, ropy expectoration (*vide* Bronchiectasis).

Dyspnea is a conspicuous symptom in aneurysm of the transverse portion of the aorta (the aneurysm of symptoms—Broadbent). It arises (*a*) most frequently in consequence of pressure upon the recurrent laryngeal nerve, (*b*) direct pressure on the trachea, and (*c*) from pressure on the left bronchus. Marked stridor may accompany the first variety.

Paralysis of the vocal bands is occasioned by compression of the recurrent laryngeals, particularly the left, while a slight degree of compression or irritation of the same nerve causes *spasm* of the vocal cords. The symptoms of these conditions are hoarseness, cough, and aphonia respectively. The laryngoscope should be employed, since paralysis of one of the abductors may be present without giving rise to appreciable symptoms.

Hemorrhage may occur as a slow oozing, either from the point of compression in the trachea or externally; in either case the bleedings are small. Profuse bleedings (producing sudden death) take place in consequence of rupture of the sac into the lung, the bronchus, or the trachea.

Deglutition may be difficult, owing to compression of the esophagus. When an aneurysm has been diagnosticated or even suspected, the esophageal sound should not be passed, lest the sac be ruptured.

Compression and irritation of the sympathetic system of nerves cause pupillary changes that have already been mentioned. With dilatation of the pupil there may be observed pallor of one side of the face, due to stimulation of the vasodilator fibers; on the other hand, with contraction of the pupil (due to paralysis of the constrictor fibers) there is hyperemia of one side of the face and unilateral sweating, with drooping eyelid. The most common cause of anisocoria is unequal blood-pressure in the ophthalmic arteries (Wall and Walker).

Clubbing of the fingers and *incurvation of the nails* (at times unilateral) are not rarely met with in thoracic aneurysm.

Physical Signs.—*Inspection.*—Visible pulsation is one of the earliest appreciable signs. It is most frequently observed at the right side of

the sternum, above the level of the third rib (second interspace), and much less frequently on the left side over a corresponding area. In aneurysm of the transverse portion pulsation may be seen at the episternal notch, though an impulse here may also be due to nervous palpitation, and have no connection with aneurysmal growths. When pulsation is associated with swelling, its diagnostic value becomes greater.

Involvement of the innominate artery produces pulsation in the neck above the sterno-clavicular junction, or less commonly above the sternum. Corresponding to the site of visible impulse, there is, sooner or later, bulging in most instances. It may, however, be so slight as to elude detection unless the keenest observation be practised, and in not a few instances the tumor itself is invisible from the front of the body, but is recognizable looking from behind or from either side. Again, on allowing the light to fall obliquely upon the chest slight prominences may be brought to view that would otherwise be inappreciable.

When the aneurysm is situated in the ascending part of the arch, the most frequent seat of the bulging—which varies in size from a hen's egg to a cocoanut—is over the first and second right interspaces near to, and frequently involving, a portion of the sternum; when seated just beyond the aortic orifice, a pulsating prominence may occupy the third interspace along the left sternal border; situated in the transverse section of the aorta, bulging of the upper part of the sternum is common. In the descending portion the swelling, when present, is in the second and third left interspaces near to the sternum, or in the left scapular zone. The apex-beat is displaced downward and outward, chiefly from pressure, though also from hypertrophy (functional).

Palpation.—The protrusion presents a more or less yielding and elastic mass, and when superficially seated fluctuation may be obtainable. The degree, and the rhythmic expansile character of the pulsation are to be noted, and also the fact that there is an alternate contraction and dilatation of the sac in every direction—a distinctive feature.

If the aneurysm is largely concealed, bimanual palpation should be employed, the palm of one hand being placed over the spine and that of the other over the sternum. In rare cases aneurysmal pulsation is only yielded when the finger-tips are used, and especially at the end of expiration. A diastolic shock is often perceived, and forms a sign of no little value. A distinct systolic shock, sometimes accompanied by a purring fremitus, can also be felt over the aneurysmal sac.

Percussion.—If the growth be deep-seated, percussion may give negative results; when, however, the tumor causes bulging or comes in contact with the chest-wall, a proportionate area of flatness is presented. The abnormal field of dulness may be the only symptom present. Aneurysms of the ascending arch give flatness to the right of the sternum; those of the transverse arch, over the upper part of the sternum and to the left; while those of the descending portion are revealed by a flat area between the spine and the left scapula. With flatness of the percussion-note there is a sense of increased resistance. There is generally a moderate increase in the area of cardiac dulness. Conversely the left ventricle has been found of diminished size at necropsy.

Auscultatory percussion (practised after the method of Sansom and Ewart) quite often gives valuable results.

Auscultation.—Since murmurs owe their origin, in great part, to the presence of fibrin in the sac, they may be absent, and this even in the case of large aneurysms. When, as is usual, a murmur is present, it is systolic in rhythm, heard with greatest intensity over the flat area or body of the tumor, and is transmitted in the direction of the blood-stream, being, therefore, distinctly audible in the vessels of the neck and along the course of the aorta. The murmur has a booming quality.

Aortic regurgitation may be considered as associated with aneurysm near the aortic ring when a double murmur is heard. In a few instances the diastolic bruit is alone detectable. A much intensified, ringing second sound is present (unless marked aortic regurgitation coexists).

The Peripheral Arteries.—The pulse in the vessels beyond the aneurysm is delayed. Hence the two radial pulses may exhibit differences in *time*. The *volume* of the pulse beyond the aneurysm is lessened, and in aneu-

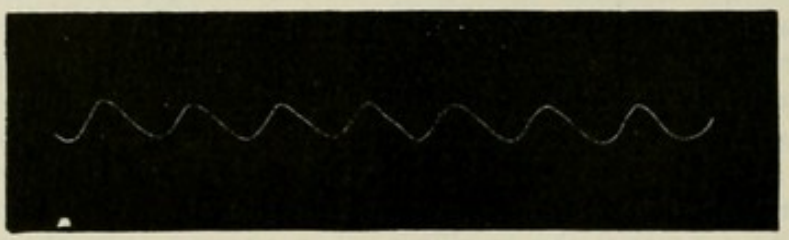


FIG. 58.—Sphygmogram of a case of aneurysm of the left subclavian artery (Foster).

rysm of the abdominal aorta or the femorals it may be obliterated. Such differences as these will not only point to the existence of thoracic aneurysm, but also its *seat*. Thus, if there be dilatation of the transverse arch with no implication of the innominate, the pulse at the right wrist is strong and almost simultaneous with the cardiac impulse, while that on the left side is small, weak, and delayed. If the reverse be true, then the aneurysm may be near to or involve the innominate. O. K. Williamson has found a marked difference in the blood-pressure of the two arms in cases of thoracic aneurysm, a variation of more than 20 mm. Hg being in favor of aneurysm.

The sphygmogram exhibits a slanting up-stroke with obliteration of the secondary wave (Fig. 58), though its characters are inconstant.

Tracheal Tugging.—This sign may be practised while the patient is sitting or standing with the chin slightly elevated. The cricoid cartilage is grasped between the thumb and forefinger and pushed upward so as to stretch the trachea. The patient must cease breathing momentarily, when, if this sign be present, there will be a downward tugging at each systole. The transmitted pulsations from the cervical vessels must not be confounded with the vertical movement of the trachea. A new method of eliciting tracheal tugging, first suggested by Ewart, has been widely adopted and possesses the advantage of ensuring greater delicacy of touch than the old. He stands behind the patient, supporting the head of the latter against his body, and grasps the cricoid between the tips of the forefingers. The method is in other respects similar to that previously described. As shown by Toulmin, the tracheal tug may be present in health and in other diseases, hence it is of little value.

Diagnosis.—In the presence of the following points the existence

of thoracic aneurysm may be confidently inferred: (1) Antecedent arteriosclerosis (with the appropriate causes of the latter); (2) History of other etiologic factors, as age (between thirty and forty-five years) and occupation (such as entail unusual muscular strain); (3) Pressure-symptoms, as pain, dyspnea, aphonia, cough (either laryngeal or bronchial), bronchorrhea, dysphagia, edema, vasomotor disturbances; (4) Physical signs of a pulsating tumor (including the abnormal area of dulness, systolic murmurs, the systolic and diastolic shock, and tracheal tugging) somewhere along the course of the arch or its great branches, with or without differences in the blood-pressure, and in the volume and time of the radial pulses. There are, however, several classes of cases which offer difficulties that are sometimes insurmountable: (*a*) Those in which the aneurysm is small and deep-seated. Here the symptoms and physical signs are indefinite. There may be thoracic oppression, in which pain may radiate to the left shoulder, and mild pressure symptoms—a group of suspicious features merely—sometimes appear. In one of my cases left-sided intercostal neuralgia was the only symptom present. (*b*) Aneurysm of the transverse arch, in which the pressure symptoms are more or less pronounced, but with no physical signs. In such, a clear history suffices to complete the diagnosis. Pressure symptoms without etiologic factors are just as likely to be due to other causes. (*c*) Those cases in which the more characteristic features are manifested intermittently. Fortunately, a proper diagnosis of aneurysm in obscure and latent cases can be often made by the aid of the *x*-rays, and it can also be excluded, in suspected cases, by fluoroscopic examination.

Extremely obscure are many of the cases, in which the only symptoms manifested point to irritation of the trachea or bronchial tubes, with paroxysmal cough, or the signs of bronchiectasis. In a recent case of this sort tracheoscopic examination revealed compression of the windpipe. In another instance the laryngoscope determined the diagnosis, in that it brought to view bilateral paralysis of the abductors of the vocal bands. A symptom, which has been designated by its author, R. V. Hoesslin, as "diastolic expiration," is due to compression of the trachea by an aneurysm, so that air can escape from the lungs only during diastole, when the sac diminishes in size.

Differential Diagnosis.—The affections from which intrathoracic aneurysm must be distinguished are *pulsating empyema*, *pulmonary tuberculosis*, *abnormal pulsation of the aorta*, and *solid tumors*. Of the latter, those simulating aneurysm are carcinoma, sarcoma, and enlarged lymph-glands. These *mediastinal tumors* may duplicate all of the pressure-symptoms, though less apt to cause bulging, and less apt still to excite abnormal pulsation; when pulsation is noted it is quick, and not heaving and expansile, as in aneurysm. Solid growths also lack the characteristic shock—both systolic and diastolic—of aneurysm. The cardio-vascular symptoms are usually wanting in the case of solid tumors, especially the moderate hypertrophy, accentuation of the second sound, tracheal tugging, and the difference between the radial pulses.

Carcinoma of the mediastinum usually gives a history of the disease in other parts of the body, with enlargement of the axillary or other superficial lymphatic structures, and later the characteristic cachexia, this being particularly marked in carcinoma of the esophagus.

Abnormal pulsation in the aorta is noted in neurotic subjects, mostly females, and in aortic regurgitation; less frequently it is associated with retraction of the right lung, with spinal curvature, and with displacement of the aorta. In the case of the latter two conditions a careful consideration of the causal states and the absence of the characteristic physical signs would lead to a correct diagnosis. *Aortic regurgitation* is frequently associated with aneurysm of the arch, and in its course there is often developed a dilatation of the ascending portion of the aorta. The diagnosis of aneurysm of the arch of the aorta should not be made, therefore, in these cases, unless the physical signs and symptoms be unmistakable. Dynamic pulsation of a neurotic origin is seen and felt in the episternal notch, as a rule, and a correct appreciation of the nervous element will prevent the observer from committing an error.

Pulsating empyema can only be confounded with large aneurysmal growths, and, as pointed out by Wilson, it does not have the same definite relation to the central long axis of the body as do aneurysms. The abnormal field of dulness is situated at the base of the lung in empyema, and is less circumscribed than in aneurysm. In empyema, moreover, the pulsation is not expansile, but is caused by pressure of the respiratory movements. Auscultation in empyema renders audible neither a bruit nor the double shock of aneurysm; the pressure-symptoms and pulse-characters are also entirely wanting.

Pulmonary tuberculosis may be mistaken for thoracic aneurysm. When an aneurysm compresses a bronchus, bronchiectasis, attended with cough, bronchorrhea, fever, and emaciation, may be the result; but in phthisis the fever and emaciation are more pronounced, tubercle bacilli are present, whilst the cardio-vascular signs of aneurysm are absent.

Prognosis.—The occurrence of perforation and consequent speedy death in unsuspected cases must be recollected. In other instances the end is approached in a very gradual manner, and cases in which rupture does not supervene sometimes pursue the general course of chronic valvular affections of the heart. The condition ends in death as a rule, and the *immediate causes* of the fatal issue are as follows: (1) Rupture of the aneurysm, followed by hemorrhage into any of the adjacent cavities or organs (pericardium, heart, large vessels, mediastinum, trachea, a bronchus, esophagus, lungs, pleura, spinal canal); it may, though rarely, rupture externally, in which case slight hemorrhages occur and life may last for weeks; (2) Gradual asthenia; (3) Direct pressure; (4) Independent diseases, either primary or secondary to, and induced by, the aneurysm. Among these *pulmonary complications*—fibrinous pneumonia, abscess, gangrene, tuberculosis—are of first importance.

Treatment.—There are two main objects of treatment—first, the promotion of coagulation of the blood, and secondly, the contraction of the sac. The clotting of the blood within the growth may be greatly favored by retarding the blood-current. Nothing so well accomplishes this object as *absolute rest* in the recumbent posture. This cannot always be rigidly enforced, but muscular exertion must be minimized, mental application regulated, and emotional excitement avoided; stimulants, arterial and nervous, are to be eschewed for like reasons. Palpitation of the heart, when present, is to be allayed by the local use of the ice-bag. The coagulability of the blood is also increased by removing

as far as possible the liquid portion of the diet. The measures already indicated tend to lessen the volume of blood and the intra-aneurysmal pressure, thus inviting contraction of the sac as well as consolidation of its contents. A. E. Wright has insisted upon the value of calcium salts to increase the coagulability of the blood (gr. x to xv—0.648 to 0.972, t. i. d., may be given). T. R. Boggs[1] thinks calcium salts increase the coagulability of the blood, the best for this purpose being the acetate and lactate of calcium.

Injections of gelatin in aneurysm may have a specially favorable effect according to certain observers. Moyer[2] reviews the literature, and in the main his conclusions are: Gelatin solutions are of some value in the treatment of saccular aneurysms, but not of the diffused forms. Solutions not stronger than 1 per cent. should be used; they should be kept in a brood-oven to determine bacterial growth, and great care should be taken in the technique. Absolute rest in bed should be enjoined. This method is worthy of extended trial, but great caution and watchfulness must be exercised in its administration.

Among medicinal agents, to *contract the sac*, ergot and potassium iodid have been employed, the latter with good effects. The exact manner in which the iodid produces its favorable results in these cases is unknown, though most probably it acts upon the vascular walls, and hence would be most efficacious when the disease is of syphilitic origin; this view accords with my own personal experience. I would advise against the prolonged internal use of ergot. Langenbeck and others have obtained good results from the direct injection into the sac of the aqueous extract of ergotin dissolved in water or glycerin, every day or two. When employed in this manner ergotin induces contraction of the smooth muscles in the wall of the aneurysm. Numerous observers have resorted to the use of horse-hair, fine wire, fine catgut, slender watch-springs, with a view to coagulating the blood as it comes in contact with these foreign bodies. Electrolysis is a method that has been warmly advocated (Loreta).

Combined wiring and electrolysis (Corradi's method) has been successfully employed by Rosenstein, Kerr, D. D. Stewart, and Herchy. The details of the method are briefly as follows:

A piece of fine wire, several feet in length, is passed from a spool through a small insulated canula, so that the wire curls up within the sac. It is attached to the positive pole, while the negative is connected with a surface pad placed over the abdomen. The current is then passed through, and gradually increased in strength to 40 to 80 milliampères. Before stopping the current its strength should be slowly decreased. Each application of the current should last from one to two hours. It is not, however, without serious dangers (hemorrhage and embolism).

Galvano-puncture has long been resorted to, and in some instances with encouraging results. The cases that receive most benefit from the above measures belong to the saccular variety; this is also true of the plan first commended by Tufnell, which is especially applicable in the earlier stages. Tufnell's method is founded upon two principles—absolute rest in the recumbent posture, and a much-restricted, dry diet. A quiet mental state should be conjoined. The diet is as follows: Breakfast, 2 ounces (64.0) of bread and butter and 2 ounces (64.0) of milk; for dinner, 2 or

[1] *N. Y. Med. Jour.*, February 1, 1908. [2] *Medicine*, March, 1899.

3 ounces (64.0–96.0) of meat and 3 or 4 ounces (96.0–128.0) of milk or claret; for supper, 2 ounces (64.0) of bread and 2 ounces (64.0) of milk.

The chief advantages growing out of this method are the lessened number and force of the heart-beats in consequence of the bodily rest, and the diminution of the blood-volume in consequence of the dietetic restrictions. It should be persevered in for several months. The bowels should be regulated, and straining at stool prohibited.

Special Symptoms.—Pain is often relieved by potassium iodid. When arterial sclerosis is present I have seen relief from pain afforded by the use of nitroglycerin (♏j to ij—0.066 to 0.133, four times a day). In the later stages morphin should be given. When there is bulging the pain may be relieved by the the ice-bag or by a belladonna plaster.

Dyspnea and great venous congestion are to be met by venesection, and tracheotomy may be required in bilateral paralysis of the abductors. In dyspnea from pressure on the trachea or bronchus, however, tracheotomy would be a valueless expedient. When the aneurysm forms a large external tumor the application of an elastic bandage to the chest may be both agreeable and advantageous, as in a case referred to by Osler.

Among subjects of arterio-sclerosis, tortuosity and elongation of the aorta may exist and give rise to some of the signs of aneurysm of the arch. Holzknecht[1] was the first to point out this pseudo-aneurysmal dilatation, but he classified them as dislocations of the aorta. Joseph Sailer and G. E. Pfahler[2] have made fluoroscopic and radioscopic examinations of eighteen cases, some of which were autopsied at a later date, and showed that pulsating hemispheric shadows above the heart were not always aneurysms, but often mere tortuosities of the aorta, which cast a shadow from one-half to two inches to the left of the fifth and sixth dorsal vertebræ.

The *symptoms* of atheroma with tortuosity are for the most part negative, but certain physical signs which strongly suggest aneurysm are commonly observed. "Probably it would be justifiable to suspect tortuosity in cases in which there is inequality of the radial pulse, slight tracheal tugging, dyspnea, and in which the symptoms are stationary for a considerable period of time and the fluoroscope shows a projection to the left of the descending portion of the arch of the aorta that has no true expansile pulsation" (Sailer and Pfahler).

ANEURYSM OF THE ABDOMINAL AORTA.

The vicinity of the celiac axis is the favorite seat of abdominal aneurysm, which is less common than intrathoracic aneurysm, though not rare. It may assume a fusiform or saccular nature.

Symptoms.—The tumor may grow backward; but more frequently it grows forward. Projecting from the *posterior wall*, it usually erodes the vertebræ, and compression of the cord is apt to take place, producing *paraplegia*, preceded by *tingling* and *numbness* of the *legs*.

Pain is the leading symptom. It may be neuralgic or of a boring or gnawing character, due to destruction of the bone. Rarely, the aneurysm perforates the diaphragm, and finally *ruptures* into the lungs or pleura. Arising from the *anterior wall*, it may early form a well-defined tumor. It may, however, when situated high up or near the diaphragm, conceal

[1] *Wiener klin. Woch.*, 1900, No. 10.

[2] "Tortuosity of the Aorta," *The Amer. Jour. of the Med. Sci.*, October, 1903.

itself until it has attained a comparatively large size, as in a case recently under my care at the Medico-Chirurgical Hospital. *Vomiting* and *gastralgic seizures* may be troublesome, and the fact that *embolism* of the superior mesenteric artery may occur and give rise to severe colicky pains must be recollected. *Jaundice* has been observed.

Physical Signs.—Epigastric pulsation may be *visible*, and occasionally an epigastric swelling. The *palpating* hand detects a heaving, expansile pulsation that may be accompanied by a thrill. When the tumor hugs the diaphragm the pulsation may be double. The femoral pulse is diminished in volume and delayed. An abnormal area of dulness may be present. In most instances a *soft bruit* is audible. The diastolic murmur and shock of *intrathoracic aneurysm* are usually absent.

Diagnosis.—A certain diagnosis demands the presence of a definite growth that is seizable and has a heaving, expansile pulsation. Mere pulsation attended with a thrill and a systolic murmur are insufficient.

Differential Diagnosis.—A *throbbing aorta*, as met with in neurotic females and in anemia (particularly in instances of the traumatic form), is sometimes distinguished from aneurysm of the abdominal aorta with great difficulty. It does not, however, present a pulsating tumor that can be held in the grasp, as in aneurysm.

When *solid growths* lie upon the aorta the latter may manifest pulsation, a thrill, and a systolic murmur, but the very general absence of pulsation (when the patient is placed in the knee-elbow position), owing to the fact that the tumor falls forward, suffices usually to differentiate the condition from genuine aneurysm. Again, expansile pulsation is not evinced by a solid growth.

The **prognosis** is very gloomy. Very rarely, however, nature effects a cure if the conditions be favorable. "Death may result from (*a*) the complete obliteration of the lumen by clots; (*b*) compression-paraplegia; (*c*) rupture either into the pleura, retroperitoneal tissues, peritoneum, the intestines, or, very commonly, into the duodenum; (*d*) embolism of the superior mesenteric artery, producing infarction of the intestines" (Osler).

Treatment.—Apart from the measures indicated for thoracic aneurysm, there is one means of cure that may be tried if the growth be low down—viz. *pressure*. This must be maintained for twenty-four hours at least under an anesthetic. It is best to make steady pressure on the proximal portion of the vessel, and unless practised with great care the sac will be damaged and death ensue.

ANEURYSM OF THE PULMONARY ARTERY.

Dilatation of the pulmonary artery is of frequent occurrence in affections that oppose obstruction to the lesser circulation (*e. g.* mitral disease, emphysema, phthisis). Very rarely extreme dilatation of the vessel is followed by semilunar incompetence, when a diastolic murmur at the pulmonary orifice (second left interspace) becomes audible.

Aneurysms involving the pulmonary artery are quite rare: such as occur are small and of the saccular and fusiform varieties.

The **symptoms** resemble those of *intrathoracic aneurysm*, though they are rarely well marked, owing to the fact that they remain of small size as a rule.

Physical Signs.—Pulsation (and, rarely, a small tumor) is detectable in the second left interspace. *Palpation* may also render appreciable a

thrill and diastolic shock. Coextensive with the area of pulsation there may be *dulness* on percussion, and over the second interspace to the left of the sternum a loud *superficial systolic murmur* is heard on auscultation, together with a diastolic shock. Before attaining to a large size, these aneurysms usually *rupture* into the heart itself.

The **prognosis** is altogether unfavorable, the treatment having reference to the principles that are appropriate in thoracic aneurysm.

The **coronary arteries** may be the seat of aneurysm, though exceptionally. The condition arises in consequence of weak points (due to arteriosclerosis) in the course of the vessels, and is unrecognizable during life.

ANEURYSM OF THE CELIAC AXIS.

This condition is sometimes observed in combination with aneurysm of the upper portion of the abdominal aorta.

ANEURYSM OF THE SPLENIC ARTERY.

This branch of the celiac axis is occasionally the seat of aneurysmal dilatation. It may be single or multiple, and, whilst it is small as a rule, may in rare cases be quite large.

The **symptoms** are indefinite, but distressing. Deep-seated abdominal *pain*, which shows a tendency to radiation, forms, with *vomiting*, and rarely *hematemesis*, the main features. By *percussion* a tumor may be mapped out in the left hypochondriac region, the dulness merging with that of the spleen and the left lobe of the liver. Usually, pulsation, and, rarely, a tumor can be *felt*, and *systolic murmur* is often heard. The condition may be confounded with *gastric ulcer*.

ANEURYSM OF THE HEPATIC ARTERY.

This is exceedingly rare, the total number of cases on record being about 20. H. B. Schmidt has recently reported a case associated with symptoms of gall-stones, in which, as shown by the autopsy, death was caused by rupture of the sac into the bile-ducts. Schmidt found records of but 5 cases of this mode of termination. Osler and Ross have reported an instance associated with multiple hepatic abscesses.

The **symptoms** are, in the main, *colicky pains*, *vomiting*, *hematemesis*, and *obstructive jaundice*. A *tumor* is rarely discernible, though an abnormal area of pulsation is relatively more frequent. The recognition of the condition during life is entirely conjectural.

Aneurysm of the superior mesenteric artery is of rare occurrence. *Pain* in the epigastric and lumbar regions, and *demonstrable tumor* near to or directly over the median line of the abdomen, are the symptoms displayed. Detached fragments of the clot may produce *embolism* of the terminal branches of the mesenteric arteries. The condition terminates usually in rupture into the peritoneal cavity.

Aneurysm of the inferior mesenteric artery runs a course similar to the above. It is quite rare and possesses slight interest.

Aneurysm of the Renal Arteries.—Small multiple dilatations are occasionally seen, but large ones are of great rarity. They are prone to rupture into the retroperitoneal cavity.

ARTERIO-VENOUS ANEURYSM.

Definition.—An artificial communication between an artery and a vein. A sac may intervene between the two vessels (*varicose aneurysm*) or there may be a direct fistulous communication without an intervening sac (*aneurysmal varix*).

In varicose aneurysm the sac is developed from the structures that mark the boundaries of the communicating duct. The majority of cases are caused by the simultaneous wounding of an artery and a vein during venesection. Hence their most frequent seat is at the bend of the elbow. Pepper and Griffith have analyzed the records of 29 cases in which the ascending portion of the aortic arch had opened into the vena cava.

Symptoms.—The symptoms are largely *aneurysmal*, and in addition there appear in rapid sequence *great swelling of the veins*, *cyanosis*, and *edema* of the upper portion of the body. A continuous *thrill* and *buzzing murmur*, with systolic intensification, are the chief signs.

In the **treatment** of thoracic arterio-venous aneurysm the same general plan is to be pursued as advised in the purely arterial variety.

CONGENITAL ANEURYSM.

This condition arises because of a defective ante-natal development of the elastic coat. It is often multiple, and the tumors are, as a rule, small in size, ranging from that of a pea to a hazel-nut. The most common situations for these growths are the coronary and pulmonary arteries. To Eppinger belongs the credit for having pointed out the fact that the aneurysmal walls consist only of the adventitia and intima. "*Peri-arteritis nodosa*," a rare condition, which Eppinger holds to be a form of congenital aneurysm, presents the symptoms of general infection rapidly developed. "On examination after death the arteries are found beset with nodules of active inflammatory products, chiefly on the outer coat" (Allbutt). The condition may be met with in children and rarely in adults.

PART VII.

DISEASES OF THE DIGESTIVE SYSTEM.

I. DISEASES OF THE MOUTH.

STOMATITIS.

CATARRHAL STOMATITIS.

(*Stomatitis Erythematosa.*)

Definition.—A simple, acute inflammation of the buccal mucous membrane. It is more commonly met with in children than in adults.

Etiology.—As a *primary* affection its causes are mainly mechanical and chemical irritation, such as the presence in the mouth of hard and sharp bodies, dental caries, acids, hot or cold food, condiments, tobacco, certain drugs (as mercury), eruption of teeth, and bad feeding, particularly in illy-nourished children. It is the result often of a neglect of the mouth-toilet, leading to the decomposition of accumulated bits of food and mucus, and many cases probably owe their origin to infection. *Secondarily*, catarrhal stomatitis may be associated with certain of the eruptive fevers (scarlet fever, measles, typhoid), also with gastro-enteric derangements, and may follow, by direct inflammatory extension, ulcerative tonsillitis, and pharyngitis.

Symptoms.—The *local symptoms* of this affection are those usually seen in an inflammation of a mucous membrane—redness, heat, swelling, and dryness, soon followed by increased secretion and soreness. The lips and gums only, or the membrane of the whole mouth, may be inflamed, and the swollen lips, cheeks, and furred tongue may be indented by teeth-marks. Enlarged and reddened papillæ on the tongue and minute vesicles inside the cheeks and lips from projecting mucous follicles are sometimes seen to terminate in small ulcers. A craving for cold drinks is nearly always noted, and distress, pain on taking food, and a disagreeable taste due to the perverted buccal secretions. *Chemical examination* of the dribbling saliva shows an acid reaction, but should the condition progress to ulceration an alkaline reaction is usual. Microscopically, desquamated epithelium that has undergone partial fatty degeneration, leukocytes, and occasionally red blood-cells are seen. The leptothrix buccalis, micrococci, and bacilli are also present. Aside from restlessness and the symptoms common to slight febrile disturbances, the *constitutional condition* is rarely disturbed, except when the stomatitis is

secondary either to inflammations lower down in the digestive tract, or to the specific infectious fevers.

The *course* of the disease is usually acute, and the duration about one week.

The **differential diagnosis** of catarrhal stomatitis is easily made by inspection of the membrane.

The **prognosis,** though favorable, will vary as to time and severity according to the cause.

Treatment.—After proper attention to the hygienic surroundings of the patient and the removal of all irritant influences, the treatment is mainly local. The first indications are to cleanse the mouth and allay the pain, and these may be met by the use of cool solutions of boric acid, sodium bicarbonate, or potassium chlorate, 5 and 10 grains (0.324 and 0.648) respectively to the ounce (32.0) of glycerin and rose-water, as mouth-washes, or for swabbing in the case of infants. When iced drinks are ungrateful and the inflammation is more intense and protracted, the use of hot milk and lime-water, mucilaginous decoctions, and sedative and antiseptic sprays of 1 or 2 per cent. solutions of cocain or carbolic acid are often beneficial; or mild astringents may be needed, as ¼ to 1 per cent. solutions of silver nitrate, alum (5 to 10 grains—0.324 to 0.648—to the ounce—32.0—of honey), and glycerite of tannin (2 drams to the ounce—8.0 to 32.0—of water), especially if there is a tendency to chronicity of the trouble, as in topers and inveterate smokers. Tender and spongy gums may be relieved by the application of equal parts of the tinctures of myrrh and rhatany on a camel's-hair brush (Strümpell). *General symptoms* must be met as they arise. Small doses of aconite or potassium citrate for the pyrexia, with a minimum dose of bromid for irritability and sleeplessness, may be all that is required. The internal use of potassium chlorate in children is not to be recommended in this affection, both because of its deleterious action upon the kidneys, and also because it seems to be unnecessary (Forchheimer, Blackader). Sometimes an associated gastro-intestinal catarrh needs correction by the use of laxatives. The administration of bland foods and mild ferruginous tonics should be continued throughout convalescence.

APHTHOUS STOMATITIS.

(*Follicular Stomatitis; Stomatitis Aphthosa.*)

Definition.—A variety of catarrhal stomatitis characterized by the eruption of one or more vesicles upon the edges of the tongue, the cheek, or the lips, rapidly passing into small round or oval discrete spots that are slightly raised and surrounded by yellowish-white bases with narrow red areolæ.

Etiology.—Though more common in children between the ages of two and six years, it is by no means rare in adults. Predisposing influences may be found in the seasons (spring and autumn), malnutrition, tuberculosis, dentition, persistent gastro-enteric disorders, anemia, and the acute exanthemata. The *exciting causes* are supposed to be certain deleterious substances, bacterial or toxic, though no special parasite has yet been isolated.

Symptoms.—The herpetic vesicles soon rupture, leaving the aphthous ulcers as described above. They are found singly, or at times as many as twenty in number, pin-head to split-pea in size, inside the lips, especially near the frenum, along the tongue-edges, and sometimes inside the cheeks near the edges of the back teeth. They are exquisitely *tender*, so that almost any motion of the affected parts causes sharp burning pain; nourishment is therefore difficult. Patches of *catarrhal stomatitis*, and even of gingivitis, are seen adjacent to the aphthous spots. There is an increased flow of the secretions of the mouth, and the breath is heavy, but seldom offensive. *General symptoms*, as slight fever, anorexia, and furred tongue, constipation or diarrhea, and irritability, are usually present, with the additional symptoms of any associated disease that may coexist. Gastro-intestinal affections, though often associated with aphthous stomatitis, are most probably due to the common cause, and are not necessarily the cause of the stomatitis in these instances. In some of the specific infectious fevers many aphthæ may appear and tend to run together; these form large irregular ulcers, and give rise to the *confluent form* of stomatitis aphthosa. The special form known as *Bednar's aphthæ*, occurring in young marantic babes, is a rare condition in America. Large white patches are seen on both halves of the posterior part of the hard palate near the alveolar processes, and these may cause large ulcers and involve the bone. Pressure of the tongue upon the thin mucous membrane during nursing, or other forms of traumatic irritation, appear to act as causes.

Aphtha Cachectica (Riga's disease).—Fede has described a form of aphtha, occurring principally in Southern Italy (a raised, gray swelling), situated on the frænum and under surface of the tongue. It affects children soon after the eruption of the lower incisors. A severe type is sometimes met, and this may terminate fatally.

The average *duration* of the ordinary discrete aphthous eruption is from four to seven days; in very ill-nourished and poorly cared-for cases the appearance of successive crops of aphthæ will prolong the distress.

Diagnosis.—This is based upon the characteristic appearance of the ulcers and the degree of soreness. Aphthæ must be differentiated from *thrush* (see page 743), where the distinguishing features will be dwelt upon in the description of the latter affection. Herpes of the mouth, so called, and aphthous vesicles are probably identical in most cases.

Prognosis.—The discrete form is mild, and favorable in its course toward recovery; confluent aphthæ is more troublesome, and follows a prolonged course on account of the general debility induced by the associated disease (Starr). In certain cases the affection is apt to recur; relapses are also frequent in those having weak digestive and imperfect assimilative functions. Recovery from Bednar's aphthæ is rare.

Treatment.—It is first necessary to remove all irritating influences, and in order to minimize the intense pain of the aphthous spots the blandest liquids and the softest foods that are consistent with the sustenance of the patient are imperative. Absolute cleanliness of the mouth, the foods, and the vehicles of administration, especially in bottle-fed children, is important. Local applications are of obvious value. Demulcents, as mucilage of sumac, or of marshmallow, with boric acid (gr. v to ʒj—0.324 to 32.0), sodium bicarbonate (gr. v–x to ʒj—0.324–0.648 to 32.0), carbolic acid, or potassium permanganate (gr. iv

to ℥j—0.259 to 32.0), are invariably useful. Swab-applications of wine of opium (♏v to ℥j—0.333 to 32.0) or of cocain (4 per cent. solution) may be necessary when the pain is intense, and prior to taking food.

To promote the healing of the ulcers a very light touch with the silver-nitrate stick or solution (gr. x–xxx to ℥j—0.648–1.94 to 32.0) is often beneficial. Much favor is deservedly given also to potassium chlorate in solution (gr. x to ℥j—0.648 to 32.0), or applied in the dry powdered form. In the confluent aphthous ulcer the use of sodium salicylate (℥j to ℥j—4.0 to 32.0) has been recommended, while an ethereal solution of iodoform (℥ij to ℥j—8.0 to 32.0) has been advised by J. Lewis Smith. For bleeding and spongy gums the mild astringents mentioned in the treatment of catarrhal stomatitis are indicated. Stronger astringents may answer for application to sluggish ulcers; thus copper sulphate, either solid or in solution (gr. x to ℥j—0.648 to 32.0), and zinc sulphate (gr. xv to ℥j—0.972 to 32.0) are useful. Potassium chlorate acts as a specific in ulcers of the mouth, and is eliminated by the buccal secretions, which keep the ulcerated surfaces constantly bathed with the drug, so that its internal use is to be recommended, though in very small doses in children, well diluted, as in the following formula:

℞. Potassii chlorat., gr. xxiv (1.55);
Tinct. myrrhæ, gtt. x (0.666);
Syr. acaciæ, f℥ij (64.0);
Aquæ dest., q. s. ad f℥iij (96.0).—M.
Sig. Teaspoonful every three hours for a child three years of age.

Constitutional symptoms are to be combated on general principles and require careful attention. Remedies directed to the correction of digestive derangements and to the stimulation of assimilation are also often required. Good food and ferruginous tonics are necessary.

MEMBRANOUS STOMATITIS.

(*Stomatitis Crouposa.*)

Definition.—In this form of stomatitis the inflammation is more intense and more extended in area than in the aphthous form, being also attended with the formation of a false membrane.

The **pathology** of these membranous patches, which are very seldom confined to the mouth alone, is embraced in the article on Diphtheria. If in the latter disease the typical false (diphtheritic) membrane is removed, it leaves a deeper ulcer than does the removal of a croupous membrane, in which the coagulation-necrosis involves the more superficial layers only.

The **etiology** of membranous stomatitis is usually specific (diphtheritic, streptococcic). Membranous stomatitis may also be due to gonorrhœal or syphilitic infection of the new-born.

Symptoms.—Some of these cases are, doubtless, true diphtheria of the oral cavity (usually secondary from extension), and an account of the symptoms presented may be found in the chapter on Diphtheria, p. 151. The writer has seen instances of extensive membranous stomatitis, in which bacteriologic examination showed the presence of streptococci (principally), and also staphylococci. The symptoms were

analogous to streptococcic membranous pharyngitis with this difference, in my cases of membranous stomatitis, that salivation was marked and distressing. The *general features* may be quite pronounced.

ULCERATIVE OR FETID STOMATITIS.

(*Stomatitis Ulcerosa.*)

Definition.—A specific ulcerative inflammation of the buccal mucous membrane and gums, attended with marked fetor of the breath, and having a tendency to extend widely and deeply.

Etiology.—The predisposing causes of this malady are principally as follows: Childhood, after the commencement of the first dentition, and usually between the ages of three and eight years; damp weather, especially during spring and autumn; unhygienic surroundings, particularly the lack of pure air, of good and abundant food and clothing, and the added detriments to health for which neglect and filth, specific infectious diseases, uncleanliness of the mouth, caries and loosening of the teeth, and congenital heart-disease (Duckworth) are responsible. An endemic type of this affection has been observed among soldiers in camps and barracks, among children in crowded eleemosynary institutions, and in jails. Its epidemic and contagious character likewise points to a microbic origin. The specific *exciting cause*, it has been held, corresponds to the hoof-and-mouth disease of cattle, the poison being conveyed in milk. Payne suggests the identity of the virus with that of impetigo contagiosa. The careless administration of mercury may also be followed by this affection. Scurvy (*scorbutic stomatitis*) and the persistent use of lead and phosphorus are also excitants.

Clinical Symptoms.—*Locally*, the disease starts, as a rule, at the edges of the gums opposite the lower incisor teeth, gradually spreading backward and to the adjoining portions of the lips and cheeks. The gingival mucous membrane is deeply red and swollen; the gums soon become spongy, bleed easily, and break down into thick, soft, grayish sloughs, which leave deep and ragged ulcers surrounding the necks of the teeth. The latter even become loosened, and in protracted cases the alveolar periosteum may become inflamed and cause necrosis of the bone. Profuse salivation, a foul breath (that once earned for the condition the term of "putrid sore mouth"), occasional slight hemorrhages from the gums, and excessive discomfort, or even pain, on mastication are nearly always present. The tongue is coated, swollen, and tooth-marked; aphthæ are sometimes seen, and the submaxillary glands are generally swollen. The *general symptoms* attending this ailment are those of a lowered state of vitality, produced by an unhygienic environment, or cachexia, or severe illness primary to it, with, usually, moderate fever. Nausea and vomiting or an offensive diarrhea may supervene as the result of swallowing the putrid discharges.

Course and Duration.—Usually acute in its course, the highly debilitating character of the disease may tend to make it chronic, especially when there is alveolar necrosis and a neglect of proper treatment. Ordinarily, with careful management, convalescence may be established in from four days to a week. Goodhart regards the occasional termination of the pyrexia by lysis, with an accompanying im-

provement of the local symptoms in such cases at least, as suggestive of the specific nature of ulcerative stomatitis.

Neurotic Ulceration.—Under the head of *stomatitis neurotica chronica* Jacobi describes an herpetic (rarely pemphigoid) eruption in neurotic subjects. Sibley has met three cases, all of which occurred in women beyond middle age. In all the condition had lasted for a number of years and produced great difficulty in talking and mastication.

Diagnosis.—Upon examining the mouth and noting the characteristic ulceration, the fetid breath and saliva, and the cachectic appearance, the disease is usually recognized, and should not be confounded with the dark, extensive, gangrenous sloughs of noma.

The **prognosis** is favorable in typical cases, though less so in marasmic and neglected cases. When cancrum oris or necrosis of the jaw occur, chronicity, deformity, and even death, may take place.

Treatment.—It is well in nearly all ill-nourished, uncleanly-kept, and sickly children, as well as in cases in which mercury is to be administered for any length of time, to prescribe mouth-washes of potassium chlorate (gr. xv to ʒj—0.972–32.0), in order to prevent the occurrence of mercurial or ulcerative stomatitis. The *hygienic treatment* of ulcerative stomatitis is important. On account of the contagiousness of the disease cases occurring in a family or in institutions should be isolated, and fresh air, light nourishment, and cleanliness are *sine quâ non* of recovery. The *local* treatment is virtually a specific one in the use of potassium-chlorate washes (gr. x–xx to ʒj—0.648–1.296 to 32.0—of rose-water or demulcent), aided by the internal administration of the same salt in small doses. For the disagreeable fetid odor the alternate use of antiseptic washes is indicated. Solutions of carbolic acid or potassium permanganate, in strength equal to or slightly over that indicated in the treatment of aphthæ, or hydrogen peroxid (ʒj–iij to ʒj; 4.0–12.0 to 32.0), or listerin and water (equal parts), are useful. Pencilling the spongy gums with such astringents as tincture of rhatany, silver nitrate (gr. x to ʒj—0.648–32.0), alum, and also with tannic-acid solutions, may be necessary. Loosened teeth should not be disturbed, as they may grow firm with convalescence, though surgical interference may be required in cases of necrosis of the alveolar process. Kissel's method in obstinate cases is to curette the ulcers and rub into them powdered iodoform once daily. Careful attention to the teeth is always requisite. During the height of the disease constitutional treatment may have to be directed toward stimulating the languid and lowered vitality. For this purpose either whiskey or brandy, in half- or one-teaspoonful doses in milk, is extremely useful; the elixir of cinchona, with some vegetable salt of iron, as the citrate or tartrate, also forms a useful combination. When there is pyrexia or a diminished urinary secretion the internal use of potassium chlorate must be cautiously employed. The following prescription is almost specific:

℞. Potassii chloratis, gr. xlviij (3.11);
Acidi hydrochlor. dil., fʒj (4.0);
Syrupi, fʒvj (24.0);
Aquæ destillat., q. s. ad fʒiij (96.0).—M.
Sig. Teaspoonful diluted, every two hours for a child three years old (Starr).

The prolonged use of tonics and cod-liver-oil emulsion with lime-salts in scrofulous, rachitic, and scorbutic subjects must be carried on in order to prevent relapses of ulcerative stomatitis.

PARASITIC STOMATITIS.

(*Thrush; Stomatitis Mycosa.*)

Definition.—A specific, contagious fungous disease, characterized by the rapid formation upon the oral mucous surfaces of small, whitish, soft, and lightly adherent spots or flakes, tending to coalesce and spread throughout the entire buccal cavity.

Etiology.—*Predisposing causes* are—infancy with its concomitant disorders of the gastro-intestinal tract (especially when unhygienic surroundings prevail), congenital syphilis, tuberculosis, and the exanthemata. The disease may attack adults and complicate the typhoid and cachectic states, as in the final stages of low fevers, carcinoma, chronic tuberculosis, and diabetes. The growth of thrush-patches is due, specifically, to the *saccharomyces albicans*, though micrococci have also been found. It is a characteristic of this fungus to develop from round or oval spores in the formation of long-branching mycelium filaments, from the ends of which a multiplication of ovoid torulæ-cells takes place by the process of simple budding. These mycelia exhibit a tendency to penetrate the deeper layers of the mucosa of the mouth and also into the mucous glands (Starr). Since the growth of this organism requires both an altered condition of the mucous membrane and an acid medium, the primary or exciting cause of thrush is to be found in whatever produces such a favorable nidus. Most important in this connection is uncleanliness, particularly in the case of poorly-nourished and bottle-fed children. The development of catarrhal stomatitis and the acid fermentation of remnants of food (especially of saccharine substances), which impair the nutrition of the mucosa and acidify the normally alkaline oral secretions, are common causes of thrush. The further growth of the fungous patches also contributes to the acid state of the already abnormal buccal fluids. The fact that the spores of thrush may be transferred to other cases by bottle-tips, spoons, and ill-kept feeding-bottles is well recognized as an explanation for the occasional endemic character of the malady.

Symptoms.—Any marked local symptoms are due rather to the coexisting stomatitis than to the thrush itself (Allchin). There will be some *soreness*, *heat*, *persistent dryness*, and *lividity* of the mucous membrane. Thrush-spots, slightly raised above the surface, begin to appear on the tongue, and grow into patches that may coalesce and spread to the cheeks, lips, and hard palate: they may even invade the tonsils, pharynx, and esophagus, and, rarely, the true vocal cords, the stomach, and cecum (Parrot). At first pearly-white in color, the curd-like flakes may become yellow and even brown, owing to slight hemorrhages caused by the irritation. Though early adherent, in a few days they become loose, and when brushed off leave a smooth surface; when complicating some serious gastro-intestinal disease or dyscrasia, however, their attachment is deeper, and the deposit may sometimes appear in successive crops. A *microscopic examination* of the thrush-patches shows inter-

lacing, irregular, and branched mycelial threads, spores, occasional bacilli, and leptothrix-filaments imbedded in a mass of granular débris and fetid particles. The buccal fluids are acid in reaction. The *general symptoms* depend upon the associated disease, and are usually those of wasting, artificially-nourished children having digestive troubles or a constitutional taint.

Diagnosis.—This may be accurately made upon the discovery of the fungus by microscopic examination. Only very rarely are portions of the thrush-organism found in the false membrane of *diphtheritic stomatitis*. *Milk curds* may be readily removed, and are not necessarily associated with the stomatitis accompanying thrush or the grave systemic states. The important point of differential diagnosis arises in the case of *aphthæ*. The following table will express the main points:

Parasitic Stomatitis (Thrush).	Aphthous Stomatitis.
Dryness of the mouth.	Salivation.
Whitish, raised spots or patches with no red areola; these are easily removed, leaving no ulcer and causing no bleeding.	An ulcer with a yellowish-white, depressed base, surrounded by a red areola. The base is removed with difficulty by forceps, and bleeding results.
Spots are numerous.	Usually few in number and discrete.
Begins in the form of minute spots.	Not so; ulcers appear, preceded by the formation of herpetic vesicles.
Ulcers not painful. Discomfort depends on the associated stomatitis.	Ulcers exquisitely tender.
The characteristic thrush-fungus is always detectable with the microscope.	No specific micro-organism determined, though probably present.

Prognosis.—This is favorable as regards the thrush alone, but, occurring in marantic children and cachectic adults, its appearance is of grave significance, and may portend a speedy death.

Treatment.—Prophylaxis is of great moment, since it is much easier to keep the mouth clean and the secretions normal, and to attend to proper food, and thus avoid creating a soil for the growth of the vegetable parasite, than it is to prevent absolutely the entrance of thrush-spores. Efforts directed toward preventing acidity are especially indicated. This is to be done by the use of mild alkaline mouth-washes, as soda-water and lime-water. The dietary should be carefully looked after, and should exclude sugars and all starchy food; the addition of lime-water to the milk (about one part to four) is a desirable precaution to take, particularly with children. Cleansing the feeding-apparatus and the mouth after each feeding is essential, both in the prevention of the formation, and in decreasing the further growth, of thrush when present. The local treatment consists in the use of alkaline and antiseptic applications, preferably by means of the spray. Solutions of boric acid or sodium hyposulphite (℥j—4.0—of either to ℥j—32.0—of water, with the addition of a little glycerin), potassium permanganate, or hydrogen peroxid, are useful. Syrupy excipients are to be excluded. Potassium chlorate may exert a beneficial effect in those cases in which stomatitis is associated, as may also pencilling with a solution of silver nitrate. Concetti[1] urges the use of a 3 to 5 per cent. solution of silver nitrate instead of the weaker strength usually employed. The use of the galvano-cautery is often serviceable.

[1] *Rev. mens. des Mal. de l'Enfance*, July, 1899.

When esophageal obstruction exists it may be necessary to gently force a rubber tube through the mass of thrush-deposit in order to give nourishment (Forchheimer).

Medicinal treatment embraces the administration of nourishing and easily digestible food, occasional stimulation, and the correction of gastro-intestinal disorders. Attention must also be paid to the primary affections to which the thrush is superadded. Iron, cod-liver oil, and acid and bitter tonics in palatable form are usually indicated in debilitated subjects, along with general hygienic measures. The internal use of small doses, frequently repeated, of calomel or mercuric chlorid may also be tried for a possible specific effect in combating thrush.

LA PERLÈCHE.

This contagious disease is confined to the angles of the mouth. It was first described by Lemaistre in 1886 as prevalent among the children of Limousin in France. It was found that the drinking-water in that locality contained cocci similar to the spherobacteria that infested the epithelial thickenings, and that these were probably conveyed to human beings by drinking-vessels. Little elevations and fissures, said to resemble those of congenital syphilis, were seen around the oral angles. The latter were the seat of smarting pain, particularly on opening the mouth suddenly or too far, and caused the patient to lick (*perlicher*) them constantly. The disease seemed to be entirely local, and lasted from two to three weeks. Alum and copper-sulphate solutions were most useful.

GANGRENOUS STOMATITIS.

(*Noma; Cancrum Oris.*)

Definition.—A rapidly-spreading gangrenous affection of the cheek and gums, of rare occurrence, usually asymmetric, and ending fatally in most cases.

Pathology.—In addition to the necrotic changes in the cheeks, the process may extend to the jaws and lips. The blood-vessels contain thrombi, thus preventing hemorrhage from the sphacelus. The submaxillary and cervical glands may be slightly enlarged and soft. Blood-changes of an uncertain character have been noted. Hemorrhagic infarctions, aspiration broncho-pneumonia, or gangrene by inhalation of gangrenous particles or metastasis, may be met in the lungs. Wharton has described an associated membranous form of colitis, and a metastatic infiltration of the cardiac muscle and purulent pericarditis may also be seen *post-mortem*. Klementorosky met with a peculiar and fatal form of gangrene limited to the gums of babes and occurring a few days after birth.

Etiology.—*Predisposing Causes.*—This uncommon affection attacks girls more frequently than boys, usually between the ages of two and five years; it appears to be endemic in low, moist countries, as Holland, though apparently it has not been regarded as contagious in the past. Children suffering from the effects of overcrowding and previous disease are especially liable to noma. Most often, however, it is secondary to measles; it may also follow scarlet fever, typhoid, small-pox, or less fre-

quently pertussis. The causative influence of mercurialization and ulcerative stomatitis has been overrated.

Bacteriology.—Jos. Sailer[1] recovered diphtheria bacilli from the gangrenous areas of noma of the buccal cavities. Guzzetti found pseudo-diphtheritic bacilli together with staphylococci and streptococci.

Symptoms.—The mucous membrane of one cheek, near the corner of the mouth, is usually first affected, a *dark, ragged, sloughing ulcer* appearing and spreading insidiously for two or three days before the substance of the cheek is involved. A *hard* and *sensitive nodule* may then be felt by grasping the thickness of cheek between the thumb and finger. Brawny induration of the skin over this nodule soon becomes manifest, and then there appear collateral edema and an unctuous-looking, deeply livid, gangrenous spot, soon becoming bullous and leaving a black eschar. Perforation of the cheek may occur on the third day, though usually not until a week has passed. There is an *ichorous discharge* of shreds of gangrenous tissue from the unhealthy wound. The *fetor* of the breath is almost intolerable and characteristically gangrenous. The necrosis may extend over one-half the face of the side affected, and may involve the gums and jaws, but seldom does it attack the opposite side of the face. The *general symptoms* of such a grave malady may be slight at a very early period, but with the formation of the eschar they become rapidly severe and typhoid in type. Great prostration, delirium, pyrexia (104° F.—40° C.), diarrhea, and edema of the feet are common. The course rarely extends beyond two weeks.

Complications.—Septic lobular pneumonia may occur from aspiration of gangrenous particles; colitis and gangrene of the genitalia in females (*noma pudendæ*) are also seen. In those very rare cases that recover granulations form, the gangrenous edges become clean, and cicatrization follows, often with great disfigurement of the face and even restricted jaw-motion.

Diagnosis.—The disease when fully established is easily diagnosed by its characteristic origin, the gangrenous ulcer-nodule, the eschar-formation, and perforation, associated with a previous history of measles or other acute infectious fever of childhood. The offensive fetid odor and severe constitutional depression are also of great value.

Differential Diagnosis.—From *anthrax* it differs in that the latter affection is more common in adults, with a history of contagion, and in the fact that malignant pustule starts on the exterior of the cheek, and perhaps in a previous abrasion in the skin. The discovery of the bacillus anthracis in the blood and discharges is conclusive. *Ulcerative stomatitis* of a severe and neglected type may be confounded with cancrum oris, but in the former the destruction of tissue is mainly of the gums and alveoli, the cheeks being simply ulcerated and no extensive sloughing taking place; the breath, though fetid, is not gangrenous, and the oral discharge, though sometimes bloody, is not mixed with shreds of gangrenous tissue (Starr). Finally, the course of ulcerative stomatitis is less severe, a fatal termination being extremely rare.

Prognosis.—Noma is seldom recovered from, the mortality being about 80 to 90 per cent. (Bogel). When recovery does take place the

[1] *Philadelphia County Medical Society*, Nov., 1901, p. 301.

development of ectropion, facial deformity, and local disability, with a protracted convalescence, render life burdensome.

Treatment.—Quarantine all cases until they are proven by bacteriologic study to be of a nondiphtheric nature, and the avoidance of mercurialization will also be of undoubted use. The primary indication in the *local treatment* is the arrest of the gangrenous process, thus causing, if possible, a healthy reaction on the part of the surrounding tissues. All sloughs should be cut away, followed by cleanliness of the mouth and wound; and by the application of strong caustics, as fuming nitric acid, the acid nitrate of mercury, solid zinc chlorid, silver nitrate, carbolic acid, a concentrated solution of perchlorid of iron, Vienna paste, and the actual cautery. For the protection of the healthy parts and for efficiency the Paquelin or the galvanic cautery is probably best. Anesthesia is requisite for such strong measures. Milder applications, however, seem to be quite adequate in some cases. Thus, bismuth subnitrate, potassium chlorate, and aristol, or the following formula by Dr. Coates, may be tried:

℞. Cupri sulph., ʒij (8.0);
Pulv. cinchonæ, ℥ss (16.0);
Aquæ, q. s. ad fʒiv (128.0).—M.

As a mouth-wash employ mild antiseptic washes of carbolic acid, hydrogen peroxid, Labarraque's solution, potassium permanganate, etc.; and for the diminution of the fetor, antiseptic charcoal poultices containing boric or salicylic acid are useful. Mild antiseptic and astringent lotions of boric acid, zinc sulphate (gr. ij to ℥j—0.129 to 32.0), or balsamic ointments with vaselin, may aid in healing the granulating surfaces in favorable cases. The internal treatment must be directed toward sustaining the strength of the patient by the administration of the most nourishing food, stimulants, and tonics. Rectal feeding may be necessary. Plastic operations may be needful after recovery to mitigate oral disabilities or facial deformities. W. C. Cahall has successfully treated a case of noma with anti-streptococcus serum. Antitoxin should be given early where the diphtheria bacillus is found.

MERCURIAL STOMATITIS.

(*Mercurial Ptyalism.*)

Definition.—An inflammation of the mouth and salivary glands, caused by the excessive use of mercury; a similar condition is rarely seen as a result of the therapeutic use of other drugs.

Etiology.—Predisposing causes are dyscrasia and occupation, mainly. The peculiar individual susceptibility of these subjects to dyscrasia will not permit the use of even minimum doses of mercury without serious and almost immediate symptoms of ptyalism. This is also seen in barometer-makers, mirror-silverers, chemists, and others who handle mercury in their daily work. The exciting cause of ptyalism is the ingestion, inhalation, or cutaneous absorption of mercury.

Symptoms.—A *metallic taste* in the mouth is first noticed by the patient. Soon the *gums* become "touched"—*i. e.*, red, swollen, tender

to the touch, and sore during the act of mastication. A marked secretion and *flow of saliva*, with a *fetid breath* and swollen tongue, follow. Very rarely in this disease the affection passes into an *ulcerative stomatitis*, and causes loosening of the teeth and necrosis of the maxilla. *General symptoms*, as constitutional depression, anorexia, diarrhea, mental anxiety, and nervousness, may supervene.

The recognition of the foregoing causal factors—predisposing and exciting—renders the *diagnosis* easy. The *prognosis* is favorable, and, although the local symptoms may be harassing, recovery is attainable within a few weeks as a rule.

Treatment.—The toxic action of mercury in the production of ptyalism can be avoided by a knowledge of individual susceptibility and by the local and internal use of potassium chlorate. Upon the first appearance of the symptoms there must be a prompt withdrawal of the mercurial influence, and a change of occupation if that be the predisposing cause. Locally, soothing, alkaline, and mildly antiseptic mouth-washes, as in the treatment of catarrhal stomatitis, may be all that is necessary. For the fetid breath solutions of boric acid or potassium chlorate may be used. Ulcers may be brushed with silver-nitrate solution. The internal treatment should be directed toward keeping the bowels soluble; in addition, alkaline mineral waters may be used, and in severe cases potassium chlorate in 5- to 10-grain (0.324–0.648) doses. Atropin (gr. $\frac{1}{100}$—0.0006) and opium have been recommended to decrease the excessive salivary secretion and to allay pain, and hot baths will aid the treatment materially. In severe cases the resulting debility and anemia should be met by the use of highly nourishing liquid foods and by tonics.

Osler points out that the condition of the teeth known as *erosion*, which sometimes follows infantile stomatitis, and especially the mercurial form, is to be discriminated from the deformed teeth of congenital syphilis. In the former the first permanent molars, and then the incisors, are observed to have small pits or discolored and eroded spots, due to a morbid deficiency in enamel-formation. The notched and irregular teeth of hereditary syphilis in children (Hutchinson) are sufficiently distinctive.

II. DISEASES OF THE TONGUE.

GLOSSITIS.

ACUTE GLOSSITIS.

(*Glossitis Acuta.*)

Definition.—An acute parenchymatous inflammation of the tongue, sometimes ending in abscess.

Etiology.—Predisposing causes are supposed to be an impaired general health and exposure to cold, humid weather. The exciting causes

are most frequently the stings and bites of insects, or burns, scalds, and the action of corrosives. I believe that many cases follow slight injuries to the tongue that allow of the introduction of inflammatory poisons or microbes. A. J. Hall describes a case of membranous glossitis complicating acute nephritis.

Symptoms.—These come on *rapidly* and with more or less local severity and danger. The *tongue* becomes much swollen, and may even protrude beyond the lips. It is very *tender* and *painful*, and coated with a thick, soft yellowish-white fur, and it may also be dry, cracked, and ulcerated. *Catarrhal stomatitis* is often associated, salivation is usually profuse, and talking, swallowing, and even breathing are rendered difficult and distressing. *Dyspnea*, even to suffocation, may be imminent. The cervical and sublingual glands may be swollen, moderate *fever* is always present, and the obstruction to breathing and administration of nutriment may assume a dangerous aspect.

The inflammation reaches its height in about three or four days, tending to subside almost entirely about the seventh day. Not rarely the inflammatory infiltration passes into suppuration with the formation of a circumscribed abscess of variable size in one-half of the tongue; fluctuation may not, however, be obtainable, spontaneous rupture being sometimes the first indication of abscess. The **prognosis** is favorable, except that serious obstruction is likely to remain.

Treatment.—When the case is seen quite early and during the congestive stage, the topical use of ice, allowed to slowly dissolve in the mouth, is serviceable. Mucilaginous mouth-washes, containing some mild antiseptic, as sodium borate with sodium bicarbonate (gr. v–xx to ʒj—0.324–1.296 to 32.0), should also be employed. A brisk saline purge, given early, will aid in reducing the inflammation, and should the tongue become alarmingly swollen, deep scarification and the use of half a dozen leeches between the hyoid bone and the jaw-angles may be of decided service. Steam-atomization, medicated with the compound tincture of benzoin or ammonium chlorid (ʒj to ʒj—4.0 to 32.0), favors resolution (Cohen). Abscesses must be incised and washed out with antiseptic solutions. Tracheotomy is rarely called for to relieve the dyspnea. Rectal alimentation with predigested foods may be necessary, and during convalescence ferruginous tonics in glycerin and bland foods should be continued for some time, in order to prevent chronic inflammation and thickening. Any local source of irritation, as from carious or sharp teeth, should be removed.

CHRONIC SUPERFICIAL GLOSSITIS.

Definition.—A chronic inflammation of the mucosa of the tongue.

Etiology.—This disease is often preceded by several acute attacks, the habitual use of tobacco, both in smoking and chewing, and of strong spirituous liquors being mainly productive of the original affection. The frequent use of irritating foods is also a factor in some instances.

Symptoms.—The surface of the tongue is continually sensitive and more or less reddened. Often there are seen ovoid patches of various size, smooth and shiny, on account of the loss of papillæ, and separated by furrows that extend to the depth of the mucosa itself. The tongue

may also be slightly furrowed in intervening spaces, especially at the base. The general health is somewhat deteriorated.

Diagnosis.—This rests upon the history of the case and upon the results of examination of the organ.

The **prognosis** is favorable as to alleviation, but guarded as to cure.

Treatment.—The blandest dietary must be insisted on, as well as absolute abstention from the causal irritants. The local use of demulcents and of mildly alkaline and antiseptic lotions, such as Seiler's tablets in solution, and of solutions of chromic acid or silver nitrate (gr. v–x to ℥j—0.324–0.648 to 32.0) in water or honey, applied once or twice daily by gentle brushing, is to be recommended. General tonics and the avoidance of irritating drinks will be indicated.

GLOSSITIS DESICCANS.

A rare disease, chronic in nature and of unknown causation. It is characterized by "the gradual development upon the surface of the tongue of a number of deep fissures and indentations, giving the organ an uneven and ragged look. The pain is due to the frequent presence of excoriations and ulcers in these fissures" (Strümpell). The *prognosis* of the affection is favorable as regards any danger. The *treatment* is hygienic, consisting of cleanliness of the mouth and the use of disinfectant mouth-lotions, together with the topical use of alterative or astringent applications, as silver nitrate or chromic acid, to any ulceration.

LINGUAL PSORIASIS (TYLOSIS LINGUÆ).

In this disease there are small regular areas of hyperplasia of the glossal epithelium, eventually causing a map-like appearance of the surface of the tongue—"lingua geographica." The trouble is obscure in its etiology and persists for years. Seldom is there any discomfort associated, although mental anxiety or hypochondriasis may develop.

LEUKOPLAKIA ORIS (BUCCAL PSORIASIS).

In this affection the mucous membrane of the mouth and tongue may be involved. On the lateral borders of the tongue white or bluish-white scar-like spots or patches, often slightly notched, make their appearance. Some of these pass away to be replaced by others, and the affection progresses despite all attempts to cure it. The true *cause* is unknown, but it has been suggested that some irritant, as the use of a pipe, may account for the condition. The malady has, however, been seen in women. A syphilitic taint is said to especially predispose to the disease (Strümpell). The affection must be carefully diagnosed from the oral manifestations of syphilis, if for no other reason than to relieve the mind of a morbidly anxious patient. Excepting some pain connected with possible ulceration, there are no annoying *symptoms*, and the *treatment* suggested for glossitis desiccans is appropriate. Kyle touches the white patches daily with pure tincture of iodin.

In children a similar tongue-affection has been named "wandering rash." The patches are circinate and enlarge peripherally, forming rings

of epithelial hyperplasia, within which is a red, glossy center, "devoid of filiform papillæ, though the fungiform remain" (Allchin).

ANGINA LUDOVICI.

(*Ludwig's Angina.*)

Definition.—A rare acute phlegmonous inflammation of the floor of the mouth.

Etiology.—The condition is more common in males, and may be secondary to specific infections (scarlet fever, diphtheria). Thomas states that insignificant lesions in the mouth (*e. g.*, carious tooth, ulcer, tonsillitis) are the usual primary foci leading to lymphatic involvement. The specific organism is generally the streptococcus, though rarely the staphylococcus is found. It may result from trauma.

Symptoms.—These are *intense* at the *outset*, and begin with swelling in the region of the submaxillary gland, with a rapid involvement of the cellular tissue of the floor of the mouth as well as of the anterior portion of the neck. *Pain* is marked, and this, with the *acute swelling*, renders articulation, mastication, and deglutition extremely difficult. Compression or edema of the larynx may often cause dangerous dyspnea. The *constitutional disturbance* is usually febrile, and may either approach the typhoid type or may be septic. The condition generally terminates either in abscess or extensive sloughing (*cynanche gangrænosa*).

The **diagnosis** is easily made when complicating a specific fever.

The **prognosis** is always grave. Of 106 cases collected by Thomas, 43 died. Relapses may follow in weakly subjects.

Treatment.—The most that can be done is to sustain the strength of the patient and secure prompt surgical interference. Tracheotomy may be demanded if asphyxia threatens life.

III. DISEASES OF THE SALIVARY GLANDS.

HYPERSECRETION.

(*Ptyalism.*)

Definition.—An abnormal increase in the secretion of saliva.

Etiology.—Salivation as an idiopathic affection is rare, and as such is considered to be a neurosis. Thus, it has been seen in emotional children of from two to eight years of age, though apparently in perfect health. According to Bohn, the secretion in these cases is mostly increased during active exercise, is reduced on lying down, and absent during sleep. Spontaneous recovery takes place in a few years. As a deuteropathic disease ptyalism may be the result of oral disease (*e. g.* noma, ulcerative stomatitis), and also of gastro-enteric, pancreatic, uterine (as gestation), centric (as diseases or tumors of the medulla or of the facial nerve), toxic, systemic (as small-pox, the use of mercury, iodids, pilocarpin, tobacco), and hydrophobic irritation and disease.

Diagnosis.—It should be pointed out that a failure in swallowing the normal quantity of saliva may cause dribbling from the mouth and simulate true hypersecretion.

The **prognosis** is favorable in itself, but dependent on the cause.

Treatment.—The causes are to be removed and the general health toned up. For stomatitic salivation potassium chlorate is first in rank as an internal and local remedy. Iron and arsenic are valuable in neurotic cases, and the bromids or hyoscin may be of supplemental use. Atropin (gr. $\frac{1}{200}$ to $\frac{1}{100}$—0.0003 to 0.0006) and belladonna are almost uniformly successful in idiopathic as well as in central ptyalism.

XEROSTOMA.

(*Aptyalism; "Dry Mouth."*)

Definition.—A morbid arrest of the salivary and buccal secretions.

Etiology.—The disease is probably due to an affection of the nerve-supply of all the glands of the mouth (Harris). It may follow sudden mental phenomena as a temporary condition. A. J. Hall collected 39 cases, of which 32 occurred in females. In most of the cases the causes were unknown. Not uncommonly xerostoma is an effect of the febrile state, of mouth-breathing (due to nasal obstruction), and of diabetes.

Symptoms.—Apart from the sensation of dryness, mastication, deglutition, and articulation are difficult. The *local appearances* show a glazed, red, and sometimes cracked condition of the tongue and labial and palatine mucosa. The teeth may become diseased and crumble.

The **diagnosis** is made on inspection, the **prognosis** depending on the removability of the cause, and rightfully being guarded on account of the frequent obstinacy of the trouble.

Treatment.—Attention to the systemic condition is requisite. Small doses of potassium iodid and pilocarpin (gr. $\frac{1}{20}$—0.003) in gelatin lamellæ or in lozenge form, allowed to dissolve in the mouth with the aid of a sip of water, have been productive of relief. In cases of centric origin the galvanic current should be tried.

Glassblowers' Mouth.—This condition is found among glassblowers and also among musicians. It occurs in about 2.5 per cent. of all glassblowers. Scheele,[1] who reports two cases, describes the condition as a hernial-outpocketing of the muscles of the cheeks. The epithelium of the mucous membranes shows the so-called *plaques opalines*. It is often combined with a distention of Steno's duct. In addition to a ballooning out of the buccal mucosa, there is likely to be a disturbance of hearing and cramp-like contraction of the cheek. The parotids may be emphysematous and crepitate on palpation.

SYMPTOMATIC PAROTITIS.

(*Parotid Bubo.*)

Definition.—A secondary inflammation of the parotid gland, generally due to septic infection and tending to suppuration.

Etiology.—Not being a primary affection, the causes giving rise to it may be mentioned as follows: (*a*) Acute infectious fevers, as typhoid, typhus, pneumonitis, pyemia, erysipelas; (*b*) Injury or disease of the abdomen or pelvis (Stephen Paget), or of the genito-urinary tract, as mild traumatisms or derangement of the testes or ovaries, or even

[1] *Berlin. klin. Wochenschrift*, Mar. 12, 1900.

menstruation or pregnancy; gastric ulcer may be accompanied by it; (*c*) Peripheral neuritis with facial paralysis (Gowers).

Most of the cases are septic and indicative of an unfavorable course in the progress of the associated disease. The *symptoms*, *diagnosis*, and *treatment* of the parotitis itself fall under the scope of surgery.

Chronic Parotitis.—Mikulicz first described this condition and reported a case in which symmetric enlargement of the lachrymal, and subsequently of the salivary, glands occurred. Kümmel and Osler have also recorded cases. It may be caused by lead or mercury and may be secondary to mumps, inflammation of the throat, and chronic Bright's disease. The condition may be painless.

IV. DISEASES OF THE TONSILS.

ACUTE TONSILLITIS.

Definition.—An acute inflammation of the tonsil or tonsils, affecting either the mucous membrane, the follicles, or the parenchyma, and ending either in resolution, suppuration, or chronic enlargement.

Pathology.—In the *superficial variety* of acute tonsillitis the mucosa is simply red, swollen, and sometimes covered with a thin, soft exudate of muco-pus. The tonsil itself may also be swollen. In *follicular tonsillitis* the lacunæ become filled with a cheesy exudate which often protrudes from the tonsillar crypts; epithelial and pus-cells, cellular débris, and occasional cholesterin-crystals are found in these cheesy masses. In older, darker-hued masses an offensive odor is given off, and numerous micrococci and bacteria are found. In adults, calcareous infiltration of the cheesy little masses may be met with. *Parenchymatous tonsillitis* is shown by a greater enlargement of the tonsil, due to a marked infiltration of all the tissues. Suppuration in the tonsil is frequent, the follicles usually bursting and uniting in abscess-formation. Pus may burrow into the cellular tissue surrounding the tonsil, and find its way even down to the clavicle. The *herpetic* or *ulcero-membranous form* of tonsillitis described by Rilliet and Barthez, DaCosta, and others, in which an eruption of herpetic vesicles on the tonsils is followed by their rupture and the formation of a lightly adherent membrane is rare. In *necrotic tonsillitis* (Strümpell) a grayish-white adherent necrotic membrane is observed, that is limited by the inflamed membrane surrounding the mucosa covering the tonsils. The latter are moderately swollen. A dirty ulcer often remains after the slough separates.

Etiology.—*Predisposing causes* are age, sex, temperament, and atmospheric conditions. The disease is most common in youth and in early adult life. Boys and young men appear to be attacked more often than the opposite sex. Tonsillitis is most prevalent during the spring season. An individual susceptibility is most distinct in lymphatic and strumous constitutions. It is aggravated by, or tends to recur especially in, the rheumatic diathesis. The proportion of cases in which tonsillitis precedes rheumatism is probably over 30 per cent. It is certain that one attack of acute tonsillitis predisposes to subsequent ones, par-

ticularly when the first attack has left some enlargement of the tonsils. Sudden and extreme climatic changes predispose to the disease.

The *existing causes* of acute tonsillitis are most commonly the following: (*a*) exposure to cold and dampness, or talking in a cold, moist atmosphere; (*b*) exposure and talking in an overheated atmosphere vitiated with smoke or other irritating vapors or gases; (*c*) bad drainage, sewer-gases; (*d*) specific infectious fevers, as scarlatina, measles, and erysipelas; (*e*) irritation from hard and sharp foreign bodies or chemical irritants; (*f*) the presence of microbes (streptococci, staphylococci, pneumococci; (*g*) epidemics of streptococcic infection have been traced to the milk-supply.

Clinical Symptoms.—Three principal varieties of acute tonsillitis occur clinically, the symptoms of which will be described separately.

(*a*) **Acute Catarrhal or Superficial Tonsillitis.**—This form is often associated with acute pharyngitis. The earliest *local symptoms* are pain and difficulty in swallowing, the former often becoming quite acute and radiating to the ear and lymphatics at the angle of the jaw, where tenderness on pressure may also be elicited. In speaking a nasal twang is often noticed. During the laborious act of swallowing the sensation of a lump in the throat, especially when the mouth is dry, is commonly complained of. Simple stomatitis may be associated, and rarely there is a slight cough with the painful expectoration of a sticky mucus which accumulates in the throat (Browne). There may be salivation, with fetor of the breath. *Inspection* shows the tonsil to be red and swollen. Though dry and glazed at first, the surfaces soon become covered with a thin exudate of muco-pus, which is easily detached by brushing, gargling, or "hawking" the throat. There is usually some accompanying redness, and also a *tumefaction* of the uvula and faucial pillars. The *constitutional symptoms* of simple erythematous tonsillitis at the outset are mildly febrile. The attacks usually come on rapidly, and last but a few days, subsidence taking place rapidly also. *Otitis media* may follow the extension of the tonsillar inflammation.

(*b*) **Acute Lacunar or Follicular Tonsillitis.**—In this form, which is quite common in children, not only the mucous membrane lining the crypts is inflamed, but that covering the surface of the tonsils also, giving rise to more or less associated *catarrhal tonsillitis*. The *local subjective symptoms* are, as in the preceding variety, pain, tenderness, and difficult deglutition. The tonsils are seen to be covered with small, slightly prominent, whitish-yellow spots or patches of a characteristic creamy exudate corresponding to the position of the crypts and numbering from two to eight or ten or more. These little masses or plugs may be pressed out of the follicles with a spatula. A predominance of pus-cocci and cells may rarely forerun the further formation of little follicular abscesses, and even of slight erosions and ulceration of the mouths of the lacunæ. Unlike simple catarrhal tonsillitis—at least in so far as simultaneous involvement is concerned (Cohen)—both tonsils are usually affected in this trouble, though one to a greater degree than the other. The whole tonsil is considerably swollen, and in severe cases the cervical lymph-glands also. The *constitutional symptoms* of follicular tonsillitis may be quite severe. The disease may be ushered in with a pronounced chill, headache, aching of the back and limbs, marked anorexia, and insomnia, along with a rapid rise in the temperature to

103° or 104° F. (39.4°–40° C.)—in children as high as 105° F. (40.5° C.). Slight albuminuria is an exceptional finding, but acetonuria is more common (25.6 per cent.—Reiche). The general depression may be so great as to simulate adynamia. Though sudden in its onset and rapid and often intensely acute in its progress, the disease seldom lasts more than five or eight days. Follicular abscesses complicate the case, while chronic swelling of the tonsils, desiccation, and bacterial degeneration of the lacunar masses may be sequelæ. Packard has reported five cases of endocarditis following acute angina. Pericarditis, pleuritis, nephritis, and skin-lesions, particularly erythema nodosum, may occur as complications. The exudate may become calcified, and may be expectorated as concretions or chalk-plugs.

(*c*) **Acute Parenchymatous Tonsillitis** (*Tonsillar Abscess* or *Quinsy*).—In this form of tonsillitis, which occurs most often during adolescence and early adult life, the symptoms reach the most pronounced and severe types. The stroma is inflamed and the tendency is toward suppuration.

Local Symptoms.—Complaint is first made of dryness of the throat, with painful and difficult deglutition. The pain is a prominent subjective sign, and may be referred to one or both ears according as one or both tonsils are inflamed. The secretion of a viscid mucus soon takes place, and as the tonsillar swelling increases, the husky voice of sore-throat and difficult articulation supervene; in cases of aggravated swelling dyspnea may often appear later. On examining the tonsils they are found to be greatly enlarged, deeply reddened, firm, and edematous. The surrounding soft parts, the faucial arches, pillars, and the uvula, manifest a deep congestion. In severe cases the tonsils may meet in the median line, pushing the uvula forward. Patches showing follicular tonsillitis are sometimes associated. The submaxillary glands may be engorged, and opening the mouth is often performed with difficulty; it is usually only partial, on account of the fixation of the jaw.

In a few days, perhaps, softening and fluctuation may be detected in the tonsils, and spontaneous rupture and discharge of the pus may occur, with almost instant relief to the patient. Resolution, however, sometimes takes place in the milder cases. The abscess may open in one or more places, and should rupture occur during sleep it may, as in one of my patients, cause suffocation by the entrance of pus into the larynx. The tonsil may regain its original size in a few days after the discharge of pus, and all the symptoms subside. The *constitutional phenomena* of parenchymatous tonsillitis are usually severe from the start, even in children, and more so than in the follicular form (Mackenzie). The temperature rises to 104° or 105° F. (40° or 40.5° C.), and the pulse-beats may reach 130 per minute. Acetonuria is often present in this disease. There may be delirium, and the symptoms generally increase until the abscess bursts or is opened, when all symptoms abate.

Course, Duration, and Terminations.—Though often severely acute in its course, quinsy seldom goes on to rupture in children, usually ending in resolution in from three to five days. If both tonsils are inflamed, only one suppurates as a rule, or but one at a time. The duration of an attack ending in tonsillar abscess is about eight or ten days in adults.

Complications and Sequelæ.—The tonsillar suppuration may invade the cellular tissue between the tonsil and the pterygoid muscles; a peritonsillar abscess may then result that may burrow as far as the clavicle.

Ulceration into the internal carotid or internal maxillary arteries with fatal hemorrhage may occur, though rarely. Edema of the larynx is also an infrequent complication. French writers, as Guble, Germain Sée, and others, have reported cases of paralysis of the soft palate and pharynx following inflammatory throat-diseases. On subsidence of the tonsillar inflammation the trouble becomes evident in the difficult swallowing and partial regurgitation of liquids and solids into the nasal passages, and in the nasal intonation of the voice. A frequent sequel is chronic enlargement of the tonsils.

(*d*) **Necrotic Tonsillitis.**—This affection is considered by Strümpell to be in some instances entirely distinct from diphtheria in its etiology, although he admits that quite frequently it is simply a mild form of the latter disease, and that often it is impossible to distinguish between the local appearances of the two conditions: these have been referred to under the heading of Morbid Anatomy. The *constitutional disturbances* are severe, especially in children, though they seldom last longer than a week, and are followed by a rapid convalescence. The cervical glands are not swollen to the same extent as in diphtheria. The occurrence later of palatal and pharyngeal *paralysis* in a supposed case of necrotic tonsillitis would point to its true diphtheritic origin.

(*e*) **Streptococcus Tonsillitis** ("*Septic Sore Throat*").—An epidemic form, which was first observed in Christiana in 1908 and later in England, has more recently been described by certain American authors, *e. g.*, D. J. Davis and E. C. Rosenow, and C. E. A. Winslow. The onset is sudden, with or without a chill. The throat presents a diffuse redness with much mucoid secretion. The crypts of the tonsils are always filled with an exudate, and a grayish membrane resembling diphtheria may spread over a large part of the tonsil. "The fever, the muscular pains, the prostration, and the constitutional symptoms were out of all proportion to what one would expect from the amount of local involvement."[1] The pulse is relatively slow and the leukocytes are only moderately increased. The acute symptoms may subside in a few days, but at the end of a week or ten days the patient suddenly manifests great enlargement of the cervical glands without suppuration, as a rule. Visceral complications occur most often in the group of cases having little or no glandular involvement but marked constitutional disturbances. Otitis media is frequent and bacteriologic examination shows an encapsulated hemolytic streptococcus of high virulence.

Diagnosis.—The appearance of the several forms of acute tonsillitis, associated with the clinical history of each case, should enable a ready diagnosis to be made in the majority of cases. A difficulty may, however, arise in discriminating follicular tonsillitis from diphtheria, and apparently transitional forms are not uncommon. The appended table gives the important points of differentiation between these diseases:

Follicular Tonsillitis.	Diphtheria.
A soft, pultaceous, yellowish-white deposit occurs in spots or patches situated over the mouth of the follicles, with areas of redness intervening.	A tough, ashy-gray, continuous, and uniform pseudo-membranous deposit covers the tonsils, pharynx, or soft palate.
The exudate is easily removed, leaving a smooth surface.	Very adherent, and can be torn off in strips only, leaving a bleeding surface.

[1] *Pract. Med. Series*, 1912, vol. vi., 110.

Follicular Tonsillitis.	Diphtheria.
The deposit is always limited to the tonsils (important).	The pillars of the fauces and uvula are involved as well.
If the creamy deposits unite to form a continuous layer, removal is either not followed by re-formation, or very late.	Removal of the membrane is followed by re-formation within twelve to twenty-four hours.
May have high temperature, lasting only a day or two, and falls after administration of sodium salicylate. Albuminuria extremely rare, if present at all.	Persistent elevation of the temperature, which is not materially influenced by salicylates; more or less albuminuria is common.
Cervical lymphatic glands seldom or slightly swollen.	Usually markedly swollen glands.
Complications rare and mild.	Complications frequent and grave (cardiac failure and paralyses).
Bacteriologic test shows no special organism; often, however, streptococci and staphylococci.	Bacteriologic examination shows presence of Klebs-Löffler bacillus.

Cases seen early, with severe constitutional symptoms and red and *swollen tonsils* having no deposit, may give rise to the question whether *simple angina* or *scarlet fever* is to follow. In such cases the latter disease may be excluded by a negative history of exposure to contagion, by the absence of a very high pulse-rate, and by the non-appearance of the scarlatinal eruption. *Necrotic tonsillitis* may be discriminated from the lacunar variety in the same manner as diphtheria—*i. e.*, by its local manifestations (*vide* Morbid Anatomy).

The **prognosis** is good as regards life, and favorable as regards complete recovery. The occurrence of either fatal hemorrhage or asphyxia in *quinsy* is extremely rare. In debilitated and strumous individuals relapses are prone to occur, and successive acute attacks of tonsillitis tend to cause permanent hypertrophy of the tonsils. In cases of *necrotic tonsillitis*, especially during the earlier periods, the prognosis should always be guarded.

Treatment.—Particularly in the lacunar and necrotic forms of tonsillitis the patient should be kept apart from others as much as possible, since both types appear to be contagious to a certain degree. Individual susceptibility to frequent attacks of sore throat may be lessened by systematic cold bathing of the neck. Constitutional and local rest is a first and constant requisite. Efforts at swallowing and talking should be reduced to a minimum. Bland nourishing liquids, as milk, broths, and the like, should constitute the only nutriment during the attack.

Medicinal Treatment.—Early in the case a free evacuation of the bowels should be obtained, and small doses of calomel (gr. $\frac{1}{8}$–$\frac{1}{6}$—0.008–0.010, repeated hourly until about gr. 1—0.0648—has been taken), followed by a Seidlitz powder or Rochelle salts in hot water, will be effective in most cases. In severe cases of quinsy relief from the pain is urgently called for, and either a Dover's powder or a hypodermic injection of morphin (gr. $\frac{1}{6}$–$\frac{1}{4}$—0.010–0.016) and atropin (gr. $\frac{1}{100}$—0.0006) will probably suffice for their relief. A high temperature must be combated by small doses of aconite, frequently repeated: this drug has been much used in the follicular tonsillitis of children. Quinin, in solution with dilute sulphuric acid, is also often given.

The administration of sodium salicylate or benzoate, of salol, or of the ammoniated tincture of guaiac in 1-dram (4.0) doses (Sajous), seems to lessen the duration and severity of tonsillitis, and even to cure some cases of the lacunar form within forty-eight hours and without local

applications. The tincture of the chlorid of iron in glycerin (4 or 5 drops to the dram—4.0—given every two hours) is regarded by Bosworth as almost specific at the commencement of an attack of acute follicular tonsillitis. During convalescence soft, light foods may be allowed; and bitter tonics and iron are to be administered if there are depression and anemia. The following is a favorite prescription:

℞. Strychninæ sulph., gr. ss (0.032);
Syr. acaciæ, ʒss (16.0);
Liq. ferri et ammon. acetat., q. s. ad ʒiij (96.0).—M.
Sig. ʒj (4.0) *t. i. d.*, in water, after meals.

Local Treatment.—If the case is seen early, the use of cold is of great value in giving local relief and in shortening the attack. Ice may be sucked and flannel wrung out of ice-water may be applied around the neck, or an ice-bag used. Lozenges of guaiac (gr. ij—0.129) or the ammoniated tincture in 1-dram (4.0) doses in milk, and used as a gargle, are indicated early, and, according to Sajous, seldom fail to control or arrest the inflammation. Equal parts of the tincture of the chlorid of iron, glycerin, and water, applied gently with a camel's-hair brush, have long been used locally on the surfaces of the tonsils, and with marked benefit. Alkaline and mild antiseptic solutions, used as gargles or sprays (preferably the latter), are generally useful. Thus, Dobell's solution, or Seiler's tablets dissolved in water, or borax and thymol, or carbolic acid, or potassium permanganate in weak solution, may be serviceable. Mild counter-irritation at the angle of the jaw by means of iodin or slightly irritating embrocations is helpful.

Early scarification of the tonsils as a depletory measure, and painting with cocain (10 per cent.), I have found useful to bring about resolution.

Astringent sprays containing alum or silver nitrate are often efficacious after a day or two. When the case is first seen and fully developed, the atomization of a warm solution of cocain (4 to 8 per cent.) or lime-water. The external application of the ice-collar is indicated. Should gargling be possible, nothing is better than hot water or milk. If, in parenchymatous tonsillitis, fluctuation be detected or suppuration be even suspected of commencing, the prompt use of the bistoury (the blade being guarded by wrapping with cotton or adhesive plaster), with the production of free bleeding or the discharge of pus, will give great relief. The patient's head, especially if it be a child, should be tilted forward during the operation, so as to allow most of the blood and pus to pass into the mouth. When incision of the tonsil fails to bring pus, it has been advised to puncture through the anterior pillar, where pus may be formed in the cellular tissue in front of or behind the tonsil. When the tonsillar enlargement threatens life through suffocation, excision of the tonsils, laryngotomy, or tracheotomy may have to be performed.

CHRONIC TONSILLITIS.

(*Hypertrophied Tonsils; Adenoid Vegetations.*)

Definition.—Enlargement of the tonsils (faucial and pharyngeal), due to chronic inflammation or hypertrophy, and usually associated with or causing a perverted local and systemic condition.

Pathology.—The faucial tonsils show a true chronic hypertrophy of the lymphoid and fibrous elements. If the latter predominate the organs will be smaller and more indurated. They may be rough on the surface from "distended lacunæ or ruptured follicles" (Berkley Robinson), the latter being in a state of chronic inflammatory thickening, and showing caseous degeneration of their contents. The growths in the vault of the pharynx are adenomatous papillomata; they are either sessile or pedunculated, and are fleshy in appearance and consistence and very vascular. They range in size from a grain of wheat to an almond-kernel (Allen), and project from the pharyngeal vault, lying in the depression posterior to and on a line with the fossa of the Eustachian tube (Rosenmüller's fossa). "Hypertrophy of the pharyngeal adenoid tissue may also be present without great enlargement of the tonsils proper" (Osler). A congestive type of nasal catarrh in adults often accompanies, or is the result of, neglected adenoid growths and hypertrophied tonsils. Chronic pharyngitis may be associated.

Etiology.—The *predisposing causes* of chronic hypertrophy of the tonsils are—(*a*) heredity, especially in the scrofulous and syphilitic diatheses; (*b*) age, most frequently between five and fifteen years; (*c*) sex, boys appear to be affected more frequently; (*d*) hygienic surroundings.

The *exciting causes* are usually previous attacks of acute tonsillitis, either simple or that which is symptomatic of diphtheria or scarlatina. According to Harrison Allen, adenoid growths from the normal lymphoid tissue of the vault of the pharynx (pharyngeal tonsils) may be congenital, and are "in some way associated with the canal which is found in early fetal life penetrating the brain-case and uniting the anterior part of the pituitary body to the lining membrane of the pharynx."

Symptoms.—*Local.*—With slight or moderate enlargement there may be few or no symptoms. There may be simply an increased secretion of mucus, and a susceptibility to fresh anginal attacks or to severe tonsillar manifestations in diphtheritic or scarlatinal attacks.

The first symptom to attract the attention is the direct effect of nasopharyngeal obstruction—*i. e. oral respiration*. This mouth-breathing is visibly labored and abnormally audible, and is especially marked at night, the child's respiration being noisy, snorting, and irregular. Sleep is disturbed by paroxysms of dyspnea, sometimes due, perhaps, to reflex spasm of the glottis. Nightmare follows as a result of imperfect aëration of the blood supplying the brain on account of the obstruction to perfect respiration. The act of swallowing is rendered difficult by the faucial obstruction, and is often painful, owing to the superadded acute tonsillar trouble that is so liable to occur in the hypertrophied glands. Indirect results of chronic tonsillar enlargement are a *laryngeal stridor* and a croupy *cough*. Sometimes *asthmatic attacks* coexist, and seem also to be due to the hypertrophy. An excessive secretion of mucus in the pharynx is a common symptom, and causes hawking in subjects past young childhood. The *hearing* is often impaired, and tinnitus aurium is complained of, being the result of pressure of the growths against the orifice of the Eustachian tube or of extension of inflammation from the nasopharynx. Absolute deafness may result, and the senses of taste and smell are likewise diminished or perverted. *Inspection* of the fauces will show the tonsils bulging as two lumps covered with thick mucus, or the

latter may ooze around the uvula from the pharynx. In mouth-breathers of long standing the superior dental arch is narrowed and the hard palate is highly arched. The breath is fetid, owing to the cheesy, inspissated exudate in the tonsillar crypts. In very old cases a tonsillar *calculus* may be felt, and is the result of calcification of the secretion.

The *facial expression* is characteristically stupid and apathetic; the disposition is dull, irritable, and stubborn; the lips are thick, and a vacant stare is in the eyes. *Speech* is slow, phonation nasal in quality, and articulation of the nasal consonants *n* and *m*, *l* and *o*, is changed or muffled. *Stammering* may be associated. The anterior nares may be dilated and present a pinched appearance above their openings.

The prolonged interference with respiration gives rise to a peculiar chest-conformation, simulating that of rickets (*chicken-breast*). The ribs are prominent anteriorly, and there is a marked forward angle at the manubrio-gladiolar junction, as well as a grooved depression at the ensiform cartilage. Depressions between the widely-separated ribs exist anteriorly also. Posteriorly, and at the base of the chest in particular, the intercostal spaces are practically absent on account of the closeness of the ribs. The upper part of the chest is very narrow and the shoulder-bones quite prominent. On percussion the hepatic area of dulness is diminished on the chest-wall, but increased downward and to the left. The first cardiac sound is weak. On inspiration there is a retraction of the intercostal spaces in the lower and lateral thoracic regions.

The resulting thoracic deformity may express itself principally as an excavation of the lower sternal area (*trüchter brust*). When chronic tonsillar enlargement leads to oft-recurring asthmatic attacks, the chest may become *barrel-shaped*, as in emphysema, at an early period of life.

The *general symptoms* of tonsillar hypertrophy are more marked when the growths exist in the pharyngeal vault alone. Developmental processes in children, such as dentition, and at puberty, particularly when the voice-changes are looked for, are often retarded or perverted. Anemia, headache, especially during study, cardiac palpitation, enuresis, and habit-chorea of the facial muscles, may be associated with general capriciousness, mental dulness, indisposition to intellectual exertion, drowsiness, and sullen irritability. The term *aprosexia* has been given to the loss of power to concentrate the mind for any length of time that is so characteristic of these cases.

Diagnosis.—Inspection of the fauces will reveal enlarged tonsils. The act of gagging, however, often causes the tonsils to rotate forward and inward, making them appear larger really than is the case. Adenoid growths of the pharyngeal vault may exist without tonsillar enlargement, and can be detected by posterior rhinoscopy or by the insertion of the finger into the naso-pharynx.

Differential Diagnosis.—It is important not to attribute the obstructive symptoms to *nasal hypertrophies* or *atresia* or to *malignant growths* in the naso-pharyngeal space. The latter are infrequent at the ages at which chronic tonsillar enlargement of the fauces and pharynx is most apt to occur—*i. e.*, early in life. Again, palpation of sarcomatous or carcinomatous growths gives marked differences in consistence, and there are usually spontaneous hemorrhages and local pain in attendance

upon these neoplasms. "*Thumb-suckers*" differ from mouth-breathers in that in the former the incisors are inclined forward and cause slight protrusion beneath the upper lip; the dental arch is flat. In mouth-breathers, however, the incisors are vertical or nearly so, or incline so as to overlap each other; the dental arch is high and curved (H. Allen). *Retropharyngeal abscess* may be confounded with tonsillar enlargement, especially in children. But in this disease the attacks of dyspnea, the dysphagia, and the local distress are more marked. Again, in the pharyngeal disease the swelling is in the median line, pushing the soft palate forward perhaps, and on palpation it may give a sense of elasticity or fluctuation to the finger. Slight fever may also be present.

Prognosis.—Tonsillar hypertrophy is not a severe disease as regards life. There is, however, an increased liability to contract colds, to recurrences of follicular tonsillitis, attacks of diphtheria, and severe scarlatinal angina. The prognosis in acute respiratory affections associated with chronic tonsillar enlargement is always more or less grave. Adenoid growths, even when neglected, tend to lessen in size after puberty. After removal the growths, as a rule, do not return.

Treatment.—The old-fashioned use of astringent applications is probably useless when there is any marked chronic enlargement of the tonsils, and active surgical treatment alone is to be recommended for the condition. The use of absorbents and caustics, either externally or by parenchymatous injection, is, I think, objectionable.

There are no more satisfactory means of doing radical good in cases of this kind than the galvano-cautery, scarification, and the removal of the tonsils with the tonsillotome, snare, or bistoury. In offensive follicular disease applications of chromic acid may give good results. Adenoid growths may be removed by means of the finger, curet, or forceps.

Constitutional treatment is often necessary in improving the nutrition of the patient. Good food, a change of air, systematic bathing, prudent habits, careful dress, and medicinal tonics and alteratives, as cod-liver oil, iodid of iron, and the hypophosphites, are usually indicated.

V. DISEASES OF THE PHARYNX.

PHARYNGITIS.

ACUTE PHARYNGITIS.

(*Pharyngitis Acuta Simplex.*)

Definition.—An acute catarrhal inflammation of the mucous membrane of the pharynx.

Pathology.—The mucous membrane is congested diffusely or in patches, and there may be an inflammatory exudate in, and a consequent swelling of, the submucosa and the contained glandular structures. The surface of the membrane is more or less coated with a viscid muco-pus.

Etiology.—*Predisposing causes* are—age, it being more frequent in adolescence and young adult life; a depraved constitution; digestive disorders, and a rheumatic, gouty, or scrofulous diathesis. The usual

exciting cause is exposure, particularly of certain portions of the body, as the neck and chest, to cold or to sudden changes of temperature and to irritating vapors. An acute naso-pharyngeal catarrh, by bathing the pharyngeal mucosa with its irritating secretions, may set up the trouble. "*Epidemic pharyngitis*" is probably a manifestation of influenza. Acute simple pharyngitis may be a complication of scarlatina, measles, and small-pox (*exanthematous pharyngitis*). Micrococci are present, the streptococci often predominating.

Symptoms.—*Locally*, the affection is ushered in with a feeling of dryness and soreness, especially on swallowing. With the production of the muco-purulent secretion a tickling sensation provokes *hawking* or a slight "*throat cough*" and efforts at exspuition. The catarrhal process may extend to the larynx and cause some hoarseness, or to the Eustachian tube, causing dulness of hearing. Movements of the neck are *painful* and *stiff*, particularly if there is, as is often the case, slight involvement of the lymph-glands. *Inspection* of the throat shows the pharynx, often the posterior pillars of the fauces and the soft palate, and even the anterior pillars and tonsillar surfaces, to be deeply reddened and tumefied; the coursing veins are enlarged, and particles of a yellowish-white secretion appear here and there. Sometimes the pharyngeal follicles become subject to acute inflammation, and appear as elevated, discrete, shiny spots (*herpetic pharyngitis*—Mackenzie).

At the *onset* of this affection there may be chilliness, followed by slight fever, headache, an accelerated pulse, a dry skin, and anorexia. The *pharyngeal symptoms* seldom last more than from three to five days, when resolution takes place, some tenderness of the pharynx, however, remaining for a time.

Diagnosis.—On examination of the throat there should neither be any difficulty in diagnosing the affection nor any likelihood of confounding the affection with simple tonsillitis.

The **prognosis** is always favorable. In weakly patients, however, there is a liability to subsequent attacks.

Treatment.—In the early stages sucking of small pieces of ice does much to allay the congestion and irritability. A spray of cocain or menthol in albolene (2 per cent.) may also be used, followed by a 4 per cent. solution of antipyrin. Eucain may be substituted for cocain (2 per cent. solution), and is preferred by Gibbs and others. Dobell's solution is always to be recommended for its alkaline, sedative, and antiseptic action. Swabbing the pharynx with a silver-nitrate solution (gr. xl to the ounce—2.59 to 32.0) is, according to Sajous, of great benefit.

In well-established cases relief is often obtainable by medicated steam inhalation, as with the compound tincture of benzoin. In rheumatic cases lozenges of guaiac (gr. iij—0.194) are useful. The sipping of hot milk in which sodium bicarbonate has been dissolved is grateful.

The *general treatment* embraces measures directed at the fever and the diathetic condition. A hot foot-bath and a calomel purge, with belladonna, acetanilid, or aconite for the fever and pain, and sodium salicylate (gr. lx–lxxx—4.0–5.1—in the twenty-four hours), may be required. The diet, of course, should either be liquid or semi-solid.

Persons susceptible to repeated attacks must exercise caution in regard to exposure to severe cold and weather-changes, irritating vapors,

and the like. Daily cold sponge-baths may be used to harden the skin. Tonic, nutrient treatment is also frequently called for.

MEMBRANOUS PHARYNGITIS.

(*Pharyngitis Crouposa.*)

Definition.—An acute superficial inflammation of the pharyngeal mucosa, characterized by the formation of a whitish false membrane, due usually to the streptococcus.

Etiology.—The principal causes of this form of pharyngitis are exposure of persons in debilitated health to cold or an impure or a septic atmosphere, particularly during epidemics of such diseases as scarlatina.

Symptoms.—The local and general symptoms are those of ordinary sore throat, though of a more severe type.

Diagnosis.—The pseudo-membrane is thin, of a yellowish-white color, and appears in small patches over the pharynx. It is easily detached, and this feature, together with the presence of small vesicles or ulcers and the absence of grave constitutional disturbances serve to differentiate this affection from diphtheritic pharyngitis.

The **prognosis** is favorable.

Treatment.—Local applications of solutions of hydrogen peroxid or potassium permanganate (gr. x to the ounce—0.648 to 32.0) are very satisfactory. For the painful dysphagia the sedative and soothing remedies suggested for simple acute pharyngitis may be used. Internally, sodium benzoate (gr. v–xv—0.324–0.972) in glycerin, elixir of calisaya, and salol have each been recommended. Tonic treatment is nearly always needed.

CHRONIC PHARYNGITIS.

Definition.—A chronic inflammation of the mucous membrane of the pharynx. It may consist of either a hypertrophic or an atrophic involvement of the follicles, or both processes may coexist.

Varieties.—(*a*) Chronic naso-pharyngeal catarrh; (*b*) chronic hypertrophic pharyngitis or naso-pharyngitis (*pharyngitis sicca*); (*c*) follicular or granular pharyngitis. The last named is probably the result of, and nearly always is associated with, chronic simple (or hypertrophic) pharyngeal (or naso-pharyngeal) catarrh.

Pathology.—The mucous membrane in simple chronic pharyngitis is either reddened, thickened, and viscid (hypertrophic form), or pale, thin, and dry (atrophic form); in both instances dilated and tortuous veins are prominently shown. In the follicular variety the pharyngeal mucous glands are swollen into little red, glistening nodules studding the congested membrane. The enlarged follicles are due to a hyperplasia of lymphoid cells and an accumulation of retained dried-up secretions.

Etiology.—A protracted impairment of the general health, especially in those who over-exert mentally and are of sedentary habits, is a common *predisposing cause* of chronic pharyngitis. Repeated acute attacks may precede the affection. It is most common in adolescent and middle life.

The *exciting causes* are frequent and prolonged over-use and strain

of the voice in clergymen, singers, teachers, army-officers, and street-venders; irritation from tobacco-smoke, chemical vapors, and continued exposure to cold air. Among prevailing causes may be mentioned post-nasal adenoids, deviations of the septum, and neoplasms. It may arise from gastric disorders.

Symptoms.—In all varieties of chronic pharyngitis the *local discomfort* is often very slight, and more annoying than painful, except when an exacerbation takes place. There is a sensation of *dryness* and *tickling* or *burning* in the throat and the desire to clear the throat of sticky mucus by *hawking* or a *short cough*. These symptoms are usually worse on rising in the morning, especially if some unfavorable influence has been exerted during the night previous, the throat being dry and a viscid secretion having collected. Swallowing is seldom interfered with.

If the larynx is somewhat affected by extension of the pharyngeal inflammation, *hoarseness* and a *dry, hacking cough* are produced. After using the voice there is a sense of fatigue, with huskiness and irritability.

The *local appearances* of chronic pharyngitis vary according to the form of the affection present in the case. In chronic catarrh of the pharynx a considerable collection of muco-pus is seen adhering to the mucosa and extending downward from the posterior nares. The senses of hearing and taste may be impaired. The uvula is frequently elongated, and its tip may rest on the base of the tongue. A nasal intonation of the voice is sometimes provoked. The posterior nares as seen by the rhinal mirror are often stopped up by foul secretions or by hypertrophy of the nasal mucous membrane. Headache and attacks of vertigo may occur.

Chronic hypertrophic pharyngitis and follicular pharyngitis ("*clergyman's sore throat*") are commonly associated. The thickened, reddened, pimply, vein-coursed appearance of the mucosa is characteristic. The follicles may be seen sometimes as polypoid elevations, and the pharyngeal tonsil may be found by the finger to be enlarged (Kölliker).

In the dry, *atrophic pharyngitis* that occurs more often in later life, and as a sequel of the simple chronic or follicular variety, a pale, smooth, relaxed, lustrous, and often quite painful membrane is observed.

The *general symptoms* are usually those of a weak, debilitated, nervous constitution, though in mild cases the general health may be unimpaired. In atrophic pharyngitis considerable cachexia may be present.

Diagnosis.—Care should be exercised in discriminating the variety of chronic pharyngitis present in any given case, so that the treatment may be planned accordingly. Careful and repeated inspection of the throat renders the diagnosis easy unless ulceration has taken place: in such cases a *tuberculous* or *syphilitic* sore throat must be eliminated by the superficial character of the ulcers, by their ready response to proper treatment, by the history of the case as to specificity, and by the absence of marked pain or symptoms pointing to tuberculosis. When due to gastric disturbance the lower throat will be deeply congested and the tongue will be irritable, with red papillæ standing over its base (Price-Brown).

Prognosis.—This should be guarded as to cure, on account of the stubborn resistance to treatment and the difficulty in removing unfavorable influences. Acute exacerbations are liable to recur unless rigid caution is practised at all times in avoiding the cause of the trouble.

Treatment.—The local use of astringent and alkaline antiseptic sprays or of the nasal douche is usually recommended, but has only a palliative effect. Silver-nitrate cauterization may be tried. The only effectual means, however, of curing the follicular or hypertrophic variety is that used by most throat-specialists—namely, the wire galvano- or actual cautery. Applications of silver nitrate (gr. x to the ounce—0.648 to 32.0) and the internal use of the oleoresin of cubebs have been recommended for the atrophic pharyngitis. Insufflation of powdered tannin or alum is also of service.

Systemic disturbances need attention according as they present themselves. Mineral baths are sometimes of great benefit, and tonics are usually indicated. All irritating causal factors must be removed or avoided before any favorable results can be hoped for from local applications. Tobacco-smokers and topers must deny themselves their habitual luxuries.

ACUTE INFECTIOUS PHLEGMON OF THE THROAT.

Definition.—An inflammation of the pharyngeal mucosa that passes rapidly into a suppurative process. It is exceedingly rare.

Its **etiology** is not definitely known. I have met with no cases except in my hospital wards, though they doubtless occur in general medical practice. The clinical features have been described by Senator.

The **symptoms** are *sudden in their onset* and quite intense. They are severe soreness of the throat, dysphagia, and hoarseness, as a rule; in advanced cases there has been difficult respiration. *Inspection* shows the pharynx to be deeply injected and the seat of marked inflammatory edema, the neck appearing greatly swollen as well. The general disturbance is correspondingly severe.

The **treatment** is wholly symptomatic.

RETROPHARYNGEAL ABSCESS.

Definition and Pathology.—A suppurative inflammation (rare) of the glands or connective tissue anterior to the cervical spinal column.

Etiology.—The disease is relatively most common before two years of age. It is usually a primary affection, occurring without assignable cause, but a certain proportion of instances are doubtless caused by caries of the cervical vertebræ. It may rarely be secondary to any of the specific fevers. Traumatism causes occasional instances.

The **symptoms** are *pain* in swallowing, *impeded respiration*, soon becoming stertorous in character, the dyspnea meanwhile constantly increasing. There may be *couyh*, and the voice may present abnormal characteristics. The signs of *stenosis* finally declare themselves with considerable violence, and an examination of the pharynx usually serves to make the diagnosis positive; the projecting tumor is visible, and the palpating finger readily detects fluctuation. In children the *general features* (slight fever, anorexia, languor) overshadow for days the *local*, while in adults the condition develops acutely with severe faucial symptoms.

The **course** of the disease may be acute, lasting one or two weeks; more frequently, however, it is subacute (rarely chronic), as, for example, when it is due to caries of the vertebræ.

The **prognosis** is favorable in all cases that are early diagnosticated. If unrecognized until the later stages have been run, suffocation may ensue, or rupture into the larynx may cause death by asphyxia.

Treatment.—As soon as fluctuation is detected the abscess should be freely opened, and preferably, as a rule, through the mouth by means of a guarded bistoury. The throat, after the abscess is thoroughly evacuated, should be washed out with some mild antiseptic solution (salicylic acid 2 per cent. or boracic acid 2 per cent.). When pointing occurs at the side of the neck, as sometimes happens, the incision should be made through the skin in that locality. Constitutional indications are to be fulfilled in accordance with general principles, and the strength of the patient is to be maintained by a highly nutritious dietary.

VI. DISEASES OF THE ESOPHAGUS.

ESOPHAGITIS.

ACUTE ESOPHAGITIS.

Definition.—An acute inflammation affecting either the mucous membrane or submucous tissues of the esophagus, or both.

Pathology.—The ordinary morbid changes of an acute esophagitis are those of a simple catarrhal inflammation of the mucosa. It is rather characteristic of the condition that there is no increased secretion, a sponginess and rapid desquamation of the epithelium taking place instead, and causing a granular appearance of the membrane. Occasionally the mucous glands are swollen, and may break down, with the formation of small follicular ulcers. Catarrhal erosions may also be seen here and there. A croupous or diphtheritic exudate is seldom found in the lower portion of the esophagus, and small-pox pustules are rarely, if ever, seen. A diffuse or circumscribed purulent inflammation of the submucosa may dissect up the mucous membrane so as to considerably diminish the esophageal caliber; pus is usually discharged into the tube. In severe cases of poisoning (*corrosive esophagitis*) sloughing may extend into the muscular layer, and may produce a foul, dark, hemorrhagic mass. A fibrinous cast of the gullet has been vomited up by an hysteric woman (Birch-Hirschfeld).

Etiology.—The causes of acute esophagitis, other than traumatic, are rare. Under the latter are included the *mechanical*, *thermal*, and *chemical* irritants, such as the presence of foreign bodies and the swallowing of hot liquids, corrosive poisons, "concentrated lye," mineral acids, and arsenic. The condition may also be the result of the following: (*a*) an extension of catarrhal inflammation of the pharynx; (*b*) specific infectious fevers, as typhoid, typhus, and pneumonitis; (*c*) diphtheria (*pseudo-membranous esophagitis*) by the extension of pharyngeal diphtheria; (*d*) small-pox, giving rise to a pustular inflammation of the gullet; (*e*) local disease, as carcinoma of the esophagus, glandular or vertebral abscess, or laryngeal perichondritis (Strümpell).

Symptoms.—*Pain* during deglutition may be referred to the region of the esophagus, and a steady, dull pain may exist beneath the sternum. *Dysphagia* and *regurgitation of food* may be caused by spasm in severe cases. Mucus, blood, and pus may be discharged later. The absence

or mildness of pain is not a true indication of the gravity and extent of esophageal inflammation.

Sequelæ.—Simple catarrhal or follicular ulcers may appear, and the necrotic form of the disease may be followed by suppurating ulcers, which, if healing takes place, may cause cicatricial stenosis.

Diagnosis.—This may be based upon the localization of pain, especially during deglutition; upon the pain occasioned by the passage of the esophageal sound; and upon the mucus, blood, or pus adherent to its bulb on withdrawal, provided carcinoma at the cardiac orifice of the stomach can be excluded. The expulsion of a pseudo-membrane (diphtheritic) from the gullet should be differentiated from esophagomycosis (thrush), especially in children. The diagnosis of the particular form of esophagitis will depend upon the facts elicited relating to the etiology.

The **prognosis** is good in mild cases, and should be guarded in those associated with grave disease. Death may occur in either the purulent or necrotic form.

Treatment.—This is entirely symptomatic, and in severe cases is of little value. A soft, bland diet, preferably of milk, may be borne in ordinary instances; if not, rectal alimentation should be resorted to. For the mild cases swallowing of bits of ice, and later of warm demulcent drinks, should be recommended. In cases of marked pain and esophageal spasm relief may be afforded by a hypodermic injection of morphin and atropin.

CHRONIC ESOPHAGITIS.

Chronic catarrh of the gullet may result from continued irritation by the causes of the acute form, and also from passive congestion due to hepatic cirrhosis, chronic cardiac or renal disease. The last-named conditions may also cause varicose esophageal veins, and fatal hemorrhage may result therefrom. Chronic alcoholism is a common cause. The increased mucous secretion may cause eructations and nausea.

Postmortem evidence of esophagitis, either acute or chronic, is found with extreme rarity.

ULCER OF THE ESOPHAGUS.

This is a consequence of a simple or follicular catarrh of the gullet or of gangrene. "Catarrhal erosions" and follicular ulcers may occur, and also necrotic ulcers, in bedridden persons opposite the cricoid cartilage. The extensive purulent ulceration following the separation of necrotic sloughs may heal and cause stenosis of the tube, or it may rupture into the trachea, the posterior mediastinum, or the aorta. Pressure ulcers (*e. g.*, from aneurysm) occur. Ulceration may also be met in uremia. Ulcers simulating those occurring in the stomach (*ulceres ex digestione*) may sometimes be found at the lower end of the esophagus. There may be localized points of pain on the passage of the esophageal bougie, with some pus and blood on the bulb after its withdrawal. Rest from swallowing should be secured as far as possible. The sipping of hot milk may be soothing, and the slow swallowing of mild boric acid and sodium bicarbonate solutions may be tried with benefit.

CARCINOMA OF THE ESOPHAGUS.

THIS is the most frequent affection of the tube, and, as it is the commonest cause of stenosis, it is important from a diagnostic standpoint.

Pathology.—Carcinoma of the esophagus is primary and of an epitheliomatous nature, the mucous membrane here being composed of pavement-cells. The new growth affects the mucosa first, and then, increasing in size and causing ulceration, it involves the entire circumference of the tube. This may either be hard and fibrous, or soft and jelly-like. The esophageal lumen is markedly diminished, though disintegrating ulceration or "flat" carcinoma may encroach upon the caliber but little. There may be a diffuse dilatation of the esophagus above the growth, as well as an hypertrophy of the circular muscular fibers. The cancerous tumor is found most commonly in the lower third of the esophagus (generally at the bifurcation of the trachea). A small percentage of the cases are surgically accessible, being situated in the neck.

Etiology.—The *predisposing causes* of esophageal carcinoma are age and sex, males past forty years of age being the usual subjects of this neoplasm. The *exciting causes* are of uncertain origin. It has been alleged that various forms of protracted irritation of the mucous membrane may cause the development of carcinoma; and especially has this point been maintained in connection with the frequent occurrence of carcinoma of the gullet in topers. It is also believed by some that as gastric carcinoma may develop from the scars of old ulcers, a like condition in the esophagus may act as a nucleus for a carcinomatous growth.

Symptoms.—*Dysphagia* is the earliest symptom of esophageal carcinoma with beginning stenosis of the tube. This gradually and steadily increases, so that liquids alone can be swallowed, and later regurgitation even of small amounts (not above three ounces) of liquid foods takes place. There may be considerable *pain*. I saw an instance with the late Dr. W. Frank Haehnlen, in which mucus was almost constantly regurgitated, and bronchiectasis developed near the close.

The *ejecta* may contain cancerous fragments, blood and mucus. The dysphagic symptoms may subside spontaneously, owing to the disintegration and ulceration of the growth, or the dysphagia may be so slight as to be masked by the prominent symptoms of hepatic or pulmonary carcinoma and gangrene secondary to a very flat esophageal carcinoma. Or, without secondary manifestations of such a growth, the esophageal symptoms may rarely be latent. The cervical glands may be enlarged.

The most important *general symptom* of esophageal carcinoma, as of this malignant growth elsewhere, is the progressive emaciation, which increases with the stenosis and obstruction to the entrance of nourishment into the stomach. Though seemingly anemic, the patient's blood may contain an excessive number of corpuscles in a given bulk. This is due to inspissation from failure to absorb water and food into the body.

Course, Duration, and Termination.—The disease is chronic, becoming progressively worse, and is often beset with grave complications (*vide infra*). It seldom lasts longer than one and a half years, and the duration of medullary carcinoma of the gullet is usually much shorter. A fatal ending is inevitable, by inanition and exhaustion, or as the result of metastasis and secondary complications.

Complications.—These follow extension of the cancerous growth to neighboring parts. Thus, involvement of the larynx, trachea, and bronchi has been noted. The cancerous ulcer may also perforate the pleura, the pericardium, or the aorta or its branches, and cause fatal hemorrhage. The vertebræ have been eroded, and compression of the cord, with resulting paraplegia, may take place.

Paralysis of the vocal cords may be the effect of pressure by the growth upon the recurrent laryngeal nerve; most frequently pulmonary gangrene is due to perforation of the lung or to the inspiration of cancerous and decomposing particles that have been regurgitated.

Diagnosis.—All other causes of dysphagia must be excluded. *Enlarged tonsils*, *pharyngeal tumors*, pressure from without by cervical intrathoracic tumors, as *aneurysm*, or by displacement of the sternal end of the clavicle, and the presence of *foreign bodies* or *cicatricial strictures* of the gullet—all figure in the production of difficult deglutition. The history of the case, the age of the patient, the progressive emaciation (cancerous cachexia), and the obstinately increasing dysphagia will enable us to exclude the other affections named. In using the esophageal bougie for diagnostic purposes great care should be exercised, as an aneurysm may thus be ruptured or a deeply ulcerated carcinoma perforated. The withdrawal of cancerous tissue upon the bulb will decide the case. The esophagoscope may be useful in certain cases. G. E. Pfahler[1] has shown that by the skilful use of diaphragms, the elimination of secondary rays and of motion by great speed, the disease can be diagnosticated by means of the Röntgen rays. The exceptional occurrence of latent cases must be remembered. *Sarcoma* cannot be distinguished from carcinoma by the clinical symptoms. By means of the esophagoscope, however, a small piece of the tumor may be removed for examination.

The **prognosis** is hopeless, and the supervention of grave complications renders the chances of an early demise very probable.

Treatment.—This is essentially symptomatic and sustentative. If feeding by the mouth is difficult on account of the extreme stenosis, although permitting the passage of an esophageal tube, the latter may be used for the passage of liquid nourishment. Rectal feeding may later become imperative. The local application of radium has been recommended. The mechanical treatment of the cancerous stricture by the passage of the graduated esophageal bougie is seldom of any avail. Soft, disintegrating, and ulcerating carcinoma should thus be treated, though with the absence of any force whatsoever. The performance of gastrostomy may prolong life in some cases in which there is formidable difficulty in passing a tube into the stomach.

RUPTURE OF THE ESOPHAGUS.

THE first recorded case of this rare condition occurred under the observation of Boerhaave in 1724 in the person of the Baron Wassemar.

Pathology.—Softening, together with a great friability, of the esophageal walls may be found, this probably being the effect produced by the solvent action of the gastric juice upon the mucous membrane at

[1] *Archives of Diagnosis*, January, 1909.

a time when the local circulation is disturbed and the vitality of the tissues thus lessened.

The *postmortem* evidence of this accident consists of a longitudinal (as a rule) tear about 5 cm. (2 inches) in length, situated in the lower half of the esophagus. Food and air may be found to have escaped into the left pleural cavity, and unless death occur at an early date signs of purulent inflammation will probably be noticed. *Postmortem* digestion of the esophagus is more common (Osler). The perforation is often large, and is located in the posterior wall of the tube.

Etiology.—Softening of the walls of the gullet (*esophago-malacia*) is suggested by Zenker as a condition that always precedes spontaneous rupture, so called. The exciting cause is believed to be violent and persistent vomiting after a particularly heavy meal or during acute alcoholism.

Symptoms.—These come on *suddenly* or soon after a full meal, and commence with *nausea* and very *severe vomiting*, accompanied by *great pain* and rapid and extreme *collapse* of the whole body, due to the shock. A cutaneous emphysema of the neck and chest is manifested soon after the rupture.

The **diagnosis,** if made at all, must rest upon the clinical history. Death usually takes place in a few hours or days at the most.

Treatment.—Pain, if excruciating, should be dulled by the hypodermic administration of morphin.

NEUROSES OF THE ESOPHAGUS.

MUSCULAR SPASM.

(*Esophagismus.*)

Definition.—A spasmodic contraction of the muscular layer of the upper or lower portions of the esophagus.

Etiology.—It is almost always a secondary affection, met with not infrequently in hysteria, hydrophobia, and rarely in chorea and epilepsy. In this case the esophageal bougie could be passed only with a great deal of difficulty during the spasm. It may be observed in aged males, and especially in those suffering from hypochondriasis. It may be due to reflex causes, originating, for example, in the uterus; thus, in some cases, it occurs only during the pregnant state. Spasm may complicate all of the lesions of the esophagus, even organic stricture.

Symptoms.—The chief subjective characteristic is *dysphagia*. Although liquids can be swallowed, solids, as a rule, cannot. Post-sternal *pain* is sometimes noticed, and choking signs are quite common. In the hysteric variety emotional disturbances are found among the prodromata. The general nutrition remains good.

Diagnosis.—The etiologic factors must be carefully weighed in connection with the symptoms and the valuable testimony gained by the use of the sound. The bougie on reaching the constriction is rather tightly gripped, though gentle pressure soon causes it to relax. After the subjective symptoms and spasm are over the sound passes without the slightest difficulty. The siphoned and vomited masses contain neither blood nor pus. An examination with the esophagoscope allows the diag-

nosis to be easily made. In some cases the dysphagia may be due to pharyngeal paresis. The *elderly hypochondriac* is, as before stated, liable to develop a similar condition, which must not be confounded with true cancerous stricture. The **prognosis** is good. Guisez speaks of severe spasms with lethal tendency.

The **treatment** is directed to the disease on which the condition is found to depend, and this must receive careful attention. The sound should be used as previously indicated under the discussion of Esophageal Stricture. Its passage has often been followed by speedy and permanent cures. A special electrode with which to apply electricity to overcome the spasm of the cardia has been employed.

PARALYSIS OF THE ESOPHAGUS.[1]

In extensive bulbar paralysis, when adjacent parts are involved, we may infer the existence of esophageal implication, though there be no objective evidence to adduce in confirmation. Doubtless the esophagus rarely shares in post-diphtheritic paralysis also. Dysphagia is the leading symptom. An invaluable peculiarity belonging to diphtheritic paralysis is the fact that solids are more readily swallowed than liquids.

DILATATION OF THE ESOPHAGUS.

Pathology and Etiology.—Diffuse dilatation of the esophagus is usually secondary to organic stricture at or near the cardiac orifice. In accordance with the common law of compensation, the first effect of the stenosis is to engender hypertrophy of the muscular layer above it with a view of overcoming the resistance caused by the obstruction. The wall of the esophagus becomes thickened, and the tube is generally somewhat narrowed, above the seat of the stenosis; but finally, as a result of degenerative changes, the muscular coat weakens, the esophagus dilates, and food accumulates above the stricture—a condition that, once begun, progresses. This condition also occurs without anatomic stenosis, and Plummer records 91 cases, and only 5 of the patients were of a neurotic type.

Congenital dilatation, in which the whole extent of the tube participates, has also been met with, though such a condition is rare indeed. It sometimes results from fatty degeneration of the muscular wall, and a predisposition to the complaint may be acquired as the result of injury or prior inflammation.

Symptoms.—The essential symptom is chronic *dysphagia*. When dilatation follows stenosis the patient often locates the point at which the food lodges in the esophagus. Most of the ingesta are regurgitated several hours after eating, and this process is often attended by more or less severe strangling. The *esophageal sound* comes upon the stricture, and is either gripped firmly or totally resisted; in the latter event the bulb can be moved about above this point with abnormal freedom. In the rare cases of spindle-shaped dilatation without stenosis the sound usually

[1] For remarks on the treatment of this complaint the reader is referred to the section on Nervous Diseases.

detects no obstacle on its way into the stomach. A *sac* is occasionally formed, however, as the result of localized bulging of the paralyzed wall, in which food may collect or the exploring sound may catch, thus leading to erroneous inferences. Dysphagia is present, though it presents peculiarities, in that the food may either pass down very slowly until it reaches the stomach, or it may lodge in the shallow pouch, as above described. In the latter event the food may be gulped up from time to time. If the sound can be easily introduced into the stomach, we may safely eliminate stricture as the cause of the dilatation.

The **prognosis** is good as long as sufficient food can be gotten into the stomach for the support of life.

Treatment.—The chief object in the treatment is to keep the patient nourished. If sufficient food cannot be swallowed, a Symond's tube should be inserted and nourishment given through it; and when this mode of feeding is no longer feasible, the physician has to choose between gastrostomy and rectal feeding. There can be no doubt that by means of nutrient enemata nutrition may be fairly well maintained, but not indefinitely, as these cases would seem to demand. In the hands of a competent surgeon, on the other hand, gastrostomy is often fruitful of brilliant results. Galvanism has been recommended on high authority. Local lesions, when present, must be dealt with in accordance with the rules governing the treatment of the several causal conditions. The sac may be washed out daily with an antiseptic solution (*e. g.*, boric acid 3 per cent.).

ESOPHAGEAL DIVERTICULUM.

(*Pharyngocele.*)

Definition.—A circumscribed sac in the wall of the esophagus.

Pathology and Etiology.—Two varieties are met with, which Zenker has termed *pressure* and *traction* diverticula; the latter are rare. Diverticula that occur at or near the inferior constrictor, and more particularly the larger ones, are *congenital* in origin. When *acquired* they are the result of a localized lesion in the muscular coat, through which the mucous membrane bulges like a hernia. This is owing to repeated slight pressure occasioned by the passage of food. When once such a process is started, various factors tend to continually enlarge the pouch. Chief among these are the morsels of food that find lodgement and naturally tend to augment the size of the diverticulum by dragging it downward. The sac may finally attain a diameter of not less than 4 inches (10 cm.). Its situation is nearly always on the posterior wall at the pharyngo-esophageal junction, and its form is usually saccular or pear-shaped. Most instances have been met with in males after middle life. The cause of the weakened area at which the diverticulum occurs is to be found sometimes in injury, but more frequently in an antecedent inflammation. Histologic changes are observed only in the mucous and submucous layers, these anatomic elements together forming the pouch.

Traction diverticula are produced by the fringe of tissues that often

becomes adherent to the upper aspect of the esophagus, and from their mode of occurrence they will obviously be more or less funnel-shaped. Their dimensions are small. They are more common in children than in adults, for the reason that in the former, more frequently than in the latter, do the bronchial glands suppurate, with subsequent cicatrization. This circumstance affords an explanation of the fact that traction diverticula are usually seated on the anterior wall of the esophagus, near the bifurcation of the trachea.

Clinical History.—*Traction* diverticula do not, as a rule, give rise to clinical symptoms. Exceptionally, however, as the result of the mechanical irritation caused by bits of food that are retained in these funnels, ulceration may occur and be followed by perforation of their apices. In this manner the main bronchi are perforated (causing pneumonia and pulmonary gangrene), also the pleura (causing empyema), and, more rarely, the pericardium (causing suppurative pericarditis).

Pressure diverticula when small cannot be recognized, owing to the absence of signs and symptoms. When they attain considerable size, however, they are often attended with severe symptoms. The earliest clinical manifestation is difficulty in swallowing; some of the food enters the sac, and, if allowed to remain, undergoes putrefactive decomposition, causing *fetor of the breath*. From time to time, and especially on attempting to swallow, the partly or wholly filled condition of the pouch excites *nausea* and *vomiting*, associated with prolonged *strangling;* this results in the ejection of a portion of the accumulated contents. These contain no hydrochloric acid. After such an attack the patient is unable, temporarily, to swallow food, and in consequence of the limited amount of food taken signs of inanition soon appear; this may finally become extreme, and is sometimes the immediate cause of death. The appearance of a *pear-shaped swelling* in the side of the neck has been observed. As the tumor enlarges it displaces the larynx and presses upon the enlarged vessels—more rarely upon the superior laryngeal nerve—giving rise to dyspnea and distressing fits of coughing.

Diagnosis.—A point in the differentiation of this affection is the enlargement of the sac after meals (not all the food passing into the stomach), and its disappearance after being emptied. Another discriminating sign is the effect of compression by the hand in causing the contents ("air and sodden food") to flow back into the mouth. In those instances in which the tumor is absent we may demonstrate its existence by the use of the esophageal sound. If the sound passes into the sac, the descent will soon be arrested. If, however, the instrument fails to enter the mouth of the pouch, it readily glides into the stomach. An elbowed sound, bent at an obtuse angle near the tip, is useful in such cases. It may be inserted in different directions, so as to avoid entrance into the sac. Schwalbe and Rosenfeld have been able to recognize the condition by the aid of radiography. Some writers advocate the methylene-blue test of Bökelmann. The esophagoscope should be used last in a routine examination, there being danger of a rupture of a possible aneurysm (Mayer).

Prognosis.—The outlook is unfavorable in the absence of operative treatment, though modern surgery gives promise of curing a certain proportion of cases. Wheeler has operated successfully in one instance at

least. The physician may prolong life by directing attention to the nutrition of the patient, but he cannot hope to promote a cure.

Treatment.—If the patient cannot swallow an adequate amount of nourishment, he may be successfully fed through a tube, which he himself should be allowed to pass. When sufficient food cannot be introduced by this method, rectal feeding should be instituted. If excision of the diverticulum be deemed impracticable by the surgeon, then the establishment of a gastric fistula is worthy of extended trial in cases in which the above-mentioned modes of feeding have failed. It has been advised to wash the sac daily with sterile water or some mild disinfectant to prevent decomposition. Stetten collected statistics of 60 radically operated cases, which gave a mortality of only 16.6 per cent.

STRICTURE OF THE ESOPHAGUS.

Etiology.—A stricture of the esophagus may be due either to (*a*) Congenital narrowing (exceedingly rare); (*b*) Squamous epithelioma, usually producing an annular constriction; (*c*) Rarely to polypi protruding from the mucosa, which almost occlude the lumen of the tube; (*d*) Rarely to specific inflammation, as syphilis and tuberculosis; (*e*) Simple stricture generally results from the ingestion of corrosive fluids, which cause extensive sloughing of the mucosa, followed by cicatricial contraction; (*f*) Rarely as a sequel of typhoid and peptic ulceration.

Clinical History.—The symptoms vary with the special cause and with the degree of stenosis. The first and most prominent indication of narrowing of the gullet is a very slowly increasing *dysphagia*. The patient for a long time complains of a *sense of pressure* at a certain substernal point on swallowing solid food, or, more rarely, an apparently healthy person will suddenly experience painful pressure in attempting to swallow a larger quantity of food than usual. By and by even fluids cause dysphagia, and the patient observes that the time required for the food to reach the stomach is lengthened. The impediment to the act of swallowing is not due alone to mechanical stenosis, but partly to the weakness of the muscular coat, sometimes owing to its partial destruction, and in exceptional cases partly to spasmodic contraction. When due to carcinoma, difficult deglutition is, as a rule, the only symptom complained of. When occasioned by corrosive fluids or traumatism, *pain* is prominent from the onset.

Above the seat of stricture the esophagus is often *dilated* and contains accumulations of the ingesta. The latter, together with considerable mucus, are regurgitated three or four hours after meals, and we may be certain that the materials thus ejected do not come from the stomach if they are alkaline in reaction. The leading clinical features are the gradually *increasing debility* and *emaciation*, finally reaching an extreme degree.

Diagnosis.—However characteristic the symptoms may be, the bougie should invariably be passed before pronouncing a positive diagnosis. By this means we ascertain the degree and the seat of the stricture. To begin with, a medium-sized gum-elastic bougie (No. 16 Eng-

lish scale) should be employed, after warming it and lubricating with glycerin. Its use should be preceded by a cocain-spray to prevent spasm. The patient should occupy a low seat, with his head supported by an assistant from in front of the operator. The head should be only slightly thrown backward. The forefinger of the left hand should then be passed back over the tongue until it touches the epiglottis, and the bougie inserted along it with the right hand, thus avoiding the error of passing it into the naso-pharynx or the larynx. When the bougie reaches the cricoid cartilage it is sometimes gripped pretty firmly even in a healthy person—a fact that is always to be remembered. No force should be applied. The instrument may pass the constriction with a jerk, or it may not only be gripped, but distinctly arrested, when a smaller bougie should be tried. By moving the instrument upward gently we may detect sometimes several strictures lying one above another. To locate the obstacle, the distance from the teeth to the point of stricture is measured on the instrument, and the results compared with the normal measurements, which are as follows: from the teeth to the cricoid cartilage, 7 inches (17.7 cm.); to the left bronchus, 11 inches, (27.8 cm.); and to the opening into the diaphragm, 15 inches (37.9 cm.).

Auscultation of the esophagus has been practised, but the clinical indications afforded are of little practical value. The stethoscope is placed to the left of the spine, and the patient takes a mouthful of water, when, if a stricture be present, a splashing, cooing sound will be heard at the seat of the stricture instead of the normal esophageal bruit.

Differential Diagnosis.—It is important for rational treatment to determine not only the existence of a stricture but also the underlying disease. First and foremost, we must exclude those affections that simulate simple and malignant stricture, in certain of which the introduction of the sound would be attended with grave dangers. *Compression of the esophagus* by enlarged or accessory thyroids, aortic aneurysms, vertebral abscess, enlarged lymphatic glands, and occasionally pericardial effusions, may produce dysphagia, and on passing the bougie resistance is offered at the seat of the external pressure. As a rule, the extent of the stenosis is moderate. If the narrowing be due to aneurysm—"(*a*) rhythmic movement is sometimes communicated to the free end of the sound introduced as far as the stenosis." Careful physical examination will often reveal the presence of an aneurysm or other pressing tumor, and should never be neglected. Passage of the sound in cases of aneurysm has caused rupture of the sac and death. (*b*) Spasm of the esophagus or paralysis (the latter rarely) may closely resemble true stenosis. These neurotic forms are almost exclusively met with in hysteric females; on the other hand, malignant strictures are found generally in males over forty years; while in simple stricture there is usually a definite history and certain etiologic factors.

To discriminate between simple and malignant stricture is not difficult, as a rule. When a clear history of gumma, of tuberculous disease, or of injury (from corrosive liquids) is obtainable, the presence of a simple stricture may be safely inferred after eliminating the affections previously mentioned. In the absence of etiologic data pointing to the simple form, cases occurring in the male after forty years of age may be looked upon as malignant.

Prognosis.—In forming a prognostic opinion the chief factor to be considered is the nature of the stricture. Practically, so long as the stenosis is dilatable, the prognosis is not unfavorable provided sufficient nourishment can be taken; moreover, not a few cases of simple stricture are curable. The majority, however, come to a fatal termination finally.

Treatment.—The chief object of the treatment is to gradually and methodically dilate the stricture. The flexible English bougie above mentioned is the best for the purpose, commencing with one of good size; conical ivory bougies, having a flexible whalebone handle, may also be used, though, being quite hard, they are apt to inflict injuries unless used cautiously. It is sometimes necessary, on account of the tightness of the stricture, to begin with a catgut sound. The method of introducing these instruments has already been given. They should be used once daily, and often can be passed successfully by the patient himself. At intervals of three or four days trials of bougies of larger size should be made. I have seen truly remarkable results from this treatment when carried forward systematically in cases due to cicatricial contraction, the patients increasing in bodily weight and strength. In annular constrictions of a malignant type, however, it is productive of temporary benefit only.

The *diet* deserves most careful attention. When the stenosis is so pronounced as to prohibit sufficient food being swallowed, a Symonds tube should be passed into the stomach, and through it liquid food is introduced. Concentrated forms of nourishment, as raw eggs, bovinin, and the various infants' foods, may be administered with milk.

When the passage of the bougie is no longer possible relief may be secured in one of two ways: (1) rectal feeding; (2) gastrostomy, if the seat of the stricture be near the stomach, and esophagostomy if at the upper portion of the gullet. I have recently witnessed favorable results from gastrostomy in a case of simple stricture operated upon by Laplace. J. McCrae advises the wearing of a permanent tube, which may be fixed by cords through the mouth and fastened to the ear. It is important that the patient should thoroughly masticate the food before introducing it into the stomach. Before resorting to operative procedures, however, careful trial should be made of rectal feeding. Various forms of nutritious enemata and other points regarding rectal alimentation will be found in the Treatment of Gastric Ulcer.

VII. DISEASES OF THE STOMACH.

METHODS OF DIAGNOSIS.

The examination is begun by the patient's narration of his past and present troubles, family history, and any special peculiarities in health. It is all important to ascertain whether the patient has lost or gained in weight, and the condition of the bowels. The necessary knowledge is best acquired by asking a series of questions which, for sake of convenience, I have arranged under the following subheadings:

Pain.—Pain, when present, may be located at the pit of the stomach (cardialgia), or in the gastric region (gastralgia). The pain may be severe, slight, or merely a discomfort and uneasiness. All important is it to know when and how (sudden or gradual) the pain appears, and what conditions excite or relieve such distress. Does the pain develop before meal time and when the stomach is empty, and is appeased by the taking of food; or is it excited by taking food, and does it appear immediately after food, or one to four hours later? Is the pain constant, and is it local or diffused? Does it radiate to the back or scapular regions?

Appetite.—The loss of appetite (anorexia), or a desire for unusual foods (parorexia), are frequently noted. When the appetite is increased, or the patient becomes hungry a short time after a meal, it is referred to as "bulimia."

One should determine further whether the appetite comes on when the patient begins to eat, or disappears at the sight of food, or after a few mouthfuls of food are taken. The taking of abnormally large amounts of food at meal times only is termed "polyphagia." Where the appetite is not satiated, even after a full meal, we refer to such condition as "acoria."

Thirst.—In certain maladies the thirst is increased, while in a second class of conditions there is little or no desire for water or other liquids. Inquire whether thirst is allayed by taking water.

Taste.—Many gastrointestinal conditions are accompanied with an unpleasant, sour, bitter, or sticky taste which may be experienced only on waking, or it may be more or less persistent.

Deglutition.—Does the patient swallow both solids and liquids naturally; also is he liable to cough while eating, and does such effort cause discomfort or pain?

Pyrosis.—This is a burning sensation in the epigastrium and sternal region. Note at what time, before or after food, it is experienced, its duration, and how it is influenced by various foods.

Regurgitation.—Note how long after taking food this annoying symptom is observed, and also whether the food tastes sour? Where the contents of the stomach are expectorated, it is referred to as regurgitation, but should it be again chewed and swallowed, it is termed "rumination."

Hiccough.—The time at which hiccough occurs, and whether or not it is accompanied with a burning sensation in the throat or by an unpleasant odor, are points of clinical value. Prolonged hiccough is of grave significance.

Nausea.—Is it occasional or frequent, and how influenced by food and by sleep?

Vomit.—Inquire carefully as to the frequnecy of the vomiting; how influenced by pain; when the stomach is empty, after soft food, solid food, or is it excited by certain odors? The quantity and consistency of the vomit, as well as whether it ever contains fresh blood (red), or blood that has lingered in the stomach for a time (coffee brown vomit)?

Such special symptoms as constipation, mental dulness, sleepy and giddy sensations, and a blurring of objects, are not infrequently observed in gastric disorders.

EXAMINATION OF THE GASTRIC FUNCTIONS.

Secretory Function.—While gastric secretion normally continues as long as food is present in the stomach, during the later stages of gastric digestion the activity of the secretory function of the stomach diminishes, and to obtain accurate knowledge of any pathologic condition of the organ, examinations of the gastric contents must be made under conditions as nearly like the physiologic as possible. Reliable results cannot, therefore, be obtained from an examination of ordinary vomita, but the contents of the stomach must be procured at a definite period after a so-called test-meal (*vide infra*).

Numerous test-meals have been offered to the profession, but those that I have found most satisfactory are "the test-breakfast of Ewald and Boas" and "the test-dinner of Leube-Riegel." The former being simpler and easier of preparation than the latter, it is the oftenest used.

The *Ewald-Boas test-breakfast* consists of one or two rolls (50–70 gm.) and one cup of tea or water (300–400 c.c.). I constantly advise the use of one roll and a glass of water. About an hour after this meal has been taken the contents of the stomach are to be withdrawn, and at such a time HCl should be the only acid present.

The *Leube-Riegel test-dinner* consists of a large plate of soup (300–400 c.c.), a large piece of beefsteak (150–200 gm.), and some potatoes (about 50 gm.) or a roll—practically, a large plate of soup, a piece of meat (preferably beefsteak), and a roll of bread. The examination is to be made about three and a half to four hours after the meal.

To obtain the contents of the stomach we should use a stomach-tube. The tube is moistened with water and the end carried back to the pharynx; the patient is now asked to swallow, and the tube is gently pushed down the esophagus, these acts being repeated until the tube reaches the stomach. The stomach is emptied by siphonage.

The method I have most frequently used is that of "expression," as follows: The patient is asked to take a deep inspiration, and then to contract his abdominal muscles as in the act of having a stool: in this way the contents are quickly expelled through the stomach-tube. This should be first examined macroscopically to detect any residue from previous meals, such as meat and the like, and the quantity obtained should be 20 to 40 c.c. After filtering the gastric contents thus obtained it is variously tested.

Among qualitative tests the following are important:

To determine the *reaction*, ordinary litmus-paper is used; if acid, the blue turns red.

The presence of *free acids* is determined—(*a*) By Congo-red, a solution of which is turned blue by the addition of liquids containing free acids.

Free HCl.—Günzburg's test—phloroglucin gr. xxx (2.0), vanillin gr. xv (1.0), absolute alcohol ℥j (30 c.c.). To two or three drops of this reagent add an equal number of the gastric filtrate in a porcelain dish, and slowly evaporate to dryness over a flame; if free HCl is present, a rose-red tint appears along the edges. Blowing at the edge will hasten the reaction. The great delicacy of this test is conclusively shown by its availability when HCl is present in the proportion of 1 to 20,000. There are no recognized interfering conditions.

Boas' Resorcin Test.—Resublimed resorcin 5 parts, white sugar 3 parts, and diluted alcohol 100 parts. The method of procedure is the same as in Günzburg's test, and a purple-red color appears. More caution is required in evaporating, but this method will also detect the presence of free HCl in the proportion of about 1 : 20,000.

Töpfer's Test.—To a few c.c. of filtered (or unfiltered) stomach-contents, 1 to 4 drops of the reagent (dimethylamidoazobenzol in a 0.5 per cent. alcoholic solution) are added; in the presence of free HCl a rose- or cherry-red color is produced. Combined HCl gives a negative result. The presence of acid salts, peptones, mucin, and starch (in the usual percentage) do not interfere with this reaction.

Lactic Acid.—Uffelmann's Test.—The reagent should always be freshly made, as follows: To 10 to 15 c.c. of a 2 per cent. aqueous solution of carbolic acid add 1 or 2 drops of neutral ferric chlorid, when an amethyst-blue color will appear. To 1 or 2 c.c. of the mixture add a few drops of the filtrate, and if lactic acid is present a canary-yellow color appears. Sources of error may be overcome by shaking 5 or 10 c.c. of the filtrate with double the quantity of ether, and, after allowing the ether to separate and pouring it off, adding more ether to the filtrate, again shaking, and repeating the washing. The ether is then evaporated almost to dryness in a water-bath. To the residue about 1 c.c. of water is added, and to this an equal quantity of the Uffelmann reagent from a pipette; and if a canary-yellow now appears, positive proof of the presence of lactic acid is afforded. Bread contains lactic acid, and hence it is better to employ a thin gruel made by adding to a quart of water flavored with salt half an ounce of oatmeal-flour. Boas states that no lactic acid is present in the filtrate several hours after this test-meal, except in cases of carcinoma of the stomach. Lactic acid in the stomach-contents also occurs with fermentation-stagnation from either obstruction or deficient motility.

A more reliable test for lactic acid than the foregoing is that of Boas, as follows: Digest the filtrate several times with ether to remove the fatty acids: add a few drops of phosphoric acid and boil. Transfer the mixture to a distillate flask; add H_2SO_4 and MgO_2; heat, and lactic acid will pass over. This can be conducted into a strongly alkaline solution of iodin and potassium iodid. The presence of lactic acid is then shown by the production of iodoform, which can be recognized by its odor and by the precipitate formed.

Fatty or Volatile Acids.—Heat to boiling a few c.c. of the filtrate in a test-tube, over the mouth of which place a strip of moistened blue litmus-paper; the presence of fatty acids will change the paper to red.

Acetic Acid.—In large quantities this acid is detected by its odor, and in smaller quantities its presence is determined by neutralizing with sodium carbonate the watery residue of the ethereal extract, and adding neutral ferric chlorid, when a blood-red color develops. Quantitative estimation of certain constituents is desirable.

Total Acidity.—To 10 c.c. of the filtrate add 1 or 2 drops of a 1 per cent. alcoholic solution of phenophthalein, and decinormal solution of sodium hydrate is added slowly from a buret until the reddish color that appears fails to disappear on shaking. The number of cubic centimeters of the decinormal solution normally required ranges from 4 to 6;

hence, if these be multiplied by 10, we have 40 to 60 as the percentage of acidity. Under pathologic conditions these numbers may be either higher or lower. This total represents both free and combined acids. If no organic acids be present, the above figures will represent the percentage of HCl. The latter is also reckoned thus: If it required 5 c.c. of the decinormal solution of sodium hydrate to be added to 10 c.c. of the filtrate to get the red color (alkalinity) with the phenophthalein, we say the acidity is 50, and multiplied by 0.003,646 = 0.1823 per cent. of hydrochloric acid. The normal range of percentage is from 0.1 to 0.22.

Estimation of Free HCl.—Mintz's method: To 10 c.c. of the filtrate add a decinormal solution of sodium hydrate from a buret until no reaction is given with Günzburg's reagent. The number of c.c. of the decinormal solution used, multiplied by 10 and then by 0.003,646, gives the percentage of free hydrochloric acid.

Töpfer's Method.—To 10 c.c. of filtered gastric juice 1 or 2 drops of Töpfer's reagent are added, and then also a decinormal solution of soda, drop by drop, until the last trace of red has changed to yellow. To estimate the percentage of HCl, the number of c.c. of soda solution required to neutralize the free HCl in 100 c.c. of stomach-contents is multiplied by 0.00365. Example: To remove the red color 4 c.c. of soda solution are required; hence, $0.00365 \times 40 = 0.14$, the percentage of free HCl.

Estimation of Combined HCl.—The difference between the total acidity and the percentage of free hydrochloric acid represents approximately the percentage of combined hydrochloric acid.

Estimation of Lactic Acid.—If the volatile acids are present, they should be removed by boiling. Take the total acidity of 10 c.c. of the filtrate; then to a second 10 c.c. add 25 to 30 c.c. of ether; shake well, allow the ether and filtrate to separate, remove the ether, and again add 25 to 30 c.c. of ether; shake, and repeat the process. Next obtain the acidity of the watery solution, and the difference between this and the total acidity, multiplied by 10×0.09, will give approximately the amount of lactic acid.

In the *gastric digestion* of the *albuminoids* (proteolysis) the proteids are converted into peptone. The degree of hydration of albumins during the various steps of digestion are of little clinical value.

In a later stage of the process of albumin-digestion peptone is produced and its detection is easy. To a small quantity of the filtrate (the propeptone having been removed) add enough sodium or potassium hydrate to render the solution alkaline; then add a few drops of a 1 per cent. solution of cupric sulphate, and, if peptone be present, a rose-red color is presented.

The Test for Pepsin.—To a test-tube containing 15 c.c. of filtrate add a small piece of egg-albumen, and keep at a temperature of about 100° F.; if present, the albumen disappears in from two to six hours. If hydrochloric acid is absent from the filtrate, add a few drops of the dilute acid. It should be pointed out that laboratory attempts to estimate the rate of albumin-digestion are unreliable.

Rennet Ferment.—To 10 c.c. of raw milk add five drops of the gastric filtrate, and keep it at a temperature of about 100° F.; if rennet is present, coagulation into a single cake occurs in from a few minutes to an hour or more.

Rennet Zymogen (which is converted into *rennet ferment* in the presence of an acid).—To 5 c.c. of gastric filtrate add enough sodium carbonate or sodium hydrate to make it slightly alkaline; then add calcium chlorid (1–2 c.c. of a 2 per cent. solution); then mix with an equal quantity of milk, and, if zymogen is present, coagulation occurs as in the case of rennet ferment. Both rennet ferment and rennet zymogen may be assumed to be present when HCl has previously been found.

Starchy Derivatives.—To 10 c.c. of gastric filtrate add 1 or 2 drops of Lugol's solution; the presence of dextrin gives a blue reaction—erythrodextrin purple, achroödextrin, grape-sugar, and maltose (intermediate substances)—showing a yellowish color. If there is a mixture of these starchy derivatives, as when the digestion of starches proceeds naturally, the first few drops of Lugol's solution may produce no color-reaction, or it may be taken up by the dextrose or maltose, while the addition of more of Lugol's solution will give a purple (if erythrodextrin be present) or a blue color, due to starch.

Indeed, if a minute quantity of the solution strikes a blue or purple tinge, conversion of starch into maltose has been abnormally tardy. I believe this is oftenest due to hyperacidity, though it may also more rarely be due to a defective ptyaline-supply.[1] For methods of detecting *occult blood*, see p. 832.

The Tests for the Motor Function.—More important than the secretory is the motor function of the stomach. There are several tests.

The oldest method is that of *Leube*. It consists in washing out the stomach from six to seven hours after a large meal, preferably consisting of beef-soup (13 oz.), beefsteak (6½ oz.), bread (1½ oz.), and water (6½ oz.), or from two to two and a half hours after Ewald's test-breakfast. Normally, the stomach should be empty within these periods of time, so that if a residue remains it denotes a lack in the motor force. Boas recommends the giving of 400 c.c. of water to which 20 drops of chlorophyll (concentrated aqueous solution) have been added. Thirty minutes after the patient has drunk this, the stomach-tube is passed. With normal motility about 50 to 60 c.c. are recovered.

To Test the Absorptive Power.—The method described by Penzoldt has been almost universally adopted: A capsule containing 0.2 to 0.3 grams of potassium iodid is given to the patient just prior to the taking of a full meal. The iodid is absorbed from the stomach and appears in the saliva, normally in ten to fifteen minutes. The saliva being tested every three minutes by strips of filter-paper wet with starch solution, the characteristic blue color being observed as the iodin enters the saliva. The reaction may be delayed for a half-hour or more and rarely may fail to appear at all. In the Sahli desmoid test the patient swallows a little iodoform or methylene-blue wrapped in rubber tissue and tied with raw catgut; the time required for their appearance in the urine corresponds to the state of the secretory functions.

[1] The tests for the estimation of the combined acids, of some of the fatty acids, and of many of the products of proteolysis are complicated and unnecessary in an ordinary clinical examination.

PHYSICAL OR EXTERNAL EXAMINATION.

This implies the well-known physical signs—inspection, palpation, percussion, and auscultation, including succussion or splashing.

Inspection.—(*a*) *General.*—This may give an idea of the nature of the illness as well as its severity by noting whether the patient appears to belong to a neurotic group, the general health often being good, or whether the patient is emaciated, or has with the latter the cachexia of a malignant growth. In diseases of the stomach attention should be directed to the mouth, and especially to the teeth, these may be of causal importance in gastric ailments, and frequently prevent their cure.

(*b*) *Local Inspection.*—In patients with thin and relaxed abdominal walls the contour of the stomach can be plainly noted; especially is this the case in very large, dilated stomachs or in those that have been displaced. The examiner is greatly aided by inflating the stomach with air or gas. The former is to be preferred, for the reason that the supply is easily regulated; he is enabled to watch the different steps of the distention, and after the examination is completed the air is allowed to escape through the tube. For this purpose an ordinary stomach-tube is most convenient, and its passage is to be effected in the same way as in removing the gastric contents. A double bulb-attachment is connected with the external end of the tube, by means of which air is readily forced into the stomach (*Runeberg's method*).

Frerichs' method is sometimes used. It consists in administering ʒj (4.0) of tartaric acid, dissolved in half a glassful of water, and immediately afterward ʒj (4.0) of sodium bicarbonate, dissolved in the same amount of water. Effervescence now occurs, with a progressive visible distention of the organ. There are many objections to this method.

The inflated stomach presents a circumscribed protuberance, usually in the epigastric, and also in the umbilical region if the organ is dislocated or dilated. The air may find its way into the intestine, producing a visible change in the contour of the abdomen. Tumors and other abdominal enlargements may also be recognized, and an idea obtained as to which organ is involved, after making due allowances for displacement, as in gastroptosis and pyloric carcinoma. Exaggerated peristaltic waves may also be noticeable in the upper portion of the abdomen, usually when associated with the stomach, and in the lower portion if it is in the small intestine. Peristalsis is increased from various causes—inflation of the stomach, external tapping, neuroses, pyloric obstruction, and the like.

The value of the gastroscope in inspecting the interior of the stomach is, I think, questionable. Gastro-diaphany (illumination of the stomach) is sometimes useful in showing the fundus extending to a lower level (at the navel) than is indicated by percussion, and in indicating the presence of tumors in the anterior wall of this organ. The Röntgen rays show the outline of the stomach, though indistinctly, after the administration of bismuth subnitrate (ʒj—31.0).

Palpation.—This elicits at times more trustworthy information than inspection. The patient should be in the recumbent position, the lower limbs partially flexed on the abdomen and the head low. The examiner should stand at the right side of the patient and use the right hand,

which should be warm. With the palmar surface down gentle pressure should be made with the fingers and the ulnar side of the hand. If the abdominal wall is tense, it is best to distract the attention of the patient from the examination by talking to him. In this manner we can corroborate inspection as to the size, shape, and position of the stomach, and can detect morbid growths as well as determine their consistency and movability.

Deep palpation, by increasing pressure with a slightly rotatory movement, elicits the degree of sensitiveness, tenderness, or pain, whether circumscribed as in ulcer or diffuse as in generalized inflammatory states (enterocolitis, peritonitis). In deep-seated tumors palpation should also be made in the knee-elbow position, and if movable they may drop to the abdominal wall. Gurgling and succussion-sounds of some diagnostic value may be elicited. In some instances relief from pain may be noted on pressure with the broad hand in neuroses. Variations in the degree of tension and of resistance are found and prove helpful.

Percussion.—The patient is placed in the recumbent position; the examiner uses his fingers and endeavors to discriminate the slightest differences in the note, and percusses lightly. If the stomach is empty or partially filled with gas, it gives a lower tympanitic sound than the colon. To ascertain the size and position of the stomach by percussion the process should begin at the symphysis pubis and follow the median line upward. The upper border of the stomach is at the ensiform cartilage, the lower about two fingers' breadth (3 cm.) above the umbilicus. If the upper margin is some distance below the ensiform, displacement of the organ is indicated; this depression may be occasioned by various diseases of the thorax. The stomach may be elevated by great distention of the gut or peritoneal sac.

It is well to trace the limits of resonance of the stomach and of any areas of dulness met with, so that their size and position may be graphically represented. The differences in the percussion-note over the stomach and colon may be greatly exaggerated by inflating the former. Runeberg's method is to be preferred. By employing light percussion the limits of the stomach can now be easily and accurately defined, unless the transverse colon be at the same time greatly distended with gas. In such instances Dehio's modification of Piorry's method is to be resorted to. It consists in giving about 1 liter (1 quart) of water in fractional doses while the patient is standing; one-quarter of the liter is swallowed and percussion practised, when a dull note will be obtained over the most dependent portion of the stomach. A second quantity of equal amount is given and a re-examination made, and so on, the object being to ascertain to what point the lower border sinks on the addition of more fluid. Boas holds that this method tests effectively the tone of the stomach, and that a *marked* descent of the lower border after each addition of water is indubitable evidence that there exists weakness or atony of its walls. If a neoplasm originates posterior to the stomach or colon, inflation of the latter may cause the previous circumscribed dulness to disappear.

By striking the abdomen in the epigastric region splashing-sounds may be produced. This sign is of diagnostic value in dilatation of the stomach, though its absence does not contradict the presence of the

dilatation. Again, if the splashing-sound is obtained in a fasting stomach, it may give a clue to some abnormal condition. The stomach may contain large quantities of fluid and no splashing-sound be obtained. Caution should be exercised lest the splashing-sound sometimes produced in the transverse colon be mistaken for that originating in the stomach; in the former the sound is usually associated with diarrhea, while in the latter constipation usually obtains. The outlines of the stomach can be most satisfactorily determined by auscultatory percussion.

Auscultation.—Various sounds are heard, none of which are pathognomonic of any diseased condition.

Succussion-sounds are produced by shaking the patient, and, if the stomach is dilated and contains fluid, a splashing sound may be audible some distance from the patient, and when heard after digestion has been completed they indicate some abnormal condition. Heard below the umbilical line, they usually indicate dilatation. In motor insufficiency (atony) of the stomach-walls splashing-sounds are audible after swallowing a few ounces of water. Partial obstruction of the cardiac orifice causes a delay of the "deglutition murmur" (a hissing sound followed in six or seven seconds by either gurgling, sprinkling, or splashing), as heard over the esophagus with the stethoscope while the patient is swallowing a liquid, "while in complete or almost complete closure of the cardia, this murmur is absent" (Ewald).

MALPOSITION OF THE STOMACH.

THE stomach may occupy a truly vertical position in consequence of the persistence of the normal infantile condition or of improper clothing—*e. g.* long-continued pressure from corsets. Unless an angular condition of the duodenum, causing obstruction to the outflow of the gastric contents, followed by dilatation of the stomach, be engendered. the malposition is of little or no clinical significance. Transposition of the stomach, with the organ occupying the right hydochondrium, is rarely met in association with transposition of other viscera.

GASTROPTOSIS.

Definition.—Downward displacement of the stomach. The lesser curvature of the organ lies normally about midway between the ensiform cartilage and the umbilicus, and the greater curvature may descend to near the symphysis pubis.

Etiology.—So far as our present knowledge extends, the conditions and circumstances contributing mostly to the origin and development of gastroptosis are—(*a*) Age and sex. Meinert of Dresden found among girls of fourteen years gastroptosis in 80 per cent., and among the women who presented themselves at his private clinic in 90 per cent. According to my observation, gastroptosis is not as frequent among American girls and women as among the Germans. "Dislocation occurs in about 5 per cent. of the male population of Dresden." (*b*) Improper clothing, particularly tight lacing. (*c*) Dislocation of the right kidney. This operates potently, and prolapse of other abdominal organs, as the liver and intestines, is often associated, and may

constitute the chief point of departure. (*d*) Repeated pregnancies, inducing a relaxed state of the abdominal wall. (*e*) Muscular strain and local injury, by diminishing the tonicity of the gastrohepatic omentum. (*f*) Abnormalities of the chest-formation (kyphosis); gastrectasis; great meteorism, and enlargement of the abdominal organs, especially of the spleen and liver. Certain chronic diseases may be active—*e. g.*, chlorosis, tuberculosis. (*g*) Congenital weakness of the supporting ligaments.

Symptoms.—Malposition of the stomach may exist without symptoms, but commonly it produces functional disturbances of clinical importance. The latter are due, first, to the difficulty that the stomach experiences in emptying its contents. Soon functional disorders arise in consequence of gastric atony, and later there is apt to be a greatly diminished gastric secretion and motility, associated with dyspepsia and neurasthenia. The stomach may be of natural or of diminished size (as the primary result of the compression of the corsets—Fleiner) or it may be dilated. *Constipation*, due to defective peristalis, and *colicky pains*, due to spasm of the intestinal muscles, are important features. Diarrhea is sometimes present.

Physical examination of the inflated stomach[1] permits the accurate demonstration of gastroptosis. The epigastrium is hollowed, while the lower quadrants of the abdomen are prominent. The percussion-note now indicates the position of the organ. It is to be borne in mind that the cardiac end remains fixed at the twelfth dorsal vertebra, while the pylorus moves downward and to the left: this will explain why the epigastrium is free of gastric tympany. Dilatation of the pyloric end is present in varying degree in most cases. Much more rarely general dilatation is found with gastroptosis. *Succussion splashing-sounds* may be heard if atony, with retained gastric contents, obtains. The differentiation of gastroptosis from *dilatation of the stomach* is also accomplished by the method of inflation, since this makes plain the course and position of the lesser curvature and of the pylorus.

The **prognosis** is not unfavorable as to life and is frequently modified by the presence of special causal agencies, and in others by the occurrence of certain complications, as dilatation of the stomach.

The **treatment** has relation to the removal of all causative conditions. Sufficient rest after confinement is an important preventive measure. The abdominal walls should be strengthened by means of suitable gymnastic and athletic exercises in childhood and youth. Massage, and recumbency, preferably on the right side, after somewhat restricted meals should be enjoined. Many cases are relieved by the support of a properly adjusted belt and pad. McCoskey advises, for the support of the viscera, a strip of zinc oxid adhesive plaster across the lower abdomen, to each end of which is attached a bandage long enough to reach around the body above the iliac crest.[2] Borgbjärg and Fischer lift the stomach by inflating a bag underneath an inelastic corset. Gastrorrhaphy and shortening of the gastrohepatic and gastrophrenic ligaments have given promising results.

[1] Inflation may be accomplished by the introduction of atmospheric air (*vide ante*).
[2] For details, see *Jour. Amer. Med. Assoc.*, Oct. 28, 1911.

DILATATION OF THE STOMACH.

(*Gastrectasis.*)

THE condition is to be subdivided, clinically, into acute and chronic forms. The normal capacity of the stomach varies within rather wide limits, though the maximum normal capacity, according to Ewald, does not exceed 1600 c.c. (1.5 quarts); enlargements above this capacity may then be said to fall under the heading of dilatation.

Etiology and Pathology.—The chief factor in the production of chronic dilatation is **pyloric stenosis.** This is usually due (*a*) to carcinoma, cicatrix of an ulcer, fibroid overgrowth and spasm of the pylorus, or the contraction consequent on the action of corrosive poisons; (*b*) to the external compression arising from carcinoma of the liver, pancreas, or gall-bladder, the omental lymph-glands, and a displaced right kidney, or from large gall-stones; (*c*) to perigastric and duodenal adhesions—*e. g.*, with the gall-bladder, and congenital pyloric stenosis.

In all such instances increased force is necessary to propel the food from the stomach into the duodenum, thus leading gradually to a hypertrophy of the muscular fibers, particularly in the immediate vicinity of the pylorus. So long as this hypertrophied state of the muscular layer compensates for the obstructive lesion, pathologic dilatation cannot occur. Just as soon, however, as the muscles prove to be inadequate on account of secondary degenerative changes, accumulation of the food in the stomach ensues. This tendency for the contents of the stomach to accumulate is very much augmented by the increasing weakness of the muscle on the one hand and the progressing degree of stenosis on the other. Chronic gastric catarrh ensues in consequence of the chemical (from putrefactive changes) and mechanical effect of the undigested food. The degree of dilatation is enhanced by the generation of excessive quantities of gases under these abnormal conditions, as well as by the great weight of the accumulated gastric contents. When produced in this manner the stomach attains enormous dimensions. Dilatation is usually general, though there may be mere diverticula.

Dilatation may also occur *independently of pyloric stenosis*, although less commonly, and the condition is not so pronounced. In this variety there is atony of the muscular coats, due to various and dissimilar causes: (*a*) repeated overstrain of the muscular layer, due to overfilling of the organ with food and drink, met with in diabetics and in those who habitually drink large quantities of beer; (*b*) chronic gastric catarrh and sclerosis, due to old ulcers, frequently weaken the muscle; (*c*) fatty and other forms of degeneration or nutritional disturbances associated with certain constitutional diseases (particularly carcinoma, anemia, and tuberculosis); (*d*) congenital weakness of the muscular coat (myasthenia); (*e*) impaired innervation, leading to imperfect peristalsis and consequent dilatation; (*f*) omental hernias (Bamberger) that drag down the stomach; (*g*) perigastric and periduodenal adhesions without narrowing of the gut or pylorus (F. Billings); (*h*) gastroptosis.

Acute dilatation has for its chief causes—(*a*) specific fevers, producing parenchymatous degeneration of the muscular coats; (*b*) the acute para-

lytic distention of Fagge, due to chronic catarrhal inflammation; (*c*) the drinking of large quantities of effervescing liquids; (*d*) following shock (Boas, Rosenheim); (*e*) sudden obstruction of the pylorus and of the duodenum (Bettmann); (*f*) dietetic errors; (*g*) trauma; and (*h*) post-operative. According to Neck,[1] there are 60 cases on record.

Clinical History.—Since the diseases causing dilatation are numerous and diverse, the clinical history presents great variations. The symptoms of dilatation are sometimes overshadowed by those of the causal affections. Among the earlier symptoms, *increased hunger and thirst* are frequently observed, partly due, most probably, to inanition. The thirst is also due, according to Von Weinig, to the fact that the stomach does not readily absorb water, and the pyloric obstruction prevents the passage of water into the intestines. *Vomiting* occurs at intervals of several days, the matter ejected amounting to from 1 to 3 gallons (4–12 liters). Occasionally the vomiting occurs more or less regularly some hours after feeding. The clinical characters of the *vomitus* are strikingly peculiar. The ejecta often contain remnants of previous meals, are, as a rule, excessively acid, emitting a sour odor, and on microscopic examination they show bacteria, sarcinæ, and torulæ in great numbers. The vomitus undergoes fermentative changes very rapidly, is ill-smelling, the odors being mainly due to sulphuretted and phosphuretted hydrogen. It consists of acetic, butyric, and lactic acids and partially decomposed food (HCl being usually absent), and on standing separates into three layers—an upper layer of brownish froth, a middle one of grayish-brown fluid, and a lower one composed of remnants of food. The acid contents of the stomach are not infrequently regurgitated, causing *pyrosis*. Eructations of foul gases are also common. A dragging pain is often present in the upper abdomen, most intense after eating.

Certain *general symptoms* almost invariably ensue. Progressive emaciation naturally follows, sometimes becoming extreme. A characteristic symptom is *muscular cramp* affecting the calves of the legs and sometimes spreading to the flexors of the arms and the abdominal muscles. Owing to the fact that but a small amount of liquid reaches the intestines, and also to the impaired absorption of the stomach, there are *constipation* and *scanty* urine, usually alkaline in reaction. The nervous phenomena of gastritis are in evidence and insomnia is often pronounced. *Loss of consciousness* has been met with. *Tetany*, with which indicanuria may be associated, particularly after lavage, has also been observed. A striking instance is reported by J. T. Whitcomb, in which nearly all the muscles of the body appeared to be in a tetanic condition. Cardiac palpitation and arrhythmia are often present and are induced principally by the effects of the dilatation. Nocturnal dyspnea (asthma?) may develop.

Physical Signs.—*Inspection* may reveal a rounded prominence just above the umbilicus, patient in the supine posture, and just below the umbilicus when standing. In the epigastric region there is sometimes a noticeable depression. The outline of the stomach may be made distinct by the patient taking an effervescing draught, and may sometimes be readily seen. The outline of the greater curvature is at times visible, "passing obliquely from the tip of the tenth rib on the left side toward

[1] *Jour. Amer. Med. Assoc.*, Nov. 10, 1906.

the pubes, and then curving upward to the right costal margin." Sometimes peristalsis is visible through the abdominal walls, and rarely the peristaltic waves are seen passing from right to left. These movements may be excited mechanically by various manipulations. *Palpation.*—The increased resistance of the walls of the stomach and their peculiar elasticity aid us in mapping out the contour of the stomach with more precision by palpation than by inspection alone. The movements of the organ can be plainly felt. A sign of considerable value is the loud splashing sound obtained by tapping the region of the stomach with the finger-tips of both hands alternately, or by shaking the body while the hand is placed over the epigastrium, though this should be distinguished from a similar sound produced in the colon. The patient may produce and maintain similar splashing sounds by voluntary efforts. *Percussion* furnishes subsidiary evidence as compared with palpation. The examiner should first percuss the empty, and then the filled stomach, if he would obtain reliable aid from this sign. When empty, an increased area of tympanitic resonance will be obtained, extending from above downward to a point several inches below the umbilicus. If now water amounting to 1 quart (1 liter) be introduced into the organ, and, in consequence, a line of dulness at or below the navel be noted where tympanitic resonance had been found, we have good evidence of the existence of dilatation. The posture of the patient should next be changed, when it will be found that the line of dulness has also altered. The stomach may be inflated by gas or air (*vide* Physical Examination) and its limits mapped out by *auscultatory percussion*. *Auscultation* reveals little that is of diagnostic value. The transmitted sounds heard over the stomach have a metallic ring. I have confirmed the observation by Franck and others, who claimed to have heard peculiar gurgling sounds produced by the heart's action and systolic in rhythm. Fluids swallowed by the patient may be heard dropping into the dilated stomach, and succussion-sounds may be elicited by shaking his body. *Measurements* made by introducing a probang into the stomach until it reaches the greater curvature are valuable only when the degree of dilatation is considerable. In health the instrument passes about 60 cm. (24 inches), reaching a point more or less nearly on a level with the umbilicus, while in extreme dilatation it may be introduced 70 cm. (28 inches). Dilatation can also be determined by an *x*-ray examination.

Diagnosis.—The diagnosis embraces, first and foremost, the recognition of the special causes. The unmistakable clinical manifestations are the characters of the vomitus and the peculiar manner of recurrence of the vomiting. The foregoing points, together with the physical signs, are adequate for a positive diagnosis.

Differential Diagnosis.—The condition is apt to be confounded with *ascites* or *overdistention* of the bowel, and in the female with *ovarian cyst*. In *dilatation of the intestines* the gastric symptoms of dilatation of the stomach are wanting; moreover, the physical signs are dissimilar. The splashing sounds on manipulation, the line of dulness below the umbilicus after filling the stomach, and other signs of gastric dilatation are absent in overdistention of the intestines. In addition, we may try the salol test, though this is now considered of little value (*vide*

Chemical Examination). From dilatation of the stomach we may discriminate *ascites* by the history and by the characteristic gastric symptoms belonging to the former affection. In dilatation the abdomen is asymmetric, the projecting prominence being in the vicinity of or just below the umbilicus. In ascites the lower portion of the belly is chiefly distended, and on assuming the recumbent posture the abdominal area becomes broadened and flattened. On palpation fluctuation may be elicited in the hypogastric and iliac regions. *Megalogastria*, or simple "big stomach," is distinguished by its absence of symptoms, and the fact that the food is passed into the intestines as quickly as in health. *Gastroptosis* may be distinguished by absence of decided motor insufficiency and by finding, on inflation, the lesser curvature lowered.

Acute Gastric Dilatation.—Acute dilatation of the stomach has a sudden onset; the first symptom is violent vomiting, accompanied by more or less intense pain in various parts of the distended abdomen. The *pulse* is small and rapid, but the temperature is normal. "The absence of a rise of temperature allows peritonitis to be excluded" (Neck). *Vomiting* is more frequent and severe than in the chronic form. Cyanosis is a common symptom, and pain often a prominent one. The patient frequently passes into a condition of collapse that may prove speedily fatal. Acute dilatation may arise in the course of chronic gastrectasis. Some cases represent a mere episode in the course of the chronic disease (Veeder, Todd).

Prognosis.—The prognosis in the *acute form* is uncertain, though the majority of cases recover; the condition may, however, tend to merge into the chronic form.

Chronic dilatation offers a bad prognosis, most instances being utterly incurable. Obviously, it depends greatly upon the causal conditions. A resort to surgical interference sometimes gives promise of a more favorable subsequent course in cases of cicatricial stenosis. Cases of dilatation that are not secondary to pyloric obstruction, however, give a more favorable prognosis on the whole.

Treatment.—One of the chief aims of the physician should be to lessen the labor of the muscular coat and to prevent the continual necessity of passing the usual contents of the stomach into the intestines. This is to be accomplished by careful attention to the character and amount of food taken and by frequent cleansing of the stomach. It is necessary to thoroughly empty the organ by lavage, repeated daily. Perhaps the best way in which to thoroughly empty the stomach is by the use of the stomach-tube, as will be detailed under Chronic Gastritis. Recently this has been replaced by the siphon apparatus as a simpler and more convenient mechanism than the former, and one not so likely to be attended with harmful effects, though perhaps less efficacious. The long course of these conditions renders it desirable that the patient should, whenever possible, be taught to wash out his own stomach. On account of the fermentative and putrefactive changes going on in the ingesta it is necessary to use weak antiseptic solutions for this purpose, suitable ones being a 3 per cent. solution of boracic acid or a 1 per cent. solution of salicylic acid. Subsequently warm water alone may be employed. Lying on the right side for an hour after meals, so that the opening in the pylorus is on a lower level with the rest of the stomach,

is worthy of trial. The *diet* should be composed chiefly of fluids, given in small quantities and at stated intervals. If the pyloric obstruction be not too far advanced, tender meats, eggs, and other easily digested albuminous articles of food may be allowed in moderate quantities. Since gastric digestion and absorption are very often markedly impaired, it is well to include those substances that are digested and assimilated after leaving the stomach, though they must be given in a fluid state. In no other manner can we bring such marked relief from gastric symptoms as by a suitable dietary, and in no other manner can the nutrition of the patient be so markedly improved. The weakened condition of the muscle-walls is due to overstrain and to degenerative processes; hence, after having minimized the labor thrown upon it, we should attempt to overcome its paretic state by the employment of such agents as strychnin and electricity. Stockton, Reed, and others have obtained good results from direct electrization of the stomach by the use of special electrodes; it improves motility and lessens the size of the organ. Exercises to develop the muscles, abdominal massage, and suitable bandages are also useful. For the associated catarrhal state the remedies recommended under Chronic Gastric Catarrh may be employed.

The deficiency of intestinal fluid is to be met by rectal injections of a weak solution (gr. v to ℥j—0.324–32.0) of sodium chlorid, not less than one pint of this solution being injected twice daily. In addition, nutrient enemata should be employed when, despite proper regulation of the dietary, loss of flesh and strength continue. For the anemia and debility tonics are indicated, particularly iron. Finally, surgical intervention often becomes necessary, and should not be too long delayed.

For *acute dilatation*, lavage of the stomach every hour or two if necessary, followed by complete rest and stimulation, with strychnin, eserine salicylate (dose, gr. $\frac{1}{50}$), and especially saline infusion, are the chief items of treatment. Place the patient upon the stomach with slight inclination to the right side so as to mechanically compress the dilated stomach and possibly unkink the duodenum (Morris).

INFLAMMATORY DISEASES OF THE STOMACH.

ACUTE CATARRHAL GASTRITIS.

(*Acute Gastric Catarrh.*)

Definition.—An acute catarrhal inflammation of the mucous membrane of the stomach, attended with more or less severe local and constitutional symptoms.

Pathology.—The postmortem evidences of an acute inflammation of the gastric mucosa are distinctive only of the graver, fatal forms. Observations upon cases of gastric fistula, however, have shown that in milder grades the morbid appearances are similar to those characteristics of acute catarrhal inflammations of the mucous membranes normally exposed to view. Thus, at first there are small irregular patches of redness, dryness, and ecchymosis. Later, serum effused from the congested vessels, and mixed with an increased quantity of mucus, escaped leukocytes, and desquamated epithelium, is present. Hem-

orrhagic erosions may be seen; the mucous membrane is now thickly swollen, softened, and covered with a tenacious mucopus. Infiltration and swelling of the solitary lymph-follicles are frequent; these sometimes form minute abscesses that rupture and result in follicular ulcers. The gastric tubules may be filled with a granular débris of epithelial cells. The above-described changes are more pronounced near the pylorus.

Etiology.—The *predisposing causes* of acute gastric catarrh embrace those various impairments of the system in which the normal functional activity of the stomach is altered or diminished. These are seen as the result of (*a*) improper hygienic surroundings; (*b*) malnutrition; (*c*) the various anemias; (*d*) in gouty and rheumatic subjects; (*e*) in the tuberculous, cancerous, and malarial dyscrasiæ; (*f*) associated with chronic passive hyperemia of the stomach due to emphysema of the lungs, cirrhosis of the liver, and renal and cardiac diseases; (*g*) in sickly and delicate children, in convalescents from acute diseases, and in enervated chronic invalids. (*h*) Persons having chronic gastric catarrh are predisposed to superadded attacks of the acute disorder.

The *excitants* are mainly (1) dietetic. These include the ingestion of much indigestible food; food or drink that is too hot or too cold (*thermal*); sour and highly-seasoned articles; the too free use of condiments; and especially the eating of decomposed canned goods and tainted meats. In cases due to the latter the fermentative and putrefactive agents (acetic, lactic, and butyric acids, and the ptomaïns) are the immediate causes of the catarrhal inflammation and tend to produce the constitutional disturbances, sometimes typhoid or septic in nature, that give rise to the so-called "gastric fever." The term "*crapulous gastritis*" has been applied to those cases due to gluttonous meals. (2) Toxic gastritis. Excessive indulgence in spirituous liquors is a common cause. Certain drugs, as the salicylates, iodids, bromids, arsenic, and mercury. (For the intense form of toxic gastritis, *vide* p. 793). (3) Acute infectious fevers, as measles, typhus fever, and scarlatina, provoke the disorder ("*infectious gastritis*"), as do also malarial fevers, especially when of the pernicious variety. (4) The influence of cold as an excitant of this disease has very probably been overestimated. (5) The *mycotic* origin of the condition cannot any longer be doubted. Among the microörganisms incriminated are the anthrax bacillus, the favus fungus, the Oidium albicans, and the yeast fungus. (6) Animal parasites (*e. g.*, ascarides, tænia, oxyurides, etc.) may cause gastritis.

Clinical History.—The symptoms of the ordinary or milder variety of acute gastric catarrh are embraced in the description of the "subacute gastritis" or "acute dyspepsia" of some writers. Soon after eating there are uneasiness, fulness, pressure, distress, and, perhaps, a dull pain referred to the epigastrium. Thirst is common, also nausea, eructations of gas or liquid, and, less often, vomiting. The *vomitus* consists of undigested food, considerable mucus, and fluid constituents that are sometimes bile-stained. The percentage of HCl in the stomach-contents is variable, although either absent or greatly diminished as a rule. The *tongue* is coated. The general condition of the patient remains unimpaired, and the average duration is less than twenty-four

hours. In *severer cases* the symptoms before stated are intensified, and particularly the nausea and vomiting. Physical exploration discloses slight prominence of the epigastric area, with more or less tenderness on palpation. The *tongue* is dry and heavily coated, the breath unpleasant as a rule, the patient complaining of a flat or bitter taste in the mouth. *Constitutional symptoms* appear early, and the onset is often marked by rigor and a febrile reaction, the temperature rising to 102° or even 103° F. (38.8°–39.4° C.). *Herpes* may appear on the lips and skin—a fact that points to the infectious nature of this complaint. The *pulse* is usually accelerated, and there are indisposition to exertion, headache, dulness, and other nervous symptoms. An erythematous cutaneous eruption is often present, particularly in febrile cases in children. The marked general disturbance is due to the toxic effects of the products of fermentation and decomposition.

Complications.—Constipation is a comparatively frequent complication, and diarrhea a comparatively infrequent one. Either coincidently or by direct extension the duodenum is similarly affected, and in some instances jaundice becomes an accompanying feature. The duration of this variety of the disease rarely exceeds four or five days.

Diagnosis.—The diagnosis of the lighter, afebrile forms of the disorder is not attended with the slightest difficulty. A logical diagnosis in cases in which well-marked local and general symptoms appear is not easy. The definite etiology, the vomiting (affording temporary relief), the pain or tenderness, the sudden rise of temperature, and the equally sudden fall at the end of a few days, however, are almost unequivocal.

Differential Diagnosis.—The absence of prodromata, of rose spots, of the peculiar temperature-range, and of enlargment of the spleen serve to distinguish this complaint from *typhoid fever*. The instances of indeterminate etiology may present a clinical picture not to be differentiated from certain infectious diseases. Here a careful analysis of the local symptoms and signs will usually lead to a correct conclusion, despite the apparently complete identity of the general disturbances. Close observation of the behavior of any obscure case for two or three days will usually enable the physician to arrive at a correct diagnosis. In children headache and vomiting are symptoms often so well marked as to create a striking resemblance to *tubercular meningitis*, but the latter can be discriminated by the history and longer duration. In children acute gastritis with an erythematous rash is often mistaken for *scarlet fever*. The final elimination of the latter disease is usually easy, however, in consequence of the absence of angina, of the typical tongue, the hard and very rapid pulse, and the peculiar desquamation affecting the hair and the nails.

Prognosis.—Quite generally the prognosis is good. When, as sometimes happens, however, the disease is purely secondary, the prognosis must depend largely upon the primary affection. Many persons suffer from repeated attacks of gastric catarrh, each increasing the liability to subsequent attacks.

Treatment.—Our chief aim should be to remove the cause and then to give the stomach complete rest. Hence, whenever the disease is distinctly traceable to errors of diet, emetics of the blandest sort should be employed; large draughts of warm water usually suffice, but lavage is

to be preferred in some cases. This should be followed by a purge made up as follows:

℞. Hydrarg. chlorid. mit., gr. j (0.0648);
Sodii bicarb., gr. xviij (1.16);
Sacchari lactis, gr. xij (0.777).
M. et ft. chart. No. vj.
Sig. One, dry on the tongue, every hour; the last to be followed in two hours by a wineglassful of Hunyadi Janos or other saline laxative.

The stomach must now have absolute rest for about twenty-four hours, when pancreatized milk or milk boiled with lime-water may be given at stated intervals. If nausea and continued vomiting prohibit the use of milk by the mouth, I resort to rectal alimentation early, and particularly in children. Certain symptoms, as *nausea*, *pain*, and *restlessness*, demand as early relief as possible, and can be most successfully met by the use of morphin in small doses hypodermically at intervals of twelve hours. When constant nausea is the symptom chiefly complained of, I have found creasote combined with bismuth or cocain in small doses to be highly serviceable. Convalescence is usually uninterrupted, and is soon complete. When protracted it is often on account of the too early return to solid articles of diet or the too early use of bitter tonics. The mineral acids should first be administered, well diluted, after the local symptoms have in a great measure subsided, and to these the bitter vegetable tonics are later to be gradually added. Locally, I employ sinapisms at the beginning of severe types of the affection, and follow these with warm linseed poultices lightly applied to the entire epigastric and hypochondriac regions.

TOXIC GASTRITIS.

Pathology and Etiology.—This is an intense form of acute gastritis, produced by the ingestion of irritant and corrosive poisons, among the former being such agents as phosphorus, antimony, and arsenic, and among the latter concentrated mineral acids and strong alkalies. When caused by the non-corrosive poisons, intense hyperemia and tumefaction, leading to desquamative changes in the glandular structure, ensue. When excited by corrosive substances necrosis of the mucous membrane may occur, leading even to an involvement of all the coats, and terminating in perforative peritonitis. Injurious retention substances, as in uremia, cholemia, and diabetes, may cause an autotoxic variety of gastritis. The lesions are either localized or general.

Symptoms.—The symptoms vary somewhat with the nature of the special poison, though they are usually quite violent. *Incessant vomiting*, great pain in the epigastric region, and, later, *diarrhea*, and excessive thirst, together with such symptoms as intense *burning pains* in the mouth and throat and dysphagia, are the most characteristic signs. The *vomitus* contains mucus, sometimes blood, and, rarely, shreds of mucous membrane. The *physical examination* reveals a marked distention of the abdomen, which is also, as a rule, very painful on pressure over the epigastric region. The *general condition* of the patient soon

becomes one of profound prostration; the skin-surface is cold and clammy, and the pulse and respiration are hurried, terminating at times in fatal collapse within a few hours. Sometimes there is a febrile movement; the temperature may reach 104° F. (40° C.); the pulse ranges from 100 to 130; and if life be spared long enough toxic nephritis, with or without hematuria, develops. The nervous symptoms (convulsions, stupor, sometimes ending in coma) may be due in part to the renal lesions, though mainly to the diminished alkalinity of the blood. Symptoms of gastric ulcer or of esophageal stricture may be sequelæ.

Diagnosis.—The diagnosis rests upon the history of the ingestion of some poison, upon the character of the symptoms (referable not only to the stomach, but also to the mouth and pharynx), and upon the results of an inspection of the mouth, pharynx, and the vomitus. A chemical examination of the stomach-contents and urine may be necessary.

Prognosis.—This depends upon the nature of the poison and its dose. When free emesis occurs early the prognosis is thereby rendered more favorable, since both the local and constitutional effects are thereby mitigated. Among unfavorable symptoms may be mentioned signs of collapse or of peritonitis. Among sequelæ (due to scar-formation) are pyloric stenosis and hour-glass contractions.

Treatment.—To ascertain, in the first place, the special cause of the gastritis, and when this is found to administer the proper antidote to that poison, are measures of prime importance. The stomach should be cautiously washed out with warm water containing some demulcent substance and a small proportion of the appropriate antidote. Subsequently measures should be employed to combat the active local inflammation. Externally, leeches, followed by the ice-bag, have proved to be the best agents in my own hands; internally, opium, bismuth, and demulcents, with bits of ice, are most useful. Rectal alimentation should form the sole method of feeding so long as the signs of severe inflammation along the upper alimentary tract are present. The indications presented by the general conditions will vary with the general effects of the peculiar poison in each case.

DIPHTHERITIC GASTRITIS.

This form of gastritis is always a secondary condition, though it is not, as has often been stated, always caused by a direct extension of the diphtheritic process from the pharynx down through the esophagus to the stomach. It arises more frequently in the course of some other acute infectious malady, as pneumonia, scarlet fever, or small-pox. Though it is regarded as a rare disease, the fact that it is unrecognizable during life renders it certain that the affection is sometimes overlooked. I have seen two instances associated with croupous inflammation of the intestines, both occurring in greatly debilitated children. Osler saw a case which occurred as a secondary process in pneumonia.

ACUTE SUPPURATIVE GASTRITIS.

(*Phlegmonous Gastritis.*)

Definition.—An acute suppurative inflammation of the submucosa.

Pathology and Etiology.—Phlegmonous gastritis is confessedly a rare, and almost invariably a secondary, disease. I have observed pathologic evidences of its presence, however, in two cases that came to autopsy, both patients having died of sepsis. It is excited by invasion with bacteria or fungi. The male sex is the more commonly affected. It may originate spontaneously or follow an injury; more commonly it is a symptom of a general septic process or a complication of an acute infectious malady. Two forms are described—namely, a *diffuse purulent infiltration* and a *circumscribed form* (*stomach-abscess*). The morbid process begins in the submucous layer, and then spreads in various directions, involving soon all of the coats. The limited variety results in the formation of abscesses that may attain considerable size and rupture either into the peritoneal cavity or into the stomach.

Symptoms.—There may or may not be an initial rigor. Whether the attack is ushered in by a chill or not, the *temperature* rapidly rises to 103° or 104° F. (40° C.), and subsequently pursues an irregular course. The symptoms of the *typhoid state* supervene, and are usually associated with the symptoms of the primary affection. Hence the clinical picture is greatly diversified. For a variable period prior to the fatal issue the patient passes into coma. The *local symptoms* and *physical signs* are rarely diagnostic. There is a constantly increasing epigastric *pain*, which is not aggravated by movement; *emesis* also appears, the *vomita* often containing a notable quantity of pus-cells, Leukocytosis is generally found.

The **physical signs** reveal but little in most instances, and vary with the form of the complaint. Inspection shows in the *diffuse* form a considerably distended abdomen. On pressure the stomach is found to be quite tender. In the *limited variety* the gastric abscess sometimes gives rise to the physical signs of a tumor, and a localized prominence may be seen over the seat of the abscess; the tenderness to the pressing finger may be confined to the same area. Palpation has served to elicit fluctuation and to define the limits of the tumor, the latter sometimes attaining the size of a cocoanut; on percussion either dulness or a muffled tympanitic resonance is elicited, varying according to the size of the mass.

Diagnosis.—The diffuse variety cannot, as a rule, be positively distinguished from certain other gastric affections. The detection of pus-cells is, however, of the utmost diagnostic value. Gastric abscess, on the other hand, is often recognizable, since the physician has not only the history to aid him, but also the physical signs, which may demonstrate the presence of a fluctuating tumor.

Course and Prognosis.—The majority of cases reach a fatal termination within one week, and those that do not terminate in death thus early pursue a subacute or even chronic course. They present such symptoms as local pain, chills, and fever, and death results, sooner or later, either from exhaustion or such complications as peritonitis and metastatic abscess with jaundice.

Treatment.—The treatment in the diffuse form is, at best, only palliative. In the circumscribed variety the aid of the surgeon should be invoked as soon as a probable diagnosis has been made.

CHRONIC CATARRHAL GASTRITIS.

(*Chronic Catarrh of the Stomach; Chronic Catarrhal Dyspepsia.*)

Definition.—A chronic catarrhal inflammation of the gastric mucous membrane, presenting various degrees of intensity and embracing the symptoms that are more or less characteristic of widely different clinical forms of gastric derangement.

Pathology.—The anatomic changes are most marked near the pylorus, where the mucous membrane often presents a distinctly wrinkled, mammillated appearance. The mucous membrane looks either red or gray (the latter hue being due to pigmentation), and is pretty generally covered by tenacious mucus, mingled with detached epithelium. Ewald describes the histologic changes thus: "The minute anatomy shows the picture of a parenchymatous and an interstitial inflammation. The gland-cells are in part eroded or show cloudy, granular swelling or atrophy. The distinction between the 'haupt' and 'beleg' cells cannot be recognized, and in many places, particularly in the pyloric region, the tubes have lost their regular form and show in many places an atypical branching like the fingers of a glove. Individual glands are cut off toward the fundus, but appear at the border of the submucosa as cysts, with a smooth membrane, partly filled with remnants of hyaline and refractile epithelium. An abundant small-celled infiltration presses apart the tubules, and is particularly marked toward the surface of the mucosa, and from the submucosa extensions of the connective tissue may be seen passing between the glands. The mucoid transformation of the cells of the tubules is a striking feature in the process and may extend to the very fundus of the glands." Hemorrhagic abrasions may be found in cases due to cardiac disease or to portal engorgement. Superficial ulcers may form, usually in the pyloric region, varying in size from a few lines to an inch or more in diameter, and nearly circular in shape. Long-standing cases also present sclerotic changes of the mucous membrane. Of these, two forms are distinguished. In the one variety the mucous membrane is perfectly smooth and atrophied; the glands are displaced, narrowed, and shortened, while the gap thus formed is more or less filled with connective tissue. There is a thinning of the stomach-wall, with enlargement of its cavity. The other form presents a hyperplasia of the mucous membrane, the glandular structure, and the submucous layer, sometimes resulting in enormous thickening of the stomach walls, with great diminution in the size of its cavity (*gastrophthisis*). The contraction of the new-formed connective tissue may cause polypoid projections.

Etiology.—It is evident that the factors which produce acute gastric catarrh will, if long continued, produce a chronic condition. The causes of chronic gastritis act either as mechanical, chemical, thermic, or biologic irritants, and fall naturally into the following classes: (*a*) Errors of diet (referring more particularly to important articles of food), its variety, and preparation; excessive alimentation; the habit of eating at irregular intervals or with undue haste, and thus not allowing time for perfect mastication of the food. The too free use of ice-water, tea, and coffee during meals plays an important *rôle* in the causation of dyspepsia in America. (*b*) The immoderate use of alcohol, more particu-

larly spirituous liquors, stands second in order of importance. Those persons who habitually indulge in alcoholic beverages to excess are prone to an irregular mode of life, which leads to digestive disturbances. Such patients are apt to suffer from the more active forms of the complaint, and, at intervals of time, from genuine acute gastritis. In the same category should be mentioned certain toxic irritants, as the overuse of tobacco and the prolonged use of tonics and purgatives. (*c*) Functional derangements of the stomach sometimes merge into the disease under consideration. This is true of that form in which there is a deficiency in the gastric juice. Under these circumstances, as also in gastric ectasy, fermentative and putrefactive changes develop in the retained stomach-contents. Stockton holds that the majority of cases of chronic dyspepsia are of nervous origin. (*d*) Local mechanical influences (portal congestion) may offer resistance or obstruction to the outflow of venous blood from the stomach to the right heart. In this way chronic gastric catarrh is a secondary process in chronic affections of the liver, heart, and lungs. (*e*) Such constitutional conditions as gout, chronic rheumatism, chronic tuberculosis, Bright's disease, diabetes, anemia, chlorosis, chronic malaria, syphilis, and chronic forms of skin disease. The explanation of the peculiar liability of these conditions to catarrh of the stomach lies in the obstruction offered to the passage of blood through the hepatic and cardiopulmonary circulation. This is true in an especial degree in chlorosis, anemia, chronic tuberculosis, and malaria; in gout, chronic Bright's disease, and syphilis it is probably due largely to the action of chemicovital irritants in the circulating medium. (*f*) Gastric carcinoma.

Clinical History.—The *local symptoms* bear a striking resemblance to those of other forms of gastric disturbance. They vary greatly in severity, though never entirely absent, as in the case of purely functional disorders. Deficient secretion of the gastric juice, due to the anatomic changes in the gastric tubules, is a potent factor in the production of the symptoms directly referable to the stomach. It is the function of hydrochloric acid, normally present in the gastric secretions, to destroy the ferment-producing spores; hence when, owing to lack of free HCl, the latter are not destroyed, deleterious products of fermentation are the result, these in turn aggravating and prolonging the course of the affection. Recent investigations go to show that deficient motor power is more important than a deficiency in the secretions in bringing about the clinical phenomena of the disease. The presence of an inordinate amount of mucus which is alkaline in reaction neutralizes in part the HCl; it may also more or less completely cover the ingesta, thus preventing the gastric secretions from reaching them, and lengthening, at the same time, the period of digestion.

Among the *earlier symptoms* directly attributable to the gastric lesions are anorexia (though at times the appetite may be moderately good or even keen); fulness and distress; burning sensations and dull pain in the epigastric region; eructations of gas, which may be either offensive or odorless, during and immediately after meals; regurgitation of fluid, either acid (heartburn), due to the presence of organic or hydrochloric acid, or a bitter form of peptones. These symptoms are usually increased in intensity after meals. The *tongue* frequently appears broad

and flabby, and almost constantly the edges and tip are somewhat reddened, whilst the papillæ are enlarged. Occasionally it is small, with enlarged and red papillæ, or it may look healthy. A bad or a persistently bitter taste and great thirst may be complained of. There may be a profuse secretion of saliva or the mouth may be dry. *Nausea* is common, and is most marked in the morning hours; it is frequent before or after meals, and often *vomiting* occurs either immediately after meals or a couple of hours later. The *vomitus* will vary somewhat with the time of the occurrence of emesis. Usually it consists of food in the first stages of digestion, mixed with large quantities of mucus. In alcoholic catarrh morning vomiting occurs, and consists mostly of saliva and mucus. This class of sufferers may exhibit well-marked evidences of salivation. In Kelson's experience, aching throat is often associated with flatulent dyspepsia. I have repeatedly found the material vomited in chronic gastric catarrh to be acid in reaction, unless, as occasionally happens, the vomiting takes place several hours after eating, when it is sometimes faintly alkaline or neutral. The acidity of the vomitus is not due to the presence of free HCl, but possibly in small measure to combined HCl, and partly and sometimes largely to acid salts (lactic, butyric) or resulting from the abnormal processes of fermentation.

Microscopic examination sometimes reveals the presence of sarcinæ ventriculi, yeast fungi, and numerous bacteria. The relations of these low forms of vegetable life to the pathologic processes going on in the stomach are not well understood. It is to be borne in mind that many of these bacteria are introduced with the food, and that certain of them contribute toward the production of gases, and of the organic acids of the stomach. Hydrochloric acid inhibits the development of bacteria.

A *chemical examination* of the contents of the stomach for purposes of diagnosis according to the methods laid down in the preliminary section (*vide* p. 778) should not be neglected. In simple chronic gastric catarrh the hydrochloric acid is found to be diminished, and lactic, butyric, and acetic acids are rarely present. In many cases of chronic catarrhal gastritis there is an abundance of mucus (*gastritis mucipara*—Boas); and in other cases there is present a normal amount of acid or even hyperacidity—the *gastritis acida* of Boas. In protracted forms free HCl is sometimes greatly diminished or entirely absent—*gastritis anacida*. According to Boas the difference between this and the atrophic form is but one of degree, all secretion being lost in the latter. In atrophic gastritis then there is little or no mucus in the gastric contents, and in established cases an absence of HCl and of the gastric ferments (*gastritis atrophicans*). Ewald has subdivided all cases into three varieties: (*a*) *Simple gastritis*, in which the fasting stomach contains only a small quantity of slimy fluid, while after the test-breakfast the HCl is diminished in quantity, and lactic acid and the fatty acids are usually present. (*b*) *Mucous gastritis*, in which class the acidity is always slight and the condition is distinguished from simple gastritis by the large amount of mucus present. (*c*) *Atrophy*. Here the fasting stomach is always empty, while after the test-breakfast HCl, pepsin, and the curdling ferments are wholly wanting.

The *absorbent* and *motor* powers of the stomach are both diminished in proportion to the degree of damage received by the stomach.

Physical Signs.—Sometimes there may be observed an undue distention of the stomach, the prominence being more marked toward the left. On making *firm pressure* over the epigastric region tenderness is often elicited. This is not present in the early stages, nor constantly later, since the degree of inflammatory action is subject to great oscillation. Diffuse tenderness in the absence of a new growth is of great diagnostic value. It is to be recollected, however, that resistance may be felt when the stomach is thickened in chronic interstitial gastritis. Dilatation of the organ may be indicated by splashing-sounds (*vide* Physical Signs, p. 787), and these are not suggestive of gastritis if detected at a time when the stomach should be empty.

On *percussion* we may note alterations in the size of the organ.

Among the general or indirect symptoms manifested the *nervous phenomena* are of first importance. So prominent are they in the clinical picture that the physician may suspect his patient to be suffering from some primary disease of nervous origin. The nervous derangements have been by many writers attributed solely to morbid sympathetic disturbances. It is altogether probable, however, that we should ascribe a share of the morbid influence to the absorption of toxic materials from the stomach and intestines. Headache is frequently complained of; it is generally frontal, though also occipital, and tends to appear before meals. The so-called sick headache more rarely occurs. Indisposition to mental or physical exertion, vertigo, depression of spirits, and well-marked hypochondriasis are common concomitants. Patients complain of wakefulness and disturbed dreams, though drowsy after meal-time. There is a sympathetic disturbance of the cardiac rhythm, and sometimes dyspnea, owing to the same cause. The urine is often highly colored, scanty, and deposits an abundant uratic sediment; occasionally (*e. g.*, in neurotic subjects), it is of low specific gravity, rather copious in amount and pale in color, owing to the influence of phosphates.

Complications.—The intestines often become involved, and usually by direct extension. Implication of the duodenum may lead to jaundice and to obstinate constipation, though only moderate constipation is the rule in catarrh of the stomach. When the process extends to the large intestines diarrhea develops. Alternating constipation and diarrhea are often observed. The nutritive system is, in confirmed cases, seriously implicated, as shown by the anemia, emaciation, and general debility present. It is particularly in examples of combined intestinal and gastric catarrh that we observe the most notable impairment of the general health, the reason being that under these circumstances all the digestive fluids are lessened in amount. The gases generated in the stomach often find their way into the intestinal canal, giving rise to distention, and sometimes to colicky pain. Perhaps many reflex sympathetic disturbances are of intestinal origin. The gastric catarrh may extend upward to the oral cavity. Under such circumstances the tongue is large and heavily coated, with impressions of the teeth upon its edges. The abnormal condition of the secretions renders the breath foul and causes thirst. Certain skin-eruptions, as eczema, lichen, and urticaria, are common. These disorders of the skin are probably due to an auto-intoxication from the intestinal tract. I have frequently observed, however, that when present their improvement has been followed by an aggrava-

tion of the gastric symptoms, and *vice versa*. A sequel of the disease is dilatation of the stomach. The *course* of chronic gastric catarrh is long, the average duration being considerably more than one year. Its duration may be much abridged by early recognition and proper treatment. The symptoms at first intermit and are mild, but later are persistent.

Diagnosis.—A positive diagnosis may be based on a clear etiology, the presence of persistent symptoms and signs of digestive disturbance, diminished (*usually*), normal, or even increased, amount of HCl (the atrophic form apart, *vide supra*), an abundance of mucus in the gastric contents, and deficient absorptive and motor power. The finding of mucus in the wash-water of the fasting stomach is truly diagnostic (Riegel). The points of difference between the more serious affections of the stomach (carcinoma, ulcer, and dilatation) and chronic gastric catarrh will be detailed when the former diseases are considered. As I have said, Ewald makes three leading forms of the complaint, based on the results obtained from an analysis of the stomach-contents, but transitional types are constantly met with.

Prognosis.—Chronic catarrh of the stomach may be said not to manifest an innate lethal tendency. It, however, aggravates the symptoms of existing forms of acute and serious forms of chronic diseases, especially other organic affections of the stomach. The prognosis depends considerably upon the stage that has been reached when first met with, since the condition is amenable to treatment only when not too far advanced. The prognosis is rendered somewhat more grave by the presence of certain complications, particularly intestinal involvement. I have seen one case that proved fatal in consequence of stricture of the pylorus.

Treatment.—It must never be forgotten as far as possible to search for and remove the causal affections in every case. When associated with grave forms of cardiac, hepatic, or renal disease these must receive careful attention primarily.

The masticating apparatus must be looked after by the physician, who must also instruct his patient in the art of eating slowly, so that insalivation of the food is thoroughly effected. Too often the quantity of aliment consumed is beyond the need of the bodily functions, and the method of preparing the same faulty. All food eaten should be fresh and pure. Such patients should eat oftener than in health, taking four or five meals in the twenty-four hours. The physician must with untiring diligence attend to every dietetic, sanitary, and therapeutic detail. The major portion of the treatment has relation to—

(1) *The Diet.*—In the matter of arranging the dietary in separate cases the general condition and peculiarities of the individual must be taken into account. The wise physican will be guided to some extent by the dictates of his patient's experience, and will not fail to avail himself of any information obtainable upon this head. The teachings of physiology direct that animal food should be allowed with a view to stimulating the secretion of HCl when found to be deficient in the gastric contents. We must, however, select the special articles of diet according to the severity and nature of the morbid process. In *severe cases* an exclusive milk diet for a period of two to four weeks often gives the best results. The daily amount requisite to meet the demands of the vital functions is 4 to 8 pints. Of this, 5 to 8 ounces are to be taken *slowly*

every two hours during the day. The beginning amount, however, must occasionally be smaller—2 to 3 ounces—to be gradually increased. A pinch of salt or from ½ to 1 ounce of lime-water may be added to each feeding, or the milk may be diluted with Vichy. The milk should not be taken iced, but warmed or at the temperature of the room. Boiled milk is objectionable. The stools are to be watched for curds, and when the digestive capacity is exceeded the amount of the nutrient should be lessened and other articles cautiously added.

When *whole* milk cannot be digested on account of an actual loathing for it, skimmed or partly skimmed milk or buttermilk should be substituted. If the latter cannot be utilized in proper amount, animal broths, together with some of the artificial foods (panopeptone, liquid peptonoids), may be added. As tolerance for a liberal amount of milk becomes established the appetite is no longer satisfied, and then I begin to add the light solids in a gradual manner; for example, white meat of chicken or game (except tame ducks and turkey), stale or twice-baked bread, milk or dry-toast or zwieback, soft-boiled eggs, oysters, fish, and, later, Hamburg steaks, stewed sweetbread, and the like. For dessert, junket or custards, sweetened with saccharin, are well borne as a rule. Subsequently, farinaceous articles, if thoroughly cooked (except oatmeal), and certain plain vegetables, may be allowed, but their effects must be minutely observed. The former are to be eschewed in cases in which acid-fermentation or flatulency is a prominent feature. Among the latter, rice, spinach, lettuce, and macaroni (stewed in milk) are to be selected. Peas and beans, if green and succulent, may be tried, but if ripe are to be discarded. The only form of fat permissible is good butter. Stewed fruits, Graham bread, and soft, green vegetables are often well borne and tend to overcome constipation. Pig's- and calf's-foot jelly may be allowed.

In light cases and in those of moderate severity, particularly if the cause of the complaint is removable, the dietary need not be rigid at the start. Indeed, to minimize the saccharine articles and starches and to avoid the coarser vegetables, hot bread, pastries, and the like, is all that is required. In the case of confirmed dyspeptics the following articles are to be scrupulously avoided: very fat meats, fat fish-foods, condiments, certain fruits (strawberries, bananas), hot bread, saccharine articles of diet and farinacea, potatoes, and coarser vegetables. Fermentable foods, as milk, eggs, and rare meats, should be avoided in selected cases.

The best drink during meal-time is simple hot water, to which a little milk may be added, or a single coffee-cup of weak tea. Occasionally cocoa is allowable, but ordinary chocolate, coffee, and strong tea are harmful. Too much liquid should not be taken during a meal, since it dilutes the gastric secretion to a deleterious extent, and cold drinks are to be interdicted during the same period. Alcohol, and particularly concentrated spirituous liquors, exert an irritating effect, and hence should be forbidden. In cases in which there is no gastric fermentation certain wines may be allowed (Oporto, Malaga, imported Hungarian Tokay).

(2) *Hygienic measures* are of signal value in this disease. Of these the most important are forms of fresh-air exercise, as bicycling, walking, boating, and horseback-riding. Suitable indoor apparatus for physical exercise is now easily obtainable at little cost, and therefore open-air

exercise may be supplemented by the latter. Physical exercise must be carefully supervised, so as to avoid the deleterious effects of over-exertion. I am convinced of the superior advantage of travel, including a sea-voyage, and an appropriate change of air—for example, to the seaside or mountains—particularly for the large class of self-centered and low-spirited dyspeptic patients. A cold sponge-bath, followed by brisk friction of the skin, is to be advised. An abdominal bandage, made of woollen or silk material and constantly worn, tends to increase the patient's comfort.

(3) *Medicinal Treatment.*—Saline laxatives, as sodium phosphate, Rochelle salts or Carlsbad salts, taken fasting in hot water, are advantageous, since they serve to regulate the bowels, to deplete the engorged gastro-intestinal vessels, as well as to rinse the stomach. Hunyadi Janos or Carlsbad waters may be substituted. Their efficacy is much enhanced when the alkaline carbonates are administered simultaneously. Patients may be advised to seek suitable watering-places, but the course should not be for too long a period. The use internally of antiseptics, combined with alteratives and mild astringents, is often beneficial. I can speak most positively in favor of the following pill:

℞. Argenti nitratis, gr. iv (0.259);
Ext. hyoscyami, gr. viij (0.518).
M. et ft. pil. No. xvj.
Sig. One about one hour before each meal, the stomach being first prepared by washing with one or more pints of a 2 per cent. solution of borax in water.

Hemmeter recommends silver nitrate, in the form of lavage (1 : 2000), or in the form of solution 0.3 to 120 of peppermint-water; of this one tablespoonful three times daily on an empty stomach.

In the *fermentative* form of chronic gastric catarrh the hyperacidity is, in reality, often dependent upon the lack of free HCl; hence this agent should be supplied. It is best administered immediately after meals, the dose being not less than 10 minims (0.666), well diluted, and this may be repeated in the course of ten or fifteen minutes in obstinate cases; it may be combined advantageously with pepsin (gr. v–x—0.324–0.648) or pancreatin (gr. x—0.648). Pancreatin is better associated with sodium bicarbonate in the form of a tablet containing each gr. ij (0.129). Of these two or three may be administered fifteen to thirty minutes after meal-time. Care is to be taken to use only the best articles of pepsin and pancreatin. When hyperacidity exists, diastase and ptyalin may be exhibited, but I have failed to obtain encouraging results from their employ. This class of cases represents an aggravated or advanced form of the disease (atrophic stage), and demands prolonged and varied treatment. At the end of the digestive process it is well to thoroughly irrigate the stomach (lavage), and more particularly if evidences of dilatation be present. The stomach may also be cleansed and prepared for the reception of the next meal in a very agreeable manner by having the patient sip a 2 per cent. solution of borax in warm water or a 2 per cent. solution of sodium chlorid half an hour before meals; indeed, the continued use of simple hot water for the same purpose has, in my hands, often given excellent results. With it must, of course, be combined the

saline laxatives and the restricted diet. Not less than 1 pint of water, hot as it can be taken by the patient, should be sipped at each sitting. Boas considers magnesium salicylate (gr. xv–xxx—1.0–2.0, t. i. d.) the best antifermentative remedy.

To assist the appetites of these patients and to stimulate the secretory function a few drops (not more than 5) of the tincture of nux vomica may be given fifteen minutes before meals, with gr. ii–iij (0.129–0.194) of sodium bicarbonate. These indications are also fulfilled by lavage once daily or bi-daily (if the patient be feeble). If hyperacidity, due to the organic acids, tends to persist, we may combine bismuth subnitrate with magnesia and a few grains of charcoal, this being administered when the stomach is empty. We may also check fermentation by the exhibition of salicylic acid (gr. v—0.324) thrice daily or creasote (gr. $\frac{1}{2}$—0.0324) thrice daily. Germain Sée has recently found strontium bromid (ʒss to ʒj—2.0–4.0) to be of great value in cases in which gaseous fermentation with hyperacidity is combined with permanent tenderness. Happy results often follow a course at some spa if the patient be under the charge of a competent physician during his sojourn. The robust or plethoric should go to Carlsbad, Ems, and Kissingen abroad, and to Saratoga at home, using more especially the Hawthorne water. The anemic should go to Franzenbad and to the iron springs at Bedford, Pennsylvania. A course of the alkaline mineral waters may be successfully taken at home in many instances, though patients are much more apt to obey the physician's injunctions as to diet, exercise, and the like when at a spa than when at home. These waters do not simply act as purgatives, but also as antacids. It has been experimentally shown that sodium chlorid, sodium carbonate, as well as carbon dioxid, promote the secretion of the gastric juice. In the more chronic cases belonging to this class or those that have resisted other forms of treatment intestinal complications are usually found. Here the alkaline waters are to be alternated with calomel in small doses, prescribed thus:

℞. Hydrarg. chloridi mitis, gr. ij (0.129);
Sodii bicarb., ʒj (4.0);
Sacchari lactis, ʒss (2.0).
M. et ft. chart. No. xij.
Sig. One, dry on the tongue, four times daily.

I have been in the habit of continuing the use of these powders for several days to one week, then returning to the alkaline waters for a period of two weeks.

In the *mucous* variety of gastric catarrh additional indications for treatment are presented. The chief aim should be to limit, as far as possible, the production of mucus and to cleanse thoroughly the stomach prior to each meal, thus preparing the organ for the reception and better digestion of food. Here, again, at least one pint of hot water, containing the substances before mentioned, should be sipped half an hour before each meal. This mode of cleansing the stomach is usually successful; if unsuccessful, however, it should be supplemented by lavage once daily, employing two or more pints of warm water. The siphon is also quite useful in cases of this sort in which stricture of the pylorus is suspected and when the food is retained in the stomach much longer than the

normal period of digestion; a condition which is enhanced by the mucus collecting upon the food and thus preventing it from being acted upon by the gastric juice. For the same reason absorption is greatly retarded. The therapy of this form of chronic gastritis requires the more potent astringents for the purpose of arresting hypersecretion of mucus. The best way to use these agents is topically. The stomach may be washed (at bed-time or early in the morning) with a 2 per cent. solution of alum or a 1 per cent. solution of tannic acid; antiseptic solutions are employed in like manner, a 2 per cent. solution of salicylic acid being especially efficacious. If lavage cannot be practised, such astringents as catechu, cerium oxalate, and silver nitrate, with small doses of opium (*vide supra*), should be tried. For use internally, one of the best remedies is atropin sulphate.

Certain symptoms belonging to all varieties of the affection may demand relief. These must be met in accordance with general principles. Vomiting, which is at times a distressing symptom, is best allayed by small doses of resorcin or creasote in combination with cerium oxalate.

As soon as the morbid irritability of the stomach has been reduced mild forms of bitter tonics, with a view to imparting vigor to the digestive organs, may be cautiously employed. Their too early use is very apt to aggravate existing symptoms, or even to reproduce such as have already disappeared. Iron is often indicated during convalescence.

GASTRIC ULCER.

(*Simple or Round Ulcer of the Stomach.*)

Definition.—An ulcer presenting sharp borders, with a tendency to extend in depth, generally without collateral inflammation, giving rise, usually, to one or more characteristic symptoms, as pain, vomiting, and hematemesis. Peptic ulcers may be single, but are oftener multiple.

Pathology.—The gross anatomic characteristics and peculiarities may be briefly considered seriatim. (*a*) In *shape* it is usually round or oval. Frequently there are several ulcers, and these may unite to form larger ones having irregular borders. They are at first superficial, though their floor (when seen at autopsy) is below the mucous membrane. Thus, the ulcer has for its base frequently the muscular or serous coats, but sometimes the ulcerative process extends through the walls of the stomach ("perforating ulcer"), in which case adhesions form between the stomach and the adjacent viscera, one or other of the latter organs occupying the base of the ulcer. The walls usually slope inward, giving rise to the characteristic funnel-shape. The edges may, however, be sharp and abrupt. The floor of the ulcer is quite generally clean. A recent ulcer presents clean-cut edges that are not the seat of collateral inflammatory edema, though an old ulcer often presents thickened margins. (*b*) The *size* is quite variable. The majority of the ulcers are not larger than a dime; others may measure as much as 10 cm. (4 inches) in their greatest diameter. The edges are almost invariably formed from the coalescence of two or more smaller ones. (*c*) The *position* is

most frequently near the pylorus on the posterior wall, and particularly in the vicinity of the lesser curvature.[1] This is the point of greatest irritation from the moving mass of gastric contents which the disturbed muscular mechanism ejects before they have become reduced to a liquid (Barker). Fortunately they rarely occupy the anterior surface.

The deeper ulcers heal by cicatrization. The resulting scar is pale and stellate, and there is puckering of the surrounding mucous membrane. If the ulcer has not extended deeper than the mucous membrane, granulation-tissue develops from the edges and base; this tissue slowly contracts, uniting the margins without a distinct scar. On the other hand, if the ulcer be large and involve the muscular and serous coats, stricture of the pylorus, followed by dilatation, may result. The stomach may present an hour-glass shape, due to the contraction of a girdle ulcer in the central part of the organ. Nearly all gastric ulcers would perforate the coats were it not for the development of a localized peritonitis with the establishment of protective adhesions. The ulcers being usually situated on the posterior wall, the surface of the pancreas forms the point of attachment most frequently, though the stomach may also become adherent to the left lobe of the liver, the spleen, omentum, diaphragm, or the transverse colon. The organs with which the stomach becomes agglutinated may be penetrated by the ulcerative process, resulting in suppurative inflammation (*abscess*); or, guided by the limiting adhesions, fistulous connections of the stomach with the transverse colon, the pleura, the pericardium, lungs, gall-bladder, and the duodenum may be established. Of these, gastrocolic fistulæ are the most common. The ulcer has perforated the left ventricle. Penetration of the ulcer through the posterior gastric wall opens the lesser peritoneal cavity, in which case the base remains limited, producing a condition known as subphrenic pyo-pneumothorax. When the anterior surface of the stomach, which has no anatomic relations with other organs favorable for the establishment of protective adhesions is perforated, general infectious peritonitis rapidly supervenes. Intense hyperemia or the erosion of small vessels gives rise to small or moderate hemorrhages. If the ulcer penetrate one of the larger vessels, then fatal hematemesis is the usual result. The development of a "protective thrombosis" may prevent this accident. In several instances small aneurysms have been found at the bases of the ulcers (Douglas, Powell, Welch).

Frequency and Etiology.—The prevalence of gastric ulcer in the various countries is shown by the statistics of C. P. Howard; he analyzed the records of 161,599 cases treated in American hospitals, and found 930 instances in which gastric ulcer was present (0.57 per cent.); Bromwell, of Edinburgh, in 43,357 cases, found 2.02 per cent. to suffer from gastric ulcer. The percentage for London is 1.24 per cent. lower than that given for Edinburgh; Breslau 0.66 per cent.; Berlin 1.33 per cent. Concerning its pathogenesis, there are two points that are generally accepted: (*a*) that the ulcer is due to a self-digestion of a circumscribed portion of the stomach; (*b*) that the resistance of the part

[1] Of 793 cases collected by Welch from hospital statistics, 288 were on the lesser curvature, 235 on the posterior wall, 95 at the pylorus, 69 on the anterior wall, 50 at the cardia, 29 at the fundus, 27 on the greater curvature. MacNevin and Herrick noted the location of the lesion in 97 fatal cases, as follows: lesser curve 47, posterior wall 30, anterior wall 17, and greater curve 3.

digested has been previously reduced or even lost. Diminished or lost resistance may be due to a lessening of the supply of alkaline arterial blood, which prevents the stomach from being digested in health; also, to *embolism* and *thrombosis* of the nutrient artery of the part, the infarct thus produced being annihilated by the gastric secretions (Virchow). Bassler thinks the lack of gastric mucus, which protects the glandular elements in hyperchlorhydria, is a factor. The experiments by Panum and Cohnheim show that ulcers produced artificially tend to heal rapidly. Stockton holds that the disease is a neurosis. Traumatic injuries have been suggested. It is probable that microbic invasion has not received sufficient attention in the past as an etiologic factor. The gastric juice, while bactericidal, does not afford universal protection. Turck claims that round ulcer of the stomach and duodenum can be produced in dogs by feeding the colon bacillus.

Predisposing Causes.—Hyperacidity of the gastric juice is doubtless most influential—a condition almost universally present in this disease; although the ulcers may not result primarily from the presence of an excess of acid, it is quite probable that further extension of the ulcerative process may be due to this factor. Peter assumes the cause of simple ulcer to be gastritis. It rarely follows cutaneous burns and also wounds of the bladder. The affection is often secondary in chlorosis, anemia, and amenorrhea. The fact that in all the different forms of anemia there is a diminished alkalinity of the blood is of great interest in this connection. Obviously, then, ulcer occurs more frequently in females than in males. It is most common between seventeen and thirty-five years; it is rare in young children, though Gorgart saw an instance in a child thirty hours after birth, and less rare in those past middle life. Cackovic states that the age was under ten years in 2.32 per cent. of 172 operative cases. It is more frequent in the poor than in the rich; occupation has also a noticeable influence, and I have personally seen instances in weavers. It is also prone to attack servants, cooks, and needlewomen among females, and shoemakers, tailors, saddlers, and carpenters among males. Exner has discovered that gastric ulcers are frequent in tabetics.

Clinical History.—In *typical cases* of gastric ulcer the clinical symptoms are almost positively diagnostic. The earliest manifestations commonly point to chronic or subacute gastric catarrh, these being followed, soon or late, by those that are characteristic, as *pain, vomiting*, and *hematemesis*. Of these, pain is most constantly present, and presents certain peculiarities that demand rather elaborate mention. It is commonly dull, at times burning, and is associated usually with great oppression. The character of pain that is most diagnostic is an *intense gnawing, burning* or *boring* in the epigastrium, more or less *periodic* and *strictly localized* in a circumscribed area. These paroxysms usually come on almost immediately after eating, occasionally one or two hours later, and disappear quite promptly when the stomach is emptied either by vomiting or by its contents passing into the duodenum. From the time of its development, the quality, and strict localization of the pain, it may safely be assumed that it is due to direct irritation, set up by the food, of the sensory fibers occupying the base of the ulcer. In addition, there are paroxysms of diffuse pain (*gastralgia*) there are often strictly intermittent, though not necessarily excited by the partaking of food. This pain is due to a sympathetic nervous disturbance or reflected irritation.

Finally, sharp, intense, lancinating pains, that are caused by local or general peritonitis, may appear suddenly. The pain in round gastric ulcer is greatly modified by numerous conditions. The *effect of taking food* has been already referred to, and it should be added that indigestible, imperfectly masticated, and highly spiced food, sweet and hot substances, cause the paroxysms to be more intense than do less irritating articles of diet. *Rest* diminishes the pain by preventing traction on the ulcer. *Certain postures* may aggravate it, and, while not a trustworthy guide, we may often determine the situation of the ulcer by the effect of posture after taking solid food. The severity of the pain is often increased by bodily fatigue or even moderate exercise and emotional influences. The situation of the pain, when strictly localized, is of the utmost importance in diagnosis. I have found it almost invariably from one to two inches below the ensiform cartilage, yet it has rarely been observed in the umbilical and hypochondriac regions. It is absent in one-half of all cases. There is a pain-point in the dorsal region (often at a level with the tenth to the twelfth thoracic vertebra) on the left side. Says Moullin: One special symptom that indicates the spread of ulceration is persistent cutaneous hyperesthesia in Head's epigastric triangle and at the dorsal pain-point.

Vomiting, next to pain, is the most frequent symptom, but unless the vomitus contains macroscopic blood, which is present in less than 50 per cent. of all the cases, or occult blood (*vide* p. 832), it has little diagnostic importance. Nausea and eructations of acid or food often precede or accompany the emesis. Vomiting usually occurs about two hours after eating, often at the height of the paroxysm of pain, which the vomiting relieves as a rule. The *vomitus* and *gastric contents*, as first shown by Riegel, commonly contains an increased proportion of HCl (hyperacidity). The total acidity is abnormally high. The withdrawn test-meal usually contains blood, either macroscopic or occult. The acidity is reduced with the age of the patient and chronicity. Kemp confirms Rubow's assertion that the amount of residue is large and extremely acid.

Hematemesis is a symptom of unequalled clinical significance, and on it alone frequently rests a positive diagnosis. When the hemorrhage is considerable, pure blood, more or less clotted, may be ejected, this being highly characteristic of gastric ulcer. Frequently, however, the blood oozes gradually into the stomach and mingles with the gastric juice, and in consequence the oxyhemoglobin of the blood is converted into hematin, the vomitus presenting the appearance of coffee-grounds. On *microscopic examination* large and small granules of blood-pigment are seen, but the red cells are incapable of recognition.[1] Vomiting of blood may occur at intervals of a few hours or on each successive day. The *amount* also varies within the widest limits according to the size of the vessel eroded. Some of the effused blood passes through the pylorus, escaping with the feces and giving to the latter a tarry, black appearance. A few cases have been reported in which all the blood was evacuated with the stools except that which was absorbed from the alimentary tract. Steele claims that gastric ulcers do not bleed as often as might be expected, and that in dubious cases the stools must be examined for several weeks before chronic ulcer could be excluded. *Intermittent* hemorrhages, how-

[1] The blood, however, can be identified by the guaiacum and other chemical tests and through its spectroscopic appearance.

ever, point strongly toward ulcer. Either as the result of a single copious hemorrhage or of repeated smaller bleedings a *pronounced anemia* is produced. As a rule, however, the evidences of anemia are only moderately well marked, and to assume that the anemia is due solely to the hemorrhages would be an error. A slight rise of temperature is often observed under these circumstances; this is the so-called *anemic fever*. The pain and the most unpleasant local symptoms have been frequently observed to disappear after its cessation. Both cardiospasm and pylorospasm, as shown by radioscopy, are not infrequent accompaniments. The appetite may be good, but the patient is disinclined to eat, owing to the pain resulting therefrom. Not infrequently convalescence sets in immediately.

Physical signs are few and slight. On *palpation* tenderness is found, though not in all cases. The spot of localized agonizing pain before alluded to is often excessively tender on pressure—a valuable sign. The true gastralgic attacks are at times relieved by making firm pressure with the broad hand over the epigastrium. Near the pyloric end of the stomach palpable tumors may be felt, due to the thickened floor of the ulcer. When these indurated masses become adherent to adjacent organs—the pancreas, for example—epigastric tumors of considerable size may be felt, suggesting the presence of carcinoma. *General symptoms* often do not appear until late in the disease. Anemia is usually noted first, to be followed by debility and emaciation; the degree of the general disturbances is in direct proportion to the severity and duration of the coexisting catarrh, hemorrhages, pain, and vomiting. The cachexia may be pronounced, and the face, on account of the prolonged suffering, assumes a gaunt appearance.

Other Clinical Forms.—These have been subdivided into numerous types, some of which merge into one another and cannot be separated clinically. The following atypical forms should be distinguished: (*a*) Latent ulcers, whose existence is not suspected during life, but which are revealed, should they come to autopsy, as open ulcers or cicatrices. (*b*) An explosive form, in which the ulcer may or may not give rise to gastric disturbances prior to the occurrence of perforative peritonitis. (*c*) A recurrent form, described by Welch thus: "In this the symptoms of gastric ulcer disappear, and then follow intervals, often of considerable duration, in which there is apparent cure, but the symptoms return, especially after some indiscretion in the mode of living. This intermittent course may continue for many years. In these cases it is probable either that fresh ulcers form or that the cicatrix of an old ulcer becomes ulcerated."

Complications and Sequelæ.—Perforation of the ulcer (most common when it is situated in the anterior wall) leads to peritonitis, which almost always ends fatally. Rarely a localized peritonitis is the result, owing to rapidly formed limiting adhesions or perforation into the lesser peritoneal cavity. The symptoms of this complication will be given in their proper place (see also *Pain*, p. 806). Hemorrhage may prove a serious complicating accident, being in not rare instances an immediate cause of death. Severe hemorrhage may also cause a diminution of free HCl, with an amelioration of the symptoms. Parotitis, due to oral starvation and multiple neuritis, have been noted.

The cicatrization of an ulcer may lead to *hour-glass stomach*, which presents features as follows. "(1) In washing out the stomach part of the

fluid is lost. (2) If the stomach is washed clean, a sudden reappearance of stomach-contents may take place. (3) 'Paradoxical dilatation' when the stomach has apparently been emptied, a splashing sound may be elicited by palpation of the pyloric segment. (4) After distending the stomach a change in the position of the distention tumor may be seen in some cases. (5) Gushing, bubbling, or sizzling sounds are heard on dilatation with carbon dioxid at a point distinct from the pylorus. (6) In some cases, when both parts are dilated, two tumors with a notch or sulcus between are apparent to sight or touch" (Moynihan).

Diagnosis.—The typical cases in which the characteristic symptoms above mentioned are conspicuous are easy of diagnosis. Hemorrhages occurring with gastralgic attacks are almost pathognomonic. A considerable proportion, however, offer formidable difficulties. Without the presence of hematemesis, visible or occult, for example, a positive diagnosis should not be made, and yet this symptom does not appear in 50 per cent. of all cases. In the absence of hemorrhage we may, however, infer the altogether probable existence of ulcer if there be a history of the more important etiologic factors; if there be gastralgia, hyperacidity, local pain and tenderness, a dorsal pain-point; and, particularly, if the latter symptoms be aggravated by the taking of food. The long course and liability to remission are strongly confirmatory.

Differential Diagnosis.—This disease may be mistaken for *gastralgia*, *chronic gastritis*, *the passage of gall-stones*, *cirrhosis of the liver*, and *carcinoma of the stomach*. The differentiation of the latter complaint will be given later. (*a*) In certain cases of cirrhosis of the liver hematemesis is met with, but here there is absence of all the other characteristic symptoms of ulcer, and the presence of a group of symptoms and physical signs pointing to disease of the liver. (*b*) Hepatic colic simulates ulcer of the stomach without hemorrhage. The sudden onset, the longer duration of the attack of pain, its sudden complete cessation, the presence of jaundice and certain physical signs presented by the liver, often suffice to distinguish this affection from gastric ulcer. The urine may contain pepsin in ulcer, and the administration of orthoform (Hemmeter) will relieve the gastric pain but not that of cholelithiasis. (*c*) Chronic gastric catarrh with hematemesis resembles ulcer of the stomach in many particulars. The great diminution in the proportionate amount of hydrochloric acid found in chronic gastric catarrh and the increased amount in gastric ulcer help materially in discriminating these two diseases. When associated with one another my observation teaches that there is an excess of HCl present; hence a proportionately diminished amount of HCl probably argues against the presence of ulcor. The vomiting in ulcer is combined with severe paroxysms of pain; not so in chronic gastritis, and the vomit in the former contains larger quantities of blood than in the latter disease. (*d*) Doubtless ulcer of the stomach has often been mistaken for neurotic gastralgia, and the discrimination cannot always be accomplished to a certainty.

Gastric Ulcer.	Gastralgia.
History of certain occupations, taking of hot drinks or irritants, as acids or alkalies, chlorosis, amenorrhea, tuberculosis, and heart diseases common.	History of neurasthenia, neuralgia, and hysteria the rule.

Gastric Ulcer.	Gastralgia.
Most frequent from fifteen to thirty-five years of age.	Most frequent before or near the menopause (in the female).
The paroxysms of pain usually come on at a definite period after eating.	Paroxysms more frequent when stomach is empty and show less periodicity.
Eating rarely relieves pain.	Eating usually brings relief.
Position of patient may relieve pain.	No decided relief.
Tenderness on pressure over a certain limited area in the epigastrium.	Tender spot absent. General hyperesthesia of the skin of epigastrium often present.
Pressure usually aggravates, and only occasionally relieves patient during paroxysm of pain—not during the intervals between seizures.	Pressure almost always relieves the pain.
In the intervals gastric disturbances, more or less severe, are present.	In the intervals between attacks no gastric disturbances present, as a rule.
Hematemesis present in nearly one-half of the cases.	Hematemesis absent.
General health often much impaired, particularly late in the affection.	General health less affected than in ulcer.
Physical signs of a mass may be present.	Signs of tumor always absent.
Dilatation may coexist in the late stage.	Dilatation never present.
Hyperacidity of gastric juice usually present.	Hyperacidity present only in certain forms (*supra*).
Improvement follows rest and regulation of diet.	Regulation of diet has no effect.

The **prognosis** is obviously uncertain. The average mortality is about 15 per cent. Such grave complications as free bleedings and peritonitis have been discussed sufficiently in the Clinical History. Among serious thoracic complications, pneumonia, tuberculosis, and left-sided perforative empyema are those most frequently encountered. The more recent the case the better the prospect of recovery. The possibility that the resulting scar may cause persistent gastralgia, and the probability that a cicatrix surrounding the whole or any part of the pylorus may cause obstruction, followed by ectasy, must be remembered. Carcinoma often develops in the floor of an old ulcer (*vide* p. 813). Innately, the disease is an exceedingly chronic one, often lasting several, and sometimes ten or fifteen, years.

Treatment.—The treatment of simple gastric ulcer embraces three leading objects: (1) Of paramount importance is *absolute rest for the stomach*. This is to be accomplished by maintaining the recumbent posture in bed, on the one hand, and by rectal feeding, wholly or partly, on the other. This mode of alimentation will be discussed presently. Perfect rest constitutes the best-known safeguard against those serious accidents that intervene suddenly in the course of this affection. It also ensures more rapid cicatrization than any other single agent. The process of repair is very slow under the most favorable circumstances; hence the patient should be informed at the outset that from six to eight weeks, at least, must be spent in bed. (2) *The careful regulation of the diet.* It is not possible for the stomach, when the seat of ulcer, to digest the normal amount of nitrogenous food without being injuriously affected thereby. Those articles of diet should be employed that are digested and assimilated chiefly in the intestinal tract. But, though the patient is fed by the mouth, this should be supplemented by rectal feeding almost from the beginning. By pursuing this combined method and giving per rectum but a limited amount of albuminous food the vital

forces can be more effectually supported. Failure to cure cases of gastric ulcer is often due to the fact that the patient's general strength early becomes exhausted. Frequently the stomach is so irritable as to render it exceedingly difficult to introduce into it even a fractional part of the amount of food necessary to support life properly; in such cases "a period of absolute abstention from food by the stomach should be inaugurated" (Lambert). Nothing but water and pieces of ice should be allowed. Exclusive rectal feeding during the first week is a method quite commonly adopted. The following dietary will be found useful: At 7 A. M. give 100 c.cm. (℥iij) of Leube's beef-solution; at 11 A. M., 200 c.cm. (℥vj) of pancreatized milk-gruel;[1] at 3 P. M., 200 c.cm. (℥vj) of peptonized milk or skimmed milk or buttermilk; at 7 P. M., 200 c.cm. (℥vj) of pancreatized milk-gruel; in addition, the following by rectal injection: at 8 A. M., 6 ounces of pancreatized milk-gruel, with ½ ounce of bovinin, and, if necessary to overcome rectal irritability, 5 to 10 drops of tincture of opium, this to be repeated at 2 and 8 P. M. If the nutrient enemata must be discontinued for a time, the regular diet must be increased proportionately. If, on the other hand, the stomach rejects the above-mentioned food, then the feeding must be, for a time, exclusively rectal; this is quite practicable if the proper choice be made of nutrient preparations. In addition to the substances before mentioned we may employ from 4 to 6 ounces (150–200 c.cm.) of Leube's beef-solution, or the same amount of defibrinated blood or pancreatized milk with brandy.

Lenhartz treated 295 cases of gastric ulcer with a more nourishing diet than that allowed by von Leube, with a mortality of 2.3 per cent. Da Costa reported recently a number of instances that were cured by a diet of ice cream. Senator advises the use of gelatine as food in gastric ulcer. Owing to the abnormally free secretion of HCl in this disease, the proteins should be limited in the dietary, while carbohydrates are indicated, physiological investigations having shown that the latter (also fats) diminish the secretion of the normal acid. It has been recommended to employ lavage when the stomach is exceedingly irritable, but the use of the stomach-tube is liable to damage the ulcer even in the most careful hands. The good effects from washing out the stomach for uncontrollable vomiting and pain have, however, been frequently witnessed. It may often be accomplished by the use of 1 pint (½ liter) of warm water containing a few grains of sodium bicarbonate, sipped slowly when the stomach is empty. If at the expiration of two months the condition of the patient indicates that the reparative process is far advanced, then well-boiled rice, stale bread, and potatoes may be allowed; and later eggs, oysters, fish, and sago, while an ordinary solid diet should not be resumed for at least six months.

(3) The *medicinal treatment*, which is altogether subsidiary to the dietetic, has reference to two ends: (*a*) Promotion of the healing process. We cannot be certain that any known remedial agents can accomplish this object, yet it is our duty to attempt it. Of the efficacy of alkaline remedies we are thoroughly convinced; in neutralizing the hyperacidity of the gastric secretions they fulfil an important indication, since the excess of HCl must have an unfavorable effect

[1] The milk-gruel is prepared with wheaten flour or arrowroot, mixed with an equal quantity of milk.

upon the ulcer. Of these, sodium bicarbonate (in full doses) or the alkaline purgative mineral waters, as Carlsbad, Kissingen, Hunyadi János, are most useful. The Carlsbad salts are beneficial, and may be prepared artificially as follows: sodium sulphate, 50 parts; sodium bicarbonate, 6 parts; sodium chloride, 3 parts—of which a teaspoonful may be taken in hot water, fasting, in the morning. The alkaline waters must not be allowed while the stomach is at perfect rest. The preparations of bismuth may be given in combination with antiseptics, which latter are especially to be recommended. Fleiner's method of giving 10 gm. of bismuth in 200 gm. of warm water on an empty stomach, then allowing the patient to drink several swallows of water, and afterward placing him in the horizontal position with the hips elevated for about an hour, has yielded gratifying results. About 200 gm. of bismuth administered in the above manner usually suffice to effect a cure (Savelieff). For the chronic gastric catarrh which may be associated with ulcer, silver nitrate is efficient, and may be combined with small doses of opium or hyoscyamus. The previous general condition of the patient is frequently unfavorable to the successful healing of the ulcers, and to combat the anemia and chlorosis that are often present we may employ iron and arsenic. Small doses of Fowler's solution of arsenic are generally well borne by the stomach; the former may also be given hypodermically. When organic cardiac diseases are concomitants they should receive careful attention, and also any other associated conditions.

(*b*) The relief of symptoms. The extract of opium, combined with silver nitrate, often relieves the pain. Mild counter-irritation is also of service, but not warm poultices. The application of the ice-bag sometimes alleviates the pain, but Hemmeter advises orthoform. For the gastralgic attacks morphin may be required. For *vomiting*, bismuth, creasote, silver nitrate, and opium are useful; chipped ice, with a small amount of brandy thrown over it, is also of value. When obstinate, the following remedies may be tried separately: cerium oxalate, potassium bromid, tincture of iodin, cocain, chloral, and hydrocyanic acid.

For the *hematemesis*, rest, rectal feeding, the application of a broad, flat ice-bag, and the use of morphin or ergot hypodermically, will usually suffice. For exhaustive hemorrhages infusion into the veins or into the subcutaneous tissue of normal salt solution is an important measure. For stopping a hemorrhage, lavage followed by bismuth is highly recommended. Operative intervention in gastric ulcer is demanded: 1. In recurring hematemesis, W. L. Rodman advises operation between attacks—always after the third bleeding; 2. In perforation, so soon as the diagnosis is clearly established. In the cases of perforation which have been operated upon within the first twelve hours during the past three years, 83.78 per cent. have been saved (Tinker). 3. Most of the cases not cured by medical treatment are savable by timely surgical intervention, but in simple ulcer of the stomach operation is not advised, "the medical treatment of which should be more careful and more prolonged than was formerly deemed necessary" (Robson[1]). 4. If gastrectasis due to pyloric obstruction or if adhesions form and persist, operation is indicated. The treatment of callous ulcer is purely surgical.

[1] *British Medical Journal*, Nov. 17, 1906.

CARCINOMA OF THE STOMACH.

Pathology.—Next to the uterus, the stomach is the most favored seat of carcinoma. In a total of over 30,000 cases studied by Welch, 21.4 per cent. showed involvement of this organ. With reference to the parts of the organ attacked, Welch analyzed 1200 cases with the following results: pyloric region, 791; lesser curvature, 148; cardia, 104; posterior wall, 68; greater curvature, 34; anterior wall, 30; fundus, 19. The forms of gastric carcinoma noted are columnar epithelial (including colloid) and the glandular carcinomata (embracing encephaloid and scirrhous). The epitheliomata grow from the lining epithelium, while the encephaloid and scirrhous are new growths from the glandular epithelium. The last two forms are, therefore, similar in structure, but differ in the rapidity of their growth; the encephaloid cancers are soft, and readily break down on their surface, forming large ulcers that have a clean floor, while the scirrhous cancers are hard and firm. Columnar epitheliomata are frequent, and are situated at the pyloric end of the stomach. They are often the seat of colloid degeneration. Squamous epitheliomata occur at the cardiac end. Secondary new growths in adjacent organs occur, the scirrhous, however, manifesting the least tendency to metastasis. Perforation of the stomach-walls occurs in 3.3 per cent. (Brinton). Welch collected 37 cases of secondary gastric carcinoma; 17 were secondary to mammary carcinoma. Atrophic gastritis ensues.

Etiology.—The factors bearing upon the etiology of gastric carcinoma may all be regarded as **predisposing causes.** Of these *age* is the most potent. Of 2038 cases examined by Welch with reference to this point, 75 per cent. occurred between the fortieth and seventieth years, 24.5 per cent. between forty and fifty years, 30.4 per cent. between fifty and sixty years, and 2.8 per cent. before the thirtieth year. The maximum liability lies between the forty-fifth and sixtieth years (Lebert). In 1069 cases collected by Osler and McCrae,[1] 2.5 per cent. developed before thirty years of age. There are records of 6 cases before the tenth year. Collingsworth reports the case of a child at ten days and death at twenty-ninth day; and Widerhofer 1 at sixteen days. I find records of 13 cases between ten and twenty years. *Heredity* stands next to age as a causal factor, though it is far less influential. Welch analyzed 1744 cases, and found that a family history of carcinoma was present in about 14 per cent. *Sex* has little if any influence. The colored *race* enjoys comparative immunity. Gastric carcinoma may follow a pre-existing chronic catarrh. More commonly, however, chronic ulcer precedes. In Haberfeld's series of 662 autopsies in cases of gastric carcinoma, 106, or 16 per cent., showed macroscopic evidence of having originated in round ulcer. Sonnichsen, quoted by Rodman, found that out of 156 cases of gastric carcinoma which came to autopsy, 22, or 14 per cent., developed in the scars of ulcers, while the statistics of Klausa (126 cases) give more than 26 per cent. that grow from either ulcers or cicatrices. The disease is most probably of bacterial origin.[2] The disease is rare in the tropics.

[1] *New York Medical Journal*, April 21, 1900, p. 581.

[2] "The Nature of Carcinoma," by the writer, *New York Medical Journal*, Nov. 21, 1908.

Clinical History.—Prior to the development of gastric carcinoma the symptoms of catarrhal dyspepsia may be present for a variable period of time. The onset, however, is oftener abrupt. Again, it may be insidious, and be marked more by the evidences of failing general health and strength than by distinct local subjective symptoms. Osler and McCrae[1] have reported cases of latent carcinoma of the stomach. A *progressive decline of the appetite* is generally observed, though occasionally it remains unimpaired. A sense of oppression, rarely true cardialgia, and eructations (*pyrosis*) come on soon after eating. In many cases but little *pain* is complained of, while in a lesser number pain is a prominent symptom throughout the entire course. Its character is very often described as lancinating, less often as burning or gnawing; the latter form of pain is due, most probably, to associated secondary ulcers. The pain is often referred to the shoulders and the back or loins. *Vomiting* is infrequent, except in the more advanced stages of the disease, when it is almost constantly present to a greater or less degree. During the early stages it is due to the catarrhal irritation, later to obstruction. When the latter is at the cardiac orifice, the pain occurs at once after eating; when at the pylorus, it appears several hours after meals. Vomiting may also be caused by the occurrence of fermentation in large accumulations. The *vomitus* has few, if any, of the physical characteristics noted in simple ulcer of the stomach. Free hematemesis is very rare; when, however, the surface of the new growth ulcerates, there is almost invariably an occasional slow oozing of blood into the stomach. It is here acted upon by the altered gastric juice, and the black hematin resulting from the transformation of the red hemoglobin gives rise to the well-known "coffee-ground" vomit of carcinoma of the stomach.[2] The chocolate-colored appearance of the vomitus is not found alone in carcinoma of the stomach. Small hemorrhages are more common in ulcerated carcinoma than in gastric ulcer.

The *chemical examination* of the aspirated stomach-contents is of prime diagnostic importance, showing as it does the almost constant absence of free HCl after the *test-meal* (see p. 778). The presence of free HCl, supposing the examinations to be properly made (by the use of the color-test) and sufficiently often repeated, speaks almost positively against carcinoma. In not one of 154 artificial digestive experiments was albumin digested in this disease. Cases do occur, however, in which free HCl is present, as when carcinoma of the stomach is secondary to an ulcer. Moreover, in the incipient stage of gastric carcinoma a small percentage of HCl is occasionally found. Free HCl is also absent in carcinoma of the esophagus, duodenum, extensive amyloid disease, advanced cases of renal disease, and the febrile state.

The leading view as to the cause of the absence of HCl is that the inflammatory degeneration of the mucous membrane, commencing as a catarrhal inflammation and advancing to interstitial change and atrophy (*Rosenheim's view*), diminishes and finally arrests hydrochloric acid secretion. Moore[3] believes that the non-production of HCl is due

[1] *Philadelphia Medical Journal*, Feb. 3, 1900.

[2] *Teichmann's test* for hematin crystals may be employed as follows: Place a drop of the "coffee-ground" material upon the slide and add a few crystals of sodium chlorid. Then introduce a few drops of acetic acid beneath the cover-glass and warm.

[3] *The Lancet*, i., 1120, 1905.

to the relative diminution of H. ions and an increase in the OH. ions and alkalinity of the blood. *Lactic acid* in excess occurs in the stomach-contents after a test-meal in carcinoma. Sick[1] concludes that the most important factor for lactic acid fermentation is the soluble albuminoids produced by the carcinoma (autolysis). The *microscopic appearances* of the vomitus and wash-water are in some ways identical with those observed in gastric ulcer, and if they be examined speedily, red blood-corpuscles may rarely be seen. The constant finding of *occult blood* with the guaiac test has great significance. Invisible hemorrhage in the stool is a fairly constant finding. The microscope, also, very seldom reveals pieces and bits of cancer-tissue, and Kaufmann and Hemmeter emphasize the frequency of long bacilli, the latter observer finding the Boas-Oppler bacillus in 94 per cent. of cases. Riegel states that sarcinæ are infrequent. Both the proteolytic and amylolytic power of the stomach are greatly diminished. The presence of pus in the gastric contents is confirmatory of carcinoma. The working power of the stomach is defective at an early stage—an important diagnostic feature. Kemp found a small amount of relics of food in the fasting stomach twelve hours after eating as a constant sign.

Physical Examination.—*Inspection* may reveal an irregular tumor in patients much emaciated. When dilatation exists, the outlines of the organ may be seen. On *palpation* the new growth, in a majority of cases, may be felt through the abdominal walls, though often not clearly, as a hard, nodular, and sometimes movable mass. Though this generally appears in the epigastrium, it must be recollected that it depends upon the part involved; also that a tumor united with the wall of the stomach, particularly if situated at the pylorus, sags downward, even to a point below the umbilicus. Less frequently it is discovered in such unlooked-for situations as the right or left hypochondriac region. Varying degrees of fulness of the stomach will alter the position of the tumor. When situated at the cardia it is beyond reach; when attached to the lesser curvature of the stomach or the posterior wall, it is rarely to be felt unless of large size. The new growth cannot be definitely made out when it assumes the form of a diffuse infiltration, though it offers increased resistance and exhibits tenderness on pressure. Usually the patient lies in the dorsal position during the examination, with the limbs drawn up, breathing regularly, while the mouth is kept open. The detection of a tumor when in an unfavorable situation may be facilitated by shifting the patient's position from the dorsal to the lateral, the standing, or the knee-elbow position respectively; at the same time one or two tumblers of some carbonated water should be given with a view to distending the stomach and carrying the tumor downward. Pulsations are frequently communicated from the aorta to the palpating hand through the tumor. If the growth is situated at the lesser curvature, a deep inspiration will often cause it to fall lower and become accessible to palpation. *Percussion* over the growth causes a muffled tympanitic resonance; superficial percussion, however, may give dulness.

The presence of *metastatic new growths* in the liver and enlargements of the supraclavicular or inguinal lymph-glands are of value in the diagnosis. In one instance that I saw in the Philadelphia Hospital a nodule

[1] *Deutsch. Archiv f. klin. Med.*, Berlin, vol. lxxxvi., Nos. 4 and 5, 1906.

the size of a walnut protruded from the umbilicus, leading to the suspicion of gastric carcinoma. Subsequently a nodulated pyloric neoplasm could be readily held in the grasp. Boas contends that enlargement of the supraclavicular glands has no value as an early indication, since he has never observed this symptom in the early stages.

General Symptoms.—Quite early in the disease such evidences of general nutritional disturbance as loss of flesh and anemia may be observed, and, obviously, cases attended with constant anorexia and vomiting will earliest manifest the wasting process. Almost from the beginning the face gradually assumes the cachectic appearance which, in the advanced stages, becomes so characteristic of gastric carcinoma. Anemia soon becomes a prominent feature. There is a waxy pallor of countenance, and the cerebral symptoms as well as the peculiar cardiac murmurs of anemia appear. The blood frequently presents peculiarities that bear a resemblance to those seen in pernicious anemia, and at times such bloods are indistinguishable from those of true pernicious anemia. I have observed leukocytosis late during the course of this malady. A differential study of the leukocytes is of no value, and the presence or *absence of digestive leukocytosis* is too uncertain to be of diagnostic importance. The blood-count is usually that of secondary anemia. Nucleated red cells (normoblasts) are not uncommon, and myelocytes are occasional findings. The anemia of cancer is accompanied by loss of flesh, while in pernicious anemia the superficial fat is preserved. The causes of the profound anemia met with in this affection are not quite plain, since frequently it becomes pronounced before the nutritional disturbances (shown by a loss of flesh) have become marked. The fact that metastatic carcinoma has been found to be abundant in the marrow of the bones is significant in this connection, as pointing to the probable interference, in some instances, with the blood-producing function of the bone-marrow. In advanced cases moderate *edema* of the ankles and of the backs of the hands is frequently observed, and is probably dependent upon excessive anemia. The *temperature* at first shows no abnormalities, as a rule, though after the cachexia has become decided it is often subnormal. Sudden elevations of temperature (103° to 104° F.—40° C.), preceded by rigors and followed by profuse sweating, are rarely observed. The explanation of their occurrence is to be found in the fact that suppuration sometimes takes place in the bases of the cancerous ulcers. The mind almost invariably remains clear to the last, though delirium may be a late-appearing symptom.

Complications.—*Intestinal symptoms* are frequently observed, and *constipation* in particular is quite common. It is apt to alternate with *diarrhea* toward the close of the disease, or diarrhea may in the later stages become a persistent and obstinate symptom. Some of the complicating conditions have reference to the *secondary* new growths. When, as frequently happens, the liver is implicated, *jaundice* is rather common, being associated with signs of hepatic enlargement. Indeed, so prominent may be the symptoms and physical signs referable to secondary carcinoma of the liver as entirely to mask the more or less hidden forms of carcinoma of the stomach. The mesenteric and retroperitoneal lymph-glands or the lungs may be the seat of secondary carcinoma, which, however, rarely gives rise to characteristic symptoms.

Occasionally the new growths spread to the peritoneum (and excite exudation) or to the rectum. Perforation may rarely occur, and we then have the pronounced and rapidly supervening symptoms of diffuse peritonitis.—*Fistulous communications* between the stomach and the transverse colon or the small intestine—the latter rarely—may also occur. *Nervous symptoms* may be regarded as complicating conditions, and sometimes hasten the fatal termination; the patient becomes somnolent or, rarely, even comatose; the breathing is difficult and the respiration deep and labored. This mode of termination I noted in one case. Traces of *albumin*, and in the later stages tube-casts, may be present in the urine. Indicanuria is a rather common symptom, while acetonuria is seldom seen. Diacetic acid is present in rare instances.

Latent Forms.—The disease may be latent, most often in feeble persons and in the aged, and accidentlv discovered on physical examination. In some cases the cachexia furnishes ground for suspicion.

General Course and Duration.—The course of gastric carcinoma is invariably toward a fatal issue, death usually taking place before the expiration of two years. The average duration of the disease is about one year. When it occurs in emaciated persons it pursues a slower course than when occurring in fleshy individuals. Cases develop only slowly in old ulcer cases. The younger the individual the more rapid the course of the disease. The symptoms are far less intense in the cases in which food stagnation is absent.

Diagnosis.—A positive diagnosis of gastric carcinoma is easily made when a tumor is demonstrable. The history, the presence of characteristic symptoms, such as pain, ectasy, coffee-ground vomit, deficient motor power (*early*), the constant absence of free hydrochloric acid, especially the almost constant presence of lactic acid after the Boas test-meal (unfortunately not an early feature) and a constant positive blood reaction in the stools, all occurring in persons beyond middle life, together with the existence of progressive cachexia, are sufficient to warrant a diagnosis in the absence of a palpable new growth. A putrid smelling tube and putrid eructations have diagnostic importance, and Hemmeter states that the early diagnosis of carcinoma of the stomach is possible in a certain number of cases from histologic examination of small fragments of gastric mucosa, if a direct invasion of the gland-substance by epithelial cells is observed. Exploratory laparotomy may be advised if improvement does not follow medical treatment in suspicious cases within a few weeks at most. Moullin has emphasized the diagnostic value of direct inspection through an incision. Kelling's method,[1] or the hemolytic serum test, was applied by Rosenbaum[2] in 70 patients, including 26 of carcinoma; he obtained favorable results. If the reaction be negative, however, cancer cannot be excluded (Gorham and Lisser). The results of recent experiments tend to discredit the reliability of this test. The meiostagmin reaction (Ascoli) has been found to be reliable by certain observers. The glycyltryptophan test is valuable for diagnosis (Weinstein). Goodman's[3] modification of the Salomon test, which shows the percentage of phosphates (over 10 mg. per 100 c.c.) in the wash-water, is corroborative in ulcerative cases. Falk and Salomon's salicylate method

[1] *Arch. f. klin. Chir.*, lxxx., No. 1, Berlin. [2] *Münch. med. Wöchen.*, March 3, 1908.
[3] *Arch. f. Verdauungskr.*, B. xv., H. 4.

is of material aid[1]. Salomon and Saxl[2] have found a reaction of neutral sulphur very constant in carcinomatous patients. Smithies[3] found in 1175 cases this test less constant than the glycyltryptophan reaction. A positive skin reaction is strong presumptive evidence of carcinoma (Lisser and Bloomfield).[4] B. K. Brown found the stools uniformly Gram-positive in a series of cases. An expert *x*-ray examination is an important aid in the diagnosis.

When malignant degeneration of an ulcer sets in, pain increases in intensity and may radiate to the thorax and back, anorexia develops, hyperacidity often gives way to achlorhydria, and hematemesis may occur early and recur at frequent intervals.

Differential Diagnosis.—A gastric carcinoma presenting a discernible mass is liable to be mistaken for a *cicatrized ulcer*, for *carcinoma of the pancreas*, of the *transverse colon*, *duodenum*, *omentum*, and the *left lobe of the liver*, as well as for *aneurysm of the abdominal aorta*. The *aneurysmal tumor*, however, is smooth, and is not nodular, like the cancerous growth, moreover, it gives rise to an expansile impulse. In aneurysm the characteristic cachexia is wanting. In *pancreatic carcinoma* the tumor is fixed (*vide infra*, p. 956). *Carcinoma of the transverse colon and omentum* will be excluded by the presence in malignant disease of the stomach of a chocolate-colored appearance of the vomitus, deficient motility of the organ, the permanent absence of HCl, and persistent presence of lactic acid in the gastric contents. For the recognition of hidden gastric carcinoma with grave anemia the reader is referred to p. 470.

Chronic ulcer may in cicatrizing give rise to a small tumor, followed by pyloric stenosis and secondary dilatation—an exact counterpart of the course of gastric carcinoma. Great reliance should be placed on the age of the patient, the presence of HCl in the gastric secretions, the points of pain (dorsal epigastric) and localized tenderness with hematemesis, and the longer duration of ulcer. Ulcer with tumor-like thickening may show an excess of lactic acid, due to associated motor insufficiency, rendering a differential diagnosis exceedingly difficult. Hypertrophic stenosis of the pylorus is also simulated (*vide* p. 819).

Simple gastric ulcer and chronic gastritis are often confounded with carcinoma of the stomach without palpable tumor (*vide* parallel differential tables below, modified slightly from DaCosta).

Chronic Gastritis.	Gastric Ulcer.	Gastric Carcinoma.
Not confined to any age. More common in middle-aged or elderly people.	May occur in middle-aged persons, but is most frequent in young adults, especially women.	Most common in elderly people; rarely occurs in persons under thirty years of age.
Pain at the epigastrium somewhat augmented by food; soreness is also present. Both are constant, although comparatively slight.	Pain at the epigastrium much augmented by food; subsides when this is digested: paroxysms of pain, not lancinating; strictly localized soreness to touch in epigastrium; sometimes a painful spot over lower dorsal vertebræ. Intermissions in the pain are frequent.	Pain frequently of a radiating kind, often paroxysmal, not infrequently severe and lancinating, but not of necessity associated with soreness. Little or not at all affected by food. Pain rarely remits; never intermits for any considerable time.

[1] *Deutsche med. Wochen.*, Jan. 11, 1912. [2] *Archives of Internal Medicine*, Oct., 1912.
[3] *Centralblatt für die Grenzgebiete der med. und Chir.*, Jena, Aug. 30, 1911.
[4] *Bull. of Johns Hopkins Hosp.*, Dec., 1912.

Chronic Gastritis.	Gastric Ulcer.	Gastric Carcinoma.
Symptoms of indigestion marked.	Symptoms of indigestion sometimes very slight.	Symptoms of indigestion marked. Anorexia; extreme acidity of stomach.
Sometimes vomiting.	Vomiting may be present or absent.	Vomiting a very frequent symptom.
No hemorrhage, or but trifling hemorrhage; at most blood-streaks in vomited matter.	Abundant hemorrhage from the stomach common. Stools may contain blood (tarry).	Hemorrhage not very abundant, but frequently occasioning coffee-ground-looking vomit.
Bowels constipated.	Bowels usually constipated; intermittent occult blood in stools.	Bowels obstinately constipated. Occult blood in feces; more constant.
No fever.	No fever.	Attacks of slight fever occur; temperature often subnormal.
Not so.	Acids taken increase pain.	Not so.
Not much emaciation; no cachectic appearance.	Frequently extreme pallor and debility, especially if preceded by anemia.	Progressive loss of flesh, and cachexia; and at times hypertrophy of the lymphatic glands, especially cervical.
Disease may be relieved or cured; is often of very long duration.	Duration uncertain; may get well, may run on rapidly to perforation; or may last for years.	Average duration one year; may be shorter, but seldom longer.
No tumor.	Rarely a tumor.	Generally a tumor.
Contents of stomach almost always contain free hydrochloric acid.	Hydrochloric acid in excess in contents of stomach.	No hydrochloric acid in contents of stomach.
No lactic or fatty acids after the rigid Boas test-meal.	No lactic or fatty acids after the rigid Boas test-meal.	Lactic acid present after Boas's test-meal.
Slight motor disturbance.	Motor function fair.	Early marked disturbance.
No dropsy,	No dropsy	Edema of ankles common.

Treatment.—The diet should be adapted to the peculiarities of the individual case. Physiology indicates that meat and meat-extracts stimulate the secretion of HCl, hence they deserve a careful trial in the earlier stages. If these fail of their physiologic effect, however, then articles of food that are digested and assimilated in the intestines should be employed. After well-marked evidences of pyloric obstruction appear we may add greatly to the comfort of the patient by limiting the dietary to liquids, and by predigesting them if they are not otherwise well borne. Should the stomach reject all food, rectal alimentation should be promptly instituted. The more troublesome symptoms—namely, pain, vomiting, hematemesis, and constipation—are to be met on general principles. The claims that have been advanced in favor of arsenic and other preparations as possessing power to control the progress of gastric carcinoma await confirmation. Coca and Gilman[1] have used a vaccine in the specific treatment of carcinoma. Neoformans vaccine is also recommended. If dilatation coexists, it is to be managed in accordance with the recommendations found under Dilatation of the Stomach (p. 790). Gastric carcinoma is usually primary and for some length of time it is a local disease. Early surgical intervention, therefore, offers promise of relief and even cure.

Hypertrophic Stenosis of the Pylorus.—By this term is meant pyloric obstruction due to hypertrophy, principally of the circular layer

[1] *Philippine Jour. of Science*, 1910, iv., 391.

of the muscularis with hyperplasia leading to secondary dilatation of the stomach. This may be (*a*) congenital; (*b*) acquired (*e. g.*, tuberculosis). The *etiology* is unknown, although spasm of the pylorus has been suggested. The *symptoms* are those of dilatation of the stomach and a pyloric tumor may be palpable. The resemblance to ulcer with tumor-like thickening and to *ulcus carcinomatosum* may be striking (*vide* also p. 818). Medical treatment—massage, electricity, and strychnin internally—should be tried, this failing; pyloric stretching or other form of operation is indicated.

Benign Cirrhosis of Stomach.—This is a rare condition and difficult of diagnosis. Sheldon states that it may be suspected in patients presenting symptoms of benign stenosis of the pylorus with contracted stomach. The symptoms pointing to it are long-standing disease, absence of hematemesis, contraction of the stomach, absence of tumor on palpation, absence of glandular or hepatic involvement, and general improvement and relief of the stomach symptoms for a period of time when rectal feeding is resorted to. The treatment is necessarily surgical.

Congenital atresia proves rapidly fatal, while the adult form may run a long course. Exceptionally other forms of gastric tumor occur—*lipomata*, *sarcomata* (a total of 56 cases—von Graff), *fibromata*, and *cysts*.

HEMATEMESIS.

Hematemesis is a symptom, hence it is hardly to be properly classed among gastric affections.

Etiology.—Among the causes of hematemesis are—1. Traumatic injury to the stomach; 2. Diseases of its coats (carcinoma, ulcer, miliary aneurysms, acute congestion); 3. A mechanical impediment to the portal circulation; 4. Vicarious menstruation; 5. Alterations in the blood; 6. A disease of some neighboring organ, such as carcinoma of the pancreas, may perforate the gastric coats and open its vessels.

Symptoms and Diagnosis.—If the fact that it is always a symptom, and not a disease, be recollected, the importance of recognizing its causal condition in each instance will be greatly facilitated. The manner of its occurrence and the characteristics presented by the blood often give a clue to its nature and origin. Thus, we have seen that the clinical signs in hematemesis due to carcinoma and ulcer of the stomach vary greatly, being almost peculiar to each. This fact must, however, be weighed with the history and symptoms of the case in which it may occur; in this manner, and in this manner only, can errors be avoided. A process of exclusion is the best way to reach a decision. If a careful inquiry determines the absence of morbid lesions of the stomach, such as carcinoma, ulcer, or chronic gastritis, then the other organs of the abdomen, and more particularly the liver, must be examined. If this and the heart be found to be healthy, attention should then be turned toward the state of the blood, as in the specific fevers. It may also be found that the menstrual or other habitual discharge has become suppressed.

Differential Diagnosis.—It is to be recollected that the source of the blood may be other than the stomach. Rarely, an abdominal aneurysm

bursts into the stomach; occasionally, too, a thoracic aneurysm opens into the esophagus, whence the blood speedily finds its way into the stomach. A careful consideration of the history and of the attending symptoms, together with a thorough physical examination, will, after excluding the various conditions causing true gastric hemorrhage, lead to a correct interpretation of the phenomena. Blood coming from the *throat*, *tonsils*, *mouth*, or the *respiratory organs*, including the nose, is sometimes swallowed, and afterward ejected by vomiting. To discriminate from this condition it is only necessary to make an examination of the lungs and elicit most carefully the history. It must also be recollected that *hysteric females* and *malingerers* have been known to swallow the blood of animals and other dark fluids, and vomit them subsequently. The vomitus may resemble dark blood in appearance when stained by bile or iron or after a free indulgence in wine. The points of contrast between hematemesis and *hemoptysis* are correlatively considered below:

Hematemesis.	Hemoptysis.
The history points to gastric, splenic, hepatic, or cardiac disease, or anemia.	History of cough and other symptoms points to pulmonary or cardiac disease.
A feeling of uneasiness, and sometimes of nausea or faintness, precedes the hemorrhage.	A feeling of weight and uneasiness in the chest, a saline taste, and a tickling in the throat precede the hemorrhage.
The blood is ejected by vomiting; violent vomiting may excite cough.	The blood is raised by coughing or clearing of the throat, though, if it be swallowed, vomiting may follow.
The blood is either clotted or fluid and dark; it may be mingled with remnants of food, and is acid in reaction.	The blood is bright-red, frothy, in small coagula, sometimes mixed with mucopus, and alkaline in reaction.

Prognosis.—Hematemesis, except it be due to rupture of an aneurysm, rarely presents a hopeless prognosis. In cases of splenic enlargement, hepatic cirrhosis, or gastric ulcer, it may prove fatal.

The **treatment** has been detailed in the discussion of Gastric Ulcer. The suprarenal extract has given specially favorable results.

NEUROSES OF THE STOMACH.

NERVOUS DYSPEPSIA.

(*Neurasthenia Gastrica.*)

Definition.—A functional disorder of the stomach, usually characterized by regularly (and sometimes irregularly) recurring attacks of gastric disturbance, followed by almost complete freedom from symptoms. Sensory disturbances of the stomach are constantly present, and with these either motor or secretory disturbances or both may be associated.

Etiology.—The majority of cases occur in highly emotional and hysteric persons, under such exciting conditions as great anxiety, violent passion, dissipation, social excesses, mental overexertion in business life, grievances, and any startling news. The condition is most commonly met with in healthy-looking, ruddy-cheeked adults. It is more common in females. Persons living amid luxurious surroundings suffer most. Gastric neuroses may be of reflex origin, arising from derange-

ment of the nervous system. Deaver[1] states that they may be manifestations of disease in the liver, gall-bladder, bile-ducts, or appendix, which will demand surgical interference.

Symptoms.—The symptoms follow immediately upon the action of the exciting cause and are largely under the influence of the emotions. In the ordinary form the gastric secretions are often normal, and the stomach is found empty after a test-meal within the physiologic time-limit. There is anorexia, which occasionally alternates with a voracious appetite. After meals the patient complains of distress and oppression in the epigastrium; eructations, and an occasional regurgitation of the acid liquid or solid contents of the stomach, with heartburn, will also be noted. Vomiting is not rare, and occurs independently both of the time of eating and of the character of the food. *Gastric peristalsis* is sometimes so well marked as to be readily felt and even visible through the stomach-wall. Kussmaul has called special attention to this symptom, (*vide peristaltic unrest*, p. 826). The increased peristaltic waves excite cooing, gurgling sounds that are a source of annoyance.

The *physical examination* sometimes reveals abdominal distention and hyperesthesia of the surface, but no localized tenderness, pressure with the broad hand usually affording relief from pain. *Nervous phenomena* always exist, and their correct interpretation is of the utmost importance in the diagnosis. Neurasthenic and hysteric manifestations are commonly associated. The mental condition is unstable and illy regulated, and this fact furnishes a satisfactory explanation of the operation of the etiologic factors. The general health is in many instances not noticeably impaired; but in those subject to frequent vomiting and complete anorexia, the general nutrition suffers considerably.

Complications.—The bowels are often constipated, distended with gas, and may be the seat of an abnormal peristalsis. The *course* of nervous dyspepsia is chronic, and it may terminate in catarrh of the stomach.

Nervous dyspepsia with hypochondriasis forms a group of cases in which the hypochondriasis may sustain a causal relation; it may, however, be secondary to the gastric disturbances. It is apt to be marked after the gastric symptoms have lasted a long time. The symptoms other than the nervous are similar to those described above.

Diagnosis.—The diagnosis is based on the following points: (*a*) The etiologic factors. Here it is important to ascertain the particular causative influence that produces the gastric symptoms, taking also into consideration any well-recognized predisposing causes. (*b*) The course of the complaint and the absence of some of the physical signs and symptoms that would point positively to anatomic lesions of the stomach. When there is a catarrhal process, the symptoms become more pronounced immediately after taking food than in neurasthenia gastrica. The influence of the ingestion of indigestible substances upon sympathetic dyspepsia is often to relieve, or is of neutral effect, whereas in catarrhal indigestion it decidedly aggravates the condition. The dull pain after eating and the tenderness on pressure are more marked in the catarrhal variety, and the stomach contains large amounts of mucus. The symptoms of the latter do not intermit, as in nervous dyspepsia, but are more

[1] *Amer. Jour. Med. Sciences*, Feb., 1909.

constant. The analysis of the stomach-contents obtained after a test-breakfast shows digestion to be normal as to time and chemism, although rarely any secretory abnormality may be present. The motor function may be either reduced or increased, but, as a rule, it is normal.

Prognosis.—If there be an absence of an inherited predisposition, and if the cause is removable, complete recovery may be prognosticated. In a neurotic constitution, however, the tendency to recurrence is very strong. The most unpromising cases are those in which the cause is irremovable, though as to life the prognosis is favorable.

Treatment.—Every causal factor must be recognized and mitigated or removed. The dietary should be generous and composed of highly nutritious articles of food, and to convince the patient that his stomach is capable of digesting a full meal is the physician's first duty. So soon as the patient realizes the truth in reference to his digestive capacity his sufferings are largely at an end. The nervous system demands especial attention, and the internal treatment of the stomach is merely placeboic. Nerve-tonics combined with nerve-stimulants are serviceable.

A change of air from the city to the country, the mountains, or the sea-coast is usually followed by improvement. In some manner the patient must be extricated from the old surroundings under the influence of which the disease was started and has continued. Sea air has seemed to me to be more serviceable than mountain air in these cases, though I believe it to be an axiom in climatic therapeutics that the latter confers more lasting benefits than the former. These patients are often averse to taking exercise, but this sanitary measure should be insisted upon. Cold sponging of the surface, followed by friction to the skin, should be practised daily for its effect upon the nervous system. Occasional lavage, hot and cold douches, electricity (intra- and extra-gastric), and gastric massage may all be tried. In highly neurotic and hysteric females the S. Weir Mitchell treatment is often attended with good results. The hypochondriac form is often intractable. Strychnin, however, if perseveringly used, and if coupled with a change of air, often proves beneficial. One of the most obstinate examples of this nature that I have seen occurred in a retired merchant living in Philadelphia. This man was finally cured in consequence of his own suggestion, resulting in his removal to the country and engaging in farming. In neurasthenia gastrica lupulin finds a special indication (Stern).

SPECIAL FORMS OF GASTRIC NEUROSES, CHARACTERIZED BY MARKED AND PECULIAR ANOMALIES OF SENSATION, MOTILITY, AND SECRETION.

NEUROSES OF SECRETION.

HYPERCHLORHYDRIA.

(*Hyperacidity.*)

Definition.—An augmentation of the secretory function of the stomach during the digestive period, resulting in excess of HCl.

Etiology.—Hyperacidity is common during digestion, and is usually due to the causative influences mentioned under Nervous Dyspepsia (grief,

great anxiety, mental overtaxation). The disease is common among the professional classes (male sex) and in the young. Highly seasoned foods and alcoholic intoxicants may occasion the condition. Lichty emphasizes organic disease of the gall-bladder and ducts as a cause.

Symptoms.—Hyperchlorhydria may be *continuous*, though more often it is *discontinuous* and lasts from a few hours to several days. After the periodic form has lasted a long time it may gradually become a permanent condition. The patient first complains of *uneasiness* in the epigastrium one or two hours after meals. Later, this amounts to *pain*, and follows every meal after a like interval. The duration of the pain is from one to three hours. Acid eructations are frequently noted. The increase of hydrochloric acid interferes with the digestion of starches, and thus tends to increase the pain. On the other hand, however, a diet composed of albuminoids often affords relief, and the salts of the alkalies also ease the pain. Associated *nervous symptoms* (headache, dizziness) are often observed, though the bodily nutrition is usually well maintained. *Palpation* of the epigastrium may show a diffused tenderness. Evidences of moderate gastrectasis may be detectable. The amylolytic power of the stomach is uninfluenced as a rule. If amylacea are taken in large amounts, stagnation of stomach-contents and even permanent hypersecretion may be produced. The amidulin reaction is intensified.

Diagnosis.—Though the diagnosis of hyperacidity is made probable by the above symptoms, it is rendered certain only by a repeated analysis of the gastric contents. The findings, according to Einhorn, are: (1) On examination of the stomach in the fasting condition, the organ either is found empty or contains only a few cubic centimeters of juice; (2) one hour after Ewald's test-breakfast the hyperacidity is increased, owing to the great amount of free HCl present. To make a decisive diagnosis the examination must be made during the height of digestion.

Gastric ulcer must be eliminated. In this disease hyperacidity occurs, but the pain is aggravated immediately after eating, and is not relieved by albuminous food nor by large doses of alkalies, as in hyperchlorhydria of nervous genesis. In ulcer, moreover, the pain often leads to vomiting, and severe, painful attacks frequently occur at night.

Gastro-succorrhœa (*Reichmann*); **Gastroxynsis** (*Rossbach*).—In this affection there is an increase of hydrochloric acid, either constantly or intermittently, when no food is present. An *epigastric gnawing pain* and nausea appear in the full bloom of health. The *nausea* soon results in the *vomiting* of enormous quantities of gastric contents. The *appetite* is lost, but the thirst is excessive, and the amount of drink taken and of liquid vomited are proportional. During the night or in the early morning hours the patient commonly vomits large amounts of a clear or bile-tinted liquid containing hydrochloric acid and the gastric ferments in excess. This may be followed by persistent vomiting, attended with much retching. After a lapse of a few hours the ejection of a large quantity of highly acid liquid may be repeated. The *pain* often becomes intense, headache is common, and a tendency to collapse is usually marked. The attacks last, as a rule, about two or three days, when they quite abruptly give place to apparent good health. *Recurrence* at the end of periods ranging from a few months to a year or more are common. A physiologic form has been advanced.

The *diagnosis* is made upon the presence of the cause (a violent psychic shock), the clinical symptoms and course, as well as upon the results of oft-repeated analyses of the vomitus. Gastric ulcer and certain organic spinal and cerebral nervous affections, in which there is excessive gastric secretion, must be excluded before diagnosis can be made.

Gastro-succorrhœa Continua Chronica.—Reichmann first described a condition characterized by a *constant secretion of gastric juice* either in the absence or presence of food. The *symptoms* are much the same as those in *hyperacidity*, but tend to become continuous, so that the vomiting finally becomes a daily occurrence. In the fasting state a highly acid secretion that contains no food-particles flows through the stomach-tube. Albuminoids are rapidly and starches slowly digested. The disease is quite rare, and must not be confounded with the organic diseases to which continuous gastric succorrhea may be secondary and upon which it is dependent. Schreiber, Boas, and others believe that this is almost always a symptom of gastric atony or gastric ulcer.

Larval Superacidity.—This variety arises early in the period of digestion; the amount of gastric contents after the test-meal is large, and consists mainly of a watery secretion with low specific gravity and with a normal acidity and presenting the amidulin reaction.

Leube has described a neurosis in which there is a constant **subacidity of the secretion.**

Gastromyxorrhœa.—The fasting stomach often contains small quantities of mucus (5 cm.), but when above 25 cm. Cuttuer considers it pathologic and terms the condition *gastromyxorrhœa*. It seems to be largely of nervous origin. There are two forms of the disease, the intermittent and the continuous. In the first, the attacks develop suddenly with severe headache, pain, and vomiting, and after a period varying from one to five days the attack suddenly ceases (Friedenwald). The other type is usually discovered in examination for chronic catarrh. The *treatment* is symptomatic, although lavage is of service in the acute form. The neurotic tendency must be combated during the intervals.

Achylia Gastrica (*Einhorn*).—The suspension of the gastric secretions may result either from gastric atrophy (common) or from a nervous derangement of secretion. The condition has been mistaken for carcinoma of the stomach. Lactic acid, however, is not present in excess. Eosinophile cells generally occur in the gastric juice. Achylia gastrica may cause chronic lienteric diarrhea (A. A. Jones).

The **prognosis** in the foregoing affections is not bad as to life, and not infrequently a cure, even, can be effected.

Treatment.—The dietetic treatment differs according to different observers. Einhorn advises three large and two small meals composed principally of nitrogenous articles, daily. Physiology, however, teaches that when milk, bread, fats, and starchy substances are taken, the amount of HCl secreted is small, hence the proper causal treatment is to limit the amount of proteids. Acids, tobacco, and spirits—substances that excite the glands of the stomach—must be excluded. The medicinal treatment should, in addition to meeting the general neurotic condition, consist of full doses of sodium bicarbonate or sodium citrate. In some cases more active alkalies than sodium bicarbonate may be needful—*e. g.*, magnesium and sodium salicylate, aluminum silicate (ʒss–j ad aqua ℥iij a. c.),

either separately or in combination. Lavage daily, before the chief meal, is beneficial and may be combined with a salt-free diet. Lemoine advises hydrotherapy and rest to strengthen the nervous system.

NEUROSES OF MOTILITY.

INCREASED PERISTALSIS OF THE STOMACH.

Gastric peristalsis is increased in various conditions, which will be considered *seriatim*, though briefly.

(*a*) **Belching and Eructations.**—These may be of *nervous origin* and are met with generally in *hysteric* subjects, and less frequently in *neurasthenics*. The air is swallowed, and then expelled with more or less noise, owing to an increased contractility of the stomach. The gas is *odorless*, and differs in this point from the gases of fermentative dyspepsia. Epigastric distress and distention often arise, and certain nervous phenomena, as anxiety or palpitation, may coexist. In hysteric subjects the belching may be from the esophagus alone.

(*b*) **Pyrosis** means regurgitation of the acid contents of the stomach.

(*c*) **Rumination** (*Merycism*).—A rare affection in which the food is regurgitated into the mouth, the cud chewed, and again swallowed after the fashion of ruminants.

(*d*) **Nervous Vomiting.**—This is a *reflex neurosis* that may affect persons of any age, though most frequently it is seen in adult females with an hysteric tendency. Without previous nausea, and independently of the character of the food taken, the contents of the stomach are readily expelled or, more correctly speaking, regurgitated into the mouth, and then expectorated. Though this usually takes place after meals, it may occur without reference to meal-time—a feature that indicates its nervous origin. The *attacks* of vomiting are separated by longer or shorter intervals of excellent health. Periodic vomiting may also occur independently of hysteria or other nervous affections, as pointed out by Leube. The *course* is rarely unfavorable.

(*e*) **Peristaltic unrest** (Kussmaul). or **spasm of the stomach**, has been referred to under Nervous Dyspepsia. It has also been observed in compensatory hypertrophy of the stomach-wall following pyloric stricture. In a case of gastric carcinoma in my own care the supermotility of the stomach caused an almost immediate expulsion of the gastric contents, and even of the rigid test-meal at certain times.

(*f*) **Cardiospasm.**—By this term is meant a painful cramp of the cardia. Two forms are distinguished: (*a*) acute cramp; (*b*) chronic cramp (exceedingly rare). Among causes are neurasthenia, hysteria, and local irritation (thermal, mechanical). Bassler found, post-mortem, firm adhesions of the pleura, particularly at the base, in association. Chronic spasm may lead to complete atresia of the cardia, and is a distressing affection. In acute cardiospasm the attacks may recur.

(*g*) **Pylorospasm.**—Cramp of the ring-musculature of the pylorus may be *primary* or *secondary*. The latter is due to intense local irritation (superacidity, hypersecretion, excess of organic acids). The painful spasm in the pyloric region induces stagnation of the ingesta, followed by atony of the stomach and consequent dilatation.

Treatment.—To the regimenal management the attention of the physician should be primarily directed. The medicinal treatment is to

be aimed at the causal nervous affection. The valerianates and the bromids often do good service. For the cramp of the cardia and pylorus belladonna or codeine are efficient; these failing, stretching of the cardia is indicated. If internal treatment fails in functional motor insufficiency, operative intervention may be indicated.

DIMINISHED PERISTALSIS OF THE STOMACH.

(*Atony.*)

(*a*) **Pyloric Relaxation or Incompetency.**—This is a rare neurosis that allows the partially digested gastric contents to pass the portals of the stomach prematurely. It likewise permits the regurgitation of the intestinal contents into the stomach. Its recognition is possible upon inflating the stomach, when gas may be seen to pass into the intestines, and also upon the regurgitation of intestinal contents into the stomach.

(*b*) **Insufficiency of the Cardia.**—This condition leads to eructations and regurgitations, and when these are of aggravated form they impair the general nutrition. Ordinarily no ill-effects follow.

(*c*) **Atonic Dyspepsia** (*Atony*).—This may occur either as a neurosis or secondary to chronic gastritis and reflexly in chronic appendicitis, carcinoma, and tuberculosis of the intestines. It implies *hypomotility*. The *chyme* is retained in the stomach beyond the natural time-limit. There is an *epigastric oppression* with a distention of the organ during digestion that tends to become permanent. There are eructations of gas, an impaired appetite, and often constipation. The stomach is found empty in the morning, and six or seven hours after Leube's test-meal it contains some chyme. In the absence of pyloric stricture the hypomotility may be shown by the administration of salol.

Treatment.—The diet is to be regulated as in chronic gastritis with dilatation. It is rarely necessary to restrict the solids to any marked extent, but the quantity of fluids should be lessened. The patient must be taught to eat slowly and masticate thoroughly. His hygienic standard of living must be high, and he must not be allowed to over-use his mental faculties. Exercise in the open air and cold baths, properly regulated, are potent for good. Of medicines, strychnin stands first, and I have found the following formula of great service:

℞. Tr. nuc. vomicæ, f℥iiss (10.0);
Inf. cascarillæ, q. s. ad f℥iv (128.0).—M.
Sig. ℥ij (8.0) three times daily.

Electricity is indicated, and intragastric faradization has given excellent results. The constipation is to be overcome by an appropriate dietary (green vegetables, Graham bread, an abundance of fruit). There is an advantage in assuming the right lateral position, which hastens evacuation. Lavage deserves a prudent trial.

NEUROSES OF SENSATION.

CARDIALGIA.

(*Gastralgia; Gastrodynia.*)

Definition.—Severe paroxysmal pain in the epigastrium in the absence of gastric lesions. There are two other forms of this disease that

are clinically identical with nervous gastralgia, the one occurring in ulcer and carcinoma of the stomach, and the other in certain chronic nervous diseases, forming the so-called gastric crises.

Etiology.—The subjects are often hereditarily predisposed to neuroses of other types. Such conditions as anemia, exhaustion from repeated hemorrhages, and syphilis exert a *predisposing* influence. The female sex is more liable than the male, and in the former it appears to be dependent upon disturbances of the menstrual function or quite frequently upon hysteric conditions. It is sometimes excited by reflex irritation, by deep grief, worry, and great anxiety. Hypochondriasis and hyperacidity are also among its frequent causes.

Symptoms.—These are *sudden in their onset* as a rule, and quite characteristic. Occasionally the attack is preceded by anorexia, or it may begin with a sense of oppression and distention in the epigastrium, lasting for a few minutes. In any event, the onset of the attack proper is marked by *agonizing pains* in the epigastrium, that dart through to the back, and at times also pass around the lower ribs. The seizure lasts from a few minutes to an hour or two, and terminates with *eructations of gas*, or, less frequently, with vomiting. From the nature of the causative factors it is obvious that the gastralgic seizures are in no wise dependent upon the character of the food taken; hence the fact that they occur more frequently when the stomach is empty need occasion no surprise. Firm pressure over the epigastrium relieves the pain. *Nervous phenomena*, varying with the etiology of individual cases, are constant attendants, but cannot be detailed here. A distinct clinical variety is found associated with that form of nervous dyspepsia in which an excess of HCl is secreted (*vide* Hyperacidity); this occurs at varying intervals. Many functional nervous disturbances are thus subject to the law of periodicity. I believe that a very small percentage of cases are caused by malaria, since I have met with two such cases in a malarial district, both of which yielded readily to quinin. The disease took on a desultory, periodic character, and was associated with other malarial symptoms.

Diagnosis.—The history, the absence of any local causes, the violent, spasmodic attacks of pain, that cease abruptly, and their occurrence at irregular intervals, will enable the clinician to render a positive diagnosis in most instances. The gastric crises that occur in locomotor ataxia closely resemble gastralgia and must be excluded. Gastralgia may be simulated by cholelithiasis (*q. v.*). To discriminate this condition from *gastric ulcer* is difficult, but stress has been laid upon the differential points in the description of the latter disease (*vide* p. 809).

Prognosis.—This depends entirely upon the causal condition. The disease itself has no intrinsic fatal tendency.

Treatment.—This is to be subdivided into (*a*) the treatment of the attack; (*b*) the management of the intervals between the seizures. The pain is, as a rule, sufficiently intense to demand morphin, which is best administered hypodermically in combination with atropin. This should not, however, be given if an idiosyncrasy exist. In mild attacks the constant or the faradic current often affords prompt relief. Under these circumstances counter-irritation, together with the internal use of Hoffman's anodyne or chloroform in small doses, may relieve the pain.

(*b*) *The Management of the Intervals.*—Here the physician's efforts

should be directed to the detection of the causes and their removal by appropriate means. In hysteric females I have obtained good results from the prolonged use of the valerianates, combining with them iron and arsenic, thus:

℞. Zinci valerianat., gr. xviij (1.16);
Quininæ valerianat., gr. xxvij (1.74);
Ferri arseniat., gr. ij (0.129).
M. et ft. pil. No. xviij.
Sig. One after each meal.

A change of air is often highly serviceable, and should be advised whenever financial considerations permit. These patients are constantly in a more or less exhausted, anemic, and run-down condition, and a tonic plan of treatment is always indicated to overcome the primary cause. In the intervals between the attacks digestion, as before stated, proceeds normally, and the stomach, therefore, requires no treatment. Constipation, if present, is a condition demanding relief, not, however, by the use of purgatives, but by such means as massage, a suitable diet, enemata, or laxative suppositories. The physician must carefully regulate the sanitary particulars of the patient's daily life.

HYPERESTHESIA OF THE STOMACH.

This is met with in functional and organic diseases, as well as in chronic gastric catarrh and other affections of the stomach. Again, it may occur as a *neurosis*, most frequently in chlorotic girls and women. There is an increased gastric sensibility, so that the mildest irritant produces *painful sensations* that may be either gnawing or burning in character. A feeling of fulness and nausea are among the common features of the complaint. Food and certain articles that are not easily digestible may afford relief, and, oppositely, fasting or restriction of diet may aggravate the condition. The complaint, however, is often aggravated during digestion, particularly after excessive indulgence in certain kinds of food (crabs, lobsters, oysters, strawberries). Cutaneous symptoms, as erythema and urticaria, may appear. *Hypochondriasis*, neurasthenia, and hysteria are often associated. The above symptoms are dependent upon an individual idiosyncrasy.

Treatment.—At first a restriction of the diet to soft and liquid articles should be tried, and later a cautious return to solid food is to be made. Of medicaments, the bromids, given for a period of two or three months, have given the best results in my own hands. For the chlorotic type iron in the form of Blaud's pill, in ascending doses, is the best treatment.

ANOREXIA.

This consists merely in a loss of appetite, and occurs in many organic gastric disorders. It may also be a primary gastric *neurosis*, the latter being often associated with gastric hyperesthesia. Anorexia sometimes leads to a repugnance to food and a degree of abstinence that may induce grave nutritional disturbance. Among exciting causes mental shock of any sort ranks first. The recognition of anorexia as a neurosis of the stomach is difficult after the general nutrition has become seriously im-

paired. Chronic dyspepsia, phthisis, and other diseases associated with emaciation and debility must be excluded before the diagnosis is established.

HYPEROREXIA.

(*Excessive Appetite.*)

This may either be symptomatic of other affections (*e. g.* diabetes mellitus) or it may be a gastric neurosis. It may also be paroxysmal (*bulimia*). The patient complains of burning sensations in the epigastric region and of an insatiable hunger. The symptoms of neurasthenia and hysteria are often in association. The local and general symptoms are relieved by food. It may also accompany affections of the brain, exophthalmos, and migraine. In *bulimia* the abnormal sensation of hunger may come on at any hour, even immediately after abundant food has been taken. When the morbid sensation of hunger develops more gradually and some time after meals it is spoken of as *polyphagia*.

Pica is the term applied to the craving for substances not used as food (slate-pencils, dirt, chalk).

Malacia represents the desire for highly spiced dishes (mustard, salads, pickles, fruits).

The above conditions are met with in neurasthenia, chronic gastric affections, and chlorosis.

VIII. DISEASES OF THE INTESTINES.

METHODS OF DIAGNOSIS.

Examination of the Feces.—Although the results are in most cases unsatisfactory, an examination of the feces should not be neglected, especially in the more serious affections of the intestine. This embraces—(*a*) a macroscopic; (*b*) a microscopic; (*c*) a chemical; and (*d*) a bacteriologic examination.

(*a*) The *macroscopic* appearances often suffice. A thorough inspection of the stools furnishes valuable points in regard to the presence or absence of coarse parasites, fragments of tumor, foreign bodies, concretions, blood, bile, fat, pus, mucus, undigested meat, and the like.

The shape, color, and consistence of the stools must be noted, and it is to be remembered that in these particulars, as well as regards their frequency, they exhibit a considerable range of normal variations, according to individual peculiarities, the character of food taken, and so on. It is to be recollected that normal stools contain fat in varying amounts, for the reason that only a limited quantity can be emulsified and taken up from the intestine. The naked eye may, at times, detect its presence from the "peculiar silvery appearance" of the feces. Fat in the stools (*steatorrhea*) is often pathologic, and the separate affections in which it is met with will be considered hereafter. The dejecta present a shining, tallowy appearance, either throughout or in circumscribed spots. Again, the fat may occur in the form of oil floating on the surface of liquid stools. Mucus is also visible, either as slimy or jelly-like masses, or as shreds and granules (sago-grains). Diarrheal

stools should be examined macroscopically with great care for gross admixtures (flakes of casein, bits of meat, etc.). Constipational dejections often assume a rounded form (*sheep's dung*) on account of their delay in the large bowel. They may attain to the size of an orange, and may be, though rarely, enveloped in mucus or blood-streaked. Their color is dark. On the other hand, the stools may be colorless in cases in which the bile-ducts are occluded; these usually contain a large proportion of fat, though not invariably. The effect of certain drugs upon the color of the stools is to be borne in mind. When blood is intimately mingled with the feces, they have a reddish, dark- or blackish-brown (tarry) color, according to the quantity and the time allowed for decomposition in the intestine. Blood, either clotted or fluid, may also be passed in a pure state. Its source is usually the lower bowel, though when peristalsis is augmented, it may come from the small intestine, as in typhoid fever. Pus may occasionally be recognized macroscopically. From a diagnostic point of view, it is most important to examine for biliary concretions in doubtful abdominal colic. "For the detection of small concretions the stools should be passed through a sieve" (Ewald).

(*b*) *Microscopic Examination.*—Diarrheal stools can be examined as discharged, but to solid and mushy dejections a solution of common salt (½ per cent.) should be added and all hard masses thoroughly broken up. Different portions of the stools are to be selected for microscopic examination. *Microscopically* we are enabled to detect the eggs of parasites, pus, blood, protozoa, mucus in the form of shining, vitreous, homogeneous, or whitish masses; and in the interior of the latter certain pathogenic bacteria, various crystals, and intestinal epithelium may be seen. Remnants of vegetable food may simulate mucous islets, but the former strike a blue color on the application of Lugol's solution. Microscopically, diarrheal stools show undigested muscle-fibers, fat-crystals, vegetable cells, starchy granules, and innumerable bacteria. Von Leersum[1] advocates the sedimentation process until the sediment finally contains most of the meat-fiber, and that the nuclei be then stained and examined to estimate the degree of pancreatic digestion. Undissolved starch in even moderate quantity points to catarrhal enteritis of the small intestine. On microscopic examination of the dejections in constipation we find "a copious detritus of brown or black color, usually numerous colorless or slightly tinged triple phosphates (phosphate of ammonium and magnesium crystallizing in the form of a coffin-lid), or, more sparse, crystals of neutral phosphate of lime." Seldom do we meet with the rhomboid plates of cholesterin, which are recognized in that they are colored from a red, dish-brown to violet by dilute sulphuric acid (1 : 5), and become blue or green on the further addition of a solution of iodin. Needle-shaped crystals of fat, single and also in the forms of tufts, are frequently met in obstruction of the biliary ducts. Bile-pigment cannot be detected. Remnants of food are sparsely present in normal feces. Epithelium from the mucous membrane, pus-cells, and blood-corpsucles, unless they come from the passage of the fecal mass through the anus (in which case they are simply adherent to the external surface of the scybala and are but little changed), are greatly altered; they are fatty, degenerated,

[1] *Münchener Med. Woch.*, February 6, 1912.

shrunken, and hardly recognizable. Rhomboid crystals of hematoidin may be at times observed. The microscopic examination for animal parasites will be referred to in appropriate sections of this work.

(*c*) *Chemical Examination.*—The presence of bile-pigment is easily detected by the Gmelin reaction. The stools must, if needful, be rendered fluid by the addition of water, then filtered, and the filtrate allowed to dry. At the margin of the drop the characteristic green color will appear. Urobilin strikes a red color. The stools in diarrhea may contain ferments capable of digesting albuminoids. The fatty acids are distinguished from fatty soaps by the solubility of the former in ether.

For the detection of occult blood, to an ethereal extract of 2 to 5 cc. of liquid feces or solid stool reduced by water, 2 cc. of a 10 per cent. solution of guaiac in glacial acetic acid is added, followed by 2 cc. of hydrogen dioxid, the mixture is thoroughly shaken and in the presence of blood turns blue. A dilution of blood, 1 in 250,000, is recognized by the Adler technique, in which to feces prepared as above add 1 cc. glacial acetic acid and 2 cc. of fresh concentrated solution of benzidin in pure alcohol, and an equal volume of hydrogen dioxid; if positive, a dirty green or deep blue develops. Boas' phenolphthalein test may also be employed.[1] In bleeding from the stomach and intestines, from the mouth, pharynx, rectum, or vagina, the ingestion of meat, watermelon, or medicinal iron must always be excluded. The indol-reaction may be increased, pointing to increased intestinal putrefaction.

(*d*) A *bacterial examination* of the intestinal contents, and particularly of any mucus or mucopus that may be discharged, may decide the diagnosis of certain intestinal disorders (tuberculosis, amebic dysentery). For the method of carrying on these investigations the reader is referred to special works on diagnosis and bacteriology.

(*e*) The *x*-rays have been shown to be of diagnostic value in many intestinal conditions (*vide* Enteroptosis, Appendicitis, Enteroliths).

Physical or External Examination.—*Inspection.*—This should be made with the patient in the dorsal position and with proper illumination. Localized prominences are to be noted (sometimes simulated by localized contractions of the abdominal muscles). The influence of respiration on these circumscribed bulgings is also to be observed. In the absence of unusual distention of the abdominal walls it is of great value to inflate the large intestine with air *per rectum*, and to note the progressive distention of the intestinal coils as a means of detecting obstructing lesions in the bowel; the position and mobility of a tumor should also be noted. It is often of marked aid to inspect the mucosa of the rectum by the use of approved specula. The volume of the abdomen may be diminished or even "scaphoid." Abnormal peristalsis may rarely be noted (important if associated with distention).

Palpation.—This is of first importance. The patient should occupy the dorsal decubitus, with the head raised, the thighs drawn up, and the mouth open, so as to relax the abdominal muscles. Something may be gained in this direction by distracting the patient's attention. I have found that placing the patient in the lateral decubitus, with the thighs flexed on the abdomen, to be the most satisfactory way of determining the degree of mobility of certain tumors. The examiner should

[1] *Amer. Jour. of Gastro-enterology*, September, 1911, p. 48.

not fail to remember the knee-elbow position in cases in which it is desired to palpate the parts occupying the bottom of the pelvic cavity and all deep-seated, movable growths. In certain cases relaxation of the abdominal muscles is only obtainable by anesthetizing the patient, and I do not hesitate to do this in cases in which the diagnosis is important. In palpating the abdomen for abnormal conditions we must keep in mind steadily the relations of the different parts of the intestines, and also that the latter may vary considerably in position—a fact particularly true of the transverse colon (*vide* Enteroptosis). In this connection Ewald's statement "that abnormally situated organs or neoplasms of parts other than the intestines will, under the pressure of the intestines filled with air or water, return to the position that the organ normally occupies," should be emphasized. New growths of the pancreas, of the spinal column, or of the pelvis, and retroperitoneal tumors will remain fixed. Palpation may detect pathologic peristalsis, and increased resistance if the coats are thickened. Tenderness, localized or diffuse, as well as peritoneal friction, is noted. The rectum may be palpated if the symptoms point to disease of that organ.

The palpation of pathologic conditions of the intestines will be considered in connection with the separate intestinal affections.

Percussion detects a fluid effusion either in the general peritoneal cavity, the position varying with the patient's posture, or in circumscribed localities; the latter must not be confounded with areas of dulness that are occasioned by splenic and hepatic enlargements, solid new-growths, or abscesses. Air in the peritoneal cavity (*meteorismus peritonei*) generally gives a pure tympanitic note, though if the tension be very strong, a non-tympanitic tone may be elicited. These sounds are general, even extending up to the fifth or fourth rib, and hence they cover the regions of the spleen and liver. The best results when the abdomen is not tense are obtained after inflation of the large intestine with air. The pitch of the tympanitic note becomes elevated with increase in the tension of the gut; it falls with relaxation of the bowel. Hence the large cannot always be told from the small intestine by percussion.

Auscultation.—Noises are often audible either at a distance or by means of a stethoscope applied to the abdomen. They are sometimes occasioned by the natural peristaltic movements or by certain voluntary or involuntary spasms of the abdominal muscle. I have repeatedly confirmed the observation of Ewald, who frequently found in those suffering with chronic intestinal indigestion a swashing or splashing noise, sounding as though air and water were being forced through a narrow space in the ileo-cecal region. These sounds may rarely be found in healthy persons. Similar noises sometimes have their seat in the descending colon, particularly if the bowel is unnaturally dilated by air or fluid. They are often audible prior to an evacuation in cases of colitis. Noises may also originate in the transverse colon, and to discriminate these it is necessary to empty the stomach if we would avoid confusion with identical gastric sounds. Direct auscultation of the intestines renders audible the peristaltic movements, and the absence of the latter indicates paralysis of the intestine, which may be local or general. Friction-sounds may be audible when inflammatory exudates are present. When obstruction of the large intestine is suspected, auscultation should be

practised while air is being forced into the rectum, inasmuch as the degree of permeability can be thus determined. Metallic tinkling and amphoric noises may be audible, particularly on making auscultatory pecussion, but these are without real diagnostic value.

ENTEROPTOSIS.

Definition.—The descent of the intestines from their normal position. The condition occurs coincidently with gastroptosis, nephroptosis, and prolapse of other viscera, constituting splanchnoptosis (Glénard's disease).

Etiology.—It is linked with gastroptosis and other forms of ptosis by common etiologic influences, such as sex (being most common in females), tight lacing, traumatism, muscular strain, numerous pregnancies, rapid emaciation, and probably the wrong use of cathartics. Either the small intestine alone or the large, or both, may be involved. Prolapse of the colon (coloptosis) is the more common; it is more frequent than gastroptosis (C. Meinert). Lying immediately above the symphysis pubis, it is sometimes elongated and tortuous—"S- or M-shaped."

Symptoms.—The condition, even when pronounced, may exist without symptoms. On the other hand, in the majority of instances the intestinal, gastric, and other bodily functions are disturbed, and yet enteroptosis is usually overlooked. Chief among the intestinal symptoms is *excessive flatulence;* not rarely, also, there is membranous enteritis, the latter probably being due to the flexures that produce an arrest of fecal masses, and this in turn causing inflammation (Boas). *Constipation* generally prevails, and sometimes alternates with diarrhea. The symptoms of gastroptosis and nephroptosis are often associated; they are loss of flesh and nervous symptoms, and the latter may simulate those of neurasthenia or hysteria.

The **diagnosis** is made upon the afore-mentioned points and upon the results of a physical examination. The position of the colon may be determined by inflation with air or gas. Again, after the injection of water (f℥viss–ixss—200–300 c.cm.) a splashing sound is audible; this is double the amount of water required in the normal condition. Glénard has pointed out that a transverse cord (which he believes to be the colon) can be felt in the upper part of the abdomen. Boas and Ziemssen assert that this cord is the pancreas, rendered palpable by the sinking of the stomach. Movable tenth rib is common, but not a distinctive sign, since it is just as frequent in nervous gastric disturbances in general. The *x-rays* are of diagnostic value (*vide* Plate VII.).

Treatment.—The bowels must be moved regularly, the tonicity of the abdominal walls must be increased by electricity, massage, and hydrotherapy, and in strongly nervous cases the treatment of neurasthenia, including the Weir Mitchell rest-cure, must be instituted. Supporting bandages have been found serviceable. The medicinal treatment aims at meeting symptomatic indications, such as flatulence and fermentation.



Skiagraph of Enteroptosis (Pfahler).

INTESTINAL CATARRH.

(*Catarrhal Enteritis; Muco-enteritis.*)

Definition.—A catarrhal inflammation of the mucous membrane of the whole or of any anatomic division of the intestinal tract. It may be either acute or chronic, primary or secondary. The chronic variety occurs less frequently than its counterpart, chronic gastritis, particularly in adult life.

Pathology.—The morbid lesions of the acute variety do not differ essentially from those met with in catarrhal inflammation of any other mucous membrane. The first stage is characterized by swelling and dryness of the mucosa; this is soon followed by a copious exudation of mucus, and more rarely of pus, which bathes the membrane more or less completely. After an abundant secretion is poured out the membrane appears rather pale, though the tips of the valvulæ conniventes in the small intestines may appear reddened. The solitary and agminated glands, as well as Peyer's patches, may stand out prominently, owing to their corrugated condition (*follicular enteritis*). The apices of the solitary glands often undergo a necrotic change, thus forming follicular ulcers. The remainder of the mucosa may also be the seat of rather extensive areas of superficial erosion, though this must not be confounded with postmortem softening of the epithelium. In some cases the desquamation of epithelium is more pronounced than the abnormal mucous secretion. In *chronic* intestinal catarrh the mucosa presents a slaty hue, with a more or less dark pigmentation of the villi and follicles; it is in most instances thickened, owing to an increase in its connective-tissue elements. In a smaller number of cases it is thinned, particularly in the intestinal catarrh of children, on account of atrophic changes affecting chiefly the glandular and muscular layers. Roughening of the inner surface of the bowel, due to projecting glands, is frequent in those forms of chronic intestinal catarrh that are attended with thickening of the coats. Polypoid cysts may develop in long-standing cases.

Etiology.—The primary form is produced by (*a*) local irritants, either mechanical or toxemic, that find their way into the intestinal canal. The chief source of these excitants is an unsuitable dietary, and especially is this the case in children. It is readily seen from this fact why the stomach and the intestines are often simultaneously involved in a catarrhal process. (*b*) Over-eating may be productive of the disease, though this often excites diarrhea by merely increasing intestinal peristalsis. (*c*) Idiosyncrasy has a positive influence, the ingestion of certain substances not difficult of digestion being invariably followed by this affection in individuals thus predisposed. (*d*) Toxic substances, whether in the form of tainted food-stuffs (spoiled meats, ice-cream, beer) or inorganic poisons (mineral acids, caustic alkalies, mercury, arsenic) or irritating cathartics, often produce intestinal catarrh. (*e*) Impure water, or water to which individuals are unaccustomed. (*f*) Atmospheric changes, particularly a prolonged high or a sudden fall of temperature, the latter being especially apt to cause it in children. (*g*) An excess or a lack of biliary secretion. Two functions of the bile (its antiseptic properties

and its power to stimulate peristalsis) must not be forgotten: the one explains how a paucity of this secretion favors the abnormal processes of fermentation that are capable of exciting catarrh, and the other makes plain the possibility of a bilious diarrhea being due to an excessive hepatic secretion. It is not clear, however, that the latter condition is attended with an actual catarrhal process. The same is true of diarrhea due to fright, excitement, or other nervous influence. (*h*) Bacteria are, doubtless, among the *excitants*—*e. g.* the normal colon-bacillus. under conditions favorable to its growth and development. The small intestinal *diplococci* probably operate to produce catarrh, particularly fermentative dyspepsia (Schmidt and Strasburger).

Secondary or **complicating forms** are caused—(*a*) By direct extension from adjacent organs (ulcers, gastritis, peritonitis, hernia, and invagination); (*b*) By general infectious processes (septicemia, pyemia, typhoid fever, dysentery, cholera, tuberculosis, pneumonia).

The **chronic forms** are met with—(*a*) In certain cachectic states (carcinoma, chronic malaria, chronic Bright's disease, Addison's disease, and profound anemia); (*b*) In connection with disturbances of the circulation, particularly such as produce stasis in the terminal branches of the portal system of vessels: among the chief diseases that tend to prevent the return of venous blood from the intestines are chronic heart-affections, diseases of the liver (especially cirrhosis), and emphysema; (*c*) Severe cases of chronic diarrhea, probably due to the *protozoon balantidium*, have been reported recently.

Among predisposing causes is the *age*, children being particularly liable to the disease. Unfavorable hygienic surroundings, especially when a high temperature prevails, and epidemic and endemic conditions also strongly predispose to the affection.

Clinical History.—From a clinical standpoint we recognize acute and chronic forms of enteritis; also special varieties (*vide infra*).

The **simple acute form** of general catarrh of the intestines (muco-enteritis) has for its two most characteristic symptoms slight *griping* or *colicky pains* in the abdomen (sometimes absent), that are followed soon by *diarrheal stools*. The *discharges* consist, at first, of feculent masses, and later of a watery, highly irritating fluid. Diarrhea is due partly to increased peristalsis and partly to the abnormal irritability of the intestinal mucous membrane. Active peristalsis of the intestines may (*vide ante*) be of purely nervous origin (*e. g.*, in neurasthenia), and produce a diarrhea that is to be distinguished from that due to catarrh, although an exceedingly difficult task in some cases. Again, steatorrhœa may be present in cases in which the pancreatic secretion is absent. The causes that produce the catarrh also produce the undue peristaltic movements. If it be true, as physiology teaches, that the stools, owing to the absorption of the watery portions of the food, are normally formed in the large intestines, then catarrh of the small intestines alone does not excite diarrhea, though both large and small are involved in the majority of the cases. On the other hand, in *acute colitis diarrhea is conspicuous*, and forms the most important clinical symptom. The vigorous peristalsis also accounts for the gurgling and rumbling sounds (*borborygmi*) that are often felt and heard by the patient himself. These peculiar noises, if pronounced, point to isolated catarrh of the small intestines. The *stools*

vary in number from two to ten or more, being increased in frequency after taking food; gases are also formed, causing tympanites. The thin or mushy stools either present a bright-yellow or a yellowish-brown color and emit offensive odors. Occasionally they are greenish in color from the presence of considerable quantities of bile-pigment or from bacterial action. In advanced cases of considerable severity there is painful tenesmus; the stools are often small and contain mucus and blood, becoming dysenteric in character, especially in colonic catarrh. Nausea, impairment of appetite, and great thirst are commonly present.

A *microscopic examination* reveals large masses of epithelium and mucus, as well as countless microörganisms and isolated leukocytes, crystals of calcium phosphate, oxalates, remnants of food (starch-granules, fat, and muscular fibers). Flakes of yellowish-brown mucus, of epithelium, and grayish-white masses of fat may often be seen *macroscopically*. The stools give an alkaline reaction as a rule.

The *physical examination* reveals on *inspection* slight tympanitic distention as a rule. The tongue is dry and furred. *Palpation* elicits considerable sensitiveness in the majority of cases, though during the colicky pains pressure with the palm of the hand often affords relief. Fluctuation may be detected if the intestines contain much fluid. *Percussion* gives an exaggerated tympanitic resonance, varying, however, with the tension of the bowel. Splenic enlargement has been described by Fischl.

The *general symptoms* are often entirely wanting, save for a slight feeling of weakness due to the diarrheal discharges. Severe forms of infectious origin often disturb the general health considerably. The patient is languid, and prostration is prominent; he suffers much from headache, and pyrexia is common, the temperature often reaching 100°–103° F. (37.7°–39.4° C.). The higher temperatures are seen among children. Additional evidences of a systemic infection are sometimes observed, such as painful enlargements of certain joints, severe muscular pains, and albuminuria.

Complications.—The symptoms of gastric catarrh (vomiting, nausea, and pain immediately after feeding) are often associated with those of enteric catarrh; the combination is then spoken of as *gastro-enteritis*. Acute nephritis has been noted as a sequel.

Special Forms.—Though the anatomic limits in the more or less local forms of intestinal catarrh cannot be made out definitely, yet the different clinical pictures observed often enable us to fix the location of the disease with considerable accuracy; it is important, moreover, from the standpoint of the treatment, to accomplish this whenever possible. The following may be briefly described:

(*a*) *Duodenal catarrh* (*duodenitis*), in which form constipation, often obstinate, is present in the place of diarrhea, the colon not being affected; merely local pain, tenderness on palpation, and uneasiness are complained of. These symptoms may frequently be overshadowed by those referable to the stomach when gastric catarrh coexists (*gastro-duodenitis*). Without *jaundice* (usually present) due to the occlusion of the common bile-duct in consequence of the swelling of the duodenal mucous membrane, we cannot render a positive diagnosis.

(*b*) Localized *catarrh of the jejunum and ileum* cannot always

be distinguished. The condition is often found to be a more or less prominent feature in general enteric catarrh, in which complaint diarrhea is a prominent symptom. The existence of this special variety may be safely inferred when certain enteric symptoms are combined with marked gastric disturbance. Under these circumstances the symptoms indicative of inflammation of the small intestines are rumbling noises (borborygmi), colicky pain, swelling, and slight tenderness over the abdomen in the vicinity of the umbilicus or over other regions occupied by the small intestines. Finally, an *examination of the stools* furnishes valuable points for differential diagnosis. It must be kept in remembrance that in catarrh of the small intestines the stools may be quite solid, despite the increased peristalsis caused by the catarrhal process (*vide ante*). More frequently, when the ileum is the seat of catarrh the colon is also implicated, this combination being attended with diarrhea, even if it be of minor severity. The thin stools "contain food-remnants, that point indubitably to implication of the small intestine." As the result of increased peristalsis of the small intestines their contents are passed into the large bowel with undue rapidity; hence the latter contains undigested food-constituents and other substances that are normally found in the small intestines. These pass from the rectum unchanged. They are mainly starch, fat, and masses of meat-fiber, the latter of which may be of sufficient size to be seen by the naked eye. This would be pathognomonic evidence of the form of catarrh in question if it were not true that increased peristalsis of the small intestines, due to other conditions, as anemia, extreme nervousness, and fever-conditions, that are not seen in ileo-jejunal catarrh, causes the same fecal peculiarities. An acid reaction of the dejecta points to catarrh of the small intestines. *Microscopically* the stools show hyaline particles of mucus, giving rise to a speckled appearance.

In health the contents of the small intestines give the characteristic color-reaction for bile-pigment, whilst the contents of the large bowel and the stools do not. There is quite often a large admixture of undecomposed bile-pigment (Strümpell) that responds to Gmelin's test,[1] a fact of considerable value in diagnosis. Nothnagel has called forcible attention to the fact that *bile-stained* stools and small *pigmented* masses of mucus are met with, and are highly characteristic of the diarrhea that marks catarrh of the small intestines.

(*c*) *Colitis.*—The joint appearance of abdominal pain and diarrhea is almost pathognomonic of this condition. These symptoms, in the absence of the more prominent and above-mentioned clinical features that have special reference to inflammation of the small intestines, point to the fact that the large intestines are the chief seat of the disease.

Physical examination is only partially confirmatory of the rational symptoms. The chief sign is tenderness on palpation over the track of the colon. An *ocular examination of the stools* furnishes important practical results. They may contain blood and mucus, and the latter often in masses large enough to be readily visible to the naked eye; it is not

[1] This consists in bringing a few drops of nitric acid in contact with the intestinal contents, when the characteristic play of colors appears. (See also Methods of Diagnosis, pp. 830–834.)

intimately mixed with the feces, as in catarrh of the small intestines, but forms separate masses. The feces are often of the consistence of soup. "If the catarrh affects the lower portion of the large intestine chiefly, it may be that the intestinal contents are already formed" in firm lumps, which may sometimes be wholly or partly enclosed in a layer of mucus (Strümpell).

Such *general symptoms* as loss of flesh, weakness, and sallowness of the skin are often observed. Simple diarrhea, lasting but a few days, as a rule, is to be classed with catarrh of the large intestines, since these affections imply increased peristalsis of the large bowel. It is not always easy, however, to discriminate diarrhea due either to purely functional influences or to catarrh of the rest of the intestinal tract.

(*d*) *Proctitis*, or inflammation of the rectum, is characterized by painful tenesmus and by the presence of large quantities of mucus and pus, particularly in the dejections. The disease may be primary, though more often it is secondary to morbid lesions either in organs that are adjacent to or in the rectum itself.

Chronic intestinal catarrh may, comparatively rarely, be a *primary disease*, developing gradually. It may also be *secondary* (*vide* Pathology) at times to one or more attacks of acute intestinal catarrh. Generally there are no other local symptoms to call attention to the condition than *chronic diarrhea*. More rarely there are in addition colicky pain and tenderness over the abdomen. The diarrhea often alternates with constipation, and this is most apt to be the case when the disease is of idiopathic origin and affects only the large intestine (Nothnagel). *Constipation* is constant in those cases in which *atrophic alterations* occur in the glandular and muscular coats, as well as in those in which the lesions are in the small intestines. When constipation is not present the stools are thin, pale, sometimes fermented, emitting offensive odors, and vary greatly in number and quantity. There is commonly present visible mucus. When the small bowels are also implicated, food-remnants are found in the dejections (*lienteric diarrhea*). *Microscopically*, the picture does not differ from that of the acute form. That form of diarrhea occurring in *organic diseases of the heart, liver, and lungs* demands brief special mention. Here the serum of the blood is made to exude into the intestines, owing to mechanical obstruction to the return of the venous blood, and this results in a liquefaction of the feces. The *stools* are apt to be most copious and numerous during the morning hours. Sometimes an irresistible desire to evacuate the bowels seizes the patient as soon as his feet strike the floor on rising in the morning; two or more serous discharges follow each other at short intervals. Subsequently, all discharges cease until the following morning, when the same symptoms are repeated. The *general nutrition* suffers visibly in chronic enteritis, and emaciation eventually becomes pronounced. I have also noticed slight pyrexia in the evening hours.

Differential Diagnosis.—Among the diseases likely to be confounded with acute catarrh of the intestines are *typhoid fever*, *dysentery* (diseases in which diarrhea is a cardinal symptom), *peritonitis*, and *colic*. The chief differential features between simple colic and enteric catarrh may be contrasted thus:

Enteric Catarrh.	Colic.
Diarrhea is generally present.	Constipation is present.
Fever may be slight or marked.	No fever.
Pain is griping, and followed by diarrheal stools.	Pain is colicky, more severe, and is not followed by diarrheal discharges.
Tenderness in the intervals between pains.	No sensitiveness on palpation.

From *peritonitis* we may readily distinguish catarrh of the intestines by the more intense pain and tenderness, by the constipation, the greater tympany, the constitutional disturbance, the anxious face, thoracic respiration, and immobility of the patient, all of which characterize the former disease. The characteristic symptoms of *typhoid fever* (the typical temperature-curve, swelling of the spleen, eruption, Widal test) and of *dysentery* (scanty, frequent stools, tenesmus) are easily separable from enteric catarrh. In children, however, the diagnosis between typhoid fever and simple catarrh of the bowels offers considerable difficulty; but the temperature-record, the enlargement of the spleen, the characteristic eruption, and the Widal reaction, taken unitedly, will warrant the diagnosis of typhoid fever and exclude acute enteritis.

In diagnosticating chronic intestinal catarrh we may have difficulty in eliminating *lardaceous disease of the bowels* and *ulcerations*. The latter condition will be excluded hereafter. Boas recommends lavage in the diagnosis (about one liter of lukewarm water through a rectal tube); the funnel is then lowered and the dejecta siphoned off. If the recovered fluid contain mucus, catarrh is present. *Amyloid degeneration* is a general disease, affecting primarily other organs than the bowel, and hence lardaceous diarrhea is always preceded by the clinical indications of disease (enlarged viscera, albuminuria) elsewhere. The condition also gives a definite *etiology* as a rule.

Prognosis.—The prognosis in uncomplicated cases is favorable, though the possibility of a merging into the chronic form must be borne in mind. Occurring in weakly subjects and in the course of debilitating affections, *acute catarrh* of the intestines may endanger life. Its duration varies much—from three to ten days or more—according as the type of the individual case is mild or severe.

The prognosis in the *chronic forms* is moderately good as to life, though as to cure it is not so, the disease often enduring for many years together, or as long as the chronic conditions producing it remain unremoved. It sometimes exhausts the system of those suffering from serious causal affections of a chronic nature, and occasionally it ultimately proves fatal. The prognosis will depend largely upon the character of the etiologic affection, but intestinal catarrh invariably renders the prospects of life more gloomy.

Treatment.—Respecting the treatment of this affection the views of the profession have undergone many changes, even within recent years; hence it may be reasonably inferred that our present therapeutic methods are by no means satisfactory.

Hygienic and Dietetic Management.—In the not uncommon mild cases, due to errors in diet, a mild purgative, followed by proper *dietetic treatment*, is all that is required. Albuminous food in liquid form, such

as skimmed milk, weak broths, and even semi-animal articles of diet, as eggs, oysters, sweet milk with seltzer, are usually well borne. In the severe forms predigested liquid foods only should be allowed. When the chief seat of the disease is in the large intestine, we may allow easily digested starches and certain green vegetables (arrow-root, sago, lettuce, water-cress); the coarser vegetables, all fats, and most fruits should be withdrawn absolutely. *Rest* in bed is especially beneficial in that it serves to keep the abdomen warm and mitigates the pain and diarrhea, and, in short, cures the disease. Sinapisms should be applied at the outset until the skin is reddened, succeeded by light linseed poultices until the local sensitiveness has, in a great measure, subsided; after this a flannel band may be applied. The local abstraction of blood by a few leeches, applied to the abdomen or anus, is beneficial in the early stages in severe types of enteric catarrh, provided the patient's strength is good.

Medicinal Treatment.—It is sound practice to prescribe a mild cathartic (castor oil, calomel, or rhubarb, followed by a saline) with a view to getting rid of decomposable intestinal contents. Combined gastric lavage and high intestinal irrigation has recently yielded excellent results in my hands; it is an appropriate method of overcoming the fermentative processes that tend to excite and maintain the condition.

If the chief tenderness be localized in the right iliac fossa, corresponding to the course of the colon, a simple enema, slowly given, will stimulate the bowel sufficiently and cleanse it more effectually than a cathartic. Subsequently, chief reliance is to be placed on intestinal antiseptics and astringents, though it must be recollected that the selection of internal remedies must, in part, be influenced by the etiologic indications. For instance, if the cause has been exposure to cold or wet, besides the efforts directed at the local condition diaphoretics and febrifuge mixtures are serviceable. I have found the following combination to be of benefit in controlling the local inflammatory action:

℞. Salol, ʒss (2.0);
Creasoti, ♏x (0.666);
Bismuthi salicylat., ʒj (4.0).
M. et ft. capsulæ No. xx.
Sig. One every three hours.

If pain be troublesome, opium or phenacetin may be combined with the above formula.

In many instances the secretions of the intestinal tube are decreased for a considerable period after the most active symptoms have been subdued. Here we must supplement the natural juices of the bowel; this may be satisfactorily accomplished by the following agents:

℞. Pancreatin, ʒj (4.0);
Sodii bicarb., ʒij (8.0).
M. et ft. chart. No. xij.
Sig. One an hour after meals.

In cases in which the large intestine is chiefly affected, and when the condition does not yield to internal medicines, treatment by medicated

colonic irrigations are useful. When there is reason to suspect that the main lesion is in the large bowel, small enemas of starch-water (℥ij—64.0), with laudanum (♏ xx–xxx—1.33–2.0), every four to six hours, are also efficacious. If colicky pain be severe, morphin (gr. $\frac{1}{8}$—0.008) should be given hypodermically in addition to the measures before suggested. If the diarrhea shows no tendency to abate after forty-eight hours of the general treatment above outlined, large doses of bismuth (gr. xxx–lx—2.0–4.0) every three or four hours should be tried. In my own hands lead acetate (gr. ij—0.129), with the extract of opium (gr. $\frac{1}{8}$—0.008) in pill-form, has proved a most efficient combination. The thirst is best relieved by chipped ice in small quantities or by carbonic acid and Apollinaris waters. For distressing flatulence we may prescribe the alkaline carbonates, or spirits of ammonia, and some carminative. The oil of cajeput is a most valuable drug in the treatment of excessive fermentation (Murrell).

In *chronic catarrh* of the intestines the local treatment is of paramount importance. Daily irrigation of the bowel with a weak solution of some antiseptic agent, as salicylic acid (gr. v–℥j—0.324–32.0), boracic acid (gr. x–℥j—0.648–32.0), creolin (♏ v–℥j—0.324–32.0), or with some such astringent as tannin (gr. v–℥j—0.324—32.0), or finally with an alterative, such as silver nitrate (gr. $\frac{1}{4}$–℥j—0.016–32.0), will be found to be serviceable. The latter solution is a most excellent remedy, but sometimes excites pain if too concentrated. I often use a mild antiseptic or astringent with the foregoing, giving each on alternate days, and thus obtain happy results. The only appliance needful is a fountain syringe with a soft-rubber end-piece, which should be gently introduced for a considerable distance into the bowel. The fluid used should be warmed to 90° F. (32.2° C.), and the quantity administered at each sitting should be not less than 2 to 3 pints (1–1.5 liters); this should be allowed to flow into the bowel slowly. The patient should, as a rule, assume the dorsal decubitus, though if the fluid is to be carried as high up as possible, the knee-elbow position may be assumed or the patient may be placed on the left side with the hips elevated. Again, turning him from side to side during the irrigating process may be warmly recommended.

The same careful attention must be paid to *hygienic details*, and especially to the diet, as is directed in the acute form. In addition, flannel should be worn next the skin both in winter and summer. If the strength will admit of it, cold baths are useful.

A stay at a suitable spa (Saratoga, Bedford, Virginia Springs, Carlsbad, Kissingen) often produces most satisfactory results.

Among internal agents, zinc oxid (gr. v to x—0.324–0.648—t. i. d.), silver nitrate, lead acetate, and alum, given with tonics, such as strychnia, arsenic, and iron, are especially to be recommended.

The management of this troublesome malady depends upon the indications furnished by the causative affections. No method of treatment, however, can succeed that is not carried out patiently, systematically, and over long periods of time.

DIARRHEAS OF CHILDREN.

ACUTE GASTRO-INTESTINAL CATARRH.

(*Acute Gastro-enteric Infection; Summer Diarrhea; Gastro-enteritis; Cholera Infantum; Mycotic Diarrhea.*)

Definition.—This is the usual intestinal trouble that prevails during the warm summer months. It usually takes the form of an epidemic, and its course is manifested by a sudden onset, high fever, irritability of the stomach, frequent watery evacuations, and symptoms of nerve-involvement. This form of diarrhea usually follows an attack of acute indigestion, in which it very frequently has its origin (*acute dyspeptic diarrhea*). Acute gastro-intestinal catarrh (*cholera infantum*) stands midway between acute indigestion and ileo-colitis.

Etiology.—Two important conditions seem to be necessary to influence the disease—*temperature* and *diet*. A general and well-recognized belief associates special danger with the second summer of children. Out of nearly 2000 fatal cases collected by Holt, only 3 per cent. were exclusively breast-fed. Generally speaking, the disease has its origin in some irregularities in artificial feeding. Heat and season are important elements in the continuation of the disorder when once commenced.

It is seen from May to September, the greatest prevalence occurring in July. The pauper element of large cities furnishes most instances.

Flexner and Holt[1] assert that the bacillus dysenteriæ may be isolated from the intestinal discharges, and from the intestinal mucosæ in a large percentage of cases developing along the Atlantic coast of the United States, during the summer months. Holt found bacillus dysenteriæ in 50 per cent. of cases at the Babies' Hospital of New York. The Flexner-Harris type of bacillus is most often encountered, while the "Shiga" type is but occasionally recovered. It is common for cultures to develop streptococci in connection with the *bacillus dysenteriæ*, and both organisms appear to grow luxuriantly together, which renders it impracticable to decide whether the lesions of the intestine and the general symptoms depend upon one or both of these organisms.

Booker, Jeffris, Baginsky, and Metschnikoff affirm that the proteus class of bacteria are commonly present, and that they are pathogenic. The "bacillus dysenteriæ" reacts with the serum of infected children.

Pathology.—A catarrhal swelling of the mucosa of the large and small bowel is present; the mucosa itself is pink in color from capillary congestion. Peyer's patches are enlarged. The whole intestinal tube shows an early stage of inflammation (ileo-colitis). In addition there is most likely some involvement of the sympathetic nerves, leading to dilatation of the capillaries and transudation of serum into the intestine, and to alterations of the pulse, temperature, and respiration. Its nature is paralytic, and closely resembles in its results experimental sections of the sympathetic nerves. The changes in the other organs are slight. Broncho-pneumonia frequently occurs. The spleen is often swollen, the brain is anemic, and the kidneys are congested.

[1] Rockefeller *Institute for Medical Research*, 1904.

Symptoms.—Clinically, there are three forms of acute enteric infection: (1) acute dyspeptic diarrhea; (2) cholera infantum; and (3) ileocolitis.

(1) *Acute Dyspeptic Diarrhea.*—There may be merely an increase in the number of stools, with or without fever; restlessness is usual at night. This condition may continue for two or three days, when the stools become more frequent and offensive, containing undigested food and curds. The *odor* by this time is very pronounced. Frequently the disease has a sudden onset, with vomiting, griping pains, and fever, which may quickly rise to 104°, 105°, or 106° F. (40°–41° C.). *Convulsions* may be the commencement of the attack. The abdomen is sensitive and swollen, and the child lies with its legs flexed on the stomach. The *stools* consist of grayish or greenish-yellow feces (mixed with curds, portions of undigested food) and some fluid. In children two years of age and older the stools may contain unripe fruit or large curds from excessive drinking of milk. Relapses are frequent, and during hot weather the frequency of the attacks may lead to a persistent entero-colitis.

In delicate children a severe attack, especially if it is accompanied by convulsions, may prove fatal.

(2) *Cholera Infantum.*—The *initial* symptoms are sudden. The child voids immense stools, at first fecal, if no preceding diarrhea have been present. Soon they become watery, light yellow or greenish in color; frequently they are so thin and colorless as to pass through the napkin without leaving a stain. At times they contain a few yellow or greenish flocculi or a mass of mucus, and in all cases they are odorless. Very often the stools are brown and liquid, with a small quantity of fecal matter, having a peculiar musty odor that clings to the napkin and child for days. The number of stools per diem may vary from six to thirty, and a most remarkable feature is the fact that they are evacuated with considerable force.

The *stomach* becomes irritable, refusing everything; even ice is rejected as soon as swallowed. The vomitus at first contains bile, while later it becomes serous. The *appetite* is, of course, entirely lost; intense thirst prevails, the little patient drinking at every chance and following the receding glass with eager eyes. The *tongue*, moist at first, soon becomes dry and pasty; the abdomen is collapsed. The *temperature* is always high—105° or even 108° F. (40.5°–42.2° C.); and the *pulse* small and very frequent—130 to 180 beats per minute. The *breathing* is shallow and irregular, and the expression anxious and staring, but soon becomes dull. The urine becomes dark and scanty.

With this array of symptoms there is a striking and appalling change in the child's general appearance. Within a few hours it can scarcely be recognized; the face has become pale and pinched, the eyes and cheeks sunken, the eyelids and lips wide apart from loss of muscular control, the muscles flabby, the bones prominent, and the skin greenish or cadaverous, hanging in loose folds from the wasted frame.

Collapse comes on soon: the hands, feet, nose, and breath become cool, the respirations more unequal, and there are drowsiness and utter apathy. When life is near its close, vomiting stops, the whole surface becoming cool and clammy as the patient sinks into a state of coma, with

injected eyes and contracted pupils. At last the end is reached quickly, preceded perhaps by a slight convulsion. The *duration* of the disease is short; it may prove fatal in from one to four days.

(3) *Ileo-colitis.*—This may follow acute dyspeptic diarrhea, cholera infantum, or complicate the acute infections of childhood. The *symptoms* develop acutely. At the outset there may be vomiting, but it is not persistent, and the stools are greenish, feculent, often showing masses of casein. Later the discharges are increased in frequency, are small, and contain also blood and mucus. In severe cases pain and straining are distressing features. The abdomen is prominent and there is tenderness along the course of the colon. The disease presents high fever.

The *course* is variable. It may be acute—three to six days—terminating either in convalescence or death due to exhaustion. In other instances the acute symptoms subside, particularly the fever, while moderate diarrhea continues and is attended with wasting and debility. Gradual recovery may ensue, though more commonly relapses occur and death follows from broncho-pneumonia or an intercurrent acute attack.

Treatment.—The treatment of *acute gastro-intestinal catarrh* divides itself into hygienic, dietetic, and medicinal measures. If a child is attacked in the city during the summer and does not yield to treatment in two or three days, it should be sent to the country or seashore. In the case of a child under two years this is absolutely imperative. Fresh air is important in all diarrheal disorders, and all cases should be kept out of doors as much of the time as possible. Children should be kept quiet. Bathing is soothing, insures cleanliness, and, what is very important, reduces the temperature.

Dietetic treatment is of great importance. It should be remembered that digestion is arrested in the early stage, hence to give food at this stage is to do harm. Thirst may be controlled by ice- or albumin-water, toast-water, or gum-water, with a little brandy. Buttermilk twice daily has given excellent results.

Medicinal Treatment.—The first step is directed against the acute indigestion and the active putrefaction going on in the intestine. The indication, therefore, is to empty thoroughly the alimentary tract as soon as possible, and no other treatment should be considered until this end has been accomplished. Whenever vomiting persists the stomach should be washed. In older children emetics will favor complete emptying of the stomach, but are never to be given to infants under two years. For the intestine calomel and soda may be used; for the colon irrigation: this is advisable in all cases, as it hastens the effect of the calomel, and removes at once much irritating and offensive material. Opium should not be used until the whole intestinal tube is cleansed, and then cautiously. Spirits of chloroform, or camphor, is a better remedy for the pain than opium in any form. In older children the hypodermic injection of morphin and atropin in appropriate doses frequently controls the symptoms. Bowles has used lactic acid in the maximum dose of $1\frac{1}{4}$ grains every hour, and found it to control the symptoms in from twenty-four to forty-eight hours. Thus far the results of serum treatment have been disappointing.

Treatment of Cholera Infantum.—In this form of infection of the

intestinal tract we are likely to forget that we are called upon to treat a case of acute poisoning. The toxic material acts both powerfully and quickly as a cardiac and systemic depressant. It also acts toxically upon the nerve-centers, and paralyzes the vaso-motor nerves. According to Holt, the leading indications are—(*a*) to empty the stomach and intestines; (*b*) to supply the body with fluid to offset the great loss by vomiting and purging; (*c*) to counteract the effect of the poison on the heart and the nervous system; (*d*) to reduce temperature; and (*e*) to treat the symptoms as they arise. In the first condition thorough stomach and intestinal cleansing is absolutely necessary. Moreover, we cannot depend on emetics or purgatives to arrest pain and to limit the effect of the poison on the nervous system; a hypodermic injection of atropin and morphin is essential. Morphin must be given with discrimination to young children, especially when the vomiting and purging are slight; it is especially contraindicated when stupor or collapse seems near. Small doses repeated are better than larger single doses. Holt gives gr. $\frac{1}{100}$ (0.0006) of morphin, with gr. $\frac{1}{800}$ (0.00008) of atropin, as the first dose in a child one year old. In supplying fluid to the exhausted tissues it is useless to attempt to give them by the mouth, or even by the rectum, as by both avenues it would be rejected. An injection into the cellular tissues of the buttocks, back, or thighs of a saline solution (40 grains—2.59—of common salt to a pint of sterilized water) is the best way to meet the drain. One pint (half liter) may be used every twenty-four hours, and larger quantities may often be used with advantage. Baths must be given to control temperature, and ice-bags should be placed to the head. Ice-water injections will aid in the control of temperature, and ice-suppositories act efficiently when the water is not retained. Stimulants may be given hypodermically. During the active stage nothing should be allowed by the mouth except iced brandy or champagne.

The dietetic management and internal treatment of *ileo-colitis* are similar to that of the preceding variety. A dose of castor oil or of calomel is to be promptly administered and followed in a few hours by copious irrigations of the colon, preferably with tepid saline solution (strength 7 : 1000). Later a small quantity of a thin starch solution, to which ♏j to iij of laudanum has been added, may be gently thrown into the rectum, to be repeated once or twice daily. After the acute stage is over a weak silver nitrate solution may be employed.

CELIAC DISEASE.

(*Diarrhœa Alba; Diarrhœa Chylosa.*)

Definition.—A form of intestinal catarrh marked by copious fetid and frothy discharges resembling gruel.

Pathology.—Although ulcers have been noted in the intestine, the pathology of the disease is not known. Says Osler: This affection resembles somewhat the disease in adults known as "hill diarrhea" or the "white flux" of India.

Etiology.—The disease is limited chiefly to children from one to five years old. The filaria sanguinis hominis has been found in the feces in cases of diarrhœa chylosa.

Symptoms.—The disease is of slow development, and the character-

istic feature consists of copious *diarrheal* (though not watery) *stools*, resembling gruel or oatmeal-porridge. These are also frothy (*frog-spawn*) and horribly fetid. The *physical signs* consist of a moderate distention of the abdomen and a boggy sensation that is imparted to the palpating finger. The *general features* may be summated in gradually increasing emaciation, debility, and pallor. The disease terminates fatally as a rule.

The **treatment** is purely symptomatic, unless the presence of parasites be suspected, when large antiseptic enemata should be given.

PHLEGMONOUS ENTERITIS.

THIS is a suppurative inflammation of the submucous layer of the intestines. It is among the rarest of grave maladies, especially as an irrelative disease. It may be diffuse or take the form of a circumscribed abscess. Rarely it occurs as a complicating condition in septico-pyemia and in malignant types of the exanthemata, resulting in the formation of abscesses that usually have their seat in the duodenum. Phlegmonous enteritis may be secondary to strangulated hernia or intussusception.

Symptoms.—The *local signs* simulate closely those of peritonitis. Among the symptoms *vomiting* is prominent, though not diagnostic; it is always severe, and may become stercoraceous. *Pain* and *tenesmus*, when due to obstruction, are intense. Rigors more or less severe have been observed. The *temperature* is high, and its curve is somewhat typical of the fever of suppuration. The disease is very fatal, the patient passing from a condition of extreme prostration to one of utter collapse.

Treatment.—The physician's task is confined to an attempt to support the powers of the patient and to relieve his inordinate suffering. The surgeon's aid should be invoked early in cases of obstruction.

CROUPOUS OR DIPHTHERITIC ENTERITIS.

Definition.—An intense inflammation of the intestinal mucosa, accompanied by a croupous exudate; it occurs in connection with a variety of conditions. If from any cause the epithelial covering is destroyed, agents that set up local inflammation may excite a croupous exudate.

Pathology.—There are two sets of morbid lesions to be distinguished: (1) The first and most important class exhibits a croupous deposit varying greatly in thickness and in area. Its color is variable, being sometimes of a *grayish* or *grayish-white hue*, frequently *grayish-yellow*, and rarely *blackish*. I have almost invariably seen these lesions in the colon. (2) In the second group the solitary follicles alone are inflamed, and covered with diphtheritic deposit.

The *etiologic* factors may be (*a*) mechanical irritants (impacted feces, intestinal sand, gall-stones); (*b*) chemical irritants (ammonia, acids, mercury, arsenic); (*c*) secondary to acute infectious and certain chronic complaints (Bright's disease, pyemia, carcinoma, diabetes, tuberculosis, and anemias).

Symptoms.—When mechanical irritants give rise to symptoms, they do not differ from those due to stercoral ulcers, and there is no way of recognizing the croupous deposits unless they be discharged *per rectum* and are detected in the stools. In cases that arise from the action of irri-

tant poisons vomiting and purging are well marked and the dejections contain blood-stained mucus. We cannot be certain about the presence of croupous deposits in toxic cases unless they be found in the discharges. When phlegmonous enteritis occurs as a complicating condition in infectious diseases, the symptoms are almost completely veiled. The symptomatology of the follicular variety cannot be separated clinically from that of follicular ulceration.

The **treatment** is that of the causal conditions or affections.

SPRUE.

(*Psilosis.*)

THIS has been defined as "an insidious, chronic, remitting inflammation of the whole or part of the mucous membrane of the alimentary canal, occurring principally in Europeans who are residing or have resided in tropical or subtropical climates" (Manson).

The principal **morbid changes** consist in patchy or general destruction of "the surface of the mucosa in all degrees, from slight erosions to complete disintegration of the villi, glands, and follicles." Congestive, catarrhal, ulcerative, and cirrhotic changes may be all combined in one and the same case.

The **etiology** is unknown, although the disease is probably of microorganismal nature. Residence in hot climates and previous affections of the alimentary tract are the main predisposing causes.

The leading **symptoms** are, according to Manson, irregular action of the bowels, and the passage of copious, pale, drab-colored, yeasty-looking, sickly-smelling stools. The complexion is dark or muddy; there is emaciation and the abdomen is distended. Weakness, loss of memory, and irritability of temper are common. The oro-cavity is inflamed and the seat of erosions, cracks, and superficial ulcerations. Brunton has pointed out that Indian Hill diarrhea differs from sprue in that soreness of the mouth and anus is absent in the former.

Early appropriate **treatment,** which is principally dietetic (milk-diet) and hygienic, checks the progress of the disease.

CHOLERA MORBUS.

(*Cholera Nostras; Sporadic Cholera.*)

Definition.—A self-limiting disease, characterized by serous vomiting and purging, colicky pains, and often muscular cramps.

Pathology.—No constant anatomic changes have been noted. They are analogous to those seen in acute gastro-enteritis, though cases have terminated fatally in which no morbid lesions were found *postmortem.*

Etiology.—Among predisposing causes, the *age* and the *season* exert the most prominent influence. The condition may appear in subjects under two years, when the term "cholera infantum" is employed; but it is oftener met with in older children and adults. It is almost invariably seen during the heated term in temperate zones, from the latter part of June to September, and it is especially prevalent during the

months of July and August. Bad hygienic environment, foul air in particular, has a noticeable effect, and, though not as yet absolutely proved, it may be safely inferred from the symptomatology and general clinical course of the affection that it is of microbic origin. Among other factors are improper food, particularly unripe fruit, cucumbers, egg-plant, and exposure to cold and wet. Various *organisms* (especially the Finkler and Prior spirillum) have been found present. No one variety, however, has been definitely found to be the cause of the condition. Virulent specimens of the bacillus coli commune, and even of the streptococcus, have been noted.

Clinical History.—The *onset* is often sudden, and is marked by abdominal pain, vomiting, and diarrhea. At first the *vomitus* consists of food, and later of bile and mucus. The *dejections* are fecal in character at the onset; though they soon become watery, and may resemble the rice-water stools of Asiatic cholera.

Physical examination reveals only tenderness on pressure over the abdomen, and particularly over the epigastric region.

General Symptoms.—Cramps in the calf muscles are common. The temperature varies greatly, ranging from 100° to 106° F. (37.7° to 41.1° C.). The skin-surface, however, and more particularly that of the extremities, feels cool, and owing to this fact the rectal temperature should be recorded. The pulse becomes rapid and feeble as the case progresses. The face is pale or even cyanotic, and the features look pinched. The extremities lose their plumpness, and the patient usually appears prostrated and mentally dull. The urine is scanty, high-colored, and sometimes albuminous, and thirst is extreme. There is a group of cases that develop subacutely, and in these the symptoms tend to persist.

Differential Diagnosis.—The symptoms of cholera morbus resemble so closely those of *Asiatic cholera* as to preclude the possibility of a differential diagnosis from the symptoms. A bacteriologic examination of the stools, however, permits a certain discrimination; and during a cholera epidemic the distinction between these affections is thus made. The effects of certain direct irritants, as in poisoning by ptomains and toxic doses of arsenic, must be excluded by the history.

Prognosis and Duration.—The duration of the disease varies from three to four hours to two days. It is rarely fatal, though in persons suffering from such chronic affections as Bright's or cardiac disease, and also in the aged, the prognosis is only guardedly favorable. An element of danger is profound collapse. Otitis media is occasionally seen, although most cases without sequelæ recover.

Treatment.—The *diet* must be rigorously restricted, and predigested milk and animal broths are to be prepared as lightly as possible until convalescence has been fairly established. The comfort of the patient is much enhanced by keeping him at absolute rest. *Local measures* are useful in combating pain and vomiting. A large mustard-paste applied to the stomach and abdomen, followed by linseed-poultices that are to be worn constantly, has a strong influence in accomplishing the relief of the symptoms before mentioned. If indigestible substances have been taken prior to the attack, prompt though mild laxatives are to be given at the beginning of the treatment. For the excessive thirst chipped ice, over which a little brandy has been sprinkled, is effective.

For controlling the morbid sensitiveness, on which the pain, nausea, and the diarrhea depend, we have a remedy *par excellence* in the hypodermic administration of morphin. The dose should vary (gr. $\frac{1}{4}$ to $\frac{1}{2}$—0.016 to 0.032) according to the severity of the symptoms, and I have rarely found it necessary to give a second dose. Not only are the pain and diarrhea subdued, but the circulation is re-established. It has also been recommended to administer opium by the mouth for these symptoms, but the results are less satisfactory. The other points in the treatment of this affection are identical with those discussed under the treatment of Gastric and Enteric Catarrh.

INTESTINAL INFARCTION.

A FEW instances of occlusion of the superior mesenteric artery by an embolus have been recorded recently. The condition produces hemorrhagic infarction of the small intestines, and is marked by grave and usually fatal symptoms. Its *causes* are sometimes obscure. The cases that have come to autopsy have shown intense congestion, with a swollen, blood-infiltrated state of the jejunum and ileum. Osler has seen three instances: in one there were numerous vegetations on the mitral valves from which the embolus was probably derived; in another the superior mesentery was plugged at its orifice; and in the third the artery was blocked by a portion of the fibrous clot of an aneurysm of the aorta near the diaphragm. The *symptoms* are urgent. Quite often diarrhea is present from the first, the dejections sometimes becoming blood-tinged. Soon the characteristically grave symptoms of intestinal obstruction supervene—viz., *great pain*, *vomiting*, and *constipation* (less commonly diarrhea), with *tympanitic distention* of the abdomen (generally). The condition cannot be recognized from the symptoms on account of their resemblance to the various forms of obstruction, yet its probable existence may be inferred from the presence of the known causes.

INTESTINAL ULCERS.

DUODENAL ULCER.

Definition.—A small, round perforating ulcer of the duodenum (*vide* p. 804). It may be primary or secondary.

Pathology.—The morbid characteristics are so nearly identical in appearance and nature with those of peptic ulcer of the stomach that they scarcely demand a separate presentation. The *seat* of the ulcer is with few exceptions above the orifice of the common bile-duct. When these ulcers heal the resulting cicatrix produces stenosis, which in turn leads to dilatation of that portion of the duodenum back of it, and finally of the stomach also. Progressive cicatricial contraction may completely close the *ductus communis* and the pancreatic duct or portal vein. Protective adhesive inflammation between the duodenum and the adjacent parts (pancreas, gall-bladder, liver) often prevents complete perforation of the duodenal wall: when perforation does occur, the peritoneal cavity may be opened, causing peritonitis, or a fistulous communication may be established with the gall-bladder, liver, or pancreas.

Etiology.—Though the duodenal ulcer has, as a rule, the same mode of origin or pathology as the gastric ulcer, it is a remarkable fact that extensive burns of the skin-surface of the body are quite prone to be followed by a perforating ulcer of the duodenum (6.2 per cent. of fatal burns, Fenwick's), while gastric ulcers are seldom caused in this manner. As in other forms of duodenal and gastric ulcers, the circulation is arrested by an embolus (from decomposing masses of blood) at some point in the mucous membrane, the acid gastric juices subsequently digesting the part thus deprived of its blood-supply. The disease is met with in chronic Bright's disease, in pneumonia, and in association with gall-stones.

The influence of *sex* and *age* as causal factors is notable and in striking contrast with their import in gastric ulcer. In the latter disease most instances occur among young females, while in duodenal ulceration they occur, as a rule, in males between the twentieth and fortieth years. These differences respecting their etiology are inexplicable. W. J. Mayo met 74 cases in a total of 231 gastric and duodenal ulcers; he found most of the latter situated close to the pylorus. They are commonly mistaken for gastric ulcers just within this aperture. In duodenal ulcer, gastric ulceration is associated in 50 per cent. (Mayo, Moynihan). Codman[1] holds that ulcer is more common below than above the pyloric orifice. In the necropsy records of 3000 cases, Paus found duodenal ulcer in 0.73 per cent. and gastric ulcer in 2.77 per cent.

Clinical History.—Perhaps no real distinction between the symptoms of gastric ulcer and those of its homologue affecting the duodenum can be said to exist in most instances. For example, Kemp found gastric hypersecretion in his 10 operative cases. A probable diagnosis of ulceration of the duodenum has, however, been repeatedly made, and sometimes verified by the subsequent autopsy. Günzburg[2] noted a tympanitic zone in the region of the quadrate lobe of the liver, presumably due to the dilated duodenum passing behind this lobe. If duodenal ulcer be classed with gastric ulcer, there is great danger that the true nature of many cases will be overlooked. The difference in the symptomatology in the two forms of ulceration is owing solely to the difference in locality.

The distinctive features of this disease may be shown by presenting its leading symptoms by the side of those characteristic of gastric ulcer:

Duodenal Ulcer.	Gastric Ulcer.
Usually occurs between 20 and 40 years, except when due to external burns.	May occur at any age after childhood.
Males are more frequent sufferers than females, in the proportion of 10 to 1.	Females are the chief sufferers.
Onset marked by intestinal hemorrhage, which may recur at intervals.	Hematemesis often occurs, preceded by other gastric symptoms, as a rule.
The melena may be preceded, or accompanied, by hematemesis, though not generally.	Blood may appear in the stools, usually after hematemesis.
Blood in the discharges often is bright red, profuse, but not marked as in gastrorrhagia; dark and tarry from the action of acid chyme when slight.	The blood in the dejections is dark and tarry from the action of the gastric juices.
Pain, due to acid, may come on late, two to four hours after meals; more often absent. It is localized a little above and to the right of the umbilicus. Pain relieved by eating, owing to absorption of acid and closure of pylorus.	Pain paroxysmal, greatly influenced by taking food. Pain sharply localized in the epigastric region, about two inches below the ensiform cartilage. Usually pain is aggravated by taking food.

[1] *Boston Med. and Surg. Jonr.*, November 25, 1909.
[2] *Deutsche medizinische Wochenschrift*, Berlin, July 14, 1910.

Duodenal Ulcer.	Gastric Ulcer.
Hyperchlorhydria with hypermotility.	Hyperchlorhydria without hypermotility.
Gastric crises occur without reference to time of taking food.	Gastric crises come on soon after taking food.
Vomiting inconstant without relation to ingestion of food, and affords no relief.	Vomiting more common (during painful crisis) and affords relief.
Jaundice occasionally present.	Jaundice absent.
No marked improvement after diet has been regulated.	Usually a marked improvement follows regulation of diet.
Dorsal pain-point absent.	Pain-point (tenth to twelfth dorsal vertebræ on left side) usually present.
Motor insufficiency, due to pyloric spasm, may occur.	Less common.

Finally, "whenever a young man in apparently good health is attacked by melena, with or without hematemesis, it is probable that the ulcer is located in the first part of the duodenum rather than in the stomach, the converse being the case in young women" (Fenwick).

Diagnosis.—Of the symptoms mentioned under Duodenal Ulcer, the *intestinal bleedings* and *violent* pains in the right hypochondrium, coming on from one to four hours after meals, are the most diagnostic. Attacks of pain before meals (hunger pains), with vomiting, may warrant a provisional diagnosis (Codman). The diagnosis is rendered more positive if the thread-test shows a distinct blood-spot—58 to 66 cm. from the teeth (Einhorn[1]). An *x*-ray examination after a meal of bismuth, in uncomplicated cases, shows the fluid passing into the duodenum at once with great rapidity, so that the stomach is empty before the pain begins. While hemorrhage is the leading single symptom, we must not, in attempting to estimate its significance in any case, neglect to eliminate *hemorrhoids*, *carcinoma*, *tuberculosis*, *dysentery*, and *hemorrhagic diathesis*—all conditions in which melena may occur. There may be an absence of symptoms until perforation occurs (*latent duodenal ulcer*). Moynihan speaks of fluid passing down in perforation, causing symptoms simulating those of appendicitis.

Sequelæ.—Dilatation of the stomach from pyloric spasm or following the healing of these ulcers, associated usually with chronic gastroduodenal catarrh, is not uncommon. Rarely, stenosis of the *ductus communis* is a sequel; more frequently tumors either compress or occlude the lumen of the bowel below the mouth of the duct. The symptoms presented differ from those due to stenosis above the duct, the most characteristic being the continual backward flow of bile into the stomach, sometimes attended by constant vomiting of biliary secretions.

Complications.—As in the case of gastric ulcer, so in the duodenal form, there is at times so much thickening about the base of the ulcer as to give rise to the signs of malignant tumor. This is especially true of those instances in which the base of the ulcer becomes attached to adjacent organs. Under these circumstances infection of the head of the pancreas with tumor-like swelling may occur and produce obstruction to the outflow of bile, with accompanying jaundice.

Prognosis.—The risk to life is greater than in gastric ulcer. Perhaps 50 per cent. of the fatal cases are due to perforation.

Treatment.—The treatment of duodenal ulcer is similar to that of gastric ulcer. Medical measures are to be tried, but when the diagnosis

[1] *Medical Record*, 1909, p. 549.

is no longer in dispute, timely operative intervention is to be advised. Pyloric spasm may be prevented by the use of atropin and alkalies.

Follicular ulcers have already been described under Catarrhal Enteritis (*vide* p. 835), and they have a similar pathology and etiology. When present in goodly numbers they give rise to a symptom peculiarly their own, and hence may be dignified by a separate though brief mention. The *symptoms* of the condition arising in the course of chronic enteritis often escape observation for a long time. The most characteristic manifestation is the appearance in the stools of conical-shaped masses of mucus resembling "boiled sago." Marked weakness and emaciation rapidly ensue. Among children the disease is common and assumes an aggravated form, the little sufferers quite frequently reaching their end as the result of inanition. An unfavorable termination may be due to perforation followed by suppurative peritonitis. The *treatment* coincides with that of chronic enteritis.

Stercoral ulcers are the result of the mechanical effect of hard fecal scybala (often enteroliths, due to a deposit of lime-salts) upon the intestinal mucous membrane. They occupy the sides or tops of the normal folds in the colon.

Symptoms.—There is, as a rule, a clear history of *chronic constipation*, though the physician is often called on account of the presence of *diarrhea;* this is caused by the retained hardened feces finding their way into the rectum. A *digital exploration* will now clear up the diagnosis. There are *tenesmus* and *colicky pain* in the abdomen, the latter symptom being also complained of when diarrhea is absent. The pain often occurs in severe paroxysms that may be attended with the discharge of flaky mucus, pus, and sometimes blood. Enteroliths may lie in the intestines for years together, or they may finally be discharged with the stools. The ulceration that is thus caused often passes unrecognized.

Physical Examination.—Palpation may in rare instances reveal the presence of a sausage-shaped tumor and sharply localized tenderness over the seats of ulcers.

The **prognosis** is good if the condition be not overlooked.

The **treatment** consists in thoroughly evacuating the bowels by salines and simple enemata, persistently used. Subsequently these cases are to be treated as other non-specific ulcers of the bowels.

Simple ulcerative colitis is a not uncommon complaint, and one that is frequently associated with chronic intestinal catarrh. The ulcers may be quite extensive, removing the greater portion of the mucous membrane, though in several instances I have observed cases at the Episcopal Hospital that were superficial; these were confined almost solely to the mucosa. The muscular layer of the gut was greatly hypertrophied and its lumen increased in every instance. The non-ulcerated portions of the mucosa looked, in part, quite pale, and in part quite dark. Polypoid growths have been observed situated between the ulcers.

The **etiology** is obscure. The disease is met with most frequently in persons past middle life, and it is quite probable that chronic enteritis

sustains a causal relation. Those whose constitutions have been enfeebled by previous disease or an improvident hygienic environment are the chief sufferers.

Symptoms.—The clinical features are ill defined at the onset, and are often erroneously ascribed to indigestion. *Diarrhea* (lienteric in character) is its most prominent symptom, and with it constipation may alternate. Pus and blood are absent with the rarest exceptions. The general health soon suffers greatly, the patient becoming weak and emaciated.

The **course** of the disease is subacute, tending to become chronic.

The **diagnosis**, apart from a consideration of the symptoms above mentioned, requires the elimination of *dysentery*—an easy task as a rule. The disease resembles most closely the amebic form of dysentery, hence in dubious cases a microscopic examination of the feces should not be neglected.

Prognosis.—This is unfavorable during the earlier stages in the aged. The tendency to chronicity of the disease must be considered.

The **treatment** embraces (*a*) a careful regulation of the diet, consisting in a restriction of the patient to liquids and semi-solids during the acute stage; (*b*) the administration of a gentle laxative, followed by antiseptics and astringents (bismuth gr. xxx—2.0—combined with salol gr. v—0.324—every four hours); (*c*) the more serviceable local measures in the form of enemata, among the best being silver nitrate (gr. ¼ ad ℥j —0.016 to 32.0) or creolin (2 per cent.).

Solitary Ulcers.—"Two instances of ulcer of the cecum, both with perforation, have come under my observation, and in one instance a simple ulcer of the colon perforated and led to fatal peritonitis" (Osler).

The **diffuse catarrhal ulcer** is inseparable from acute enteritis; the **cancerous ulcer**, **tuberculous ulcer**, and **amebic ulcer** are alluded to under their respective heads.

APPENDICITIS.

Definition.—A catarrhal, ulcerative, or interstitial inflammation of the appendix vermiformis. It must be confessed that, according to our present views, appendicitis is a surgical rather than a medical affection, particularly from the standpoint of treatment. Knowing from personal experience and observation, however, that general practitioners are constantly meeting with cases of appendicitis, its prompt clinical recognition by the latter is not only a matter of interest, but also of great practical importance for two reasons: First, in order that surgical intervention can be instituted at the proper moment; and secondly, because appendicitis is the leading serious disease of the intestinal tract.

The term "appendicitis" includes the affections *typhlitis* (inflammation of the cecum) and *perityphlitis* (a similar involvment of the connective tissue behind the cecum) by reason of the fact that with few exceptions when the symptoms of the latter affection are presented the appendix vermiformis is the part primarily affected. To the physicians

and surgeons of America belongs the credit of having first established the truly important rank of appendicitis.[1]

Anatomic.—Without any known function the human appendix vermiformis represents the remains of the enormous cecum of inferior animals, especially rodents and herbivora. Clado asserts that the vermiform appendix is kept in position by two folds of peritoneum, a meso-appendix, which is attached to the iliac fossa, and a second fold, perpendicular to the first, which is attached to the posterior portion of the small intestine.[2] A lymphatic gland generally occupies the angle formed by the appendix, cecum, and the small gut; this receives all the lymphatic vessels of the appendix. In the female a lymphatic connection may exist between the appendix and the right ovary. The size of the appendix varies greatly. Ferguson,[3] after measuring 200 appendices, gave as the average length 4½ inches (11.4 cm.), and as the diameter, that of a No. 9 English sound—about a quarter of an inch (0.62 cm.). Berry's studies, which are partly based upon personal examination of 100 bodies, and partly upon comparison of his own results with those obtained by other investigators, gives the average length in all the observations as 9.2 centimeters (3.6 inches). The caliber is ordinarily of the size of a goose-quill. Very exceptionally, as in a case reported by Swan, there is a congenital absence of the appendix. Its two fibromuscular coats (external longitudinal and internal circular) are thick; its mucous membrane contains lymphoid elements in abundance. The blood-supply is derived from the ileo-colic artery at the valve, a single branch running to the end of the appendix, while the nerves are derived from the superior mesenteric plexus of the sympathetic. Shortly after middle life the cavity of the appendix becomes obliterated. Its blind extremity points most frequently toward the spleen. The appendix may lie behind the cecum, and sometimes partly to its inner side, its tip almost touching the liver or the gall-bladder. In not a few instances it dips downward, passing over the brim of the pelvis. There is no adjacent organ to which it may not become adherent, and in rare instances it is twisted like a loop around the small gut, causing constriction or even strangulation.

Pathology.—Three pathologic varieties are recognized:

(1) **Catarrhal or Obliterative Appendicitis.**—This may be acute or chronic. The term "catarrhal inflammation" is still retained, though scarcely applicable, since, as a rule, appendicular inflammation tends to spread quickly to all the coats, including the serosa. Obliterative appendicitis is descriptive and in every way preferable. The *mechanism* of the inflammation is briefly as follows: The mesentery being too short, the exit is too small, and in consequence of swelling of the coats (especially the mucous) the venous return is greatly impeded, then the arterial, followed often by abscess-formation. In the female a branch is supposed to be furnished by the ovarian artery, making a more perfect blood-supply. The appearances are, in the beginning, identical with those of catarrhal inflammations elsewhere in the bowel. Within twenty-four

[1] The following names will long be connected with this disease: Pepper, Fitz, McBurney, Porter, Willard Parker, Weir, Sand, Bull, Warren, Keen, Morton, White, Price, Deaver, Senn, and many others.

[2] *Sajous' Annual*, vol. i., 1893.

[3] "Some Points regarding the Appendix Vermiformis," *American Journal of Medical Sciences*, Jan., 1891.

hours all the layers are swollen, with marked cellular infiltration, causing the appendix to become firm and often rigid. The mucosa may be denuded of its epithelium and present a granular surface. The external coat (serosa) is usually hyperemic, and not uncommonly the seat of fresh or old adhesions. The tube may become completely obliterated by pressure, resulting in a union between the granular surfaces, in this manner rendering subsequent attacks impossible (Hawkins). It is in cases in which this fortunate result is not reached, however, that acute appendicitis leads to the chronic form with relapses. Two additional terminations may be observed: First, an obliteration of the lumen may occur near the valve, in which case the appendix becomes dilated, and sometimes enormously so (cystic). The contained liquid may be either serous or purulent. Second, obliterative appendicitis may lead directly to ulceration of the mucous membrane, and often in the absence of a fecal concretion or foreign body. Again, the cystic appendix may ulcerate, with or without perforation. Obviously, the more marked the stenosis of the appendix the less favorable the conditions for natural drainage, and the greater the liability to recurrences of attacks of appendicitis. This variety then may end in resolution, complete obliteration, stenosis, or ulceration, and the latter sometimes in perforation.

(2) **Ulcerative Inflammation.**—Like the preceding, this variety may be acute or chronic. It may be a sequel of the obliterative form, and often accompanies chronic obliterative appendicitis. More commonly, however, it is seen in connection with concretions, and sometimes with foreign bodies also. By no means invariably, however, does the presence of these substances excite ulceration of the appendix. Microorganisms play an important rôle in this variety (*vide* Etiology). The submucosa or muscularis usually forms the base of the ulcer. The termination may be in healing, with tendency to stricture. Again, the ulcer may extend in depth until perforation occurs.

(3) **Interstitial or Parietal Inflammation.**—This may be preceded by the obliterative or the ulcerative form, which may be followed by anemic necrosis and sloughing. Concretions or foreign bodies are often found, though specific bacteria are of greater etiologic importance. The gravest, most common, and hence the most important lesions are the gangrenous, which are usually limited to a circumscribed part of the tube. Interstitial inflammation has a single termination—perforation—and leads to appendicular peritonitis of a virulent and infectious type.

It may be that neither necrosis nor gangrene may supervene. When perforation occurs, one or more openings, ranging in size from one to several millimeters, may be observed, while the remainder of the appendix may present no abnormalities; more often, however, it is blood-injected and swollen. The appendix may slough *en masse*. The histopathologic changes may be characterized by intense cellular exudation, necrosis, or purulent inflammation. Pathologically considered nearly all cases are suppurative. The muscular coat is hypertrophied, and chronic thickening of the appendix may result.

Consequences of Perforation.—A common result of all forms of appendicitis is a localized peritonitis, and this is a constant effect of the severer forms, either leading to (*a*) circumscribed peritonitis or to an (*b*) acute diffuse peritonitis.

(*a*) *Circumscribed Peritonitis.*—At first the surface of the peritoneum is opaque and velvety. Soon a fibrinous exudation covers the appendicular peritoneum, and quickly establishes adhesions between the appendix and the adjacent parts (abdominal wall, intestinal coils). The process may not proceed any further. Generally, however, it is soon followed by a serous or sero-fibrinous exudation, which becomes sero- or fibrino-purulent, and often forms the so-called perityphlitic abscess. The seat of the abscess is always near the tube, and is as varying as the position of the appendix; its size is also extremely variable, as it sometimes contains enormous amounts of pus. Among the most common locations are—McBurney's point, the vicinity of the cecum, the coils of the small intestines (near the umbilicus), and, more rarely, in the pelvis below. The pus contained in the abscess is rarely thick, grayish-yellow in color, and emits a fecal odor; more commonly it is thin, turbid, dark-gray or greenish in color, and has an extremely fetid or even gangrenous odor. The process of gangrenous sphacelation *en masse* is often completed after the limiting wall of adhesion has formed, when the entire appendix is found free in the pus-cavity.

The abscess may be *subperitoneal*, as when perforation occurs into the retro-cecal connective tissue, and the term "iliac abscess" was formerly applied to these *extra-peritoneal* purulent collections. They are rare, however, since the early operation has been employed. Their situation and dimensions depend upon the direction taken by the appendix. The latter may pass downward, and the pus is then apt to accumulate in the lower part of the iliac fossa, and may point and finally burst in the neighborhood of Poupart's ligament, with subsequent recovery. Occasionally under these circumstances a fistula remains for an indefinite period of time. The appendix may touch various abdominal structures, and the pus in following the line of least resistance may cause spontaneous rupture into the rectum, bladder, or the vagina when it points inward; and into the perinephric region or into the pleural cavity (through the diaphragm) when it points upward; or even into the cecum or colon. The contents of the abscess may also find their way through the abdominal wall in the vicinity of the umbilicus. The psoas muscle may conduct the abscess downward, and it may then point at the hip-joint or gain the gluteal regions or the scrotum, producing the so-called "scrotal appendicitis." The appendix has also been found in a hernial sac. Among the rare lesions to be noted are erosion of one of the arteries of the iliac region (causing fatal hemorrhage) and pylephlebitis. From the thrombi in the mesenteric veins in the latter condition infectious emboli may be conveyed to the liver, giving rise to hepatic abscess; this occurred in a case of my own at the Episcopal Hospital, Philadelphia. The abscess may also be due to an extension of the thrombo-phlebitis of the mesenteric veins that lead from the appendix to the portal vein. Thrombosis of the iliac veins with edema of the corresponding leg may also arise, and these veins may, during the process of healing, become compressed, with a resulting edema of the leg, as in two of my cases. It rarely happens that suppurative processes are both extra- and intra-peritoneal.

(*b*) *Acute Diffuse Peritonitis.*—This follows perforation when previous adhesions have not taken place, or when, having formed, they yield.

Generalized peritonitis may also follow the circumscribed form, the lesions being propagated to the entire membrane by extension. The morbid changes are described under Acute Peritonitis (*q. v.*). Since the early operation has been employed peritonitis has been the result, usually, of direct perforation before protective adhesions have been formed.

Etiology.—**Predisposing Causes.**—(*a*) Doubtless certain *congenital structural defects* aid in the producton of appendicitis. Among them are unnatural length, location, and arrangement of the organ; also the shape of the meso-appendix and Gerlach's valve. These factors tend to obliterate the lumen of the canal by producing kinks and twists, thus favoring the collection of material within the appendix. (*b*) *Strictures*, particularly near the cecal end of the tube, and old adhesions, especially peritonitis, operate in the same manner as (*a*), only with greater power. (*c*) *Fecal concretions* are the main cause in nearly one-half, while *foreign bodies* play a small rôle, having been present in 7 per cent. only of 1400 cases (J. F. Mitchell). The calculi form in the appendix itself (Rochaz). The foreign bodies are very various, and consist of seeds, worms, gall-stones, pills, bristles, and, more rarely, pointed bodies, as fish-bones or pins. The presence of fecal concretions and foreign bodies is often tolerated by the appendix without symptoms or local pathologic changes; hence they are looked upon rather as a predisposing than as an exciting cause. (*d*) *Ulcers* (tuberculous, typhoid, and, rarely, actinomycotic) may also produce this affection. (*e*) *Straining Efforts and Traumatism.*—Not uncommonly excessive muscular exertion, traumatism, or jarring of the body, as in jumping, act as favoring causes. (*f*) *Age.*—The disease is especially frequent in young adults between the fifteenth and thirtieth years. It is not infrequent in childhood after the third year, and it has been seen in persons over seventy years of age. (*g*) *Sex.*—Appendicitis attacks males oftener than females (4 to 1); this fact has been explained (*vide supra*). In the female it is rarely of adnexal origin. Adhesions between the tube and ovary and the appendix may occur, the morbid process then extending to the latter. (*h*) *Gastro-intestinal Disturbance.*—Indiscretions in the diet may precede a primary attack, and are of paramount etiologic importance in the recurrent forms of the malady. (*i*) *Heredity.*—That this plays a rôle in cases of appendicitis I have long felt convinced. This serves to explain cases in which rheumatism and uric-acidemia seem to act as causal agents. (*j*) Evidence to show that influenza and other affections may cause appendicitis is not wanting. (*k*) It is not improbable that *poor blood-supply* and retrogression of the organ, plus torsion and the like, are the leading predisposing factors. Any slight interference with the circulation tends to block it, with dire consequences. (*l*) The negro enjoys comparative immunity. (*m*) The immoderate use of meat (MacLean).

Bacteriology.—The combined results of several experimentalists show that no special organism plays an exclusive *rôle* in this disease, but the studies of Hodenpyl indicate that the *Bacillus coli communis* is most generally present: it is well known, moreover, that this bacillus becomes pathogenic when it escapes into tissues in which it does not naturally belong. A. O. J. Kelly found this organism present alone in 73.4 per cent. in 94 instances of acute appendicitis; alone in 89.71 per cent. of 107 cases of chronic appendicitis. Barbacci emphasizes the etiologic importance

of the passage of the intestinal contents into the peritoneal cavity—*i. e.*, the chemical factor. Of other specific bacteria, those of *typhoid* and *tuberculosis* are not uncommonly found to be present. The *streptococcus pyogenes* may also produce the most virulent infection, and the *staphylococcus pyogenes aureus*, the *proteus*, and other organisms have been found. The great frequency of appendicitis is rendered appreciable by the numerous favoring factors (including the congenital conditions) acting upon the appendix, which naturally has an exceedingly low vitality; also by the constant presence of organisms that are known to become pathogenic in the presence of a slight lesion.

Clinical History.—Doubtless many cases are overlooked because of the extreme mildness of the symptoms. These are often attributed to intestinal indigestion or to a "cold," to which the patient pays little attention unless he displays unusual susceptibility.

The *onset* of acute appendicitis may be slow and gradual, but oftener it is *quite sudden*. A clear history of some obvious cause (an error in diet or muscular effort) may be obtainable. Again, preceding the onset of the definite symptoms and extending over a day or two, there may have been *certain prodromes*, as impaired appetite, nausea, constipation, or diarrhea. In slow cases the local and general symptoms are at first slight, but gradually increase in severity. Indeed, in the latter class the patient may go about his customary duties during the attack with ill-defined rational symptoms, while in reality suffering from periappendicular abscess. These patients run two serious dangers—first, spontaneous rupture of the abscess into the peritoneal cavity may occur; and secondly, the slow septic absorption may suddenly overwhelm the system. As a rule the sudden cases develop in seeming perfect health, and are sometimes heralded by a rigor or chilliness.

The characteristic features of the invasion are *abdominal pain*, *fever*, *tenderness over McBurney's point*, *circumscribed resistance*, *gastric disturbances*, and, as a rule, constipation. The *pain* varies in intensity from a mere feeling of soreness to that of the most agonizing suffering. It may be paroxysmal, though oftener it is constant, with moderate exacerbations. Severe pain points to an involvement of the peritoneum and signalizes a danger of perforation. At first the pain may be referred to any point in the abdomen for the reason that the superior mesenteric plexus, that furnishes the nerve supply to the appendix, sends numerous twigs to the small intestines; later, within forty-eight hours, it becomes more distinctly localized in the ileo-cecal region.

Elevation of Temperature.—The exacerbations may at first touch 102°, 103°, or even 105° F. (38.8°–40.5° C.), and particularly in children; more commonly they range from 100° to 102° F. (37.7°–38.8° C.). The degree of fever is unreliable, however, as a criterion of the severity of the case, since the worst cases may show a subnormal temperature.

An elevation of temperature, however trivial, is most significant, pointing as it does to inflammation as the cause of the local symptoms. The pulse-rate is somewhat higher than the elevation of temperature would lead one to expect, and in bad cases the pulse is usually much quickened. Sometimes, however, it remains at 80 to 90 per minute, and may be full and soft, even though the patient be practically moribund.

Fixed *tenderness* is practically constant on pressure over a limited

area, midway on a line between the anterior superior iliac spine and the umbilicus (*McBurney's point*), and is a valuable sign. The seat of the tenderness may be found at other points rarely, depending upon the location of the appendix. I have twice observed it in the lumbar, once in the right hypochondriac region, and once far below the usual point, in the right iliac fossa. It has been found in the umbilical and left iliac regions, in the pelvis, and in the groin. In several instances, although I have found it elsewhere in the early stage, it has shifted to McBurney's point later. On the other hand, it may move from the usual position in cases that are allowed to drag on. When the sensitive area is at McBurney's point, as is the rule, the gentlest pressure often suffices to elicit exquisite tenderness, but when it is situated elsewhere firmer pressure with the finger-tips is

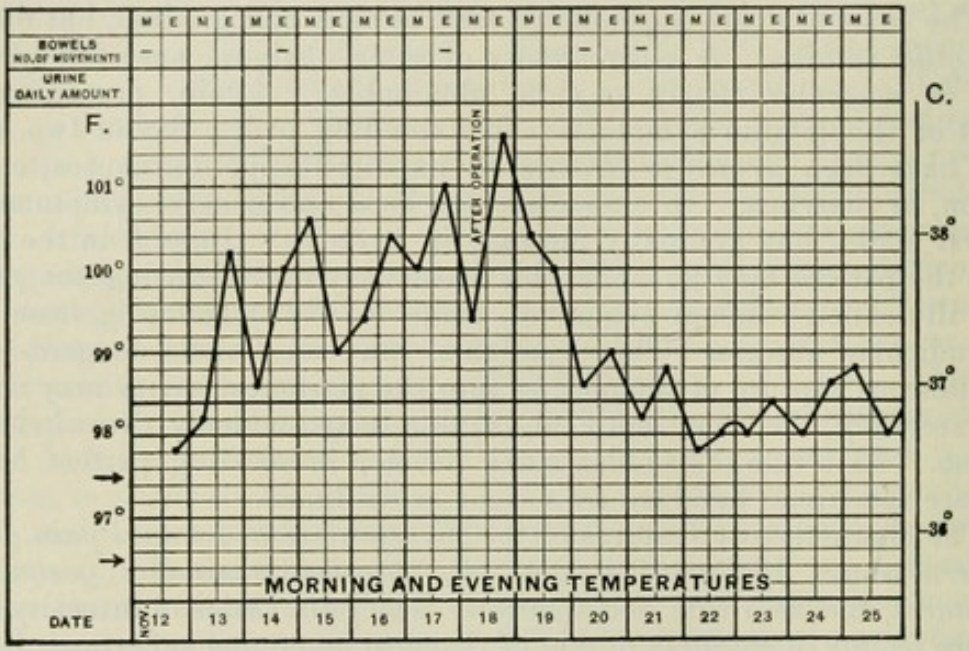


FIG. 59.—Temperature-chart of a case of appendicitis. M. M——, aged thirty-five years; motorman. Laparotomy, by Prof. E. Laplace, disclosed catarrhal appendicitis with adhesions.

usually required. Deep pressure always reveals localized tenderness at some point in the abdomen if the case is one of appendicitis. *Palpation* also detects an abnormal rigidity of the right rectus abdominis muscle. On or about the second day a *circumscribed induration* manifests itself, followed soon by a fulness and swelling tending to obliterate the depressions above and in front of the anterior iliac spine. The position of the indurated area varies according to the location of the appendix. Sometimes a questionable mass the shape of an enlarged appendix is palpable. In such cases peritoneal exudation has not as yet occurred to any great extent. Induration may gradually assume the circumscribed form; it may, moreover, be so deeply seated as not to be appreciable. The degree of tenseness of the two recti muscles—right and left—should be compared, though an absence of tension of the right rectus does not eliminate appendicitis. The results of *percussion* furnish no certain guide.

Vomiting usually occurs at the beginning, unless there be diarrhea, and is attended by more or less nausea; it may continue throughout the course of the attack. In most cases, after a few paroxysms of vomiting

the symptom disappears, although it may recur if errors in diet be committed or if peritonitis supervene. Constipation is the rule during the attack, though diarrhea, which sometimes precedes appendicitis, may also occur at a late stage as a septic symptom. There is anorexia and the tongue is coated. The *decubitus* is dorsal, with the right leg flexed. Frequent micturition (early) and retention of urine (later) are not uncommon, the urine having a deep color-tint, and sometimes contains albumin. Moderate leukocytosis (*e. g.*, 10,000 to 15,000 per c.mm.) usually exists, but may be absent. Daniells,[1] in 120 cases of uncomplicated appendicitis, found that a rapidly increasing leukocytosis means that the inflammation is increasing and extending.

The case may follow a mild *course*, terminating in resolution with recovery; or it may be of a severe type and develop perforation, with the formation of abscess or diffuse peritonitis. It is impossible to obtain statistical evidence of the relative frequency of these alternatives, and hence the frequency of treatment by abdominal section. In more than one-half of the cases it is probable that the course is favorable.

If not operated upon early, the fever may continue for three to five days, and then subside, with simultaneous abatement of the severe local and general symptoms and with the establishment of convalescence. The same amelioration of the symptoms may be brought about by free purgation early, as the result of salines or, less often, spontaneously. In these instances resolution takes place even after invasion of the peritoneum. Small abscesses may be absorbed, and usually in cases terminating in resolution perforation has not occurred. Infection of the peritoneal membrane directly through the appendix is not uncommon.

In severe attacks perforation may occur, with the development of localized peritoneal abscess or generalized peritonitis (*vide* Pathology), and it must be remembered that cases that begin gradually may also show a tendency toward perforation. When this event occurs during the course of appendicitis, the symptoms of *local* or *general peritonitis* are superadded. If early, the symptoms pointing to peritonitis are intense; the abdomen swells quickly, and is exquisitely tender, while the physical signs of a tumor are absent. The temperature often falls, when vomiting and circulatory collapse appear. The generalization of the peritonitis is usually marked by less violent symptoms. Starting from the seat of circumscribed inflammation, the pain and tenderness advance noticeably from day to day until every portion of the peritoneum has been invaded. Besides progressive augmentation in the local features, including the pain, there is a gradual failure in cardiac power, as shown by the condition of the pulse; vomiting also returns, and at last becomes fecal. Death results from asthenia, and sometimes suddenly when unanticipated. If perforation occurs later, sufficient time has been allowed usually for the inflammation to become circumscribed, in which case the localized abscess is generally intra-peritoneal; it may, however, rarely be extra-peritoneal. The local symptoms intensify, the pain becomes excruciating, and the spot of tenderness may rapidly extend itself in all directions, particularly downward. Vomiting sets in, and may become troublesome, and constipation is absolute, not even gas escaping.

Physical Signs of Localized Abscess.—*Inspection* shows distention of

[1] *Columbus Med. Jour.*, September, 1906.

the belly, the affected area being prominent, with an obliteration of the natural depression in the right iliac region. A dark and swollen appearance of the subcutaneous and deeper veins has been noted by Skinner. *Palpation* discovers induration and great tension that soon yield to pressure (doughy), and edema of the skin. If the abscess is superficially seated, fluctuation may be appreciable. Deep-seated tumors are not uncommon, and then fluctuation is detected with difficulty. An examination *per rectum*, with a view to determining whether the abscess occupies the pelvis, is important, and in doubtful cases bimanual examination should not be neglected. *Percussion* reveals dulness if the abscess be superficial. A tympanitic note, however, is often elicited, and is due to an intervening coil of intestine.

If active peritonitis and septicemia do not develop, the constitutional as well as the local symptoms may abate, and the patient leave his bed, carrying with him, however, the abscess. The latter may point somewhere in the right lower quadrant of the abdomen or in the lumbar region. Spontaneous rupture into the rectum, bladder, vagina, or cecum may also occur. Often, preceding the discharge of pus into these organs, the latter display marked irritability, particularly the rectum and bladder. There is always the danger that the contents of the abscess may find its way into the general peritoneal cavity. The symptoms of hepatic abscess may develop. The pus may traverse the abdomen in the upward direction until it touches the diaphragm, when the symptoms of subphrenic abscess may be manifested. Extension through the diaphragm, causing pleurisy or pericarditis, and a pleuro-fecal fistula may occur. The lung complications originate, as a rule, from emboli. Sonnenburg found that out of 740 cases of appendicitis, 28 had some lung complication, and of these, 14 were cases of thrombosis. The early recognition of post-operative lung-emboli is important.

The general symptoms undergo a modification, due to the *suppurative process*. Rigors or a decided chilliness may occur. Diarrhea often succeeds to previous constipation, and drenching sweats to a dry skin. Improvement and even spontaneous cure may ensue if spontaneous rupture into one of the outlets of the body should occur. The fever (Fig. 60) may be either remittent or intermittent, and if the localized inflammatory process be active, the usual pronounced features of septicemia are predominant in the clinical picture. The latter specially grave condition often drifts into an extreme typhoid state with a hopeless course.

Diagnosis.—Typical cases of appendicitis are readily diagnosticated. Their recognition rests upon a few cardinal symptoms—viz., the acute development of severe pain in the right iliac fossa, coming on in a person previously healthy and usually under forty years of age; appendicular tenderness, unilateral induration, fever, vomiting, and constipation, or, more rarely, diarrhea. Atypical cases, however, may offer difficulty, although it is my belief that errors in diagnosis are less frequent than in almost any other disease. The pain may for a time be referred to a circumscribed area far removed from the site of the appendix, and rarely it continues without a change of situation throughout the attack. In the latter case the morbid lesions may occupy the usual position, or more often perhaps some quite unusual position. Thus, when the pain is referred "due east," or to the left iliac fossa, with

bilateral induration, the appendix will be found in the pelvis (Deaver). In such instances a rectal and a bimanual vaginal examination are imperative. It should be an unvarying rule in all cases of severe abdominal pain to palpate with the finger-tip every square inch of the abdomen if necessary, to find the localized tenderness when it is not found at McBurney's point. The degree of tenderness sustains a close relationship to the severity of the local inflammation as long as the condition remains strictly localized, but this relationship is lost when generalization occurs. With the appearance of a circumscribed induration and of the intense local tenderness and pain it is reasonably sure that per-

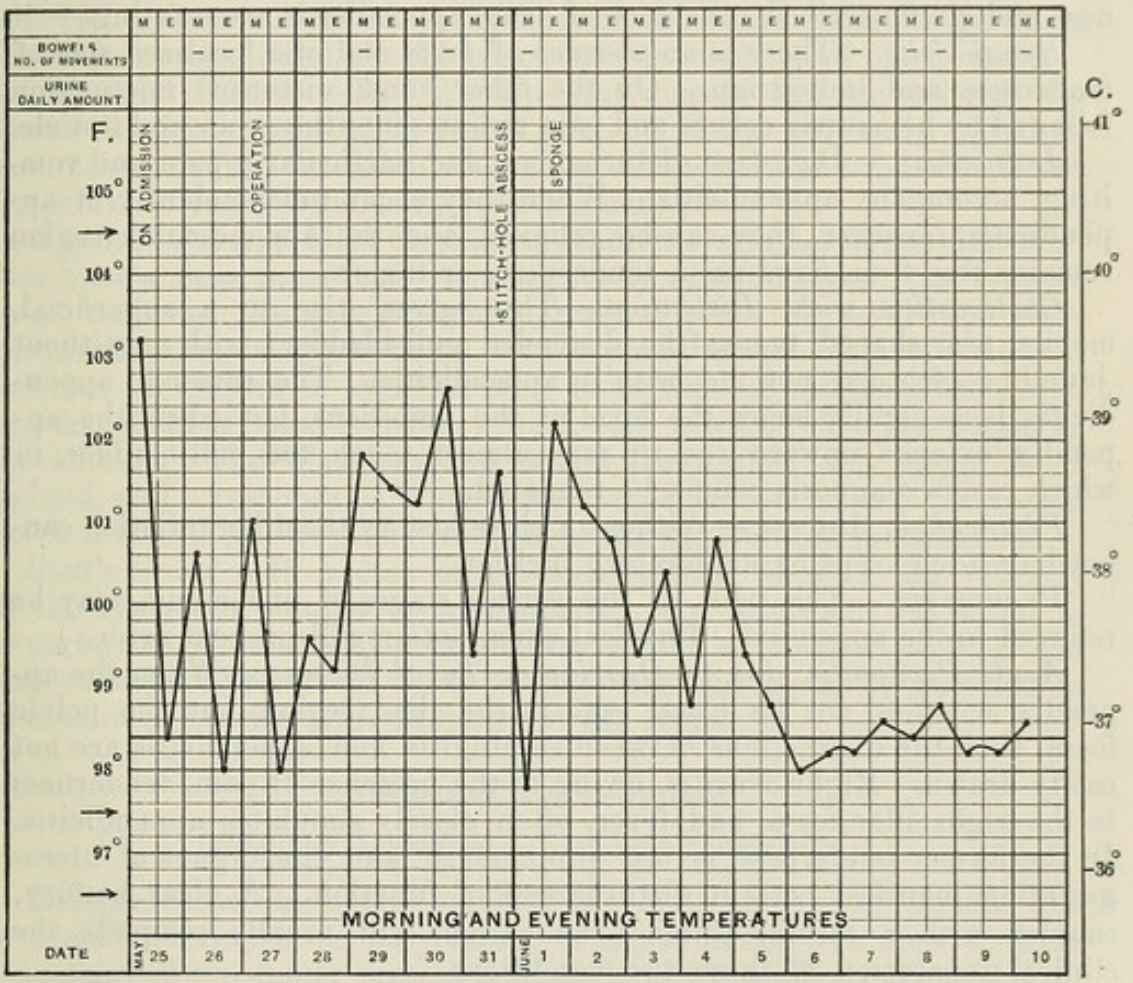


FIG. 60.—Temperature-chart of a case of appendicitis. R. C——, aged nineteen years; carriage-builder. A peritoneal abscess was found, while the appendix was becoming gangrenous.

foration either has occurred or is impending. Perforation may occur without local induration, however, and even after subsidence of the acute pain and excessive tenderness. Gangrenous appendicitis is *most deceptive*. The very acute symptoms, including the fever, may disappear, and unless the physician be upon his guard the patient will be considered convalescent and be allowed to go about. Rupture of the abscess now occurs unexpectedly into the peritoneal cavity, intestines, or some other direction, or a large-sized abscess develops with the usual signs and symptoms. In dubious cases the *x*-rays should be employed for diagnostic purposes.

Differential Diagnosis.—*Typhlitis, and especially the Massing of*

Feces in the Cecum.—These are truly rare conditions. According to McBurney, 99 per cent. of all typhlitic abscesses are of appendicular origin, and of 400 autopsies by Einhorn 91 per cent. had this origin. Ball and others have performed laparotomy for ulcerative cecitis, but this condition cannot be recognized during life. Stercoral typhlitis is discriminated from true appendicitis by the precedent constipation, which may become absolute, by the dragging character of the pain, the late-appearing fever, and the physical signs, which indicate the presence of a superficial, sausage-shaped tumor that is often doughy and extends vertically from a point near the right costal border "southward." Percussion elicits dulness over the seat of the tumor. The localized tenderness and circumscribed resistance of acute appendicitis are wanting.

Renal Colic.—There is an absence of fever and of a localized spot of tenderness and induration. On the other hand, disturbed micturition followed by hematuria occurs and pain radiates into the groin and testicle.

Indigestion.—Digestive disturbances, and particularly pain and vomiting, accompany appendicitis. When they occur independently of appendicitis, however, they can be relieved, and the appendicular region remains free from fixed pain, tenderness, or tumor.

Cholecystitis with Distention.—This gives rise to a superficial, mobile, pear-shaped tumor (the distended gall-bladder), with or without jaundice—features not met with in appendicitis. The tumor in appendicitis is generally below the level of the umbilicus, but when the appendix extends upward the tip may almost touch the gall-bladder, in which case a diagnosis cannot be rendered.

Perinephric Abscess.—Without a clear history the differentiation cannot be made except by exploratory incision.

Pneumonia.—The pain in the earlier stages of pneumonia may be referred to the appendix. Physical examination will prevent error.

Acute Peritonitis, due to Ovarian or Tubal Disease.—When the appendix occupies, not its usual seat in the iliac region, but the pelvic fossa, then the distinctions between salpingitis and appendicitis are not easily drawn. Right ovaritis, owing to the presence of pain, tenderness in the right iliac fossa, and fever, often closely simulates appendicitis. In the former tenderness is less pronounced, and the organs of utero-gestation manifest certain disturbanees of function. A clear history, coupled with a careful pelvic examination, will usually complete the clinical separation of these two conditions.

Extra-uterine Pregnancy.—In this condition the menstrual history furnishes important information. There is, in addition, profound collapse, due to hemorrhage, when rupture of the adhesions occurs. Elevation of temperature is absent. The localized tenderness and increased resistance are lower in the pelvis than in appendicitis.

Acute Tuberculous Peritonitis.—As in appendicitis, so in tuberculous peritonitis, pain, tenderness, and fever are present, but in the latter the onset is more gradual, and the signs of tumor and increased resistance in the ileo-cecal region are absent. Movable dulness may be present in the tuberculous affection, but not in appendicitis until the peritonitis is generalized. The lungs generally show lesions in tuberculous peritonitis.

Acute Intestinal Obstruction.—When this is due to intussusception there may be signs of a tumor, but not at McBurney's point; the ten-

derness over the site of the mass is less intense, while the frequent bloody discharges that are seen in this condition, accompanied by tenesmus, do not characterize appendicitis. When obstruction is caused by strangulation stercoraceous vomiting is apt to occur, and is absent in appendicitis. Pain, local tenderness, and, not uncommonly, signs of a tumor appear, but elsewhere than at McBurney's point.

Intestinal Lithiasis.—This can be diagnosticated, as a rule, by the presence of intestinal sand in the movements (Bottentuit). Circumscribed resistance is absent in this connection.

Acute Hemorrhagic Pancreatitis.—This affection simulates appendicitis with generalized peritonitis. But the deep-seated epigastric pain, followed by circumscribed resistance in the same region (a grouping absent in appendicitis), should arouse strong suspicion of pancreatitis.

Hip-joint Disease.—In both hip-joint disease and appendicitis the dorsal decubitus with flexed leg is noted. If the patient be anesthetized, however, full extension of the leg and a normal condition of the hip-joint are easily demonstrable in appendicitis.

Typhoid Fever.—Mild cases of appendicitis with accompanying diarrhœa bear a close superficial resemblance to typhoid fever. In typhoid fever, however, the onset is more gradual and the fever-type more continuous than in appendicitis. In typhoid the stools are somewhat peculiar, the spleen is swollen, there is dulness of intellect, bronchitis and the characteristic eruption attend,—all features that are absent in appendicitis. The diazo-reaction would strengthen the diagnosis of typhoid, and a response to Widal's test would be conclusive. In appendicitis the local features, and in typhoid the general, are predominant.

Dietl's Crises.—In a case of movable kidney which I saw recently all the symptoms pointed to appendicitis. An operation was about to be performed when a sudden subsidence in the abdominal swelling and local induration occurred. The kidney was subsequently detected in an abnormal location (*vide* Mobility of the Kidney).

CHRONIC APPENDICITIS.

(*Relapsing Appendicitis—Recurrent Appendicitis.*)

Relapses occur in nearly one-half the total number of persons who have suffered from a primary attack of appendicitis. In most of these cases there is constantly present a slight local discomfort during the interval. When successive attacks occur in the same individual at intervals of considerable duration (*e. g.*, a year or more), each new attack is spoken of as a *recurrent appendicitis.* Severe attacks may succeed to light ones and even prove fatal, or conversely, mild recurrent, may follow severe primary attacks. The *local symptoms* in those having had an antecedent peritonitis are more pronounced than in the first attack, but after a number of recurrences the symptoms are likely to be less severe with each new attack. The most constant symptom between attacks is a subacute form of *pain* that is liable to manifest exacerbating periods with slight fever. Physical fatigue, a strain, and errors in diet, causing gastro-intestinal disorder are very likely to induce a relapsing or recurrent appendicitis. Chronic appendicitis strongly favors the retention of fecal matter in the cecum, thus forming so-called *stercoral typhlitis.*

In the intervals between the attacks the appendix can be readily appreciated on *palpation*, the method employed by Edebohls being preferable: "The patient lies upon his back with the examiner at his side; the latter places his right hand upon the patient's abdomen over the right rectus muscle, opposite the anterior superior spine of the ilium, and presses the left hand upon the right, so that no force is used by the right hand and the tactile sense of its fingers is left undisturbed. The hands are drawn slowly outward, allowing the contents of the abdomen to slip from underneath them. The coils of intestine can be felt as they escape from under the hand as it presses against the posterior abdominal wall."[1] In this way the appendix may be felt as an elongated tumor of the size and shape of the little finger. If there be only a slight exudation present, the appendix often appears to be immediately beneath the abdominal wall. It may, however, be deep-seated, even though the exudation with adhesions be absent. *Bastedo's test* in suspects,—the passing of a colon tube 10 or 12 inches into the rectum and injecting air by means of an atomizer bulb. If now pain and tenderness to finger-point pressure become apparent at McBurney's point, there is chronic appendicitis. Both pain and tenderness are pronounced, and particularly if pus be present.

Here should be mentioned a form of appendicitis which is chronic from the start and not preceded by acute attacks. The inflammation, slowly developed, may be due to various causes, such as influenza (rare), floating kidney (common), and errors of diet, which produce a condition of enterocolitis, to which the chronic appendicitis is secondary. The symptoms are,—an unpleasant, dull pain accompanied by a dragging sensation, which may affect the entire right side of the abdomen or be circumscribed in the region of the appendix, obstinate constipation, emaciation, and marked neurasthenic features. Attacks of appendicular colic, with or without vomiting, may arise from time to time. A differential leukocyte count and frequent thermometric observations, which may show slight elevations of temperature, are aids to the diagnosis. The *physical examination* reveals tenderness on deep pressure over the vermiform appendix, with which an equal degree of tenderness, however, up near to the costal arch (suggesting gall-bladder disease) may be associated. More or less resistance may also be noted, but seldom a tumor.

In so-called *appendix dyspepsia*, in which the appendix is the seat of chronic inflammation, the symptoms exhibited may be those of gastric or duodenal ulcer. This mimicry is due to an exaggerated action of the pylorus (Moynihan). Removal of the inflamed appendix is generally followed by relief of the foregoing dyspepsia. Sailer[2] states that many cases of movable cecum, due to kinks or folds that give rise to partial or complete obstruction, are wrongly diagnosticated chronic appendicitis. The removal of the appendix is not followed by relief of the symptoms.

The results of chronic appendicitis upon the general health and nutrition of the patient are quite noticeable, and tend to augment as time passes, if the attacks be frequent or the intervals between them grow shorter. The chief symptoms are those of a nervous type; emaciation and debility are also observed. The associated nervous symptoms are

[1] B. Farquhar Curtis: *Twentieth Century Practice of Medicine*, vol. viii.
[2] *Amer. Jour. Med. Sciences*, February, 1912, 157.

those of neurasthenia. These patients often become introspective and exceedingly irritable, the mental condition being accounted for, to a great extent, by the consciousness that there is ever present the overhanging danger of a fresh attack with serious possibilities.

Differential Diagnosis.—*Carcinoma of the Appendix and Cecum.*—This presents many points of similarity to chronic appendicitis. I have under my care at present a lady aged sixty years suffering from chronic appendicitis, whose case had been diagnosticated as carcinoma of the cecum, and for a considerable time my own view coincided with that of my predecessor. The occurrence, however, of relapses, during which the feces were massed in the cecum and fever arose, soon indicated the correct diagnosis. Besides the absence of periodic attacks of fever, the general features—loss of flesh and strength, anemia—are more steadily and rapidly progressive in carcinoma of the appendix or cecum. The history of the mode of onset also aids in the distinction. Pain, tenderness, and a resistant tumor are common to both affections. Lane's kink of the ileum may present symptoms simulating chronic appendicitis, especially marked constipation, colicky pains, and meteorism.

Hypochondriasis and Hysteria.—Hypochondriasis and hysteria may lead to the manifestation of morbid feelings simulating those of appendicitis. Such cases may show merely a greatly exaggerated uneasiness, or such an increase of sensibility as to cause the patient to complain of pain in the right iliac fossa. In addition, there may be localized tenderness. I recently witnessed the removal of the normal appendix from an hysteric female in whose family two genuine cases of appendicitis had occurred not long previously. Hypochondriasis and hysteria distinguish themselves by the antecedent history and by the absence of a tumor-mass and of increased resistance; there is also an absence of localized tenderness if the patient's attention be withdrawn. In such subjects oxaluria is not infrequent, and it is possible that irritation of the right ureter by the passage of crystals of calcic oxalate, as mentioned by Cabot, may explain the localizing of the discomfort (Wood and Fitz). I saw a case of this sort in a medical student.

Prognosis.—Unlike many of the acute infectious diseases, the height of the temperature and, to a lesser degree, the rate of the pulse are unreliable guides in appendicitis. Broadly speaking, however, in the severer forms the local process exhibits a strong tendency to spread; the temperature and pulse are relatively high, and there is an intense appendicular intoxication. These are the cases that suppurate or result in perforative peritonitis (often rapidly spreading), and in pericecal abscesses. Of this fatal group of cases not less than 68 per cent. die before the eighth day. The development of *fulminant peritonitis* or of a peritoneal abscess after perforation is attended by a falling temperature, though subsequently the latter may mount high or become markedly irregular.

On the other hand, in the mild forms that are included in the name *catarrhal appendicitis* recovery is the unvarying rule. These lighter cases often lead to adhesive peritonitis—a circumstance that strengthens the view that they are of an infectious nature. The temperature is only moderately elevated as a rule, and the pulse-rate correspondingly quickened. Both pulse and temperature indicate marked im-

provement on the third or fourth day, while the pain and localized tenderness disappear. In this connection the deceptiveness of gangrenous cases must be recollected (*vide supra*, Diagnosis). The complications and antecedent and associated conditions may decidedly influence the issue. As to age, "the younger the child the worse the prognosis" (Finney). The general mortality of appendicitis is about 14 per cent. (Fitz). Improved methods, chiefly surgical, of dealing with the disease have, however, greatly reduced its death-rate. The prognosis in *chronic appendicitis* is most uncertain; after the patient has survived several attacks it is on the whole more favorable.

Treatment of Appendicitis.—Whether imminent danger of perforation exists or not, the physician who is called to a case of appendicitis should at once request the services of a competent surgeon. Few surgeons subscribe to the doctrine that all cases demand operation; but, since it may become necessary to perform celiotomy at any hour thereafter, the latter should help to settle the important question: "When is it necessary to operate in the case?" The physician who does not pursue the course above recommended falls short of his duty, both toward the patient and toward the surgeon on whose skill he relies to remove safely the source of danger. Surely, in a disease that so often baffles both physician and surgeon, suddenly developing, as it sometimes does, a fatal virulence without previous unfavorable symptoms, they should stand guard together from the moment the case is diagnosticated or appendicitis is strongly suspected. Unfortunately, both the medical and surgical treatment of appendicitis have each been recommended with great earnestness by their respective advocates.

With rare exceptions, prompt surgical intervention should be recommended. The indication for an immediate operation is undoubted in all cases of acute appendicitis, whether marked by sudden and severe or mild invasion-symptoms, if seen at the beginning of the attack, and free purgation at the earliest possible moment is not followed by decided relief. A waiting policy and medical treatment are also perilous in doubtful cases. Obviously, the conditions are less favorable for operation after a case has progressed to the beginning of abscess-formation—*i. e.* from the third to the fifth day of the illness. It is at this period that the peritoneal inflammation tends to circumscribe itself by the formation of adhesions. Hence, as Richardson states, it is "too late for an early operation, and too early for a safe late operation," since there is great risk of infecting the general peritoneal cavity. Whether it is wise to allow the appendix to remain after adhesions have been formed in some cases, and merely to drain, cleanse, and pack the cavity, cannot be discussed here. The lightest grades of appendicitis, in which doubt may surround the diagnosis and all factors possessing an unfavorable prognostic import are absent, scarcely require immediate operation.[1] The mild attacks that develop in the course of chronic appendicitis after numerous previous seizures need not excite alarm. In relapsing and in recurrent appendicitis operation should be undertaken between attacks, when the mortality is practically *nil*. On the other hand, in cases that have been allowed to drag on until general

[1] "Factors Influencing Mortality in Appendicitis, from a Medical Viewpoint," *Archives of Diagnosis*, January, 1911, by the writer.

peritonitis has set in, treatment by operation is not advisable. Moreover, the most ardent advocate of immediate operative treatment is sometimes compelled to rest satisfied with medical measures. Such cases are those in which there are associated chronic affections (advanced diabetes, Bright's disease), not to speak of those in which the patient declines operation. King has obtained encouraging results in the treatment with high-frequency currents. Appendectomy alone does not cure in patients with chronic constipation, a long dilated cecum, or enteroptosis.

General Management.—The patient should be kept in bed in a quiet, well-ventilated apartment, and in no affection is the value of *absolute rest* in the treatment of inflammation greater than in appendicitis. Neither food nor drink should be allowed from the moment the patient is first seen until early convalescence. At the start, and particularly if a sausage-shaped tumor be present, intestinal irrigation, oft-repeated, with a view to removing the fecal matter, must be carried forward carefully. I avoid the use of high enemata in *progressive* cases, since they are more apt than salines to induce rupture of the sac. To relieve thirst, enteroclysis by the drop method may be employed, and when stimulants are needful, whiskey or liquid meat extracts may be added to the physiologic saline solution.

As regards the use of opium professional opinion is not united, though a general tendency toward the limitation of its use to the minimum amount necessary to alleviate pain is happily noticeable; unless demanded by excessive suffering it had better be omitted altogether. When necessary, it is best administered hypodermically in the form of morphin (gr. $\frac{1}{12}$–$\frac{1}{8}$—0.0054–0.0081). The greatest objection to the use of opium is its effect in veiling the symptoms that assist the physician in forming a judgment as to the prospects and progress of the case. Gastric irritability may be sufficiently marked to demand special measures, such as the swallowing of small pieces of ice, spirits of chloroform, menthol, listerine, and the well-known combination of cereum oxalate (gr. iij—0.1944) and cocaine (gr. $\frac{1}{8}$—0.0081) every third hour may be used.

Local Measures.—The suspended ice-bag is an excellent means of combating the pain, and often obviates the necessity of an internal use of opium. Instead of the ice-bag, cloths wet in cold water may be applied and changed every few minutes. In the early stage a few leeches may be beneficial in their effect upon the local inflammation. Blisters, however, are rarely advisable, and are particularly objectionable should the patient afterward be submitted to an operation. Mild forms of counter-irritants (mustard-paste) are preferable, though these also render the skin and underlying tissues hard and leathery.

Management of Convalescence.—The patient should not be allowed to leave his bed for several days after the disappearance of all symptoms; even the mildest forms of exercise should not be undertaken for at least one week subsequent to getting out of bed. During convalescence the diet must be carefully guarded, and the bowels, at all hazards, kept in a soluble condition. It is questionable whether drugs will aid in the absorption of the exudate or assist in resolution.

TYPHLITIS.

By typhlitis is meant inflammation of the cecum without involvement of the appendix (rare); and in some cases ulceration due to pressure by retained fecal matter or foreign bodies ensues. The history of previous constipation or of some dietetic error is frequently obtainable.

The *symptoms* are pain of a dull character, nausea, and obstinate constipation with moderate fever. The *physical signs* are, a prominence in the ileocecal region, tenderness to pressure, and those of a doughy, sausage-shaped tumor in the cecal region. After two or three days the tumor gradually diminishes; also the active symptoms, but tenderness persists for a week or longer. When ulceration attacks the inflamed cecum a pericecal abscess is the usual result. To this condition the term "perityphlitis," which is now practically obsolete, was formerly applied.

The *treatment* of typhlitis is that of obstinate constipation. Eserine (gr. $\frac{1}{80}$ every fourth hour) has proved serviceable. For this purpose enemata administered high in the bowel are most effective. We may employ the so-called "ox-gall" enema, as follows:

℞. Powdered ox-gall,	gr. xx	(1.29);
Glycerin,	fl. ℥j	(32.0);
Water and soapsuds (105° F.),	Oj	(1082.08).

My own best results have been obtained from the use alternately of an enema of olive oil (℥viij at a temperature of 100° F.) administered through a rectal tube high in the bowel while the patient occupies the left lateral-prone position, and one composed as follows:

℞. Sulphate of magnesia,	℥iss	(48.0);
Glycerin,	fl. ℥j	(32.0);
Spirits of turpentine,	fl. ℥ij	(8.0);
Hot water (100° F.),	Oj	(1082.08).

The diet should be of the blandest sort, such as albumin-water, peptonized milk and the like, given at stated intervals in small, fixed quantities. Arterial stimulants may be required during the latter stages.

INTESTINAL CALCULI.

Intestinal calculi are rarely passed with the feces, and may be in the form of small concretions, hepatic calculi that have entered the intestine, and as sand which is formed in the saccules of the colon and folds of the cecum and rectum. Their origin may depend upon the deposition of calcium and magnesium salts upon particles of undigested food.

A teaspoonful or more of gritty sand may escape with each stool. But four cases of true enterolithiasis have been reported. The condition is likely to arise when the diet is exclusively milk. "Sand" may be produced through the ingestion of the banana (Myer and Cook).

INTESTINAL OBSTRUCTION.

(*Ileus.*)

Definition.—An acute or chronic, complete or partial, occlusion of the intestinal canal.

Pathology and Etiology.—The causes of intestinal obstruction may be divided, at once most simply and practically, into the (1) *acute* and (2) *chronic* forms. In the former variety the narrowing or closure develops very suddenly or rapidly, and usually in the small bowel; in the latter, the large bowel is commonly affected by pathologic conditions that develop gradually and narrow its lumen (usually in advanced years).

Acute.—(*a*) *Strangulation.*—In the order of frequency, this is first among the causes of acute intestinal obstruction. It is produced most often by bands of adhesion, the result of a former recent or remote peritonitis, and is most commonly situated in the right iliac fossa. Incarceration of the bowel from flexions and adhesions not rarely follows upon abdominal section for the treatment of pelvic disease in women.

The usually free end of Meckel's diverticulum is sometimes attached to the abdominal wall, and may thus cause constriction of a loop of bowel. This diverticulum is the remains of the fetal omphalo-mesenteric duct, and arises from the ileum about half a meter (1.64 ft.) from the ileo-cecal valve. A similar constricting band is formed by a cord representing one or more of the obliterated omphalo-mesenteric vessels. The adhesive attachment of the free end of the appendix vermiformis may also form an opening through which the bowel may be caught.

Internal strangulation (hernia) may be the result of forcing a portion of bowel through a slit in the omentum or mesentery, or into peritoneal diverticula and openings, such as the duodeno-jejunal fossa (*Freitz's retro-peritoneal hernia*) or the foramen of Winslow.

Diaphragmatic herniæ are not of extreme rarity, and may be either of congenital or traumatic origin. Most cases of intestinal strangulation occur in males during early adult life.

(*b*) *Intussusception.*—*Invagination* is the descending "telescoping of one section of the bowel into another," probably caused by a circumscribed, irregular peristalsis of the intestine. The effect of the latter state in producing invagination may be either a thrusting forward of the receiving portion by a contraction of the longitudinal muscular coat (Nothnagel), or a thrusting inward and downward of the portion immediately above by means of an increased or spasmodic peristaltic action. Thus, a cylindric or sausage-shaped tumor results, varying from a half inch to over a foot (1.3–30 cm.) in length. The layers met with in intussusception are the outer or receiving, called the *intussuscipiens*, the middle or returning layer, and the inner, called the *intussusceptum.* The seat of invagination is most commonly at the ileo-cecal valve, though it is often found in either the ileum or colon alone. Sometimes the intussusception is detected in the rectum. A lateral or partial invagination may also occur, due to the attachment of a tumor within the bowel.

The intussuscepted portion of intestine is usually the seat of peritoneal adhesions, so that in pronounced cases the parts are so firmly agglutinated that reduction is wellnigh impossible. The engorgement may

pass into an intense local inflammation, with final necrosis and sloughing, and even the discharge *per rectum* of the invaginated portion.

Intussusception occurs most frequently in children prior to ten years of age, and males suffer more than females. Invagination is an occasional consequence of the operation of circular enterorrhaphy (Robinson).

(*c*) *Volvulus.*—Twists of the intestine are met with most commonly at the sigmoid flexure of the colon. An unusually long or relaxed mesentery predisposes to the condition, so that the axis of twisting may either consist of the mesentery itself or frequently of the bowel. Not rarely the pedicle of the volvulus contains both a twist and a sharp bend in the bowel, causing complete acute strangulation. The latter condition may be pronounced in such cases, or at least be hastened, by the accumulation of the intestinal gas and of masses of feces,—by bowel-adhesions to an adjacent stump of omentum (Nieberding). The passive reactive pressure of the coils of intestine and of the abdominal walls tends also to further confine the enormously dilated and twisted loop of bowel to its abdominal state. Knots may be formed by the association of loops of the ileum with each other or about the pedicle of a twisted cecum.

Males between forty and sixty years are especially the subjects of volvulus. Acute intestinal obstruction invites bacterial invasion, which is the probable cause of the general symptoms.

Chronic.—(*a*) *Fecal Impaction.*—*Intestinal Concretions.*—Accumulation of feces (*coprostasis*) is a common cause of intestinal obstruction, the impaction taking place usually in the cecum or sigmoid flexure.

Though not infrequent in children, fecal obstruction is more common in adults (particularly in females), in the hysteric, the demented, and the hypochondriac. Congenital dilatation of the colon may predispose to coprostasis, and an acquired dilatation, which in some cases becomes enormous, is often the result of paresis of a portion of bowel caused by over-distention for a long period of time.

Among other causes of obstruction due to abnormal contents may be mentioned *enteroliths.* These are intestinal concretions formed of various nuclei, as gall-stones, hardened feces, phosphates of lime and magnesia, various foreign substances, and organic derivatives. Balls of tangled ascarides may mass sufficiently to cause obstruction.

Foreign bodies, as pins, buttons, coins, fruit-stones, may also cause obstruction of the bowel. It is stated that even insoluble mineral medicines, as bismuth or magnesia, have caused obstruction.

(*b*) *Tumors.*—Tumors cause a form of chronic obstruction that may at any time develop suddenly into the acute type. They may do so either as—(1) *new growths* in the wall of the intestine itself, or by (2) *compression and traction from without.* Again, the intestinal neoplasms may be *malignant* or *benign* in nature. *Carcinoma* of the bowel is at once the most frequent and important of these. It may be either circumscribed and annular, causing a gradual narrowing of the bowel-lumen, or a diffused infiltration of the intestinal wall, commencing either in the mucosa or in its glands (cylindric epithelioma). Its most common seat of growth is the large bowel, about the sigmoid flexure.

Sarcoma usually attacks the small bowel, starting beneath the mucosa, and is of the recurrent variety. Regional infection of the mesenteric and retroperitoneal glands (*Löbstein's cancer*) is also a usual

consequence of sarcoma. It may occur in children or in young adults.

Benign tumors may be polypoid, adenomatous, fibromatous and lipomatous. Intestinal obstruction due to compression or traction may be caused by tumors (omental) or by adhesions of the pelvic viscera.

(*c*) *Cicatricial strictures* cause chronic intestinal obstruction, as after the healing of various ulcers, the cicatrices of which slowly contract. Cicatricial stenosis of the colon is commonly due to the cicatrization of dysenteric ulcers. In the rectum the stenosis is usually a result of a syphilitic lesion. Tuberculous and rarely, typhoid ulceration may be followed by stricture of the small intestine.

(*d*) *Congenital stricture* is rare, and is more purely surgical than the preceding cases. It is often an occlusion or an imperforate condition of the anus (*atresia ani*), and is only mentionable in this connection.

(*e*) *Paresis of Peristalsis.*—This condition—called also *adynamic obstruction*—while it is a functional affection, is held to be either a circumscribed or diffuse paresis of the intestinal muscular coat. It is caused by some such inflammatory disturbance as enteritis or peritonitis, or even by the manipulations employed in abdominal sections. Here the obstruction is due to an accumulation of feces and gases in the paretic portion of the bowel, causing tympanites, vomiting, and constipation. Intestinal stasis may be due to the presence of kinks, as shown by radiography (Jordan).

Special Pathology.—The pathologic changes that accompany nearly every form of intestinal obstruction are briefly stated as follows: Accumulative dilatation—with hypertrophy in chronic cases—of the intestine above the seat of disorder, and an emptiness, narrowing, and even atrophy below the obstruction. The affected walls of the bowel are inflamed, and there is a surrounding acute or chronic peritonitis. Catarrhal and sometimes diphtheritic inflammation of the mucosa may develop. Gangrene, ulceration, and perforation of the bowel, with resulting generalized peritonitis, may also ensue.

Symptoms.—Acute Obstruction.—There is a suddenly developed *abdominal pain* that may follow some abrupt or severe exertion. *Early vomiting* and *absolute constipation* are also conspicuous and important symptoms. If the obstruction is high in the small bowel, distressing hiccough and eructations may precede the vomiting. Except for the possible discharge of the intestinal contents below the seat of obstruction, the constipation is usually complete and obstinate. The early symptoms, however, are caused by strangulation rather than by obstruction. Accompanying the latter condition there is tympanites, which is most marked in obstruction of the colon. Intermittent and colicky at first (partial obstruction—Treves), the *pain* soon becomes agonizing and constant. *Vomiting*, also, alternating with *painful retching*, is more constant and severe after several hours. The *material* at first ejected is gastric and mucous; it then becomes bilious, and finally is characteristically stercoraceous.

The *constitutional symptoms* develop early, are intensely threatening to life, and cause rapid and profound collapse. The pinched and pallid features, cool and moist skin, Hippocratic expression, rapid and feeble pulse, the usually subnormal temperature, shallow and accelerated breathing, marked thirst, scanty urine, great anxiety and prostration—all indi-

cate the gravity of the condition. McClure's experiments show the symptoms to be due to absorption of bacterial toxins.

The *physical examination* will discover a swollen, extremely tender, and tympanitic belly. Exaggerated peristalsis of the intestine above the obstruction may be visible on the surface of the abdomen. Borborygmi, gurgling, and splashing may be heard on auscultation.

Chronic Obstruction.—The symptoms are more dependent upon the special causes operating than in acute obstruction. The fact that early in the case only partial obliteration of the intestinal lumen may be rightly inferred in many of the chronic forms of obstruction has given rise to the discriminating term of *intestinal constriction*. In general, the clinical history is one of *increasing* and *intractable constipation*, sometimes alternating with diarrhea, due to catarrhal inflammation of the mucosa above the obstruction. Paroxysms of colicky *pain* and, later, augmenting *tympanites*, *vomiting*, and *prostration*, attend. These symptoms may merge suddenly into those of the acute form of obstruction. The bowel-movements in chronic obstruction are irregular, infrequent, slight, and sometimes accompanied by pain and tenesmus. The *stools* consist often of small, hard, ribbon-like, or scybalous masses, and may contain blood and mucus. When the stenosis is in the small intestine the constipation is less apt to occur on account of the fluidity of the contents. Sometimes, and particularly in old people, the rectum becomes distended with hardened accumulations of feces; there is in such cases a constant feeling of fulness and a harassing desire to defecate, but the attempts thereat are ineffectual (*vide* Typhlitis, p. 870).

In *cicatricial stenosis* there are a prolonged and variable history of constipation, occasional vomiting, localized pain, and meteorism.

Physical Examination.—*Inspection* shows the abdomen to be distended from meteorism, the movements, and contour even, of the coils of intestine in active peristalsis above the seat of stricture being evident. A tumor or the throbbing aorta (excited, perhaps, by pressure of the distended bowel or growth) may be *palpated*. Tympany and borborygmous noises may also be noted.

Diagnosis.—Locality of the Obstruction.—Given the symptoms of a sudden, severe, and exacerbating pain in the abdomen; of marked, and later feculent, vomiting; of absolute constipation and of tympanites and profound, early, systemic depression,—a diagnosis of acute intestinal obstruction may be made. The determination of its seat is often very difficult. First may be mentioned the *differential diagnosis* between obstruction occurring in the small and in the large intestine. In the former vomiting occurs early, is scanty, and later feculent, while in the latter there is less vomiting and the vomitus is seldom feculent. Again, in obstruction of the small gut the distention is both less marked and higher situated, while in that of the large gut tympanites is often quite marked, is more central, is associated with tenesmus, and sometimes with mucus and blood. If the cause of obstruction be a tumor or stricture, the locality may be successfully palpated or the lower limit of the active coils of hypertrophied intestine may be defined.

Examination *per rectum* with the finger or with the rectal tube, by means of liquid distention or gaseous inflation of the colon, may enable us to determine the seat of obstruction in certain cases. The detection of a

deeply-seated incarcerated hernia (in the abdominal fossæ and pouches, diaphragm, or obturator foramen) is often made only *postmortem*.

Nature of the Obstruction.—This is even more difficult of discovery than the preceding. The following causes of obstruction with their differentiation may be referred to in attempting a diagnosis: *Strangulation* often affords a previous history of peritonitis or abdominal section or of recurrent attacks of abdominal pain, occurring mostly in young adults. Early fecaloid vomiting is common.

Intussusception usually gives a negative previous history. The suddenness of the attack, without appreciable cause, occurring in a child, and associated with colicky pain, tenesmus, and the presence of mucus and bloody stools, and of an elongated cylindric tumor in the right iliac or umbilical regions often render this condition easy of diagnosis. It is to be noted that absolute constipation and meteorism are here unusual. The intussusception may be felt in the rectum.

In *volvulus* it may be helpful to elicit a history of former constipation and flatulence, with evidences of atony of the bowel, in persons of advanced years, along with marked abdominal tympany, tenderness over a distended coil, which may perhaps be outlined (Wahl), a rigid abdomen, and sometimes dyspnea from great gaseous distention.

The history in cases of *fecal obstruction* is nearly always one of obstinate, habitual constipation, and occurs especially in females and neurotic subjects. The onset is gradual; pain is less acute; and tympany and fecal vomiting are less prominent and late in appearance.

Obstruction due to *large enteroliths* or *foreign bodies* may be only surmised: especially is this true when symptoms of appendicitis arise.

Biliary calculi may give a history of previous attacks of hepatic colic and of recurrent jaundice.

In the chronic obstructive form of *stricture* of the bowel due to cicatrices or neoplasmata the history of dysentery, tuberculosis, sarcoma, or carcinoma should be considered (*vide Carcinoma Intestinalis*).

In obstruction caused by *intestinal paresis* there is generally a history of a previous enteritis, peritonitis, or celiotomy. The abdomen is smooth, though tympanitic throughout, and there is no perceptible peristalsis.

Not rarely it will be of therapeutic as well as of diagnostic importance to ascertain whether an attack of acute obstruction is primary, or whether it is the terminal exacerbation of a chronic condition, such as carcinoma of the bowel. Here a study of the past history of the patient, as well of the present signs of a probable nature, will afford considerable aid.

Differential Diagnosis.—Acute intestinal obstruction must be discriminated from *acute generalized peritonitis*.

Acute Generalized Peritonitis.	Acute Intestinal Obstruction.
Etiology.	
There is a history of causal conditions or diseases (ulcer, appendicitis, pelvic infection).	There is a history of previous chronic obstruction or hernia. (The young are most liable to intussusception.)
An early and considerable rise of temperature; later variable or may be absent.	No early rise (except in volvulus), but later with advent of peritonitis, and subnormal temperature develops later.
Pain continuous and diffuse and increased by movements.	Pain in short paroxysms and localized.
Vomiting, but not stercoraceous.	Vomiting becomes characteristically stercoraceous early.

Acute Generalized Peritonitis.	Acute Intestinal Obstruction.
Etiology.	
Collapse occurs late.	Earlier onset of collapse.
In septic cases, leukocytosis with increase in polynuclear cells.	There may be increase in number of leukocytes.
Distention of the abdomen is usually general and marked.	Less marked, unless the obstruction be situated in the lower segment.
Visible peristaltic waves absent.	Present and pronounced when the seat of obstruction is low, and course of wave may be reversed.
Tenderness decided and general.	Tenderness localized and usually slight.
Signs of effusion appear.	Less common, due to secondary peritonitis.
Auscultation negative.	Loud gurgling and splashing sounds audible over the abdomen (colon).

It must also be differentiated from *acute enteritis*, in which (particularly when due to toxic minerals) there is more apt to be diarrhea with considerable mucus and blood, an elevated temperature, intense gastric pain, associated with traces of the poison in the vomitus, and an absence of marked tympanites and fecal vomiting.

The various forms of *abdominal colic*, as enteralgia, hepatalgia, and nephralgia should not be mistaken for acute intestinal obstruction.

Course, Complications, and Prognosis.—A case of acute obstruction usually terminates within from two to seven days. The chronic form may last weeks, and even months, with progressive emaciation and anemia, until the superaddition of more or less acute symptoms, lasting from ten to fourteen days. The *prognosis* is unfavorable in the acute cases unless recognized early. The chronic forms, due to fecal or other impaction, often recover. Life may be prolonged by surgical interference in certain cases if they are taken in their inception.

Complications that may occur, as secondary peritonitis, gangrene, perforation, septico-pyemia, and enteritis, are all grave.

Treatment.—The treatment of acute intestinal obstruction is surgical. The only indication for therapeutic interference in acute obstruction is presented by the *incessant vomiting*. For this symptom no other measures are comparable to gastric lavage and starvation. It is well in most cases to withhold food for some hours to prevent retching, and aggravation of the condition. The lavage is strongly advised by Kussmaul, who claims that both the tension above the seat of stricture and the inordinate peristalsis are thus greatly diminished and exceptionally cured. It may be repeated every six hours. Hypodermic injections of morphin for the pain induce deceptive tranquillity. When the cause or character of the obstruction is unknown, cathartics should absolutely not be given. If it has been determined that fecal impaction is the trouble, it is still prudent to avoid purgatives until the main mass has been moved, as in many cases there are both paresis and inflammation at the seat of impaction, so that this class of agents would in most cases at least be useless, if not harmful. High rectal injections, copious, steady, and regularly repeated, are to be practised, using for this purpose preferably "a warm saline solution of olive oil" (particularly if scybala be present) administered while the patient is in an inverted position by means of a fountain syringe, so that the flow is readily controllable. The abdomen should be methodically kneaded and the patient at times well

shaken. This method of treatment, by hydrostatic pressure, can and must be carried forward without undue violence, and if it be unsuccessful, the intestines are to be inflated from a large india-rubber bag with air or hydrogen gas (Senn), of which two to three gallons may be cautiously introduced. Thorough manipulation of the abdomen from below upward, particularly if it be a case of intussusception, may be combined. In the latter condition inflation, early and perseveringly applied, cures the majority of instances. If not promptly relieved, immediate operation is to be encouraged and advised. Although the statistics of Fitz show the mortality in cases without operation to be lower (69 per cent.) than with operation (83 per cent.), I am convinced from personal observation that the less favorable results from abdominal section would not obtain if it were performed in due time.

In chronic obstruction the treatment of the underlying or etiologic conditions and various complications is to be conducted on general principles. Additionally, the patient's dietary is to be arranged with care, and the bowels moved with unfailing regularity, by the use of unirritating laxatives and enemata. If total obstruction persist despite medical treatment, surgical treatment—enterectomy, enterotomy, or other operation, as the circumstances of individual cases may dictate—is required.

The after-treatment consists in keeping the bowels active and regular by habit, diet, and an aperient pill if needed. Massage and electricity to the abdomen are found useful at this time.

Stenosis of the Duodenum.—Duodenal stenosis invariably develops secondarily upon morbid processes either in the duodenum or adjacent organs. Extensive studies of the subject have been made by Leichtenstern, Perry and Shaw, Wilms, Boas, Laffer, and others. The *causes* are principally intra-duodenal—ulcer of the duodenum (53.44 per cent.), carcinoma of the duodenum, sphincteric action of the muscular layer of duodenum, and gall-stones. Among extra-duodenal factors are "diseases of the pancreas, compression by the root of the mesentery, adhesions, morbid growths, kinking, and gall-stones."[1] *Symptoms.*—When the stenosis is suprapapillary or at the ampulla of Vater, marked dilatation of the stomach and of the duodenum above the stenosis usually occurs. When at the ampulla, obstruction may show acholic stools containing enormous numbers of fatty acid crystals and jaundice. Neither blood nor bile are found in the vomitus, as a rule. Stenosis below the ampulla of Vater is characterized by absence of meteorism, biliary, but never fecal, vomiting, the temporary disappearance of meteorism in the epigastrium, after vomiting, and early anuria. Stenosis due to kinking causes sudden severe pain, frequent vomiting, and decided shock. The *prognosis* is dependent on the special cause, while the *treatment* is appropriately surgical.

CARCINOMA OF THE INTESTINE.

(*Carcinoma Intestinalis.*)

CARCINOMA of the intestine is the commonest cause of chronic intestinal obstruction. The stenosis is usually partial. Primary intestinal carcinoma is rare in comparison with that of gastric carcinoma.

[1] *American Journal of the Medical Sciences*, Sept., 1912, by the writer.

Pathology.—When carcinoma attacks the intestine it is usually in the form of a cylindric-celled epithelioma, although it may assume the various forms as found in carcinoma of the stomach—namely, scirrhous, medullary, and colloid. The growth may be annular or semipolypoid, or it may occur as a diffuse infiltration of the bowel-walls. Ulceration of the surface of the carcinoma may take place, and the glandular structures of the abdominal cavity may reveal metastatic growths. The most frequent seat of intestinal carcinoma is the rectum, and next in order of frequency are the sigmoid flexure, the transverse and descending colon, the *papilla duodenalis*, the ascending colon, and the lower and middle portions of the ileum. The bowel is dilated above the constriction and filled with fecal matter. The muscular coat is hypertrophied. Below the narrowing the intestine may be atrophied. Rectal adenoma may develop into carcinoma (adenosarcoma).

Etiology.—Heredity and advanced age are of chief importance as predisposing causes. Antecedent intestinal ulceration may afford a nidus for carcinomatous growths. Carcinoma may invade the appendix. Harte, from statistics based on 101 cases, concludes that carcinoma occurs in from $\frac{1}{3}$ of 1 per cent. to 1 per cent. of all cases operated on for chronic appendicitis.

Symptoms.—A description of the course of *rectal carcinoma* belongs to surgical works. The symptoms of carcinoma of the bowel *above the rectum* are often vague, and vary according to the portion involved by the neoplasm. With or without an appreciable tumor in the abdomen the history is usually that of *chronic obstipation* of the intestines. There are irregular attacks of *sharp, colicky pains*, especially a few hours after eating, distressing defecation, obstinate constipation, perhaps alternating with diarrhea, sometimes vomiting, which may be feculent, and not rarely slight meteorism. The special symptoms of carcinoma of the *papilla of Vater* are vomiting, jaundice, and colic. The progressive emaciation and debility are marked. In advanced cases of stenosis the *feces* are passed in small, compressed lumps resembling sheep's dung.

Physical Examination.—*Inspection* of the abdomen may show the presence of a tumor along the line of the sigmoid flexure or colon; peristalsis may be seen above the site of the carcinoma, communicating its movements to the abdominal walls. *Palpation* may be resorted to in order to confirm the above, and the growth is then frequently found to be nodulated. A sign which is practically diagnostic of stenosis is a sudden appearance of small coils of bowel which vanish very quickly and reappear again (Boas). *Percussion* may give either dulness or a muffled tympany over the tumor and for some distance above (often sharply defined), on account of accumulated masses of feces.

Diagnosis.—This may rest, in some cases, upon heredity, the age, the evidences of the cancerous cachexia, sharp, radiating abdominal pains, bloody stools, and the detection of a firm and nodular tumor. Patchy pigmentation of the skin and small angiomas are corroborative features. An *x*-ray examination may prove an aid in the diagnosis. The prospects for early diagnosis are unfavorable (J. Boas[1]).

Differential Diagnosis.—(*a*) Carcinoma of the bowel above the rectum needs to be discriminated from other abdominal tumors. For example, *sarcomata, fibromata, myomata, adenomata*, and *cystomata* may produce symptoms of obstruction like those due to carcinomatous growths. The

[1] *Fortschr. der Med.*, February, 1906.

cancerous cachexia may be simulated by other conditions. The advanced age of the patient and the rapid and downward progress of the disease will, however, point toward malignancy. *Enteroliths*, *foreign bodies*, and old *peritonitic adhesions* may need to be excluded also. Fecal masses may exist above and overshadow the presence of carcinoma.

(*b*) *The portion of the bowel involved* by the neoplastic growth is also difficult of definite diagnosis. The locality of the tumor as detected by palpation, associated with special symptoms, is of value in arriving at a diagnosis of the diseased portion of the bowel. Heulin[1] has studied carefully primary cancer of the duodenum, and asserts that the comparative frequency of duodenal involvement is due to limited motion of the organ, being thus subject to injury. When it occurs above the papilla of Vater the symptoms greatly resemble those of *dilatation of the stomach*. An important point separating carcinoma above from that below the papilla is the presence or absence of bile in the vomit, being absent if situated above. When the carcinoma involves the papilla of Vater symptoms of biliary obstruction necessarily follow. A hard, nodular mass may sometimes be felt in the lower epigastric region; this coupled with increasing gastric dilatation and marked persistent jaundice would indicate carcinoma of the duodenum. It is apparent, however, that *carcinoma of the pylorus*, of the left lobe of the liver, or of the omentum or mesenteric glands, or a thickened cecum might all be easily confounded with carcinoma of the bowel at various adjacent parts of its course. The injection of fluid into the bowel may be resorted to in locating the probable situation of the growth. Thus, if obstruction from carcinoma exists in the sigmoid flexure, liquid will be arrested there and the rectum distended; while, if the stenosis be high up in the large or small intestine, the colon will be found comparatively emptied of feces and will be distended with the injected liquid. Carcinoma of the appendix usually gives rise to the symptoms of appendicitis with slight fever. These cases generally occur between the ages of twenty and thirty years (Kernhadjian).

Course and Complications.—Carcinoma of the intestine sometimes runs a rapid course, and may last but a few months; in the scirrhous variety, however, the disease may last two or three years.

Intestinal carcinoma may perforate the bowel and cause fatal purulent peritonitis. Or, owing to extreme distention by fecal accumulation between a cancerous stricture of the sigmoid flexure, for instance, and the resistant ileo-cecal valve, rupture of the colon, followed by peritonitis, may result. Extension of the growth into surrounding tissues, with ulceration, may lead to cellulitis, phlebitis, and pyemia; and extension from the rectum may cause purulent vaginitis and cystitis.

The **prognosis** is almost hopeless.

Treatment.—This, from a strictly medical standpoint, is simply palliative. The diet should be highly nourishing and easily assimilable, but when the symptoms of acute obstruction supervene the administration of food by the mouth is contraindicated. Opium or cannabis indica for the pain, and stimulants for the depression, may also be serviceable.

Lavage of the stomach gives decided relief for regurgitation on account of the damming back of accumulated food detritus.

[1] *Gaz. hebdom. de Méd. et de Chir.*, February 13, 1898; *Thèse de Paris*, 1897; Saunders' *Year-Book*, 1899, p. 194.

Carcinoma of the bowel may be treated surgically by colotomy, excision, lateral anastomosis of the bowel, enterostomy, and, if the growth be situated in the rectum, by extirpation by means of sacral resection (*Kraske's operation*). Operability does not necessarily depend upon the duration of the disease.

HABITUAL CONSTIPATION.

(*Costiveness.*)

Definition.—Chronic fecal retention, habitual infrequency, irregularity, difficulty, or insufficiency of the evacuations of the bowels.

Although constipation is a symptom, and although habitual constipation is frequently a symptom of chronic disease, the causal elements of the latter may be so indefinite and obscure that the former takes on all the individual importance of a functional affection. I describe habitual constipation, therefore, as a disease *sui generis* ("idiopathic").

Etiology.—In the majority of cases habitual constipation is the direct effect of a lack of expulsive or peristaltic power, and also of a deficiency of the hepatic and intestinal secretions. Schmidt, Strasburger, and Lohrisch claim that too thorough digestion and absorption of foodstuffs is one of the primary factors in the production of habitual constipation. The more recent investigations of Pletneu,[1] however, throw doubt upon the latter theory; he thinks that the more rational explanation is a deficient secretion in the gastro-intestinal tract. Two sets of causes operate to bring about these conditions of abnormal defecation:

General Causes.—(*a*) *Temperament:* it has been observed often that people of a nervous and "bilious" or motive temperament, of the dark type, are much troubled with constipation. Anemic brunets—persons having pale skin and dark hair combined—are particularly so affected, although alternating periods of diarrhea may supervene, owing to the hydremic state of the blood. "Torpid liver" and "sluggish bowels" are commonly held to be synonymous with these physical characteristics. (*b*) *Habit:* a sedentary life conduces to secretive inactivity. Thus, a lazy life, in which the calls of nature are irregularly attended to or habitually neglected, leads to frequent over-distention of the rectum and paresis, a common cause of chronic constipation. Again, the feminine false modesty (so called) that prompts a postponement and suppression of the desire to defecate in public places tends to obtund the sensibility of the rectum to fecal masses. The accumulation of these fecal masses causes paralytic over-distention, their hardening into scybala, and difficulty of expulsion. (*c*) *General bodily weakness, and diseases,* as neurasthenia, hysteria, anemic brain- and spinal-cord affections (causing inhibitory disturbances of the intestinal nerve-supply), acute fevers, hepatic disorders, especially the presence of jaundice, and the habitual dependence upon and use of purgatives. (*d*) *Diet:* the constant use of concentrated articles of food, as meats, in which little residual matter is left to stimulate the bowel to peristalsis. On the other hand, a very coarse diet may leave such an excess of residue as to cause fecal impaction. (*e*) A change of drinking-water, or water from chalky regions. Constipation is also caused by the use of an insufficient amount

[1] *Zeitsch. f. experim. Path. u. Therap.*, Band v., Heft 1, p. 186, 1908.

of water during the intervals between meals. (*f*) *Abundant and prolonged diuresis and diaphoresis*, by causing loss of fluids, also may induce chronic constipation.

Local Causes.—(*a*) *Atony of the abdominal muscles* from obesity or, in females, as a result of improper dress and many pregnancies. (*b*) *Atony of the large bowel* (the sigmoid flexure in particular) from chronic colitis. (*c*) *Pressure by tumors.* (*d*) The presence of *intestinal stenosis* from external or internal constriction. (*e*) *Congenital stricture* or *giant growth* of the colon, with coprostasis (as in Formad's case). (*f*) Tonic contraction of the muscular coat, as in basilar meningitis and lead-poisoning. (*g*) Enteroptosis. (*h*) Retention of the gastric contents.

Symptoms.—In cases in which there is no adequate cause for habitual constipation other than a constitutional or inherent peculiarity there may be the true appearance of perfect health. Nothing is complained of save the fact that an evacuation of the bowels occurs too infrequently. The term "constipation" is, individually speaking, almost wholly a relative one—*i. e.*, one person may enjoy good health with but one evacuation every other day, another with two passages per diem, while still another must have one stool a day, *cæteris paribus*, to feel perfectly well. The last is usually considered an average normal state with most people.

Symptoms of habitual constipation may be direct or reflex. *Direct* or *local* troubles are seen in the feeling of fulness, weight, and pressure in the perineum and abdomen. Flatulence, colicky pains, and alternating diarrhea occur not infrequently. The hurried and inattentive performance of defecation gives rise to the so-called "cumulative constipation," in which the accumulated feces are but partially evacuated with the movement, and the rectum consequently is not emptied. A sense of fulness then remains, and complete relief is not felt in these cases.

Reflex and *general* symptoms are malaise, languor, hebetude, irritability of temper, headache, facial flushing, palpitation, cold extremities, anorexia, vertiginous attacks, paresthesia, menstrual distress in women, sleeplessness, and bad dreams. Pressure on the sacral and visceral nerves may cause neuralgias. The tongue is coated. Palpation of the abdomen often shows the presence of doughy-like fecal tumors at the cecum or at the hepatic, splenic, and sigmoid flexures, or of bologna-like masses at intervening places. In marked cases attacks of nausea and vomiting, with diarrhea, may ensue; fever may also be present.

Complications and Sequelæ.—Hemorrhoids, ulcerative colitis, perforation, and enteritis may be associated with chronic constipation. Not rarely do we have as results dilatation of the colon or sacculation, with the presence, in old people mainly, of *enteroliths* (calcified scybala); also intestinal obstruction and typhlitis, or cerebral hemorrhage or hernia from violent straining efforts.

Diagnosis.—Bearing in mind the relativity of constipation in different individuals, the diagnosis is read at sight. The detection of the causes is not difficult, though sometimes tedious. An etiologic diagnosis may require modern methods, such as an *x*-ray examination, the examination of the feces according to Schmidt and the procedure known as rectoromanoscopy. "In spastic constipation, hypertonicity is found in the distal portion of the colon, and normal tonicity with hypermotility in the proximal portion" (Singer and Holzknecht). Hypochondriasis or melancholia should be carefully placed either as precedent to or conse-

quent upon chronic constipation, the nervous condition often acting to produce the latter, and *vice versâ*.

The **prognosis** is usually favorable, but should be guarded.

Treatment.—Hygienic.—Causative factors must, of course, be removed, modified, or lessened. Systematic regularity as to time and frequency and sufficiency of movements of the bowels should be enjoined upon and practised by the patient. Exercise is of signal value, and particularly horseback riding or gymnastic motions that bring the abdominal muscles into play. Attention to the calls of nature should be esteemed a duty, and proper time and heed must always be given to the completeness of defecation. The *dietetic regimen*, if properly looked after, often avails much in relieving this affection, and foods calculated to be easily digestible, but leaving a moderate residue after digestion, are to be recommended. Such are bread made of unbolted flour, plenty of vegetables and fruits, butter, and such laxative articles as figs or honey. Certain substances which swell from imbibing water, but are not digested or absorbed, such as agar-agar, liquid vaseline, coarse, raw, wheat bran, and the like, may be advantageously taken with the food. Prof. L. B. Mendel, experimenting with agar-agar, found that the greatest part of it was excreted in the feces unchanged; this substance resists intestinal enzymes and bacterial decomposition, and is recommended for chronic constipation. Gomperts[1] has had experience of its use, and advises 15-gram doses twice daily, eaten with milk or cream, the same as a modern breakfast food. After regular movements of the bowels have begun, the dose of agar is reduced. Luke[2] has had good results with the use of sour milk. A glass of cold water taken regularly at bed-time and in the morning before breakfast is efficacious and a point of common knowledge.

Remedial.—The methods and means offered for the cure of chronic constipation number legion. From the little aperient pill or "peristaltic persuader" to the cannon-ball rolled externally along the course of the large bowel is made up such a list of drugs and measures as to leave untenable any plea of lack of resource. *Drugs occupy a subordinate part in the treatment of habitual constipation.* Indeed, their use should be restricted to those periods when the bowels become unusually obstinate. The constant use of laxative and purgative drugs tends to a confirmation of the condition.

I have found of value in lithemic and dyspeptic subjects the laxative bitter waters (Hunyadi Janos, Kissingen, Friedrichshall, Carlsbad).

Among those laxatives and cathartics most commonly used may be mentioned aloes, rhubarb, Rochelle and Epsom salts, compound licorice powder, castor oil, jalap, senna, mercury, colocynth, and podophyllin. Important adjuncts in combination with one or more of the above are the extract of nux vomica (or strychnin) and the extracts of belladonna, hyoscyamus, and physostigma. The much-used aloes, strychnin, and belladonna pill can be used for a considerable length of time in the hope of stimulating a normal intestinal and sphincteric activity, and thus inducing even a cure in some cases. The formula is as follows:

℞. Aloin., gr. iij–v (0.194–0.324);
Strychninæ sulphat., gr. $\frac{1}{3}$–$\frac{1}{2}$ (0.0216–0.0324);
Extr. belladonnæ, gr. ij–ijss (0.129–0162).
M. et div. in pil. No. xx.
Sig. One pill at bedtime.

[1] *Amer. Jour. Med. Sci.*, October, 1909.

[2] *Practitioner*, 1910, lxxxiv., 653.

Sulphur in confection, along with the official pill of aloes and iron, has been recommended for the habitual constipation of anemia. In senile atony of the bowel, with much flatulence, a laxative pill having in combination asafetida or capsicum is often beneficial.

The subjoined formulæ are also serviceable:

℞. Ext. cascar. sagrad., ʒss (2.0);
Ext. nucis vomicæ, gr. iv (0.259);
Ext. physostigmat., gr. iij (0.194);
Ext. belladonnæ, gr. ij (0.129).
M. et ft. in pil. No. xx.
Sig. One at night, or night and morning.

(Aloes, gr. j (0.0648), or podophyllin, gr. ii–iij (0.129–0.194), or ext. colocynth. comp., gr. ii–iij (0.129–0.144), may be substituted for cascara in the foregoing formula.) Spastic constipation (*e. g.*, that due to lead intoxication) may at times be relieved successfully by the use of sedatives, such as bromides, valerian, asafœtida, and opium.

The **mechanical** means of relieving habitual constipation, as by *enemata*, are injurious if long continued, by reason of their irritating effect on the rectal and colonic mucous membrane. At times when the stomach is weak or irritable, a loaded bowel may be relieved by an ordinary enema of soap and water or by one containing ½ to 1 ounce (16.0–32.0) of castor oil, with 1 or 2 drams (4.0–8.0) of oil of turpentine if there be some flatulence. Glycerin enemata, containing from ½ to 2 ounces (16.0–64.0) of the agent, may be used. Fleiner has suggested oil-injections. From 2 to 4 ounces of sweet oil warmed to body-heat by standing the containing bottle in a vessel of hot water, may be injected slowly through a piston-syringe on retiring and retained until next morning. Riesman,[1] who has had a considerable experience of their use, speaks strongly in favor of oil-injections. Paraffin injections of the consistency of a salve at the temperature of the body are warmly recommended by Lipowski; they tend to prevent absortion of fluids in the rectum. *Suppositories* of soap, molasses candy, or glycerin are included in the armamentarium. *Massage* also claims an important part in the relief of habitual constipation. It acts by stimulating the peristalsis and the abdominal muscles, and should be employed at set times in the day preceding a desired evacuation of the bowels. The hand of the *masseur*, or that of the trained patient even, when systematically used in this way, may be effectual when all other means have failed. The regular rolling of a metal ball along the course of the greater gut may be mentioned for its novelty as well as for its undoubted efficacy. The application of the faradic current to the abdominal walls or galvanization of the lumbo-abdominal circuit deserves proper trial in many cases. Hydrotherapeutic measures, or cold sponging and baths, are nearly always useful adjuncts.

DILATATION OF THE COLON.

(*Ectasia of the Colon.*)

This is usually a chronic condition, though not rarely it is acute. It may also be general, but in the majority of cases it is confined to the

[1] *The Therapeutic Review*, February, 1904.

colon, and particularly to the sigmoid flexure. The *postmortem* findings are those of hypertrophic dilatation of the bowel, and rarely ulcerative and catarrhal lesions of the intestinal mucosa are noted. The sigmoid flexure is prone to become dilated in subjects in whom it is congenitally elongated. Atony of the muscular coat is a leading causative element. The most distinctive features are *constipation*, which generally dates from infancy, and great *abdominal distention*. Peristaltic waves may be visible upon the surface. The condition may exhibit constipation alternating with regular daily movements, and the distention changing to a normal softness of the abdominal parietes. I have recently seen a case of this kind in a male aged twenty-seven. In the *treatment* of the constipation, lavage of the intestine with a very long tube is superior to laxatives or purgatives. A *diet* calculated to prevent or relieve constipation is indicated. Vegetable foods leaving a gross residue should be prohibited. Massage, galvanism, and hydrotherapy are all capable of beneficial effects in suitable cases. Strychnin is a valuable remedy, as is also betanaphtol for its antifermentative action, and in cases attended with constriction surgical measures should be considered.

COLOPTOSIS.

(*Displacement of Colon.*)

Coloptosis.—Displacement of the colon in various directions is occasionally observed. In 7 cases reported by W. W. Babcock[1] the stomach was not found to be displaced. Certain sections of the colon show accessory loopings and tortuosities, as the result of an unusually long mesentery. The transverse colon and cecum are most often seen to deviate from their normal course and may depend upon anomalous formation; while tympany and obstinate constipation may favor the condition. The symptoms are those of dilatation of the colon (p. 883).

Diagnosis.—Irregular abdominal distention with tympany over such areas. Inflation of the colon with gas or water may enable one by careful percussion to detect with some certainty this condition. At times the position of the colon is shown by skiagraphs taken after the colon has been well filled with an emulsion in which bismuth is contained.

Treatment is practically that of dilatation of the colon.

INTESTINAL AUTO-INTOXICATION.

THIS condition was alluded to in the discussion of lithemia and also that of chronic gastritis, but it demands brief special description. Although not a pathologic entity, it is readily recognized clinically and known to be due to the absorption into the circulation of toxic bodies, namely, albumoses and leucomaines, which are formed during intestinal digestion. If these substances enter the circulating blood rapidly and in sufficient quantity, acute intestinal auto-intoxication is the result; this often assumes the nature of a bilious attack or migraine in the course of the chronic variety, or it may follow obstinate constipation.

[1] *International Medical Magazine*, March, 1901.

The chronic form is the most common, if we except children, in whom the reverse is true.

The **etiology** is varied and often obscure in the case in question. The ingestion of proteins in excessive amounts is doubtless the most potent causative factor. Fat and sugar taken in amounts above the physiologic capacity of the organism are also responsible for the condition in certain cases, and the same may be true of certain abnormalities of metabolism. Moreover, obstructive conditions of the intestinal tube, constipation, and chronic appendicitis or any pathologic changes that will interfere with the function of motion, may act as causes.

Fortunately for mankind, certain protective functions often prevent the development of auto-intoxication. Thus the liver destroys the toxic bodies as a rule, but deficient hepatic function leads to their formation in excessive amounts. Again, the digestive tract, skin, and especially the kidneys are normally active in the elimination of these poisonous substances. It is the putrefactive products not oxidized to indican that constitute the toxins.

Diagnosis.—"The physician should never make the diagnosis of intestinal auto-intoxication until he has made a careful differential diagnosis eliminating everything else" (Forchheimer). The group of symptoms most commonly observed is as follows: Headache (often of the type of migraine), vertigo, a high-tension pulse, constipation, signs of hepatic congestion, flatulence, indicanuria, at times albuminuria, furred tongue, and the presence of chronic ulcerative stomatitis (Rigg's disease). Unless the indican and conjugated sulphates in the urine be excessive in amount, they cannot be regarded as indicative of intoxication. In a considerable proportion of cases vomiting and often diarrhea are associated with fever and skin eruptions (erythema, urticaria), or arthritis resembling rheumatism, or distressing myalgic pains occur. The constipation may alternate with diarrhea or mere irregularity of bowel action exists. "A careful *physical examination* of the colon, more particularly by light percussion, will indicate the presence of an overfilled condition of the bowel, most commonly in its descending portion. Palpation may detect a doughy mass or masses in one or more sections of the colon, and after removal of these fecal accumulations more or less thickening of the intestinal walls, due to a catarrhal state with infiltration, may be detectable."[1] The nervous manifestations most prominent in the clinical picture are a feeling of languor, insomnia, loss of physical and mental energy, vertigo, and irritability, with occasional headaches.

Treatment.—In the *acute form* the treatment of the cause suffices as a rule. Purgation by means of mercurials followed by a saline are indicated first of all. The *diet* must be fluid and much restricted in amount, milk and gruels being especially serviceable. Water is to be taken freely, if retained. In the *chronic form* digestible solids, composed principally of vegetables, milk (particularly sour milk), bread, and cereals should form the protein in the diet. These, with fruits, bacon, and butter, in right proportions, will give 65 grams of protein daily. While it is the proteins in abnormal amount that excite the fermentative and putrefactive processes, I have observed instances in which the carbohydrates (sugar, starch) seemed to act as the cause, hence the latter must

[1] *Journal of the Indiana State Medical Association*, by the writer, July, 1908.

sometimes be excluded from the dietary. Laxative articles of food and such as leave a maximum residue in the intestines are efficacious in overcoming constipation. The use of two or three glasses of cold water or mineral water on retiring, on rising, and between meals favors elimination, both through the bowels and kidneys.

The function of the latter is of the highest importance as a channel of elimination. If the above suggestions do not afford thorough relief daily to the bowels, warm saline laxatives before the morning meal are to be advised. Among the most efficient are sodium phosphate and sodium sulphate, or the aperient waters, as Hunyadi, Apenta, Carlsbad, Veronica, and the like. It may be necessary to administer a mercurial from time to time to maintain biliary secretion. Gastric lavage and colonic irrigation should be resorted to in the acute gastro-intestinal attacks. The mouth condition and gastric features must be treated as recommended in appropriate sections of this work. Cutaneous elimination is to be aided by hot baths or Turkish or Russian baths; they must be carefully adapted to the individual cases. Physical exercise deserves proper trial in most cases and massage is also a useful adjunct when active exercise is unsuitable.

For the flatulence which often proves annoying, intestinal antiseptics are indicated. Of undoubted value for this purpose are β-napthol, salol, benzosol, and menthol. These remedies should be administered as suggested by Forchheimer, namely, in the form of an intestinal pill or one dissolved only in an alkaline medium, and Waldstein recommends a coating with an alcoholic solution of shellac containing salol to accomplish this object. It has been suggested to raise the content of Bacillus coli communis in the bowel "by instillation either of the autogenous mixed forms or strains from other individuals" (Bassler). The underlying and etiologic conditions and complications must receive due attention in every case.

NEUROSES OF THE INTESTINE.

As in the case of the stomach, these embrace derangements of (*a*) secretion, (*b*) sensation, and (*c*) motion.

(*a*) SECRETORY DISTURBANCES.

Unquestionably the intestinal secretion may, through a purely nervous influence, be augmented. This manifests itself most frequently in the primary morbid secretion of mucus (*mucous colic*) and in membranous enteritis. Moreover, the fact that an actual catarrh of the intestinal mucosa may supervene as a secondary event is undeniable.

MUCOUS COLIC.

(*Colitis Colica, Enteritis Membranacea.*)

Definition.—A peculiar pathologic condition, chiefly of the large intestine, attended by a morbid secretion of mucus.

Pathology.—In the truly primary form there are no morbid lesions discoverable in the mucosa. From mucous colic we must distinguish membranous enteritis, which is associated with an inflammatory process

of the mucosa and accompanies typhoid fever, dysentery, and many other affections. This is a catarrh of the colon, while true mucous colic, the disease under consideration, is a functional (secretory) disturbance.

Etiology.—Sex has a decided influence, 80 per cent. of cases are observed in neurotic women. It is rare in children. Direct mechanical irritation of the rectum (horseback-riding, bicycle-riding, hardened scybala, etc.). Bacteria are believed to play a causative rôle, particularly the Bacillus coli communis.

Symptoms.—I have found the condition associated with a constipated habit—a fact that may, in part, explain its occurrence, since time is thus allowed for cast-formation. The important feature is the *passage*, at *varying intervals*, of *long*, *ribbon-like threads of mucus*, or of more or less *perfect casts of the gut*, with *tenesmus* and severe *colicky pains*. The stools consist of a turbid ground-substance, which, on the addition of acetic acid, becomes opaque and striped; cellular detritus, consisting partly of granules and partly of cellular elements, including blood. Symptoms of neurasthenia are present and are often quite pronounced.

The individual paroxysms vary in *duration* from one to ten days or more. In one case observed by me the attacks lasted about two days, recurring regularly at the end of every three months. Ordinarily the recurrence is after a shorter interval.

Diagnosis.—A microscopic examination of the pieces of membrane insures the diagnosis. It is to be recollected, however, that membranes are not passed with every attack, and that there is a complete absence of the signs of organic disease between the attacks of colic.

Course and Prognosis.—The disease pursues a very chronic course and lasts for many years. The bodily nutrition suffers considerably if the attacks are frequent and severe, but, as a rule, this does not occur until a late stage in the affection. The risk to life is slight.

(*b*) SENSORY DISTURBANCES.

It may be noted here that the sensory nerves of the intestines, as well as the inhibitory and vaso-motor dilators, are traceable to the splanchnics. Increased sensibility of the sensory nerves produces—

ENTERALGIA.

(*Neuralgia of the Intestine.*)

Etiology.—This is commonly met with in hysteric, neurasthenic, and anemic subjects. It occurs as a reflex neurosis, as in the case of cold, gout, and irritative lesions of the pelvic organs (kidneys, liver). Enteralgia is symptomatic of many local affections and conditions that induce direct irritation of the sensory nerve-filaments of the intestine; among these are inflammation of the mucosa, foreign bodies, gall-stones, abnormal distention with gas, and enteroliths. Under these circumstances the condition is associated with increased activity of the motor nerves or heightened contraction of the muscularis, forming true intestinal colic. In lead colic it is probable that the lead acts directly upon the nerves or their ganglionic cells. I have repeatedly observed the action of certain exciting causes (*e. g.*, nervous shocks).

Symptoms.—Enteralgia may develop very *suddenly*, but oftener it

sets in less abruptly, and is then attended with eructations of gas, expulsion of flatus, and the like. In the fully-developed attack the *pain* may attain to great violence, causing the patient to "bend double" or even faint, and its character is variously described as boring, tearing, or cutting. The pain may be confined to a circumscribed spot or may be diffuse. The attacks are sometimes brief, or they may be characterized by a sudden subsidence. At other times they last for days or perhaps weeks, and then subside gradually. *Recurrences* are common.

Hypogastric neuralgia is a term applied to neuralgia affecting the sensory nerves lying in the most dependent segments of the intestine. Here the nerve-fibers entering into the hemorrhoidal plexus are involved. It is caused chiefly by tabes, by hemorrhoids, and by the neurotic state so common to females. This form of neuralgia has its seat in the hypogastric region, and is accompanied by a distressing sensation of pressure in the rectum and bladder, and by an irresistible desire to go to stool; pains also radiate to the sacrum, thighs, and perineum.

Diagnosis.—The various organic diseases and conditions mentioned under Etiology, in the course of which colic is a common symptom, must be separated from the true neurotic enteralgia. The former are distinguished from the latter by a group of symptoms peculiar to themselves (fever, aggravation of the pain upon pressure, vomiting, constipation, or diarrhea), and by the usual definite causes furnished by the history.

Renal and *hepatic colic* bear a superficial similarity to enteralgia. The former conditions, however, are distinguished first by the seat and direction of the pain, and secondly by the appearance of jaundice in hepatic colic and of hematuria in renal colic. *Rheumatism* of the abdominal muscles is easily eliminated, since it is generally combined with rheumatism in other parts of the body; the pain is also greatly increased upon throwing the muscles into contraction, as in stooping or rising; finally, it vanishes in response to the action of the salicylates.

DIMINISHED INTESTINAL SENSIBILITY.

This implies diminished peristalsis or constipation. A greater or less degree of anesthesia of the bowel attends, with a loss of desire to go to stool and an accumulation of feces in the rectum. This is a usual concomitant in many diseases of the brain and cord, with which paralysis is associated. Motor innervation may remain intact, and when atony of the intestine is absent spontaneous movements of the bowels occur; when atony is present, however, to a marked degree (motor paralysis), the feces must be artificially removed.

(*c*) DISTURBANCES OF MOTILITY.

When the contractility of the muscularis is increased from purely nervous causes the result is—

NERVOUS DIARRHEA.

This condition presents no morbid lesions. The increased contractility results from an exaggerated irritability of the motor nerves of the bowels. It may also result from morbid processes in the central nervous

system and in other organs of the body; in short, the condition may be a reflex one.

Examples of this sort are caused by tabes, by gastric disturbances, as after certain foods and drinks, by dentition, and the like. Most cases, however, are encountered in persons having an abnormally irritable nervous organization—*i. e.* the neurasthenic and hysteric classes. In such the effect of mental excitement, of fright, and similar psychic influences is to induce diarrheal evacuations.

Symptoms.—The *stools* vary in number from two or three to twenty-four or more daily. In rare instances they are soft—not truly diarrheal—and formed, yet they may be quite frequent. Blood and mucus, pus, and other morphologic elements are absent from the dejections. It is characteristic of nervous diarrhea that the stools follow one another in rapid succession, usually during the morning hours, and then discontinue for the greater part of the day. The bodily nutrition is often well preserved.

In the **diagnosis** organic affections of the bowel are to be carefully eliminated.

ENTEROSPASM.

(*Spasm of the Intestine.*)

By this term is meant a concurrent spasm of both the longitudinal and circular muscular fibers, usually inducing spasmodic constipation, and sometimes total, though temporary, occlusion of the bowel.

Its *causes* are similar to those of nervous diarrhea, and the condition is *clinically* related to enteralgia. Neither pain nor constipation, however, is a constant feature. The stools may assume the form of a ribbon or of large rounded masses (sheep's dung), but they are not pathognomonic. They may also be covered with mucus. Ewald distinguishes between an idiopathic and a secondary or symptomatic spasm, the latter being a concomitant of basilar meningitis and of chronic lead-poisoning (see also Constipation, p. 880). Another variety affects the rectum (*proctospasm*), and is generally secondary to some other rectal affection, as fissure of the anus; it may, however, occur as a neurosis in the hysteric and nervous class of subjects.

The *diagnosis* of true functional enterospasm can only be made after all organic causes that may produce spasm of the bowel have been excluded.

CONSTIPATION.

This is a common condition as a neurosis. It is due to an abnormity of function of the intestinal nerves that leads to a weakened peristaltic action, and is met in hysteria, neurasthenia, and the various forms of psychoses. Central nervous affections often manifest atony of the intestine as a symptom; hence this form is not a disease *sui generis*. Cases of this class do not respond to any variety of cathartics (Ewald).

Paralysis of the external sphincters is a common concomitant in a great variety of local (catarrhal) and central nervous diseases. Under these circumstances the act of defecation may be purely *reflex*, owing to

loss of control of the voluntary muscles; or it may be *voluntary*, except when the person affected is not upon his guard, or during mental excitement, micturition, sneezing, and like influences.

Treatment of Intestinal Neuroses.—A suitable change of environment, including an appropriate arrangement of the dietary, is of primary importance, and is uniformly applicable in this class of sufferers. Further, the treatment of special cases has peculiar reference to the character of the nervous derangement. After making an accurate diagnosis a search for the factors of the greatest etiologic importance should be made, and these must then be vigorously assailed.

In the *secretory neuroses* an associated mucous colic must be corrected, the digestion is to be improved if faulty, and the obstinate constipation overcome. For the latter symptom enemata containing ox-gall, either alone or in combination with salines, are especially serviceable. Kussmaul and Fleiner have obtained the best results from regular large oil-enemata administered once or twice daily. During the painful attacks copious enemata of normal saline solution to which has been added oil of peppermint (5 drops to the pint at a temperature of 100° F.) will sometimes bring speedy relief from the pain and other distressing colonic symptoms, and will assist nature's efforts at separating the cast-formations. Pain must be at times relieved by morphin. The results of treatment of mucous colic, however, are unsatisfactory. Surgical measures have been adopted in selected cases.

In the *sensory disturbances* in which the activity of the sensory nerves is increased (enteralgia and hypogastric neuralgia) the treatment may be considered under two headings: first, the relief of the neuralgic pains; and secondly, the correction of the causes or conditions on which the enteralgia depends. If the pain be severe, opium or morphin may be required. Especially good as an antispasmodic is codein, which may suffice in all save the severer cases. The object should be to give the minimum amount of the opiate that will meet the necessities of the case, with a view to obviating a resultant constipation. In hypogastric neuralgia I have found suppositories containing opium to be little short of magical in their effects.

In cases in which there is constipation due to diminished sensibility, with a loss of motor innervation (atony of the bowel), the feces must be artificially removed unless the underlying condition can be successfully overcome. It is especially important that the environment—physical and psychic—be so regulated as to bring about an improvement in the general condition of the patient. It may become necessary to employ tonic preparations of strychnin, iron, or arsenic.

The treatment of nervous diarrhea involves the same principles, so far as the indication presented by the peculiar nervous organization is concerned, as in the sensory and secretory neuroses. It is especially important to prevent the operation of the direct causes—fright, mental excitement. Astringents and intestinal antiseptics are not called for, unless the bodily nutrition be affected thereby. Enterospasm is to be met by the same remedies that are used to control enteralgia.

IX. DISEASES OF THE LIVER.

ANOMALIES IN SHAPE AND POSITION.

Altered Shape.—Malformations of the liver may be either the result of disease or of pressure of adjacent structures. The former "may be due to syphilis, fœtal peritonitis, or possibly to tuberculosis" (Rolleston). Of the latter class the most important cause is tight lacing, met with almost exclusively in women and producing the so-called "corset-liver." The lower part of the right lobe of the liver is usually the part affected; the hepatic parenchyma is atrophied, owing to continued compression, and shows deep grooves that correspond to the position of the lower ribs. The connective-tissue capsule and the peritoneal coat are both thickened at this point. In marked cases the right lower lobe may become converted into a dense fibrous band. Among other acquired causes of anomalies in shape may be mentioned deformities of the vertebræ and ribs, or tumors of the ribs or adjacent structures (pylorus) pressing against the liver. Moser invites attention to multiple lobulation, as many as 16 lobules having been found; this is due to pathologic causes and is not a morphologic phenomenon.

Diagnosis.—Rarely, clinical symptoms are present. "A constant *sensation of pressure* and *pulling* is felt in the hepatic region, and sometimes, as a result of venous stasis, there is a temporary but decided swelling of the isolated portion, and, possibly, *violent pain* and indications of irritation of the peritoneum, such as *vomiting* and an approach to *collapse*. Jaundice is rare in consequence of this deformity" (Strümpell). The danger of this condition lies in a possible mistaking it for an *abdominal tumor* (Pepper), *amyloid disease*, *passive congestion*, or *new growths* of the organ (Strümpell).

Primary alterations in the shape of the organ may be due to active or passive congestion, hereditary syphilis, hypertrophic or atrophic cirrhosis, acute yellow atrophy, carcinoma, abscess, or hydatid cyst. The accompanying *symptoms* would, of course, be those of the special disease causing the deformity.

Anomalies of position are not infrequently met with, the organ being displaced upward, downward, or laterally. The most common cause of lateral displacement is an abnormal lengthening of the suspensory ligament. The organ may occupy the epigastric region or be displaced into the lower part of the abdominal cavity, but a change in the posture of the patient or external pressure is often sufficient to replace the organ. The *symptoms* (if present at all) consist of a dragging sensation, often amounting to pain that may be severe and referred to the right shoulder. On *physical examination* palpation may reveal a fissure between the right and left lobes, together with a movable tumor presenting the size and normal outlines of the liver, which by manipulation may be reposited. Percussion gives tympany over the normal hepatic area, which changes to flatness when the organ is pressed, or falls into its natural position.

Displacement upward may result from gastric or intestinal distention,

marked ascites, or an abdominal tumor; while downward displacement may be due to a mediastinal tumor, emphysema, or a pleural effusion.

Diagnosis.—Among the conditions likely to be confounded with movable liver may be mentioned *carcinoma of the omentum* or of the *pylorus*, *dermoid cysts*, *tumors of the ovary* and *uterus*, *hydro-* or *pyonephrosis*, *tumors of the kidney*, and *chronic proliferative peritonitis*. By a careful study of the symptomatology, and in the absence of the normal physical signs over the hepatic area, the diagnosis can usually be established, although marked fatty degeneration or atrophic cirrhosis may coexist with any of the above conditions and cause marked diminution in the area of hepatic dulness. Steele's careful studies of 100 cases of floating liver show that colicky pains, often accompanied by jaundice and simulating *hepatic colic*, occur in nearly 40 per cent. of the cases.

The **treatment** of movable liver is merely palliative, and consists in the application of a suitable bandage for preventing the displacement.

JAUNDICE.

(*Icterus.*)

Definition.—A condition in which the tissues and secretions are stained with bile-pigments. Jaundice is not a disease, but a symptom.

Hepatogenous or *obstructive jaundice* is more commonly seen in—(1) Inflammatory swelling of the duodenum or of the lining membrane of the duct, which is by far the most common factor in its causation, and demands separate consideration (*vide infra*, Catarrhal Jaundice); (2) Foreign bodies within the ducts, as gall-stones or parasites; (3) Stricture or obliteration of the duct; (4) Tumors within the duct or obstructing its orifice; (5) Pressure on the duct from without, as by a tumor of the liver, stomach, pancreas, or omentum; also by fecal accumulations, displaced organs (*e. g.* floating kidney), a pregnant uterus, enlarged glands in the fissure of the liver, and, more rarely, by abdominal aneurysm; (6) Lowered blood-pressure in the vessels of the liver favoring resorption of bile, as in simple icterus of the new-born (Frerichs).

CATARRHAL JAUNDICE.

(*Hepatogenous Jaundice; Icterus Catarrhalis; Duodeno-cholangitis; Inflammation of the Common Bile-duct.*)

Definition.—A condition characterized by a discoloration of the tissues from retention and absorption of bile and resulting from a catarrhal inflammation of the lining membrane of the ducts, more especially the larger, and of the duodenum.

Pathology.—On examining a liver and gall-bladder *in situ* the former is usually found enlarged, lighter in color than normally, and of a distinct icteroid tint. On making a longitudinal section drops of bile can be collected on the edge of the section-knife.

The gall-bladder is found distended with bile, and on firm pressure a

tough plug of mucus is usually expelled from the common duct into the duodenum, after which bile flows into the intestine freely. The mucosa lining the *ductus communis* is swollen and inflamed, and the catarrhal process may extend to the cystic, and in some cases to the hepatic, duct. As a rule, that portion of the common duct lying in the intestinal wall is more frequently and more deeply involved. If the disease becomes chronic, a formation of connective tissue occurs, owing to the irritation caused by the retained secretion, and atrophy of the liver-cells, with biliary cirrhosis, may result. Suppuration is rare.

Toxic (hematogenous) jaundice, so-called, has for its lesion extensive catarrh of the intra-hepatic bile-ducts from their origin. Here duodenal catarrh is not necessary for the production of jaundice. It was formerly assumed that the pigment (hemoglobin) was liberated in the blood; but Stadelmann and others have shown that the bile containing the poison, or its irritant products (toxins), excite inflammation of the finer ducts.

Etiology.—Simple catarrhal jaundice results in a majority of cases from extension of inflammation in gastro-duodenal catarrh, and the principal *predisposing causes* are as follows: (*a*) Exposure to cold and wet; (*b*) The use of improper foods, including faulty cooking and improper mastication; (*c*) The excessive or prolonged use of irritants (tea, coffee, alcohol); (*d*) Prolonged anxiety and mental or physical overwork; (*e*) Certain acute diseases, as pneumonia, relapsing fever, typhoid fever, and malaria (toxic jaundice, *vide supra*); (*f*) Portal obstruction, occurring in chronic heart- or kidney-disease; (*g*) More rarely it has occurred in epidemic form, although said to be common around the Mediterranean Sea. Barker and Sladen[1] found that food (probably meat) was the most likely source of the infectious agent in an epidemic.

Symptoms.—Preceding the development of the distinctive features by several days, dyspeptic symptoms are in evidence (*vide* Gastro-hepatic Symptoms). The principal symptoms in detail are: (*a*) *Icterus*, or tinting of the body surface may be the first symptom noticed in this condition, appearing usually on the forehead and neck and rapidly spreading over the entire body. The conjunctivæ also early become discolored, and the general hue, though variable, is commonly a bright lemon-yellow. In chronic cases the color is apt to change to a bronzed or deep-green tint.

(*b*) *Secretions and Excretions.*—The urine and sweat are often found to contain bile-pigment, the patient's linen frequently being discolored. In extreme cases the urine may be dark-green in color, while in those of average severity it is of a lighter or deeper greenish-yellow hue. The shaken specimen foams, and the froth has a yellow color-tint. Often the presence of bile is detected before any noticeable coloring of the conjunctivæ occurs. In cases of intense or long-standing jaundice albumin and tube-casts may be present, and the latter may be bile-stained.[2] Hyaline casts are often found in cases of moderate intensity.

[1] *Bulletin Johns Hopkins Hospital*, October, 1909.

[2] *Tests for Bile.—Gmelin's test*, or the play of colors, consists in bringing a few drops of urine in contact with the same quantity of commercial nitric acid on a plain white slab, whereupon various shades of yellow, green, red, and violet are produced.

Rosenbach's test is made by filtering the suspected urine and touching the filter-paper with a drop of nitric acid. If bile be present, a green circle will form at the point of contact. (See also Choluria.)

The bowels are constipated, and the stools are pale-drab or slate-colored; they are usually very fetid. Diarrhea, however, may be present, owing to the production of irritating substances and decomposition.

The tears, saliva, and milk are rarely stained with bile-pigment. The expectoration also is rarely tinted, unless pneumonia or some form of pulmonary infiltration coexists.

(*c*) *Circulation.*—The pulse, although not appreciably altered in volume or tension, is usually slow (often 30 or even 20 beats per minute), though this is not an unfavorable symptom.

(*d*) The *temperature* is usually normal, although slight elevations may occur (100°–101° F.—37.7°–38.3° C.).

(*e*) *Gastro-hepatic Symptoms.*—Dyspeptic symptoms—viz. anorexia, a sense of fulness after eating with flatulence, acid eructations, nausea and vomiting, accompanied by a dull, heavy pain over the hepatic area, with some tenderness on pressure—are present. These often develop insidiously; rarely they occur suddenly with a severe rigor or chill, violent headache, and vomiting—*e. g.*, at the onset in the epidemic form.

(*f*) *Cutaneous Phenomena.*—Pruritus or itching often becomes a troublesome symptom, being more common, however, in the chronic forms. Lichen, urticaria, furuncles, and sweatings (diffused and localized) may develop, the latter being often limited to the skin covering the abdomen and the palms of the hands.

A peculiar disease of the skin called *xanthelasma* or *bita higoidea* may also occur. It consists of bright-yellow spots, slightly elevated, appearing on the eyelids, and rarely on other parts of the body.

In the severer forms ecchymoses and sometimes profuse hemorrhages may occur into the skin and mucous membranes. These are usually associated with symptoms of a grave type. In chronic forms the coagulation time of the blood is much delayed.

(*g*) *Nervous Symptoms.*—Headache and vertigo are common; irritability of temper, despondency, and wakefulness or mental dulness almost equally so. With the oncoming of darkness vision may grow indistinct (*hemeralopia*) or it may attain unnatural clearness (*nyctalopia*). Rarely, objects look yellow (*xanthopsia*). The nervous phenomena observed in catarrhal jaundice are attributable to the effects of the bile-acids. In certain cases, however, associated with destruction of the hepatic substance, as in acute yellow atrophy, carcinoma, cirrhosis, and fatty degeneration, grave cerebral symptoms (acute delirium, convulsions, and coma) may develop suddenly and prove fatal. This class of symptoms has been named *acholia*, *cholemia*, or *cholesteremia* (the latter owing to the mistaken supposition that cholesterin is the poisonous product). The true nature of the toxic agent in the blood is unknown. In some fatal terminations of this character death was due directly to a renal complication.

The **physical signs** in a case of simple catarrhal jaundice show on palpation and percussion an increase in the hepatic area, the lower border of the liver projecting in some instances several fingers' breadths below the ribs. Rarely, the distended gall-bladder projects below the lower lobe of the liver, as when there is complete obstruction near or at the duodenum, and then it can be distinctly palpated.

Diagnosis.—The etiology (errors in hygiene and diet), a history

of previously existing gastro-intestinal catarrh, the age of the patient (young adult life), and the appearance of the jaundice unaccompanied by pain or general emaciation, together with an absence of symptoms pointing to *cirrhosis*, *carcinoma*, or *acute yellow atrophy*, form a characteristic grouping of clinical indications.

Duration and Prognosis.—The *duration* of catarrhal jaundice varies from two to eight weeks. If the symptoms continue longer than two months, grave doubts may be entertained as to the case being one of simple jaundice. The prognosis is guardedly favorable. A rise of temperature usually indicates mischief (Pepper), while hemorrhages of the skin and mucous membranes always influence the issue unfavorably.

Treatment.—The *diet* and *hygiene* are the first considerations in the treatment. Rich, highly seasoned foods, rich pastries, fats, and sweets, are to be interdicted; starchy foods, lean meats, bread, soups (containing no fat), and green vegetables may, however, be used in moderation. Skimmed milk, butter-milk, and alkaline drinks (Vichy and Saratoga mineral waters) may be used freely, while sour wines, lemonades, and tamarind-water are allowable. Systematic bathing (Turkish or Russian baths, under supervision) and regulated hours of sleep exert a beneficial effect. The free use of pure water often does good by increasing the flow of bile and by dislodging plugs of mucus.

Gerhardt and Kraus have recommended the faradic current, applied over the region of the gall-bladder; manipulation has also been tried with a view to removing the obstruction, but without success.

The first *therapeutic indication* is to keep the bowels freely soluble by the use of saline aperients, as Hunyadi water or Carlsbad salts ($\frac{1}{2}$ to 1 teaspoonful in hot water before meals). The latter remedies tend to lessen the catarrhal inflammation by depleting the mucous membranes. In obstinate constipation calomel or rhubarb may be employed.

Conspicuous among other remedies may be mentioned the alkalies, sodium bicarbonate, salicylate, and phosphate, which tend to increase the flow of bile and render it less thick; hydrochloric acid (which, according to Ewald, by aiding digestion prevents the formation and consequent absorption of toxic substances), in combination with the bitter tonics—gentian, quassia, and nux vomica; ammonium chlorid, which sometimes proves beneficial; and silver nitrate (gr. $\frac{1}{8}$–$\frac{1}{4}$—0.008–0.016, thrice daily).

Injections of cold water (60°–70° F.—15.5°–21.1° C.), daily, in quantities of 1 or 2 quarts (1–2 liters), are highly recommended as promoting the secretion of bile; while lavage, practised daily and over a protracted period of time (one to two months), has proved highly beneficial, especially when gastro-duodenal catarrh has existed.

Itching.—This troublesome symptom may often be relieved by the external application of a solution of borax or sodium bicarbonate (ʒss–Oj—16.0–512.0), or of menthol and alcohol (gr. x–ʒj—0.648–32.0). Internally, large doses of the bromids or the continued use of pilocarpin, as recommended by Witkowski, are worthy of a trial.

Flatulence.—To this end it is important to regulate the diet, avoiding starches and sugars. Charcoal tablets, bismuth subnitrate or salicylate, and betanaphtol are all useful. Irrigation of the colon with some efficient antiseptic in solution is often a factor of service.

Headache is caused by the circulation in the blood of some toxic

principle. Of drugs, caffein citrate, camphor monobromate, and phenacetin, either singly or in combination, may be recommended.

When the obstruction is due to mechanical causes (biliary calculi, tumors pressing on the duct) the treatment is surgical.

OTHER FORMS OF JAUNDICE.

Hemohepatogenous Jaundice (*Hemolytic, Toxic*).—The labors of Stadelmann, Hunter, Naunyn, and others have definitely shown that all jaundice is hepatogenous, since bile-pigments are formed only in the liver. To the cases, however, in which destruction of erythrocytes due to toxemia furnishes the hemoglobin from which the bile-pigments are formed in excess, the term hemohepatogenous jaundice has been given. This toxic or hemolytic jaundice occurs in many infections, both acute and chronic (typhoid fever, yellow fever, septicopyemia, malaria, pneumonia, so-called infective jaundice), and in pernicious anemia and chlorosis. In the same category belong the intoxications—*e. g.*, snake venom, cold-tar products, chloroform, poisoning with ptomains, mushrooms, phosphorus, and the like.

Chronic Family Jaundice.—There is a chronic jaundice probably of hemolytic origin, which appears in several members of a family, frequently in two, three, or four generations.[1] It dates either from birth, or is first noticed during adolescence, and persists throughout life. "Bilious attacks," enlargement of the spleen, and a moderate grade of anemia are the chief features. The stools are not colorless and contain no bile, but urobilin. The prognosis as to life is good, but treatment is without avail.

Experiments conducted by Münzer, Starling, Hopkins, and others tend to show that the liver-function is not suppressed by many of the conditions and affections mentioned above; but, on the other hand, that increased secretion (*polycholia*) and increased formation of bile-pigments (*polychromia*) may prevail. Again, the poisons or toxins may cause swelling of the cells and compression of the biliary capillaries; this would cause obstructive jaundice.

ACUTE INFECTIOUS CHOLECYSTITIS.

Definition.—An acute inflammation (infective) of the gall-bladder.

Pathology.—Five pathologic varieties — catarrhal, suppurative, phlegmonous, gangrenous, and membranous—are recognized. The gall-bladder progressively enlarges and becomes filled with mucopurulent or purulent or (rarely) hemorrhagic material. The cystic duct is often occluded. In the supparative form ulcers may coexist and perforation, followed by localized peritonitic abscess or acute diffuse peritonitis, may occur. The lesions of cholangitis, either catarrhal or suppurative, and also cholelithiasis, may be associated. Cholecystitis may exist without gall-stones.

Etiology.—The *bacterial* excitants include the streptococci, staphylococci, the pnemococcus, the colon bacillus, and the typhoid bacillus.

Among predisposing conditions are many of the acute infections, as typhoid, typhus, malaria, sepsis, pneumonia, puerperal fever, and cholera.

[1] *Vide Gaz. d. Hop.*, May 14, 1910, Chalier; *Amer. Jour. Med. Sci.*, June, 1910, Tileston and Griffin.

DaCosta has collected 58 cases of typhoid cholecystitis. His figures show that it may occur at almost any *age*, and of 48 cases in which the sex was stated, 26 were males and 22 females.

Symptoms.—The onset is abrupt, with pain (often paroxysmal) in right side of the abdomen or epigastrium. The region of the gall-bladder is acutely sensitive, and with the development of spreading peritonitis the tender area grows correspondingly. Rigidity of the right rectis may be observed. In many cases a *tumor* occupies the seat of the gall-bladder. It is detected on palpation as a firm, pear-shaped tumor or as a "mere resisting mass below the costal margin." The latter is often due to peritonitic abscess following perforation.

Nausea and *vomiting*, which may be persistent, are usual symptoms at the outset. *Jaundice* occurred in 17 out of 58 cases (DaCosta). Among the *general symptoms* chills are conspicuously absent. *Fever* may be present, but by no means always; the pulse becomes rapid and feeble, the abdomen distended, and prostration profound. Bayard Holmes noted cardiac disturbance as the most characteristic symptom in 46 cases. In the suppurative form a blood examination generally shows leukocytosis. The writer[1] has reported three cases of cholecystitis complicating lobar pneumonia. Jaundice occurred in two of the cases. This serious affection may be entirely latent.

Differential Diagnosis.—*Appendicitis* may be mistaken for cholecystitis, particularly if the appendix be situated abnormally high up. The discrimination would here rest upon the history (following typhoid or other infection), the presence of a *tumor* and marked sensitiveness in the region of the gall-bladder, corroborated by jaundice.

Acute intestinal obstruction may be closely simulated in cases in which adhesions between the gut and gall-bladder are present. In such cases exploratory celiotomy is to be advised or at least considered with a view to clearing the diagnosis. *Recurrent cholecystitis*, a not uncommon complaint, gives the history of recurring attacks of pain simulating cholelithiasis. In one of my cases Laplace operated and found the gall-bladder somewhat enlarged and the seat of catarrhal cholecystitis. Osler suggests that in some of these cases gall-stones may have been present and have passed before the operation (see also p. 898).

Prognosis.—This is dependent upon the special variety, although it is among the most fatal of diseases. A fatal result is the rule in purulent and phlegmonous cholecystitis. In the catarrhal form recovery is not infrequent (DaCosta). Pneumococcal cholecystitis is more acute and severe than that due to colon or typhoidal infection (Richardson). Gangrenous cholecystitis is rare and quite fatal.

Treatment.—This embraces absolute rest, rectal alimentation, the relief of pain by the judicious use of morphin, and of other symptoms as they arise. Stimulants are necessary as a rule. If the diagnosis of suppurative or phlegmonous cholecystitis can be established, surgical intervention is imperatively demanded as a rule.

Chronic Cholecystitis.—By this term is meant chronic inflammation of the gall-bladder, either secondary to an acute cholecystitis or a chronic low-grade infection from the beginning. It cannot be differentiated clinically from cholelithiasis, with which it is associated in the majority of cases.

[1] *American Medicine*, vol. ix., No. 11, March 18, 1905.

CALCULOUS CHOLECYSTITIS.

(*Biliary Calculi; Gall-stones; Cholelithiasis.*)

Definition.—Concretions formed in the gall-bladder, usually due to infection of its walls; they set up characteristic disturbances (*cholelithiasis*).

Etiology.—Catarrhal inflammation of the gall-bladder excites pathologic production of cholesterin by perverting metabolism inside the mucus-secreting cells in its walls. Schürmayer believes that cholelithiasis is really an expression of a metabolic disease of the liver. Infective catarrh of the small intrahepatic ducts leads to an albuminous exudation, which precipitates bilirubin-calcium calculi in the bile. Scheel found gall-stones in 15 per cent. of 2753 cadavers. In 67 per cent. of those showing gall-stones the bile passages were apparently not pathologic. The *exciting* cause is an infective inflammation of the gall-bladder and bile-ducts due to various organisms—*e. g.*, the *colon bacillus* and the *typhoid bacillus*. Among **predisposing causes** are: (*a*) *Female sex*, especially between the ages of forty and sixty. Senac's statistics, out of a total of 311 individuals, give 227 women (Dujardin-Beaumetz). (*b*) *Stagnation of bile*, due to an *excessive diet of starches and of fats*, a sedentary life, *constipation*, *tight lacing*, *pregnancy*, *obesity*, *chronic obstruction* to the outflow of bile (tumors, visceroptosis). Kehr claims that stones can develop in sterile bile, when the flow is obstructed. (*c*) It may occur during childhood. (*d*) Disorder about the pancreas may be the cause of gall-stone formation (Croftan). (*e*) Insanity, particularly chronic melancholia. (*f*) Incidence. Brockbank found among 13,047 completed postmortem records 7.4 per cent. were gall-stones.

Composition and Appearance.—Gall-bladder calculi are formed principally from cholesterin mixed with some bilirubin-calcium from the earliest stage. Certain salts (lime, potash, soda, traces of iron and copper) also enter into the composition. Those formed in the hepatic ducts are composed of bilirubin-calcium alone. In *size* they vary from the smallest particle of sand to that of a goose-egg. Fagge reports a calculus weighing, in a dry state, 462 grains (30.0). The *color* varies from white or light-yellow to that of a dark-green (pigment-lime calculi), and may present any variation between these two extremes. The *nucleus* often consists of cholesterin, the outer layer being usually the harder, and made up, for the most part, of lime-salts. The cholesterin gall-stones cut like wax, are white, and the cut section presents a crystalline appearance. Other forms are apt to be brittle. The *surfaces* may be smooth, striated, or hollowed out, solitary calculi being usually round or ovoid, while multiple stones often present smooth facets, due to the massing together of the calculi (Dujardin-Beaumetz). They are usually olive-shaped, but may be pyramidal, cylindric, lenticular, pisiform, cubic, finger-shaped, or olivary. Their *seat* is usually the gall-bladder, but they may be found anywhere along the biliary passages.

Symptoms.—The passage of a calculus through the duct, if it sets up a "perialienitis" or inflammation of the structures surrounding it (*cholecystitis*), will give rise to *hepatic colic*, whereas a permanent blocking of the duct will cause symptoms of chronic obstruction (*vide infra*).

Hepatic Colic.—When a gall-stone becomes impacted in a bile-duct

the patient experiences *agonizing pain* (tearing, cutting, or lancinating in character) in the right hypochondriac region, radiating to the right shoulder, and accompanied often by profuse *sweating*, *vomiting*, and a *feeble, running pulse*. The most common *seat* of the pain is two to three inches to the right of the median line and about an equal distance below the ensiform cartilage. Less frequently it is in the region of the gall-bladder. This happens in cases in which the gall-stone is impacted in the cystic duct, and may be due either to distention of the gall-bladder, or, more commonly, to associated cholecystitis. Hepatic colic, however, may occur independently of the passage of biliary calculi, as from non-calculous cholecystitis (Stockton, Riedel). If pain is severe without relation to meal time, you should suspect cholelithiasis. On the other hand, large calculi have been found in the dejecta without having excited hepatic colic. I saw an instance in which the gall-stone was the size of an English walnut. A *rigor* or *chill* often precedes the attack, which is usually accompanied by moderate *fever* (Charcot's intermittent fever), the temperature reaching 101°–102° F. (38.3°–38.8°). If the stone passes through the common duct without becoming impacted, jaundice and pain may be absent. When, however, occlusion of the common duct occurs, the *jaundice* becomes intense. This symptom may be present, though less marked, before the gall-stones reach the *ductus communis*. Jaundice occurs in about 50 per cent. of the cases (Fitz), and it sets in within forty-eight hours after the onset of the attack. *Physical examination* reveals on inspection a slight prominence in the hepatic area, and on palpation the edge of the liver can often be distinctly felt below the costal margin—at times as low as the umbilical level; it is sensitive on pressure, and particularly the gall-bladder, which can be often palpated. If the latter viscus contains many calculi, and the abdominal wall is relaxed, crepitation may be noticeable to the palpating fingers (rarely). Tenderness in Boas' area to the right of the spine between the tenth and twelfth rib is a valuable confirmatory sign. The swollen organ, after the cessation of the colic, quickly subsides. Tenderness over Mayo Robson's point at the junction of the lower third with the upper two-thirds of a line drawn from the tip of the ninth rib to the umbilicus is a characteristic feature. *Recurrences* of the attacks after varying intervals of time are common, and in the female, especially at the menstrual period. Finally, the gall-stone may be expelled and the colic cease to return. Multiple stones, however, may be passed. Hyperchlorhydria is commonly present.

Rupture of the duct, followed by fatal peritonitis, has been known to occur. Localized peritonitis results from extension of inflammation through the walls of the gall-bladder. Biliary colic is of variable *duration*, lasting from a few hours to a few days or one or more weeks even. Examination of the *urine* after the paroxysm reveals bile, uric acid, and urates. The *pulse* often becomes slowed. Exner found about 0.4 per cent. of sugar in the urine in 39 out of 40 cases of gall-stones. On the other hand, Kausch has found glycosuria in only one of 85 cases.

The **prognosis** as regards life is good, but as regards recovery only guardedly favorable. Cardiac distress with palpitation may occur during hepatic colic and form a serious complication. Fatal syncope has also been known to occur, and gall-stone ileus, especially near to the ileo-cecal valve, may terminate life. If evidences of an infectious inflam-

mation arise, the outlook is then more serious. The sequelæ will be discussed hereafter (*vide infra*).

Diagnosis.—The diagnosis of gall-stones is sometimes difficult on account of the obscure clinical symptoms and the absence of physical signs. When, however, the calculus becomes impacted in the duct, symptoms of biliary colic—intense pain in the epigastrium and right hypochondriac region, radiating to the back and right shoulder—usually appear. The attack is of brief duration, with abrupt cessation. There are also fever, vomiting, and in one-half the instances jaundice. The urine should be examined early, since bile may be present many hours before icterus occurs. Biliary calculi are not often found in the dejecta.

Differential Diagnosis.—*Gastralgia* occurs in neurotic individuals, and is characterized by severe paroxysmal pains in the epigastrium, extending to the back and base of the chest. It occurs when the stomach is empty and is relieved by eating. Firm pressure over the epigastrium often alleviates the pain temporarily, and the absence of fever, jaundice, stones in the dejecta, and the negative urinalysis, together with the history of former attacks, would tend to differentiate it from hepatic colic.

Renal Colic.—The pain in this condition starts in the flank of the affected side and is transmitted down the ureter, and there is localized tenderness. The testicle and inner side of the thigh are very painful, the former being often retracted. Micturition is frequent and sometimes painful, and the urine is scanty in amount and often mixed with blood.

Intestinal Colic.—In this variety the pain is of a boring or twisting character, usually centering about the umbilicus. It is relieved by firm pressure. Abdominal distention is often present, and relief comes with the passing of flatus. Usually there is a history of an indiscretion in diet. When due to *lead-poisoning*, the history, the blue line on the gums, and the presence of wrist-drop would tend to confirm the diagnosis.

Reflex colic, due to uterine or ovarian disease, may also occur. The recurrence of the attacks, together with causes and symptoms pointing to pelvic disease, would establish the identity of the condition.

CHRONIC OBSTRUCTION OF THE DUCTS BY GALL-STONES.

The obstruction may exist either in the common or the cystic duct.

1. **Obstruction of the Common Duct.**—**Pathology.**—The result of the irritation produced by the presence of the stone is a catarrhal process (*cholangitis*) that may either remain chronic or terminate in suppuration (*suppurative cholangitis*). In a case of simple obstruction the gall-bladder is often moderately enlarged, though rarely extending below the lower border of the liver. The common duct is greatly distended, the stone being usually located near its termination. Occasionally two or more calculi are present, completely obliterating the canal. The hepatic duct and its branches are greatly dilated, and often contain thin, colorless mucus, the lining membrane being smooth and clear. The liver in these cases is firmer in consistency than normal, showing some increase in the connective-tissue element (biliary cirrhosis). Following moderate enlargement of the organ progressive atrophy may rarely occur. When *suppuration* has occurred the mucous membrane is greatly swollen and reddened, and in some instances shows erosion or ulceration (Ulcerative Angio-

cholitis). The process often ascends the hepatic ducts into the liver, with infection of this organ, and in the severer cases abscess-formation. On the other hand, it may extend to the gall-bladder, giving rise to empyema of the latter. In some instances the gall-bladder has been perforated and abscesses have formed between the liver and stomach. *Diverticula* are sometimes found postmortem, containing biliary calculi.

While cholelithiasis is a common cause of catarrhal, suppurative, and ulcerative angiocholitis, it not rarely complicates hydatid disease, carcinoma of the bile-ducts, and the acute infections, particularly typhoid fever (*vide* Acute Infectious Cholecystitis, p. 896). Rarely foreign bodies (fish-bones, lumbricoids) operate as excitants.

Symptoms.—Chronic obstruction by gall-stones, with coexisting *catarrhal inflammation* (catarrhal angiocholitis), is characterized by a distinctive group of symptoms, among the most prominent of which are—

Jaundice.—This may be constant and very intense, or intermittent and slight, depending upon the amount of obstruction present. There are periodic elevations of temperature accompanied by a deepening of the jaundice, when this symptom already exists (ball-valve action of the stone). *Itching* is, as a rule, a most distressing feature. A stone low down produces obstruction also of the pancreatic ducts, in which case the stools will contain a great amount of fat and undigested muscle-fibers.

Pain, occurring in paroxysms and referred to the region of the liver. This is accompanied by fever that may reach a high degree (102°–103° F.—38.8°–39.4° C.), also by chills and sweating, resembling the paroxysms of malaria. Painful points in the right side posteriorly may be annoying; these are either constant or paroxysmal.

The *chills* are often intense, and may present a quotidian, tertian, or quartan form. The temperature of the intervals is normal. The peculiar exacerbations of temperature were first described by Charcot, and to them has been given the name of *Charcot's intermittent fever*. Concerning their nature Murchison writes: "These paroxysms may be more or less periodic, and may extend over several months, without necessarily indicating pyemic hepatitis, the patient ultimately recovering." He adds that they are probably analogous to febrile paroxysms produced in passing a catheter along the urethra. Charcot believes the etiologic factor to be a septic poison, bacterial in origin and the result of chemical changes in the bile. Various microörganisms have been detected in the bile in such cases (bacterium coli commune, streptococcus pyogenes, *et al*).

Gastric Disturbances.—These may excite alarm during the paroxysm. Intense pain is complained of in the epigastrium, accompanied often by nausea and vomiting, which, however, usually subsides at the close of the paroxysm, while the jaundice at this time deepens. Lichty found disturbance of the gastric secretion in 75 per cent. of the cases, of which two-thirds showed hyperchlorhydria. Gastric motility was disturbed in about the same proportion of cases.

The symptoms of *suppurative cholangitis* are intense. The *paroxysms* of fever occur more frequently, the temperature merging into the remittent type. Grave constitutional symptoms, indicating septicopyemia, are present, and the case rapidly tends to a fatal issue. The attacks of colicky pain occur with jaundice, but the latter symptom is

less intense than in the catarrhal form. As to hepatic enlargement, the converse is true; this organ takes on progressive enlargement and "may descend as low as the umbilicus, the swelling being uniform and smooth and tender to pressure" (Robson). It should be borne in mind that pain may be absent when the disease is not dependent on gall-stones. Pneumonia and empyema are serious and not uncommon complications. In *ulcerative angiocholitis* severe hemorrhage may occur, resulting either in melena or hematemesis. Mayo Robson reports a case in which hematemesis was the only antemortem symptom and had been the cause of death. The process being a septic one, it leads to the constitutional disturbances of septicemia or septico-pyemia. *Pancreatitis* may be caused by stones in the common duct.

2. **Obstruction of the Cystic Duct.**—This almost invariably causes distention of the gall-bladder (dropsy of the gall-bladder). If obstruction of the cystic duct alone occurs, *jaundice* may be entirely absent, the bile in the distended tissues being replaced by a thin, mucoid fluid. This is more apt to exist as the obstruction becomes more chronic. In some instances the distention is so great as to reach below the umbilicus, and the dilated viscus has even been mistaken for an *ovarian tumor*. Osler records a case in which 18 oz. (556.0) of fluid were removed from the gall-bladder. The contents are neutral or alkaline in reaction, albumin being often present in abundance. Catarrhal inflammation of the gall-bladder may be associated, causing *pain*, at times being so severe as to simulate hepatic colic, and *sensitiveness* in the region of the organ, although, as a rule, few symptoms are presented. The examiner can *feel* an elastic, gourd-shaped tumor closely connected with the liver, movable in respiration in the vertical, and also, under the influence of the palpating fingers, in the lateral direction. Occasionally Riedel's tongue-like projection of the anterior margin of the right lobe is palpable. Given a gall-bladder well filled with stones and a relaxed abdominal wall, gall-stone crepitus may be detectable.

The writer has reported some cases giving a more or less characteristic clinical history of cholelithiasis, in which gall-stone crepitus on palpation furnished proof of stones in the gall-bladder. In one case he combined auscultation with palpation and detected a grating sound.[1]

If the obstruction persist for a length of time, calcification or atrophy of the bladder are common sequelæ. Complete obliteration of the cavity of the gall-bladder may ensue.

Among rarer sequelæ of chronic obstruction may be mentioned—(*a*) *Empyema of the Gall-bladder.*—When this takes place the organ becomes greatly distended, and has been known to contain as much as a pint of purulent material. The *symptoms* of suppurative cholecystitis simulate and accompany those of purulent cholangitis; they are sometimes preceded by those of catarrh of the gall-bladder and ducts. Perforation may occur, giving rise to circumscribed periportal abscesses or to generalized peritonitis (see also *Acute Infectious Cholecystitis*, p. 894).

More Remote Effects of Gall-stones.—These will be spoken of under three headings:

1. Stricture of the duct, resulting from ulceration and cicatrization produced by the passage of a stone.

2. Intestinal obstruction, due to impaction of stones or volvulus.

[1] *International Medical Magazine*, Dec., 1899.

3. Biliary fistulæ resulting from perforations.

1. **Stricture of the Duct.**—Obliteration of the common duct may result from the passage of a gall-stone, giving rise to ulceration and cicatrization, or the stone may become impacted and lead to adhesions and permanent closure of the duct below it (Murchison). When due to ulceration the seat of the stricture is usually low down in the common duct.[1]

Symptoms.—The symptoms are those of chronic obstructive jaundice (Osler). In many cases there will be an antecedent history of the passage of gall-stones. In all cases in which the symptoms of gall-stones are followed by permanent jaundice without pain it may be suspected either that the calculus has become firmly impacted or that it has produced organic stricture or closure of the duct.

2. **Intestinal Obstruction from Impaction of Gall-stones.**—The ileum is commonly the seat of obstruction by gall-stones, that may give rise to intussusception or cause ulceration and gangrene of the bowel with perforation and fatal peritonitis. The latter event, however, occurs more frequently when the biliary concretions are situated in the cecum. Rarely they are found in the appendix, and may produce appendicitis. Cases of impaction in the rectum of biliary calculi have been recorded. I have recently seen a case with Dr. R. Bruce Burns.

Symptoms.—If the impaction occurs in the small intestine, the abdomen becomes tympanitic and tender on pressure. The contents of the stomach are first vomited, followed by bile and stercoraceous matter. Symptoms of peritonitis develop and continue until either the impaction disappears or death ensues. Ileus, the result of biliary concretions, is common in females of advanced age, and adhesions about the gall-bladder region may obstruct the lumen of the bowel. The history of previous acute attacks would tend to confirm the diagnosis. The pain is intense. The duration of the last attack is often short.

3. *Perforation* may occur with the establishment of *fistulous communications* between the gall-bladder and stomach, intestinal canal, bladder, vagina, lungs, abdominal parietes, or portal vein. Fistulæ between the gall-bladder and stomach are rare, though cases are recorded by Oppolzer, Frerichs, Cruveilhier, Murchison, and others. Cruveilhier states that vomited gall-stones necessarily reach the stomach through fistulous tracts, as the passage through the pylorus would be impossible.

Fistulæ into the duodenum are of much more common occurrence, ulceration taking place usually in the fundus of the gall-bladder and in the descending or third portion of the duodenum: 39 cases are recorded of fistulous communication with the colon (Osler). I have reported a fortieth case.[2] In 6 of 9 cases reported by Murchison carcinoma of the gall-bladder was present. Fistulæ into the urinary passages may occur, 2 authenticated cases being reported. The distended gall-bladder may come in contact with the urinary viscus, or the stone may perforate into the pelvis of the kidney and pass through the ureter into the bladder.

Fistulous openings through the abdominal parietes are the most common, the place of exit of the biliary concretions being usually in the region of the gall-bladder or at the umbilicus, to which it may be directed

[1] In vol. ix. pp. 22 and 130, *Pathologic Transactions*, two cases are recorded in which the strictures were exactly similar to those of the urethra, one being situated in the hepatic duct of the left lobe and the other in the common duct.

[2] *Clinical Lecture, International Clinics*, vol. ii., third series, p. 27.

by the suspensory ligament of the liver. As many as 600 stones have been removed from the gall-bladder in this manner. Advanced life and female sex are said to be predisposing causes. Courvoisier's statistics show 184 cases, in 78 of which recovery took place.

Fistulæ into the pleura, bronchi, and vagina have been recorded, but are extremely rare. Courvoisier records 24 cases of fistulæ into the lungs, only 7 of which terminated in recovery. Fauconneau, Dufoesne, Frerichs, Bristowe, and Murchison mention cases of fistulæ into the portal vein, with the presence of biliary concretions in the latter.

Diagnosis.—I would strongly urge an exploratory celiotomy as an accurate means of diagnosis in obscure cases.

Treatment of Foregoing Conditions.—The indications for treatment in *cholelithiasis* are (*a*) to remove the cause; (*b*) to relieve the paroxysms of hepatic colic; and (*c*) to adopt palliative or radical measures for the removal of the stones.

Preventive Treatment.—This has reference to the removal or mitigation of the predisposition. The diet should be as simple as possible, consisting largely of skimmed-milk, lean meat, eggs, fruit, and green vegetables. Fatty foods, sugars, starches, and pastries are to be strongly interdicted. All foods should be thoroughly masticated, so as to digest easily, and meals should be taken at regular intervals. Systematic exercise in the open air is of signal value, as it stimulates the flow of bile. Punkhauer strongly recommends horseback-riding for obstructions in the common duct. Among the drugs mostly used in the treatment of predisposing conditions, as hepatic torpor and the like, I would advise the following: Sodium sulphate, combined with the extract of taraxacum (Harley); ox-gall (Dubney), in 5- to 10-gr. (0.324–0.648) doses, three times daily (to relieve flatulency and stimulate the biliary secretion), or sodium salicylate (gr. x to xv—0.648 to 0.972, three times daily). In my own experience a dram (4.0) of sodium phosphate dissolved in a glass of hot water in the morning on rising has yielded excellent results. The same dose may be repeated before the noon and evening meals, if required. The bowels should be kept freely soluble, constipation being carefully avoided.

To prevent recurrences a course of alkaline treatment at some of the more noted mineral springs (Bedford, Vichy, Carlsbad) is often attended with good results. The efficacy of the Carlsbad treatment lies in reducing inflammatory processes, and not in the expulsion nor solution of the gall-stones. "As the result of Carlsbad treatment, Fink, in 375 cases, had good results in 291 = 72.8 per cent., of which 20 cases, or 4.95 per cent., had relapse" (Forcheimer).

Treatment of the Paroxysm of Biliary Colic.—At the very onset of an attack of hepatic colic the prompt exhibition of morphin or of codein may greatly mitigate an attack. The former may be given hypodermically in $\frac{1}{8}$- to $\frac{1}{4}$-gr. (0.008–0.016) doses every hour until relief follows; the latter is exhibited by the mouth in doses of 1 gr. (0.0648) every hour. Inhalations of chloroform, with morphin hypodermically, the former being continued until the latter has taken effect, is the typical treatment of an attack. Gilman Thompson recommends chloroform (♏ xx—1.33) by mouth for the relief of pain.

Hot baths and hot applications (with counter-irritation) over the liver

are valuable aids in the treatment of hepatic colic, being given at a temperature of 98° to 100° F. (36.6° to 37.7° C.), and continued for twenty minutes, if endurable, so as to effect relaxation. If cardiac depression results and the pulse becomes weak, the baths should be discontinued. Hot flaxseed-poultices, cloths wrung out of hot water, hot hop-bags, or turpentine stupes may be applied over the hepatic region until the attack subsides. Ice-poultices have been advised by Buchetan.

If shock or syncope should develop, the body-temperature must be maintained by hot bottles or bricks placed in contact with the surface of the body, together with strychnin (gr. $\frac{1}{30}$—0.0021), atropin (gr. $\frac{1}{150}$—0.00042), and brandy (1 dram—4.0) hypodermically.

Nausea and vomiting may be reduced by 15-drop doses of spirits of chloroform every half hour; also by brandy or champagne.

In mild cases sodium salicylate (gr. viij–xv—0.518–0.972 in twenty-four hours), recommended by Prevost and Binet, or codein (gr. j), with phenacetin (gr. x), every few hours gives relief. The free use of olive oil or glycerin in hepatic colic has been followed by a beneficial effect (Rosenberg, Goodhart). The former is given in quantities of 4 to 6 oz. (128.0–192.0) by the mouth every three or four hours, nausea being prevented by concealing the taste with lemon-juice; the latter, recommended by Ferrand, is given in doses ranging from 1 to 2 tablespoonfuls, repeated in the same length of time. Purgation and remedies presumed to act as cholagogues, given during an acute seizure, are harmful in their effects. The aim should be to reduce the inflammatory process in the gall-bladder. D. D. Stewart well says: "The treatment of recent cases of stone in the common duct belongs to the physician but a short time only." If the gall-bladder is palpable after an attack of hepatic colic, the cystic duct is probably obstructed and the treatment is surgical.

Treatment for Removal of Gall-stones.—Solvents for the stones have been tried at various times, among them being *Durande's method* (turpentine and ether), but, so far, all such methods of treatment have been unsuccessful. The free use of pure water by the mouth, together with copious rectal injections of cold water daily is to be advised. It may be rendered alkaline by sodium bicarbonate or borate in a 3 per cent. solution.

Willoughby reports a case in which prompt recovery ensued from the use of toluylenediamine after three years of unsuccessful treatment; he began with 1 grain daily, and increased to 2 grains.

Of the various surgical measures for the removal of gall-stones the following are the chief: (*a*) Removal of the stone from the common duct (choledochotomy); (*b*) Removal of the stone from the cystic duct (cholecystotomy); (*c*) Establishing a fistulous opening between the gall-bladder and the bowel (cholecystenterostomy); (*d*) Extirpation of the gall-bladder (cholecystectomy), the latter operation giving a mortality of 17 per cent., according to Murphy's statistics. And operative procedure is indicated in infectious (suppurative) cholecystitis as well as in infectious (suppurative) cholangitis; *e. g.*, evacuation and drainage. W. Mayo has operated in 510 cases of cholelithiasis with a death-rate of only 3 per cent. Of 326 cases of gall-stone complicated with biliary infection and malignant disease, 16, or 5 per cent., proved fatal. Kehr has never had a recurrence in 900 operations for cholelithiasis; Toeplitz has had recurrence in 14.2 per cent.

CARCINOMA OF THE BILE-DUCTS.

Carcinoma of the gall-bladder and bile-ducts may occur as a primary disease and exist over a long period of time without being recognized.

Pathology.—The gall-bladder, as the result of obstruction of the duct, is often greatly distended, measuring as much as 7 inches (17.7 cm.) in length (in a case reported by Harley) from the entrance of the duct to the fundus, and being filled with a cloudy liquid, somewhat resembling barley-water, that contains flakes of epithelium, granular matter, and particles of inspissated bile. If the growth be near the duodenal orifice, the common and cystic ducts are often greatly distended, and the dilatation may extend into the hepatic ducts and their branches. The liver may be enlarged, and in more than one-half of the instances presents the secondary nodules that are characteristic of the disease. Microscopically, carcinoma of the gall-bladder exhibits marked variations in different cases; "it may be either columnar or spheroidal celled" (Rolleston).

Etiology.—The causes of carcinoma of the bile-ducts are the same here as elsewhere, and among these the mechanical or inflammatory theory of Virchow must be accepted. Tight-lacing and mechanical irritation by gall-stones are followed in many instances by cancerous degeneration; Osler states that "biliary calculi are present in at least seven-eighths of all cases." Among other factors, heredity and age (after forty) play an important part. Although carcinoma of the *liver* undoubtedly occurs more frequently in males, Musser found that out of 100 cases of carcinoma of the *ducts*, 75 occurred in the female; and Ames found the ratio to be 4 to 1 in favor of females.

Symptoms.—The signs and symptoms, according to Harley, present nothing characteristic to distinguish them from other causes of obstruction in the ducts. On *palpation* in the early stages the gall-bladder is found moderately enlarged, but later it rapidly undergoes diminution in size. *Jaundice* becomes very intense, and remains permanent. Throughout the course of the disease all the symptoms referable to chronic obstruction of the duct by gall-stones (paroxysmal pain, gastric disturbance, rise of temperature, Charcot's fever) may develop.

Examination of the urine and feces reveals the presence of *bile-pigment* in the former and its absence in the latter. The urine often shows the presence of bile-stained casts (*vide* Fig. 64).

Ascites not rarely occurs during the later stages, with the involvement of surrounding organs by contiguity, as well as with the appearance of secondary nodules in the liver and the development of cachexia.

Diagnosis.—Carcinoma of the biliary ducts cannot always be detected by physical examination. Distinct evidence of chronic obstruction of the duct, as persistent and intense jaundice (which occurs in three-fourths of the cases), the development of cachexia and the absence of cancerous involvement of other organs, however, will tend to characterize it. Often a hard tumor-mass is present in the region of the gall-bladder, projecting in the direction of the umbilicus. It should be recollected that the bile-ducts are oftener the seat of the primary affection than the liver. Cholelithiasis may simulate carcinoma in all its symptoms, except those due to metastasis.

Prognosis.—The prognosis of carcinoma of the bile-ducts is, like that of other organs, absolutely fatal, though the course of the disease is not so rapid as that of carcinoma elsewhere until secondary involvement of the liver occurs.

Treatment.—The treatment is merely palliative. Operative measures are rarely justifiable, since the disease is rarely recognized before the liver becomes involved. As seven-eighths of the cases follow obstruction of the duct by gall-stones, the preventive treatment of the latter should be carefully observed whenever symptoms of disordered liver-function manifest themselves.

The treatment of the pain, anemia, and emaciation will be described in the discussion of Carcinoma of the Liver.

STENOSIS OF THE BILE-DUCTS.

Stenosis may result from any of the following causes: (*a*) *Round-worms* in the duct (rarely); (*b*) Foreign bodies, as seeds; (*c*) Ulceration and cicatrization following the passage of gall-stones (most commonly); (*d*) Pressure from without, as from tumors (carcinoma chiefly) of the head of the pancreas and pylorus (rare); (*e*) Abdominal tumors; (*f*) Aneurysm of the abdominal aorta or of the celiac axis (rare); (*g*) Secondary enlargement of the lymphatics of the liver (common); (*h*) More rarely in man than in the lower animals distoma hepaticum of liver-flukes and echinococci; (*i*) Adhesions due to chronic peritonitis.

Pathology.—If the stenosis is of recent origin, the liver is enlarged and shows more or less congestion, with some increase of the connective-tissue elements. The substance is firmer than normal, the color varying from an olive-green to a deep bronze. If, however, the obstruction be of long standing, the presence of the dilated intra-hepatic ducts and the increase of connective tissue cause secondary atrophy of the hepatic cells, with a diminution in the size of the organ.

Symptoms.—The symptoms vary greatly according to the cause of the stenosis, but in the main they are those of chronic obstruction of the duct—viz. paroxysmal pain in the region of the liver, referred to the right shoulder; jaundice of varying intensity, but gradually deepening after each attack; and gastric disturbance, with ague-like paroxysms (fever and sweating), the latter being most frequently met with in occlusion from gall-stones.

Diagnosis.—The pathognomonic symptoms determining the nature of the stenosis are very often wanting, and the diagnosis is rendered correspondingly difficult. On the other hand, stenosis or complete occlusion of the bile-passages calls for diagnosis principally on account of the special cause or causes of the given case.

When the condition is due to *lumbricoid* worms reflex symptoms usually appear, as pruritus of the nose and anus, grinding of the teeth during sleep, and convulsions.

In *carcinoma of the head of the pancreas or the pylorus* pressing on the ducts the growth may be detected by palpation, together with a rec-

ognition of other more or less characteristic features (*vide* Carcinoma of Pancreas), and the rapid course of the disease.

Abdominal aneurysm may give rise to obstruction of the duct without being evidenced by physical signs. Usually, however, when the sacculation presses against the bile-duct, the throbbing in the epigastrium, the tumor (which can often be grasped), and the expansile pulsation on palpation will tend to establish the cause of the obstruction.

When due to *cancerous nodules in the liver* there is usually a history of primary carcinoma of the stomach, mammary gland, rectum, or of one of the pelvic viscera. Osler records a case in which jaundice (thought to have been catarrhal in origin) developed seven weeks previously. On careful examination "a small nodule was detected at the umbilicus, which on removal proved to be scirrhus."

When the stenosis is due to *ulceration* following the passage of gall-stones, the history of biliary colic, and of paroxysmal pain with jaundice and intermittent fever, will serve to establish the cause.

If the fever be of the continued type and the liver uniformly enlarged, with the development of jaundice, the case is probably one of *hypertrophic cirrhosis;* whereas if the enlargement be progressive and nodules can be detected on palpation in addition to the appearance of cachexia and jaundice, *carcinoma* is undoubtedly present.

Physical signs aid but little in the diagnosis, as obstruction of the common duct is usually unattended by any great enlargement of the gall-bladder.

In many cases only by remembering the various causes and eliminating them carefully, one by one, can a diagnosis be rendered.

Prognosis.—The prognosis varies according to the cause of the stenosis. Generally speaking, the outlook is rather grave, since many of the causative conditions are fatal. If the obstruction is due to cicatricial contraction, the prognosis is guardedly favorable as to life, but hopeless as to recovery. If the obstruction is permanent, the case ends fatally.

Treatment.—The treatment of occlusion of the bile-ducts varies according as it is due to cicatricial contraction following ulceration or to foreign bodies (seeds or lumbricoid worms), or to gall-stones or tumors pressing upon or involving the ducts or adjacent organs (pancreas, pylorus). If the stenosis follows ulceration in the duct, and is sufficient to cause almost complete occlusion with biliary retention, the operation of cholecystenterostomy may become necessary in order to prevent dilatation of the gall-bladder with resorption of bile.

Foreign bodies in the duct may be removed by free purging, aided by the liberal use of alkaline mineral waters. In critical cases the operation of cholecystotomy is recommended.

Gall-stones form the most frequent cause of stenosis, and the treatment, both for the prevention and removal of calculi, has already been described in the discussion of Biliary Calculi (*vide* p. 904).

ICTERUS NEONATORUM.

Definition.—By the term icterus neonatorum is meant jaundice occurring in the new-born. It is seen in about two-thirds of all new-born infants, is unaccompanied by any other lesions, and pursues a favorable course. Icterus neonatorum must not be confounded with jaundice occurring in the new-born and dependent upon various pathologic causes—*e. g.*, congenital stricture or absence of the duct, syphilitic disease of the liver, duodenal catarrh, and septicemia, as a result of infection through the umbilical vein. In this form the skin and conjunctivæ are more or less icteroid, the urine is loaded with bile-pigment, while the feces are of a pipe-clay variety. Hence it differs in its symptomatology from true icterus neonatorum.

The secretion of bile, like the secretion of urine, begins long before birth, and Zweifel has found bile-pigment and bile-acids in the contents of the intestines of a three-months' fetus. Hence children may be born laboring under an attack of well-marked jaundice.

Etiology.—The following are the main causes: 1. The ductus venosus may remain patulous, allowing some of the portal blood, containing bile, to flow into the systemic circulation (Quincke). 2. Diminished pressure in the portal vessels from ligation of the umbilical vein causes increased tension in the hepatic capillaries and absorption of bile. 3. It is probable that the external conditions are in some way concerned in the appearance of the disease (Oser). 4. The destruction of numerous red corpuscles may be followed by an increased amount of bile-pigment in the liver.

Symptoms.—The skin is tinted greenish-yellow, resembling somewhat that of chlorosis. The mucous membranes are pale and the conjunctivæ pearly-white, except in the severer cases, when they show a slight discoloration. The icterus usually appears on the second or third day of life. The pulse is feeble and sometimes rapid. Auscultation over the base of the heart often reveals a soft systolic murmur, associated with a venous hum in the neck. According to Murchison, icterus neonatorum differs from the pathologic form in that—1. The conjunctivæ are of a natural color; 2. The urine is free from bile-pigment; 3. The yellow color gradually fades from the skin after a few days; 4. The child is quite well and the bowels are acting properly.

Prognosis.—The jaundice gradually disappears spontaneously at the end of three or four days.

Treatment.—As a rule, nothing beyond hygienic measures are required. The diet need not be restricted.

VASCULAR (CIRCULATORY) AFFECTIONS OF THE LIVER.

ANEMIA.

The physical symptoms of this condition are absolutely *nil*, and its existence only discoverable *postmortem*. Its most common causes are those of general anemia, fatty and amyloid degeneration.

HYPEREMIA.

Definition.—An excess of blood in the liver. This may be of two varieties: (*a*) *active* and (*b*) *passive*, the latter being the more common.

ACUTE HYPEREMIA.

(*Active Congestion.*)

Definition.—An excess of arterial blood in the liver.

Etiology.—Among the common causes are luxurious living, sedentary habits, alcoholism, traumatism, acute infectious diseases (typhus, typhoid), and pernicious malaria. The condition may also be vicarious, due to a sudden cessation of menstruation or of hemorrhage in other parts of the body. A *physiologic* condition is the temporary hyperemia that occurs during the ingestion of a full meal.

Symptoms.—There are no symptoms characteristic of this condition; those present in the different cases are varied and referable to disturbances of other viscera, as in coexisting cardiac hypertrophy or gastro-intestinal catarrh. There is a sense of *fulness* and *distress* in the right hypochondrium, most marked during the height of the digestive process, with *tenderness* on palpation over the margin of the organ.

Prognosis and Course.—It is impossible to make any definite statement as to the course and prognosis of active hyperemia, these depending wholly upon the cause of the affection. When due to errors of diet and hygiene the condition is easily remedied; the prognosis of hyperemia accompanying hepatic cirrhosis, however, is decidedly grave.

PASSIVE HYPEREMIA.

(*Passive Congestion.*)

Definition.—An increase of venous blood in the liver.

Pathology.—The organ is enlarged and changed to a deep-red color, while its substance is firmer than the normal. The center of the lobule (the area of the hepatic vein) becomes deeply pigmented, the periphery (occupied by the portal vein) being lighter in color, sometimes owing to fatty infiltration. Because of its mottled appearance this has received the name of the "nutmeg liver."

In long-standing passive congestion there is an increase of connective tissue, due to a proliferation of round-cells, causing atrophy of the parenchyma. The blood in the central capillaries becomes altered, the capillaries themselves are distended, and brown pigment is deposited about the center of the lobules. The organ becomes very much darker in color, and to this condition the name "cyanotic induration" or "cardiac liver" has been given. Later, contraction of the connective tissue occurs, causing a diminution in the size of the organ, and forming the so-called "atrophic nutmeg liver."

Etiology.—The causes that lead to passive hyperemia are both *local* and *general*. Among *local* causes may be mentioned the following:

1. Pressure over the portal area from without, as from a tumor or cyst.
2. Disease of the walls of the veins, as in syphilitic phlebitis.
3. Coagulation of the blood in the veins (thrombosis).

Among the *general* causes are—

1. Chronic valvular disease affecting the right side. Passive hyperemia also occurs in mitral disease.

2. Pulmonary emphysema and cirrhosis of the lung.

3. Intrathoracic tumors, which by their mechanical action cause an increased pressure in the efferent branches of the hepatic veins.

Symptoms.—Often the patient experiences a sensation of *fulness* and *weight* in the region of the liver that amounts in some instances to actual *pain*. *Jaundice* is usually present, but varies in intensity, and is due to obstruction of the smaller ducts from distention of the hepatic venules. *Hematemesis* and also *hemorrhoids* (bleeding) may occur, and symptoms of gastro-intestinal disturbance are usually present. In marked cases the *stools* are *clay colored*, showing the absence of bile; the *urine* is loaded with bile-pigment; and jaundice deepens with the development of *ascites* or *anasarca* from portal obstruction. On *palpation* the organ is tender and increased in size, extending in some instances fully a hand's breadth below the costal margin. In pronounced cases the whole organ pulsates, owing to the regurgitation of blood into the hepatic veins (see also p. 659).

Diagnosis.—The diagnosis of passive congestion, *per se*, is often very difficult, but when secondary to heart and lung diseases it is rendered more plain.

The **prognosis** and **treatment** depend upon the causal factors.

DISEASES OF THE PORTAL VEIN.

THROMBOSIS AND EMBOLISM.

Pathology.—In the early stages the clot presents a grayish-red or yellowish appearance, and on loosening it is found to adhere more or less closely to the inner coat of the vein. Later it becomes a mass of small white fibrin tightly adherent to the sides of the blood-vessel, which itself undergoes fibroid change (adhesive pylephlebitis). Organized thrombi are rarely found, except in the smaller branches of the portal area. If the thrombus obstruct the vessel, collateral circulation may be established for years. Septic softening, however, is a very common result, and pylephlebitis even more so. If a parietal or channelled thrombus be formed, partial or complete circulation may be re-established and recovery take place. Hemorrhagic infarction may occur, but it is rare.

Etiology.—*Thrombi* are rare occurrences in the portal vein. Among the *causes* that lead to their formation, however, may be mentioned—(*a*) Traumatism; (*b*) cirrhosis; (*c*) carcinoma of the liver, stomach, and pancreas; (*d*) pressure from without, as in proliferative peritonitis involving the gastro-hepatic omentum, abscesses, enlarged glands, or impacted calculi pressing on the veins; (*e*) it may be occasioned by ulcerative affections of the bowels and appendicitis, and pylephlebitis may precede its occurrence; (*f*) slowing of the circulation due to splenic diseases, such as marasmus.

Symptoms.—Symptoms may be almost lacking in portal obstruction, or the condition may simulate cirrhosis of the liver. In ordinary cases the symptoms are very slight, the hepatic circulation, as shown by Cohn-

heim and Litton, being "sufficient for the nourishment of the liver and secretion of the bile" (Henry).

If the occlusion be *complete*, *edema* followed by the rapid development of *ascites* may occur. In such cases loss of strength is persistent and progressive, and death may result from exhaustion. *Hemorrhages* due to venous stasis may occur from the nose, stomach, and intestines. *Jaundice* and *diarrhea* occur frequently, the former being the result of obstruction to the biliary passages from the same causes that produce the thrombosis or of the diminished pressure in the portal area. On *palpation* the liver is found slightly enlarged and tender on pressure, and projecting below the lower margin of the ribs; the *spleen* is also enlarged. *Percussion* also reveals enlargement of the splenic area. If ascites is present, percussion will reveal dulness in the flanks, changing with the position of the patient; and on gently tapping one side of the belly-wall, with the hand on the opposite side, a wave of fluctuation will be felt.

Diagnosis.—The diagnosis of portal thrombosis is often extremely difficult. "A suggestive symptom, however, is sudden onset of the most intense engorgement of the branches of the portal system" (Osler).

Sequelæ.—If the emboli are septic in origin, an abscess, with all its accompanying symptoms, will be the result. Hemorrhagic infarction may occur, but is very rare, since a free anastomosis exists between the lobular plexuses and the hepatic artery.

"Pylethrombosis may be regarded as probable if no other possible cause of the portal obstruction seems likely, and if we are able to discover a cause for thrombosis, like a former attack of circumscribed peritonitis" (Strümpell).

The **prognosis** is always unfavorable, although certain cases have been demonstrated by autopsy to have improved temporarily.

Course and Duration.—Nothing definite can be stated in regard to the course and duration, since these depend entirely upon the cause.

Treatment.—The symptoms resulting from portal congestion, due to thrombi in the portal vein, are those described under Cirrhosis of the Liver, and the treatment is identical with that of interstitial hepatitis. Septic emboli rarely give rise to abscesses; the treatment is symptomatic. It has been recommended to take the coagulation period of the blood, and if found to be abnormally brief, citric acid should be employed.

SUPPURATIVE PYLEPHLEBITIS.

Definition.—A purulent inflammation of the portal vein or its branches.

Pathology.—If noted in the early stages, the coats of the portal vein are distended and thickened, and the connective tissue surrounding the portal area is infiltrated and the seat of minute ecchymoses. The inflammation usually originates in the smaller veins of the portal system or in the hepatic branches of the vein itself; the main trunk is attacked least often. Numerous thrombi are found obstructing the vein and its branches, which finally undergo suppuration. From these, emboli enter the circulation and are carried to all parts of the liver, forming metastatic abscesses. In advanced cases the whole organ (especially the peripheral parts) becomes infiltrated with pockets of pus, that communicate

with the portal vein or its branches, and extend in some instances into the mesenteric or gastric veins. A single large abscess may be present, but multiple abscesses are the rule. The contents may be very fetid and bile-stained, or, as in many instances, they may be composed of thick, creamy laudable pus. From this focus of suppuration metastatic embolic abscesses may occur in the lungs, brain, kidneys, and joints.

The macroscopic appearance, with the organ *in situ*, is sometimes practically normal. The liver may present a uniform enlargement, the surface being of normal color and the capsule non-adherent. More commonly, however, the cortex presents a mottled appearance, and numerous yellowish-white spots are seen beneath the capsule.

Etiology.—The most frequent source of purulent pylephlebitis is appendicitis with abscess. Rarely the disease arises idiopathically.

Among other causes are—(*a*) A secondary (becoming a general) pyemia. (*b*) Ulceration of the intestines, occurring in dysentery and, more rarely, in typhoid fever. (*c*) Gastric ulcer. (*d*) Pelvic abscess; abscess of the spleen. (*e*) Specific infection through the umbilicus, occurring in the newborn. (*f*) The condition is more frequent in males.

Symptoms.—The symptoms vary according as to whether the case remains one of suppurative pylephlebitis or terminates in hepatic abscess. If the condition is part of a general *pyemia*, the symptoms referable to the liver may be almost negative. The *liver* is usually enlarged and tender on pressure; this enlargement is most marked when an hepatic abscess coexists. Though *pain* is present, it is not always severe; it is frequently referred to the epigastrium, and may radiate laterally or downward. *Percussion* in the left axillary line shows splenic enlargement, and the organ can in some instances be felt below the costal margin, constituting the "*acute splenic tumor*" of septicopyemia.

The *fever* is of septic type; the elevation in temperature is accompanied by rigors or chills and followed by profuse sweating. *Jaundice* of varying intensity is present, although usually it is not pronounced, the complexion being merely doughy or muddy. *Diarrhea* is not an infrequent symptom of this condition. Nausea and vomiting are often marked. As the case advances the pulse becomes rapid and small, and delirium develops, followed by stupor, coma, and death.

Duration and Prognosis.—The duration of suppurative pylephlebitis is usually from one to three or four weeks or longer. The prognosis is absolutely fatal.

Diagnosis.—The diagnosis of suppurative pylephlebitis is sometimes extremely difficult, unless the case is complicated by hepatic abscess, as enlargement of the liver is not constant in the former condition. The etiology, septic temperature, enlargement of the spleen, jaundice, and pain in the region of the liver would all, however, point to this affection.

The *differential diagnosis* of hepatic abscess will be spoken of later. Typhoid fever and the typhoid form of ulcerative endocarditis (without murmurs), as well as malaria, must be excluded.

Treatment.—Unfortunately, the treatment of suppurative pylephlebitis can only be palliative. Surgical measures are rarely curative, unless the abscess is single and localized and shows signs of pointing. The circulation is to be supported by free stimulation. The leading symptoms should be met as they arise.

STENOSIS.

Obstruction of the portal vein may be due, as before mentioned, to (*a*) thrombosis; (*b*) cicatricial contraction from cirrhosis or syphilis of the liver; and (*c*) tumors pressing on the portal area. The first cause is the more common, chiefly because mechanical obstruction, by causing a stasis of the blood-current, induces the formation of a thrombus.

The **symptoms** of portal stenosis may be *nil;* if the stenosis occurs slowly, the hepatic artery furnishes sufficient blood to carry on the functions of the liver, and the compensatory circulation is established by means of the systemic vessels. If due to thrombosis, the symptoms of portal engorgement appear suddenly with the development of edema and ascites. The liver is rarely enlarged.

Prognosis.—This depends wholly upon the cause of the affection. Thrombi in the portal vein often give rise to a suppurative pylephlebitis, terminating in hepatic abscess; tumors are rarely accessible; whereas fibroid conditions of the liver causing cicatricial contraction are incurable. As a rule, the prognosis may be said to be guardedly unfavorable.

AFFECTIONS OF THE HEPATIC BLOOD-VESSELS.

Osler records a case of *stenosis of the hepatic veins* that was associated with fibroid obliteration of the inferior vena cava, with a greatly enlarged and cirrhotic liver.

Among other affections of the hepatic veins are (*a*) Emboli, originating from a thrombus in the right auricle, and (*b*) Dilatation, from stasis of the blood-current flowing to the right heart, due to enlargement of the latter.

Affections of the *hepatic arteries* are exceedingly rare, but may occur in one of the following forms: (*a*) *Aneurysm.*—Only 10 or 12 cases of aneurysm have been reported. (*b*) *Hypertrophy and Dilatation.*—These may occur in connection with general hepatic cirrhosis, the cicatricial bands obstructing the lumen of the artery, and causing thickening in some places, and ampullæ, or sac-like dilatations, in others. (*c*) *Sclerosis.*—This may form a part of a general arterio-sclerosis, though it occurs oftener in connection with cirrhosis or syphilitic hepatitis.

ATROPHY AND HYPERTROPHY OF THE LIVER.

(*a*) *Atrophy.*—Simple atrophy of the liver may result from pressure (corset-liver), syphilis, advanced cirrhosis, senility, and from the toxic action of phosphorus, arsenic, or chloroform—all factors that induce rapid fatty degeneration with cell-destruction.

(*b*) *Hypertrophy* is of two kinds—(1) *true* and (2) *false.* (1) *True hypertrophy* may be subdivided into *simple* and *numerical* (hyperplasia), the latter referring to an increase in the number of the parenchymatous

cells, and not, necessarily, implying an increase in the size of the organ.

The two causes of simple hypertrophy are active and passive congestion. The principal causes of numerical hypertrophy are as follows: Leukemia, hypertrophic cirrhosis, atrophic cirrhosis (hyperplasia), syphilis, diabetes, and malaria.

(2) *Pseudo-* or *false hypertrophy* occurs in amyloid and fatty infiltration, carcinoma, and abscess, and consists in an increase in the tissues least concerned in the function of the organ.

HEPATIC INFILTRATIONS AND DEGENERATIONS.

AMYLOID INFILTRATION.

(*Waxy, Lardaceous, Bacony, or Albuminoid Infiltration; Amyloid Disease.*)

Definition.—A deposit in the hepatic connective tissues of a peculiar substance, which was formerly held to resemble starch, but has recently been shown to be related to coagulated albumin. Recklinghausen, who is also supported by other investigators, contends that at first there is hyaline change only, and that later amyloid transformation occurs. Amyloid material contains nitrogen, hence is related to the proteids.

Pathology.—The organ is increased in all of its diameters and of firmer consistence than the normal. The edges are rounded and not well defined, and the surface is of a light color, presenting in some instances a mottled appearance. On section the surface presents a grayish-brown, glistening appearance, which when scraped fails to exude oil-droplets, as in the fatty liver.

On microscopic examination the connective-tissue trabeculæ and the intima and media of the capillary walls (the starting-points) are chiefly affected, the lumen of the latter being lessened; this decreases the blood-supply to the liver, and often directly induces fatty degeneration. The hepatic cells may be atrophied.

Etiology.—Amyloid degeneration is most probably of microbic origin. Thus animals artificially infected with bacteria have shown amyloid change in the liver, spleen, etc.

Krawkow and, later, Davidsohn injected staphylococcus cultures, Gouget injected proteus cultures, and Carriere, the tubercle bacilli, and all obtained amyloid degeneration. Tuberculous foci that remain closed off are rarely attended with amyloid change, while ulcers of the intestines, the trachea, and the larynx show it with remarkable frequency.

Predisposing Causes.—Amyloid infiltration may occur primarily in the liver, but it is often a part of a general infiltration, affecting especially the spleen (*sago spleen*) and kidneys. It is also found in some syphilitic scars and in certain tumors and old thrombi.

It is a frequent sequel to long-standing and exhausting suppurating and cachectic affections, as necrosis of the bones, hip-joint disease, and pyelitis; "especially is this the case when they occur in an hereditary

tuberculous or syphilitic constitution" (Harley). Amyloid disease may also complicate chronic malaria, leukemia, and pseudoleukemia. In children tuberculosis and rachitis not uncommonly contribute to amyloid infiltration.

Tests and Characteristics of Amyloid Material.—Iodin gives a blue color upon the addition of sulphuric acid; "this reaction is often not obtained at all or it may be atypical" (Oser). Lugol's solution (the aqueous solution of iodin and potassium iodid) gives a brown tint to amyloid liver-substance and stains ordinary hepatic tissues a yellow color. Gentian-violet gives a reddish or pinkish hue to amyloid substance, while normal tissue is stained blue.

The following is taken from Harley's *Comparative Table of Amyloid Tests:*

	Starch.	Amyloid.	Cholesterin.
Water.	Dissolves on boiling.	Dissolves on boiling.	Unchanged.
Ether.	Insoluble.	Insoluble.	Dissolves.
Heat.	Dries up.	Dries up.	Melts.
Sulphuric acid.	Chars.	Swells up, reddish-brown.	Becomes green, blue, etc.
Iodin.	Becomes blue.	Blue color with H_2SO_4, which is destroyed by excess.	Remains unchanged.
Sulphate of indigo.		Amyloid tissue soaked in it becomes a brilliant blue, while with ordinary liver-tissues the blue fades to a pale green.	

Symptoms.—When amyloid disease occurs in *children* the subjects are poorly developed and puny, the complexion is, as a rule, muddy or sallow, and the abdomen usually prominent. Occasionally the *skin* is exceedingly transparent. At any age *gastro-intestinal symptoms* occur, prominent among which are marked constipation and a capricious appetite. *Mental phenomena*, as impairment of memory and inability to concentrate, are not unusual in this disease. *Pain* about the hepatic region is a rare symptom. The *spleen* is usually enlarged from coexistent amyloid infiltration. The *urine* often contains albumin (globulin is nearly always present), renal epithelium, and waxy tube-casts; it is of somewhat lowered specific gravity, but may be scanty and dark colored. Diarrhea, with slimy dejecta, is commonly present. The *physical signs* show an increase in the area of hepatic dulness; the edges of the organ extend below the costal margin and have a rounded outline. Sometimes, however, the edge, even in a very great enlargement, is sharp. Wilks speaks of an amyloid liver weighing 14 lbs.—6.35 kgms. (Osler). In rare instances the liver is reduced in size.

Diagnosis.—The foregoing symptoms and physical signs, in conjunction with an ordinarily clear etiology (syphilis, tuberculosis, or other primary process in some other part of the body), and amyloid degeneration elsewhere, are sufficient to establish the diagnosis.

Treatment.—As amyloid disease is almost invariably a secondary

condition, the treatment must be directed to the removal of the primary cause, whether syphilis, tuberculosis, or rickets. It has been shown recently that amyloid degeneration may disappear if the primary cause can be removed. The *diet* should consist of nitrogenous or animal substances, with a minimum amount of fat. French rolls and bran- or gluten-bread are allowable, together with lean meat, wholesome cereals, and green vegetables. Stimulants are to be strictly avoided. Moderate exercise, with the judicious use of Turkish (hot-air) and Russian (hot-vapor) baths, is also of great value.

Many drugs are mentioned in the treatment of this disease, among the more important being the ammonium salts (the chlorid, gr. v to x—0.324 to 0.648—three or four times a day) and other alkalies.

When syphilis has been clearly established as an etiologic factor of the disease, the tincture of iodin in 10- to 15-minim (0.666–0.999) doses, well diluted, has been recommended to be given three or four times daily. Cod-liver oil as a nutritive, if tuberculosis be associated, has been tried with good effect. Of tonics, the dilute mineral acids, given in moderate doses, have probably achieved the best results.

FATTY LIVER.

The term fatty liver embraces (*a*) *fatty infiltration*, or a deposit of fat in the otherwise normal hepatic tissues; and (*b*) *fatty degeneration*, in which a conversion of the albuminates of the cells into fat occurs. It should be recollected, however, that the latter condition is invariably preceded and accompanied by the former, so that most instances of fatty liver partake of the nature of both processes.

FATTY INFILTRATION.

Pathology.—The infiltration occurs often in localized areas, and may be so intense that the organ when cut presents a shiny, oily appearance. The liver is often evenly enlarged, and may weigh twelve to fifteen pounds. The edges are rounded and the substance less firm than normally. Portions of the liver-substance float in water. The color is light yellow or grayish. Microscopically, the protoplasm of the cell is seen to be pushed to one side by the fat droplets, which tend to coalesce. Fatty infiltration may end in fatty degeneration.

Etiology.—(*a*) Fatty infiltration may form part of a general obesity or it may follow gastro-intestinal disorders even in childhood. (*b*) It often occurs in wasting diseases, as carcinoma, syphilis, chronic malaria, and tuberculosis.

Symptoms.—The subjective symptoms of fatty infiltration may be entirely wanting, since the function of the liver is not impaired to any extent. When they are present progressive *anemia* and *debility* are noted, and are accompanied by *nervous irritability* and *insomnia*. In marked cases the cardiac rhythm is disturbed, causing a *feeble* and *irregular impulse*.

The *physical signs* are well defined, and the area of hepatic dulness is uniformly increased, extending in some instances as low as the umbilicus. The enlargement, however, is not so great as in amyloid disease.

Differential Diagnosis.—Fatty infiltration of the liver is not apt to be mistaken for any other affection of this organ. The occurrence of

general obesity, together with an entire absence of symptoms of obstruction to the portal vessels or bile-ducts or of other evidences of *fatty degeneration* (particularly feeble heart-sounds), will help to distinguish it from this latter condition. The etiologic factors above mentioned will also aid in the differentiation.

Prognosis.—This is decidedly favorable, as the function of the liver in many instances is not impaired in the slightest degree.

Treatment.—As the disease is of gradual development and long duration, a modification of the *diet* constitutes the first essential of the treatment. That prescribed under the Treatment of Amyloid Liver is admirably suited to this affection. Saccharine and farinaceous articles of food (potatoes, oatmeal, and sweetmeats) must be eschewed. Wheat-bread must be partaken of sparingly, and in its place gluten- and bran-bread or crusts of French rolls should be used. Fish, lean meats, fresh vegetables, and fruits are also allowable. Alcoholic beverages must be interdicted. When fatty liver develops in tuberculous subjects, the ingestion of fats and carbohydrates should be restricted.

Graduated daily exercise to stimulate metabolism and Turkish or Russian baths, judiciously used, are important factors in the treatment. Medicinally, the salts of the alkalies are highly recommended: sodium sulphate (in dram—4.0—doses, taken on an empty stomach) and ammonium carbonate (gr. xv to xxx—1.0 to 2.0—in twenty-four hours).

FATTY DEGENERATION OF THE LIVER.

Pathology.—On examining a liver that is the seat of marked fatty degeneration the organ is found smaller than normally, and the substance is light yellow in color, soft, pliable, and easily torn. On section the relation between the interlobular connective tissue and the acini is lost, the latter being replaced by fat-cells and oil-droplets. Scattered areas of pigmentation may be observed throughout the organ.

Microscopically, the cells lose their shape and become globular; the nuclei tend to coalesce, and finally disappear, together with the cell-wall, giving rise to compound globule-cells, which do not tend to coalesce and are stained black by osmic acid. Crystals, granular débris, Lener's spheres, cholesterin, tyrosin, and phosphatic crystals are also found in this form of granular change. Histologic differences are recognized at the present day between this disease and acute yellow atrophy.

Etiology.—The following are among the recognized causes of the affection: (*a*) The excessive use of beer or alcoholic liquors. (*b*) It may be a sequence of amyloid disease. (*c*) Diminution of the oxygen-supply to the tissues, occurring in phosphorus-, chloroform-, or arsenic-poisoning, and in certain wasting diseases (carcinoma, phthisis, and chronic dysentery). (*d*) It may occur as a complication in the grave anemias, especially pernicious anemia, in acute infectious diseases, and the intoxications, as well as in pregnancy.

Symptoms.—I feel convinced that partial or *mild cases* of fatty degeneration of the liver present no morbid symptoms of diagnostic import. Pain, jaundice, and ascites may occur separately or conjointly, but form the exception rather than the rule. The *severe forms* are characterized

by the symptoms seen in phosphorus-poisoning and acute yellow atrophy, to the discussion of which the reader is referred.

Complications.—The disease may be complicated with fatty change in the *kidneys.* Under these circumstances the *urine* is diminished in amount, of low specific gravity, and contains an abundance of albumin, fatty or oily casts, and crystals of cholesterin, leucin, and tyrosin. In marked cases there is a very *feeble* and *irregular cardiac impulse*, accompanied by attacks of *vertigo* and *syncope*, the latter symptoms indicating beginning degeneration of the cardiac muscle. *Edema* of the lower extremities and *anasarca* may occur as complications of this condition.

The **physical signs** elicited by *palpation* and *percussion* show increasing diminution in the size of the liver as the disease advances.

Diagnosis.—The chief diagnostic points of fatty degeneration may be summated thus: (*a*) A history of alcoholism, of poisoning by drugs (arsenic, phosphorus, or chloroform), or of an acute infectious disease (acute yellow atrophy); (*b*) Grave general symptoms, as albuminuria, edema, ascites, cardiac failure, terminating often in acholia or cholemia; (*c*) Progressive diminution in the size of the organ. When these occur conjointly the diagnosis is established beyond a doubt.

Prognosis.—The prognosis is entirely dependent upon the curability of the cause. If due to an excessive use of stimulants, the process, if recognized early, may be arrested; if associated with an acute infectious disease, the outlook is unpromising.

Treatment.—The indications for treatment may be divided into the *dietetic*, *hygienic*, and *medicinal.* The same precautions regarding diet should be observed as in fatty infiltration. An open-air existence, short of injurious exposure, aided by hot salt-water, Turkish, or Russian baths, under restriction, is sure to improve the general condition of the patient.

The *medicinal* treatment varies according to the cause of the disease. If due to grave anemia, iron (tinct. ferri chlorid. or syrup. ferri. iodid.) may be given in ascending doses. Poisoning by drugs that produce fatty degeneration of the liver is to be combated by their respective antidotes. Gastro-intestinal disturbances, if coexistent, demand appropriate treatment. For the latter Frerichs recommends highly the salts of the alkalies (sodium sulphate in dram—4.0—doses taken on an empty stomach and ammonium carbonate). Ascites and cardiac asthenia, when occurring as complications, must be met by suitable measures.

PERIHEPATITIS.

ACUTE PERIHEPATITIS.

(*Pyo-pneumothorax Subphrenicus.*)

Definition.—An inflammation, either suppurative or fibrinous, of the peritoneal covering of the liver and the corresponding portion of the diaphragm.

Pathology.—The morbid changes may consist in a purely plastic inflammation, the serous layers being thickened, opaque, and covered with a fibrinous exudate leading to adhesion. In the majority of cases,

however, the inflammatory product is chiefly purulent, and is ribboned by fibrous bands so as to form circumscribed areas, filled with pus, lying between the liver and the diaphragm; this constitutes the *subphrenic abscess*. The latter is found more commonly to the right than to the left of the suspensory ligament. It may contain much pus (1 quart—1 liter—or even more), which in most cases is mixed with air or gas derived from the gastro-intestinal canal. Rarely, bilirubin-crystals are found, betraying the presence of bile. If the latter be present in large amount, the pus assumes an ocher-yellow hue.

Etiology.—The *fibrinous variety* may result from the direct extension of one or other of the acute forms of inflammation of the liver (abscess, hydatid cyst), from a pleurisy spreading along the lymphatics in the diaphragm, or from traumatism—particularly a blow. The *suppurative form* (*pyo-pneumothorax subphrenicus*, Leyden) may be caused in the same manner as the former, but far oftener—in more than one-half of the instances—it follows perforation of a gastric ulcer, and far less commonly perforation of a duodenal or colonic ulcer. Appendicitis and penetrating wounds are not infrequent causes. Perihepatitis is a grave complicating event in carcinoma (of the stomach, esophagus, and intestines), in lobar pneumonia, and purulent pleuritis.

Symptoms.—Those of the *acute fibrinous variety* are either altogether missing or too vague to admit of correct interpretation. The coappearance, however, of severe *pain*, increased on deep breathing, and *tenderness* over a circumscribed area either in the right hypochondrium or the epigastrium, after the action of some known cause or the occurrence of one of the causative affections, is suggestive of this form of the complaint. A *friction-sound* may at times be heard below the seventh rib in the mammillary or over the epigastrium, as in two of my cases. It is of short duration, and is limited usually to the end of inspiration. Plastic pleurisy may, however, be an associated condition.

In *suppurative perihepatitis* the symptoms are sometimes screened by those of the special causative complaint; but in my experience, in cases due to perforation, the *onset* is rapid and severe and is marked by *acute pain* referred to a circumscribed spot in the hepatic region, great *tenderness*, rapid, embarrassed, and *painful respiration* (owing to implication of the diaphragm), by *vomiting* (often bilious, though at times hemorrhagic) or *nausea*, and by faint *jaundice* in some cases. Shortly the *general features* of circumscribed peritoneal abscess also appear—rigors, irregular fever, sweats, and progressive prostration and emaciation.

Physical Signs.—*Inspection* discloses bulging of the right hypochondrium and often of the epigastrium. The same regions are immobile, but this is best appreciated by *palpation*. Palpable friction may at times be obtained. The anterior edge of the liver is felt even as low as the umbilical level. *Percussion* reveals a variable increase of hepatic dulness upward, sometimes touching the fourth rib. The upper level of the fluid is movable on change of posture, and this is particularly striking if gas be contained in the abscess; the presence of the latter also causes a zone of tympanitic resonance above the dull area, while overlying the latter there is the semitympanitic area of the retracted lung. *Auscultation* reveals a *friction-sound* and an absence of breath-sounds and of the

vocal resonance over the dull and tympanitic areas, while the respiratory sounds over the displaced lung are broncho-vesicular.

Differential Diagnosis.—Acute perihepatitis often remains unrecognized during life. It may be confounded with *empyema* of the right side, but the two conditions have different modes of development. Perihepatitis is preceded and accompanied by abdominal symptoms; empyema manifests thoracic symptoms—*e. g.* cough and pleuritic pain. At a later stage the exaggerated respiratory murmur above the dull area, the slighter cardiac displacement toward the left, and the greater hepatic displacement downward in suppurative perihepatitis aid in the differentiation. The introduction of the trocar in the seventh or eighth intercostal space in the mid-axillary line may also be helpful, especially if the exudate be found to contain bile-pigment. *Pfuhl's sign*—the more ready escape of the fluid during inspiration on aspiration of abscesses below the diaphragm—may not be without value. The points narrated above may likewise serve to separate pyo-pneumothorax from suppurative perihepatitis (see also Pneumothorax, p. 607). To differentiate from *acute plastic pleurisy*, Cantlie's sign, or grasping the liver between the hands and moving it backward and forward, thus causing pain running up into the supraclavicular fossa, may be employed.

Course and Prognosis.—In the milder or fibrinous variety the outlook is favorable and the course is brief. On the other hand, the suppurative type due to perforation, if not early brought under proper surgical treatment, often terminates unfavorably by gradual asthenia. Rarely the pus is resorbed, or it may find an outlet through the lungs, abdominal walls, or other avenue, followed by slow recovery.

The **treatment** is the same as for localized peritonitis. The first evidence of the presence of pus is the signal for surgical interference—evacuation and drainage, and Wakar[1] favors the transpleural method.

CHRONIC PERIHEPATITIS.

(*Zuckergussleber.*)

This affection is a chronic inflammation of the perihepatic fibrous membrane, which becomes opaque and thickened. Contraction of this capsule ensues, with compression of the liver and atrophy to one-half the size of the normal organ (as in a case reported by Rumpf[2]), and partial or total occlusion of the vessel and bile-ducts. These changes are most marked in cases that follow acute suppurative perihepatitis. Genuine instances show no hyperplasia of the interstitial connective tissue; hence the condition is closely related pathologically to "Glissonian cirrhosis."

The main **causes** of chronic perihepatitis are great and protracted local pressure, as from a corset, and certain occupations. It may represent a portion of a more general chronic inflammation of the serosæ. I believe that syphilis is the leading single cause, and could discover no other factor present in two cases that yielded to antisyphilitic treatment. A circumscribed form (benign) may occur from local pressure.

The **diagnosis** is generally problematic. Of especial clinical worth are the etiology, pain in the right hypochondriac region—particularly in cases due to syphilis—absence of the signs of stasis of the gastro-intestinal tract, and the very protracted course.

[1] *Deutsche Zeitschrift für Chir.*, Leipsic, January, 1912

[2] *Deutsch. Arch. f. klin. Med.*, March 13, 1895.

The **treatment** is purely palliative, apart from the effort to remove the special cause, whether this be syphilis, occupation, or other influential factor.

ABSCESS OF THE LIVER.

(*Hepatic Abscess; Suppurative Hepatitis.*)

Definition.—A circumscribed collection of pus in the hepatic parenchyma.

Pathology.—If examined *in situ*, a liver that is the seat of abscess-formation is usually found to be symmetrically enlarged, and on careful palpation one or more areas of fluctuation (either deep or superficial, according to the location of the abscess) may be detected. If single, its position is usually in the right lobe near the convexity of the organ (70 per cent. of cases). The tissue surrounding the abscess-wall is usually deeply injected, the wall itself in acute cases being poorly defined, but grayish in color, irregular and shreddy, and composed of necrotic liver-cells, pus-corpuscles, and often amebæ. In chronic cases it becomes greatly thickened and often cartilaginous in appearance.

The amount of fluid contained in a liver-abscess may exceed 2 or 3 quarts (2–3 liters), and its color varies from grayish-white to a creamy, reddish-brown. The collection in some instances resembles healthy pus. I have spoken of the methods of infection and of some of the different varieties of hepatic abscess in the discussion of Dysentery (see p. 72).

Various odors are described, depending largely on the extent of bacterial invasion and the degree of necrosis. Here it may be said that in amebic dysentery, hepatic abscess is often single (involving more often the right lobe), whereas, in general pyemia, multiple abscesses are the rule. Multiple tropical abscess, however, is not uncommon and is indistinguishable from those that are met in temperate climates as the result of infection *via* the portal vein.

In these instances the surface of the organ presents many small yellow areas beneath the capsule, varying from 5 to 15 mm. ($\frac{1}{5}$—$\frac{3}{5}$ inches) in diameter. Usually, in such cases too, the appearances of a suppurative pylephlebitis present themselves. If thrombi have formed in the portal tributaries, localized necrotic areas are the result, but more often the invasion affects the whole portal system, the liver being riddled with abscesses. If the abscess is secondary to obstruction by gall-stones or inspissated bile, the ducts and the gall-bladder are greatly distended, their walls and immediate vicinity infiltrated with round cells, leading to suppurative pericholangitis and invasion of the hepatic parenchyma.

Microscopically, the hepatic cells are altered in shape and devoid of nuclei; they undergo rapid degeneration. A round-celled infiltration occurs about the blood-vessels, their walls being filled with small emboli containing innumerable staphylococci and streptococci. As the suppurative process continues liquefaction-necrosis occurs, resulting in complete destruction of the hepatic parenchyma.

Etiology.—Idiopathic abscess of the liver is rare even in tropical climates. The affection, when apparently excited by mechanical causes,

as traumatism or obstruction by gall-stones, is invariably a micro-organismal affection, and the principal germs are streptococci, staphylococci, and the ameba coli.

Gastric ulcers, typhoid fever, or appendicitis may be followed by a purulent portal pylephlebitis, resulting in abscess-formation. On analyzing 500 cases of suppurative hepatitis Kelsch found that in 85 cases in 100 the disease was associated with dysentery. Manson records a total of 3680 autopsies made on dysenteric patients in tropical countries, and of these 21 per cent. showed abscess of the liver. Among Europeans in tropical climates the general average is 12 per cent. In general pyemic processes or in bone-suppurations of long standing infection of the liver occurs. Suppurating wounds of the head may be followed by abscess of the liver. Among other causes may be mentioned *foreign bodies* travelling up the ducts, as parasites, round-worms, liver-flukes; also, more rarely, suppuroperforation by mechanical irritants (needles, pins, fish-bones, and the like), and suppuration occurring in the course of an hydatid cyst. Leick has tabulated 19 cases of hepatic abscess caused by the ascaris lumbricoides. Among other factors are: Age (adult life), male sex, alcoholism, and malaria.

The manner of infection is variable; it may be (*a*) through the portal vein (most commonly); (*b*) through the bile-ducts; and (*c*) the metastasis may take place through the blood (hepatic artery). (*d*) Exceptionally infection may occur *via* the lymph-channels.

Symptoms of Solitary Abscess.—In a typical case of hepatic abscess the most prominent symptoms are—*hectic temperature*, *pain*, *tenderness*, and *enlargement of the organ*, and often *slight jaundice*, although it must not be forgotten that any or all of these may be absent during its development. The multiple abscesses occurring in pyemic conditions, which are frequently diagnosed when in view upon the postmortem table, form an instance of this. The present description, however, has reference chiefly to the large, solitary abscess.

To facilitate the subject I shall consider the more important symptoms *seriatim*: *Pain* is circumscribed to the hepatic region, and radiates to the right shoulder in conjunction with the other symptoms and physical signs; it is very characteristic, although not pathognomonic of hepatic abscess. In the earlier stages this symptom is not pronounced unless the abscess or abscesses lie superficially. It is usually of a dull, boring character, differing in severity with the patient's position; it is usually aggravated by pressure over the costal margin and by lying on the left side, this tending to drag the liver by its own weight from its normal position. Luschka explains the radiation of pain to the right shoulder by stating that filaments of the phrenic nerves that distribute themselves in the suspensory ligament and Glisson's capsule are irritated. The phrenic arises from the third, fourth, and fifth cervical nerves, and, as the fourth supplies sensation to the right shoulder, the impression is thus transmitted through the central nervous system.

In acute cases accompanied by rapid destruction of the hepatic tissues the *temperature* usually rises rapidly, reaching 103° or 104° F. (39.4°–40° C.) in the course of from twenty-four to thirty-six hours. Its course, however, is irregular and intermittent, and it may be hectic in character; just as often it resembles a tertian or quartan intermittent or

a remittent malarial fever. *Rigors* or *decided chills* frequently accompany the rise of temperature, and during the decline profuse sweatings may take place. In chronic abscess of the liver pyrexia may be entirely absent. Less commonly the temperature may remain continuously high, with slight morning remissions. The *pulse* is usually rapid in proportion to the temperature.

The physical signs in a case of hepatic abscess are always present to a greater or less degree, and are often pathognomonic.

Inspection may reveal nothing during the entire course of the disease, although if there be intense congestion involving the anterior surface of the right lobe, bulging of the ribs on that side will occur, with a marked prominence in the hypochondriac region extending three or more finger-breadths below the costal margin.

Palpation confirms inspection and reveals tenderness on pressure below the costal margin in the mammary line. The liver, if projecting below the edge of the ribs, is usually enlarged uniformly, unless the abscess involves the surface of the margin. As the upper right lobe is more often involved, the increase in diameter is upward, thus rendering palpation negative. In rare instances the abscess gives rise to fluctuation on palpation, and a friction fremitus if the peritoneum be inflamed.

Percussion.—The area of hepatic dulness may be increased uniformly, but it is usually most marked upward and to the right (fifth rib), and posteriorly to the level of the angle of the scapula. This high position of the upper boundary of dulness which starts about the nipple-line serves to differentiate abscess from other affections of the liver, in which the enlargement extends in a downward direction.

Other Symptoms.—The *skin* is pale and shows slight icterus, the *conjunctivæ* being often bile-stained; intense *jaundice*, however, is rare. Progressive loss of flesh and strength, with *gastro-intestinal disturbance* (fulness in the epigastrium, flatulence, water-brash, nausea, and occasional vomiting), are common symptoms at the onset. The *bowels* are variable, and constipation usually alternates with diarrhea, the stools in some cases containing the ameba coli. *Ascites* may develop from pressure on the inferior vena cavæ, but such cases are rare. The *spleen* may undergo active hyperplasia in acute abscess-formation. *Pulmonary symptoms* (severe cough, characteristic reddish-brown sputum, resembling anchovy sauce, broncho-vesicular breathing, râles) are commonly present; they are due to compression of the base of the lung by the abscess pressing upon the diaphragm. In fatal cases certain *nervous symptoms* (muttering delirium, cephalalgia, subsultus tendinum, stupor, coma) make their appearance. A marked *leukocytosis* is generally present.

Complications and Sequelæ.—The abscess may perforate into the pleural cavity (pyothorax), bronchi, lungs, intestinal tract, stomach, pericardium, peritoneal cavity, or externally through the abdominal wall, giving rise to various symptoms. If rupture occurs into the intestinal tract, sudden diarrhea, with the discharge of large quantities of pus, takes place. If the rupture is into the lung, the physical signs will reveal the sudden development of weak, tubular breathing over the base, with increased tactile fremitus and percussion-dulness, together with the occurrence of profuse and typical expectoration. Reese, Lafleur,

and Boston found the ameba coli in the bronchial discharge. Rupture into the abdominal cavity gives rise to the development of a fatal peritonitis. Cerebral abscess may occur.

Diagnosis.—The clinical symptoms of hepatic abscess are of diagnostic importance only when taken in the aggregate, since the pain, fever, enlargement, and even hectic symptoms occur singly in other conditions unaccompanied by suppuration. The principal points in the establishment of the diagnosis of the affection may be summed up as follows: Residence in tropical countries, the previous existence of typhoid or dysenteric ulceration (or other gastro-intestinal inflammation), the characteristic expectoration, enlargement of the liver, with pain and tenderness on pressure, and in some instances fluctuation on palpation. Pain in the liver, often radiating to the shoulder, if the patient be shaken, is a certain sign (Malbot). Skiagraphy shows the right leaflet of the diaphragm to be displaced upward and unaffected by respiration. Lastly, exploratory aspiration with a medium-sized needle is to be resorted to in dubious cases; it may reveal pus-corpuscles, hepatic cells, staphylococci and streptococci, the ameba, and bile-pigment, which when found are pathognomonic; if the abscess be secondary to an echinococcus cyst, the presence of hooklets will be detected. The patient should be anesthetized, since the puncture may have to be repeated.

Differential Diagnosis.—Hepatic abscess may be misdiagnosed for *empyema*, *suppurative pylephlebitis*, *malarial fever*, and *hepatic calculi*.

Empyema.—In empyema there may be the history of a perforating wound of the chest, the rupture of a bronchiectatic or tuberculous cavity, or the pre-existence of a sero-fibrinous pleurisy; whereas hepatic abscess may be preceded by an attack of amebic dysentery, intestinal ulceration from other forms of infection, impacted gall-stones, traumatism, or a pyemic process. In both there may be the occurrence of a hectic temperature, with chills and sweating; but in empyema cough and dyspnea are prominent, and, if the pleural cavity communicates with a bronchus, profuse mucopurulent expectoration containing pus-cells, staphylococci, streptococci, and tubercle bacilli may be observed. Rarely an abscess of the liver penetrates the diaphragm and is expectorated. The recognition of hepatic abscess under these circumstances is to be based mainly upon clear previous evidence of the affection, and copious, blood-tinted, purulent expectoration. The detection of the ameba coli in the sputum alone would set the diagnosis at rest. The contents of hepatic abscess obtained by aspiration consist of the micro-organisms of suppuration, broken-down liver-cells, bile-pigment, and in some cases the ameba coli. Inspection in empyema reveals bulging of the intercostal spaces on the side implicated, and there is movable percussion-flatness over the base of the chest, rising posteriorly. On the other hand, in abscess of the liver, the lung is slightly displaced upward, being often bound to the diaphragm by adhesions; and the upper boundary of dulness is lower, particularly in front, and is immovable.

Suppurative Pylephlebitis.—In hepatic abscess there are present certain physical signs (swelling, fluctuation), and a history of amebic dysentery, rather than of appendicitis, as in suppurative pylephlebitis.

Hepatic Abscess.	Malaria.
History of traumatism, dysentery, intestinal ulceration, or residence in tropical countries.	History of previous attacks. Residence in warm, damp climates among the lowlands.
Hectic character of the temperature—high every evening and low every morning; irregular chills, followed by fevers and sweatings.	Regularly recurrent rise of the temperature (intermittent or remittent, quotidian, tertian, quartan, or septinarian), followed by profuse sweating; chills more often in morning.
An irregular, fluctuating tumor or multiple nodules in the liver; no splenic enlargement; rapid emaciation, with or without jaundice, but no cachexia.	The spleen is enlarged; also there is a yellow-brown coloration of the skin, more or less marked; and, in long-standing cases, the occurrence of *cachexia*.
Blood shows simple anemia and leukocytosis, and in marked cases disintegration of red blood-cells.	The presence of the hematozoa of Laveran and free pigment in the blood; usually a leukopenia.
Abscess-contents show the staphylococci, streptococci, amebæ, or bacillus coli communis, and pus.	Absent.
Quinine is resisted.	Quinine acts as a specific.

Impacted Calculi.—In this condition attacks of *hepatic colic* are often first noticed, followed by jaundice, and, if impaction be not absolute, by the occurrence of stones in the feces. In abscess the pain is not paroxysmal, but dull and boring in character, increasing in severity as the disease progresses. In *chronic impaction* jaundice, dull pain over the hepatic area, distention of the gall-bladder (which in some instances may be palpated), and clay-colored feces, constitute the principal symptoms. There occurs also an intermittent fever as in hepatic abscess, but it is occasional—*i. e.* the febrile paroxysms recur at longer intervals. Again, the course of intermittent hepatic fever associated with biliary calculi is much more chronic than the fever-stage of suppurative hepatitis. On the other hand, in abscess of the liver jaundice is comparatively rare, and, unless the abscess rupture into the gastro-intestinal tract, the stools show nothing abnormal. In some instances biliary abscesses may follow impacted calculi, and it is always a secondary affection.

Among other liver-conditions that are liable to be mistaken for hepatic abscess may be mentioned *carcinoma*, *hypertrophic cirrhosis*, *hydatid cyst*, and *pancreatic cyst*, the differential diagnosis of which will be spoken of under these diseases.

Prognosis.—The prognosis of hepatic abscess is unfavorable, the disease generally progressing to a rapidly fatal termination. Prompt evacuation of the abscess when its location can be detected, however, may be successfully performed. The mortality ranges from 50 to 60 per cent. In rare cases the walls of the abscess become calcified and the disease remains latent. The single large abscess that most often follows dysentery offers the best opportunity for surgical measures.

Treatment.—Barring operation, the treatment of abscess of the liver is purely symptomatic. The temperature often responds to repeated spongings with cool water (65° F.—18.3° C.). For the pain, mustard-poultices, the turpentine stupe, or hot fomentations over the hepatic area, in conjunction with full internal doses of opium, prove beneficial. Full and free stimulation and the free exhibition of quinin as soon as the condition is detected proves supportive and controls, in a measure, the

pyemic process. L. Rogers holds that 90 per cent. of amebic abscesses of the liver can be prevented by large doses of ipecac (gr. xxx–lx—2.0–4.0, daily, in freshly made pills). If the abscess be single and localized, prompt evacuation should be resorted to. Patients who have been thus cured should not return to a climate in which tropical dysentery occurs, since, as in a case reported by Marshall, recurrence may take place.

ACUTE YELLOW ATROPHY.

(*Malignant Jaundice; Icterus Gravis.*)

Definition.—An acute and probably infectious disease (rare), characterized by a rapid destruction of the parenchyma of the liver and by a diminution in the size of the organ; also by jaundice, hemorrhage, and grave cerebral phenomena.

Pathology.—*Macroscopically*, in a case of acute yellow atrophy the liver is seen to be much reduced in size, weighing but 15 or 20 ounces (480.0–640.0), instead of its normal weight (50 oz.—1.6 kgms.). The capsule is shrivelled and the organ is of a pulpy consistence, and changed in appearance from a mahogany-brown to a grayish-yellow hue. Sometimes the liver is primarily enlarged. The cut section often presents areas of red and yellow discoloration, the so-called "red atrophy" and "yellow atrophy," the former being a later stage of the latter. The red appearance is due to an excess of blood in the capillaries, with free pigment that has been liberated by destruction of the red blood-cells. *Microscopic examination* reveals destruction or necrosis of the hepatic cells. The nuclei have disappeared, and the cell-wall contains a number of fat-globules of various sizes containing free pigment. In advanced cases, accompanied by total disintegration of the cells, fat-droplets, granular débris, cholesterin-plates, leucin-spheres, tyrosin-needles (first discovered by Frerichs, both in the cells and in the blood-vessels), and crystals of bilirubin may be found. Findlay[1] found the fibrous tissue to be increased and in the periphery of the lobules attempts at regeneration (proliferation of the hepatic cells). The common duct is patulous.

In well-marked cases both the *heart* and *kidneys* show evidences of fatty degeneration. The *spleen* is greatly enlarged from active congestion, giving rise to the so-called "acute splenic tumor." The splenic substance is soft and easily torn. The *skin* and *mucous membranes* may be the seat of numerous ecchymoses, and dropsy of the serous cavities is frequently noted. The *blood* is dark and fluid (disintegrated). *Microscopically*, it is seen to contain crystals of leucin and tyrosin.

Etiology.—The causes of acute yellow atrophy are both primary and secondary. *Primary* or idiopathic acute yellow atrophy is rare and its etiology as yet unsettled. Among the *secondary* predisposing causes are age (being most common from fifteen to thirty-five years), female sex, mental worry, nervous shock, parturition, syphilis, chloroform anesthesia (?), and certain acute fevers (puerperal fever, typhoid, septicemia, malaria). Acute phosphorus-poisoning sometimes presents changes resembling those of acute yellow atrophy. The disease rarely accompanies cirrhosis of the liver and may follow a debauch. Rarely, an endemic

[1] *British Medical Journal*, June 2, 1900.

form is assumed, but the exciting cause is thus far unknown. The disease is probably microörganismal or toxic in nature, and although various germs have been discovered, their claim to specificity has not been established.

Symptoms.—The clinical history of acute yellow atrophy varies considerably in the early stages of the disease, the graver symptoms of the later stage alone being pathognomonic. The attack is usually ushered in by *headache*, *malaise*, *anorexia*, *nausea*, and *vomiting*, moderate *fever*, and after a few days *jaundice* appears. *Physical examination* at this time shows the area of hepatic dulness to be normal or only slightly increased. After a period varying from a few days to two or three weeks (during which the typical features of catarrhal jaundice have been present), grave *nervous* and *cerebral* symptoms present themselves, as restlessness and violent headache, followed by delirium, which often becomes maniacal. *Convulsions* then appear, and are succeeded by stupor and coma, the latter occurring usually within forty-eight hours from the onset of the period of cerebral excitement. Often *coarse tremors* are noticed in the voluntary muscles, and with the onset of the second stage the jaundice usually deepens.

The *temperature* often remains normal until just before death, when it may rise one or two degrees. The *pulse* is much diminished both in volume and tension, and is rapid in proportion to the temperature. The *tongue* at the onset is covered with a light coating, most marked on the dorsum and tip. Later, it changes to a thick yellow color and becomes dry and fissured, with the development of a typhoid state. Vomiting appears usually during the premonitory stage and often becomes intense; the *vomit* consists at first simply of the gastric contents, which later in the disease becomes mixed with blood (hematemesis). *Hemorrhages* also occur into the skin (ecchymoses) and from the mucous membranes, giving rise to epistaxis, hematuria, melena, hemoptysis, and menorrhagia. *Constipation* with clay-colored stools is common.

The *urine* in acute yellow atrophy is often scanty in amount, high colored, and shows an increase in specific gravity (1028–1032). The urea is greatly diminished, but bile-pigments and albumin, tube-casts, *leucin* and *tyrosin* are found both on chemical and microscopic examination. The latter can be easily demonstrated by allowing a drop of the urine to evaporate on a cover-glass and examining under the microscope. Tyrosin-crystals are deposited in the form of sheaves and rosettes, leucin as globular masses. These bodies are not constantly present. Thus, out of 34 cases collected by Thierfelder, in which the urine was examined in this relation, "in 7 the result was negative; in 17 both were found; in 3 tyrosin only; in 7 leucin only." Among other products found in the urine worthy of mention are creatinin, lactic and sarcolactic acids, and other bodies belonging to the fatty acid series.

Acute yellow atrophy of the liver is a striking example of acid-intoxication due to rapid and widespread destruction of proteids as the source of the fatty acids—sarcolactic, lactic, diacetic, and β-oxybutyric. The rare nervous phenomena of the disease are, in part at least, due to the diminished alkalinity of the blood arising from the abnormal metabolism.

The **physical signs** reveal tenderness over the hepatic region, often amounting to actual pain. During the second stage, in extreme cases, the

edges of the organ cannot be palpated under the costal margin. Percussion, moreover, shows a great diminution in the size of the liver, the area of dulness in a case recorded by Harley extending over but 1 inch (2.5 cm.) in the mammary line and 1¼ inches (3.1 cm.), measured perpendicularly, in the mid-axillary line.

The left lobe is often the first to show physical signs of atrophy, percussion giving tympany instead of flatness in the upper epigastric region. As the atrophy continues the tympany exends below the seventh rib from above and advances upward from the costal margin, leaving but a small circumscribed area of hepatic dulness. The atrophy is usually progressive until death occurs, although favorable cases have been recorded in which the liver increased in size perceptibly during recovery (Harley, p. 260).

Diagnosis.—The symptoms occurring during the second stage of the disease are usually so characteristic as to leave little doubt concerning the diagnosis. The occurrence of gradually increasing jaundice with vomiting, grave delirium, hemorrhages, the presence of an immense amount of bile, with leucin and tyrosin, in the urine, and greatly diminished size of the liver, all combine to form a typical symptom-complex. Unfortunately, leucin and tyrosin are also found in the urine in acute phosphorus-poisoning and rarely in severe acute infections.

Differential Diagnosis.—In *hypertrophic cirrhosis* the onset is more gradual. There is generally a negative previous history; and an examination of the urine fails to reveal leucin and tyrosin; fever is rarely present in cirrhosis, and the physical signs often show a considerable increase in the area of hepatic dulness.

The differential diagnosis between this disease and phosphorus-poisoning is given under the latter condition (*vide infra*, p. 931).

The **prognosis** is almost invariably fatal, since every case of true yellow atrophy is associated with a destruction of liver-cells that is accompanied by acute toxemia.

Treatment.—As yet no specific treatment has been discovered, all remedies used being directed to the relief of symptomatic indications. The gastro-intestinal system should be relieved at the onset by divided doses of calomel. For the vomiting cracked ice, with 1-minim (0.066) doses of the wine of ipecac repeated every half hour or divided doses of opium, may be given. Marked nervous phenomena with delirium I have seen controlled by cool baths and the ice-cap, together with camphor, chloral, or other antispasmodics used internally. Free stimulation should be begun early and persisted in throughout the course of the disease.

THE LIVER IN PHOSPHORUS-POISONING.

FOLLOWING the ingestion of a dose of phosphorus varying from gr. ⅛ to gr. 1 (0.008–0.0648) symptoms of poisoning manifest themselves (Taylor, Wormley) as follows:

After a period of time varying from three to twelve hours a sense of wretchedness, nausea, abdominal pain (not intense), and often vomiting,

occur. The *vomitus* consists of the gastric contents, with bile, and during the first few hours it may contain phosphorus, which gives it a luminous appearance in the dark.

After the second or third day the vomiting usually ceases with the appearance of *jaundice*, which may become intense as the process continues. Later in the course of the case emesis recommences, the vomita consisting of altered blood, giving rise to the so-called "black vomit." At this stage *nervous symptoms* usually manifest themselves (headache, insomnia, vertigo, and delirium, with convulsions and coma in fatal cases), death closing the scene usually in from thirty-six to forty-eight hours.

The *bowels* are constipated, although attacks of diarrhea may supervene, and the evacuations are in some instances phosphorescent.

Fever is irregular and usually is not marked, the temperature swinging from 99° to 101° F. (37.2°–38.3° C.). In fatal cases the temperature may become subnormal just before death.

The *urine* is scanty, of high specific gravity, and contains bile, bile-acids, albumin, sarcolactic acid, and in rare cases leucin and tyrosin (Wood). Renal epithelium and free fat-globules have also been found. When occurring in pregnant women, abortion or miscarriage invariably follows.

Physical examination reveals a liver uniformly enlarged and tender on pressure. In protracted cases atrophy of the organ may rarely occur.

Etiology.—The most common causes are—(*a*) Occupation, workers in match-factories being the most frequent sufferers; (*b*) The accidental swallowing of phosphorus (*e. g.* rat-poison, friction-match heads).

Pathology.—On opening the abdominal cavity in a case of phosphorus-poisoning the liver is seen to extend below the costal margin, its surface being lighter in color than normal and mottled in appearance, and its substance softer in consistence and friable.

The cut section presents marked evidences of fatty degeneration, the acini being lighter in color than the interlobular tissue. Portions of the hepatic parenchyma are deeply bile-stained, and on scraping the cut surface bile- and fat-globules will be found on the edge of the knife. The gall-bladder may be either full or empty. *Microscopically*, disintegrated liver-cells, fat-globules, granular débris, biliary coloring-matter, leucin-spheres, cholesterin-plates, and tyrosin-needles are noted.

The *gastric mucosa* is found thickened, opaque, and yellow-white in appearance, due, as pointed out by Virchow, to a universal gastro-adenitis, and not to the local action of the poison. Ulcerative or erosive gastritis is very rare in phosphorus-poisoning.

The *kidneys* may show beginning atrophy, the epithelium in the cortices undergoing granular and fatty degeneration, with final destruction of the cells.

The *blood* is dark, fluid, and not easily coagulable. Concato found that during life the white corpuscles are increased in number, and that the red are changed in shape and smaller than normal (Wood). Petechiæ and ecchymoses frequently appear in all parts of the body.

Diagnosis.—The diagnosis of acute phosphorus-poisoning is always extremely difficult and often impossible. The disease with which it is

most apt to become confounded is *acute yellow atrophy of the liver.* The differential points may be summated as follows:

Acute Phosphorus-poisoning.	Acute Yellow Atrophy.
There is a history of accidental ingestion of poison (friction-match heads, rat-poison) or occupation.	There may be an endemic history.
The onset is sudden; violent nausea, vomiting, and pain over the region of the liver. Jaundice appears on the second or third day.	A slow onset—malaise, slight fever, with nausea and vomiting; jaundice is a beginning symptom.
Nervous symptoms appear late in the disease—always preceded by jaundice.	Nervous symptoms may appear early, even before the occurrence of jaundice.
The vomit and stools are phosphorescent. Black vomit precedes death.	Black vomit occurs early and persists throughout.
Temporary arrest of symptoms between the occurrence of jaundice and black vomit.	Progressive march of symptoms with no remission.
Sarcolactic acid is present in the urine, and rarely leucin and tyrosin.	Leucin and tyrosin are common in the urine.

Prognosis and Duration.—The prognosis in phosphorus-poisoning is bad, as small a dose as gr. $\frac{1}{8}$ (0.008) of white phosphorus having caused death (Wormley). The duration is usually from one to six days, although the symptoms have been known to persist for twelve days before death. In violent cases the end may come within twenty-four hours.

Treatment.—The initial plan of treatment is by causing emesis to free the system of the poison that still remains undigested. For this purpose copper sulphate (gr. x—0.648) in divided doses (gr. ij or iij—0.129 or 0.194—every five minutes) should be given until free vomiting occurs. As copper sulphate is a chemical antidote, forming with phosphorus black copper phosphid, it should be continued in less frequently repeated doses (gr. ij—0.129—every half hour) and guarded by morphin to prevent vomiting. If emetics by the mouth fail to afford relief, apomorphin muriate (gr. $\frac{1}{5}$—0.0129), hypodermically, may be resorted to. The free evacuation of the stomach should be followed by the administration of the French oil of turpentine. Wood recommends that 1 part be given to every 100th part of the poison ingested. Ordinary turpentine is useless, but combined with mucilage of acacia, 2 fluidrams (8.0) of French oil of turpentine may be given every fifteen minutes until 1 ounce (32.0) has been taken.

Alkalies (magnesia) have been given, but are practically valueless. Free purgation should be effected if possible by Rochelle salts or magnesium citrate. Demulcent oils are never allowable, as they dissolve the phosphorus and hold it in solution. After absorption of the poison and degeneration of the tissues have taken place all known remedies are futile.

CIRRHOSIS OF THE LIVER.

(*Sclerosis of the Liver; Nutmeg Liver; Gin-drinker's Liver; Interstitial Hepatitis.*)

Definition.—A chronic disease of the liver, characterized, pathologically, by an excess of connective tissue. It presents various biliary, gastro-intestinal, circulatory, and cerebral symptoms.

Pathology.—There are three pathologic varieties: (*a*) atrophic cir-

rhosis, or "gin-drinker's liver"; (*b*) hypertrophic cirrhosis; and (*c*) biliary cirrhosis.

(*a*) **Atrophic Cirrhosis** (*Laennec's*, or *alcoholic cirrhosis*) is the most common form, at least in the earliest stages, as Foxwell's studies teach; the alcoholic (indurative) liver is more commonly enlarged than decreased in size. Morse[1] examined the records of 37 cases of cirrhosis, and found that among these there were 13 instances of enlarged liver, 11 of normal size, and 12 smaller than normal. In typical examples the capsule is thickened, the organ greatly reduced in size, hard, granular, and much altered in shape. On section (which resists the cutting-knife) the surface presents grayish-white bands of connective tissue surrounding yellowish areas (acini) that project above the surface from compression (hob-nails); hence the term "hob-nailed liver."

Microscopically, the process is seen to commence as an increase in the connective-tissue element surrounding the terminal branches of the portal vein. Compression of the liver-cells and of the portal veins, with consequent obstruction of the circulation, constantly increases with the progress of the proliferation of the connective tissue and its secondary contraction. Atrophic changes in the hepatic cells, however, are often comparatively slight. The biliary canaliculi may be increased in number. Weigert and his disciples contend that atrophy or degeneration of the acini is often the primary change, and the connective-tissue production the secondary—filling the gap, so to speak.

In alcoholic cirrhosis the liver is sometimes large, smooth, or slightly granular, soft rather than hard, as ordinarily the case, and presents a light yellow color (*fatty cirrhosis*). Histologically, this is a form of true cirrhosis, as shown by the presence of an increase in the connective tissue, with which, however, fatty infiltration of the acini is associated.

(*b*) **Hypertrophic Cirrhosis** (Hanot).—On examining the liver *in situ* during hypertrophic cirrhosis the various diameters of the organ are increased (the left sometimes more than the right), the lower border projecting several fingers' breadths below the ribs. The margin of the liver is well defined, the substance firmer than normal, and it cuts with difficulty. The organ is lighter in color than in health, and presents a yellow or mottled-green appearance. On treating a section with compound iodin solution (Lugol's) the color changes to that of a deep mahogany-red. The acini are darker in hue than the interstitial tissue.

Microscopically, the peripheral zones of the acini are seen to be the seat of a round-cell infiltration, with the formation of embryonal tissue; later, the interlobular connective tissue undergoes hyperplasia, causing obstruction of the biliary ducts with retention of bile, but the parenchyma is unchanged. New-formed bile-ducts are proliferated.

(*c*) **Biliary Cirrhosis.**—French writers have described "biliary cirrhosis" as opposed to a "portal cirrhosis" (atrophic). It results from obstruction of the bile-ducts; this causes retention of bile with swelling of the organ as a consequence. The action of the chemical irritants that are the result of stasis of bile starts a cirrhotic process around the small bile-ducts (reactive inflammation). The *microscopic appearances* of the organ simulate those of hypertrophic cirrhosis; but the hepatic

[1] *Boston Med. and Surg. Jour.*, March 10, 1898.

cells are more deeply bile-stained. *Microscopically*, the first discoverable changes are spots of insular necrosis in the peripheral zones of the acini (Stengel). These are shortly replaced by proliferation of the interlobular connective tissues. The formation of new-ducts and liver-cells is common.

There is also a so-called Glissonian cirrhosis (perihepatitis) in which the capsule of the organ is surrounded by a dense white fibrinous membrane, which contracts, reducing the size of the liver and altering its shape. This I have described elsewhere (*vide* Chronic Hepatitis, p. 921). Syphilitic cirrhosis of the organ receives special consideration (p. 396).

Etiology.—(*a*) **Atrophic Cirrhosis.**—1. *Alcoholism.*—Freyhan, Osler, and I myself have found this causal factor operative in nearly all cases. Clinical history tends to prove that the stronger the alcoholic beverage (*e. g.* raw spirits) and the larger the amount consumed the sooner cirrhosis develops, although the quantity necessary to produce the disease varies greatly in different individuals. Doubtless by the side of alcoholism all other causes combined are comparatively insignificant.

2. *Spicy foods* are, according to some, classed as predisposing agents. Tiraboschi records a case that had long been induced by the use of spicy foods and by over-eating. In many cases ptomains, the products of malassimilation through faulty digestion, are supposed to be the exciting cause.

3. *Male Sex and Middle Life.*—The cases produced by alcohol occur chiefly in males. According to my experience, females who misuse potable alcohols, particularly the more concentrated liquors, are less susceptible to the poison than males. Two-thirds of the fatal cases occur between the ages of 35 and 50 (Hawkins), although cases have been known to occur at both extremes of life. Toxins of bacterial origin may cause liver cirrhosis. Micro-organisms have also been found in cirrhotic livers, especially the colon bacillus, but no specific causative action can be ascribed to it.

4. It may follow the acute infectious diseases, notably scarlet fever.

5. Certain *chronic diseases* (syphilis—congenital lues in particular—rickets, diabetes, gout, malaria, carcinoma, tuberculosis) that favor the formation of connective tissue are apt to be complicated by cirrhosis, usually partial.

6. *Passive congestion*, secondary to chronic cardiac lesions or to obstructive lung-disease, not infrequently gives rise to hepatic cirrhosis.

7. Fatty cirrhosis results from the abuse of malt liquors in some cases, and is often associated with more or less obesity.

(*b*) **Hypertrophic Cirrhosis** (Hanot).—In most cases there is an absence of recognizable causes. Sex is a strongly predisposing cause, males being the most frequent victims, in the proportion of 6 to 1. It is not uncommon in young adults. In catarrhal jaundice the morbid processes may rarely extend to the liver and there persist, giving rise to hypertrophic cirrhosis. Cases are met with in children, in whom it may follow the acute infectious diseases. Alcohol plays an unimportant rôle in the causation of Hanot's cirrhosis. The disease is most common among the inhabitants of warm climates, and is also hereditary.

(*c*) **Biliary Cirrhosis.**—This form is produced by chronic obstruction of the bile-ducts (see also Obstruction of the Common Duct, p. 900).

Symptoms.—**Atrophic Cirrhosis.**—The symptoms of this variety of cirrhosis may present nothing characteristic as long as the sclerotic

process does not interfere with the portal circulation. In some cases the collateral (compensatory) circulation is maintained throughout the long course and symptoms fail to arise. Among the *prodromal symptoms*, a gradual loss of flesh, anorexia, constipation, a coated tongue, slight jaundice, dyspepsia, and occasionally hematemesis are to be mentioned.

As the obstruction of the portal circulation becomes marked the mucosa of the gastro-intestinal tract is congested, and gives rise to augmenting *nausea* and *vomiting* (most marked in the morning), and *hemorrhages* from the stomach (hematemesis, visible and occult) and intestines (melena), which may be frequent and profuse, but are rarely fatal. Severe hemorrhages may also occur from enlarged varicose esophageal veins. The *tongue* is coated. Uneasiness and even pain may be experienced in the hepatic *area*. Owing to the establishment of a compensatory circulation the superficial epigastric and internal mammary veins enlarge and form about the umbilicus ("caput Medusa").

Hemorrhoids are common and are due to passive congestion of the hemorrhoidal veins. As the disease progresses the *general emaciation* becomes more marked. The face assumes a pinched expression, the tip of the nose has a purple tinge from distended veins; the eyes are sunken, the cheeks hollow, and the skin presents a sallow tint (*hepatic facies*). The failure of the compensatory circulation gives rise to *ascites*, or hydroperitoneum. The *spleen* becomes enlarged. At any stage, although generally in advanced cases, *toxemic symptoms* may develop, due to some poisonous product in the blood of unknown nature: these are violent headache, followed by wild, noisy delirium, convulsions, stupor, and coma. They may occur without jaundice, and have been mistaken for uremia. With or without hemorrhages secondary anemia, more or less profound, is observed. Rogers states that leukocytosis is common.

Fever may be absent throughout the course of the disease, but is often present, and may reach 100°–102° F. (37.7°–38.8° C.).

Examination of the *urine* shows it to be of increased specific gravity, loaded with urates, and containing bile. In a small proportion of cases it is slightly albuminous, and contains casts, though out of 28 urinalyses in cases of cirrhosis Henry discovered the presence of albumin in but one. The amount of urea is constantly diminished, owing to the disturbance of the urea-forming function of the liver. An excess of indoxyl sulphate in the urine is a frequent occurrence. Roque and Cordier found the ascitic fluid to be tuberculous in nature in 22 cases of Laennec's cirrhosis.

The *physical examination* in a typical case of atrophic cirrhosis reveals a distention of the abdomen; there may be also an extreme enlargement of the superficial veins over the surface of the body. An icteroid tint of the skin is present in about 25 per cent. of the cases.

Palpation of the liver and spleen may be greatly interfered with by the large amount of peritoneal fluid present. On withdrawal of the latter, however, the spleen is found greatly enlarged. Palpation commonly detects hardened arteries, and W. W. Ford,[1] in an analysis of 500 autopsies, finds that practically all the cases of beginning cirrhosis of the liver are associated with renal disease and cardiac affections.

The liver may show slight enlargement in the beginning of the disease; but it soon atrophies, and in emaciated subjects with lax abdominal

[1] *University of Penna. Med. Bull.*, Philadelphia, February, 1904.

walls its finely granular or nodular edge can be *felt* above the margin of the ribs. *Percussion* shows its vertical diameter, which normally extends from the sixth interspace to the costal margin, and averages about 4 inches (10 cm.), diminished, especially toward the median line. Posterior dulness begins lower than normally. It must be recollected that the liver is often enlarged in otherwise typical cases. An alcoholic hypertrophic cirrhosis without ascites (Gilbert, 1899), in which there is a marked collateral circulation in the abdominal wall, occurs, and all its symptoms are those of a bivenous hypertrophic cirrhosis.

Fatty cirrhosis, in which the organ is sometimes enlarged, may be latent and remain unrecognized or be discovered on the post-mortem table. In five of my six cases the symptoms resembled those of the ordinary form. Among *complications* of this variety may be mentioned tuberculosis, pleurisy with effusion, and chronic nephritis.

(*b*) **Hypertrophic Cirrhosis.**—In this variety of the disease there is usually an absence of any alcoholic history, and it is apt to be met in young adults and even children (*vide* Etiology). *Moderate enlargement* of the liver may be present before any *digestive disorders* are observed. The latter may be absent, except the presence of slight jaundice and an occasional disturbance of digestion, until late in the course of the disease. Intense jaundice, fever, and hepatic enlargement may then appear, with the rapid development of a grave general condition. The *urine* contains bile-pigment, but the stools are not typical (pale drab or slate colored). Paroxysms of *pain*, resembling mild hepatic colic, may occur at irregular intervals. *Hemorrhages* into the skin from the mucous surfaces (due to passive congestion) are also common. In long-standing cases albumin and tube-casts may be present in the urine. Leucin and tyrosin have also been found, but are not constant. These symptoms are probably due to recent inflammatory infiltration arising in the course of an old cirrhosis. Splenic enlargement occurs, but ascites is exceedingly rare. The cases run an extremely chronic course, and in a patient of mine, a lad of fourteen years, the grave symptoms mentioned above suddenly developed and terminated life after four years of slight, though decisive, attacks of jaundice, with moderate hepatic enlargement. The stools were bilious looking, and hemorrhages from the mucous surfaces frequently occurred. There was a leucocytosis.

Physical examination shows a uniform and progressive enlargement of the organ; the lower border is felt distinctly outlined below the costal margin, its edges being rounded and at times granular.

Percussion shows an increased area of hepatic and splenic dulness.

Late in the disease, in addition to the grave symptoms described above—icterus gravis, high fever, hemorrhages, and the like—serious nervous symptoms, as delirium, convulsions, stupor, and coma, may supervene. The temperature now usually ranges from 102° to 104° F. —38.8°–40° C. (*febrile jaundice*)—although fever may sometimes be absent throughout the course of the disease. Death results either from an intercurrent disease or progressive asthenia.

Hemochromatosis (Opie).—Recklinghausen first called attention to hemochromatosis in connection with cirrhosis. Its association with diabetes mellitus and bronzing of the skin I have previously referred to (p. 415). There are cases, an illustration of which was reported by Opie, in which bronzing of the skin, cirrhosis of the liver, and chronic

interstitial pancreatitis occur without diabetes. Opie's conclusions may be cited: (1) "There exists a distinct morbid entity, hemochromatosis, characterized by the widespread deposition of an iron-containing pigment in certain cells and an associated formation of iron-free pigments in a variety of localities in which pigment is found in moderate amount under physiologic conditions. (2) With the pigment accumulation there are degeneration and death of the containing cells, and consequent interstitial inflammation of the liver and pancreas, which become the seat of inflammatory changes accompanied by hypertrophy. (3) When chronic interstitial pancreatitis has reached a certain grade of intensity, diabetes ensues, and is the terminal event in the disease."

(*c*) **Biliary Cirrhosis.**—*Symptoms and Diagnosis.*—The clinical interest of this form centers principally around the symptoms of the causative condition—chronic obstruction of the bile-ducts—which have been given in detail elsewhere (*vide* p. 900). With the latter may be associated the features of either catarrhal or suppurative cholangitis. *Jaundice* is usually more intense than in the hypertrophic form, particularly during the earlier stages. *Intermittent hepatic fever* is commonly observed. The *physical signs* are similar to those of Hanot's cirrhosis.

The *diagnosis* of biliary cirrhosis rests on the presence of the characteristic features of prolonged obstruction of the bile-ducts, from impaction by gall-stones, a tumor or stricture of the duct, and the like, with slow and gradual, smooth, or slightly granular, hepatic enlargement. It is to be recollected that when obstruction of the gall-ducts becomes complete, or "acute fermentative changes" are set up in the retained bile, the cases may terminate acutely (*e. g.*, in acute atrophy).

General Diagnosis.—(*a*) **Of Atrophic Cirrhosis.**—An assured diagnosis may be based on the following points: 1. A clear history of the most common causes (inebriety, male sex and middle life, rickets, diabetes, gout, malaria). 2. The combined presence of ascites, with Hippocratic facies, and diminution in the size of the liver, as shown by the physical signs. 3. Absence of the characteristic features of acute disease, and the negative character of results from an examination of the heart, lungs, and kidneys. It is to be recollected that the volume of the liver is not invariably decreased, and even may be increased. Hohlweg[1] and Brun[2] suggest the method of testing the tolerance of the liver for levulose, the normal liver not permitting this substance to pass unmodified in the urine.

With the atrophic form of cirrhosis, *chronic peritonitis with effusion* is most liable to be confounded. In the latter disease there are characteristic abdominal tenderness, fever, and usually associated tuberculous lesions of other organs (lungs, kidneys, intestines); but the hepatic facies and clearly indicative history of atrophic cirrhosis are absent. A large peritoneal effusion is in favor of cirrhosis

(*b*) **Of Hypertrophic Cirrhosis.**—The principal diagnostic points are an absence of the usual alcoholic history, slight icterus, extending over a variable and oftentimes long period, paroxysms of pain, mucous and cutaneous hemorrhages, moderate enlargement of the liver and spleen (without ascites), and the development of grave symptoms at any stage—intense jaundice, fever, sometimes marked nervous phenomena.

Differential diagnosis of hypertrophic cirrhosis may be confounded with *carcinoma of the liver*, *hydatid cyst*, *hepatic abscess*, and *fatty cirrhosis*.

[1] *Deutches Archiv. f. klin. Med.*, B. xcvii., H. 5 & 6. [2] *Riforma Med.*, April 18, 1910.

HYPERTROPHIC CIRRHOSIS.

Absence of recognizable causes.
Occurs in young adults and in childhood.
Usually a primary affection.
Jaundice is slight unless grave symptoms develop; there is no cachexia.
Paroxysms of pain. The case runs a slow course, usually lasting many years.
Enlargement is uniform.

CARCINOMA OF THE LIVER.

Hereditary history.
Usually occurs after forty years of age.
Often occurs as a secondary growth.
Anemia is present, and also the development of a typical cachexia.
Pain more constant with rapid emaciation. The case terminates usually within one year.
The liver is irregularly enlarged, and contains umbilicated nodules.

(See Fig. 61.)

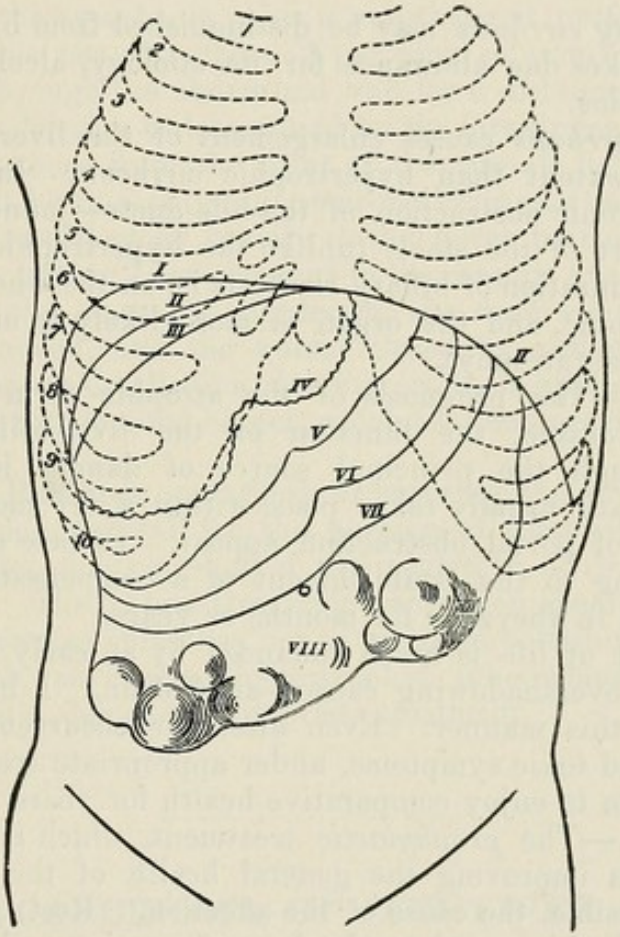


FIG. 61.—Showing approximate enlargement of the liver corresponding to the different diseases described in the text (after Rindfleisch): *I*, position of the diaphragm to the maximum enlargement (carcinoma); *II. II*, normal situation of the diaphragm; *II, III*, relative dulness; *IV*, border of the liver in cirrhosis; *V*, border in health; *VI*, lower border of the fatty liver; *VII*, of the amyloid liver; *VIII*, of carcinoma, leukemia, and adenoma.

HYPERTROPHIC CIRRHOSIS.

History negative as to alcohol. More common in warm climates.

Occurs idiopathically.

Fever, jaundice, and ascites may be present singly or together.
Anemia and emaciation slowly progressive. There is a leucocytosis.
Regular enlargement of the liver. No fluctuation nor thrill.

Aspiration is negative.

MULTILOCULAR HYDATID CYST.

History of ingestion of the embryo of tænia echinococcus with the food.

Simultaneous occurrence in colonies or in others in the vicinity.
No fever, pain, jaundice, or ascites.

Emaciation not marked; no leukocytosis.

On palpation an irregular, fluctuating tumor is felt over the hepatic area, giving an "hydatid thrill."
Aspiration gives a clear, serous fluid, rich in chlorids, and containing hooklets.

Hypertrophic Cirrhosis.	Abscess of the Liver.
Etiology usually negative. May rarely follow acute infectious diseases.	History of dysentery, traumatism, or pyemia.
There are tenderness on deep pressure and paroxysmal pain.	Severe and constant pain; marked tenderness.
Hectic symptoms absent although a continued fever may develop usually late.	Hectic symptoms appear early (fever, chills, and sweating).
Slow course, lasting months or years.	Acute course, lasting a few weeks.
Slow enlargement, regular, or slightly nodulated. No fluctuation.	Rapid development of a fluctuating tumor in the hepatic area.
Aspiration gives negative results.	The aspirating-needle reveals pus.

So-called *fatty cirrhosis* may be distinguished from hypertrophic cirrhosis, if one makes due allowance for its etiology, alcoholism, and the absence of jaundice.

(*c*) *Biliary cirrhosis* causes enlargement of the liver, but to a much more moderate extent than hypertrophic cirrhosis. In the former the symptoms of chronic obstruction of the bile-ducts—jaundice and loss of color on the part of the stools (unlike the hypertrophic form)—are in evidence. The duration of biliary cirrhosis is, on the whole, shorter than that of hypertrophic, and the organ is more likely to undergo terminal diminution in size (atrophy).

Prognosis.—The prognosis of the atrophic form of cirrhosis is decidedly unfavorable, the function of the liver-cells having been impaired, although the principal source of danger is probably the ascites; and death usually takes place within a few months or a year after symptoms of portal obstruction appear. In rare cases the symptoms abate, owing to the establishment of a compensatory circulation, and may remain in abeyance for months or years.

The prospect of life is much enhanced by an early recognition and removal of the overshadowing cause—alcoholism. I have seen a few cures made in this manner. Even after the occurrence of jaundice, hematemesis, and toxic symptoms, under appropriate treatment patients have been known to enjoy comparative health for years.

Treatment.—The *prophylactic* treatment, which is of first importance, consists in improving the general health of the patient and in removing, if possible, the cause of the affection. Rest, graduated exercise, systematic bathing, and regular hours for eating and sleeping should be inaugurated and strictly adhered to. Alcohol, strong coffee, spices, and gastro-intestinal irritants of every nature must be interdicted. H. C. Wood states that tavern-keepers and bartenders who are unable or will not cease using alcohol may greatly prolong life by substituting hard cider for all other drinks. The *diet* should be simple and easily digestible. An exclusive milk-diet has been highly recommended (Semmola).

The *medicinal* treatment is largely symptomatic, no remedy having been discovered to prevent the formation of, or remove, the new-formed connective tissue. The chief object is to deplete the portal system and prevent, if possible, the occurrence of ascites. The bowels should be kept freely open by the use of saline purgatives (concentrated solution of Epsom salts), elaterium, or compound jalap powder. The skin is to be kept active by means of Turkish or Russian baths (under supervision), and in extreme cases by the steam bath or hot pack, employed just short

of the point of exhaustion. The kidneys should also be kept active by the hydragogue diuretics, as potassium acetate, squills, calomel, digitalis in the form of the infusion, or Niemeyer's pill. Klemperer and others have also recently recommended urea as an efficient diuretic, and from 20 to 30 grains (1.29–1.94) may be given in solution.

If the case be syphilitic in origin, potassium iodid should be exhibited.

Ascites calls for free diuresis, diaphoresis, and catharsis; and if not relieved in the course of a few days, tapping should be resorted to.

The operation of *paracentesis abdominis* is free from danger. The bladder having been emptied, a spot over the linea alba about 3 inches (7.5 cm.) above the symphysis pubis is anesthetized preferably by means of the hypodermic use of cocaine (2 per cent. solution) and a trocar is quickly thrust through the abdominal wall for a distance of about 1 inch (2.5 cm.). The distance is determined by the fore-finger, which is placed at the desired distance from the point of the cannula before its insertion. The patient must be in a sitting or semi-reclining position, so as to allow the ascitic fluid to collect by gravity in the lower part of the abdominal cavity. A tube having been attached to the cannula to convey the liquid to a receptacle, the trocar is withdrawn, the fluid allowed to run out, the cannula removed, and the wound closed by antiseptic gauze or a pledget of cotton. A collodion dressing is then applied to the site of puncture, and the abdominal binder, which has been previously applied, is tightened.

Epiplopexy (Roberts's operation), or suturing the great omentum to the anterior abdominal wall for the purpose of establishing a collateral venous circulation, for the relief of the ascites in cirrhosis is useful in advanced cases. The Talma-Drummond operation should be undertaken earlier, but its precise value has not as yet been determined.

Complications, as cardiac hypertrophy, tuberculous peritonitis, or chronic meningitis, demand appropriate treatment.

CARCINOMA OF THE LIVER.

Definition.—A malignant growth of the liver, occurring usually after the age of forty, and characterized by pain, progressive emaciation, cachexia, and the appearance of a nodular mass in the hepatic parenchyma. It may be primary or secondary, though the former is rare.

Pathology.—Histologically, the cells are not distinctive, being identical with those of carcinoma elsewhere; they are epithelial in character, having a small vesicular nucleus and much protoplasm. They are altered greatly by pressure, and vary in shape, being hexagonal, polyhedral, or amorphous. Large giant-cells and spots of pigment known as "brownish granules" are not uncommonly found in the cancerous mass. The so-called colloid cancers are nearly always mucoid, and the cells have undergone a mucoid change; the stroma of connective tissue surrounding the cancer-nests in some instances undergoes hyaline or myxomatous degeneration. In other instances the interstitial tra-

beculæ completely surround the epithelial nests, which are separated by a basement-membrane; this variety is termed *adeno-carcinoma*.

When examined microscopically, *medullary cancer*, either in a large mass (primary) or in secondary nodules scattered throughout the organ, is the most common variety found in the liver. On examining a liver that is the seat of carcinoma, one of two conditions usually presents itself: *First*, the organ may be apparently normal with the exception of one lobe (usually the right), which contains a dense whitish growth of firm consistence, being distinct and sharply defined from the surrounding liver-tissue. On section the tumor is often of uniform density, bluish-white in appearance, and exudes a milk-white fluid known as "cancer-juice," which, when examined microscopically, is found to contain large, nucleated, and irregularly-shaped cells containing free granular matter. The center of the tumor may have undergone liquefaction-necrosis, with the formation of a cyst, or it may be the seat of an abscess. Various smaller nodules may be scattered throughout the organ by metastasis from the primary growth. The *second* and most common condition is secondary carcinoma of the liver, the primary lesion being situated in the mammary glands, pylorus, or the cervix uteri. The organ is greatly enlarged, as a rule. Numerous nodules are scattered throughout, and can usually be seen projecting beneath the capsule, those superficially situated having received the name of "Farre's tubercles." In the center of these nodules characteristic pits or umbilications are often present, caused sometimes by contraction of the interstitial trabeculæ and sometimes by a central softening. On section they are usually grayish-white in color and of firm consistence, although cysts, hemorrhages, pus-cavities, or areas of hyaline and fatty degeneration are often found. The cells are identical with those of the primary growth, and are composed for the most part of cylindric epithelium.

In rare instances carcinoma occurs simultaneously with *cirrhosis* in the same liver, the organ presenting an uneven, nodular appearance, and being slightly increased in size and of firmer consistence than normal. When examined *in situ* the external appearance does not differ materially from that of cirrhotic liver, but on section the whole organ is found to be infiltrated with various-sized cancer-nodules surrounded by bands of cicatricial tissue. In some cases the excess of connective tissue and the amount of contraction are extreme, and the size and weight are reduced below the normal. Eggels[1] has collected 163 cases of primary hepatic carcinoma, and calls attention to the frequent association of atrophic cirrhosis and carcinoma; he regards the cirrhosis as the primary process.

Etiology.—Among the more important predisposing factors are—

(*a*) *Age.*—The disease is most common after thirty-five or forty years of age, although cases are not rare between twenty and thirty-five years. Descroizilles reports the case of a child eleven years old who died with a tumor in the right hypochondriac and iliac region, the autopsy revealing a liver studded with numerous nodules, as was demonstrated microscopically.

(*b*) *Sex.*—Men are more often the victims of carcinoma of the liver (primary form) than women. The secondary variety, however, is slightly more frequent in women, following carcinoma of the uterus or mammary gland.

[1] Ziegler's *Beiträge*, 1901, xxx., p. 506.

(*c*) *Heredity.*—Lichtenstein found an hereditary predisposition in 192 out of 1137 cases (17 per cent.).

(*d*) *Traumatism* may contribute.

(*e*) *Mechanical Obstruction.*—Primary carcinoma of the gall-bladder and bile-ducts commonly follows chronic obstruction by gall-stones.

Symptoms.—There may either be almost no symptoms of carcinoma involving the liver, or its manifestations may be intense and varied according to the extent and location of the growth or growths. Associated gastric symptoms, often due to a primary growth at or near the pylorus, which increase as the disease advances, usually attend. A more or less marked cachexia may be the first noticeable feature. The chief symptoms may be considered in detail, as follows:

(*a*) *Jaundice.*—Discoloration of the skin is often by no means intense, and may be entirely absent. Harley states that true icterus was present in only 6 out of 100 cases seen by him, though few observers agree with his view as to the rarity of this symptom. The reason given for its lack of intensity is that in most cases the growth is situated in the right lobe and does not compress the bile-ducts.

(*b*) *Pain* is usually present to a marked degree. It is dull and boring in character, and localized generally in the right hypochondriac region. In some instances (as in the case of impacted biliary calculi) it may radiate to the right shoulder and the scapular region. It usually increases as the hepatic enlargement progresses, although cases of enormous-sized cancerous tumors of the liver have been known to occur without pain. The character and location of the pain are of diagnostic importance, and will be spoken of under the differential diagnosis.

(*c*) *Ascites.*—When the cancerous growth compresses the portal vessels, and also in cases of cirrhosis with carcinoma, obstruction to the portal circulation occurs, and results in the development of ascites. This may cause distention of the abdominal cavity to such an extent as to occlude the physical signs of hepatic enlargement. The cancerous growth may also invade the peritoneum and cause an effusion. This symptom, however, is not frequent, at least two-thirds of all cases terminating without the appearance of ascites.

(*d*) *Fever* is usually absent until the later stages of the disease. It may then appear and rise to hyperpyrexia (105° F.—40.5° C.), but it is usually moderate in degree, irregular, and intermittent in type.

(*e*) *Cachexia.*—In every case of carcinoma, at some stage of the disease, cachexia develops; when pronounced, it is almost pathognomonic. The destructive effect of the neoplasm, or the toxic substances produced by it, may play a rôle in the causation of the cachexia. At all events there is a distinct increase in the excretion of nitrogen.

(*f*) *Cerebral Symptoms.*—These may be absent throughout. In the advanced stages, however, such striking symptoms as violent headache, mental hebetube, or delirium (less frequently), which may be maniacal in character, appear. These symptoms resemble those of cholemia (*vide* Hepatic Cirrhosis, p. 931). The patient may die in sudden coma.

(*g*) The development of *metastases* (*e. g.*, in the peritoneum).

(*h*) *The Blood.*—There is a decrease in the erythrocytes and the

hemoglobin. *Per contra*, the leukocytes are both relatively and absolutely increased.

Physical Signs.—*Inspection* often reveals enlargement of the superficial veins over the abdomen, and a prominence in the upper epigastric and hepatic regions, varying with the degree of enlargement, may also be seen. In the nodular form and late in the disease, when emaciation has become extreme, elevations that are movable with respiration can be noticed beneath the skin.

On *palpation* the organ can be distinctly felt projecting below the costal margin and extending in some instances to a point below the level of the umbilicus. During respiration (forced) the liver can be felt to move downward and upward, the organ being under the influence of the diaphragmatic excursions. In emaciated subjects the cancer-nodules are readily appreciable, and in some instances the central pits or depressions are palpable, forming a characteristic sign. Cancerous infiltration of the anterior margin is most easily felt, and in enormous enlargements of the organ I have detected them on the posterior surface as well. Rarely the liver is found to be uniformly large. Palpation may also show splenic enlargement, due to passive congestion.

Percussion.—In primary carcinoma (usually found in the right lobe) the percussion-dulness is increased irregularly downward and generally to the right. On the other hand, in secondary growths (usually massive) the nodules are oftener distributed equally throughout the liver. In such cases the area of dulness may extend across the epigastrium to the left hypochondriac region, the heart and other viscera being now displaced. Posteriorly, dulness may extend upward on a level with the fourth rib, and anteriorly downward to the iliac fossa. The organ may now weigh from 15 to 20 lbs. (6.5–9 kgms.), while in the average case the carcinomatous liver weighs from 3 to 6 lbs. (1.3–2.6 kgms.).

Diagnosis.—In forming a diagnosis the family tendency, the history of primary carcinoma elsewhere in the body (stomach, colon, esophagus, pancreas, gall-bladder, uterus, mamma—Rolleston), the age, the localization of the pain in the right hypochondrium, the blood findings, the metastases, the cachexia, and the progressive enlargement of the liver, presenting umbilicated nodules, are the reliable points. The appearance of jaundice or ascites, or both, is confirmatory.

Differential Diagnosis.—Among affections of other organs that are likely to be mistaken for carcinoma of the liver may be mentioned—(1) carcinoma of the *pylorus;* and (2) carcinoma of the *colon and omentum.* The chief diseases of the liver itself apt to be diagnosed as carcinoma are—(*a*) *abscess*, (*b*) *syphilis*, (*c*) *benign growths* (*adenomata, angiomata*), (*d*) *hydatid cysts*, and (*e*) *hypertrophic cirrhosis.*

(1) *Carcinoma of the Pylorus.*—In carcinoma of the pylorus the physical examination frequently shows a hard nodular tumor that is most plainly outlined in the epigastric region. In a typical case, on deep inspiration, the tumor is pressed downward by the liver, but is not pulled upward by forced expiration, as in hepatic carcinoma. In many instances, however, adhesions bind the stomach firmly to the liver, which may be the seat of secondary involvement. The presence of jaundice, as well as the negative results from an examination of the gastric contents, would tend to eliminate pyloric carcinoma.

(2) *Carcinoma of the Colon and Omentum.*—Secondary carcinoma of the intestine affects most frequently the sigmoid flexure. The symptoms of intestinal obstruction arise, constipation being followed by attacks of serous diarrhea due to irritation, and later by the presence of blood in the stools. In carcinoma of the liver, on the other hand, the bile-ducts may be obstructed, causing clay-colored stools, but otherwise the dejecta are normal; the seat of the nodular enlargement and pain is located in the right hypochondrium. Jaundice and ascites are absent in carcinoma of the colon. The tumor, if palpable, in the latter condition is more movable and is less under the influence of the diaphragm. It does not give an absolutely flat percussion-note, as does hepatic carcinoma. Carcinoma of the omentum is usually secondary. The absence of small movable tumors in the umbilical, lumbar, or hypogastric regions, ranging in size from that of a pea to a walnut, aids in the elimination of carcinoma of the omentum. As the latter affection advances the abdomen becomes distended and painful to the touch, the bowels are obstinately constipated, and the physical signs reveal the presence of an effusion which, when aspirated, is generally serous, but sometimes bloody. Microscopic examination may possibly reveal the presence of cancer-cells, though their recognition is difficult. The liver, unless primarily involved, is not enlarged, and cachexia does not usually appear until late.

From *hepatic abscess* the points of differentiation are—

Carcinoma of the Liver.	Hepatic Abscess.
Is often hereditary. There is a history of a primary growth or chronic irritation.	There is a history of traumatism or of intestinal ulceration, as in dysentery.
Occurs usually after the age of forty.	Occurs at any age.
Jaundice is rare.	Jaundice is sometimes present.
Fever is absent or slight.	Hectic temperature, chills, and sweating.
Cachexia is present and almost pathognomonic.	Anemia may be present, but *never cachexia.*
Pain is dull and boring in character, and more constant.	Pain is sharp, lancinating, and paroxysmal.
A nodular, umbilicated tumor or tumors may be detected.	A fluctuating tumor may sometimes be detected below the costal margin.
The enlargement is downward.	The enlargement usually upward.
The duration is a few months to one year.	The duration is usually a few weeks.
Microscopic examination reveals disintegrated liver-cells, cancer-nests, and in some cases the micro-organisms of suppuration.	The microscope reveals pus, liver-cells, staphylococci and streptococci, the bacillus coli communis or the amœba coli.

Benign Growths (*Adenomata*, *Angiomata*).—Occasionally growths are detected in the liver, and may occur at any age; when these are present at or about the age of forty, they may be mistaken for carcinoma. The absence, however, of a primary growth in some one of the other viscera, together with the duration of the growth and the absence of cancerous cachexia, would tend to differentiate them from cancerous involvement. An examination of the blood may be of service, leukocytosis being more common in carcinoma.

The **prognosis** is invariably fatal, the disease terminating rapidly in from a few months to a year. The most rapid course is run by secondary carcinoma of the organ. In exceptional cases growths favorably situated have been removed without recurrence.

Treatment.—The treatment is, with rare exceptions, purely sympto-

matic. An easily digested, nutritious diet should be given, together with active stimulation to support the system. The pain may be relieved by the free use of morphin, given by the mouth, rectum, or hypodermically. For the nausea and vomiting that are apt to supervene the carbonated waters, cracked ice with champagne, or repeated doses of creasote (beechwood), dilute hydrocyanic acid, or wine of ipecac (2 minims—0.133—every hour until relieved) may be given. If violent delirium should occur during the later stages of the disease, cold compresses to the forehead or vertex, and bromids and chloral hydrate given in rectal enemata, may prove efficient.

OTHER NEW GROWTHS IN THE LIVER.

(*a*) **Angioma, Adenoma, and Cyst.**

Occasionally, benign growths occur in the liver, and often with an absence of symptoms unless their increase in size gives rise to mechanical obstruction. One of the most common of these is angioma, which is often found in the livers of old people. Angiomata consist of tortuous and dilated capillaries in the hepatic connective tissue; they rarely attain to a size larger than a crab-apple, and usually cause no symptoms. Although most common in adults, they have been known to occur in children.

Adenomata and cystomata may also occur in the liver. They are both benign growths. The former is of the tubular variety, consisting of connective-tissue nests lined with cylindric epithelial cells. Von Bergman removed a portion of a tuberous adenoma of the liver with perfect recovery and non-recurrence of the growth. Cysts are quite rare. Lippmann,[1] who searched the literature, found reported 3 retention cysts, 9 cystic adenomas, 1 lymph cyst, and 3 cases that could not be classified.

(*b*) **Sarcoma.**

Of the many varieties of sarcomata, those occurring most commonly in the liver are the small and large round-celled and the melanotic variety, the latter often being secondary to sarcoma of the choroid coat of the eye. These grow rapidly, causing a widespread destruction of the liver-structure, with a change in the size and shape of the organ that is often demonstrable by palpation. E. R. Axtell reports a case in which at the *postmortem* the upper two-thirds of the liver revealed an entire absence of hepatic structure, and consisted of three tumor-masses. On section the tumor is seen to be of firmer consistence than the surrounding liver-tissue, and presents a dark, grayish-white, striated appearance. If the growth be of the pigmented variety, patches of a deep black or of different shades of pigment may be scattered throughout the mass. Metastasis is rapid and widespread (lungs, kidneys, heart, skin), as is shown by the fact that other organs are invariably found involved at the time of the growth and development of the sarcoma in the liver. Melanosarcoma may, in rare instances, appear as a primary growth, and attain to a considerable size, as shown by a case reported by Bramwell and Leith.

[1] *Deutsche Zeitschrift für Chirurgie*, February, 1900.

The *symptoms* are those of mechanical obstruction, and consist of gastro-intestinal disturbances due to passive congestion, edema, and ascites. Anemia and emaciation may become marked late in the disease, but cachexia does not develop. The passage of an intensely dark-colored urine (melanuria) has been noted in some cases. Secondary nodules may appear on the skin-surface.

The *diagnosis* can often be made from the primary growth (melanosarcoma of the choroid or sarcomata of the lymphatic glands) and from the rapid development of the tumor. From *carcinoma* of the liver melanosarcoma may be distinguished by the presence of ocular symptoms, particularly blindness of one eye, by the rapid widespread metastasis, the melanuria, perhaps, and by the absence of a true cancerous cachexia.

The *prognosis* is absolutely fatal, and the *treatment* merely palliative.

X. DISEASES OF THE SPLEEN.

DISEASES of the spleen are mostly secondary to other diseases, the consideration of which embraces an appropriate description of the associated splenic disorders. The intimate relation between the spleen and blood accounts for the frequency with which this organ is involved in many of the blood-diseases.

DISLOCATION OF THE SPLEEN.

(*Floating Spleen.*)

Etiology.—This may be either congenital or due to the increased weight of an enlarged spleen, to tight-lacing, to relaxation of the ligaments, or to traumatism, and is often met in splanchnoptosis. Carcinomatous enlargement of the left lobe of the liver caused it in my case.

Symptoms.—The symptoms are vague, and are the result of pressure. Distinct symptoms of gastro-enteritis and neurasthenia may result from a wandering spleen. By *physical examination* we discover with the touch the spleen as a mobile tumor pendant from the left hypochondrium; the tumor is superficial, blunt-edged, and notched on its anterior border, and may be replaced by the hand in its normal position. On percussion over the splenic area the normal dulness is found to be absent.

Diagnosis.—It is important to distinguish between *floating spleen* and simple enlargement; also between the former and *movable kidney*.

The **prognosis** is guarded as to cure, though favorable as to life. Twisting of the pedicle has been followed by strangulation, with the development of intense pain and other alarming symptoms (necrosis). Intestinal obstruction, due to pressure, may appear.

The **treatment** must be mechanically supportive, consisting of pads and bands. Splenectomy has given excellent results.

SPLENIC HYPEREMIA.

Acute or active hyperemia may be found as the result of the acute infectious diseases, giving rise to the *acute splenic tumor*, or as the result of amenorrhea, or of injuries and inflammation (*circumscribed hyperemia*). The organ is uniformly enlarged (except in the last-named cases), and is darker in color and softer in consistence; the capsule also is tense. This condition merges insensibly into *acute splenitis*.

Chronic or passive hyperemia is due to some mechanical obstruction of the portal circulation caused by tumors, cardiac, hepatic, and pulmonary disease, and pylephlebitis. The spleen is enlarged, firm, dark-red in color, and the capsule is somewhat thickened.

The *symptoms* are vague, and may consist of simply a sense of weight, fulness, and pressure, and some tenderness in the left hypochondrium. In cases of extravasation of blood and rupture of the spleen the symptoms of intestinal perforation, hemorrhage, and collapse may supervene.

On *physical examination* the edge of the spleen may be palpated below the margin of the ribs. The percussion-dulness is increased in area, especially downward and forward, and may encroach upon the slightly curved umbilico-axillary "resonant line."

The detection of acute or chronic splenic hyperemia (enlargement) is often of invaluable aid in the diagnosis of the causative disease.

The *prognosis* and *treatment* are embraced in those of the disease causing the congestion. The *x*-rays have been used.

SPLENITIS.

Definition.—This term comprises acute and chronic (hypertrophic) proliferative splenitis and suppurative inflammation.

Pathology.—Next to the kidneys, the spleen is the favorite seat of metastatic inflammation and *embolic infarction*. Splenitis, due to a benign embolus originating in the left side of the heart or from the aorta above the splenic arteries, is usually circumscribed to a zone of sero-hemorrhagic infiltration about the resultant infarct. The latter is hemorrhagic at first, and later becomes particolored or *mixed*, and is of a yellow color, owing to partial fatty degeneration; still later it may become whitish and remain as a wedge-shaped (the base being peripheral), cheesy (necrotic softening), or even calcareous mass or as a fibrous cicatrix. Infection of the infarcts by pus-micrococci leads to the development of *small abscesses*, and the trabeculæ surrounding the latter may give way until finally one large pus-sac may be formed. Embolism may follow primary splenic arterial or venous thrombosis.

Perisplenitis generally follows, and sometimes with adhesions attached to adjacent hollow organs, as the stomach and colon, through which the perforating abscess may discharge its purulent contents. An unfortunate termination is the bursting of the abscess into the peritoneal cavity; a more fortunate ending results in an external opening. In acute splenic tumor there is an active congestion, with round-cell infiltration and some proliferation of the splenic cells. The spleen is moderately enlarged, dark, soft, pulpy, and friable.

In cases of intense vascular engorgement, as in the acute splenic tumor of severe typhoid fever, intermittent fever, and epilepsy (during the paroxysm), *hemorrhagic extravasation* may occur, and there may finally be even a rupture of the capsule and a passage of the blood into the peritoneal cavity. In chronic splenic tumor there is a persistent hyperplasia of the splenic cells, and frequently also of the trabecular cells, minus the acute engorgement. *Cirrhosis of the spleen* (chronic interstitial splenitis) differs characteristically from that of other organs (as the liver and kidneys) in that there is *enlargement instead of contraction.* Added to the increase in the size of the spleen, there are in both forms of chronic splenitis thickening of the capsule, patches often of old perisplenitis, and a slaty color of the tissues, with more or less pigmentation.

Etiology.—The disease probably never starts primarily in the spleen itself. *Acute proliferative* or *hyperplastic splenitis* (*acute splenic tumor*) is seen as the result of the acute infectious diseases (typhoid, typhus, relapsing, malarial fevers.) *Chronic proliferative splenitis* (*chronic splenic tumor*) is due to chronic malarial infection or repeated acute attacks, to splenic anemia, chronic passive congestion of the spleen, and leukocythemia. The leukemic spleen represents a somewhat different form of chronic proliferative splenitis from the ordinary forms. *Acute suppurative splenitis* (*abscess*), either diffuse or circumscribed, is usually secondary to infectious (pyogenic) emboli, as in ulcerative endocarditis and pyemia. Again, as the result of simple valvulitis of aortic thrombosis, *embolic infarction* of the spleen may be found, which may soften and break down in abscess-formation from subsequent infection. Abscess of the spleen may also follow traumatism, perforation of a gastric ulcer, and the extension of adjacent inflammation.

Symptoms.—These are indefinite or absent in most cases. Usually there is no pain or tenderness unless perisplenitis exists. Considerable enlargement of the spleen may be attended with a *sense of weight, tension,* or *distress* in the left hypochondrium, and perhaps by slight *dyspnea.* Any *suppurative fever* present will most probably be disassociated from the idea of abscess of the spleen, provided the local signs of pus be absent. *Sudden pain* appearing in the gastric region, followed by the *vomiting* of *pus* and *blood,* in the course of an infectious disease, with *splenic enlargement,* may be due to the rupture of an abscess of the spleen. *Ascites* may also be present.

The **physical examination** may reveal some bulging on inspection, and a fluctuating tumor may be palpated. The enlargement may be sufficient to enable one to feel the notch in the spleen, and also the anterior and lower borders, reaching even to the umbilicus. The percussion-dulness is correspondingly increased.

Diagnosis.—This may be made from a consideration of the physical signs in conjunction with a study of the primary disease. In cases in which pus is suspected an exploratory puncture may clear the diagnosis. The splenic inflammation is rather an aid to diagnosis than a condition essentially needful of recognition in itself.

Differential Diagnosis.—Acute suppurative splenitis might be mistaken for *gastric* or *pancreatic disease;* but the previous history in the former, as contrasted with that of the latter affection, conjoined with the

local symptoms that are more or less characteristic of the organ involved, will generally furnish an accurate means of differentiation.

The huge enlargements of chronic splenitis may be confounded with *hepatic, renal, omental,* or *ovarian growths.* Here a careful, discriminating observation of the constitutional state and of the physical signs is requisite for a diagnosis. Splenic enlargement must not be assumed when a *large pleural effusion* on the left side is causing the depressed lower border of the organ to be felt. Finally, *fecal accumulation in the splenic flexure* of the colon may be mistaken for moderate enlargement of the spleen. The former gives an irregular, doughy tumor, tympanites, vomiting, and a history of constipation alternating sometimes with diarrhea; there is no increase in the splenic area of dulness.

Prognosis.—This will depend upon the primary systemic condition. Abscess is a grave complication, the danger consisting of rupture and fatal peritonitis. Even in acute splenic tumor of a violent type there may be a hemorrhagic extravasation so severe as to burst the capsule. Chronic splenitides are not in themselves grave disorders.

Treatment.—This is to be directed mainly at the causative condition. Quinin and arsenic are often useful in the malarial form, and the chalybeates, iodids, and ergot have been recommended for the various chronic splenic enlargements. Strapping the affected side affords comfort. Abscess must be treated by splenotomy and drainage. Splenectomy may be useful in certain cases of simple hypertrophy, but records show only about 20 per cent. of recoveries from the operation. The state of the patient must be well considered. Splenectomy is never justifiable.

AMYLOID DEGENERATION OF THE SPLEEN.

(*Sago Spleen.*)

This occurs as a part of the cachectic condition attending amyloid degeneration of other organs (liver, kidneys). The condition develops in the course of cases of prolonged and wasting discharges (phthisis, empyema, suppurative ostitis, syphilis, chronic peritonitis, chronic enterocolitis). The spleen is, as a rule, greatly enlarged, putty-like, and rotund. The capsule is tense and glistening. There are two forms of waxy degeneration—namely, the so-called "*sago*" *spleen* and the *diffuse waxy* or *lardaceous spleen.* In the former the Malpighian bodies are chiefly affected and appear on section like sago-granules; in the latter the whole splenic pulp, and even the trabeculæ, are degenerated, and on section the spleen appears pale, smooth, and homogeneous (boiled-ham appearance). This may be but a late stage of the "sago" spleen. The spleen gives a characteristic reaction with iodin.

The *symptoms* are those of general cachexia, and the *diagnosis* rests upon the detection of an enlargement of the organ associated with evidences of amyloid disease in other organs.

The *prognosis* is unfavorable, and the *treatment* does not differ from that indicated for the underlying and causative disease.

MORBID GROWTHS OF THE SPLEEN.

THE principal new-growths are the granulomata, as tubercles and syphilitic gummata; also secondary carcinoma, melanotic sarcoma, and hydatid and other cysts. Lymphadenoma (*e. g.*, in leukemia), angioma, and fibroma may be included among tumors of the spleen.

These affections of the spleen are all of rare occurrence, and are not readily, if at all, discoverable during life. They are of no clinical or therapeutic interest apart from the general or primary disease. It may be stated that *carcinoma* of the spleen is always secondary; it may be diagnosticated by a physical examination, showing the organ to be enlarged, with the unmistakable signs of the primary carcinoma, as of the stomach. Secondary sarcoma is more common, and is recognized by an irregular enlargement and the presence of a primary tumor.

Syphilitic gummata of the spleen are often associated with amyloid degeneration and enlargement.

RUPTURE OF THE SPLEEN.

THIS may occur as the result of an intense hyperemic engorgement, both in splenitis from the rupture of an abscess and from traumatism. In the acute splenic tumor of typhoid fever, in malaria, and during an epileptic paroxysm, rupture of the capsule has been known to occur on account of the extravasation of blood. The *symptoms* are usually mistaken for those of intestinal perforation with internal hemorrhage. The *treatment* is surgical, though palliative pending the surgeon's arrival.

XI. DISEASES OF THE PANCREAS.

ACUTE PANCREATITIS.

THREE varieties of acute pancreatitis—hemorrhagic, gangrenous, suppurative—will be described below, following the usual classification, but it is to be recollected that, in the majority of instances, these are indistinguishable clinically, and represent but different stages of a single disease.

HEMORRHAGIC PANCREATITIS.

Pathology.—The pancreas is enlarged, usually firm, and somewhat chocolate-colored. Irregular areas show the circumscribed as well as the diffused form of hemorrhagic infiltration of the interstitial fat-tissue, with thrombosis of the pancreatic veins in some cases (Day). There is also an infiltration with round-cells of the interlobular tissues. Some cases are examples of degeneration (non-inflammatory). The gastro-intestinal mucosa may be hyperemic, ecchymotic, or in a slightly catarrhal state. Evidences of a localized peritonitis (*peripancreatitis*) are not frequent. Hemorrhage with inflammation is to be distinguished from true hemorrhagic pancreatitis. Opie and Meakins consider hemorrhagic pancreatitis to be primarily a necrosis, the inflammatory changes being secondary.

Disseminated fat-necrosis is quite commonly associated with hemorrhagic pancreatitis. Small areas of a peculiar (tallow-like) substance, ranging from the size of a miliary tubercle to that of a pea or even larger, are found scattered in the fatty interlobular pancreatic tissue in the omentum, mesentery, and sometimes in the abdominal fat. H. U. Williams and Kätz and Winkler, from experimental researches, conclude that probably the fat-splitting ferment is capable of causing changes similar to fat-necrosis. H. Coenen believes that the necrosis is the result of autolysis from activation of the pancreatic secretion by bacterial action, the bacteria penetrating into the pancreas in infected bile or intestinal juice.

In infectious fevers the pancreas may show diffuse, parenchymatous, and granular degenerative changes. Chiari has pointed out that *post-mortem* digestion is frequent in the pancreas.

Etiology.—Most of the cases reported have occurred in *men*, and in persons past *fifty years* of age. An especial *predisposition* to the disease seems to be the result of cases of severe and obstinate dyspepsia (gastro-duodenal), alcoholism, obesity, glycosuria, and traumatism. Hemorrhage into the pancreas may cause the disease. Opie emphasizes the etiologic importance of gall-stone and gall-duct disease, and suggests that pancreatitis may be the result of the entrance of bile into the pancreatic duct. It is seen occasionally *post-mortem e. g.*, in acute tuberculosis and the specific fevers (metastatic infection). The *direct cause* is probably an infection through the ducts of the gland, although when due to impaction of a calculus bacterial infection may be absent. Flexner[1] injected acids. alkalies, and bacterial cultures into the duct of Wirsung and the interstitial tissue, and produced acute pancreatitis.

Symptoms.—The *onset* is sudden and violent. It is characterized by *excruciating, deep-seated pain*, usually in the epigastrium or between the xiphoid and umbilicus. There are also *nausea* and *severe retching* and *vomiting, constipation*, and speedy *collapse*, ending fatally within a few days (second to the fourth—Fitz). The vomitus may consist at last of slimy mucus or dark blood. *Fever* is generally slight at first, though it may touch 103° or 104° F. later. *Dyspnea* and a rapid, feeble *pulse*, with jactitation and *marked anxiousness* or an *afebrile delirium*, may perhaps be present. In some cases there may be *diarrhea*, with thin and watery stools containing free fat. Instances may be repeated in which, owing to the coincident presence of gall-stones, there may be *jaundice* and *colicky pains* over the right hypochondrium. The jaundice, however, may sometimes be due to swelling of the head of the pancreas, which presses upon the bile-duct. *Tympanites* occurs in a majority of the cases. *Hiccough* and *albuminuria* have also been noted. The pain and collapse may be due either to a circumscribed peritonitis or to pressure upon the solar plexus. Cyanosis of the face and abdominal walls is common (Halsted). Localized tenderness and moderate rigidity above and to the right of the umbilicus are sometimes present.

Diagnosis.—This is at all times difficult, since many or all of the symptoms enumerated may be present in other affections. A careful inquiry into the previous history is important. The sudden development of an intense, deep-seated pain in the epigastrium, followed by vomiting, collapse, abdominal distention, with circumscribed resistance in the epigastrium, and the presence of constipation and slight fever,

[1] "Experimental Pancreatitis," Festschrift in honor of William Henry Welch.

should point strongly to hemorrhagic pancreatitis. The detection of free fat in the dejections, and the discovery of scattered points of tenderness, are significant. Cammidge's reaction is corroborative evidence where a pancreatic disorder is suspected. Other affections may give a positive response (pneumonia, appendicitis with diffuse peritonitis). Cammidge[1] advises that the urine examination be controlled by an examination of the feces.

Differential Diagnosis.—The temperature is apt to be higher and the pain and tenderness less localized and more constant in *peritonitis*. Fecal vomiting would indicate *obstruction of the bowel*. Here also we may determine the patency of the bowel by injection or inflation. Intestinal obstruction is of comparatively rare occurrence in the epigastrium, where the pain and distention of acute pancreatitis are localized; there are likely to be present more marked and general tympany, including the flanks, and a circumscribed distention of the intestinal coils.

In perforating gastric or duodenal ulcer there is a history of pain after eating, hemorrhages from the digestive tract, and of anemia.

Corrosive poisons may be excluded by the history of the case and by an examination of the mouth and vomitus. *Hepatic colic* must be excluded; the pain is intermittent, and referred more to the right side than in pancreatitis. There is in pancreatitis also an early collapse.

Acute gastro-duodenitis is characterized by fever, by a history of injudicious eating, followed by mild inflammatory symptoms within a few hours, and by an absence of the sudden prostration and collapse so common to hemorrhagic inflammation of the pancreas.

Prognosis.—Acute hemorrhagic pancreatitis in most cases ends in death. In view of the ease with which the disease may be overlooked it is quite possible that certain cases of milder type may recover; in these the recovery has been said to follow a different affection. Osler reports a case diagnosticated as one of intestinal obstruction in which abdominal section was performed and recovery followed. Thayer and Korte have also reported cases of cure in which a celiotomy decided the diagnosis.

Treatment.—The treatment as for shock by the use of external heat and of warm saline injections, hypodermics of morphin, atropin, strychnin, and of diffusible stimulants may probably be of some avail. Early operation with a view to establishing free drainage is the important factor in the treatment (Ochsner).

SUPPURATIVE PANCREATITIS.

Pathology.—The suppuration may be diffuse, with numerous small abscesses; more commonly a single abscess exists in the head or body of the pancreas, which may be enlarged and its structure extensively destroyed. The abscess may communicate with peripancreatic areas of suppuration, or it may evacuate either into various organs (duodenum, peritoneal cavity) or externally. Pylephlebitis and hepatic abscess or pyemia may follow. A disseminated fat-necrosis is sometimes found.

Etiology.—Most of the cases occur in adult males *prior* to fifty years of age. Intemperance, trauma, and dietetic errors are among the *predisposing* causes. Infection takes place through the ducts, or from extension of neighboring septic foci. Cholangitis, due to gall-stones, may extend to the pancreatic duct, producing suppurative pancreatitis.

Symptoms.—These may be *acute*, *subacute*, or *chronic*. Acute

[1] *Brit. Med. Jour.*, May 19, 1906.

cases occur less frequently than the latter. **Acute suppurative pancreatitis** usually begins *suddenly*, with *severe epigastric pain*, *vomiting*, *hiccough*, *chills*, and an *irregular pyemic temperature*, *progressive tympanites* (at times limited to the left half of the abdomen), and perhaps acute splenic enlargement. *Constipation* may be followed later by *diarrhea* (sometimes fatty), and slight jaundice or glycosuria may appear. Brugsch and Koenig [1] report a case in which the feces showed a decided diminution in the absorption of fats. *Prostration* is generally great, and death may set in within one week from the onset.

Not seldom, however, the *course* is prolonged to three or four weeks, the symptoms persisting with progressive emaciation and final exhaustion. *Rupture* of the circumscribed peritoneal abscess, evidenced by copious dejections in which the sloughing pancreas has been found, and rapid diminution in the size of the abdomen, may take place.

Again, the onset may be less severe, and yet the case progresses steadily downward with little pain, slight suppurative fever, anorexia, anemia, and gradually increasing debility, lasting for months or even a year, and ending in anasarca and death. A tender swelling is often *palpable*.

Diagnosis.—A limitation of the pain and a tender mass to the epigastrium, irregular fever, and the evidences of sepsis (leukocytosis, suppurative type of fever) are probably all that can be relied upon in arriving at a diagnosis. In fact, the diagnosis is hardly made *antemortem*.

For the differentiation from *circumscribed* peritonitis, *perforative gastric ulcer*, and *obstruction of* the *bowel*, *vide* p. 951.

The **prognosis** is unfavorable and the **treatment** surgical.

GANGRENOUS PANCREATITIS.

Pathology.—The pancreas may be found in various stages of necrosis, depending upon the duration of the disease. It may be a dark-brown, soft, friable, shreddy, and putrid mass, with areas of hemorrhagic infiltration and yellow softening, and surrounded by a dirty-greenish, thin, purulent, and ichorous fluid. In cases lasting for from three to seven weeks the gland may be found completely sequestrated, lying in the omental cavity as a small, thin, brownish-black, shreddy, and foul-smelling detritus, soaked in a purulent fluid. The peri- and para-pancreatic tissues are usually involved with acute peritonitis. Splenic thrombo-phlebitis is commonly associated, and disseminated fat-necrosis is frequently seen.

Etiology.—Males and females are equally liable, and persons past thirty years of age are most commonly affected. Hemorrhagic pancreatitis is the most frequent antecedent of the gangrenous form. The disease may result also from perforative ulceration of the gastro-intestinal or biliary tract, or from the extension of a catarrhal inflammation of those tracts into the pancreatic duct (Fitz). Traumatism is a cause.

Symptoms.—These are essentially the same as those of hemorrhagic pancreatitis. The *course* may last longer, however, so that death may not occur until the second or fourth week, preceded by symptoms of collapse. The necrotized pancreas may be discharged per rectum, followed at times by recovery. An epigastric tumor usually appears.

[1] *Berlin. klin. Woch.*, December 25, 1905.

CHRONIC PANCREATITIS.

Pathology.—The pancreas is indurated from an increased development of interstitial fibrous tissue. The glandular substance may be nearly obliterated, and, owing to pressure upon the duct of Wirsung, pancreatic cysts may be formed. Interstitial hemorrhages and peripancreatic adhesions may be present. In *chronic suppurative pancreatitis* there may either be small circumscribed abscesses or one large pyogenic cyst.

Etiology.—Chronic pancreatitis may be due to several attacks of the acute disease or to chronic inflammation of the pancreatic duct, often secondary to gastro-duodenal catarrh. Since the distribution of inflammation often corresponds to that of the lymphatics the infection may be lymphatic- rather than duct-borne. Persistent inflammation of contiguous structures may excite it. The majority of cases, however, arise from *disease of the biliary passages*, especially cholelithiasis. Chronic alcoholism, syphilis, tuberculosis, and trauma probably lead to this disease. The condition may be limited to a part of the organ.

Symptoms and Diagnosis.—The symptoms are hardly indicative of the disease. The symptoms of *chronic gastric catarrh*, frequently attended by *diarrhea* and *large stools*, may compose the early clinical picture. Later there may be paroxysms of *deep epigastric pain*, *slight fever*, *marked anemia*, with *great anxiety* and *faintness*, occurring at irregular intervals. Some *ascites* and *jaundice*, due to pressure, may be observed. The detection of *free fat* in the dejections (*steatorrhea*) and undigested muscle-fibers (*azotorrhea*) in the absence of diarrhea, are of great semiotic importance in the interlobular form. It has been suggested that the effect of the administration of the pancreatic ferments may be taken as a valuable diagnostic aid. On the other hand, the occurrence of *glycosuria* without pancreatic disturbance of digestion in the intestines (*e. g.*, in interacinar pancreatitis) and *lipuria* would be of distinct diagnostic value. The presence of glycosuria probably indicates an extreme degree of destruction of this gland (Fitz). Walko regards the muscle-nucleus test as being of diagnostic value. Stadtmüller advises Sahli's glutoid capsule test. Klieneberger[1] advocates the casein test for trypsin.[2] The Mayos found 81 per cent. of their cases accompanied by gall-stones. A *cachectic* appearance may be associated. *Circumscribed resistance* on palpation in the pancreatic area has been noted. Evidences of hepatic cirrhosis or of chronic renal and arterial disease may be present.

Differential Diagnosis.—

CHRONIC PANCREATITIS.	PANCREATIC CARCINOMA.
History of acute onset in some cases.	More gradual onset of symptoms.
Absence of, or gradual and incomplete, obstruction to flow of bile.	Evidence of complete obstruction of rapid development.
Distention of gall-bladder, gradual and of moderate degree.	Gall-bladder distention more rapid and and often marked.
Azotorrhea less common.	More common.
Enlarged cervical glands absent.	Present in certain proportion of cases.
Emaciation and weakness less marked and of slow development.	Rapid, becoming pronounced, and characteristic of the disease.

The **prognosis** is grave. The greater portion of the gland may

[1] *Medizinische Klinik*, Berlin, January 16, 1910.

[2] For the methods of obtaining the pancreatic secretion from the duodenum, see special works on diagnosis.

become functionless, however, as the result of progressive fibrous change, without much impairment of the health.

Treatment.—The major treatment is *dietetic*. Fat and starches, since they demand the pancreatic ferment for their conversion, are to be interdicted, or, if permitted, are to be, so far as may be, artificially digested by the administration of tablets of pancreatin and soda (gr. v–x—0.324–0.648) fifteen or twenty minutes after meals. Malt diastase, combined with alkalies, should also be tried. Minced pancreas promotes the digestion of fat (Abelmann). Fey reports recovery in syphilis of the pancreas under treatment with mercury and potassium iodid. The surgical treatment consists in the relief of tension by opening and draining the gall-bladder (Deaver).

PANCREATIC HEMORRHAGE.

(*Pancreatic Apoplexy.*)

It is only in recent years that this fatal affection has been isolated and defined, and mainly through the observations of Fitz, Zenker, and Draper.

Pathology.—The pancreas may or may not be enlarged; it may also be soft and friable. The hemorrhage is apt to occur into circumscribed areas of the gland—*e. g.*, its head, the interstitial and subperitoneal tissues. Extensive hemorrhage may be found in the omentum, transverse mesocolon, in the retroperitoneal fat-tissue, and adjacent mucous surfaces. Secondary reactive inflammations and necrosis are commonly noted.

Etiology.—*Slight hemorrhages* into the pancreas may be found secondary to excessive chronic passive congestion, to hemophilic or purpuric cases, and acute infective diseases. These have, however, no clinical import. The etiology of *marked hemorrhage* into the pancreas is not known. Most cases have occurred in males (in 25 of 34 instances collected by the writer), and in adult or advanced middle life (the age in 13 of 30 cases having been over forty-five years). In the majority of cases the previous health was apparently good. Traumatism may be a direct cause. Again, some local vascular lesion (*e. g.* necrosis), superinduced by alcoholic habits or a rich diet in an atheromatous person; or some corrosive action of the pancreatic secretion may operate as causes. It has been ascribed to the action of the glycerin set free during the fat-splitting process in the production of fat-necrosis. There was a history of chronic alcoholism in 12 of 18 cases (66.6 per cent.). Severe *symptomatic* pancreatic hemorrhage is dependent on a variety of primary affections of the gland—*e. g.*, acute pancreatitis and carcinoma.[1]

Symptoms.—The patient may have been in apparently robust health when the attack comes on with *sudden* and *startling* gravity. The most prominent early symptom is *intense epigastric pain*, together with a sense of *constriction*. *Nausea* and *vomiting* may be associated, and the latter is usually obstinate and gives only temporary relief. *Tympanites* may also occur. There are early and constant *general evidences* of *internal bleeding*—an anxious countenance, restlessness, depression, yawning, pallor, cold sweat, a lowered surface-temperature, and a small, rapid, and weak pulse. *Prostration* and *syncope* follow, and death ends the case in from half an hour to twenty-four hours. Death is caused by reflex paralysis of the heart, due either to some coincident vascular affection, or to pressure, perhaps upon the solar plexus and semilunar ganglion (Zenker).

[1] "Pancreatic Hemorrhage," *Jour. Amer. Med. Assoc.*, Dec. 2, 1899, by the writer.

My table includes 24 cases, exclusive of 16 reported by Fitz, in which the condition led to speedy death from shock or from compression of the solar plexus. Owing to its "idiopathic" character in many cases, and quick destruction of life, pancreatic hemorrhage assumes intense medico-legal importance. A few cases reach death more gradually.

Diagnosis.—Given the suddenly developed signs of a concealed internal hemorrhage, with pain referred distinctly to the epigastrium, and vomiting and rapid collapse, a *probable* diagnosis may be made.

Treatment.—This consists in relieving the pain and in overcoming the collapse by free stimulation. An exploratory operation is advisable.

CARCINOMA OF THE PANCREAS.

Pathology.—Primary carcinoma is the more frequent variety. It is of the scirrhous form in most cases, and usually involves the head of the gland, which may attain to the size of a child's head. Not rarely the adjacent organs are found affected, either by direct or metastatic extension of the disease, or by the pressure of the growth; the liver, peritoneum, stomach, portal vessels, bile-ducts, and aorta may thus be involved. The pancreatic duct may be occluded, so as to form retention-cysts. In 1000 autopsies at the Johns Hopkins Hospital there were 5 cases of secondary carcinoma of the pancreas. Simple extension of carcinoma of the stomach or of the duodenum may involve the pancreas.

Etiology.—*Men* from forty to sixty years of age are most liable to carcinoma of the pancreas, though it has been met with in the *new-born*. Mirallié has collected 113 cases of primary carcinoma of this viscus (Fitz).

Symptoms.—These are scarcely ever sufficient to indicate the disease with certainty. There are usually a *stubborn dyspepsia*, a *progressive loss of flesh* and *strength*, *anemia*, and a *dull*, or sometimes *neuralgic*, *epigastric pain*. *Nocturnal paroxysms* of pain are common, and are often accompanied by signs of *collapse*. In some cases *vomiting* and *diarrhea* are present. The *stools* may be light in color and greasy, and may contain blood. There may also be found an abundance of *undigested muscular fibers* in the stools in the absence of diarrhea; this is an incontestable proof of faulty pancreatic digestion. *Steatorrhœa* is not commonly present. Among the *pressure-effects* due to carcinoma of the head of the pancreas there may be *jaundice* (pressure upon the common duct), which persists and "is associated with an enlargement of the liver and gall-bladder." *Ascites* may appear from pressure on the portal vein. Chylous ascites, from pressure upon the thoracic duct, has been observed. The inferior vena cava may be compressed, causing *dropsy* of the lower half of the body; also the duodenum, followed by *gastrectasis* or by signs of *intestinal obstruction*. Carcinoma of the tail of the pancreas may be a cause of *hydronephrosis* of the left kidney, from pressure upon the ureter (Fitz). *Marasmus* and the *cachexia* develop rapidly in pancreatic carcinoma, and emaciation may become so extreme as to permit of a satisfactory *palpation* of the *tumor*, which occupies a position near the median line above the umbilicus. The growth, however, is palpable in about one-third of the cases only. *Glycosuria* may be associated. The quantity of indican in the urine is diminished.

Cammidge[1] describes carefully two tests for glycerose in the urine, and considers these as reactions *A* and *B*. Both of these reactions are modifications of the phenyl-hydrazin test for glucose in the urine, which necessitates that the urine be freed of any glucose before applying the test. Cammidge claims that diseases of the pancreas are recognized through these reactions. Reaction *A* may occur with the urine in case active tissue changes are taking place; *e. g.*, in cancer, pneumonia, etc. Treating the urine with perchlorid of mercury prevents reaction *A* in inflammatory pancreatic maladies, but has no effect upon the crystals that form in case of pancreatic cancer. Should no crystals form by either *A* or *B* method the pancreas is healthy; crystals formed by *A* and not by *B* method suggest active inflammation of the pancreas; crystals formed by *A*, due to acute inflammation, dissolve in one-half minute in 33 per cent. of sulphuric acid; but in case of chronic inflammation it requires two minutes to dissolve these crystals; while in pancreatic cancer the crystals obtained by *A* and *B* methods require fully three to five minutes for dissolution. Should the organ be damaged, as a result of previous disease, these crystals dissolve in from one to two minutes. Crystals forming in urines from persons who suffer from some malady foreign to the pancreas were found to dissolve in about one minute.

Diagnosis.—Carcinoma of the pancreas is probably present in a given case in which there are rapid and progressive emaciation, deep-seated epigastric pain, muscular fibers in the stools without diarrhea, persistent jaundice, enlargement of the gall-bladder, and the detection of a deeply-situated, fixed, and firm tumor in the region of the gland.

Aortic abdominal aneurysm may be mistaken for carcinoma of the pancreas because of the transmitted aortic pulsation. But in aneurysm the impulse is expansile instead of two and fro, while the cancerous cachexia is absent. *Chronic pancreatitis* is distinguished by the history of gall-stone attacks, greater tenderness, and the less marked cachexia.

It is sometimes difficult to differentiate a malignant tumor of the pancreas from *carcinoma of the pylorus*, of the *stomach*, or of the *transverse colon* or *omentum*; the following points will help in the differentiation of the former two:

Carcinoma of the Pancreas.	Carcinoma of the Pylorus.
The tumor is deep-seated and fixed; later it becomes slightly movable. It is not associated with gastric dilatation.	The tumor is more freely movable, and is usually associated with dilatation of the stomach.
Symptoms of chronic dyspepsia appear.	There are more marked gastric symptoms.
The vomitus is bilious; rarely contains blood.	There is "coffee-ground" vomitus; it is seldom bilious.
HCl is present, while there is an absence of lactic acid.	HCl is absent from the gastric contents; lactic acid is present.
The stools contain undigested muscle-fibers and sometimes fat. There is an absence of pancreatic secretions. The urine may contain sugar.	Usually the bowels are constipated, with occasional diarrhea. The stools are black after a hemorrhage. The urine does not contain sugar.
There is usually jaundice; sometimes ascites is present.	Usually there is no jaundice or ascites.
Inflation of the stomach shows the absence of a pyloric growth.	Inflation shows the presence of a pyloric tumor.
The course is more acute. Death may occur within a few weeks or months.	The course is more chronic, and secondary growths often appear in the liver.

[1] *The Lancet*, March 19, 1904, p. 782.

Neoplastic growths of the transverse colon are also more often superficial, and are movable and definable with the palpating fingers. There are symptoms of intestinal obstruction here, and inflation of the colon will show the relation of the tumor to the gut. In carcinoma of the colon the urine generally contains an increased amount of indican.

A discussion of the **prognosis** and **treatment** is unnecessary. Robson records 14 cases in which the portion of the gland affected was removed with 10 deaths.

Other Tumors of the Pancreas.—Exceptionally, sarcoma, adenoma, and lymphoma occur. Sarcoma is rarely primary. Secondary nodules are more common. According to Körte, of 10 cases of tumor of the pancreas operated upon of late years, 6 recovered.

PANCREATIC CYST.

Pathology.—Pancreatic cysts may be single or multiple, and large or small. When large they develop chiefly to the left of the median line. Single cysts may grow to an enormous size, containing as much as several gallons of fluid. The contents may at first consist simply of retained pancreatic juice, and usually the liquid is dark gray or dark brown, alkaline, and hemorrhagic or albuminous. A hematoma may be converted into a serous cyst. The specific gravity is from 1010 to 1024. Atrophy of the pancreas may ensue. Examined *microscopically*, the contents reveal leukocytes, red blood-corpuscles, oil-drops, fatty degeneration of the epithelium, and crystals of fatty acids and cholesterin.

Etiology.—Cysts of the pancreas may be due to occlusion of the pancreatic duct or its branches by compression from within or without the gland. They may also be due to tumors, to impaction of biliary or pancreatic calculi, to cirrhosis or angular displacements of the gland, or to the obstructive swelling from extension of catarrh of the bowel (Krecke). Of 121 cases collected by Körte, 33 were traced to traumatism. Lloyd suggests that the cysts that follow local injury are instances of encysted peritonitis involving the lesser omentum or that portion covering the pancreas (*pseudocysts*). Cysts of the pancreas usually occur in adults—in 66 of 116 cases in the third and fourth decades of life (Körte). Railton, however, met a case at six months of age.

Symptoms.—*Pain* may be absent, or it may occur as colicky paroxysms, referred either to the epigastrium, the left hypochondrium, or even the left shoulder. *Jaundice* and *ascites* are present in large tumors. *Vomiting*, *constipation*, or *fatty diarrhea* (rarely), with undigested proteids in the dejecta, or clay-colored, pasty, and offensive stools, may be present. *Albumin* and *sugar* may be found in the urine. *Emaciation* is not infrequent. *Intestinal hemorrhage* may occur and recur. A late and constant symptom is a *feeling of pressure* in the epigastrium. Rarely there is increased salivary secretion (pancreatic salivation). Occasionally all subjective symptoms are absent, and these cysts may temporarily disappear.

On **physical examination** a smooth, elastic, lobulated *tumor* is discov-

ered in the region of the pancreas if the growth is moderate in size. Sometimes a very large cyst develops in a remarkably short space of time—*i. e.* in a few weeks. When very large in size fluctuation is easily elicited. It may be slightly movable in the grasp and during inspiration. It usually presents between the stomach and transverse colon an area of dulness, and unless the tumor be of large size it is surrounded by tympanitic resonance of deeper timbre above than below. Auscultation may reveal a murmur caused by compression of the aorta. When the cyst attains enormous dimensions the usual *mechanical pressure-effects* are produced. Körte points out that cysts without any inflammatory or traumatic etiology may exist for many years or even decades.

Diagnosis.—The diagnosis rests on the typical physical signs—the discovery on palpation of a smooth, elastic, lobulated, or rounded tumor that is slightly movable, and on percussion of a dull area that is not continuous above with the spleen- and liver-dulness. Resort has been had to filling the stomach with air and the colon with water (after purging), and thus proving by *palpation* the deep-seated situation (behind the stomach and omentum) of the tumor. If pancreatic fluid be obtained from the supposed cysts, it will digest albumins and emulsify fats. This test is not wholly reliable, however. A pancreatic cyst may be mistaken for an *ovarian cyst*, for *hydatid cyst* of the left lobe of the liver, of the mesentery, *renal tumors* (*cysts*), *dropsy of the gall-bladder*, and *retroperitoneal sarcoma* (*Lobstein's cancer*). The differentiation must be made by a careful study of all the points in the case.

The **prognosis** is good under proper treatment—incision and drainage. Of 31 reported cases thus treated, only 2 proved fatal.

PANCREATIC CALCULI.

Pathology.—These are grayish-white, rounded concretions, consisting principally of calcium carbonate. The calculi may be as fine as dust or as large as an almond. Among their pathologic effects are fistulous communications with the colon, peritoneal cavity, and stomach; also cystic dilatations of the duct and abscess-formation. Atrophy of the organ and carcinoma due to irritation of the stones may be associated.

Etiology.—Pancreatic calculi presuppose a catarrhal condition of the pancreatic duct, with retention or anomalies of the pancreatic secretion, or some form of obstruction of the duct. The condition is rare, and, unlike gall-stones, more common in males.

The **symptoms** are developed when, during the passage of the stones along the duct to the duodenum, the latter excite inflammation. In consequence, paroxysms of *pain* occur (*pancreatic colic*) that are usually attributed to gall-stones, and we are often unable to differentiate the two conditions. The *radiation* of pain along the lower left costal border to the back rather than to the right side, and possibly the *detection of free fat in the stools* or *glycosuria*, may aid markedly in the diagnosis.

The finding of characteristic *calculi* in the stools is entirely confirmatory. Minnich has reported a case in which the calculi were found in the stools. Jaundice rarely appears in pancreatic lithiasis.

The **prognosis** is mainly dependent upon the associated lesions and upon certain sequelæ—pancreatic cysts and chronic pancreatitis.

The indications for **treatment** do not differ materially from those of hepatic colic. Surgical intervention should be considered.

XII. DISEASES OF THE PERITONEUM.

ACUTE PERITONITIS.

Definition.—An acute inflammation of the peritoneum. The condition may be primary or secondary. Clinically, two varieties—general and circumscribed—are recognized, while, pathologically, the disease is classified according to the nature of the exudate.

Anatomic and Physiologic Peculiarities.—The surface area of the peritoneum is quite extensive, being almost equal to that of the skin. Fluids of all sorts are rapidly absorbed by the peritoneum, and thus, if they be poisonous, constitutional infection is speedily propagated.

Pathology —Upon opening the abdomen in **acute generalized peritonitis** vascular injection both of the serous covering of the intestine and of the parietal layer is observed. Even in the most recent cases the coils of intestine may be feebly glued together by lymph, while in those of longer duration the adhesions are quite firm. As in the analogous inflammation of the pleuræ or pericardium, we distinguish the following forms pathologically: (*a*) A *plastic* or *fibrinous*, in which there may be also a small amount of serum present. (*b*) *Sero-fibrinous* (inflammatory ascites), chiefly characterized by considerable sero-fibrinous fluid; additionally, the coagulated fibrin forms a covering for the parietal and visceral layers of the peritoneum. (*c*) *Purulent* (most frequent). The amount of inflammatory exudate varies greatly, and is frequently enormous, exceeding 30 liters (quarts). Putrefactive decomposition of the pus may occur, especially in cases due to gangrene of the gut or to puerperal peritonitis (violent forms), giving rise to a thin fluid that is grayish-green in color, is sometimes distinctly sanious, and ill-smelling. Offensive gases are present with relative frequency. These may come from the intestinal canal, following the track of perforations; or they may be due to decomposition of the purulent exudate. (*d*) *Hemorrhagic.* This form is common in cases that are of a carcinomatous or tuberculous nature, and in subjects whose vitality has been lowered by various primary affections. It may also be of traumatic origin.

Changes in the Intestines.—The effect of acute peritonitis is to thicken the coats by inflammatory edema; soon the musculature is paralyzed. An associated catarrh of the mucosa of the intestine is sometimes observed.

The different pathologic varieties above described may be limited to definite portions of the peritoneal sac, when they are termed "encapsulated" or **localized acute peritonitis** (*vide supra*). In localized purulent peritonitis further extension of the process is arrested by the rapid formation of circumscribed adhesions due to the exudation of lymph; there are also undoubted instances of circumscribed, aplastic peritoneal abscesses. The milder forms of limited plastic and sero-fibrinous peritonitis pursue a slower course than the purulent variety, and commonly lead to the development of firm adhesions (*adhesive peritonitis*). Since the histologic changes in acute peritonitis do not differ from those ob-

served in other inflammations of serous membranes, the reader is referred to the section on Pleurisy (p. 580) for their consideration.

Etiology.—The irritants causing acute peritonitis may be—(*a*) **Organized inflammatory agents** (*bacteriologic irritants*). These may be *specific* or *non-specific*. Among the non-specific agents are the pyogenic bacteria. Grawitz has shown that the latter can only cause peritonitis under certain conditions: they excite the disease when injected into the peritoneal cavity or when poured out from the diseased or injured membrane more rapidly than the peritoneal tissue can dispose of them; also when the epithelial layer has from any cause been removed. Absorption may be interfered with, while the pyogenic micrococci continue to enter from the bowel or other viscera in great numbers. Unfortunately, the clinical practitioner often meets with cases of peritonitis in which these pyogenic organisms are the only positive agents. These essential conditions obtain when the membrane is wounded by the perforation of gastric and intestinal ulcers, and also in perforation of the gall-bladder, in rupture of the liver, kidneys, and spleen, when the latter are the seat of abscesses, and, with uncommon frequency, in appendicitides, in purulent inflammation of the ovaries and of the Fallopian tubes. "There are instances in which peritonitis has followed rupture of an apparently normal Graafian follicle" (Osler). These perforative forms of peritonitis are at the same time the most serious and the most important. "Death may result from the injection into the peritoneal sac of putrid liquid if the dose be large enough; but it is practically the same whether the fluid is injected into the blood-stream at once or allowed to find its way into the peritoneal cavity, and the result follows nearly as quickly in the one case as in the other" (Moullin). The rapid absorption of liquid substances gives full opportunity for the phagocytic action of the white blood-corpuscles.

Among specific organic irritants the *tubercle bacillus* deserves especial mention, though, as before intimated, a discussion of its characteristics is not in place here. The *streptococcus pyogenes* is probably responsible for the most violent forms of peritonitis (*e. g.* those occurring in puerperal sepsis and post-operative varieties). The *staphylococcus pyogenes aureus* (or *albus*) has also been found in such instances.

The *bacterium coli commune* (always present in the intestinal tract) is frequently the leading factor in peritonitis of intestinal origin, and usually in association. The streptococcus is often present also in these cases. In 12 cases of primary peritonitis, 11 were instances of mono-infection; and in operations upon the peritoneum (not involving the intestine), 25 of 33 cases were mono-infections, the staphylococcus aureus being present alone in 12 and the streptococcus in 5 (Flexner). Occasionally other organisms, as the *pneumococcus*, the *bacillus of Friedländer*, or the *bacillus pyocyaneus*, *typhosus*, and *proteus*, the *gonococcus*, the *aërogenes capsulatus*, and the *anthrax bacillus*, have been found. Multi-infection is quite common. The bacteriologic classification of peritonitis would be desirable from the standpoint of treatment (*vide infra*), but it cannot be applied clinically.

(*b*) **Chemical Irritants.**—These are rather numerous and varied, though all produce their effects in one of two ways. First, the irritant acts upon the membrane, exciting an exudation of lymph. Here constitutional intoxication is secondary. Secondly, the chemical irritant may be quickly absorbed, and produce systemic intoxication immediately (rare).

(*c*) **Mechanical irritants,** as, for example, a hernia, which may produce a localized peritonitis.

(*d*) Peritonitis may be due to a direct extension of infective processes from the intestinal tract or other adjacent organs (*secondary peritonitis*). The bacteria often penetrate the intestinal wall and gain the peritoneum by way of the lymph-channels. The disease is often secondary to pleurisy, the irritants passing through the diaphragm along the course of the lymphatics. Peritonitis may be secondary to chronic Bright's disease, gout, and arterio-sclerosis; in such cases the special irritants probably reach the membrane through the general circulation.

(*e*) The disease is very rarely *primary* (*idiopathic*). It has been attributed to exposure to cold or wet (*rheumatic peritonitis*). These so-called idiopathic cases are probably instances of cryptogenetic infection.

Clinical History.—The symptoms are both of a *local* and a *general* nature. In sthenic cases of perforative peritonitis they occur simultaneously with great severity and suddenness. On the other hand, in asthenic cases, such as occur frequently in those already afflicted with some serious disease that is apt to result in perforation (for example, typhoid fever), both the local and constitutional symptoms are more or less overshadowed by the disturbances due to the primary affection. Again, circumscribed abscesses of the peritoneum often lead to diffuse suppurative peritonitis, and the change may take place so insidiously as to defy detection. These anomalies from the typical onset and course of the disease are by no means exceptional, and should ever be distinctly borne in mind by the physician.

Local Symptoms.—Among these, *pain* is the chief. The seat of greatest intensity of the initial pain corresponds, in most instances, with its point of origin. Hence the character of the causal disease is often betrayed by the location of the chief pain. For instance, if this appears in the region of the stomach and is referred to the back or shoulders, we would think of gastric ulcer; if in the ileo-cecal region, of appendicular disease; and so on. It follows that quite commonly the severest pain is in the lower half of the abdomen. It is almost constant, increases in severity, and finally becomes general and excruciating; it is also much increased by deep respirations, by pressure, and by bodily movements. It remits, but does not intermit, though it may be slight in asthenic (*secondary*) cases. Here the patient is excessively weak, while his sensibilities are greatly blunted by the primary infection. Gastro-intestinal symptoms are prominent, more particularly *vomiting*, which occurs early and is apt to recur with comparative frequency. It may follow the taking of food, though, in my experience, it has more commonly taken place spontaneously; the *vomitus* then consists of a watery liquid greenish in color and containing mucus. In rare instances it is a dark-brown liquid. Vomiting may sometimes be absent, however, owing to the presence of marked *asthenia* or *coma*. *Eructations* and, later, *hiccup*, are common, and *constipation* is usually present and may become exceedingly obstinate. On the other hand, there may either be *diarrhea* throughout the disease, or this symptom may precede the constipation. It is to be ascribed to an increased peristalsis due to intestinal catarrh. Constipation is due chiefly to paralysis of the musculature of the intestine. The *apex* of the heart is elevated; the *tongue* at first is furred and moist, and later it is dry, brown, and often fissured.

Constitutional Symptoms.—At the onset the patient in *sthenic* cases is seized with a *rigor* that may be repeated. The *shock* sustained by the nervous system in acute peritonitis is most intense; the *temperature* rises immediately, though it does not, as a rule, attain to a high level, and it frequently presents a curve more or less characteristic of suppuration. The rectal temperature is often relatively high; the respirations are shallow (costal) and much accelerated, ranging from thirty to forty per minute. We have, as factors to account for this increased frequency, (*a*) a crowding upward of the diaphragm, (*b*) the greatly enfeebled heart, and (*c*) the pain occasioned by throwing the diaphragm into action. The *heart* becomes weak, the pulse-rate at first, however, ranging from 100 to 130, with a rise in the blood-pressure. The pulse toward the close becomes exceedingly frequent (130 to 150 beats per minute) and is almost imperceptible. Other evidences of more or less marked *circulatory collapse* soon manifest themselves. The patient wears an anxious facial expression, the eyes are sunken, the features pinched and cool, the lips cyanotic, and the extremities are likewise cold and somewhat livid. The patient invariably assumes the *supine position*, with the lower extremities drawn up, so as to lessen the tension of the abdominal muscles, and thus to secure the greatest possible comfort. The *urine* is scanty in amount, high-colored, and contains indican. There may be a retention of urine; though oftener, perhaps, micturition is more frequent than in health. Leukocytosis occurs, if we accept the fulminating cases in which leukopenia may be found. Marked *nervous symptoms* do not appear; indeed, the mind usually remains quite clear to the close. Moderate delirium, however, which sometimes gives way to mild stupor, is met with occasionally. In the *asthenic form* of acute peritonitis the constitutional features differ from those described above. The *temperature* is usually subnormal (except in the rectum), the pulse is exceedingly feeble and running, and the signs of collapse are well marked from the onset.

Physical Signs.—*Inspection* reveals the gradually increasing abdominal distention, that frequently becomes excessive if the intestinal walls are more or less completely paralyzed. Often the amount of effusion soon becomes large, when the abdomen appears widened. The degree of distention bears a definite relation to the severity of the inflammatory process, and is in inverse ratio to the development of the abdominal muscles. Thus, when the latter are poorly developed or greatly relaxed the expansion is enormous. On the other hand, when they are strong the muscles are apt to be quite tense, permitting of a relatively slight enlargement; the abdomen may even show a small concavity, in which case the walls are of a board-like hardness. The cardiac apex-beat is displaced upward and outward, occupying the fourth interspace.

Palpation elicits extreme tenderness, more particularly in the vicinity of the umbilicus. Rigidity of the abdominal wall is the most important symptom in perforative peritonitis (J. C. Wilson). In not a few instances of acute peritonitis have I been able to detect a distinct friction-rub. *Percussion* gives at first an exaggerated tympanitic note. There is often an absence of liver-dulness in the mammary line, and rarely also it is absent in the mid-axillary line. In pneumo-peritoneum, resulting from perforation of the gut or stomach, we often meet with an absence of liver-dulness, especially when a large purulent effusion co-

exists. Again, a great diminution in, or even the total effacement of, the dull area may be caused by coils of intestine forcing their way up between the anterior surface of the organ and the abdominal wall. When air is present within the abdominal cavity and the patient lies upon his right side, splenic dulness disappears from displacement by the air. The lower level of cardiac dulness is as high as the fifth rib.

By means of *percussion*, sooner or later, fluid effusions are usually detectable in sthenic cases. On the other hand, there may be in markedly asthenic cases an amount of liquid exudation present that is often too small to admit of detection. When the effusion is considerable in quantity, there is percussion-dulness over the most dependent parts; when tympanitic distention is excessive, however, even a copious effusion may be so effectually hidden as to elude discovery in this way. I have elsewhere reported one such instance.[1] When the decubitus can be altered, the line of dulness will be found to be movable, but the degree of mobility varies exceedingly, depending upon the extent of the peritoneal adhesions present. The effused material is partly contained in pouches, giving rise to areas of circumscribed dulness.

Course and Prognosis.—Asthenic forms, with rapid pulse, leukopenia, and persistent low temperature, are perhaps invariably fatal. Though the local signs are not marked, the characteristic evidences of collapse or of septicemia appear. The *duration* in sthenic cases rarely exceeds one or two days; in asthenic cases it is longer, lasting from four or five to six or eight days. Death sometimes occurs quite suddenly, owing to cardiac exhaustion or primary shock. The clinical peculiarities and the course are greatly influenced by the etiology—*e. g.*, cases due to *Streptococcus pyogenes*, *Bacillus pyocyaneus*, and *Bacillus coli* are extremely fatal, while those due to the gonococcus and pneumococcus are more benign. Acute generalized peritonitis arising from perforative appendicitis, puerperal sepsis, or from external injuries is usually of a violent form and ends fatally, unless subjected to early operation. Perforation of a gastric or duodenal ulcer gives a better prognosis, since the number of colon bacilli steadily diminishes from the ileocecal valve to the stomach (Cushing and Livingood). When the disease is traceable to rheumatism or exposure, recovery may take place. A case of the sort occurred in my own practice in which acute serofibrinous peritonitis with considerable effusion was associated.

Peritonitis in Children.—Syphilitic peritonitis may be congenital, and peritonitis caused by an inflamed cord may be met in the newborn. In children the common causes are trauma and appendicitis.

The *symptoms* differ from those presented in the adult. However severe the pain, the child merely utters a short cry or whine. Constipation and vomiting are less conspicuous features. Meteorism is pronounced and fever high. Convulsions not rarely occur. The condition is extremely grave in young children.

LOCALIZED OR PARTIAL PERITONITIS.

(*Circumscribed Peritonitis; Visceral Peritonitis.*)

This is a localized form of inflammation of the peritoneum that is coextensive only with the serous covering of single organs, and involves a limited portion of the membrane. Hence, to the various forms of cir-

[1] *International Medical Clinics*, vol. iii., second series, p. 82.

cumscribed peritonitis such terms as perihepatitis, perisplenitis, perinephritis are applied. The condition is found in its most important form in *appendicitis*, but the points that are characteristic of localization in this disease have been mentioned elsewhere (*vide* Appendicitis, p. 863). Localized peritonitis may also be caused by carcinoma.

Pyo-pneumothorax subphrenicus is the term applied to a circumscribed peritoneal abscess containing air, situated between the liver and diaphragm. The condition is described under the heading Acute Perihepatitis (p. 919).

Local pelvic peritonitis (perimetritis) is the most frequent variety, and is secondary, as a rule, to inflammation about the uterus, Fallopian tubes, and ovaries. The leading causes are tuberculosis, puerperal septicemia, and gonorrhea. F. Billings points out that when abdominal rigidity is absent in this form rectal examination will disclose rigidity of the pelvic muscles.

Symptoms.—The *local* clinical features do not differ from those described under the diffuse form, but their area of distribution is more or less strictly limited to definite regions. By eliciting the *physical signs* with care fluid collections are sometimes demonstrable. The *constitutional symptoms* are likewise similar in character, though less marked than those belonging to the diffuse variety. There may be *rigors*, and *pyemic symptoms* appear, together with the temperature-curve peculiar to this condition. The danger of involvement of the general peritoneal cavity as the result either of rupture or of an extension of septic inflammation is a constant menace. When the peritonitis remains localized these cases may pursue a subacute or even a chronic course, though grave constitutional disturbance finally develops.

Diagnosis.—In attempting to diagnosticate acute generalized peritonitis it is of the utmost importance to keep in remembrance the sthenic and asthenic forms of the affection. The character and gravity of the symptoms are such as to render the diagnosis of the sthenic form entirely easy. Especially valuable local features are the *constant pain*, the *marked tympany*, the *excessive tenderness under pressure*, and the *vomiting at intervals of a greenish fluid material*. Of equal importance are general disturbances previously depicted, particularly the *cool, sharpened features* and the *ever-increasing weakness* and *rapidity of the pulse*. These clinical manifestations clearly foreshadow cardiac exhaustion or fatal collapse. When the cases are not seen until the advanced stage has arrived, however, the diagnosis presents many difficulties. Nothing is now more important than the consideration of the previous history, with a view to determining the point of origin and the probable cause of the disease (usually some such primary disease as appendicitis or gastric ulcer), as well as the accompanying symptoms and physical signs.

The smaller number of cases belonging to the adynamic type are from the start extremely difficult of diagnosis. Here a history that is clearly indicative, the presence of moderate tenderness, and augmented tension of the abdomen, with profound collapse, would point to this condition.

General Differential Diagnosis.—*Hysteric peritonitis* (so-called) simulates the genuine form so closely as to make the distinction an insurmountable difficulty, unless there be accompanying hysteric manifestations. Previous similar attacks point to hysteria. In my experience the tenderness has been out of proportion to the gravity of the constitu-

tional disturbance. The patient often complains bitterly before the abdomen has been touched; on the other hand, when his attention has been otherwise engaged, firm and prolonged pressure can be made.

Acute generalized peritonitis occasionally supervenes on *typhoid fever*. In such cases it is caused either by perforation of the intestine or by a direct extension of inflammation from a deep typhoid ulcer. If consciousness be retained, sudden severe pain, tenderness followed by excessive tympany, a peculiar indescribable *facies*, and signs of collapse will establish the diagnosis. Peritonitis, however, develops more often in those grave cases of typhoid that are attended with coma, marked meteorism, and profound adynamia, and under such conditions it often remains unrecognized (*vide* Typhoid Fever, p. 35).

In *acute enteric catarrh* the meteorism and sensitiveness under pressure are usually less pronounced; the disease also lacks the marked constitutional symptoms of acute peritonitis. The pain is colicky, is characterized by exacerbations, and even intermits in entero-colitis, while it is constant in peritonitis. The pain in acute enteric catarrh is often followed by diarrheal stools.

Intestinal colic is distinguished by the flatulence, the borborygmi, and the wandering pain in the absence of all other phenomena.

Rheumatism of the abdominal muscles excites pain, which, however, is superficially located (the disease affecting the muscular layer), and is frequently associated with rheumatism in other parts of the body. There may also be a clear history of previous rheumatic attacks.

Pleuro-pneumonic diseases may simulate peritonitis, since the early symptoms, especially the pain, may be referred to the abdomen. The temperature is apt to be higher and the respirations more rapid in intrathoracic affections—points that should lead to a thoracic examination.

Tubal pregnancy (*after rupture*) has also been confounded with acute peritonitis, but its differential diagnosis is fully discussed and must be looked for in speciai works on gynecology and obstetrics.

Rupture of an abdominal aneurysm and *embolism of the superior mesenteric artery* are also conditions that give rise to peritonitic symptoms—meteorism, recurrent vomiting, and violent collapse.

Acute generalized peritonitis in its symptomatology bears a close resemblance to acute intestinal obstruction, and the discriminating points have already been tabulated (*vide* p. 875).

Prognosis.—This is less grave than in the diffused form, and recovery may often be expected. Timely surgical intervention, particularly if a tendency to spreading be shown, is often helpful or may even lead to prompt recovery.

Sequelæ.—If recovery should take place, the inevitable result is the formation of adhesions and fibrous bands, the contraction of which may cause constriction of the bowels, bile-ducts, and other structures.

Treatment.—Hygienic and Dietetic.—The patient should be placed in the position that will give him most comfort, and should be kept absolutely undisturbed. The sick-room should be of good size and well ventilated; the temperature should be kept at from 65° to 70° F. (18.3°–21.1° C.). The diet demands careful attention. Pancreatized milk in accurate dosage (℥iv–vj—128.0–192.0—every two hours) should be administered, and if the stomach will not bear the introduction of nourishment, recourse should be had to rectal alimentation. Other

liquid food-stuffs, as meat-juices and egg-white (diluted), may also be allowed. In asthenic cases alimentation must be generous, although solid articles of food are to be avoided.

Medicinal.—Surgical measures are recommended by most writers in the treatment of generalized peritonitis, although it is now generally conceded that in cases due to mild infection by the gonococcus, the colon bacillus, and the pneumococcus, nothing is to be gained by operation. This is especially true of a gonococcus peritonitis as shown by Hunner and Harris.[1] Whenever, however, there is reasonable doubt regarding the diagnosis, operation should not be delayed. Formerly the opium method of treatment, first instituted by the late Alonzo Clarke, was followed by the bulk of the profession. His plan was to administer ½ gr. (0.0324) of morphin or its equivalent (gr. ij—0.129) of opium, and repeat the dose every second hour until the respirations were lowered to ten or twelve per minute. The pupils were then observed to be contracted, the pulse from 76 to 80, the pain relieved, and peristalsis arrested. This latter effect was obtained, even though in the case of some patients larger doses of opium than here indicated were necessary; in others smaller doses sufficed. The bowels were absolutely let alone. It is explained that in favorable cases the bowels moved spontaneously at the end of one week, and that the patient then entered upon convalescence. This method of treatment is at present adhered to only by the ultra-conservative element of the profession. Stockton[2] advocates the opium treatment in the milder cases before the peritoneal inflammation has become generalized. The moderate use of opiates, however, results only in covering up symptoms, not in curing the patient. Stockton believes that the proper treatment of oncoming septic peritonitis is immediate operation.

The saline method is deservedly popular at the present day. *Saline purgatives* are exhibited in divided doses in concentrated solution (ʒj–ij—4.0–8.0—every two or three hours) until the irritating intestinal contents, should any be present, are removed, and additionally several copious serous discharges occur daily. Purgatives do good when given in this manner principally by causing a rapid exosmosis of serum from the blood-vessels of the intestines, by removing the collateral edema, and by indirectly relieving the congestion of the peritoneum, thus promoting a rapid absorption through the latter membrane. By increasing the peristaltic movement they also diminish the danger of peritoneal adhesions. The remedies to be selected will depend upon two primary considerations: *first*, the etiology of the individual case (whether a communication has or has not been established between the peritoneal cavity and the bowel), or an intra-peritoneal abscess or abscess-cavity in one of the abdominal viscera; and *secondly*, the type of the case, whether sthenic or asthenic. If perforation is known to have taken place or the occurrence of this accident is strongly suspected, a prompt laparotomy, followed by the free use of salines, is the proper treatment. After the *primæ viæ* have been looked after by the surgeon, salines, for the reasons before stated, are to be used with a free hand. For a like reason they are most serviceable in peritonitis due to extension of the inflammation, and also in the puerperal form. If the patient be robust, with a full, tense pulse, we may begin the treatment by the use of mercury, the

[1] *Bulletin Johns Hopkins Hospital*, 1902. [2] *Jour. Amer. Med. Assoc.*, April 11, 1908.

best preparation being calomel, exhibited in fractional doses (gr. ss—0.0324) every hour until its purgative action is obtained; this is to be followed by the salines. The object of the *calomel treatment* is to defibrinate the exudations as well as the blood of the patient. Indications demanding the *opium treatment* do not often present themselves. In cases in which the vital forces are profoundly depressed, as shown by the symptoms of collapse and there is not even a reasonable suspicion of perforation, opium should be tried. When, however, the evidences of perforation into the general peritoneal cavity are complete and competent surgical skill is not at hand, large doses of morphin are imperative, with a view to relieving pain, keeping the patient at absolute rest, and sustaining the heart against the exhausting effect of shock. The bowels should now be relieved by simple large enemata. The value of serum-therapy in this disease is as yet uncertain (Fowler). For the systemic collapse, and for combating thirst and vomiting I can warmly recommend *saline infusion*, preferably according to Murphy's drop method of rectal irrigation.

Local Treatment.—At the onset, if the patient be strong, from twenty to thirty leeches are to be applied to the abdomen. The ice-bag or ice-poultices are often of distinct service in the earlier stages. Later, in localized peritonitis, blisters may be useful, although objectionable in the event of surgical intervention becoming necessary. In cases in which meteoric distention is not great I have also made repeated trial of an ointment containing ung. ichthyol (℥j—32.0); ung. belladonnæ (℥ss—16.0); ung. hydrarg. (℥ij—64.0); this is applied thrice daily.

In order to relieve the *tympany* turpentine stupes are serviceable. I have also had favorable results from the insertion of the long rectal tube (soft esophageal) well up in the colon. *Large* high enemata should be used; and turpentine combined as follows may prove efficacious:

℞.	Turpentine,	ʒij (8.0);
	Ox-gall,	ʒij (8.0);
	Milk of asafetida,	℥iv (128.0);
	Warm water,	℥vj (192.0).

Puncturing the abdomen with a hypodermic needle in order to relieve tympany, as recommended by Loomis, may also be resorted to, though I have had no personal experience of its use.

Pain.—No matter what general plan of treatment is pursued, the pain must be relieved by opium in some form. *Thirst* is to be relieved by chipped ice, over which a little brandy may be sprinkled. The *vomiting* is best treated by carbonated water exhibited in small quantities, or by iced champagne similarly administered. One-drop doses of creosote are also of value.

CHRONIC PERITONITIS.

Definition.—Chronic inflammation of the peritoneum.

Pathology and Etiology.—The anatomic characters presented by different cases are greatly varied, though for convenience of study they may be considered under two divisions (as in the acute form): 1. *Local;* 2. *General.* The latter may be (*a*) **Adhesive,** when the peritoneal layers are inseparable and indistinguishable, with an obvious thickening, and the intestinal coils are everywhere seen to be grown together.

The cause is usually a previous acute attack, and, doubtless, the condition is commonly produced by the *acute progressive form* (Mikulicz), which is *localized* at the start. Rheumatism is also an occasional factor, and adhesive peritonitis, confined, as a rule, to small circumscribed areas, may be engendered by the trocar used for tapping in ascites.

(*b*) **Proliferative Peritonitis.**—"The essential anatomic feature is great thickening of the peritoneal layers, usually without much adhesion" (Osler). It has been found to be associated with cirrhosis of the stomach, liver, and other abdominal organs. The amount of liquid effusion, varying in composition from serum to pus, is usually moderate, and it may, owing to adhesions, be loculated. The omentum is sometimes rolled up in the form of a massive cord, its long axis taking the transverse direction. In an autopsied case of chronic peritonitis apparently secondary to hepatic cirrhosis I observed in the thickened membrane numerous small hard nodules that were at the time regarded as being tuberculous in nature. It is to be pointed out, however, that a number of cases of pseudo-tuberculosis have been recently reported. In several of these an operative incision was followed by recovery, and this was put down as a cure of tuberculous peritonitis till the microscope showed the nodules to be fibrous. Among *etiologic factors* chronic alcoholism stands first. In one case that I saw, acute followed by chronic rheumatism seemed to be the only assignable cause. The condition is sometimes secondary to chronic nephritis, to syphilis, or a general fibroid process.

(*c*) **Cancerous Peritonitis.**—Quite often in connection with cancerous growths in the peritoneum a well-marked peritonitis is evident. There may be a liquid exudation, which is apt to be bloody and chylous.

(*d*) **Chronic Tuberculous Peritonitis.**—This is the most important variety, and it may be part of a multiple serositis. The inflammatory lesions are quite pronounced, as a rule, and lead to marked thickening of the layers—changes that are to the naked eye identical in appearance with those noted under the preceding forms, but which on histologic examination show the presence of tubercles and caseous degeneration. The amount of liquid effusion varies within wide limits, and is usually blood-stained. The frequent association of hepatic cirrhosis with tuberculous peritonitis should be remarked. From tuberculous peritonitis, tuberculosis of the peritoneum is also to be distinguished clinically; the latter may be acute or chronic, and the lesions consist in the deposit of various sized tubercles without much collateral inflammation. Acute and chronic tuberculosis of the peritoneum have received due consideration in their appropriate place (p. 281).

(*e*) **"Chronic Hemorrhagic Peritonitis."**—This term should be limited in its application to that form first described by Virchow, in which the peritoneum is at intervals partly covered by a membrane of new connective tissue that alternates, as it were, with layers of hemorrhagic extravasation. A similar condition results from the frequent use of the trocar for ascites.

Chronic Localized Peritonitis.—This is of frequent occurrence, and is confined most commonly to the serous covering of the spleen, liver, and certain portions of the bowel, particularly of the appendix. The condition results in the formation of *firm adhesions*, with matting of the intestinal coils and fibrous bands. It is usually the *sequel* of localized

acute peritonitis occurring in connection with inflammatory diseases of the different abdominal organs.

Symptoms of the General Forms.—Whether chronic peritonitis follows the acute form or not, it always develops insidiously. Most cases remain quite obscure; and not a few are totally devoid of clinical manifestations. The patient may complain of disorders of the *alimentary tract*, and especially of *constipation*. On the other hand, *diarrhea* is observed in tuberculous peritonitis from associated intestinal ulceration. Rarely pressure, from the traction force of the adhesions, on the common duct or portal vein gives rise to obstructive *jaundice*, or *ascites*, as the case may be. I saw an instance recently in which compression of the veins leading to the lower extremities caused unilateral *edema*. *Subjective abdominal sensations*, as uneasiness, oppression, heat, and pain (often colicky in character), are experienced. Sometimes pain is entirely absent.

General symptoms appear, though they are quite vague as a rule. An irregular fever, hectic in type, is occasionally observed. Later, increasing general weakness, emaciation, and general nervous disturbance become rather prominent clinical features. Some of these phenomena, however, may be due to associated affections. When the peritonitis is tuberculous we frequently see clinical evidence of the primary process in other parts of the economy (*vide* Tuberculous Peritonitis, p. 281).

Physical Signs.—*Inspection* usually shows the belly to be slightly, though unequally, enlarged. As in acute peritonitis, so here we may find the belly flat, or even concave, with great tension of its walls. Fluctuation is sometimes obtainable over limited areas only, since the fluid is not free, but encapsulated. The rolled-up and shrunken omentum may be *palpable* as a sausage-shaped transverse coil, and thick bands of adhesion may also not rarely be felt, in different places, as hard, uneven masses simulating neoplasmata. The *percussion-dulness* varies considerably with the amount of effusion, its arrangement, the degree of peritoneal thickening, as well as with the character and locality of the fibrous bands. It follows that in some cases irregular areas of tympanitic percussion-resonance and of dulness are to be found side by side. Obviously, too, changing the patient's posture would not give movable dulness, owing to sacculation of the fluid. A marked sense of resistance is experienced on percussion over the dull area. *Friction-fremitus* can sometimes be elicited, and less frequently *friction-sounds* also during forced breathing.

Symptoms of Chronic Local Peritonitis.—This condition is often entirely latent. When not so, the most characteristic indication is constant *pain*, distinctly colicky in nature and often quite intense. The *physical signs* are negative, as a rule. Very rarely a resistant, ill-defined mass, corresponding with the seat of greatest pain, can be felt. A fibrous band may be so arranged as to form a snare through which a knuckle of bowel may pass, with resulting strangulation. Fitz's analysis of 295 cases of strangulation showed 63 to be caused in this way.

Differential Diagnosis.—That form of chronic peritonitis (serous or granular) most frequently seen in females at the commencement of puberty is hard to discriminate from *tuberculous peritonitis*, since the latter may be more or less latent. Tuberculous peritonitis is generally

attended with fever, more pain and tenderness, and there is a more rapid accumulation of the exudate. Again, the general features, debility and loss of flesh, progress more rapidly than in granular peritonitis. The detection of conclusive evidence of the disease in persons closely related, or on physical examination of associated pulmonary tuberculosis or pleuritis, would render the diagnosis of tuberculous peritonitis almost certain. In obscure cases the guinea-pig should be inoculated with the exudate (see Pleurisy, p. 580).

Course and Prognosis.—The milder varieties of simple chronic peritonitis may, though rarely, reach a favorable issue. In cases belonging to this category the disease takes a chronic course, and leads gradually to a condition of extreme debility, even if it does not, as is usually the case, materially shorten life. Tuberculous peritonitis has, until recently, been regarded as being almost uniformly fatal at the end of several months. Cures that must be attributed to the surgeon's work, however, are at present by no means uncommon. Rarely, spontaneous cures also occur, particularly in peritoneal tuberculosis without fever or with only slight fever. "This form runs in itself a favorable course" (C. Fenger).

Treatment.—The patient should be enabled to enjoy the benefits of good sanitary surroundings. Close attention is to be paid to the *diet*, the coarser vegetables and sweets being prohibited, since they increase the pain by exciting the production of gas. A change of air has improved the condition in several instances occurring in my own practice. The usual constipation may be relieved by simple enemata or by the use internally of the fluid extract of cascara sagrada. Tonics and alteratives, the latter with a view to promoting the absorption of the exudate, may also be employed, and I would recommend especially for this purpose the double iodids, as in the formula given in the discussion of Pleurisy (*vide* p. 595). In the early stages some degree of relief, or even a curative effect, may be secured by *local means*, as the application of equal parts of belladonna and iodin ointments until mild counter-irritation is produced. Ichthyol ointment is also serviceable. After all, however, little is to be gained from therapeutic measures, and it is to surgery that we must look for fresh triumphs in the treatment of this truly distressing complaint. Cases of chronic localized peritonitis with adhesions have been operated upon successfully by W. E. Ashton, H. A. Kelly, and others. Instances of chronic generalized peritonitis, whether tuberculous or not, in which the fluid effusion reaccumulates rapidly after repeated tappings, also furnish adequate indications for operative procedures.

ASCITES.

(*Hydrops Peritonæi; Dropsy of the Peritoneum.*)

Definition.—An accumulation of serum in the peritoneal cavity, resulting from stasis (obstruction) in the branches of the portal vein.

Pathology.—The quantity of liquid contained in the peritoneal cavity is quite variable, though it often amounts to several gallons. It is clear and transparent, or slightly opalescent, especially on standing,

and the specific gravity ranges from 1010 to 1014. In color it often has a faint lemon-yellow tint; it may, however, be either distinctly yellow, brownish (in cirrhosis), bile-stained (as when jaundice is present), or slightly blood-stained. In reaction it is usually alkaline; very rarely it is either acid or neutral.

The ascitic fluid usually contains much albumin, resembling in this respect blood-serum, as would be expected from its source. The percentage of albumin may be approximately ascertained by noting the specific gravity of the fluid by the urinometer. Thus, in true ascites the specific gravity ranges from 1010 to 1014, and the variation in the percentage of albumin is from 1 to 2. In effusions due to *peritonitis* the percentage of albumin ranges higher (2.5–6 per cent.); hence the specific gravity ranges correspondingly higher (1015–1024). The standing specimen may show to the unaided eye a minute coagulum of fibrin. In the lowest layer of the fluid the microscope discloses leukocytes, red blood-corpuscles (in abundance when ascites is due to general venous stasis), fat-cells, endothelium, and cholesterin-crystals. In ascites the microscopic appearances of the peritoneum are usually normal, while in instances of peritonitis the membrane, including the subperitoneal fibrous tissue, is opaque and slightly thickened.

In the so-called *chylous ascites* the fluid resembles milk; it contains fat-droplets, a few lymphocytes, and sugar (Hodlmoser [1]). This condition may be associated with a collection of milky fluid in the left pleural sac, when there is thrombosis of the subclavian vein at the point at which the thoracic duct enters. The term *ascites adiposus* is applied to a milky fluid, in which the origin of the fat is the débris of degenerated epithelial cells, with few fat-droplets and no sugar (Quincke and Senator), to the exclusion of other morphologic elements.

In long-standing cases the abdominal and the thoracic organs become atrophied from pressure exerted by the dropsical fluid.

Etiology.—Among the chief causal factors are those that hinder or arrest the return of venous blood from the peritoneal membrane, as the following: (*a*) Pressure upon the branches of the portal vein within the liver, due to contraction of surrounding tissues, as in hepatic cirrhosis (including malarial atrophy—De Brun), syphilis of the liver, and cancerous infiltration. (*b*) Numerous conditions in the course of which pressure may be made upon the portal vein external to the liver, as enlargement of the glands in the fissure, carcinoma, hydatids, or abscesses of the liver. Tumors of any adjacent organs (*e. g.*, pancreas) may produce it. (*c*) Thrombosis of the portal vein. (*d*) Pressure upon the inferior vena cava after it receives the hepatic trunk (Roberts), or upon the latter itself, or the lymphatics. (*e*) The portal circulation is also impeded in chronic pulmonary affections (cirrhosis and emphysema) and heart diseases (*e. g.*, ascites due to "pericarditic pseudocirrhosis of the liver"—Pick). (*f*) A new growth in the peritoneum may compress the smaller veins or the root of the mesentery. (*g*) Diminished resistance of the walls of the portal vessels, due to chronic affections that diminish the albuminous constituents of the blood and impair the nutrition of the peritoneum, as Bright's disease, carcinoma, syphilis, chronic malaria, pernicious anemia, leukemia, amyloidosis. (*h*) Chylous ascites

[1] *Wiener klin. Woch.*, 11 *Jahrg.*, No. 49.

is caused either by a leakage of the lacteals (due to ulceration, injuries, or the presence of filariæ) or by the obstruction of the thoracic duct (due to thrombosis, cicatrices, compression). Cases of lactescent ascites in which the fluid is milky (not chylous) have recently been reported. The nature of the fluid is as yet unknown. (*i*) Adipose ascites has for its direct cause fatty cellular degeneration, such as is found in carcinoma and tuberculosis of the peritoneum.

Leyden has (1897) described an ameboid organism.

Symptoms.—Slight peritoneal dropsy gives rise neither to symptoms nor to abnormal physical signs. When the sac contains 1 quart (1 liter) of fluid or over, however, the first subjective *symptoms* that are due to the mechanical effect of the fluid appear. They are a sense of weight and fulness, with slight uneasiness. As the proportion of transuded serum becomes gradually increased these symptoms become more pronounced. There may in addition be a *dragging pain* in the loins, *gastro-intestinal disturbance* (meteorism, constipation), and dyspnea (owing to the resistance opposed to the descent of the diaphragm, resulting in compression of the lungs). The latter symptom is much increased upon exertion or on assuming the recumbent posture. Since the *heart* is displaced upward, an embarrassment of its action (rapidity and irregularity) would be expected. *Syncope* is not infrequent for similar reasons. Frequent *micturition* from pressure upon the bladder is common, and the kidneys, owing to compression of the renal vessels, secrete an *albuminous urine*, which is greatly lessened in amount.

Physical Signs.—After the serum has collected in considerable amount the physical signs afford characteristic evidence of the condition. From *inspection* we learn many valuable points: (*a*) The belly is uniformly prominent (the degree depending upon the amount of serum present), giving it a rounded form. Changing the posture of the patient shifts the point of greatest pouching. (*b*) The skin is seen to be tense, smooth, and shining, and sometimes shows lineæ albicantes; the umbilicus commonly bulges forward; less frequently it is obliterated, and the surface-veins are often enlarged. (*c*) The thorax appears small, except at the base, where it is distended, and the ensiform cartilage is sometimes abruptly curled up. (*d*) The respirations are hurried and are of the thoracic type, the abdominal movements being slight or entirely wanting. As soon as the belly-walls become moderately tense *fluctuation* is elicited by placing the palm of the left hand vertically upon one side of the abdomen, and then, with the finger-tips of the right hand, tapping lightly the opposite side; impulses thus sent through the fluid will be distinctly felt by the hand in contact with the abdomen. When the dropsical fluid is small in quantity the patient should assume the erect posture during the examination. In palpating the solid organs (liver, spleen, abdominal tumors) when ascites is present, the tips of the fingers only are placed upon the skin, and then are suddenly "dipped," displacing the fluid, thus touching the solid organ or new growth. *Percussion* gives flatness over the fluid, although some degree of resonance may be transmitted from the subjacent bowel. The upper level of dulness, in the recumbent posture, is not represented by straight transverse lines, but presents a concavity that is pointed to the head. The dulness is extremely movable, shifting with change of posture. When the decu-

bitus is supine the most dependent portions of the abdomen give dulness. Again, if the patient be made to lie on either side, the opposite or uppermost flank will be found clear, the ascitic fluid always gravitating to the bottom of the sac. Tyson has observed that the flanks are tympanitic with considerable frequency in ascites, and my experience has been similar, tympany over the head of the colon being almost constant, except in pronounced cases. Moreover, to obtain reliable results, if the layer of fluid be thin, the pleximeter finger is pressed lightly upon the surface, and the gentlest percussion only is allowable. The patient should be placed on the hands and knees if the fluid be small in amount, when a zone of dulness will be found around the umbilicus. The cardiac region may present percussion resonance as high as the fourth rib, and occasionally a murmur is heard at the base. The condition should be regarded as the counterpart of hydrothorax and not of pleuritis.

Diagnosis.—In order to arrive at a positive diagnosis a clear history of one or the other of the known causative conditions is requisite, joined with distinct evidence of the presence of fluid—viz. fluctuation and movable dulness. For the early diagnosis of ascites the patient should be placed in the knee-elbow position, when dulness can be readily elicited in the umbilical region.

The **diagnosis of chylous ascites** and **ascites adiposus** rests upon insecure ground unless aspiration be resorted to, although the presence of the causative conditions in the case may afford a basis for suspicions.

Differential Diagnosis.—Ascites is most apt to be mistaken for an *ovarian cyst*. The accompanying table presents the principal points of discrimination:

Ascites.	Ovarian Cyst.
Clinical History.	
General health is bad prior to the appearance of the enlargement.	General health is good before the development of the tumor; failure afterward.
History of disease of liver, lungs, heart, kidneys, or other organ.	Frequent history of dysmenorrhea, negative as to organic affections.
Swelling begins below and gradually extends higher; more noticeable when sitting than in the standing posture.	Swelling is unilateral at first, gradually becoming more central.
Physical Signs.	
Enlargement is symmetric, the abdomen being rounded and most prominent about the umbilicus; in the supine posture the abdomen flattens, with lateral bulging; the umbilicus is often pouched and thinned.	Enlargement is asymmetric or irregular, unless the tumor be very large, when it may fill the entire abdomen. The greatest circumference is below the umbilicus, which never bulges.
Fluctuation is general from side to side and in a vertical direction.	Fluctuation is circumscribed, corresponding to the limits of the tumor.
No aortic pulsation felt.	Aortic pulsation is sometimes evident.
Vaginal examination often shows the uterus to be movable. A pouch may project into the vagina, but no cyst is detectable.	Vaginal examination shows the uterus to be displaced. A cyst may be felt and outlined in the pelvis.
When standing, the upper line of dulness presents a concavity; rarely shows irregularities due to fluid running up into "bays" between coils of intestine.	When standing, the upper line of dulness is generally a convexity.

Ascites.	Ovarian Cyst.
Physical Signs.	
In the supine position the flanks are especially dull with tympany in front.	In the supine position dulness is still in front and the flanks are resonant.
Percussion-dulness shifts its position with that of the patient.	Percussion-dulness not movable.
Ascitic fluid has a specific gravity of 1010–1014, and is usually clear. It is of a pale straw color.	Ovarian fluid has a specific gravity of 1018–1024. It is of a thick, turbid character, and the color is variable.

Large cysts may also spring from the *pancreas* and *liver;* the elimination of the latter conditions, however, does not, as a rule, offer marked difficulty. Ascites must be distinguished in practice from the exudation due to *chronic peritonitis*, and the points of differentiation have been arranged thus:

Ascites.	Chronic Peritonitis.
A previous history of organic disease of the liver, heart, kidneys, or other organ is obtainable.	There is a previous history of acute peritonitis, tuberculosis, or inflammatory diseases of the female pelvic organs; sometimes a history of injury.
No pain is experienced.	Pain is a prominent symptom.
The abdomen is symmetrically enlarged.	Abdomen is irregularly prominent, and rarely flat.
Fluctuation is general in the transverse or vertical directions.	Fluctuation is often limited to circumscribed areas due to loculation of fluid.
Palpation detects no hard masses of irregular prominence.	Palpation often detects resistant, uneven prominences.
Percussion-dulness is always movable upon altering the position of the patient.	Dulness often not changeable on varying the position owing to adhesions.
Fluid serous, limpid, specific gravity of 1010–1014, is pale straw-yellow in color, greenish tinge at times. Contains 1 to 3 per cent. of albumin. Few cellular constituents (lymphocytes, endothelial cells, erythrocytes). Cryoscopy, freezing-point higher.	The fluid is either sero-fibrinous, sero-purulent, or milky in nature. It is often viscid, its specific gravity is 1018–1024, and its color variable; 3 to 6 per cent. of albumin. Cytologic studies show more polynuclear neutrophilic leukocytes. Freezing-point lower.

Overfilling of the bladder has been confused with ascites, and this organ has been tapped under the mistaken notion that the condition was one of dropsy of the peritoneum. Catheterization of the patient before tapping for ascites will obviate this error. Ascites may be mistaken for a deposit of fat in the abdominal wall. It is to be distinguished by pinching up the belly wall within the grasp of the hand.

Prognosis.—The duration of ascites may be many months or even years. In most instances the prognosis is unfavorable, though modified by the character of the causal condition in individual cases. The immediate cause of death may be either syncope, asphyxia, pulmonary atelectasis, or it may be the primary disease.

Treatment.—**Dietetic.**—The diet should be largely nitrogenous, light, nutritious, and given at stated periods with a view to maintaining the normal proportion of albuminous material in the blood.

Medicinal.—By means of therapeutic measures we should aim to accomplish two things: First, the improvement or cure of the original disease; and secondly, to relieve the chief symptoms by removing the

ascitic fluid on which they depend. Though the causative affection is usually chronic and incurable, every effort should be made to remove or mitigate its pernicious activity in accordance with the principles laid down in appropriate portions of this work. Of medicines used to remove the transudation, hydragogue cathartics are most potent for good, and particularly when the ascites is due to cardiac or renal disease. Calomel and jalap in combination, or salines in full doses, administered after the Matthew Hay method, should be tried. Diuretics are recommended, but are often disappointing in their effects. Rolleston points out that they sometimes appear to succeed after paracentesis. English authors greatly praise copaiba and its resin. The bitartrate and other salts of potash, either alone or in combination with juniper and digitalis, are of value Equally important with the exhibition of the above remedies is the use of tonics, including hematinics, to promote the general nutrition of the patient. I have reported one instance, occurring at the Philadelphia Hospital, in which a cure was effected perhaps solely as the result of measures intended to assist the nutritive processes. Based upon the experiments of Fleischer and Loeb, which indicate that adrenalin injected intraperitoneally hastens absorption from the peritoneal cavity, T. M. Tyson and H. D. Jump[1] employed such injections in two cases with encouraging results. On the other hand, autoserotherapy is said to retard transudation into the peritoneum and produce lasting polyuria. The fluid is to be withdrawn from the peritoneal cavity with a sterile hypodermic syringe and at once reinjected subcutaneously. The dose should be progressively larger (*e. g.*, 3, 5, 8, and 10 c.c.), and repeated at six-day intervals for two months. In ascites due to cirrhosis of the liver recourse should be had to *paracentesis abdominis*, not as a last resort only, but "as a systematic method of treatment" (Roberts). A single tapping is rarely sufficient, and a repetition of the measure from time to time, until the collateral circulation is established, is to be advised and encouraged. In cases in which the transuded serum has rapidly re-formed after its removal by tapping, Southey's tubes, by means of which permanent drainage is secured, have been used with good results. Drummond affirms that ascites due to liver-cirrhosis can be cured, and has proposed an operation whereby adhesions between the abdominal contents and its parietes are secured, in which new blood-vessels are formed, thus establishing a collateral circulation.

NEW GROWTHS IN THE PERITONEUM.

THE most frequent and important of the new growths of the peritoneum are (*a*) carcinoma and (*b*) tuberculous deposit and tuberculous peritonitis, the latter two having been already considered.

CARCINOMA OF THE PERITONEUM.

There occur the usual varieties—scirrhous, encephaloid, and colloid—the latter most frequently involving the omentum. Primary carci-

[1] *Therapeutic Gazette*, January, 1911.

noma of the peritoneum is rare. Primary endothelioma, however, is occasionally met with. It resembles true carcinoma in macroscopic as well as in microscopic appearances, though it is in reality to be ranked with the sarcomata on account of its origin. Carcinoma of the peritoneum is almost always secondary to carcinoma of the stomach, liver, or pelvic organs. The peritoneum may either be the seat of numerous small round miliary tumors, or, less commonly, of larger nodular masses, the most extensive development being presented by the colloid variety. Cancerous peritonitis is often found to be an associated condition, and the retroperitoneal lymph-glands may show cancerous development.

Etiology.—More cases occur in the female sex than in the male. Age has also a potent influence, most cases appearing late in life. Trauma may operate as an influential causative factor.

Symptoms.—When *primary*, carcinoma of the peritoneum is obscure during the early part of its course. Local *pain* and discomfort are complained of, and clinical evidences of the *cancerous cachexia* develop early, but these symptoms are not at first striking enough to be entirely characteristic. Later, however, the *nodules* can often be plainly felt (unless the liquid effusion be too marked), and the *ascites* (blood-stained), *loss of flesh*, *weakness*, and *anemia* are now sufficiently developed for diagnosis. In the colloid variety ascites is often absent, the abdominal cavity being the seat of a large, semisolid, non-fluctuating mass.

The *secondary form* usually follows carcinoma of the stomach or the ovaries, and the cachexia will have been developed before the peritoneum is secondarily involved in consequence of the presence of the primary growth. Hence, any symptoms referable to the general abdominal cavity are strongly suspicious. Among other *constitutional symptoms*, apart from those already mentioned, is fever (rarely absent), which may be due in small measure to the anemia, though in a greater measure to the associated peritonitis.

Physical Signs.—The abdomen protrudes if effusion be present or if the carcinoma be of the colloid form, though not invariably. Even when the tumor is large, dropsy of the peritoneum may make its detection impossible. On practising palpation after tapping, however, the nodules can be made out, either extending from side to side or being more or less localized and not adherent to underlying structures.

Differential Diagnosis.—It will be remembered that an oblong tumor lying transversely across the abdomen below the stomach is met in certain forms of *chronic peritonitis*. This offers the same physical signs that are presented by peritoneal carcinomata, unless the tumor-masses in the latter affection be of considerable size. Carcinoma, however, is most apt to occur in persons past middle life, while nodular tuberculous peritonitis appears almost exclusively in children and young adults. Evidences of tuberculous disease elsewhere, past or present, and particularly suppuration about the umbilicus, would point to tuberculous peritonitis. Moreover, in all forms of abdominal carcinoma the inguinal glands are apt to be indurated and enlarged. Cyto-diagnosis might serve to distinguish carcinoma from tuberculosis of the peritoneum. *Proliferative peritonitis* usually gives a history of chronic alcoholism. The differentiation of *hydatid cysts* of the peritoneum from carcinoma depends upon the history of the case, the presence of hydatid fremitus, the find-

ing of the hooklets in the fluid, the less rapid growth of the tumor, and the lessened amount of pain, fever, and cachexia in the latter disease. *Carcinoma of the intestine* may simulate somewhat the disease under consideration, but the signs of increasing stenosis, as evidenced by the colicky pain, the discharge of blood and pus with the stools, and the ribbon-like character of the feces, will serve to separate the conditions. *Retroperitoneal tumors* (sarcomata) are discriminated with the greatest difficulty. As pointed out by J. D. Steele, in tumors behind the peritoneum the signs of intestinal obstruction, coupled with neuralgic pains or edema of the lower extremities from pressure upon their nervous and venous supply, are important discriminating features. Moreover, tumors of the peritoneum, whether of the omentum or mesentery, are movable, while those behind the peritoneum are generally fixed. In retro-peritoneal sarcoma "the tumor may fluctuate and may move with respiration, or be movable by palpation." Omental tumors lie in front of the intestines (as can be shown by inflation of the bowel); mesenteric new growths sometimes have a coil of intestine in front of them. On the other hand, retro-peritoneal tumors are always crossed by loops of intestine (colon). Peritoneal tumors (particularly the omental) follow the movements of respiration, while the retro-peritoneal are, as a rule, immobile. The latter always cross the central long axis of the body, while the former may be confined to one or the other side. Finally, the only sure method of determining the character of tumors behind the peritoneum is by an exploratory celiotomy.

The prognosis is always unfavorable.

Treatment can accomplish nothing beyond a more or less complete relief from the distressing symptoms.

Other Tumors of the Peritoneum.—Primary sarcoma produces larger or smaller areas of thickening of the peritoneum. Secondary sarcoma, the commoner variety, assumes the form of large nodular masses or of numerous miliary growths. The symptomatology has been given under Carcinoma of the Peritoneum.

Fibromata and *lipomata*—the former as fibroid nodules varying in size from a millet-seed to a split pea, the latter as localized overgrowths of fatty tissue showing great variation in their size—are among peritoneal and retro-peritoneal neoplasms. The lipomata, however, are the more frequent. Mr. Anderson points out that fibromata may merge, on the one hand, into the lipomata (*fibro-lipomata*); on the other, into the myomata (*fibro-myomata*). It is probable that lipomata usually spring from the retro-peritoneal tissue in the neighborhood of the kidneys and iliac fossa. Less commonly, however, they "originate in the subperitoneal tissues of the mesenteric or omental folds, where general fatty overgrowth in varying degree is frequently observed" (Allchin).

Peritoneal lipomata may be associated with extreme obesity, but this is by no means invariably the case. The *diagnosis* is rarely made, particularly in the female, owing to the close resemblance of these growths to ovarian cysts and other tumors found in connection with the female genitalia. They have been mistaken also for ascites, which is not rarely a symptom of fibromatous and lipomatous neoplasms. The *prognosis* is unfavorable, although, if early recognized, the tumors may be successfully removed.

PART VIII.

DISEASES OF THE URINARY SYSTEM.

I. DISEASES OF THE KIDNEY.

MOBILITY OF THE KIDNEY.

(*Movable Kidney; Dislocated Kidney; Floating Kidney; Wandering Kidney; Ren Mobilis; Nephroptosis.*)

Definition.—A distinction is made between two common varieties of mobile kidney, according to the degree of displacement, as follows: (1) *Movable kidney*, the upper end of which can be felt during deep inspiration, and which can be pushed down in the retro-peritoneal space to the level of the umbilicus; (2) *Floating kidney*, which is freely movable below or beyond this point—*i. e.* possessing a larger arc of mobility. In the so-called *palpable kidney* the lower edge of the organ can barely be felt on deep pressure.

Etiology.—The condition may be congenital (rare). An abnormally long renal artery may predispose to the development of a movable kidney. Emaciation with a marked wasting of the fatty capsule in which the kidney is imbedded is a frequent underlying cause of movable kidney. Women are oftener affected than men, and relations from multiple pregnancies, tight lacing and girdling, and traumatism (falls, heavy lifting, and the like) have frequently caused displacement and mobility of the kidney. Suckling[1] observed that a number of girls who served beer, and were therefore obliged to stoop and immediately stand upright with considerable frequency, were likely to have movable kidney. Heavy tumors of the organ, the pressure of adjacent tumors (as of the liver), and the traction of hernias may likewise cause the condition. Watson's[2] series of experiments proved that the structures vital to the restriction of the kidney's mobility within its normal excursion are those which form the attachments along its posterior surface and upper pole.

In *enteroptosis*, or Glénard's disease, in which there is a downward displacement of all the viscera, mobility of the kidney is often associated. Although either kidney, or even both kidneys, may be abnormally mobile, the right one is usually affected. Sometimes a floating kidney becomes fixed by peritoneal adhesions in an abnormal position, as in the right iliac fossa; an instance of this occurred in a seaman, under my care, admitted to the Medico-Chirurgical Hospital of Philadelphia.

"The body form is an important etiologic factor of movable kidney,

[1] *Edinburgh Med. Jour.*, Sept., 1898.
[2] *Boston Med. and Surg. Jour.*, vol. cxlv., No. 12.

and not only explains the greater frequency in women than in men, but also the reason why the right organ is more often displaced than the left" (Ashton). The body cavity is subdivided into three zones by two transverse planes, and in women the middle zone is liable to be contracted in various directions with a consequent displacement downward of the organs occupying this region. The right kidney is pushed downward, owing to backward compression of the liver, which tilts its superior pole forward. Becker and Lenhoff, from a study of the relation between the length and circumference of the body cavity, found that the greater the contraction of the middle zone, the higher will be the body index; this is arrived at as follows:

$$\frac{\text{Distance from suprasternal notch to symphysis}}{\text{Circumference of body at lower border of tenth rib}} \times 100 = \text{body index.}$$

They concluded that when the body index was below 75 no displacement occurred, but when the index was above 77 the kidney was almost invariably situated abnormally low.

Symptoms.—Movable kidney may exist without any symptoms whatever. It may be discovered accidentally by physical examination, and not infrequently it is found *postmortem* in a similar manner.

The symptoms of movable kidney are local, reflex, and general, the *local* and *reflex symptoms* being the most prominent in the average case. The reflex symptoms, though usually abdominal, may become general. The local symptoms are most marked in extreme mobility of the kidney (floating kidney), although many of the symptoms are due to the associated sagging of other organs, while in moderate mobility the reflex symptoms usually predominate over the local.

Most frequently there is a troublesome dragging pain, or a sense of weight or pressure in the loins or abdomen, especially after long walking or standing or hard labor; this may be referred to the sacral region. Sometimes the pain may be quite sharp and colicky in nature. Pain in the kidney itself is seldom complained of, due to congestion by pressure or traction upon the renal veins or obstruction of the ureter.

Reflex gastro-intestinal disturbances are common. *Indigestion* is usually complained of, and occasionally vomiting and nausea are noted. Dilatation of the stomach may possibly be caused by a dislocated kidney pressing upon the duodenum. Pressure-jaundice is also an unusual concomitant of the floating kidney, and J. Hutchinson, Jr., records two cases that caused both hepatic colic and obstructive jaundice. *Cardiac palpitation*, constipation, flatulence, and edema of the lower extremities (from pressure on the inferior vena cava) may attend, and disturbances of the pelvic viscera have also been noted occasionally (dysmenorrhea, abortion, and irritable bladder). Improvement is usually considerable in pregnancy. Some cases of displaced kidney are characterized by sudden and severe attacks of nephralgic or gastralgic pains, chills, fever, vertigo, nausea and vomiting, and general collapse. These attacks are often periodic, occurring sometimes at the menstrual period, and are known as "*Dietl's crises*" or "*incarceration symptoms.*" They may be excited, also, by a too free indulgence in eating and drinking. It is most probable, as Dietl himself suggested, that these cases are due to a twisting or bending of the renal vessels or of the ureter, or, perhaps, to circumscribed inflammation of the mobile kidney. An acute hydrone-

phrosis may thus develop. The urine is concentrated, and may contain uric acid or oxalates in excess. After three or four days, as the attack subsides, micturition becomes free, the kidney becoming movable once more. These attacks of *transitional hydronephrosis* may occur intermittently (*vide* Hydronephrosis). Pyonephrosis has also been noted and in rare instances may result in gangrene by occlusion of blood-vessels. When a loose kidney causes rigidity of the muscles of the abdominal wall ("splint-belly"), it may produce gastric hyperacidity, congestive appendicitis, or parenchymatous nephritis.

Floating kidney associated with Glénard's disease, in which the transverse colon, pancreas, stomach, intestines, and other viscera are prolapsed, gives rise to symptoms similar to those stated above, only with the addition of greater discomfort and nutritive and nervous disturbances. Sometimes there is albuminuria and rarely hematuria.

The *general symptoms* of movable kidney are those of neurasthenia or hysteria. Mental anxiety, leading to melancholia, sometimes follows the discovery of a movable abdominal tumor. Cephalalgia, backache, paresthesias, neuralgias, nervous dyspepsia, hypochondriasis (in men), and hysteric manifestations may arise and prove a perpetual annoyance. The condition, however, may arise in a previously neurasthenic or hysteric subject.

The **physical signs** of movable or floating kidney are highly important and diagnostic. *Palpation*, especially bimanual, as by Israel's method. The patient lying in a semi-recumbent position, counter-pressure (the left hand being placed over the lumbar region, the right next the skin in front, manipulating the abdomen from above downward) may detect a firm, movable tumor of renal size and shape in either flank (usually the right) just below the ribs (movable kidney), or in the inguinal or umbilical regions (floating kidney). Or, the patient may stand and, grasping the back of a chair, may lean slightly forward, while the examiner, at the patient's side, presses with one hand over the loin, and with the other feels over the abdomen below the ribs. Though comparatively easy to outline, the tumor is nevertheless hard to grasp; it is often, however, readily pushed into place. Deep breathing may affect a palpable or movable kidney, but has no effect upon one that freely wanders about the abdomen (floating kidney.) Pulsation of the renal artery may be felt in the last-named cases.

Inspection and *percussion* of the lumbar region in movable kidney are uncertain, and therefore unreliable. Visible depression here is rarer than a visible tumor anteriorly. I have noted increased tympany over the affected side as compared with the opposite side.

A diagnosis is possible only after a careful and thorough physical examination. When this is made, an abnormally mobile kidney is usually discovered without difficulty. The size and shape of the organ, its right-sided position, and its mobility, associated with a train of local, reflex, or general nervous disturbances, especially in a thin, emaciated woman, are quite distinctive. A knee-elbow posture is sometimes more favorable than the recumbent position for determining a movable kidney.

Differential Diagnosis.—Floating kidney is, of course, more easily diagnosticated than the movable type, and partly because of the fact that in instances of the latter *tumors of the gall-bladder* especially and *wandering spleen* must first be excluded. The absence of a well-defined splenic notch, the presence of pulsation of the renal artery,

a tympanitic note over the usually intervening colon, and an unchanged area of splenic dulness will assist in the diagnosis; in addition there is the fact that wandering spleen is a comparatively rare affection.

Tumors of the gall-bladder are frequently mistaken for movable kidney; occasionally the opposite error is made; sometimes both conditions may exist. They are both common to women; they both may present as tumors in the right hypochondriac and umbilical regions; they are more or less movable, firm, smooth, slightly tender, round or oval in shape, with variable percussion-signs and dyspeptic symptoms; and either may give rise to paroxysms of severe colic or to jaundice. Jaundice, however, is rare in movable kidney, while emaciation and general nervous disorders are more common; the floating tumor may vary in size (hydronephrosis), the diminution being accompanied by a marked increase in the flow of urine. If the gall-bladder be filled with calculi, the consistence is firmer than that of the kidney, and fremitus may be felt. Moreover, the movements of the gall-bladder are usually lateral within a short arc of a circle, the center of which is a point beneath the edge of the right lobe of the liver; while those of floating or movable kidney may be either vertical, oblique, or lateral in arcs of a much larger radius. Again, tumors of the gall-bladder descend with inspiration, as is not the case with wandering kidney.

In some cases it is necessary to distinguish between "Dietl's crises" and *renal*, *hepatic*, or *intestinal colic*, acute intestinal obstruction, affections of the genital organs, and appendicitis. *Tumors of the ovaries and bowel* are rarely confounded with wandering kidney.

Prognosis.—In uncomplicated cases life is never endangered, and a cure may be effected in numerous cases in which suitable combined medical and surgical treatment is pursued. The general nervous symptoms are usually very obstinate, but after relief is afforded from the accompanying local symptoms, they subside or cease altogether.

Treatment.—Since emaciation and loss of perirenal fat is a frequent cause of wandering kidney, it is often advisable to resort to measures that will tend to increase the weight and fat of the body. The "rest-cure," with its forced feeding, may be all that is necessary in highly nervous subjects having but a slightly movable kidney. In all cases more or less prolonged intervals of rest (lying down) throughout the day aid markedly in ameliorating the symptoms. Other hygienic measures, as the avoidance of over-exertion, extreme bodily movements, straining—as at stool—and so forth, should also be enjoined.

For several years, and until recently, the operation for anchoring the mobile kidney has been advised as appropriate in nearly all cases. This is now perhaps wisely deprecated; and a reversion to the careful, patient, and constant use of suitable abdominal pads and binders in certain cases is meeting with much success. Watson states that from 90 to 95 per cent. of movable kidney producing symptoms can be relieved by a suitable *corset*. Gallant [1] recommends a corset as long in front as can be worn; specially made or straight-front corsets being chosen. It must be at least two inches less than that formerly worn, and laced at the back from top to bottom as an open V. Having put the corset around the waist,

[1] *Saunders' American Year Book*, 1903, p. 453.

the patient lies down, draws up the knees, and then fastens the corset from below upward, drawing the lax abdominal wall up at each step. In severe cases of renal displacements, in which recurring attacks of hydronephrosis, strangulation-crises, pain with marked gastro-intestinal disturbances, profound nervous and mental disturbances, or other grave renal complications occur, some such surgical procedure as nephrorrhaphy should be strongly urged. This may prove an effectual cure, although the anchorage is often torn loose by a sudden or severe physical effort. Suckling states that surgery offers the only cure for nephroptosis. The hypodermic injection of morphin and atropin and the external application of heat are indicated in the crises of Dietl.

CIRCULATORY DISORDERS OF THE KIDNEYS.

ACTIVE HYPEREMIA.

(*Acute or Active Congestion.*)

Definition.—An acute, temporary engorgement of the vessels of the kidneys, with little or no exudation.

Pathology.—The kidney is swollen, deep-red in color, and engorged with blood, which flows freely on section. *Microscopically*, there may be seen cloudy swelling of the cortical epithelium.

Etiology.—Acute renal congestion is due mainly to the action of irritants present in the circulation, as in the acute infectious (especially the eruptive) fevers. The stimulating diuretics and certain poisonous drugs, as copaiba, squills, cantharides, potassium chlorate, and carbolic acid, also sudden contraction of the peripheral blood-vessels by exposure to cold while the body is overheated, act as causes. Post-operative acute hyperemia (ether?) is frequently met with. When prolonged the congestion passes into an acute nephritis. It may be caused in one kidney as a result of either nephrectomy of its fellow or blocking of the ureter by a calculus, clot, etc., of the opposite side.

Symptoms.—There may be a dull pain in the lumbar region, with a slight elevation of the temperature and pulse-rate. The ***urine*** either is scanty, or, as in cantharides-poisoning, it may be altogether suppressed. It is dark, the specific gravity is increased, and it contains some free blood, a trace of albumin, and a few hyaline tube-casts.

Diagnosis.—The absence of a marked quantity of albumin, of the numerous and various casts, of dropsy, and of uremic symptoms distinguishes active hyperemia from *acute nephritis*.

The **prognosis** is quite favorable upon the removal of the cause. A frequent repetition of the attacks, however, may lead to a nephritis.

Treatment.—Absolute rest and a liquid diet should be ordered. Cupping over the loins or the use of hot fomentations should be practised. The free use of water and other diluents or mucilaginous drinks should be encouraged. Saline laxatives to freely open the bowels, and the use of hot air or a hot pack to promote sweating, are important aids in relieving the congested kidneys.

PASSIVE HYPEREMIA.

(*Chronic or Passive Congestion.*)

Definition.—A chronic venous engorgement of the renal vessels, generally secondary to diseases of certain other viscera.

Pathology.—There is in the later stages a characteristic condition of the kidneys called "cyanotic induration." Earlier in the case the organs are enlarged, firm, and of a dark, bluish-red color. The capsule is usually non-adherent. On section the medullary substance is seen to be darker red than the cortex and coarsely fibrous in appearance. Microscopic examination shows the capillaries (both glomerular and medullary) somewhat dilated and the walls thickened. The epithelium may either be unchanged or a little cloudy and swollen, or, later, even fatty; the interstitial tissue may be slightly increased.

Etiology.—Most commonly the renal congestion is a part of a general venous engorgement due to chronic cardiac, pulmonary, or hepatic disease. It is found in mitral valvular disease with ruptured compensation of the heart (common); in pulmonary emphysema, fibroid phthisis, and chronic adhesive pleurisy; and in cirrhosis of the liver. Less frequent causes of congested kidneys are tumors, the pregnant uterus, and ascites, all of which bring about the condition through pressure upon the renal veins. Again, angulation, as in nephroptosis, kyphosis, and the like, may be a cause. Only rarely may passive renal congestion be due to thrombosis or embolism of the ascending vena cava or of the renal veins.

Symptoms.—These are accompanied by those due to the primary diseases that are manifested in the general venous congestion, as *edema* of the lower extremities. There may be a sensation of weight in the loins. The *urine* is diminished in quantity, of a higher specific gravity, and darker in color; it contains a little albumin, some blood-corpuscles, and a few hyaline casts and epithelial cells, depending upon the chronicity and intensity of the congestion. Urates may be deposited in the standing urine.

Diagnosis.—From *nephritis* passive renal congestion may be differentiated by the comparative absence of albumin, casts, general dropsy and uremia, and by the undiminished quantity of urea.

Prognosis.—This depends upon the primary cause. Chronic congestion may pass into chronic nephritis with fluctuation of the oliguria and albuminuria according to the functional activity of the heart.

Treatment.—Rest and a light and easily assimilable diet, together with cardiac tonics and diuretics, are indicated. The infusion of digitalis serves a good purpose by increasing the quantity of urine and clearing it of albumin. Basham's mixture is a useful adjuvant.

EMBOLIC INFARCTIONS.

Anemic and hemorrhagic infarctions of the kidney are of pathologic rather than of clinical significance. Cicatrices may result from these infarctions—"embolic contracted kidney." Very rarely the *sudden appearance* of a slight amount of *blood* in the *urine*, associated with cardiac disease, and tenderness of the kidney, and possibly with a sudden severe pain over the loin, may point to hemorrhagic infarction.

SPECIAL PATHOLOGIC STATES OF THE URINE.

HEMATURIA.

Definition.—The presence of blood in the urine.

Etiology.—(1) *Local* or *renal causes* of hematuria include congestion (including that due to torsion of the renal vessels in certain cases of floating kidney), acute inflammation of the kidneys, and acute exacerbations of chronic nephritis, embolic hemorrhagic infarction, renal calculi and pyelitis, tuberculosis, malignant renal disease, diffuse myxangiomatous condition of the pelvic submucous tissue (Myles), actinomycosis (O. Israel), hydatids, traumatism, and parasites (the *filaria sanguinis hominis* and *distoma hæmatobium* (Billharz).

(2) *Affections of the Urinary Tract.*—*In the ureter*, calculi or lacerations due to traumatism, as in protracted abdominal sections; *in the bladder*, calculi, malignant tumors, acute cystitis, ulceration and rupture of varicose veins at the vesical neck; and *in the urethra*, gonorrhea, calculi, parasites, and traumatism—may all cause hematuria.

(3) *General Diseases.*—Acute specific fevers and certain blood-dyscrasiæ (purpura, gout, scurvy, hemophilia, malaria, and leukemia) may produce hematuria. Malarial hematuria in mild form is not an uncommon feature of paludism in the Middle States of this country, and may occur after the manner of intermittent malarial paroxysms. That due to the renal congestion of chronic heart-, lung-, or liver-disease is not a marked condition, and has not been of frequent occurrence in my experience.

(4) *Essential Hematuria.*—Senator describes a form of hematuria that is sometimes seen in young persons whose health may be quite fair, the blood often appearing paroxysmally and without apparent cause ("renal hemophilia"). The view is gaining ground, with added experience, that so-called symptomless bleeding from the kidney is usually due to localized disease in the cortex. There is an idiopathic (family) or congenital hematuria. Hematuria may be also a manifestation of vicarious menstruation.

(5) *Endemic hematuria* is that variety found in some of the tropical regions where the distoma hæmatobium (a trematode worm) abounds.

Diagnosis.—This has for its object the discovery (1) of blood in the urine, and (2) of the source of the hemorrhage. Bloody urine varies in color according to the quantity of blood present, to its condition (coagulability), disposition, and the length of time present in the urine. A light reddish tinge or "smoky" hue may indicate a slight quantity of blood. A dark coagulum may be at the bottom as a sediment, with small clots floating above in a deep-red, turbid layer, above which, again, the urine may show but the slightest tint of red. *Microscopically*, the blood-corpuscles are readily discovered, establishing the diagnosis from hemoglobinuria, in which condition they are absent.[1] When red corpuscles are associated with tube-casts, renal hemorrhage may be positively diagnosed. In ammoniacal urine the corpuscles are very pale and shadowy (dissolved hemoglobin). After remaining in ordinary acid and diluted urine they lose their disk-like shape and swell into spheres of a smaller diameter. Urine containing blood always shows the presence of albumin. According to Newman[2] a ratio of albumin to hemoglobin in

[1] Hutchinson and Rainy, *Clinical Methods*, p. 337, point out a source of fallacy: "In alkaline urines, especially if they have stood for some time, the red cells are apt to swell up and disappear."

[2] *The Lancet*, July 9, 1898.

excess of 1 to 1.6 indicates not only an independent albuminuria but also a renal affection as the cause of the hematuria.

Chemically, the blood-pigment may be detected by Heller's test, which consists in adding liquor potassæ, boiling the urine, and observing the flakes of precipitating phosphates, which become reddish-yellow or brown from the added hemochromogen. The guaiacum test is also used. The spectroscope is sometimes employed to discover the bands of alkaline hematin in the precipitate which is conclusive. Leede recommends the accumulation of as many erythrocytes as possible by filtering a large amount of urine and applying the guaiac, or other chemical, tests to the residue left on the filter.

The *source of the blood* in hematuria is of great diagnostic and therapeutic importance. In *renal hemorrhage* the blood is thoroughly mixed with the urine, giving a uniformly red, "smoky," or brown color (due to methemoglobin), as in hemorrhagic nephritis. Blood-casts and leukocytes may also be found. The disease causing hematuria may be traced sometimes by a study of the urine; thus, in valvular cardiac disease the sudden appearance of hematuria would indicate *infarction* of the kidney. The discovery of a few red blood-corpuscles in a concentrated urine would point to renal congestion. In profuse renal hemorrhages clots representing moulds of the renal pelves and of the ureters may be discharged. Hemorrhage due to *calculus* is usually small in amount and appears at more or less prolonged intervals. Tubercular hemorrhages may occur. Blood from the ureters is usually moulded in clots in the shape of curved cylinders, and appears like small dark worms in the urine. Casts from the ureters are often secondary to hemorrhages; in such cases the hematuria may alternate with the passage of clear urine, owing to temporary hemorrhages or to the blocking of the ureter on the diseased side. (See also Fibrinuria.)

Vesical hemorrhages may be copious. The blood and urine are not intimately mixed, and large clots settle on standing. The first portions of urine discharged may not be bloody, while the last may consist of pure blood. *Urethral* blood is discharged before the urine, and either comes away freely or may be "milked out" independently of urination.

The endoscope has been used successfully to determine the source of the hemorrhage (which kidney?). It is especially useful in women.

Prognosis.—This varies with the primary source of the hematuria.

The **treatment** consists primarily in rest in bed. The application of dry cold to the loins is useful, and the hypodermic injection of ergotol is to be recommended for trial. Internally, such hemostatics as the extract of hamamelis virginica, the extract of hydrastis canadensis, gallic acid, lead acetate, calcium chlorid, ergot, and opium may be used. Cantharides tincture in 2- to 5-drop doses has been tried with good results in hematuria due to renal congestion. The good results following the use of a 10 per cent. solution (a pint daily) have attracted some attention recently.

HEMOGLOBINURIA.

Definition.—The presence of blood-pigments, especially methemoglobin, in the urine.

Etiology.—The direct cause of hemoglobinuria is a condition of the blood in which, as a result of the dissolution of the red corpuscles, the hemoglobin is set free and is excreted by the kidneys.

(1) The **causes of the hemolysis** are principally *toxic*, and include the following: (*a*) Poisons (carbolic and pyrogallic acids, potassium chlorate, naphtol, phosphorus, arseniuretted hydrogen, and carbon dioxid). (*b*) The ingestion of poisonous fungi or of tainted edible mushrooms (*Helvella esculenta*). (*c*) The poisons of certain infectious diseases (scarlatina, typhus and typhoid fevers, yellow fever, syphilis, scurvy, purpura). (*d*) Extensive burns, the absorption of hemorrhagic effusions, and the transfusion of animal blood. (*e*) Rarely it may be due to exposure to cold and to violent physical exertion. (*f*) The so-called *epidemic hemoglobinuria* (Winckel's disease) occurring in the newborn.

(2) **Paroxysmal hemoglobinuria,** a rare variety, may occur without any apparent cause in persons enjoying otherwise good health. It appears as an independent disease and the pigment present in the urine consists largely of methemoglobin. Hemolytic experiments by Ruziezka and Levadet show that the phagocytes attack the erythrocytes, in consequence of the union of an intermediary body (toxin) with the red corpuscles in this disease. The toxin is driven into the blood during or before the paroxysm by marked exertion or chill. It is held by some to be a manifestation of Raynaud's disease, uricemia, and by others to be due to syphilis.

(3) It appears as a symptom of *malaria* in the southern part of this country. This is termed *malignant malarial hemoglobinuria* or *hemoglobinuric fever*. In Africa it is called *black-water fever*.

Symptoms.—These are generally the symptoms of the condition that accompanies hemoglobinuria. In paroxysmal hemoglobinuria the attacks are usually sudden, brief in duration, and sometimes *intermittent*, especially when of malarial origin. An anemic condition seems to be essential to the production of malarial hemoglobinuria. *Jaundice* may be an associated symptom. The hemoglobinuria seldom lasts for more than two days, though very grave cases take on the aspect of a pernicious malarial attack. There may be lumbar pains, chills and fever, and gastric disturbances. Urticaria and purpura have also been noted, as has anemia in cases in which frequent attacks have taken place.

Diagnosis.—This is made by an examination of the urine. Macroscopically, it is of a red-brown color, slightly turbid, with a reddish-brown or brownish-black sediment. The reaction is usually acid, and the specific gravity slightly lowered. The microscopic features that distinguish hemoglobinuria from hematuria are variable. In the former condition few or no red corpuscles are present, and the few that may be seen are usually colorless ("shadows") or fragmentary. Small flakes or granules of disintegrated hemoglobin are found; and are brownish-black in color. There may be also brown-tinged casts and epithelium. Chemically, the urine is found to contain albumin, for the discovery of which Heller's and the guaiac tests for blood-pigment may be tried. The former has been described in the preceding discussion of Hematuria. The *guaiac test* consists in overlaying with urine a mixture of the tincture of guaiac and hydrogen peroxid or the oil of turpentine (equal parts). When the blood-coloring matter is present, an indigo-blue ring is formed above a white resinous deposit. When shaken a lighter blue color develops throughout the contents. By means of the spectroscope the three absorption-bands of methemoglobin may be seen (red, green, and yellow). The blood-serum in hemo-

globinuria may be somewhat red-tinged on account of the dissolved hemoglobin. The hemoglobinuria is further marked by the aplasticity of the red corpuscles, by their pallor, by poikilocytosis, and by the presence of the irregular flakes of hemoglobin.

The **prognosis** is favorable in the ordinary paroxysmal form. Malignant malarial hemoglobinuria, however, is often fatal.

Treatment.—Hemoglobinuria is rather intractable. During the paroxysms external warmth is needed, along with hot drinks to encourage perspiration. In malarial cases quinin, and in syphilitic the iodids, should be administered; although by some it is believed that quinin may aggravate the syndrome in particular cases.

ALBUMINURIA.

Definition.—The presence of albumin in the urine.

Pathology and Etiology.—The immediate cause is the escape of the normal blood-constituents, serum-albumin and serum-globulin, from the vessels into the renal tubules. This transudation of albumin indicates either a transient and slight or a permanent and grave nutritional disturbance of either the epithelium lining the glomeruli or of that of the contained tufts of capillaries, or, possibly, of the *membrana propria* or the epithelium of the uriniferous tubules. These changes induce and offer an abnormal perviousness to the albumin of the blood.

The principal *causes* of albuminuria are—(1) Those associated with definite lesions of the kidney; nephritis, acute and chronic; renal congestions, active and passive (the latter being secondary to chronic liver-, heart-, and lung-disease, pregnancy, or tumors); and certain *toxemias*. Among the last-named are included scarlet fever (scarlatinal nephritis) and gout. Other causes are—amyloid and fatty degeneration of the kidney, suppurative nephritis, and renal tumors (cystic kidney).

Albuminuria occurs also in conditions in which (2) the renal lesions are either slight or undemonstrable: (*a*) Thus, it is present in blood-changes, as in chronic lead-, mercury-, and arsenic-poisoning, scurvy, purpura, syphilis, leukemia, or extreme anemia, and in cases in which urobilin or bile-pigment and sugar (glucose) circulate in the blood. Again, slight albuminuria may be present in pregnancy, in saccharin diabetes, and after etherization. In certain affections of the *nervous system* albumin is found in small quantity, as after an epileptic paroxysm, in tetanus, apoplexy, and exophthalmic goiter.

(*b*) The so-called *accidental* or *spurious* albuminuria is due to the presence of pus or blood; in such cases the condition is not a true renal albuminuria, since it is commonly associated with cystitis, pyelitis, urethritis, or is the result of hemorrhage from the pelvis of the kidney, from the ureters, bladder, or urethra. Chemical injury of the bladder may cause a reflex albuminuria (Evans, Wynne, and Whipple).

(*c*) *Febrile* albuminuria is of rather frequent occurrence in diseases accompanied by pyrexia, especially when long continued. Among these are typhoid fever, small-pox, yellow fever, diphtheria, and even influenza, follicular tonsillitis, and pneumonitis. The renal changes in these cases are, I believe, merely a transitory cloudy swelling in the glomeruli, which, together with the albuminuria, rarely lasts longer than the fever.

(*d*) Other forms of albuminuria have been styled *physiologic* or *functional*, *transient*, *dietetic*, *neurotic*, *intermittent*, and *cyclic*: in these no

definite lesions of the kidney are found, and are denied by some to exist. Recent observers are inclined to believe that trivial, non-progressive renal changes occur in these cases. Slight albuminuria certainly does occur in some cases after a heavy meal rich in albumin, after marked and prolonged muscular exertion, intense emotion, and cold bathing.

(*e*) *Cyclic albuminuria* has come to be of greater interest and importance in later years, particularly as it bears upon the prognosis and upon life-insurance risks. In this variety there are a periodic appearance and absence of albumin in the urine. The albuminuric paroxysms are very variable, recurring usually after meals or on exertion, according to some, largely the result of the assumption of the upright posture upon rising from bed, but generally being absent during rest at night and during the evening hours. The albumin is present in but small quantity, and only rarely are casts (hyaline) found. The accompanying signs and symptoms common to nephritis are absent. Cyclic albuminuria is most common in adolescent anemic males of poor nutrition (gastro-intestinal auto-intoxication?), dyspeptic, neuralgic, often neurotic, and even hysteric. Under careful management these cases ordinarily recover. There is, however, a class of cases in which the albuminuria is persistent even after fasting, though but a mere trace of albumin may be detected. After the administration of a diuretic or on stimulating the heart the albumin may decrease in amount (Edel). However, an insidious degeneration of kidney-structure may manifest itself many years later. Albuminuria may rarely be *hereditary* (Renault). Slight *senile albuminuria*, without evidence of renal disease, is not uncommon.

Orthostatic Albuminuria.—This is a variety of albuminuria caused by the upright posture (Aubertin). It appears only after standing. Aubertin reports 4 cases, and he concludes that orthostatic albuminuria represents the terminal stage of a nephritis, or some disturbance of the renal circulation. Engel ascribes orthostatic albuminuria to an existing nephritis, in which the cells of the kidney, however, recuperate when the patient lies down. Gillett has well said the majority of cases of cyclic albuminuria are also orthostatic. Lordosis, with or without movable kidney, may be responsible for the development of the albuminuria (Jehle).

Diagnosis.—This rests upon the discovery of albumin in the urine. For the diagnosis of cyclic albuminuria, specimens of urine passed at different times of the day must be examined.

Differential Diagnosis.—Inquiry and careful inference concerning the etiology of a given case must be made. *Renal albuminuria* is persistent and of considerable quantity, except in chronic interstitial nephritis. Tube-casts are usually present. *Functional albuminuria* is slight and inconstant. Tube-casts are usually absent in the latter. Again, in the former variety, general symptoms, as dropsy, cardiac hypertrophy, anemia, and uremic prodromes, are present. It is true that slight edema is sometimes found in cyclic albuminuria, but this is probably due to the marked anemia that is so often seen. Such conditions as gleet and leukorrhea must also be excluded.

Tests for Albumin.—Two samples of urine, one of the morning before any food is taken, and one of the evening before the patient retires, should be examined. The smallest quantity can be detected only by its coagulum rendering the urine turbid; hence any turbidity present before the given test is made should be removed by filtration, unless this tur-

bidity be due to urates, when a little warming of the tube will render the urine clear.

(1) *Boiling Test.*—This is the commonest and I think the most reliable practical test for albumin. The tube is filled about two-thirds full of urine. If alkaline or neutral in reaction, a drop of acetic or nitric acid is added; an excess of acid must be carefully avoided, lest the albumin (if present) be converted into a non-coagulable form. The tube, held aslant, is then applied to the flame, and slowly revolved with the fingers, so that the upper portion of the column of urine is brought to the boiling-point. A comparison of this with the lower portion of the urine is made. Any turbidity is due to albumin or phosphates. If albumin, adding a few drops of nitric acid will increase and thicken the coagulum; if phosphate, the opaqueness will be cleared at once.

(2) *Heller's Nitric-acid Test.*—This is both delicate and satisfactory. About 1 c.cm. of nitric acid is poured into a tube, and some urine is allowed to flow slowly from a pipet and settle upon the acid. The presence of albumin is indicated by a white ring at the point of contact of the two liquids. Uric acid, urates, and certain urinary coloring-matters form a pink or deep-red ring or zone; this forms, as a rule, above the juncture of the acid and urine. Hemialbumose also gives a white zone, but does not respond to the boiling test as does serum-albumin.

Boston's Pipet Method.[1]—"Reagents: (1) concentrated nitric acid, or (2) nitric acid 1 part and saturated solution of magnesium sulphate 9 parts.

"Albumin causes a white cloud to appear in the form of a ring at the zone of contact of the two liquids (reagents and urine), and this test, when carefully applied, must be regarded as one of great value.

"1. A pipet is filled for a distance of from one inch to one and one-half inches with the urine to be tested. The urine is then removed from the surface of the pipet by washing or by wiping.

"2. The pipet, with its contained urine, is then placed near the bottom of a bottle containing nitric acid, when the pressure of the index-finger is lessened and the acid allowed to flow gradually up into the pipet.

"3. When the pipet is seen to contain about equal amounts of acid and urine, the finger is again pressed firmly upon the top of the pipet, which is then removed from the bottle and held toward the light on a level with the eye. If albumin is present, a distinct white ring of coagulated albumin appears at the junction of the urine and the reagent."

(3) *Johnson's Picric-acid Test.*—To filtered urine in a test-tube are slowly added a few drops of a saturated watery solution of picric acid. Immediate turbidity indicates albumin. Some authorities prefer that a dram or two (4.0–8.0) of the yellow fluid be placed gently on the surface of the urine, when, if albumin is present, a white zone at once is apparent, together with a haziness that spreads downward with the diffusion of the liquids. Heating emphasizes the evidence of the test, which is extremely sensitive.

(4) *Roberts' nitric-magnesium test* is also very delicate. It consists in using the following mixture, just as in Heller's test: one volume of concentrated nitric acid, added to five volumes of a saturated solution of magnesium sulphate.

(5) *Trichloracetic-acid Test.*—This will discover minute traces of albumin, but has the disadvantage that it responds to nucleo-albumin

[1] *Medical Diagnosis*, Anders and Boston, p. 605.

as well as to serum-albumin. A few crystals may be dropped into the urine, or a saturated solution may be used after the "contact method," when, if albumin be present, a white coagulum forms. This and the Geisler test-papers (Vierordt) constitute portable and handy tests.

(6) The *acetic-acid and potassium-ferrocyanid test* is minutely sensitive, but gives a precipitate with other albuminoid bodies. The urine is first acidulated with acetic acid. A few drops of a freshly prepared solution of potassium ferrocyanid are then added, and if either albumin or hemialbumose be present, it will be precipitated.

(7) *Quantitative Test.—Esbach's Albuminometer.*—This consists in using a graduated test-tube, into which definite amounts of urine and a reagent composed of 10 parts of picric acid, 20 of citric acid, and enough water to make 1000 parts are carefully mixed by reversing several times the stoppered tube. After allowing this to stand about twenty-four hours, the height of the precipitated albumin is read off on an etched scale, which will indicate approximately the parts per thousand. Not less than 0.5 part per thousand can be estimated correctly, however. Tsuchiya has suggested the use of a solution of phosphotungstic acid as follows: Phosphotungstic acid, 1.59 grams; hydrochloric acid (conct.), 5 c.c.; ethyl alcohol, q. s. ad 100 c.c. This solution is substituted for the picric acid solution, and is used in Esbach tubes. Should there be a hematuria, if the percentage of albumin by Esbach's method, divided into the number of red cells per cubic centimeter of urine, is less than 30,000, it suggests a purely hematuric albuminuria; if greater, it suggests an independent albuminuria (Goldberg).

Prognosis.—Etiologic considerations bear heavily in this matter. The febrile, hemic, cyclic, and paroxysmal varieties usually clear up with convalescence and with advancing years (in the latter case). The persistence of albumin in these cases, however, even in slight amounts or at variable periods, should cause suspicion. Personal observation leads me to believe that in many cases the function of the renal epithelium has suffered. Especially is this true when there is associated a gradually increasing arterial tension. The presence of tube-casts is conclusive of structural change in the kidneys.

PROTEINURIA.

(*Albumosuria.*)

Protein may appear in the urine as a result of pathologic conditions, especially myeloma, and is dependent upon the decomposition of organized proteids. It is a body more or less closely allied to peptones, globin, histon, and the digestion albumoses, but it displays certain characteristics unknown to these substances. The nature of the *exciting cause* is unknown; it may be bacterial or chemical (phosphorus).

Anders and Boston[1] have reviewed all the cases of albumosuria available in the literature—30 in number—and gave an account of three examples that fell under their observation. These studies appear to warrant the following inferences: Nearly all cases of albumosuria manifest themselves after forty years of age. Males are affected in 80 per cent. of the cases. Multiple myeloma figured in 80 per cent., hence albumosuria is suggestive of myeloma and may be diagnostic.

[1] *Transactions of the College of Physicians*, vol. xxiv., *The Lancet*, January 10, 1903.

Symptoms.—Proteinuria may be persistent, transitory, or, less commonly, remittent, and it occurs in variable degrees at different hours during the day. The urine may show the presence of combined serum-albumin, but tube-casts are present in rare instances only. Pain is an almost constant feature and is aggravated on pressure over the affected bones. Bence-Jones protein (myelopathic albumosuria; Kahler's disease) is symptomatic of certain infections (tuberculosis, pneumonia). Of interest is the proteinuria of pneumonia, in view of the recent theory that resolution in this disease is the result of the action of certain ferments. Proteinuria is absolutely diagnostic of disease of the marrow of the bones, but it only points toward multiple myeloma when other symptoms of the latter condition are present. It is distinguishable from ordinary albuminuria by the production of cloudiness in the urine when heated to 50° or 60° C., and by solution of the precipitate at higher temperatures. It is of grave prognostic significance and runs a fatal course within two years.

INDICANURIA.

Definition.—The presence of a pathologic quantity of indican in the urine. Indican occurs in the urine in health in very small quantities, and is, chemically speaking, indoxyl-potassium sulphate.

Pathology and Etiology.—Indican is increased abnormally in the urine by any disorder whereby large quantities of albuminous matters are decomposed. Thus, it occurs in ileus, which produces a stagnation of the contents and a consequent decomposition from bacterial action. Under such circumstances indol and phenol are formed. The former, being absorbed and oxidized into indoxyl, finally appears in the urine in combination with potassium sulphate. Acute and chronic peritonitis, wasting diseases, and cachectic conditions in which there is a considerable destruction of albuminoids (as in Addison's disease, neoplasmata, cholera Asiatica, and empyema) usually have an associated indicanuria. Increased indicanuria occurs when there is an impediment to peristalsis of the small intestine, hence is not seen in simple uncomplicated constipation. Since the pancreatic secretion peptonizes the proteids from which arise leucin and tyrosin, and these in turn are decomposed into skatol, indol, and phenol, it is stated (Piseuti) that any obstruction preventing the flow of the pancreatic juice into the bowel would be reflected in a diminished quantity of indican in the urine. An increased indicanuria is encountered when anachlorhydria or hypochlorhydria exists (*e. g.*, gastric carcinoma—Simon).

Diagnosis.—This depends upon the demonstration of indican by adding strong oxidizing agents, which decompose this product and set the indigo or pigment free. At times the urine may present a cloudy, bluish, or even blue-black appearance. This may be seen in urine that has been standing for some time, the sediment giving a bluish reflection, or there may be a blue turbid film on the surface. Porter[1] holds that a decidedly bluish-black color shows an intense type of putrefactive fermentation with the production of highly toxic substances, capable of giving rise to severe toxemia. A reddish shading of the blue or a reddish-green color shows obstruction to the free flow of bile through the duct. A greenish tinge indicates an obstruction to the internal or intrahepatic branches of the bile-ducts. These patients bear surgical interference badly.

[1] *Archives of Diagnosis*, April, 1908.

Tests.—*Jaffe's* well-known test consists in mixing equal volumes of urine and hydrochloric acid, and then adding, drop by drop, a concentrated solution of chlorinated lime, shaking the tube after each addition. A strong indigo-blue color appears if there is much indican.

A good modified test is the use of fuming nitrohydrochloric acid and urine (equal parts) and a saturated solution of chlorinated potash, used as in the above method. A blue-black cloud or ring appears below the surface. If a few drops of chloroform are then added and the mixture is agitated slightly, a blue color settles at the bottom, owing to the chloroform carrying with it the oxidized indican. H. Strauss[1] gives a new and convenient clinical method for the quantitative determination of indican in the urine. A correct interpretation of indican reactions serves as a guide both as regards diagnosis and prognosis.

PYURIA.

Definition.—The presence of pus in the urine.

Etiology.—Pyuria is due to (1) suppurative inflammation along some portion of the genito-urinary tract, or (2) to the rupture of adjacent abscesses into the tract.

Pyelitis and Pyelo-nephritis.—Pus from the pelvis of the kidney may be due to calculous, tuberculous, or other irritation. It is associated at times with the "railed" or transitional epithelium usually seen early in the case. In pyelo-nephritis casts may indicate renal involvement, although it should be borne in mind that in abscess of the kidney pus may be discharged continuously without the appearance of any casts in the urine whatsoever. One such case came to necropsy under the observation of H. S. Anders, in which small uratic calculi were discharged now and then for several years. Later, several larger stones were removed from the bladder by Willard by suprapubic cystotomy. The abdominal opening healed in a few months, but pyuria persisted. Death occurred, and it was found *postmortem* that a large abscess occupied the lower third of the left kidney, which was filled with small, dark, and irregularly-shaped calculi. A thick pyogenic membrane surrounded the purulent and calculous contents. No casts were found at any time during life, and renal symptoms were altogether absent.

The pyuria is sometimes *intermittent*, one ureter becoming temporarily occluded (on the side of the disease), the clear, normal urine from the healthy kidney passing until the ureteral obstruction is relieved, when pus again appears. Purulent urine from the kidney is usually acid in reaction, except when the pyelo-nephritis is secondary to cystitis, when it is more apt to be alkaline and to contain a decided quantity of mucus.

Cystitis.—Pyuria in this affection is fetid in most cases. Bladder-symptoms are marked. The urine is alkaline, and a stringy, tenacious muco-pus comes with the last portions. Triple phosphates are often found. The pus and urine are not so intimately mixed as in pyelonephritis.

Urethritis.—The pus is in small quantities, is passed in advance of the urine, and can be "milked out." There is a history of gonorrheal infection, and the gonococcus may be demonstrated in most cases.

Rupture of contiguous abscesses into the urinary tract is accompanied with a sudden discharge of a large quantity of pus in the urine, preceded by symptoms of abscess elsewhere, as in the pelvis or right iliac fossa

[1] *Deutsche med. Woch.*, April 17, 1902.

(suppurative appendicitis) or perinephric abscess. The pyuria disappears as abruptly as it came on, or lasts but a few days. The *strongylus gigas* in the pelvis of the kidney causes pyuria as well as hematuria.

Diagnosis.—Pus gives a greenish-yellow or yellowish-white tinge to the urine and sediment, the latter very often becoming very tenacious or jelly-like from the presence of mucus. It may resemble a phosphatic precipitate, as in cystitis; the latter, however, is white, lighter, more granular, and not so thick or tenacious. *Microscopically*, a positive diagnosis is made by the discovery of pus-corpuscles (or leukocytes) with their granular protoplasm, which has the faculty of clearing up and showing one or more nuclei upon the addition of acetic acid. The corpuscles are either more or less swollen and clear, or opaque, granular, or even nucleated, according to their number, the length of time in the urine, and the degree of alkalinity or acidity of the latter. A few phosphatic crystals and epithelium may be seen.

Chemically, there is slight albuminuria, a marked amount of albumin usually indicating renal disease. Reinecke has proposed a method for determining whether all the albumin can be accounted for by the pus. After shaking up the twenty-four-hour specimen to diffuse the pus evenly through it, he counts the cells present by means of a hemocytometer. He finds that 100,000 pus-cells per c.mm. should correspond to 1 per cent. of albumin (Esbach). It is obvious that this method falls short of accuracy, although approximately reliable. Nephritis may be diagnosed in connection with pyuria by the discovery of casts. On the addition of liquor potassæ to urine containing pus the latter is converted into a clear gelatinoid substance; mucus, on the other hand, becomes thin and flocculent. Mucus may also be distinguished from pus by its failure to react to cold nitric acid, whilst the albumin of purulent fluid coagulates.

CHYLURIA.

Definition.—The presence of chyle in the urine.

Etiology.—This interesting condition may be either *parasitic* or *non-parasitic* in origin. The former type is more common in the tropics, and is caused by an engorgement and rupture of the bladder or renal lymph-vessels, due to obstruction of the larger branches of the thoracic duct or in the duct itself, by the *filaria sanguinis hominis* (*vide* Filariasis). It is held to follow injuries to the lymphatic ducts, and may be associated with pregnancy.

Diagnosis.—The urine is increased in quantity, and has a milky turbidity (*galacturia*) due to the emulsified fat. After standing for a time a light coagulum settles to the bottom and a creamy pellicle of fat rises to the surface. The sediment contains also the fibrin of the chyle. Sometimes as much as 2 or 3 per cent. of fat is present (*lipuria*); this may be tested by agitating a portion of the urine with ether, whereupon the turbidity disappears. Owing to the serum-albumin in the chyle, the various tests for that substance would show traces of its presence in chyluria. Hematuria may be associated with chyluria, especially in parasitic cases, and both the blood and urine should be carefully examined for filaria. *Microscopically*, chyle-containing urine resembles milk in its millions of fine granules and fat-droplets.

Prognosis.—Chyluria is intermittent in its appearance, corresponding to the times of rupture of the vesical lymphatics, and may last for years. The prognosis of non-parasitic chyluria is good as to life, but unfavorable as to cure.

CHOLURIA.

Definition.—The presence of bile-pigment in the urine.

Etiology.—Choluria may be caused by any disease, local or general, in which *jaundice* is a symptom.

Diagnosis.—Bile-stained urine has a color varying from a greenish-yellow to a brownish-green or brown-black, resembling porter. When shaken its foam assumes a characteristic yellow or greenish-yellow color. White filter-paper dipped in the urine is stained yellow.

Tests.—The *chloroform* test consists in adding this substance to the urine and allowing it to settle to the bottom of the tube. If bile or pigment be present, the gravitated chloroform will be colored yellow.

Gmelin's test is most commonly employed, though it is not the most delicate. A few drops of urine and nitric acid are allowed to run together on a white porcelain plate; if bile-pigment (bilirubin) be contained in the urine, a play of colors ensues, the green predominating, followed by the blue, violet, and red, each shade representing a new form of pigment. The first color noticed (green) corresponds to the biliverdin or normal bile-pigment of herbaceous animals. This oxidation of bilirubin into biliverdin is better accomplished by nitric acid containing a little nitrous acid. Hence, the test may be improved by adding enough fuming nitric to ordinary nitric acid to form a yellow trace of the nitrous acid. This may be placed in a test-tube, and some of the urine added gently from a pipet. Bile-pigment will be indicated by successive rings of green, blue, violet, and red from above downward; this occurs, however, only when considerable bile-pigment is present.

Rosenbach's test is a modification of Gmelin's, and is more distinct. The urine is first filtered, and a drop or two of the nitric-nitrous acid is then poured upon the filter-paper, when the characteristic colored rings will appear if bile be present. According to Penzoldt, the Gmelin-Rosenbach test is made more distinct by acidulating the filtrate with acetic acid and pouring a thin layer into a white shallow dish. The acetic acid assumes a greenish-yellow, and later a green, or even a blue-green, shade if bile be in the urine. This reaction is quickened or intensified by the application of heat to the liquids.

In the *Marechal-Rosin test* a mixture of one part of the tincture of iodin and ten parts of alcohol is spread in a deep layer over the suspected urine in a test-tube or glass. A grass-green ring forms at the point of contact in choluria.

Bile-acids.—These are principally the glycocholic and taurocholic acids. Traces are found in normal urine, and their clinical significance or diagnostic importance, as far as is known, is practically *nil.*

When testing for bile-acids the Stranburger modification of Pettenkofer's method may be used, as follows: "After isolation cane-sugar is added to the extract, which is then filtered. A drop or two of strong sulphuric acid is spread on the dried filter; a violet or purple color appears" (Musser).

Other constituents of the urine in choluria of long standing are slight quantities of albumin and icteric or yellow bile-stained hyaline or finely-granular casts.

A point in **differential diagnosis** should be noted. *Certain drugs*, as rhubarb and santonin, when given internally, may produce a discoloration of the urine similar to that caused by the presence of bile. On agitation, however, there will be no yellow foam and no reaction to the tests for bile, while the addition of liquor potassæ causes a red color.

UROBILINURIA.

Definition.—The presence of pathologic quantities of urobilin in the urine. Urobilin is the principal coloring-matter of the urine, and hence is present in normal urine in small quantity. It is derived from bilirubin as a product of the reduction of this substance.

When present in large quantities urobilin gives to the urine a red-brown color. This is seen in fevers, varying in depth of shade according to the degree of pyrexia; also in diseases of the liver, after hemorrhagic effusions (due to resorption), in the hemorrhagic diathesis, in purpura, and in progressive pernicious anemia. Cavazza[1] has examined 20 cases of urobilinuria in chlorosis. He found a temporary, marked increase in acute chlorosis and in exacerbations of the disease.

When deposited in the tissues it gives rise to a form of jaundice in which there is a brownish skin—called *urobilinicterus*.

Diagnosis.—The presence of urobilin is best detected by a spectroscopic examination. A marked absorption-band between Fraunhofer's lines (f and b), fading off from the green into the blue, is characteristic. Chemically, the addition of a few drops of a watery solution of zinc chlorid to the urine will cause the peculiar red-green fluorescence of urobilin to appear.

GLYCOSURIA.

Definition.—The presence of sugar (glucose) in the urine. Normally, a trace of sugar is present in the blood (glykemia), but it may be doubted whether any is excreted in the urine in health, except after the ingestion of an excess of food rich in saccharine or starchy substances. Uric acid may give the same reactions as glucose in the urine.

Etiology.—The causes of glycosuria may be enumerated as follows: (1) Diabetes mellitus—the most common. (2) Certain diseases, like gout (*intermittent glycosuria*), cholera, typhoid, typhus, and scarlet fevers, whooping-cough, diphtheria, malaria (*paroxysmal glycosuria*), tetanus, phthisis, hepatic cirrhosis, and organic nervous diseases, especially those affecting the medulla and involving the floor of the fourth ventricle. Glycosuria may also result from psychic causes, as excessive mental exertion, extreme emotional activity (grief, worry, and shock), from injuries and after operations,[2] as cerebral concussion and hemorrhage, and fracture of the skull, from apoplexy, cerebro-spinal meningitis, and after epileptic paroxysms. (3) Pregnancy (40 per cent. of the cases—Wormmüller). (4) Certain toxic agents cause a transient glyco-

[1] *Centralbl. f. innere Med.*, March 15, 1902.
[2] T. R. Brown, *Johns Hopkins Hosp. Bull.*, May, 1900.

suria, among these being carbon monoxid, morphin, atropin,[1] hydrocyanic acid, amyl nitrite, curare, chloral, alcohol, mercury, arsenic, turpentine, copaiba (Bettman), adrenalin, phloridzin, and various coal-tar derivatives, as salicylic acid and salol. This source of glycosuria has been experimentally demonstrated in dogs by Paul Gibier. (5) Obesity and thyroidismus may cause a temporary glycosuria (lipogenic). (6) Pancreatic disease (chronic interstitial pancreatitis and, less commonly, pancreatic calculi, carcinoma, and cysts). (7) Glycosuria may occur in exophthalmic goiter, and, according to Lyman, may be present for a short time in (8) diabetes insipidus. (9) Heredity probably plays a part in predisposing to glycosuria in certain cases, particularly in the permanent affection. (10) Dietetic or alimentary glycosuria—due to ingestion of alcohol, carbohydrates, or glucose.

Diagnosis.—The daily quantity of the urine of typical glycosuria—*i. e.*, when masking saccharine diabetes—is greatly increased (60 fluid-ounces—2 liters—and over *per diem*); it is of high specific gravity (1025 and over), of a clear, pale-yellow color, a "ripe-fruit" odor, a sweetish taste, and an acid reaction that is intensified on standing, owing to the fermentation of the sugar. Albuminuria may be associated with glycosuria, and the albumin should be removed before testing for sugar.

Tests.—The most important of these depend mainly upon the peculiar property of glucose in reducing the blue oxid of copper to the orange or red suboxid.

(1) *Fehling's Test.*—Two solutions are used, equal parts being mixed to form the Fehling's solution, as follows:

Solution I. contains 34.64 gm. of cupric sulphate, dissolved in enough water to make 500 c.cm. Solution II.: 173 gm. of Rochelle salt are dissolved in 480 c.cm. of sodium hydroxid (sp. gr. 1.14); this is then diluted with water up to 500 c.c.

Application: Dilute 1 c.c. of Fehling's solution (about 10 drops of each of the above solutions) with about 1 dram (4 c.c.) of water in a test-tube, and heat to the boiling-point. If the clear blue color remains, the solution is ready for use; should it change color, however, the solution is unfit for use and should be discarded. The suspected urine is added, drop by drop, heating occasionally, when, if glucose be present, the blue color will be discharged by a yellow turbidity, which increases until finally a deep-yellow or orange red precipitate falls. Bluish-white flakes and a greenish discoloration of the mixture simply indicate cupric hydroxid, and not glucose. This test serves for the detection of .001 per cent. of glucose (Wormley). It cannot be applied to strongly ammoniacal urine.

(2) *Trommer's Test.*—To about 5 c.c. of urine in the tube add one-third or one-half its volume of potassium or sodium hydroxid, and then, drop by drop, add a 10 per cent. solution of cupric sulphate. If a bluish-white precipitate falls, either filter or agitate the liquid until it assumes a slight and uniform turbidity; then heat, and, if sugar be present, a yellow or red deposit of cuprous oxid falls: .01 per cent. of glucose may be detected in this way.

There are certain other substances which when present in urine make the copper tests fallacious by reducing the cupric to cuprous oxid (mucin,

[1] F. Raphael, *Deutsche med. Woch.*, July 13, 1899.

lactose, pyrocatechin, hydrochinon, bile-pigments, glycosuric acid, the products of elimination after the ingestion of chloral—urochloric acid—and benzoic and salicylic acids). Among normal constituents that can reduce cupric oxid are uric acid, creatinin, and hippuric acid. "Alkapton" urines also reduce Fehling's solution.

(3) *Böttger's Bismuth Test.*—This may be performed as a counter to the copper tests. Albumin, however, interferes with the test on account of the contained sulphur, which forms a black bismuth sulphid: hence, if present, it must first be removed. This may be done by acidulating the urine with acetic or nitric acid, boiling, and then filtering. Böttger's test is then made by adding to the non-albuminous urine or to the filtrate from one-half to an equal quantity of liquor potassæ and a few grains of bismuth subnitrate. Boil for several minutes, and if glucose be present black metallic bismuth will be precipitated.

(4) *Nylander's reagent* may be employed. This consists of 2 parts of basic bismuth nitrate and 4 parts of sodium tartrate to 100 parts of an 8 per cent. solution of caustic soda. One part of the reagent is boiled with 10 parts of the urine for a few minutes, when a change from the original to a brown or black color will indicate the presence of glucose. This test is quite distinct, but has the fallacy that is common to all the bismuth tests, of forming a black precipitate with the sulphur compounds.

(5) *Fermentation Test.*—Though not always convenient to apply, this is, nevertheless, a most reliable test. It depends upon the action of yeast in breaking up glucose into alcohol and carbonic-acid gas (carbon dioxid). It is performed easily by adding a small piece of compressed yeast to the urine in a test-tube, inverting the latter in a dish of the same, and standing aside for twelve to twenty-four hours, the temperature being kept at about 80° to 100° F. (26.6°–37.7° C.). The evolution of gas resulting from the fermentation of the sugar takes place, with a consequent reduction of the specific gravity of the urine. The yeast may be tested simultaneously for its purity and strength by placing one portion in a test-tube containing about two-thirds mercury and filling with normal urine, and a similar portion in a second tube with mercury and a thin, watery solution of sugar or glucose; the fermentation test of the suspected urine may be made at the same time, and all three tubes inverted over a dish of mercury. Obviously, the first tube should not show the presence of carbon dioxid if the yeast was free from sugar; but the second tube should show this gas to be present or the yeast was inert.

Other tests, such as *Moore's liquor-potassæ-and-boiling test*, *Johnson's picric-acid test*, and the *phenyl-hydrazin test*, are more intricate.

The *quantitative estimation of sugar* may be made with Fehling's solution in two parts, as recommended above for the qualitative test. This method is based upon the fact that the cupric oxid of 1 c.c. of Fehling's solution will be reduced by not less than 0.005 gm. of glucose. Place 1 c.c. of the solution in a test-tube and dilute with 4 c.c. of water (5 c.c. dil. sol.). Heat to the boiling-point, and add 1 c.c. of urine, and heat the liquid again. If reduction has taken place, 0.005 gm.—0.5 per cent. or more—glucose is present; if no reduction has occurred, less than 0.5 per cent. is present. If 2 c.c. urine are used

before the color of the Fehling solution is discharged, there will be 0.25 per cent. glucose. If ½ c.c. is used, 1 per cent. is present. If $\frac{1}{10}$ c.c. urine is all that is required (about 2 drops), then 5.0 per cent. of glucose is present.

Roberts' differential-density method depends upon a loss in the specific gravity of the urine, due to the fermentation of glucose. According to Roberts, each degree in specific gravity lost is equivalent to 1 grain of glucose in 1 imperial fluidounce (437.5 gr.) of urine, or one degree represents 0.23 per cent. glucose. Pavy's method is also convenient for clinical purposes. (See works on Urinalysis.)

Circumpolarization.—Finally, sugar may be determined by the saccharimeter or polariscope. Glucose polarizes light to the right. The percentage may be calculated by reading the vernier scale indicating the degree of reflection, and multiplying the number read by the factor of the apparatus used, after making any required corrections.

ACETONURIA, DIACETONURIA, AND OXYBUTYRIA.

Acetonuria, diacetonuria, and β-oxybutyria are so closely allied with glycosuria, and especially with diabetic coma (acetonemia), that they may be considered together. In the first-named condition the urine contains acetone; in the second, diacetic or aceto-acetic acid; and in the last, β-oxybutyric acid.

Acetonuria may exist to a minute degree in health, the acetone being a product of the normal metamorphosis of albumin. It may be present also in—(1) diabetes; (2) carcinoma; (3) febrile conditions; (4) inanition; (5) psychoses; (6) auto-intoxication, especially with fatty acids in the stomach and intestines (enterogenous acetonuria); (7) pregnancy; (8) after anesthesia; and (9) in cyclic vomiting. Urine that contains acetone in pathologic quantities has a fruity (apple-like) odor or one resembling that of chloroform.

Tests.—(1) *Gerhardt's* original test consisted in the addition of a few drops of the tincture of the chlorid of iron, which produced a Burgundy-red color with acetone, or rather with the aceto-acetic acid.

(2) *Nitro-prussid Test.*—To a fluidounce (32.0) of the urine add a dram or two (4.0–8.0) of a solution of sodium nitro-prussid (gr. v to ℥j —0.324 to 32.0) and a few drops of strong aqua ammoniæ. On standing a rose-violet color appears. According to Legal, proportionately smaller quantities of urine and the reagent may be used, and strong liquor potassæ. A bright-red color develops, and fades rapidly, but upon adding acetic acid this changes to purple or violet-red (Vierordt). This is a better test.

(3) Perhaps the most accurate and, at the same time, satisfactory test for acetone is the following: Distil the urine with a little phosphoric acid, and add to the distillate a few drops of sodium hydroxid and of Lugol's solution. If acetone be present, yellow crystals of iodoform will form, with the characteristic odor.

Diacetonuria and **oxybutyria** never occur normally. They are often associated with acetonuria in diabetes, and sometimes in fever, or occur as an independent disease (V. Jaksch). It is believed β-oxybutyric acid is the immediate cause of diabetic coma. "The persistent

excretion of more than 25 grams of β-oxybutyric acid indicates impending coma" (Simon). Stadelmann affirms that of like value in diabetes is the determination of a marked and increasing amount of ammonia in the urine (1 gram—gr. xv—and more *per diem*), as indicating the imminence of diabetic coma. Diacetonuria is found to occur in certain acute diseases of children, accompanied with convulsions.

Tests.—The presence of diacetic acid is demonstrated by the chlorid-of-iron reaction, as in the case of acetone, except that the urine is boiled previously. This is done to avoid fallacy, since in unboiled urine acetic, formic, and oxybutyric acids may strike a Burgundy-red also; in urine that has been previously boiled these do not react, while the diacetic acid does, if present. Diacetic acid is usually present simultaneously with acetone and β-oxybutyric always, and is formed first. Tests for the latter, therefore, need not be detailed here (*vide* Tests for Acetone).

LITHURIA.

Definition.—A persistent excess of uric (lithic) acid and urates (lithates) in the urine. Uric acid occurs in the urine in combination with alkalies, but may become free, separating out as a crystalline deposit.

Normal urine contains about 0.4 part of uric acid to 1000 parts of urine (about gr. x—0.648—*per diem*), or it exists in the proportion of about 1 to 45 of urea, the principal solid constituent.

Etiology.—The causes of lithuria, as seen in certain conditions in which this metabolic change occurs, may be put down to be chiefly as follows: (1) Lithemia (uricemia; uric- or lithic-acid or gouty diathesis); (2) gout and rheumatism; (3) fever; (4) leukemia and pernicious anemia; (5) pulmonary affections in which the interchange of gases is interfered with; (6) a highly nitrogenous diet. Certain other conditions of the urine may diminish its power of dissolving the uric acid shortly after voidance, and may cause a deposit that should not be mistaken for an excess. Such are—(*a*) temporary increase in the quantity of uric acid from an over-indulgence in nitrogenous food; (*b*) temporary high acidity; (*c*) deficiency in mineral salts.

Diagnosis.—The urine has a high specific gravity, a deep red-yellow color, and a marked acid reaction, although, rarely, uric acid is formed in neutral or alkaline urine (Vierordt). Albumin may be present in small amount at the same time. On standing the uric acid is deposited in yellowish-red or "Cayenne pepper" grains, composed of microscopic uric-acid crystals. Chemically pure uric acid is colorless, but that deposited from urine has a yellowish-red appearance, both to the naked eye and under the microscope. Examination with the latter shows a great variety of rhombic prisms—"whet-stone-shaped," "crosses," "lozenges," and others—single and in agglomerations.

Test.—The *murexid reaction* may be obtained by evaporating a little urine in a watch-glass or porcelain dish, adding a few drops of strong nitric acid, and heating to dryness again; this is allowed to cool, and a drop of liquor ammoniæ added, when a beautiful purple shade of murexid will appear if uric acid be present. Martinet gives the following simple and reliable method of estimating the acidity of the urine as a basis for treatment in various conditions: To 20 drops of decinormal

sodium hydrate solution are added 2 drops of a 1 per cent. alcoholic solution of phenolphthalein and the whole heated to boiling. Urine is then added, drop by drop, until the distinct pink color vanishes entirely. The total acidity is expressed in terms of sulphuric acid, H_2SO_4, by dividing 98 by the number of drops of urine required for decoloration.

Reference should here be made to the "*nucleins*" or *xanthin bases*. They result from the disintegration of nuclein, as does uric acid, but differ from the latter in being more strongly basic. Along with uric acid, these substances are often spoken of under the term "alloxuric bodies." There is great variability in the behavior of the xanthin bases as compared with that of uric acid, although they are usually increased in conditions in which uric acid is present in excess.

Urates.—These are increased in pathologic conditions that give rise to uric acid in excess, and are usually present with the latter in some quantity. It is not rare, however, in healthy individuals for a deposit of urates to occur in concentrated urine exposed to a cool atmosphere. Urates appear also in the scanty urine from any cause, *e. g.*, profuse perspiration, diarrhea, fever, and after a meal rich in albuminous elements.

Urates occur principally as acid sodium urate, calcium urate, and ammonium urate. They appear macroscopically as a flesh-colored or "brick-dust" (lateritious) sediment; this is usually abundant and very finely granular in appearance, while the urine above is cloudy. Upon heating such urine it becomes clear, the urates being completely dissolved. *Microscopically*, the sodium and calcium salts of uric acid occur as needle- or dumb-bell-like crystals or as fine, dark, amorphous granules. Ammonium urate is found in alkaline urine, often with triple phosphates when some putrescence has ensued. It is seen in dark-brown or green spiculated spherules; these are sometimes called "hedge-hog" or "thorn-apple" crystals. On the addition of a drop of hydrochloric acid under the cover-glass uric-acid crystals may be seen to develop.

OXALURIA.

Definition.—A persistent excess of calcium oxalate in the urine. A few crystals may occur in normal urine (about one urine out of every three), especially after standing for a long time.

Transient oxaluria may follow the ingestion of sub-acid fruits, as pears, or of vegetables containing oxalates (tomatoes, asparagus).

Pathology.—Oxaluria has been described by some English physicians as an independent disease or special diathesis in which marked dyspepsia and hypochondriasis or neurasthenia are associated. The condition is better explained, probably, as one of a disturbed metabolism—particularly of the fats and carbohydrates—in which the oxaluria and the nervous symptoms are manifestations analogous to the lithuria and the irregular gouty symptoms of lithemia. Oxalates and lithates are not infrequently found together in the urine of those subject to the gouty habit. Uric acid may be oxidized to oxalic acid. The ultimate source seems to be the nucleins and nucleo-albumins. Oxaluria is also present in wasting diseases, as in tuberculosis and diabetes mellitus, and in the cancerous cachexia; it may appear in catarrhal jaundice, spermatorrhea, also with the "mulberry calculi," and in general paresis of the insane. Slight albuminuria is not infrequently associated.

Diagnosis.—Oxalate-of-lime crystals appear in the urine in two

forms—most commonly as minute, regular, highly-refracting octahedra, or, more rarely, as hour-glass- and dumb-bell-shaped crystals.

The octahedral crystals have two crossed axes, giving a star or envelope-like appearance. Oxalates sometimes give a glittering and scintillating effect to floating mucus in urine that has undergone fermentation. The finding of calcium oxalate in the urine does not necessarily imply an increased excretion of this salt. The precipitation is due to the absence of the sodium phosphate which keeps it in solution.

The **prognosis** is usually favorable.

Treatment.—Nitro-hydrochloric acid in 2-drop doses is a useful agent.

PHOSPHATURIA.

Definition.—A persistent excess of phosphates in the urine.

Phosphoric-acid salts may be precipitated in normal urine that has become temporarily alkaline. These acid sodium and potassium phosphates in normal acid urine are derived from the alkaline phosphates (neutral sodium and potassium phosphates) of the blood. In normal urine 1.2 parts of alkaline phosphates per 1000 and 0.8 part of earthy phosphates are appreciable.

Etiology and Pathology.—Conditions that produce an alkaline fermentation of the urine cause a deposit either of *amorphous earthy phosphates* (of calcium and magnesium) or of *alkaline phosphates* (of potassium, sodium, and ammonium). They are also found in the decomposing urine of chronic cystitis, of phosphatic vesical calculi, of paralysis, and in undue retention of urine. In this alkalinity, due to the ammoniacal fermentation of urea, ammonium carbonate reacts with the phosphates of magnesium to form the triple ammonio-magnesia phosphatic crystals, the commonest variety of phosphaturia. Here the phosphates are deposited before or immediately after the urine is passed, giving a milky appearance to the last portion. Deposits of phosphates, and especially of triple phosphates, however, do not indicate an actual phosphaturia. This must be determined by chemical analysis. *Amorphous carbonate of lime* in small quantity may be present also if the urine is strongly alkaline and ammoniacal (Beale). The *calcium phosphates* are generally more abundant than the magnesium, and may be found in cases of nervous or atonic dyspepsia, neurasthenia, and other debilitated conditions. The alkaline phosphates (which represent three-fourths of the phosphoric acids), being easily soluble, do not form a deposit.

A quantitative estimation of the daily output of phosphates shows a decided increase in wasting diseases, as tuberculosis, leukemia, chronic articular rheumatism, and acute yellow atrophy of the liver. The phosphoric acid, however, is not increased. The so-callad "phosphatic diabetes" is characterized chiefly by excessive phosphaturia.

Diagnosis.—Phosphatic urine has usually a stale, ammoniacal odor, a whitish turbidity, and a copious light-colored granular sediment falls on standing. *Microscopically*, the calcium phosphate crystals appear singly as "knife-blade," "arrow-head," or "slender wedge-shape," or in stellate clusters. Acetic acid dissolves them. The ammonio-magnesium phosphate crystals are transparent rhombic or triangular prisms,

large and small—"coffin-lid-shaped." These also are soluble in acetic acid; oxalate-of-lime crystals are not so.

On heating phosphatic urine an increased cloudiness is produced that simulates albumin, but on acidifying, as with a drop of nitric acid, this is cleared up at once.

LEUCINURIA AND TYROSINURIA.

Definition.—The presence of leucin and tyrosin in the urine. These are strictly pathologic substances, and are usually found together. They are products of the decomposition of albumin.

Etiology.—The principal causes of leucinuria and tyrosinuria are acute yellow atrophy of the liver, acute phosphorus-poisoning (in both of which fatty degeneration is conspicuous), specific infectious diseases, as typhoid fever, small-pox, and yellow fever, and pernicious anemia.

Diagnosis.—Leucin is the more soluble, hence is rarely found in the urinary sediment. Tyrosin, on the other hand, may be discovered sometimes as a fine greenish-yellow deposit. Bile-pigment and a trace of albumin may be found not infrequently in urine containing leucin and tyrosin. Urea is, as a rule, markedly diminished. Leucin and tyrosin may be detected by evaporating a few drops of urine on a glass slide and examining microscopically. Leucin appears in the form of slightly glistening, greenish-yellow spheres that may show radiating lines and concentric rings. Tyrosin is recognized by the slender tufts of fine, needle-like crystals arranged in star- or cross-like fashion.

If the residuum after evaporation be heated with a drop of nitric acid, slowly evaporated to dryness, and then touched with a drop of sodium hydroxid, the leucin, if present, will assume a yellowish-brown hue. Tyrosin becomes red in color when boiled with Millon's reagent of mercurous nitrate, and a violet color when warmed with a little sulphuric acid, and then treated with a drop of the solution of phenic chlorid.

CYSTINURIA.

Definition.—The presence of an excess of cystin in the urine. This is rare, "but when it occurs it may be copious, and is not unlike a sediment of fawn-colored urates" (Hutchison and Rainy). The *causes* of cystinuria have not been well made out, though *hereditary influences* seem to have an important bearing on the etiology. Insufficient nitrogen metabolism, as occurs similarly in such allied conditions as gout and obesity, seems to give rise to cystinuria.

Brieger points out a probable significance in the discovery of the associated presence of ptomaïns with cystinuria. Thus, in certain infectious diseases, as intestinal mycosis, a ptomaïn-cystinic product is supposed to be formed, then to be absorbed, and finally decomposed in the urine, thus setting free the cystin. Cystitis may be caused by ptomaïns.

Diagnosis.—The sediment is light, and not very unlike that of the amorphous urates. It is not dissolved by heat, however, though soluble in ammonia. Under the microscope cystin occurs in the form of thin, transparent, hexagonal crystals. Care should be exercised in forming a diagnosis of cystinuria that a contamination with iodoform be excluded,

since the microscopic appearance of that substance is similar to that of cystin. On account of the sulphur contained in cystin, a test may be employed by which hydrogen sulphid is liberated, as by boiling the suspected urine with a solution of lead oxid and sodium hydroxid, black lead sulphid resulting from the reaction if cystin be present.

VARIOUS OTHER CONDITIONS.

Urea.—This occurs in solution in the normal urine as a product of the perfect decomposition of the nitrogenous elements of food and tissues. In 1000 parts of urine about 20 parts are constituted of urea (2 per cent., equivalent to about gr. 450—30.0—daily). The quantity of urea is *increased* in the urine after the ingestion of a considerable quantity of proteid food; sometimes after exertion; in acute inflammation and in fevers—either relatively or absolutely, as in pneumonitis; in diabetes and other morbid conditions in which metabolism is accompanied by an increase in the tissue-waste. In febrile states its excretion increases or diminishes with the exacerbations and remissions of temperature respectively.

Urea is *diminished* in quantity in all forms of nephritis, and markedly so in uremia; in organic liver-diseases; in cachectic and anemic states; and in dropsy, inanition, and allied conditions.

The quantitative estimation of urea may be made according to one or more of several methods: Fowler's hypochlorite test (with Labarraque's solution) is perhaps the most practical for ordinary clinical purposes.[1] Fowler's method is based upon the loss of specific gravity upon the liberation of the nitrogen of the urea. The mean specific gravity of a mixture of 1 part of urine and 7 parts of the solution of sodium hypochlorite is taken while quiescent, and is then subtracted from the specific gravity of the mixture taken after agitation several times during about two hours. The difference which is due to the liberation of the nitrogen (as is shown by the effervescence), multiplied by the factor 0.77, gives the approximate percentage of urea in the urine.

Urine evaporated to a syrupy consistence and then treated with nitric acid shows crystalline quadratic plates of urea nitrate.

Chlorids.—About 10 parts of the chlorids of sodium and potassium in 1000 parts of urine are excreted daily. They are *increased* in the urine after muscular exertion, during the resorption of mechanical or inflammatory transudations and exudations, and in intermittent fevers, owing to the destruction of the red corpuscles.

Pathologic *diminution* in the quantity of chlorids occurs in fevers, in the nephritides, in cachectic conditions, and especially in such diseases as pneumonitis, pleuritis, and rheumatism. In the last-named class the chlorids diminish as exudation continues, and may even totally disappear from the urine in extensive pneumonic consolidations, to reappear again with the resorption of the exudate.

Test.—The chlorids may be detected, after first removing any albumin that may be present, by acidulating with a few drops of nitric acid (to keep the phosphates in solution), and by then adding, drop by drop, a strong solution of argentic nitrate. According to the abundance of

[1] See works on Urinalysis.

the resultant white, curdy precipitate of argentic chlorid a rough estimate may be made of the total quantity of chlorids in the urine.

Lipuria is a term applied to the presence of fat in the urine. It may result from the steady use of cod-liver oil or of fatty food, or it may be found in pyonephrosis (Ebstein); in phosphorus-poisoning; in prolonged suppuration; in the lipemia of diabetes mellitus; in the "large white kidney" with fatty degeneration of chronic Bright's disease; in beer-drinkers; and in chyluria. Fatty urine becomes clear upon agitating after the addition of ether.

Lipaciduria, or urine containing volatile fatty acids (acetic, butyric, and propionic), is as yet without diagnostic significance.

Melanuria, or urine containing the pigment melanin, is found in cases of melanotic sarcoma. The urine is dark, either just after being voided or after some exposure and oxidation.

Hematoporphyrinuria (*Urospectrin*).—This term implies the presence of hematoporphyrin (iron-free hematin) in the urine. It occurs after long-continued use (even in small doses—Müller) of saffron and certain coal-tar products (sulfonal, trional). Stockton found it in acute ascending paralysis.[1] In addition to the gastric and nervous *symptoms* in poisoning from these substances is a cherry-colored or dark blue-red urine, the abnormal appearance of the latter being due to the presence of hematoporphyrin resulting from the destruction of the red blood-corpuscles. The condition has proved fatal in cases in which the kidneys were diseased. The urine is always quite acid. According to Garrod, hematoporphyrin is a scanty though constant ingredient of normal urine. He extracts it by adding 100 c.cm. of urine to 20 c.cm. of a 10 per cent. solution of sodium hydroxid. This precipitates the phosphates, which are washed with water and redissolved with rectified spirits. After acidulation with hydrochloric acid the solution shows spectroscopically bands of acid hematoporphyrin. The *treatment* consists in the withdrawal of these drugs and the administration of alkalies.

Pneumatinuria, or gas-formation in the bladder, rarely occurs. Heyse[2] records a case of myelitis in which this condition was present.

Fibrinuria.—In certain conditions of the genito-urinary tract, particularly pyelitis and ureteritis, fibrinous (and mucous) shreds are found in the urine. Fibrinuria may follow nephro-lithiasis (v. Jaksch).

Typhoid bacilluria occurs probably in about 25 per cent. of the cases of typhoid fever (Horton Smith, Gwyn).

Bacteriuria.—There are probably few specimens of urine that do not contain bacteria. Engel has found a great variety of organisms in the nephritides, and believes a special coccus to be responsible for many instances of the sort beginning as mild forms of "*bacterial albuminuria.*" Warburg[3] reported a case of chill and fever with turbid urine due to the Bacillus lactis aërogenes. The tubercle bacillus is not uncommon in the advanced stage of pulmonary and in renal or vesical tuberculosis.

Lactosuria.—Lactose is found in the urine of some puerperæ.

Inosituria.—Inosite occurs in the urine in diabetes mellitus, diabetes insipidus, and chronic interstitial nephritis.

[1] *Amer. Jour. Med. Sciences*, July, 1900.

[2] *Zeit. f. klin. Med.*, 1894, xxiv., p. 130, quoted in *The American Year-Book of Medicine and Surgery* for 1896.

[3] *Münchener Med. Wochen.*, July 18, 1899.

Alkaptonuria.—Alkapton is an obscure substance (so called by Bredeker) that is sometimes found in the urine of phthisical cases, or at times in that of patients without any apparent local or general disease. Alkaptonuria is congenital in a few cases. On exposure the urine darkens in color from above downward, also upon the addition of liquor potassæ. It gives the sugar reaction with Fehling's solution (Osler). It gives a dark-brown ring in Ehrlich's diazo-test (C. Mitchell[1]).

Urine as affected by the administration of drugs—as carbolic acid, salol, antipyrin, and potassium iodid—responds to certain chemical tests, for the study of which the reader is referred to works on urinalysis.

Cholesterinuria has been found in cases of pyonephrosis, hydronephrosis, renal hydatids, epilepsy, and severe dyspepsia.

THE NEPHRITIDES.

BEFORE considering the several varieties of nephritis, and especially the clinical history peculiar to each variety, it may be well first to describe certain general manifestations of renal diseases that are more or less common to all. Reference to these symptoms under the different forms of nephritis will, it is hoped, thus make possible a clearer apprehension of their significance and clinical importance, as well as render unnecessary any further elaboration.

One of these conditions has already been described—viz., (1) *Albuminuria.* It remains to speak of (2) the *Morphologic constituents* of the urine in nephritis, (3) *Edema* (*anasarca, dropsy*), and (4) *Uremia.*

THE MORPHOLOGIC CONSTITUENTS OF THE URINE IN RENAL DISEASE: CASTS, EPITHELIUM, ETC.

1. **Tube-casts.**—These are undoubtedly the most important morphologic elements in the urine of a nephritic. Albuminuria is coincidentally present, and the occurrence together of these two pathologic constituents furnishes indisputable evidence of renal disease. Although, on the other hand, hyaline casts may occur in many pathologic states minus albuminuria. According to the nature and number of the casts also may be determined the character and variety of the affection of the kidneys in most instances. Casts, as their name implies, are simply cylindric bodies moulded in the renal tubules, and composed essentially of the coagulable substances in the blood-serum. The coagula of the tubules are mostly albuminous. Other morphologic elements may be mixed with casts—epithelium, red blood-cells, pus-cells, and the granular matter and fat-droplets due to degeneration of the renal epithelium.

Singly, the casts are invisible to the naked eye, but in acute nephritis they may be so abundant as to form a cloudy sediment.

(*a*) Microscopically, the *unmixed* or *hyaline* cast—the commonest—appears either long or short and narrow or broad, of a clear, homogeneous substance, delicate in outline, and often showing ends with a cheesy or wax-like fracture. They may be straight or slightly curved and tor-

[1] *Medical Record*, May 21, 1910.

tuous. Rarely, a cast may be found equal to a millimeter in length. The so-called *narrow casts* are about equal in width to the diameter of a leukocyte, while the *medium* and *broad casts* are from three to four times this size. Hyaline casts are usually associated with other varieties of casts, though in fevers, renal congestion, chronic interstitial nephritis, and in amyloid kidney they may occur unassociated with other forms. Burry's India-ink method of staining for the detection of casts is highly recommended by Stövesandt.[1]

(*b*) *Granular* casts are nothing more than hyaline casts with fine or coarse granules superadded. The granules represent minute, opaque particles of urates, albumin, fat, cellular débris, and even bacteria (*bacterial casts*). It should be remembered, however, that granular casts may be simulated by casts of coagulated albumin covered with particles of hematoidin or of urates, especially in acute nephritis. The hematoidin can be recognized, however, by the brown-yellow coloration.

(*c*) *Epithelial* casts are hyaline casts more or less covered with renal epithelium, indicating desquamative nephritis (Fig. 67). The epithelial cells may show evidence of granular or fatty change.

(*d*) *Blood-casts* consist of soft hyaline casts having red blood-cells imbedded in them. These are present in acute hemorrhagic nephritis.

(*e*) *Waxy* casts are similar in appearance to hyaline casts, though better defined, broader as a rule, and of an opaque, slightly yellowish tint. They often show broken ends (Fig. 67). They do not necessarily indicate amyloid disease of the kidney, as was formerly held. They may, however, sometimes show the amyloid reaction with iodin and potassium iodid, and are always suggestive of serious renal disease.

(*f*) *Fatty* casts are such as have left upon and in them fat-droplets or granules (Fig. 66), which, if abundant, are indicative of fatty degeneration of the kidney. Cells showing granulation may be seen.

Rolled casts or *pseudo-casts* (sometimes made by sliding a cover-glass over a specimen of urine) of urates should not be mistaken for genuine tube-casts. Blood-casts (due to hemorrhage), consisting of fibrin and epithelial pseudo-casts (hollow), in cases of desquamative nephritis, also belong in this category. Cylindroids are distinguishable from hyaline casts by their greater length, tapering ends, and by being at times beset with leukocytes, red corpuscles, epithelial cells, and certain crystals. Cylindroids are met in renal congestion and are related to true casts.

2. **Epithelium.**—Renal cells are found in the urine of those forms of nephritis that are characterized by a catarrhal or desquamative and exudative process in the tubules. Epithelial cells from the kidney are polygonal or spheric in contour, with an indistinct cell-wall; they have a large oval nucleus, and are either abundantly granular or show a fatty change. These cells are about the size of the white corpuscle.

3. **Leukocytes.**—Only when attached to casts can it be positively affirmed that leukocytes are of renal origin (Strümpell). The pus-cells are frequently seen to be without nuclei in marked or chronic pyuria.

4. **Red Blood-corpuscles** (*vide* Hematuria, p. 984).—In acute hemorrhagic nephritis and in severe renal congestion free red blood-corpuscles are generally to be found.

5. **Fat-globules** and **fatty degenerated cells** are seen especially in sub-

[1] *Practical Medicine Series*, 1911, vol. i., p. 384.

acute and chronic nephritis with fatty degeneration of the proliferated epithelium, or in the fatty stage of large white kidney.

DROPSY OF RENAL DISEASE.

Since, as in other conditions, renal dropsy or edema is an abnormal accumulation of watery fluid transuded from the blood-vessels into the cellular tissues and lymph-spaces, the question arises, "What is the rationale of its development in nephritis?" On the ground that in most forms of nephritis the urine is diminished, it was formerly held that the dropsy was due to the saturation of the tissues with the water that was not excreted by the kidneys. This theory is not tenable, however, for there are some cases of edema unaccompanied by any diminution in the daily quantity of urine; on the other hand, certain instances of renal disease in which there is a state of almost anuria show no evidence of dropsy whatever. Landerer holds that the relaxation of the tissues (which may be caused by the increased transudation of stasis, or by hyponutrition from hydremia), and their consequent loss of elasticity, prevent that forcing of the lymph into circulation that exists in the normal state, and as a result a watery infiltration of the tissues is permitted. From recent experiments edema is due, at least in part, "to toxic substances accumulating in the blood and exciting an injurious action on the endothelium of the capillaries" (Edsall). Pearce[1] concludes that plethoric hydremia and vascular injury have equal value with nephritis in the production of edema, and that none of these three factors acting alone, and no combination of two acting together, is sufficient to cause edema. The chloride retention theory, namely, that there is an abnormal amount of sodium chloride in the tissues of persons manifesting renal dropsy (in consequence of which an accumulation of water in order to keep the chlorides in solution occurs), is widely accepted at the present day.

The dropsy of the nephritides may be either slight or marked, local or general (anasarca), and sudden or slow in onset. It is purely renal in origin perhaps only in acute Bright's disease or in the earlier stages of chronic Bright's disease. In all forms of chronic nephritis the dropsy may be due, in part, to the venous stasis of cardiac incompetency. In chronic interstitial nephritis, especially, edema is slight, and usually is the result of weakness and dilatation of the heart. I desire to mention here those rare cases of dropsy that simulate Bright's disease in which no satisfactory causative lesion is apparent or discoverable, and also those cases, rarer still perhaps, that have a peculiar family or congenital origin.

Physical Signs.—The recognition of edema is made possible by both *inspection* and *palpation*. Renal dropsy is manifested first by puffiness of the skin of the face, and especially of the eyelids. At other places where there is loose subcutaneous cellular tissue, and in particular where the parts are dependent, dropsy is most apt to be seen early, as under the malleoli of the ankles, the dorsum of the foot, and the scrotum. Later, the limbs and the lower part of the back become swollen, and the whole body is involved in severe cases. The skin has a peculiar waxy pallor and a glossy appearance. When vascular or cardiac changes exist, so as to permit of increased dropsy from engorgement, as in cirrhotic kidney, a cyanotic or muddy color of the skin may prevail. Palpation detects pitting due to loss of elasticity in edematous tissues.

[1] *Archives of Internal Medicine*, Chicago, June, 1909.

Pathologic Features.—Dropsy is most constant and most persistently decided in the large kidney of subacute or chronic nephritis; it is most uncommon and irregular in chronic interstitial nephritis (contracted kidney). There is also a doughy or putty-like consistence. In very marked cases of dropsy the deeper parts, such as the muscles, become affected. The serous cavities also in general anasarca show evidences of effusion, and thus give rise to hydro-thorax, hydro-peritoneum, and hydro-pericardium. Less frequently there may be edema of the larynx, uvula, conjunctiva, and other mucous membranes. Edema of the brain, either local or general, may be the cause of grave uremic symptoms in chronic nephritis, or of unilateral convulsions or paralysis and apoplectic seizures. The dropsical liquid is chemically similar to a diluted blood-serum. A minute quantity of albumin and urea is present.

UREMIA.

Definition.—Uremia is the term applied to a group of manifestations, mainly nervous and either acute or chronic, resulting from a toxemia due to the retention in the body of certain products of urinary or renal origin.

Although most common in Bright's disease, uremia may arise also in other diseases, as in gout (gouty kidney), scarlet fever (scarlatinal nephritis), typhus fever, yellow fever, and cholera, in which the kidneys and blood may be seriously affected. Kidneys which, on account of marked structural changes, fail to eliminate the normal quantity of solid constituents are directly or indirectly responsible for uremia.

Our present knowledge of the **pathology** and **etiology** of uremia, as of renal edema, is based solely upon theoretic views. The theory that attributes uremic symptoms to the retention of the excretory products appears to have the strongest proofs to support it; but the positive nature of these substances, or which is the most toxic, or whether several are concerned in the causation or not, remains to be determined.

Not only some of the solid urinary constituents accumulate in the blood in uremia, but the water also is only partly eliminated, and its presence in the blood renders the latter hydremic and of lower specific gravity. Notwithstanding the fact that most cases of uremia may be traced to a marked simultaneous diminution in the quantity of urine passed, there remain still certain instances of renal disease in which uremic symptoms appear without any such perceptible diminution. Even more frequent perhaps are those perplexing cases of anuria now and then reported in which no uremic symptoms appear. In the latter instances it is probable that the elimination of products normally excreted by the kidneys may be accomplished through other channels, as by the skin and bowels; in the former it is still likely that the solid urinary constituents are retained.

Traube's theory of the cause of uremia, particularly of the nervous or cerebral manifestations, was that it is an acute edema of the brain—local or general—with cerebral anemia. This would seem to explain certain cases of nephritis, as already mentioned, in which a fair amount of urine and solid constituents are passed; also cases of anuria due to urethral obstruction in which no uremic symptoms appear; and certain cerebral disturbances. Hughes and Carter, from an experimental study,

reached the conclusion that uremia is caused by an albuminous product unlike anything found in natural urine. Strauss found both the ammonia in the blood and the retention nitrogen in the hydremic serum markedly increased in uremia. According to Croftan, metabolic disturbances dependent upon an acid intoxication must be incriminated with producing many of the fulminating signs of uremic coma. "In general terms, however, it may be assumed that interference with albuminous metabolic processes, followed by disturbed hepatic function and renal function, explains the etiology of uremia most satisfactorily."[1] Delafield attributes the sudden violent motor symptoms of acute uremia to a contraction of the arteries from some unknown cause.

The **symptoms** of uremia may be either acute or chronic in onset, severity, and course. In **acute uremia** the *severest nervous symptoms* come on suddenly; they last but a comparatively short time, and terminate fatally, with convulsions and coma, dyspnea, feeble cardiac action and pulse, fever, and pulmonary edema. These acute symptoms, however, are not infrequently preceded by mild *uremic prodromes*, as headache, somnolence, nausea, malaise, slight dyspnea, and uneasiness. Curschmann[2] claims that in threatened uremia the Babinski reflex often becomes positive before either mental disturbance or an increase of the tendon reflexes appear.

Chronic uremia is characterized by the *absence* of the marked symptoms referred to above, the milder manifestations alone appearing and lasting over a considerable length of time. Here the general prostration, the feeble cardiac and arterial states, the occasional stupor and delirium, transient dimness of vision, anorexia and nausea, irregularly hurried breathing, and muscular twitchings, indicate the grave condition of the patient. To gain a more thorough knowledge of this condition a divisional study of the symptomatology is necessary.

Cerebral Symptoms.—These vary from a slight headache, tremors, and the restlessness of anxiety to the most violent maniacal delirium and convulsions; from somnolence, low muttering, and mental stupor to profound coma; and from slight visual disturbances to complete amaurosis. The onset of a noisy delirium, and less commonly of a marked mania, is often abrupt, and may be the first manifestation of Bright's disease in an individual. Delusional insanity (*folie Brightique*) is seen in a few cases. Bischoff has observed only two cases of purely uremic psychoses among 3000 cases of insanity. Melancholia and the delusion of persecution, with suicidal and homicidal tendencies, may occur. The most characteristic symptom of uremia, however, is the convulsion (uremic eclampsia). *Uremic convulsions* are epileptiform in type, although they may be either unilateral or local. They are supposed to be due to a local or general edema of the brain, and are probably allied to the *apoplexia serosa* of early writers (Osler). The convulsions of uremia may come on suddenly or may be preceded by headache, vertigo, dropsy, nausea, and vomiting. As in the epileptiform convulsion, after the early tonic rigidity there may follow at short intervals the clonic spasm, with cyanosis, fever, and contracted arteries, and the intervening periods of unconsciousness, shallow or noisy respiration, and slow, hard pulse.

[1] "Uremia," *The Therapeutic Gazette*, November 15, 1907, by the writer.
[2] *Münch. med. Woch.*, 1911, lviii., 2054.

Coma may come on gradually as well as during the convulsive attacks. It may be preceded by headache, apathy, and insomnia, and continue progressively to deepen. A *typhoid state* not infrequently accompanies uremic coma. The temperature is usually lowered, and moderate dilatation or contraction of the pupils may be evidenced.

Uremic Amaurosis.—Blindness may follow uremic convulsions, or, rarely, it may come on without motor disturbances. It is of purely centric origin (the cortex of the occipital lobe), and its duration is short, lasting but a few days in most instances. Retinal hemorrhage may occur. *Uremic deafness*, which is probably also of centric origin, is a less common manifestation. Other nervous phenomena, as hemiplegia, monoplegia (from cerebral or spinal congestion or edema), contractures, aphasia, pruritus, paresthesiæ, and cramps in the calf-muscles are not so frequent in occurrence.

Circulatory Disturbances.—The pulse is moderately slow, tense, and full in uremia, but with the onset of acute and severe symptoms, as convulsions, it usually becomes accelerated, small, and feeble. The heart's action is labored and feeble. Indican has been demonstrated in the blood in uremia; this is absent in health and other pathologic states.[1]

Respiratory Symptoms.—Renal dyspnea, which is sometimes called "uremic" or "renal asthma," is a marked, and often an early symptom, of uremia. I believe that it is the most constant symptom of this serious condition. The respirations are deep and often stertorous in coma, or they may be irregular, accelerated, and shallow, sometimes assuming the Cheyne-Stokes type. Dyspneic attacks are especially apt to occur at night. In chronic uremia slight dyspnea may be continuous for a long time. Again, alternating paroxysmal exacerbations may arise. The uremic dyspnea is probably due in most cases to the toxemia affecting the respiratory nervous centers. It may, however, be the result of cardiac weakness or of dropsy or pulmonary edema.

Gastro-intestinal Symptoms.—Uremic stomatitis is generally seen. The breath is foul, the tongue, lips, and gums are red, swollen, and painful, and the saliva is increased. Uremic vomiting is also usually of centric origin, though it may be provoked by the irritation of the gastric mucosa, caused by the vicarious elimination of the urea and the decomposition of the latter into irritating ammonium carbonate. The vomiting may come on suddenly and be persistent. Uncontrollable hiccough and sometimes uremic diarrhea may be associated. The irritant action of the ammonium carbonate on the intestinal mucous membrane may produce a catarrhal or diphtheritic inflammation, and ulceration even (Grawitz). Uremic diarrhea may also exist apart from any gastric disturbances.

General Symptoms.—The *skin* of the face is usually pale in uremic coma. *Urea* may be excreted by the sweat-glands, and may be seen as minute glistening crystals in some of the cutaneous furrows after the evaporation of a free sweat. The skin is often harsh and dry, as in chronic interstitial nephritis. Uremic pruritus is probably the result of the peripheral irritation of the cutaneous nerves by crystals of urea. The *temperature* is generally lowered, but uremic fever frequently accompanies the convulsions or they may be preceded by "uremic chills." In some cases the temperature rises to 105°–107° F. (40.5°–41.6° C.) just before

[1] For test, see *Zentralblatt für Innere Medizin*, Dec. 23, 1911, by W. von Moraczewski and E. Herzfeld.

death, whilst in other cases, characterized by coma that deepens into collapse, the temperature may be so low as 91° or 93° F. (32.7°–33.8° C.).

There is not infrequently an *ammoniacal odor* about a uremic patient. The *urine* is diminished in quantity, is generally highly albuminous, and deficient in urea. A previous dropsy is sometimes markedly reduced upon the appearance of acute uremic symptoms.

Duration and Prognosis.—Acute uremia is manifested by coma and convulsions, seldom lasting more than a few days. Chronic uremia, in which milder nervous symptoms, nausea and vomiting, and dyspnea are more prominent, may persist, however, for many weeks. While a grave condition, uremia, even in its most acute and violent forms, is not at once necessarily fatal, for under proper treatment—as by venesection, for instance, followed by judicious hygienic measures—life may be considerably prolonged. Sooner or later, however, barring a possible death from some intercurrent affection, a fatal result is inevitable.

Diagnosis.—Uremia may be recognized by the history, the marked arterial tension, and the accentuated second sound of the heart; also by the albuminuria (the urine has to be withdrawn), the temperature, and the odor of the breath. The presence of dropsy in some cases is a valuable indication of the nephritic origin of uremic manifestations.

Differential Diagnosis.—Uremic unconsciousness coming on suddenly, as in chronic interstitial nephritis, may simulate *alcoholism*, *cerebral hemorrhage* (*apoplexy*), *cerebral tumor*, or *meningitis*. The points of dissimilarity between the first two conditions and uremia are here tabulated (Herrick):

Cerebral Hemorrhage.	Alcoholic Narcosis.	Uremia.
Pupils unequal or dilated.	Pupils contracted or dilated; eyes injected.	Pupils generally dilated; albuminuric retinitis.
Stertorous, puffy breathing, and flapping cheek.	No stertorous breathing.	Sharp, hissing stertor.
No odor.	Odor of alcohol.	No odor, unless urinous.
Paralysis; hemiplegia.	No paralysis, usually.	No paralysis.
Unconsciousness absolute.	May be aroused.	May or may not be aroused.
Pulse slow and strong or irregular; arteries often atheromatous.	Pulse frequent and feeble.	Pulse at first strong, later weak and rapid; tension strong; arterio-sclerosis.
Coma sudden and deep.	Coma gradual.	Coma gradual or sudden.
Convulsions late; may be unilateral.	No convulsions.	Preceded by general convulsions, headache, etc.
Urine generally negative.	Urine generally negative.	Urine albuminous.
Apoplectic habit; heart may show hypertrophy.	Red face and nose, heart often weak, dilated, myocarditic.	Edema and pallor; heart hypertrophied.

In *meningitis* the mode of onset, the rigidity of the neck, incoherence or mild delirium, photophobia, and pronounced fever point to the distinction.

Uremic coma must also be differentiated from *opium-poisoning* and *diabetic coma*. Chronic uremia must not be confounded with the asthenic state of *typhoid fever* and *acute miliary tuberculosis*. In *opium-poisoning* the pupils are contracted and do not respond to light. Again, in opium-poisoning the respirations are slow, deep, and full, and the patient may answer rationally when aroused. In uremic coma, it will be remembered,

consciousness is abolished. In *diabetic coma* the history must be learned, the harsh, dry skin and emaciation noted, and especially are the ethereal odor and the Burgundy-red reaction of the urine (acetone) with the tincture of the chlorid of iron to be observed; sugar is also present.

The **prognosis** is grave, but guarded; it is even favorable in many cases, so far as immediate results are concerned.

Treatment.—This will be detailed in the discussion of the various forms of nephritis. Suffice it to say that the supreme indication is the prompt elimination of the poisons in the blood. When diaphoresis and catharsis fail, venesection should be employed; the latter measure is also probably the most reliable in urgent cases of uremic convulsions or coma. The counter-injection (intravenous) of normal salt solution may be indicated in cases of profound weakness threatening collapse. Bäumler advises against the introduction of salt solution and the use of salt in the diet. Frey recommends lumbar puncture to evacuate toxins and reduce pressure on the brain in the cerebral type.

Bozzoli recommends the subcutaneous injection of sterilized serum because of the gratifying results secured in a number of cases of uremia.

AMYLOID KIDNEY.

Definition.—Amyloid (waxy or lardaceous) degeneration of the kidneys is usually coexistent with a similar degeneration of other viscera.

Pathology.—Macroscopically, the amyloid kidney appears pale, greenish or yellowish-white, firm, and uniformly enlarged, and the surface is smooth, glistening, and often mottled, owing to the prominence of the stellate veins. On section a homogeneous, anemic, or "bacon-like" surface presents itself, particularly in the cortical region. The cortex is wider than normal; the pyramids may be red in color and slightly infiltrated; and the glomeruli may show an infiltration by the glistening, translucent amyloid (albuminoid) material. On the application of Lugol's solution of iodin to the amyloid areas a mahogany-red color is produced. Brushing over the amyloid substance with a solution of iodin, and then with dilute sulphuric acid, gives a blue or violet tint. Similarly used, a 1 per cent. solution of methyl-violet strikes a red color. The capsule of the kidney is thickened, though not always adherent.

Microscopically, the amyloid change is generally found in the early stages to affect the walls of the capillaries of the Malpighian tufts. The walls are swollen with the homogeneous material and the vessel-lumen is diminished or obliterated. The straight uriniferous tubules are also infiltrated later perhaps, the deposit occurring primarily in the membranæ propriæ. A diffuse nephritis is nearly always an associated condition. The tubules generally contain hyaline casts. Fatty degeneration of the epithelium, glomerulites or waxy glomeruli, and a thickening of Bowman's capsule are common in markedly amyloid kidneys. In advanced cases most of the secretory structure becomes atrophied. Amyloid infiltration of the smaller granular kidney is less common than of the large white kidney, with intense parenchymatous changes.

Hypertrophy of the heart is not always present in amyloid disease of

the kidneys. Amyloid infiltration of other organs, however, as of the liver and spleen, is usually associated with waxy kidneys.

Etiology.—The causes of amyloid kidney are those of the amyloid change affecting (either simultaneously or nearly so) other organs, as the spleen, liver, and intestines.

Commonly, amyloid disease is marked also in the other solid organs named above; it is secondary to wasting diseases, cachexiæ, and the like. Perhaps the most frequent cause of the waxy kidney is tuberculosis, especially of the lungs ("chronic ulcerative phthisis"): tuberculosis of the intestines also is often associated. Next in order are the prolonged suppurations, particularly of the bones, as in osteitis of the vertebræ and hips (usually tuberculous). Chronic empyema, intestinal ulcers, vesico-vaginal fistulæ, and other purulent affections, chronic in nature also, have the same etiologic effect. Amyloid kidney is often present in syphilis, especially in the tertiary stage, when ulceration of the mucous surfaces and of the bones is present. Rarely, gout, malaria, leukemia, cancer, and chronic valvular endocarditis with insufficiency seem to produce amyloid disease.

Symptoms.—These vary greatly according to the extent to which the amyloid degeneration has encroached upon the normal kidney-structure, and may be overshadowed partially or completely by those of the dominant causal affection.

The *urine* is pale yellow, clear, and variable in quantity, and the amount passed in twenty-four hours is sometimes normal or may be slightly diminished. More frequently, perhaps, it is increased, and especially in marked or advanced cases. The specific gravity is apt to be low (1015–1005), and there is seldom any sediment.

Serum-albumin and *globulin* may both be present in the urine; but a highly significant condition, and one that is seemingly diagnostic, is the high proportion of globulin as compared with the serum-albumin (Salkowski, Senator). *Tube-casts* may be found, but their presence may be only temporary; they are usually wide, hyaline, fatty and granular, and very few in number (Fig. 67). The amyloid reaction may be elicited with the hyaline casts; symptoms referable to the kidney are often absent in comparison with those of the nephritides. *Dropsy* is not invariably present, and when present is but moderate in degree and generally in the legs only. It is proportionately prominent with the increase in the anemia, circulatory depression, and wasting of flesh and strength. The latter manifestations, constituting a cachectic appearance, are quite commonly observed in amyloid kidney.

The associated enlargement and the firm, sharp outlines of the liver and spleen are of diagnostic significance. *Marked diarrhea* may be due to coexisting amyloid infiltration of the intestines or to tuberculous intestinal ulcers, and is often seen in advanced cases.

Diagnosis.—This can seldom be made upon the urinary manifestations alone. Important and often necessary adjuncts are the histories of causation and of the associated symptoms and physical signs. Thus, there will be evidenced in most cases tuberculosis, chronic bone-suppurations, or syphilis, while coexisting hepatic and splenic enlargements, wasting, and cachexia are usually present. In any of the diseased conditions mentioned amyloid kidney may be diagnosticated with reasonable cer-

tainty upon the development of an increased quantity of pale clear urine of low specific gravity and containing a large amount of albumin, or even with slight albuminuria.

From *parenchymatous nephritis* amyloid kidney is to be differentiated by the history, by the more marked and generally distributed dropsy, and by the albuminuric retinitis that characterize the former. In *chronic interstitial nephritis* there are less marked albuminuria and dropsy, and there are present arterio-sclerosis, cardiac hypertrophy, and a pronounced tendency toward uremic symptoms.

Prognosis.—This varies with the cause. Incipient bone-disease or tuberculosis, with only slight evidences of amyloid change in the kidneys, may be controlled. As a rule, however, the structural alterations are so far advanced, and the constitutional powers of resistance so much enervated, before the amyloid infiltration can be distinctly apprehended that in the majority of instances the prognosis is entirely unfavorable. In decided cases death ensues in from several weeks to as many months.

Treatment.—This also depends upon the causal affection. Hygienic and dietetic measures are always useful, however, with a view to improving the general nutrition. The iodid of iron has been recommended as an alterative, and easily assimilable and palatable fats and tonics may also be tried. Tuberculous cases require creasote or allied preparations; syphilitics require mercurials and iodids; while malarial subjects do best under the systematic use of arsenic, iron, and quinin.

NEPHROLITHIASIS.

(*Renal Calculi; Pyelitis Calculosa; Renal Colic; Gravel.*)

Definition.—A condition characterized by the formation of fine or coarse concretions in the kidney-substance or in the renal pelvis by the precipitation of certain of the solid urinary constituents.

Varieties.—According to their size, renal concretions are variously termed—(1) **Renal sand,** of which the particles are fine and pulverized; (2) **Renal gravel,** consisting of coarse grains or even of pea-sized concretions; (3) **Renal stone, or calculus,** when larger masses than the preceding exist, either more or less rounded or as stony casts or moulds of the pelvis of the kidney, its infundibula, and calyces (*dendritic* or *coral calculi*).

According to their composition, the chemical varieties of renal concretions are—(1) *Uric-acid* calculi, the most frequent in occurrence. Urates are often associated in the calculus with uric acid, thus producing stratification. These concretions may occur as sand, gravel, or large stones; they are usually quite hard, reddish-brown or black in color, and have a smooth though irregularly shaped surface. The fracture is crystalline. Pure uratic stones may occur in children. Mackarell, Moore, and Thomas have shown that uric acid is not the most common constituent of renal calculi, but calcium oxalate and calcium phosphate.

(2) *Calcium-oxalate* concretions occur more rarely in the kidney. They constitute the so-called "mulberry calculi," from a fancied resemblance to the mulberry, owing to their dark-brown or black color and very irregular and nodulated or prickly appearance. They are also quite dense; lamination, however, is not common, although they are sometimes formed about a uric-acid nucleus.

(3) *Phosphatic* calculi of the kidney are still less common than the oxalate, but they are more common in the bladder. They may consist of calcic phosphate or ammonio-magnesic phosphate, and may possibly be associated with calcic carbonate. Phosphatic salts are most often deposited secondarily about uric-acid or oxalate calculi in the alkaline urine of a cystitis set up by the irritation of the true renal stones. Phosphatic calculi are grayish-white in color and are comparatively soft.

(4) Renal stones composed of *cystin*, *xanthin*, *carbonate of lime*, *fatty or saponaceous matters* (urostealith), *indigo*, and *fibrin*, though of extreme rarity, have been occasionally reported. Cystin calculi have a pale-yellow color and a waxy luster.

Pathology.—The anatomic changes of the kidney vary with the degree and persistence of the irritation, the size of the calculi, and their passage or retention. Sometimes numerous granular and pea-sized concretions are found in the renal pelvis, with desquamated epithelium and a turbid urine. Interesting cases are those in which a dendritic stone occupies a great portion of the atrophied kidney-substance, as well as the entire pelvis of the organ. In one of my own patients the left kidney was, apparently, nearly twice the normal size, owing to the presence of a large coral-calculus (uric acid and urates), connected by an isthmus with a rounded stone in the inferior portion quite as large as a large walnut. The pelvis of the right kidney also contained a dendritic calculus.

Secondary Lesions.—Perhaps the most usual result of renal concretions is a pyelitis: this may be simple catarrhal, diphtheritic, or purulent, with or without hemorrhages, depending upon the intensity of the mechanical irritation. A pyelo-nephritis may follow in severe cases, as may even a general suppuration (pyonephrosis) or perinephric abscess and perforations. Renal pus-cavities are sometimes found *postmortem* containing numerous small stones. Hydronephrosis is another important pathologic sequel, in which the cause is to be attributed to the blocking of the ureter by an erstwhile passing stone or by the closing of the aperture of a ureter from within the pelvis. Pressure-necrosis and perforation may thus be induced. Owing to the prolonged pressure of a dendritic calculus, there is commonly a distinct and marked atrophy of the renal parenchyma, resulting in chronic diffuse nephritis with little or no exudation.

Etiology.—The definite causation and the exact manner of formation of renal concretions are still unestablished. We may infer not a little, however, with some good reason, since the *predisposing causes* are rather distinct. Thus, in children and in advanced life (before 15 and after 50 years of age) the occurrence of calculi is most common, the uratic variety being most frequent in the former and the uric acid in the latter. Men are subject to nephrolithiasis more often than are women. The uric- or lithic-acid state (lithemia), gout, and the various influences that induce these conditions, as an excessive meat (proteid) diet or a sedentary life, seem to predispose to stone. Heredity, I believe, plays a prominent part in many cases.

Broadly speaking, any habit of the system that encourages the precipitation of insoluble abnormal ingredients or of normal ingredients in excess, owing to chemical changes in the urine, tends to the formation of calculi. It should be stated, however, that the *primary causes* of calculus-formation is the presence of some substance in the urinary tract

that affords a nucleus about which the successive layers of crystals may deposit and adhere, such as bits of mucus, colloid material, epithelial shreds, parasitic ova, bacteria, blood-clots, and tube-casts.

It is generally believed that the requisite conditions for the formation of a uric-acid renal calculus are—a highly-acid urine, a low percentage of salines, and deficiency of the normal urinary coloring-matters.

Symptoms.—These may be slight, progressive, and chronic, or they may be intensely acute and comparatively short in duration, though subject to repetition—*i. e. renal colic.* It is not unusual for patients to pass uric-acid sand and gravel for years without much complaint. A sudden blocking of a ureter, however, or a slowly-passing stone of distending dimensions produces great agony at times. A smooth, snugly-fitting dendritic calculus in the pelvis may not cause any symptoms for years until the destruction of tissue by its weight and mechanical irritation ensues; there is then a progressive failure of health, a constantly increasing pain in the back, *occasional hematuria*, tenderness on pressure over the diseased kidney, both anteriorly (deep) and posteriorly, and finally *uremia* and death.

The characteristic symptoms of stone in the kidney appear as an attack of *renal colic.* This happens when a calculus in its passage down the ureter acts as a mechanical irritant, or when it is caught and stopped in the passage. The large "gravel" or pea-sized and more or less rough stones usually cause the attack, which comes on, as a rule, quite suddenly, although it may be preceded by a chill and some general uneasiness or by slight pain in the region of the kidney. It may be excited by a sudden muscular effort. The pain is tearing in character, and rapidly reaches an agonizing maximum of severity, starting from the lumbar region and extending down along the ureter into the groin, and often into the testicle and inner side of the thigh. The paroxysm may appear in the form of a diffuse abdominal and lumbar pain in some instances. There is local tenderness on pressure, and nausea and repeated vomitings are frequent. The patient is often collapsed, and perspiration, a rapid, small, and feeble pulse, trembling, anxiety, bodily twistings about, convulsions even, and syncope may ensue. There may be moderate fever. The *urine* is scanty or may be suppressed for a time, and is often bloody. Frequent and painful attempts at urination are made, with the passage of but a few drops at a time, owing perhaps, in part at least, to a reflex spasm of the vesical sphincter (vesical tenesmus). The presence of pus and of pelvic epithelium in the urine indicates a pyelitis. When a large quantity of clear urine is passed it may be looked upon as having come from a healthy kidney.

The *paroxysm* of renal colic ends when the impacted stone passes out of the ureter. This may occur within a few hours or it may take several days; or colic may be intermittent.

Recovery is not always complete upon the evacuation of the stone. The previously retracted testicle may remain painful, and there are apt to be aching and soreness over the affected kidney and ureter.

In certain severe cases of mechanical irritation the symptoms of pyelitis, pyelo-nephritis with abscess, or hydronephrosis may be superadded. Anuria and uremia may result.

Nephrolithiasis as a *chronic affection* may exist for many years, with recurring paroxysms of renal colic. I observed a case for five years

that had extended over a period of thirty years, until it finally came to necropsy. Between the attacks of colic the patient may be entirely comfortable, save perhaps an occasional burning in the urethra on micturition, owing to a highly-concentrated, acid urine or to the passage of minute uric-acid granules. There are apt to be pain and tenderness over a kidney containing a large imbedded stone. A smoky-hued urine, due to slight hematuria, is also sometimes present in long-standing cases of renal calculus, particularly after exertion.

A *renal intermittent* fever may occur in nephrolithiasis, and is analogous to the hepatic intermittent fever of cholelithiasis.

Pyelitis—simple or purulent—with late involvement of the kidney-parenchyma (pyelo-nephritis) is a frequent concomitant of chronic nephrolithiasis. The presence of pus in the urine is constant, with an absence of renal epithelium in cases of an abscess-cavity of the kidney. In ordinary pyelitis the pyuria is often intermittent.

The general health of patients with nephrolithiasis is, as a rule, remarkably good. Anorexia is not only seldom present, but such persons are habitually free and good livers. Persistent headaches with nausea, however, should warn one of uremia. Splenic and hepatic enlargement may be found with prolonged suppurative pyelo-nephritis, indicating amyloid disease.

Diagnosis.—This resolves itself into a study of the diagnostic characters of (*a*) the attacks of renal colic, (*b*) of the underlying systemic condition in general, and (*c*) the renal condition in particular that renders these attacks possible. The latter can be discovered only by a careful and continuous study of the clinical history and urinary manifestations as outlined in previous paragraphs.

Nephrolithiasis may be positively diagnosed in a case in which, after sudden, agonizing, colicky pain, referred to either lumbar region and radiating down the ureteral course to the testicle, a concretion is found to have passed with the urine. It is therefore necessary in a suspected case of renal colic to pour the urine through a fine sieve as soon as passed. The more recent improvements in the operative technic for producing the Röntgen rays enable us to detect renal calculi with accuracy as to their number, size, and relative position. The injection of collargol through an opaque sound, which has previously been introduced into the pelvis of the kidney, enables an *x*-ray picture to outline the pelvis and calices. In the case of urinary calculi consisting of uric acid alone, however, the findings are totally negative on Röntgenoscopy (A. Seelig[1]).

Differential Diagnosis.—Renal colic must not be taken for *biliary or intestinal colic*. The antecedent history is of great value in arriving at a diagnosis. In biliary colic there may be jaundice, and pain referred to the upper rather than to the lower abdominal zone, both of which symptoms are absent in renal colic; while in the latter the disturbance of micturition and the character of the urine, especially the hematuria, are characteristic.

In intestinal colic the griping pain is usually most intense in the umbilical region, is often relieved by pressure, and is associated with tympanites and constipation; it has usually a dietetic origin, while the renal and urinary symptoms are absent. The exclusion of *lumbodynia* and *lumbo-abdominal neuralgia* is not so difficult. The differentiation of the

[1] *Zeitschrift für Urologie*, Berlin, April, 1912.

varieties of calculi from the symptoms is not positive. It has been suggested, however, that the oxalate stones usually cause the sharpest pains and the hematuria. Right-sided ureteral pain felt over the lower abdominal region may be confounded with *appendiceal colic*. Musser has found the pain of renal colic to be more paroxysmal and less uniform in location than in the latter. Early *renal tuberculosis* (*vide*), with its hematuria and pyuria, must be differentiated from renal calculus also. Cases of supposed stone in the kidney with most of the typical symptoms in which, however, no stone was found at operation (false stone), have been reported by James Tyson.[1] In all instances adhesions were found between the capsule and the kidney itself, and all were relieved by operation.

Prognosis.—This should always be guarded, owing to the possible dangers and complications that frequently attend nephrolithiasis in all of its forms. Thus the passage of gravel without marked symptoms tends to persist or recur—in both events an unfavorable tendency, since subsequent formations are apt to be larger and cause serious symptoms. An attack of renal colic may itself be fatal. Large latent calculi (dendritic), of long standing, are nearly always incurable, and in most instances lead to such grave complications as pyelo-nephritis, pyo- and hydronephrosis, perinephric abscess, and uremia.

Treatment.—Paroxysms of renal colic call for prompt relief. This is best afforded by hypodermic injections of morphin and atropin, coupled with hot baths or fomentations applied to the loins. The free use of hot drinks, as lemonade, soda, or plain water, is also helpful in promoting the passage of the stone. Drinking large quantities of glycerin mixed with water has proven of service in some cases. Cases of excessive suffering require the inhalation of chloroform.

The treatment of the nephrolithiasis without or between attacks of renal colic is most important. First to be considered are the **hygienic** and **dietetic** measures, for in mild and uncomplicated cases much can be done to prevent the aggravation of the disorder, and at least the formation of larger concretions may be delayed. When the tendency is to uric-acid gravel (the commonest variety), the patient should live a regular, calm, steady, and temperate life. Exercise should be so managed that it may be taken rather moderately in the open air, and with a view to preventing additional weight in persons of fair nutrition and to promoting a reduction of weight in the obese. In short, the exercise should be sufficient to use up all nitrogenous food, so that the formation and elimination of urea may be increased to normal. Hence I would strongly advise a clinical study of the percentage of urea in the urine (*vide* p. 1003).

Over-indulgence in food, particularly in red meats (liver, sweetbread, and similar nuclear food), should be prohibited, owing to the ready formation of uric acid from the latter. Alcohol should not be taken. On the other hand, since the urine is apt to be scanty and highly acid, the patient should be encouraged to drink freely of plain and alkaline waters. The value of various pure spring-waters as diluents is undoubted, the Buffalo, Londonderry, and Otterburn Lithia, the Saratoga, Bedford, and Poland waters, all being distinguished for their purity. More marked and more generally useful for their alkilinity are the Carlsbad, Vichy, and carbonated waters. In cases characterized by occasional hematuria

[1] *New York Medical Journal*, May 26, 1906, p. 1106.

the Rockbridge alum-water may be tried. Plain soda-water and lemonade may be used as adjuvants.

The **medicinal** treatment of nephrolithiasis is aimed to secure a solvent and disintegrating action upon the stones; it is symptomatic. It is extremely doubtful whether stones once formed in the pelvis of the kidney and remaining there are ever dissolved, though certain drugs would seem to have had an eroding effect in some instances, and they are to be recommended as useful in preventing the formation of new deposits. Lithium citrate or carbonate in 5-grain (0.324) doses in tablet form, three or four times daily, has been generally employed for the purpose. Sodium phosphate and the vegetable salts of potash, as the citrate, acetate, and tartrate, are useful. Much water, especially the carbonated, should be drunk, along with doses of the above, in order to facilitate the solvent action, and in this way relieve, in a measure, the local distress and pain. Recently piperazin, lycetol, and urotropin have been brought forward as uric-acid-calculus solvents by some clinicians, and that they have such action as is claimed has been proved beyond a doubt in certain cases. Whilst they deserve a further trial in nephrolithiasis, it is too much, however, to expect successful results in every case.

Recently, Van Noorden and Strause have recommended calcium carbonate (gr. x–xv—0.648–0.972—or more thrice daily). The theory is that the calcium unites with the acid phosphates in the intestines, and thus reduces the deuterophosphates in the urine, leaving the protophosphates to dissolve the uric acid. They report excellent clinical results.

The reaction of the urine must be tested at stated intervals and kept faintly acid. Should the urine become alkaline, the alkaline treatment must be suspended for a while, or a secondary deposit of phosphates about the uric-acid stone may be induced. Nagging lumbar pains may be relieved by occasional doses of such analgesics as phenacetin, belladonna, hyoscyamus, codein, and indirectly by the sweet spirits of niter, buchu, and uva ursi. Renal hemorrhage may be controlled effectually by the use of the fluid extract of ergot, or by alum in 10- or 15-grain (0.648 or 0.972) doses, or by gallic acid in 20- or 30-grain (1.29–1.94) doses.

Efforts to acidify the urine are indicated when the calculus happens to be composed of phosphates or of calcium carbonate. This is more difficult of accomplishment than when it is necessary to reduce the acidity. Saccharin in 2- or 3-grain (0.129–0.194), and benzoic and boric acids in 5- to 15-grain (0.324–0.972) doses, in capsules, seem to be most useful for this purpose. It is claimed for calcium carbonate, again, that it diminishes the phosphates without making the urine alkaline.

The question of surgical interference must be decided in not a few cases; thus, it may be briefly stated that in protracted and obstinate cases of calculous renal disorder, with persistent local pain, a gradually decreasing capacity for work, and evidences of severe pyelitis, pyelonephritis, or, worse, of perinephric abscess, the surgeon must operate. In the simplest cases a nephrotomy or nephro-lithotomy may be performed and the stone removed. Where the renal structure is much damaged it may be necessary to do a nephrectomy. To avoid the increased perils of the latter operation, however, it were better that a nephrotomy were done as early as consistent with the diagnosis of incarcerated pelvic stone and the condition of the patient.

ACUTE NEPHRITIS.

(*Acute Bright's Disease; Acute Diffuse Nephritis; Acute Parenchymatous Nephritis; Exudative, Catarrhal, Tubal, Desquamative, and Glomerulo-nephritis of Acute Course.*)

Definition.—An acute inflammation of the kidneys, more or less diffuse in nature. It may be either of a mild, severe, or grave character. Delafield describes three varieties of acute renal inflammation under the common synonym of acute Bright's disease, as follows: (1) *acute degeneration of the kidneys*, (2) *acute exudative nephritis*, *and* (3) *acute productive nephritis*. At present writing the three forms following are recognized by many writers; (*a*) Acute tubular; (*b*) acute glomerular and, (*c*) acute diffuse nephritis. The last-named variety manifests the symptoms of the first two. Moreover, it is not possible to recognize clinically either acute tubular or acute glomerular nephritis in many instances, but I shall incidentally give a brief description of the morbid changes and clinical features which they present.

Pathology.—From the very mild to the gravest cases of nephritis there is an intermediate series of continuously more marked pathologic changes in the renal tissues. These depend greatly on the amount of poisonous material circulating in the kidneys and eliminated by them.

In the mildest cases the *macroscopic* appearances of the kidneys may present nothing distinctly abnormal. As a rule, however, the organs are slightly enlarged, swollen, and somewhat softened. These conditions are more evident when the interstitial exudation is abundant and when inflammatory edema is evident. The kidneys may be reddened and congested and appear bloody on section, or they may be pale and mottled. In examples of the former, hemorrhages may be formed beneath the capsule (*acute hemorrhagic nephritis*), though it is more common to see red patches of hyperemia alternating with opaque, whitish portions on both the outer and cut-surfaces of the kidneys. The cortex especially is swollen, turbid, and pale, or slightly congested in the mildest cases, and is deeply mottled (red and pale glomeruli) or hyperemic in severe instances. The pyramids usually show an intense redness. The surfaces are smooth and the capsule non-adherent.

Microscopically, in mild cases, there is simply a cloudy swelling or a granular (parenchymatous) degeneration of the epithelium of the Malpighian tufts, Bowman's capsule, and of the cortical uriniferous tubules (*acute tubular nephritis*). These changes may be almost exclusively limited to the glomeruli, as in some cases of scarlatina, and hence the term *glomerulo-nephritis* (*acute glomerular nephritis*). The cells are swollen, opaque, and irregular in shape, while the cell-contents are granular (albuminoid or fatty). A further advance in the process is seen in cellular coagulation-necrosis or disintegration, desquamation of the cells, and hyaline degeneration of masses of them in the tubules. Acute degenerative changes are frequently found in the acute infectious diseases or when inorganic poisons have been introduced into the body. In phosphorus-poisoning actual fatty degeneration of the epithelium may be found. A rapid necrosis of cells is also met with in severe cases.

True acute nephritis is not only characterized by changes of the renal epithelium (the parenchyma), described above, but the inflammatory exudate (serum, leukocytes, and erythrocytes) is found between the tubules. The kidneys show different stages of the process in different portions. In some places there is only a slight cellular infiltration of the intertubu-

lar tissues; in others, besides the desquamation of necrotic epithelial cells and the presence of hyaline casts in the tubules, the interstitial tissue is swollen by the coagulated sero-fibrinous exudate, abundant leukocytes, and some red blood-corpuscles. It should be stated that the inflammatory exudate collects also in the Malpighian bodies and tubules. The epithelium lining the latter, especially the convoluted portion, is often flattened, and the tubules themselves may be dilated and choked with degenerated cells or, more frequently in the straight tubules, with hyaline casts. The white blood-cells that are found infiltrating the stroma of the kidney are not usually equally diffused, but are collected in foci in the cortex.

In most cases of diffuse exudative nephritis new epithelium appears, and a restoration of the glomerular function takes place. In the *productive variety* of acute diffuse nephritis, however, according to Delafield, the lesions—consisting of a cellular growth in the capsules and of connective tissue around thickened arteries—are more permanent in character from the first, and hence the increased gravity of the disease. In the more intensely acute cases the new tissue between the tubules is largely cellular; in those of a subacute type it is relatively dense and fibrous.

Anasarca and pleural, pericardial, and peritoneal dropsy are also found in those dying of acute Bright's disease. Complicating conditions (lobar pneumonia, meningitis) are sometimes seen *postmortem*.

Etiology.—Acute nephritis may occur at any time of life, though it more often makes its appearance before than after middle life. Males are more susceptible than females, and particularly when engaged in occupations requiring exposure to cold and wet. The habitual use of alcoholics is generally a predisposing cause of acute Bright's disease.

The principal exciting causes of acute diffuse nephritis are the following: (1) *Those acting on the skin*, as cold and dampness, extensive burns, and chronic skin-diseases. In many cases it is difficult to estimate whether the influence of alcoholic intemperance predominates or the exposure incident to it. Thus, acute intoxication from beer-drinking itself may cause an attack of acute nephritis. The disease may also be attributed, at times, to exposure to cold and wet irrespective of alcoholic indulgence. It may be presumed with reason that in such cases there is some inherent or acquired weakness or a susceptibility of the kidneys, rendering them the weak links in the visceral or systemic chain. Watson[1] states that cold is a factor producing, not acute nephritis, but the onset of acute symptoms in an already existing chronic nephritis.

(2) *Biologic Toxic Agents.*—These embrace the poisons of the acute infectious diseases, though in the majority of cases scarlet fever is the primary affection. Nephritis may supervene during the height of scarlatina, but more often it occurs in the second or third week of convalescence. Other infectious fevers may also cause acute nephritis (smallpox, typhus, typhoid, relapsing fever, epidemic influenza, cholera, diphtheria, yellow fever, measles, chicken-pox, erysipelas, septico-pyemia, acute lobar pneumonia, cerebro-spinal meningitis, dysentery, acute articular rheumatism, and tuberculosis: syphilis and malaria are rare causes). Acute infectious nephritis may also occur as a primary disorder, and the brunt of the affection may fall either upon the kidney, rather than upon any other part, or upon the organism as a whole, as in the fevers.

(3) *Chemical Toxic Agents.*—Among the principal irritants of this

[1] *British Med. Jour.*, 1912, i. 822.

class are turpentine, cantharides, carbolic and salicylic acids, iodoform, the mineral acids, potassium chlorate, and such inorganic poisons as phosphorus, lead, arsenic, and mercury. The excessive ingestion of highly-acid, spiced, or adulterated foods (as from salicylic acid and lead chromate) may in certain individuals cause acute renal inflammation. Ether anesthesia may induce acute diffuse nephritis.

(4) *Pregnancy.*—Here the nephritis (*gravidarum*) comes on in primiparæ, usually in the last months of pregnancy. It is probably caused by renal engorgement due to mechanical pressure, as well as to nutritive disturbances in the kidney, owing to the altered blood-condition.

(5) Latent and insidious *chronic nephritis* may be the cause of an onset of a manifest acute nephritis.

(6) Finally, traumatism to the kidney may cause acute nephritis, when the urine may contain hematoidin crystals.

Symptoms.—The *onset* varies with the cause of the nephritis, though generally it is rather sudden. Chilliness, nausea and vomiting, pain in the back, and, within twenty-four hours, dropsy, are seen in some cases. Children may be seized with *convulsions* (uremic), and adults are not less liable to them in severe attacks. Fever may be present, although it is neither constant nor high. The characteristic symptom is the early appearance of *edematous puffiness* of the eyelids and face, with pallor of the skin. Soon (and sometimes at first, even) a swelling is noticed about the ankles and legs, and in marked cases the whole body becomes dropsical, so that pitting on pressure may be observed pretty much all over the bodily surface. In such instances the scrotum and penis or the labia may become enormously distended, the skin having almost a translucent appearance.

Local symptoms, as pain and tenderness in the lumbar region, are often wanting and are never marked. There may be a desire to micturate often, accompanied by slight burning and vesical tenesmus, due to the concentrated urine. In very severe dropsy the tense, dry skin, as of the limbs, may be sensitive or even painful to the pressing finger. Movements of the body are often difficult, painful, and distressing in marked anasarca. Intense headache and backache may precede the onset of uremia. In mild cases the renal condition may be overlooked unless a urinary examination is made. The characteristics of the urine in acute nephritis are all-important. The total *quantity* passed in twenty-four hours is diminished, and may be very scanty, sometimes amounting to not more than from 5 to 25 ounces (150–740 c.c.). Suppression occurs in some cases of toxic origin, when an acute degeneration or necrosis of the renal epithelium takes place, and in the most severe exudative inflammations. The *specific gravity* is increased to 1025 or more, early in the case; later it may be as low as 1010 or 1015. The *color* is darker than normally, and is usually smoky-red or reddish-brown, according to the amount of blood passed. If the morphologic constituents are present in great quantity, a more or less abundant flocculent sediment appears on standing.

Microscopically, some red blood-corpuscles and renal epithelium are found, along with the characteristic *blood*, *epithelial* and *granular tube-casts* (Fig. 62). Typical casts may rarely be found without the presence of albumin. Chemically the urine is *acid*, and on boiling a *thick, curdy precipitate of albumin* forms. The percentage of the latter by Esbach's method varies from 1 to 1.5 per cent. The urea and gross solids are diminished. The molecular concentration or osmotic pressure of the

urine is usually reduced (hyposthenuria), so that the freezing-point (cryoscopy) is 1° or less than 1° C. (instead of the normal 1.3° to 2.3° C.) below that of distilled water (0° C.) (A. O. J. Kelly).

Other symptoms may develop during the course of acute Bright's disease. If *great general edema* is present, physical signs of hydrothorax, ascites, and hydropericardium may be elicited. The first-mentioned condition is bilateral and causes dyspnea; the second increases the dyspnea by pressing the diaphragm upward; and the last impairs the heart's action. Strümpell describes a form of pneumonia—a "stiff inflammatory edema"—midway between lobar pneumonia and broncho-pneumonia, that sometimes develops in severe cases of acute nephritis. Edema of the conjunctivæ, soft palate, and larynx may also occur. Recently, Lapinsky reported a fatal case of acute parenchymatous nephritis in which severe bilateral sciatic neuritis was associated.

The *pulse* is often hard and tense, and, though slow at first, it may become accelerated later. Cardiac hypertrophy of a slight degree may be detected. The second aortic sound is accentuated. The arterial pressure is considerably elevated. Epistaxis is an occasional symptom and subconjunctival hemorrhages are sometimes seen as a result of uremic convulsions that may not have been witnessed. A very constant symptom is the dry, anemic skin. *Uremic manifestations* may ensue at any time during the course of the disease. They appear early in the most severe cases, with intense headache and backache, vomiting, and convulsions.

The *clinical course* in other cases differs somewhat from the above, which may be considered as the common form resulting from exposure. Acute nephritis *occurring as a complication of the infectious fevers*, except scarlatina, may be characterized by the very slight degree, or even by the absence, of dropsy. Albuminuria, hematuria, anemia, and uremia supervene in the graver affections; this is the *acute tubular nephritis*. In *scarlatinal nephritis* we have an illustration of *acute glomerular nephritis;* anasarca is common, and slight edema at least is quite constant. During the period of convalescence tube-casts (granular or fatty granular) may be found in the urine (Fig. 63). In mild affections simply a little albumin and a few hyaline casts reveal the parenchymatous degeneration. In cases of *degenerative nephritis* due to mineral poisoning the subsidence of the acute toxic symptoms may be followed by the *typhoid condition*. In the so-called *nephro-typhoid condition*, where typhoid fever begins with pronounced symptoms of acute nephritis, hematuria may be marked. The *nephritis of pregnancy* is usually gradual in its onset, and the albumin increases from month to month. Some hyaline or faintly granular casts are found (Fig. 65), and erythrocytes rarely appear in the urine. *Danger of eclampsia* is constant until the albuminuria has subsided.

Acute productive nephritis (Delafield), in which there is a tendency to the formation of patches or wedges of fibrous tissue, is characterized by higher fever, by cerebral and circulatory disturbances of a typhoid nature, and by anemia, dropsy, and a highly-albuminous urine, even though blood may be absent and casts may be few. The dropsy is most apparent in the legs. Dyspnea, vomiting, diarrhea, and a progressive and rapid loss of flesh and strength ensue until convulsions or coma, sometimes preceded by acute maniacal excitement, end in death. Milder cases, lasting from two to four weeks, apparently get well, albumin and casts persisting, however, until, after an interval of weeks or months,

another and similar attack occurs. In short, the first acute attack is liable to chronic repetition until a fatal one takes place.

Diagnosis.—The condition cannot be overlooked when the urine is carefully examined both chemically and microscopically. The dreaded *eclampsia gravidarum* can, however, be recognized only by repeated urinary examination, especially during the last months of pregnancy. Acute Bright's disease should be suspected, and the urine examined in every case showing pallor of the skin and puffy eyelids, whether general prostration of the health is apparent or not. The characteristic symptoms of acute diffuse nephritis are the following: headache, restlessness, muscular twitching, dyspnea, nausea and vomiting, a tense pulse, moderate fever, dropsy, and anemia. Tube-casts and albuminuria are almost constant, except in rare instances of puerperal eclampsia (J. Hirst). It should be borne in mind that slight albuminuria occurring in the course of pregnancy or during any of the acute specific fevers, *without casts*, is not a true nephritis, although a more or less remote consequence of the glandular degeneration of the renal epithelium associated with the febrile albuminuria. In addition to the presence of albumin and hyaline and cell-casts, however, a diminished quantity of sooty-looking urine and the discovery of red and white blood-corpuscles will render the diagnosis positive. The history of the case and the causal factors are also to be taken into consideration.

The diagnosis of the particular sub-variety is sometimes possible. Thus *acute tubular nephritis* commonly results from an intoxication, or more rarely from a severe infection or chilling. The urine is scanty, turbid, and often reddish-brown in appearance, and the sediment is composed largely of renal epithelium and tube-casts. Edema is absent. The effects of the toxic agent on other viscera is shown by the presence of jaundice, myocardial weakness and the like. In *acute glomerular nephritis* the urine is scanty, the amount of albumin large, but few, if any, tube-casts and renal cells are found. General edema is commonly present.

Prognosis.—The *duration* of ordinary exudative or tubal nephritis following exposure to cold and wet varies from a few days to three, four, or six weeks. The albuminuria steadily decreases, and with the casts finally disappears, while the daily quantity of urine increases, as does the excretion of urea. The prognosis depends much upon the primary disease or causative condition, and also upon the intensity and character of the renal inflammation. Scarlatinal nephritis is less likely to be recovered from than nephritis due to exposure to cold after alcoholic excesses. The acute parenchymatous degeneration that accompanies typhoid fever, diphtheria, and other infectious fevers, as well as pregnancy, is usually a mild affection and recovery takes place easily. But in acute yellow atrophy, yellow fever, cholera, and in severe phosphorus- or mercurial poisoning death may occur from the intense and widespread necrosis of renal epithelium. In favorable cases of ordinary exudative nephritis the dropsy and albuminuria gradually diminish, while the color of the skin and the quantity of urine and urea increase, so that in the course of from three to four or six weeks recovery is established. After the disappearance of the dropsy the albumin may persist for some time, and then slowly disappear; but rarely, in unfavorable cases, even when dropsy has disappeared, albuminuria may continue and the affection become a chronic parenchymatous nephritis.



FIG. 62.



FIG. 63.



FIG. 64.

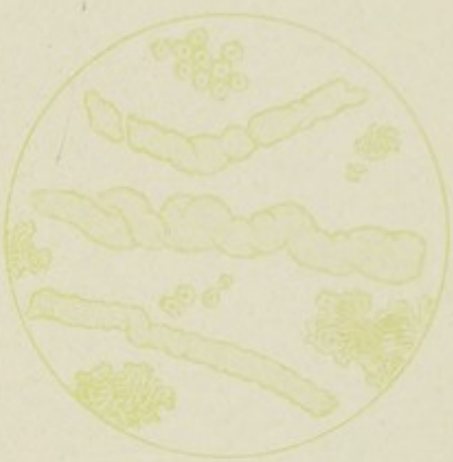


FIG. 65.



FIG. 66.

FIG. 67.

FIG. 62.—A. G., aged fifteen, male, suffering from acute nephritis. Urine showing granular casts (Queen obj. $\frac{1}{6}$; eye-piece ij.).

FIG. 63.—C. A., aged nine, male. Scarlatinal nephritis, third week of convalescence. Urine showing granular casts (Queen obj. $\frac{1}{6}$; eye-piece ij.).

FIG. 64.—J. D., aged fifty-four, male, suffering from cancer of the common duct and head of the pancreas. Urine showing bile-stained casts (Queen obj. $\frac{1}{6}$; eye-piece iv.).

FIG. 65.—B. J., aged twenty-two, female, suffering from puerperal eclampsia. Urine showing large, finely granular casts (Queen obj. $\frac{1}{6}$; eye-piece ij.).

FIG. 66.—S. A., aged fifty-eight, male. Urine showing granular and fatty casts; post-mortem showed chronic parenchymatous nephritis (Queen obj. $\frac{1}{6}$; eye-piece iv.).

FIG. 67.—C. C., aged forty-two, female, suffering from septicopyemia with amyloid kidney. Urine showing epithelial and (so-called) amyloid casts (Queen obj. $\frac{1}{6}$; eye-piece ij.).

[L. Napoleon Boston.]

Serious and often dangerous symptoms of acute nephritis are—severe general edema, dropsical effusions into the serous sacs (as hydrothorax), uremia (especially when beginning with cerebral manifestations, as coma or convulsions), and finally inflammation of the internal organs, as pleuritis, pneumonitis, pericarditis, peritonitis, and meningitis. In the absence of uremia recovery in cases of marked general dropsy is quite common. Suppression of urine, however, lasting more than twenty-four or forty-eight hours, is usually a fatal symptom. The prognosis is unfavorable also in cases in which the nephritis has a productive character. Life may, on the other hand, be prolonged for several years.

Treatment.—I shall not include here the management of the primary affection of which the nephritis may be either a complication or consequence. Bland liquid *foods* only should be allowed in the acute stage, and the patient should be encouraged to drink freely of water (plain or distilled), skimmed milk, or buttermilk; these are especially valuable when hot. If dropsy be present, a salt-free diet should be adopted.

Since the renal function is diminished by the congestion and inflammation, the first object in the treatment is to relieve these conditions and thus restore the excretory function. The single or combined use of diaphoretics and cathartics is practised, therefore, not that the skin and bowels should be made to perform the work normally done by the kidneys, but in order to restore the functional equilibrium by the antiphlogistic effect produced.

Absolute rest in a warm bed and in a warm room is of primary importance. Woollen underwear and blankets should be provided, so as to promote a constant free action of the sweat-glands. These hygienic measures should be carried out both in the mild and in the severer cases.

Local bloodletting, as by leeches or cupping over the loins, I seldom employ; in rare cases, however, when much pain is complained of, it may be useful, although hot fomentations may be more so. Diminution of the edema and the elimination of urea and other urinary constituents that may be retained in acute nephritis are best obtained by exciting a profuse perspiration. The hot-air or hot-water bath and the hot wet-pack may be used to accomplish these results, and in most cases the last-named method suffices. It is easily applied by wringing a blanket out of hot water, wrapping the patient in it, and then with a dry blanket, and finally a rubber-cloth cover, surrounding all. This furnishes a steam-bath in which the patient may remain until copious sweating has lasted an hour or so, according to the condition. Children suffering from scarlatinal nephritis may be treated thus, or quite readily also by immersion in hot water, for twenty, thirty, or forty minutes; the skin should then be lightly dried, and the child wrapped in warm sheets or blankets and warmly covered in bed. Hot vapor or air may be generated alongside the bed, and transferred under the raised or cradled bed-clothes by means of a tin funnel and pipe. The sweating will be aided by the drinking of hot lemonade or soda-water or of water containing spirit of Mindererus. Should the skin fail to respond to these measures, as in uremia, perspiration may be started by a hypodermic injection of pilocarpin (gr. $\frac{1}{8}$ to $\frac{1}{6}$—0.008 to 0.0108), after which it will continue to pour out on the application of heat. The heart and pulse should be watched after the injection of pilocarpin, as serious collapse sometimes attends its use. The sweatings should be repeated

until the dropsy disappears and as often as the patient's strength will permit. The beneficial effects of sweat-baths are not due to the removal of nitrogen through the skin, but rather of sodium chloride. A useful adjunct to the above is the administration of hydragogues, as the saline cathartics, elaterium, and compound jalap powder. Elaterium extract (gr. $\frac{1}{6}$–$\frac{1}{4}$—0.0108–0.0162) is prompt in action, and magnesium or sodium sulphate (℥j—4.0), given in hot concentrated solution every hour, or a calomel purge, may be recommended. It may be necessary to aid in relieving the tension and distress of extreme edema by multiple punctures or by the use of a small trocar and canula, with a drainage-tube (Southey) attached to the latter after the trocar is withdrawn. Aspiration must be performed if either hydro-thorax, hydro-pericardium, or ascites assumes serious proportions. Half-ounce (16.0) doses of the spirit of Mindererus (liq. ammon. acetat.) in water may be added to the diaphoretic treatment; this, combined with aconite, aids in controlling the fever that may be present and in preventing the vaso-constriction that is often premonitory of uremic symptoms.

Uremic convulsions that do not soon yield to prompt diaphoresis and catharsis should be treated by venesection. As much as a pint or two (.5–1 liter) of blood may be withdrawn and life saved thereby. Sometimes chloroform-inhalations are needed to subdue the very violent convulsive seizures, as in eclampsia. Their return may be prevented by rectal injections of potassium bromid and chloral, consisting of 1 dram (4.0) of the former and ½ dram (2.0) of the latter. Contraction of the arteries with increased tension and beginning muscular twitchings calls for the use of nitroglycerin, chloral hydrate, or, possibly, morphin.

Diuretics other than the simple diluent drinks mentioned have very little use in the therapy of acute diffuse nephritis, at least early in the disease. Later, as adjuvants to the diuretic properties of water, potassium bitartrate or acetate, sodium benzoate, and cardiac stimulants, as caffein citrate and the infusion of digitalis, may be given, well diluted.

During convalescence care must be exercised that the patient does not catch cold. The diet must not be increased to solids too suddenly nor too rapidly, and particularly in the matter of meats. Light watery vegetables, fruits, and cereals may be gradually added to the diet-list, although milk should be mainly used. Ferruginous tonics are indicated for the anemia, and Basham's mixture is an admirable preparation at this stage.

A change of locality to a warmer, drier, and more equable climate, and careful habits of dress, diet, and exercise, are necessary in cases of recovery from the very serious forms of nephritis, in which the renal parenchyma is shown to have been somewhat damaged by the persistence of slight albuminuria at intervals.

ACUTE INTERSTITIAL NON-SUPPURATIVE NEPHRITIS.

("*Lymphomatous Nephritis*"—*Wagner.*)

Increasing interest and importance attach to the etiologic and pathologic features, principally, of this affection.

Definition.—"An acute inflammation of the kidneys, character-

ized by cellular and fluid exudation into the interstitial tissue, accompanied by but not dependent upon degeneration of the epithelium; the exudation is not purulent in character, and the lesions may be both diffuse and local" (Councilman).

Pathology.—The kidney is enlarged, pale, and somewhat mottled. The essential lesion consists in an acute proliferation of the cells in the intertubular tissue, with but little change in the parenchyma. The proliferation takes place mainly from the venous and capillary endothelium. The cells are found chiefly in the intermediate zone of the kidney, between the pyramids and the cortex. Howard[1] noted the occurrence of the following: (1) plasma-cells, lymphocytes, polymorphonuclear leukocytes in the exudation; (2) lymphocytes and plasma-cells in the dilated vessels; (3) mytosis and evident ameboid activity of plasma-cells in both blood-vessels and tissues. A special observation was the discovery of large numbers of typical eosinophilic leukocytes in the interstitial exudation and in the blood-vessels.

Etiology.—Most of the cases of acute interstitial nephritis occurred in children suffering from diphtheria and scarlet fever. The first case described, by Biermier in 1860, occurred in a case of scarlatina. Other acute infectious diseases, as typhoid fever, lobar pneumonia, measles, and epidemic cerebro-spinal meningitis, may have acute interstitial nephritis present. The Bacillus coli and pus streptococci have been found in some of the kidneys; and a general streptococcus infection following abortion has been noted in several cases of this form of nephritis. Bacteria may play no part in the etiology of this affection, and the powerful toxins of the mixed infection commonly met with are possibly responsible for the chemotactic cellular proliferation.

Emulsion-albuminuria.—The urine is chylous in appearance, and upon microscopic study it presents a faint haze, caused by minute globules of fat. No volatile liquid results from destructive distillation (acrolein test), but a questionable reaction is obtained with Fehling's solution.

Boiling causes a heavy precipitate; while artificial peptic digestion removes the turbidity.

CHRONIC NEPHRITIS (EXUDATIVE).

(*Chronic Bright's Disease; Chronic Parenchymatous Nephritis; Chronic Diffuse Nephritis with Exudation; Chronic Tubal and Chronic Desquamative Nephritis; Chronic Glomerulo-nephritis; Large White Kidney; Secondary or Fatty and Contracted Kidney.*)

Definition.—A chronic diffuse inflammation of the kidneys, attended with epithelial degeneration, exudation from the blood-vessels, and permanent connective-tissue changes in the stroma. According to Delafield, this is the *chronic productive* (*or diffuse*) *nephritis with exudation*—one of two varieties of chronic Bright's disease.

Pathology.—Although there are several types of pathologic kidney in this disease, the anatomic differences depend upon the causation and duration of the nephritis.

The first type of kidney to be mentioned is the *large white kidney* (without waxy degeneration). It is either enlarged or normal in size,

[1] *Amer. Jour. Med. Sciences*, Feb., 1901.

and pale or yellowish in color. The surface is smooth, and the capsule is easily stripped off. On section the cortex is broader than normally, yellowish-white throughout, or it may present opaque yellowish or whitish areas with mottlings of red. The pyramids are congested in some cases. Microscopically the renal epithelium is swollen, hyaline, granular, or fatty, and more or less disintegrated or flattened; the glomeruli are enlarged from the growth of the capsule-cells and of the cells covering the capillaries, and in some cases, owing to the connective-tissue thickening of the capsule, the tuft of capillaries is found to be atrophied. The interstitial tissue shows some thickening of the arterial walls and a moderate growth of connective tissue in patches around the glomeruli and tubules; the latter contain hyaline and granular casts.

The *small white kidney*, or secondary contracted kidney, in most instances is probably a later stage of the preceding, in which the degeneration of epithelium is more advanced and the growth of connective tissue and resultant cicatricial contraction are prominent features. The surface is slightly granulated, and the capsule is proportionately adherent. While this kidney is usually grayish or yellowish in color (*pale, granular kidney*), there may be some mottling due to red spots. The consistence is firm and the cut-surface shows yellowish-white foci of the fatty degenerated epithelium in the narrowed cortex, "small, granular, fatty kidney." Under the microscope we find extensive degeneration and disintegration of the epithelium of the glomeruli and convoluted tubules, with atrophy of the parenchyma, and a corresponding increase of the interstitial connective tissue. Waxy degeneration may be associated.

Another variety is the *large red or variegated kidney of chronic hemorrhagic nephritis*. The organs are usually enlarged, swollen, red, and congested-looking or mottled, and frequently "bumpy" or slightly bossellated. The capsule is slightly adherent to the depressions between the bosses. Red spots, due to small hemorrhages, may be noticed. The section shows also congested portions and gray or yellow spots corresponding to the anemic and fatty degenerated portions. Small cortical hemorrhagic areas or striations, brownish-red in color, are distinctive of the kidney. The microscopic appearances are those of the large white kidney plus those of acute nephritis. Or, there may be inflammatory edema and cellular infiltration of the intertubular tissue, and dilated tufts of capillaries with surrounding cellular hyperplasia.

Etiology.—The disease may follow either the acute diffuse nephritis, as of scarlet fever or pregnancy, or simple chronic congestion and chronic degeneration of the kidneys. Watson believes that acute nephritis is not a common disease, is usually due to the direct effect of some infection and that the majority recover completely. More often chronic parenchymatous nephritis arises insidiously, in a subacute manner and without any previous acute manifestation. Males are more frequently subjects than females. Children affected with the disease have usually had scarlatinal nephritis. Young adults are more commonly affected, however, with the usual variety, developing subacutely. Drinkers of beer and other malt and alcoholic intoxicants seem to be liable to the disease. It is not improbable that some toxic or infectious agency, acting slowly and persistently, may in the insidious cases be the cause of the nephritis, although manifestations elsewhere may be absent. I have observed it in certain individuals living in malarial regions.

Persons working under exposure to cold and wet, and those living in humid and low, marshy localities are more liable than those who are better protected from climatic vicissitudes. Tuberculosis, syphilis, and chronic suppuration may give rise to the "parenchymatous" form of chronic Bright's disease, and it is usually combined with amyloid disease.

Symptoms.—There may be a persistence, in a lesser degree, of the symptoms of an acute parenchymatous nephritis, particularly the anemia, dropsy, and the albuminuria, until the affection becomes chronic. In most cases, however, the disease develops slowly and gradually, in a *subacute* manner, though the earlier symptoms seldom indicate any renal derangement. There may be simply a general impairment of health and strength, loss of appetite, nausea, and attacks of indigestion, headache, dulness, and perhaps some pallor. Soon there is puffiness of the eyelids or swelling of the feet or ankles, or both, and the complexion takes on a blanched appearance. The *edema* gradually extends up the legs, and is often worse as the day grows, while on rising in the morning it may be found to have disappeared during the night's rest and recumbency. The *quantity of urine* is diminished in the majority of cases, though in the later stages it may be nearly or quite normal, and even slightly increased in long-standing instances or when absorption of the dropsy is taking place. Superadded acute nephritis may cause a very scanty or a suppressed secretion of urine. The specific gravity is, of course, increased in scanty urine, and *vice versâ*. *Albuminuria* is often quite marked. The amount of albumin may be from one-fourth to three-fourths of the volume of the urine, or from 1 to 3 per cent. by weight, so that the daily loss of albumin may be considerable. The nitrogen coefficient, *i. e.*, the relation of the urea to the total nitrogen, is reduced from the normal (80 to 82) to 60. The color of the urine is turbid, sometimes smoky-yellow, and urates, casts, red and white blood-cells, epithelial cells, granular débris, and fatty granular cells are found in the usually abundant sediment. The *tube-casts* are of different varieties, but narrow or broad hyaline, fatty granular (Fig. 66), and epithelial casts are commonly observed.

The *edema* is prominent and persistent. It gradually extends all over the body, so that pitting can be obtained on the limbs, chest, abdomen, and back. The loose subcutaneous tissues, as of the penis, scrotum, and eyelids, are particularly distended. In chronic hemorrhagic nephritis, only, the edema may be absent or very slight. The *pasty, pallid complexion* and *anasarca* are most characteristic of chronic exudative nephritis, especially with large white kidney. The dropsy may be moderate and about stationary for several months; then, despite all treatment, it becomes insidiously worse, death ensuing in a month or two.

Dropsy of the serous sacs, with its attendant distressing symptoms, may be present in serious cases, and edema of the larynx and lungs may come on suddenly and cause death. Dyspnea may be toxic and nervous, as well as mechanical or cardiac in origin. *Cardiac dyspnea*, due to failure of the heart's action, as seen in many cases, is usually worse on lying down. It may be provoked by vaso-constriction, and is then a danger-signal of uremia. Catarrhal bronchitis may be associated with cough and expectoration.

There is *moderate hypertrophy of the left ventricle*, and later dilatation of both ventricles. The *aortic second sound is accentuated* and

the *pulse-tension increased*. The blood-pressure is persistently exalted prior to failure of the left ventricle.

Uremic symptoms are frequently manifested, except the convulsions which are common to chronic nephritis without exudation. Headache, vertigo, sleeplessness, nausea and vomiting, diarrhea, and stupor, coma, or delirium, may develop and precede a fatal termination.

Albuminuric neuro-retinitis, as evidenced by dimness of vision and field-defects, occurs in quite a number of cases. The skin of the legs becomes subject to a red eczematous eruption in some cases of great dropsical distention. In the absence of complicating inflammations, such as pericarditis, endocarditis, pneumonitis, and ulcerative colitis, which are rare, the temperature is practically normal.

The **course** of chronic exudative nephritis may either continue from bad to worse, until death ends all in a year or two, or anemia, dropsy, and albuminuria may attack one who for years previous has had apparent good health, after a first attack the second proving fatal within a few months. Again, some patients, having a little pallor, slightly diminished urine of high specific gravity, with albumin, may complain of nothing for years, until decided attacks, lasting for several months, may occur, with intervals during which the dropsy, dyspnea, etc. may be absent, although some albuminuria persists.

The average *duration* of the disease varies. The duration of chronic hemorrhagic nephritis may be somewhat longer (eight months to two or three years) than that of the large white kidney (six to eighteen months), but it is shorter than the secondary, contracted kidney, which lasts from one and a half to three or even five years.

Diagnosis.—The diagnosis of the disease itself is not difficult, but of the stage or the variety of kidney it is almost impossible to tell correctly in some instances. The urinary examination, coupled with the symptoms of dropsy and anemia, is sufficiently diagnostic of chronic diffuse nephritis (with exudation). The fact that, as shown by Czyhlarz and Donath,[1] methylene-blue is retarded in its elimination from the kidneys in nephritis, may have diagnostic value in some cases.

In cases of *large white kidney* the urine passed is, as a rule, less in quantity and is of higher specific gravity than in the small, pale, and contracted kidney. Edema is usually greater in the former also, while in the latter cardio-vascular changes are more marked. The transition of the disease from the earlier to the later stage may be thus noted. The casts in the latter may be narrower and more darkly granular than in the large white kidney. The existence of *hemorrhagic kidney* may be inferred from the history of alcoholism and the persistent presence of erythrocytes and blood-casts in the urine.

Chronic parenchymatous is distinguished from chronic interstitial nephritis by the following points of difference:

Chronic Parenchymatous Nephritis.	Chronic Interstitial Nephritis.
Occurs in early or middle life.	Occurs later in life.
There is a previous history of an acute attack, of scarlet fever, or perhaps of acute alcoholism.	A previous history of gout, chronic lead-poisoning, syphilis, excessive eating and drinking (spirits), nerve-strain.
The onset is gradual or markedly manifest.	The onset is very slow, insidious, and indefinite.
Dropsy is a constant symptom.	Dropsy is rare.

[1] *Wien. klin. Wochen.*, June 15, 1899.

Chronic Parenchymatous Nephritis.	Chronic Intestinal Nephritis.
Vascular changes and cerebral symptoms are comparatively uncommon.	Arteriosclerosis, cardiac hypertrophy, and cerebral symptoms are common.
Albuminuric retinitis common.	Retinal hemorrhage and choking of disc.
Marked albuminuria; many tube-casts, chiefly short, thick, granular.	Very slight albuminuria and few casts, chiefly hyaline (long, narrow).
Urine but little increased in quantity, often diminished; specific gravity is increased or slightly diminished.	Urine of very low specific gravity and excessive in quantity.
Anemia occurs earlier and is marked.	Anemia slowly progressive and less marked.
Uremic symptoms are generally less severe—amaurosis, vomiting, diarrhea, headache.	Uremic symptoms are generally severe—coma and convulsions, great dyspnea.
Runs a shorter course—from two to six or seven years.	Has a more chronic course—seven to thirty years.

Prognosis.—This is invariably bad as to cure, though life may be prolonged in certain cases. In severe cases death may take place in from three months to a year, either from uremia, dropsy, cardiac dilatation, or complications. Cases of a year's duration almost never recover, and, *a fortiori*, those in which advanced secondary contraction of the kidney may be inferred are incurable, and may soon terminate fatally. Complete recoveries from the disease, particularly in children that have had scarlet fever, may occur but rarely. The prognosis depends greatly on the quantity of urine passed daily, the excretion of urea and total solids, and upon the amount and persistency of the albumin, as well as upon the degree of cardio-vascular and retinal changes. F. Widal[1] reëmphasizes the importance of determining the amount of urea in the blood, more than 0.5 grams per liter of blood-serum implying nitrogen retention, which the organism cannot tolerate.

Treatment.—The indications for treatment are similar to those in acute nephritis. The dropsy and uremia must be treated symptomatically, and the *diet* is of importance. Skimmed milk and buttermilk, or "zoolak," with dried bread, crackers, and zwieback, perhaps, should be depended on as much as possible when dropsy is pronounced. When dropsy is slight, more solid food, meats sparingly and vegetables, rice and other light cereals and fruits, and out-of-door life should be recommended. Until recently not enough proteid food was allowed in chronic nephritis, but milk is still the best article. The reduction or complete absence of salt in the diet has a strong influence in reducing edema (Rovighi). The output of water and salt, however, should be recorded, and the intake of the latter restricted as indicated. A patient with edema should remain in bed. Residence in a warm, dry climate may aid in prolonging life. Woolens should be worn next to the skin, and severe exercise should be forbidden.

The infusion of digitalis, strophanthus, spartein, adonidin, or convallaria may be needed in cardiac weakness, or nitroglycerin for contracted and tense arteries with a tendency to uremic twitchings. Unirritating diuretics, such as Trousseau's diuretic wine and Basham's mixture for the anemia, are useful. Strontium lactate in doses of from 15 to 20 grains (0.972–1.29), three times daily, I have found useful in some cases. Diuretin has also been tried lately with favorable results. Von Hösslin states that sodium bicarbonate without much fluid reduces albuminuria.

[1] *Bull. et mem. Soc. med. d. hôp. de Paris*, 1911, 3 s., xxxii., 627.

CHRONIC NEPHRITIS (NON-EXUDATIVE).

(*Chronic Interstitial Nephritis; Chronic Bright's Disease; Primary, or Genuine, Contracted Kidney; Cirrhotic Kidney; Red Granular Kidney; Chronic Productive (Diffuse) Nephritis without Exudation (Delafield); Gouty Kidney.*)

Definition.—A chronic diffuse inflammation of the kidneys, attended with a growth of connective tissue in the stroma, degeneration and atrophy of the parenchyma, and marked cardio-vascular changes. Two additional varieties are recognized; the arteriosclerotic type and the secondary contracted kidney. The last named was described under chronic parenchymatous nephritis, of which it is a sequel.

Pathology.—In genuine primary contraction of the kidneys there is a reduction in size and weight about equal in both organs. They may be only one-half or one-third the size of normal kidneys, and the two kidneys together may not weigh over two ounces. They are often found imbedded in thick adipose tissue, the capsule being thick, opaque, and very adherent, so that on stripping it off it brings away portions of the renal cortex. The surface of the kidney is red, irregularly granular, or finely nodular, and occasionally small cysts are present. The consistence is firm, dense, and resistant to the knife. The cut surface shows a thin, atrophied cortex, and dark, reddish streaks alternating with pale portions. The pyramids are also diminished, and darker than the cortex. In the gouty kidney the pyramids show fine striations of sodium urate or of uric acid, or crystals representing uric-acid infarctions.

Microscopically, the essential changes are an increased production of connective tissue, especially in the cortical substance, and a more or less proportionate degeneration and atrophy of the renal parenchyma, the destruction of which is due to the circulation of noxious agents, but which is replaced by cicatricial fibrous tissue (Weigert).

The new tissue occurs in irregular masses around the shrunken glomeruli or between the tubules. The distribution of connective tissue in the pyramids is diffuse. In the earlier cases the cells of the tufts and capsules are swollen and multiplied, and a small-cell infiltration is seen around the glomeruli and tubules. Later this infiltration of cells becomes fibrillated and ends in fibrous thickening. Glomerular atrophy is due partly to the changes in the capillary, intracapillary cells, and those around the tufts; partly also to capsular thickening and hyaline or waxy degeneration; and partly to the thickening and occlusion of arterioles.

The tubules show marked changes. Some are included in masses of connective tissue, so that there is compression-atrophy and even total obliteration of the lumen. The intertubular connective tissue constricts the tubules in certain places, so that the lumen is elsewhere increased. This dilatation is especially prominent in the granules seen on the outer surface of the kidney, and, owing to the damming back of urine in some of the tubules thus obstructed, little cysts are visible to the naked eye here and there. The epithelium lining these tubules shows granular, fatty, or waxy degeneration, and may be either flattened, cuboid, or swollen. The tubes may contain granular or fatty débris and tube-casts.

An important change in most cases is the growth of fibrous tissue in the walls of the arteries, causing sclerosis. The arteries and capillaries are thus mostly occluded by the obliterating *endarteritis*. Waxy or

hyaline degeneration is seen also (*vide* Arteriosclerosis). These arterio-capillary changes may be the primary condition, and may represent the renal effects of a general arteriosclerosis or fibrosis forming the arterio-sclerotic type of the disease. Interstitial nephritis is "but one lesion of a generalized process of fibrosis" (Watkins). An almost constant accompaniment of chronic productive nephritis is cardiac hyperthrophy. The degree of the latter depends upon the extent of the renal, and also of the general, arterial sclerosis. The whole heart may become so large that the term *cor bovinum* has been fittingly applied to it. In moderate enlargements the left ventricle only is hypertrophied.

Complicating lesions that may be mentioned are cerebral hemorrhage, cirrhosis of the liver, pulmonary emphysema, chronic endocarditis, chronic endarteritis, pericarditis, bronchitis, and gastric catarrh.

Etiology.—The cause of the slow diffuse degeneration, atrophy, and fibroid contraction of the kidneys is sometimes quite obscure. (*a*) In some cases it would seem to be "only an anticipation of the gradual changes which take place in the organ in extreme old age" (Osler)—the "senile kidney." (*b*) *Heredity* undoubtedly plays a part in the causation of certain cases, even to the third or fourth generation. (*c*) *Age and Sex.*—The disease is more common in males; it is seldom manifested symptomatically until about fifty or sixty years of age, and is therefore an affection of advanced life. (*d*) Individuals having a special tendency to sclerotic degeneration of the arteries, from whatever injurious influence, are more liable to chronic interstitial nephritis, although the prolonged irritation of deleterious (especially chemico-toxic) agents may give rise to the disease in those whose cellular nutrition is usually not defective. Thus, the disease has been attributed to the following causes: alcoholism, uric acid, and lead, giving rise to chronic poisoning. Chronic syphilis and chronic malaria probably are also causative factors. (*e*) Habitual overeating and drinking, owing to the imperfect metabolism of the substances ingested, cause a constant excretion of irritating products by the kidney, and no doubt frequently cause granular atrophy and sclerosis of the organ. The continuous and even moderate use of alcohol for many years, especially of spirituous liquors, is a widespread cause of the disease. It is equally likely that the excessive use of red meats in the diet leads to the production of the uric acid that induces the renal disorder (*uricemia; lithemia*), by deranging the function of the liver (Murchison). (*f*) Allied to the above is gout, which causes chronic Bright's disease—in England perhaps more than in this country, lithemia and nervous dyspepsia being more common there. (*g*) According to Strümpell, severe acute articular rheumatism is sometimes followed by contracted kidney. (*h*) Chronic Bright's disease with renal sclerosis is favored in origin and development by the anxieties, worries, and high nervous tension connected with modern business activity and "social functions," the latter particularly acting their part among elderly ladies. Associated with these are usually over-indulgence in rich foods and wines, and sedentary habits. (*i*) Emerson[1] has presented experimental evidence which shows the influence of repeated disturbance of the circulation as an accessory etiologic factor to the

[1] *Archives of Internal Medicine*, June, 1908.

action of toxic substances. (*j*) The cold, moist climate of New England and the Middle States would seem, according to Purdy, to predispose to contracted kidney. (*k*) A chronic productive nephritis without exudation, though not the true "contracted and red granular" kidney, may be caused by hydronephrosis, chronic pyelitis, and chronic congestion of the kidney, as from heart disease.

Symptoms.—These may be latent for years, while the morbid productive changes in the kidneys are slowly effected. The first symptoms may not appear until late in life, although the kidneys may be in an advanced stage of degeneration. Or some complicating or intercurrent affection may set in, as pneumonia or pericarditis, and cause the development of grave or fatal renal symptoms. More commonly, however, there is an attack of *uremia*, with headache, stupor, or convulsions, dyspnea, nausea and vomiting, and a tense pulse. This attack may be recovered from. Then there is an interval of variable duration, during which the health is more or less impaired, and lassitude, drowsiness, disordered digestion, headache, failing vision, dyspnea, and frequent micturition are complained of. This is followed by another uremic attack, severer than the first, or perhaps fatal; if not fatal, the general health is still more reduced, and confinement to the house or bed is necessary, until the vital forces can no longer compensate for the destruction of the renal parenchyma.

Spasmodic dyspnea (uremic; cardiac) is sometimes the first manifestation of contracted kidney. The gradual onset of periods of uncontrollable drowsiness during the day is often marked. An attack of hemiplegia may also be the first indication of renal disease. Sometimes progressive loss of flesh and strength, with a *dry, harsh, wrinkled skin*, may be from the beginning the only clinical features of the affection until death results from sheer feebleness and emaciation. The complexity and variability of the symptoms make it best to describe them under the heads of the various systems:

Urinary System.—The daily *quantity of urine* is usually increased so much that patients are troubled with a desire to urinate frequently, not only during the day, but two or three times during the night. This complaint may be aggravated by the hyperacidity of the urine and the irritability of the prostate (especially in advanced age) that are so often associated with cases of renal cirrhosis. The urine voided during the twenty-four hours may measure several quarts (2 to 4 liters) in well-marked cases of the disease. Early in the attack, when the incipient degeneration and destruction of the parenchymatous cells is taking place, the quantity of urine may be slightly decreased; but as the "blood-flow to the parts that remain must, *cæteris paribus*, be as great as it would have been to the whole of the organs if they had been intact," excessive pressure is brought to bear within the capillaries by the compensating hypertrophy of the heart, and the secretion of the urine, especially of the watery elements, becomes more active. The *polyuria* may give rise to a suspicion of diabetes. The urine is clear and pale-yellow in color, the *specific gravity* being seldom above 1010 or 1012, and it may be as low as 1002 or 1005. *Albumin* is found only in traces or it may be absent altogether (*glomerular atrophy*), especially in urine voided in the early morning. The urea is diminished, and there is

little or no sediment. A very careful microscopic examination may reveal a *few*, usually *narrow*, *hyaline* or *granular casts*, perhaps some *leukocytes*, and rarely a few *erythrocytes*. In the later stages of the disease or upon the supervention of an uremic exacerbation or of a complicating inflammation the urine may be decreased, the albumin increased, and numerous casts be discovered in a more apparent urinary sediment. Hematuria is rare.

Circulatory System.—The freezing-point of the blood is lowered, due to the retention of products normally eliminated by the kidneys. It is to be recollected that the freezing-point in health is —.56° C., and in nephritis it may be found to be —.58° C. or lower. The *physical signs* of cardiac hypertrophy are present. Symptoms referable to the heart are absent, unless dilatation and feebleness, sudden arterial contraction, cardiac complications, or endocarditis occur. *Inspection* and *palpation* of the *hypertrophied heart* show an apex-beat displaced downward and to the left, and an increased, heaving, and rather circumscribed apical impulse. These signs may be less evident in cases of coexisting emphysema. The left border of the deep cardiac dulness extends outside the nipple-line in the fifth or sixth interspace. The first sound of the heart is loud and may be duplicated. A distinctive auscultatory sign is the *accentuation of the aortic second sound*, indicating increased vascular tension. In quite a majority of the cases I observe, sooner or later, a mitral systolic murmur; it is due to relative insufficiency.

The *pulse* is *increased in tension*, and is hard, incompressible, and persistent, the duration of each pulse-wave being increased (*pulsus tardus*). The radial artery itself—and this is true of most of the palpable arteries—feels hard, thickened, and often tortuous, on account of the arteriosclerosis. The systolic blood-pressure is decidedly high, often exceeding 200 mm. Hg. As soon as compensation of the heart fails, symptoms of breathlessness (especially on exertion), palpitation, and the like, appear, and sometimes in paroxysmal attacks ("cardiac asthma"). The resultant stasis gives rise to transudation into the lungs (bronchorrhea; pulmonary edema), and later to edema of the extremities.

Respiratory System.—Epistaxis may be a serious symptom. Sudden edema of the larynx may also occur, and is always grave. Transudations into the pleural sac (hydrothorax), as well as into the lungs (*vide supra*), may precede death. Dyspnea, which is either cardiac or uremic, is usually worse at night, and a true orthopnea, together with Cheyne-Stokes breathing, may be observed associated with uremic stupor and coma.

Nervous System.—Symptoms referable to the nervous system are very important, since they are usually indicative of grave uremia. Cephalalgia is frequent, and neuralgic pains throughout the body, and insomnia, may be complained of. Later great *drowsiness* is often a premonition of uremic coma. Convulsions may be preceded by muscular twitchings, which should attract attention to the imminent danger of the former. Cerebral apoplexy with hemiplegia may be the first symptom of contracted kidney. It is especially apt to occur in cases of marked hardening and weakening of the arteries. There may be an hemorrhagic pachymeningitis, as well as a hemorrhage into the brain-substance. The hemiplegia may persist until death; or it may disappear in a short time, and be followed by subsequent attacks at in-

tervals ("shifting paralyses"). *Formication*, *numbness*, and pallor of one or more fingers (the "dead finger") I believe, with Dieulafoy, to be sometimes the earliest symptoms of chronic Bright's disease.

Of the **special senses**, *nephritic retinitis* is often the earliest evidence of chronic Bright's disease. The patient may or may not have had slight dimness of vision (mistiness) prior to the ophthalmoscopic examination. The loss of vision affects both eyes, and is usually partial (*amblyopia*). Sudden and complete blindness may come on in grave cases —*uremic amaurosis*—the condition being due to neuro-retinitis. The optic papilla is swollen, and surrounded by retinal hemorrhages or by white dots and streaks ("feather-splashes"). Exophthalmos without thyroid enlargement has occasionally been noted (Barker and Hanes, Gordinier). Tinnitus aurium, deafness, and *vertigo* are not uncommon.

Digestive System.—Anorexia, nausea, and annoying dyspepsia are often complained of. Severe vomiting may usher in an uremic attack. Catarrhal gastritis may exist for some time, the tongue being coated and the breath heavy and urinous. *Uremic diarrhea* may also occur.

The Skin.—Edema is usually absent in renal sclerosis; when it does occur, however (as in the ankles and limbs), it is due to dilatation and failure of the heart. The skin is dry, and minute lustrous scales of urea may be seen around some of the pores. A certain degree of pallor is noticed, and often the skin has a cyanotic tinge. *Pruritus* and troublesome eczema are frequently present, and *muscular cramps*, occurring especially in the calves of the legs and at night, may also be associated. The general nutrition gradually fails, so that in advanced cases the debility and emaciation are extreme.

Uremia may come on at any time during the course of the disease, and may be the first symptomatic manifestation; it may either be sudden and severe in its onset (acute uremia) or mild, insidious, and gradual (chronic uremia). Moderate fever may attend an uremic attack, or the temperature may be normal; in chronic uremia, with prostration, coma, delirium, and feeble pulse, it may be even subnormal.

Among the **complications** that may occur are the following: pneumonia, either lobar or lobular; pleuritis, pericarditis, bronchitis, gastritis, enteritis, peritonitis, meningitis, endocarditis, emphysema, phthisis, acute dermatitis exfoliativa (Duckworth), and hepatic cirrhosis.

Diagnosis.—This depends in great part upon the physical, chemical, and histologic examination of the urine. Both the morning and evening urine should be examined repeatedly for albumin and casts, since one examination—and especially that of the morning urine—may give negative results, owing both to the scarcity of these two pathologic elements and to the fact that one or both may be altogether absent in some instances. The mere discovery of a trace of albumin or of a few casts is not always positive evidence of chronic Bright's disease, as both may exist in other conditions. But the age, habits, and symptoms of the patient must be studied in connection with frequent urinary examinations; and a persistent slight albuminuria, with casts, and the passage daily of large quantities of clear, pale urine of low specific gravity, afford sufficient grounds for making the diagnosis.

Contracted kidney should be suspected in all cases in which, during middle life, either one or more of the following symptoms and signs may be noticed: frequent headache, congestive disorders, repeated epistaxis,

vertigo, dimness of vision, intractable conjunctival irritation (Alleman), impaired strength, dyspneic attacks, gastro-intestinal dyspepsia, noises in the ear, itching of the skin, cramps in the calves, muscular twitchings, growing mental dulness, increasing pulse-tension, and rigidity and tortuosity of the temporal and radial arteries. Sudden coma, convulsions, amaurosis, apoplexy, vomiting, or dyspnea in persons in the middle period of life, with or without a history of polyuria, should create the suspicion of chronic Bright's disease. It will be found in such cases that there has been a diminution in the urinary flow before the attack. Persons of lithemic, gouty, rheumatic, or alcoholic habits, with evidences of cardiac hypertrophy, an accentuated aortic second sound, and a hard pulse are often readily diagnosed as subjects of contracted kidney when an examination of the urine is made.

If the first examination of the patient is made during a sudden uremic or apoplectic attack, catheterization should be done if necessary, and the detection of albuminuria will then clear the diagnosis. To determine accurately the permeability of the kidneys, Schapira[1] recommends the hypodermic administration of phloridzin or indigo carmine, followed by ureter catheterization.

In order to differentiate between primary renal affection with secondary cardiac hyperthrophy and *primary heart disease with a secondary congested kidney occurring late in the case*, the general features, course, symptoms, and signs must be carefully and judiciously balanced. Prominent cardio-vascular changes would indicate an arteriosclerotic kidney. The presence of a diastolic murmur would tend to exclude primary contracted kidney of toxic origin; on the other hand, an albuminuric retinitis would point to a primary renal complaint. The symptoms of ordinary non-inflammatory *senile kidney* may not be unlike those of chronic interstitial nephritis, though not so severe; and yet, from excessive eating and drinking at times, uremic attacks may supervene to cloud the diagnosis.

Prognosis.—The *duration* of chronic interstitial nephritis varies. In uncomplicated cases it may last for five, ten, twenty, or possibly thirty years. Complications or intercurrent affections may, however, shorten the duration very much. The *postmortem* examination may show the characteristic kidneys in one who during life had no symptoms indicating renal disease, and whose death was caused by some intercurrent disease. The gradual destruction of the renal parenchyma and its replacement by scar-tissue cause irreparable damage to the organs. On the other hand, the fact that the process is usually a slow one and its duration long is compatible with the preservation of life for many years, and with comparative comfort, even, in many instances. The prognosis in a given case depends very much upon the general constitutional condition, the cardio-vascular state, and the presence or absence of uremia and inflammatory complications. Cardiac dilatation and insufficiency indicate a not far distant end. Convulsive and apoplectic seizures are often fatal, and hemorrhages, persistent vomiting, and diarrhea, *retinitis nephritica*, coma, and delirium render the prognosis as to further systemic tolerance of the degenerated kidneys exceedingly grave.

Treatment.—An early recognition of the disease and the steadfast practice of careful hygienic measures will prevent, to a considerable degree, the advance of the cirrhotic changes. Noxious substances enter-

[1] *Jour. Amer. Med. Assoc.*, Jan. 15, 1910.

ing into the etiology of the affection must be avoided and removed as far as possible. The formation of uric acid must be reduced by dietetic management, alcoholics must be interdicted, and lead—when the cause of the condition—must be kept from further poisoning the system by a change of occupation. By diminishing these irritants the heart and blood-vessels are also conserved—a point of vital importance.

The **hygienic treatment** must embrace a regulation of all the habits of body and modes of life. The patient must be treated, and not his malady, since that is incurable. A dietary that is suitable for each individual case must be made out; it must be the aim to maintain the nutritive equilibrium of the patient, without producing irritation of the renal epithelium. Vaughan holds that a salt-free diet protects the kidneys by decreasing their labor. Saundby's rule is a good guide: "Eat very sparingly of butcher's meat; avoid malt liquors, spirits, and strong wines." Red meats are probably no more injurious than white in their effects in this disease; although the former should be allowed in smaller quantities than the latter. An exclusive milk diet may be necessary for short periods when gastric irritation is present, but in such a chronic disease undue weakness would result from a restriction to milk alone. The larger the quantity of proteid food consumed, the greater the amount of albumin in the urine and also the greater the accumulation of urea in the circulation. Vegetables, greens, fruits, and light, well-cooked farinaceous articles may also be partaken of, and tea, coffee, and cocoa may be drunk. The use of natural mineral waters aids in the renal circulation and keeps the kidneys flushed. In general a mixed diet will be of advantage; the nitrogenous and carbohydrate elements (sugars and starches) are used in limited amounts, while pure fats and fruits (raw or cooked) are to be recommended. I would add that whole milk, diluted, should make up a considerable portion of the diet, that meats be allowed in small quantity once daily, as a rule, and that we should draw largely upon the vegetable kingdom for aliment. Stout persons and those leading sedentary lives should have less food than those taking exercise, and gastric disorder requires the elimination of all but soft, bland foods, or a liquid diet until digestion is restored. As elsewhere stated, it is impossible to lay down a dietary that would be suitable for all cases, on account of the peculiarities presented by the individual cases. The effect of a given diet is to be noted by a careful observation of the bodily weight and by oft-repeated examination of the urine.[1] Extremes of bodily and mental activity should be avoided, and physical exercise should be moderate, regular, and taken in the open air, provided the latter be warm and dry.

A change of residence to a warm, mild, and dry climate is often of service. The variability and humidity of temperate climates, particularly during winter, aggravate this disease, while a sea voyage or a sojourn at some southern, western, or European resort, where the soil is dry and sandy and the climate equable, may be very beneficial.

The indications for **medicinal treatment** are principally as follows: The bowels should be kept free by the aid of laxatives (*e. g.*, trituratio elaterini, gr. ¼–j—0.0162–0.0648) or laxative alkaline mineral waters. Papoid, peptenzyme, and other digestants, with bitter tonics, are useful in some cases in which a furred tongue and indigestion are troublesome.

[1] *American Medicine*, October 31, 1903, vol. vi., No. 18, pp. 697–699, by the writer.

Acids or alkalies, according to special indications, may also be used simultaneously. An increased vascular tension (vaso-constriction), such as to place a serious strain upon the heart; the other extreme, of a very low tension, that induces dropsy; and complications, usually uremic (convulsions, dyspnea, headache), also call for therapeutic assistance. High tension is to be met by the cautious use of nitroglycerin in gradually ascending doses, beginning with 1 minim (0.066) every three hours. Headache, vertigo, and the so-called renal asthma (dyspnea) are also often relieved by this drug. A too great reduction in the arterial tension is undesirable, being attended with danger of uremia and serous effusions, owing to insufficient urinary excretion.

Low tension, with signs of cardiac dilatation, scanty albuminous urine, and edema, requires heart-tonics and stimulants, in conjunction with purgatives. Digitalis (preferably in infusion) has good effects, especially when combined with strychnin nitrate or with caffein citrate. Calomel and the salines should be given for the dropsy.

Uremic symptoms should be treated as in acute Bright's disease by causing profuse sweating and free catharsis, and in some cases by phlebotomy. Inhalation of amyl nitrite or chloroform, or, what is often a useful and necessary measure, the hypodermic injection of morphin (gr. $\frac{1}{6}$—0.0108), may be tried in convulsions, severe headache, or dyspnea. White and Wilcox[1] have shown that morphin does good in nephritis by diminishing the oxidizing functions of the body metabolism. Le Fevre extols chloral for its more lasting action than chloroform.

Contracted kidney of a probable malarial or syphilitic origin may be benefited somewhat by the use of arsenic and the iodids respectively; but no drugs can possibly restore the destroyed renal parenchyma or transform connective-tissue cells into secreting kidney-cells. Renaut[2] claims to have treated chronic nephritis successfully with a maceration of young pigs' kidneys. The dose recommended for adults is two kidneys per day (each weighing not over 160 gm.). The maceration has an energetic diuretic action and an unmistakable antitoxic property. Spillmann claims a specific effect for the internal secretion of the kidney as obtained from the venous blood of the organ, diuresis being greatly increased.

Certain recent writers (Rose, Ferguson, Wolff) had observed the disappearance of casts and albuminuria after the operation of nephropexy in which a portion of the capsule had been removed. In 1898, Edebohls first proposed the cure of chronic nephritis by operation—decapsulation. He reports 18 cases thus treated, and in each operation (except 2) stripped off about one-half of the capsule. The beneficial and curative effects indicated by an increased flow of urine and the disappearance of dropsy tube-casts and albumin, do not show themselves usually before the tenth day. It is not a helpful operation in advanced cases and its precise value as a therapeutic measure in chronic nephritis has not as yet been determined. While the majority of the cases treated surgically belong in the category of chronic interstitial nephritis, decapsulation is quite as appropriate in suitable cases of the parenchymatous variety.

The Rose-Bradford Kidney.—In 1904 Rose-Bradford described a form of fibrotic kidney, inflammatory in origin, and quite distinct from the contracted kidney of advanced life. The etiology is unknown. Power suggests an underlying blood-carried toxin as the cause. "The disease occurs in

[1] *Internat. Clin.*, vol. ii., 20th Series. [2] *Revue de Méd.*, last indexed, xliv., p. 140.

young subjects and is markedly latent. Polyuria with much albumin, absence of edema, cardiovascular and fundus oculi changes, with a fatal termination from an acute uremia, complete the story" (Rose-Bradford).

PYELITIS.

(*Pyelonephritis; Pyonephritis.*)

Definition.—Inflammation of the pelvis of the kidney. The compound terms above represent inflammation of the kidney-structure as a result of, and combined with, pyelitis.

Pathology.—In the mildest varieties of pyelitis (the catarrhal) the morbid changes consist simply of a reddened, swollen, and turbid mucous membrane, covered with an exudation of viscid muco-pus and desquamated epithelium. Ecchymoses are sometimes seen. The urine in the pelvis of the kidney is also turbid from the admixed pus-corpuscles and pelvic epithelium. In calculous pyelitis purulent inflammation and ulceration prevail, and the kidney structure is also involved by extension (pyelonephritis). Renal abscesses are thus formed, and small dark calculi may be found mingled with the pus in small abscess cavities; or, perhaps, as noted before (*vide* Nephrolithiasis), one large abscess cavity may replace the destroyed renal parenchyma (*pyonephrosis*).

A diphtheritic inflammation, with the formation of a false membrane and sloughing of the pelvis, sometimes follows the severe acute infections. Marked hemorrhagic areas may be seen also. In tuberculous pyelitis there is usually an association of nephritis with areas of tuberculous softening and ulceration, and later pyonephrosis. In very chronic cases the pyelitis may be followed by an infiltration of the kidney structure with cheesy masses that may become the seat of calcification.

Persistent obstruction leading to pyelitis is associated with dilatation of the pelvis from retention of urine or of pus (pyonephrosis). This, in turn, from prolonged pressure, causes marked atrophy of the secreting structure of the kidney. There is also an increase in the interstitial tissue. The so-called *surgical kidney* is found when an acute bilateral pyelitis, following a severe cystitis, has excited an acute suppurative inflammation of the kidney. Acute suppurative or interstitial inflammation of the kidney due to metastatic or miliary abscesses is considered under the heading *Pyemia* (*vide* p. 170).

Etiology.—Pyelitis rarely is primary or independent in origin, as after exposure to cold and wet. The secondary causes of pyelitis are as follows: (1) renal calculi (the most frequent); (2) extension upward of urethritis, cystitis, or ureteritis, particularly when gonorrheal in origin; (3) retention of decomposed urine in the pelvis of the kidney; (4) renal affections, as tubercle, carcinoma, and acute nephritis; (5) specific fevers; (6) foreign bodies, other than stone in the pelvis; (7) irritating diuretics. To point out briefly certain additional facts bearing upon the causation of pyelitis in the order named, it should be mentioned that *calculous pyelitis* may result from the irritation of the constant presence and passage of small stones ("gravel"), or even of uric-acid "sand," as well as from the large dendritic concretions that send offshoots into the calyces. Extensions of inflammation to the pelvis from lower portions of the urinary tract may occur in protracted cases of such affections as gonorrheal urethritis and puerperal

and calculous cystitis. *Obstructive pyelitis* sometimes follows the impaction of renal calculi or of other foreign bodies in the ureter when there is pre-existing inflammation of the tract, or when, as usually happens, there is chemical irritation from the decomposition of the accumulated urine. There may be obstruction in the bladder and urethra, as from enlarged prostatic tumors, stricture, phimosis, and paralysis of the sphincter vesicæ, or as in paraplegia. Under the consideration of tuberculosis and carcinoma of the kidney is included the involvement of the pelvis by these conditions. *Infectious pyelitis* may result from smallpox, diphtheria, typhoid fever, and scarlatina, and it depends upon the irritating effect of certain substances eliminated by the kidneys. It is usually associated with more or less nephritis (pyelonephritis). Parasites, such as the echinococcus (hydatids), distoma, strongylus, and filaria, may give rise to pyelitis. Cantharides, cubebs, copaiba, turpentine, and diabetic urine even, may rarely excite a pyelitis.

Symptoms.—These are frequently overshadowed by those of the primary causative condition: they are varied also for the same reason. The clinical manifestations of a simple catarrhal pyelitis are slight pain and tenderness in the region of the affected kidney or kidneys, mild fever, with a *turbid urine of acid reaction*, showing a few pus-cells, a little mucus, rarely some red blood corpuscles, and a trace of albumin.

In the severer varieties, as in calculous pyelitis, especially when there are attacks of renal colic, the urine frequently shows to the naked eye the presence of *blood* and a marked amount of *pus*, some *mucus*, and at times the transitional *caudate epithelial cells* from the middle layers of the mucosa. In obstructive pyelitis the urine sometimes flows freely and normally for a while, until the developing pain over the inflamed kidney ends in relief by the expulsion of the obstacle and the passage of purulent urine. This *alternation* of normal with pyoid urine is indicative of a unilateral pyelitis. *Ammoniacal urine* is met with in *cysto-pyelitis*. *Albuminuria* is decidedly shown according to the degree of pyuria.

In chronic suppurative pyelitis or pyelonephritis the pyuria is variable both in quantity and constancy. *Intermittent pyuria* may be due to the temporary blocking of the ureter by a stone (*vide* Obstructive Pyelitis). The pus is seldom mixed with epithelium in chronic purulent pyelitis. The associated intermittent fever may be like that of tuberculous pyelitis, and marked prostration, anemia, and emaciation are concomitants. Evidences of amyloid change may be revealed in long-standing, chronic cases.

In severe pyelitis the *pain* is often acute, coursing *down the ureters*. The fever is moderate, and there are present the common symptoms described under Nephrolithiasis (*vide* p. 1016).

The *fever* in purulent pyelitis (pyonephrosis) and pyelonephritis takes on a *hectic* or *typhoid* type. Paroxysms of rigors or chills, followed by a rapid rise in temperature and ending in perspiration, may be observed; or there may be marked prostration and feebleness of circulation, delirium, and stupor. The temperature-curve runs an irregular course, with marked remissions, in pyemic cases.

The term *ammoniemia* has been applied to that complexus of nervous symptoms that is supposed to arise from the decomposition and absorption of urinary substances. These symptoms may be similar to the manifestations of diabetic coma.

Distinct *enlargement* and *fluctuation* of the diseased kidney may be

determined in some cases of pyonephrosis. This may also be intermittent, being detectable while there is obstruction to the flow of pus, and *vice versâ*. According to A. H. Smith, at the menstrual periods pyelitis may be subject to marked exacerbations, simulating renal colic. In chronic pyelitis with atrophy of the kidney the onset of uremia may terminate the case.

Diagnosis.—This embraces the discrimination from other affections, and the possible detection of the variety—etiologically considered—of the pyelitis. It is most important to pay attention to the clinical history of any case with a view to the discovery of the cause; also the urinary condition must be carefully studied. In the very nature of this affection it is often impossible to exclude other affections of the urinary tract, as *nephritis*, *cystitis*, and *urethritis*.

Epithelium from the pelvis of the kidney cannot be distinguished from transitional bladder-cells; but, given the indications of a pyelitis, its calculous cause is at once made clear upon the passage of the characteristic uratic or oxalatic concretions. It may happen that the urine from one kidney is prevented from flowing by the impaction of a stone in the ureter. The urine may now flow clear from the other and vicariously acting kidney until, the stone having given way, it suddenly increases in quantity and changes in character, owing to the return of the morphologic elements of the pyelitis (corpuscles, desquamated epithelium, crystals, and débris).

Catheterization of the ureters and renal pelves, as described and practised by Pawlik and Kelly, is a most certain method of determining in doubtful cases from which side the purulent urine arises. Urine from the diseased kidney freezes at a point higher than does that from the healthy organ.[1] Palpation of the ureters through the lateral and anterior fornix of the vagina will sometimes reveal thickening and tenderness in cystopyelitis, and ureteral distention sometimes may be felt in pyelitis calculosa.

Casts and albumin are usually present when the kidney-structure is involved by extension of the pyelitis, while marked pain in the region of the kidney indicates predominant pyelitis, though it does not exclude the possibility of coexisting nephritis. Marked vesical irritability points to associated cystitis, but in intense pyelitis with much pus and an acid urine vesical tenesmus may also be troublesome. Tuberculous can be discriminated from calculous pyelitis by finding tubercle bacilli in the pus. Tubercle bacilli were found by Flick and Walsh in the urine in 73.3 per cent. of consumptives, though lesions of the kidneys were often wanting. The presence of a fluctuating tumor in the lumbar region is significant enough of pus; but it may be difficult to determine whether it is due to pyonephrosis or perinephric abscess, although pyuria and the previous history of pyelitis, as well as the more circumscribed and less edematous character of the swelling of the former, are important distinguishing points.

Differential Diagnosis.—The *hemorrhagic pyelitis* of Senator, Delafield, and others, described as occurring in milder forms, and particularly in girls of neurotic types, may be distinguished by the intermittent hematuria and the occasional lumbar pain, lasting but a few days or a week, and followed uniformly by recovery.

Difficulty is sometimes experienced in diagnosticating pyelitis when coexistent with cystitis—*pyelo-cystitis*. These affections will not be con-

[1] Tinker, *Johns Hopkins Hospital Bulletin*, June, 1903.

founded, however, when it is recollected that their histories differ. There is pain in one lumbar region in the former, and in the bladder in the latter.

According to Rosenfeld: (1) an alkaline reaction is not found in uncomplicated pyelitis; (2) the limit of albumin in the urine, even with severest cystitis, is 0.1 per cent. (maximum, 0.15). Stress is laid upon the relation of the albumin-contents, which is from two to three times greater with pyelitis than with cystitis.

Prognosis.—Renal complications always make the pyelitis a serious affection. Catarrhal cases recover. Calculous pyelitis tends toward chronicity. Pyelo-nephritis and pyonephrosis are apt to end fatally from exhaustion or uremia. Perforation and the discharge of pus into the peritoneal cavity, pleural sac, intestine, and bronchi even, may precede death. The gravity of all cases of pyelitis depends upon the causes and upon the tendency to consecutive suppuration.

Treatment.—This varies according to the cause: the latter needs to be removed, its effects counteracted, and its return avoided. The treatment of calculous pyelitis is essentially the treatment of nephrolithiasis. Primary inflammation of the lower portions of the urinary tract must be combated; causes of retention of decomposed urine, as an urethral stricture or enlarged prostate, must be diminished; infectious fevers must be judiciously handled and irritating diuretics withheld.

Local measures are of value in all forms of pyelitis. Hot-water bags, fomentations, poultices, and dry cupping are often of great service. Internally, the use of diluents is to be recommended, especially the alkaline mineral waters, flaxseed tea, barley-water, skimmed and buttermilk, and lemonade.

Potassium citrate, uva ursi, pichi, buchu, and pareira brava are sometimes selected for their soothing properties. But, practically, none of the remedies named nor any other drug is of any avail when suppuration is once established. Irrigation by means of Kelly's ureteral catheter may be practised with good results in females. Hypodermoclysis of normal salt-solution may be of sustaining value at critical times in infectious pyelonephritis. In chronic pyelitis salol and the oils of turpentine, sandalwood, juniper, copaiba, urotropin, methylene blue, and erigeron have been used for their stimulating and alterative effects upon the mucous membrane. Surgical intervention is necessary in severe purulent pyelitis, pyelonephritis, and pyonephrosis.

HYDRONEPHROSIS.

Definition.—An obstructive accumulation of urinary fluid in the pelvis and calyces of the kidney; it may cause dilatation, pyelitis, or inflammation and atrophy of the renal structure.

Pathology.—Hydronephrosis is usually unilateral. The pathologic changes consist of a dilation of the pelvis of the kidney, associated with a degree of atrophy of the renal tissue depending upon the degree and persistence of the pressure. The accumulated fluid causes flattening and atrophy of the papillæ, and gradually of the tubules and glomeruli, as the dilatation and distention increase, until in extreme cases

remnants only of the renal structure remain in the walls of the hydronephrotic cyst. The mucous membrane lining the pelvis and calyces first becomes thinned, and later thickened, by the growth of connective tissue, thus forming the dense sac-wall. There is also a growth of connective tissue in the renal parenchyma, medullary and cortical, a chronic nephritis with degeneration and atrophy of the renal cells being set up.

A *nephrydrotic cyst* may be very large, containing as much as several gallons of liquid. Sometimes in medium-sized sacs the external appearance of the walls may be lobulated; the interior, however, usually shows only partial septa projecting from the wall into the cavity of the sac. According to the seat of obstruction one or both ureters may also be dilated. If one kidney is affected, its fellow is often hypertrophied.

The fluid contained in the sac varies in composition, but usually is a clear, thin, yellowish, watery urine. The specific gravity is low, and the reaction is often slightly alkaline. Traces of albumin, urea, uric acid, and salts are found. Turbidity may be present, owing to admixture with pus, blood, or epithelium, but only in instances in which previous inflammatory conditions, as a calculous pyelitis, or subsequent complications of like nature have existed.

Etiology.—Hydronephrosis—or, better, *nephrydrosis*—is in most instances secondarily produced by diseases—congenital or acquired—that cause occlusion of the ureter. Probably from 20 to 35 per cent. of cases are congenital (Roberts). In these cases the causal condition is one of stricture, due to obstruction caused by a defective development or malformation in the urinary passage of one or both sides, usually the latter. Thus, there may be a valve-like formation or a very acute insertion of the ureter into the kidney. The dilation has occasionally become so great in the fetus as to cause considerable mechanical difficulty during labor.

Among adults, women are more often subject to hydronephrosis than men, and especially women who have borne children. The condition may be bilateral, as from a stricture low down and due to gonorrheal urethritis, but more often it is unilateral. The causes of these acquired cases are as follows: (1) Impacted calculi in the ureter or renal pelvis. (2) Disease of the ureteral walls, as inflammatory thickening and cicatricial stenosis from ulcers. (3) Flexion and twisting of the ureter, as from movable kidney. (4) Pressure upon the ureter from without, as by tumors and constricting bands (pelvic adhesions). The gravid and retrodisplaced uterus, uterine and ovarian neoplasms, enlarged and prolapsed spleen, and similar conditions causing compression or traction and obliteration of the lumen of the ureter, are found in this class. (5) Calculus of the lower portion of the ureter. (6) Diseases and tumors of the bladder that involve the ureteral orifices, particularly carcinoma and papilloma, or that cause retention, as prostatic enlargement. (7) Urethral stricture.

Symptoms.—These depend somewhat upon the cause and extent of the hydronephrosis. Marked bilateral disease, when congenital, may render the fetus inviable. The unilateral variety may be overlooked for years, and no symptoms may point to the trouble until a tumor can be made out by inspection and palpation, or until the ureter of the remaining kidney may become obstructed and symptoms of uremia super-

vene. The latter are more apt to come on, and earlier too, in double hydronephrosis.

Locally, the patient may complain of frequent and severe *pains* that shoot about the affected loin and downward toward the thigh. Sensations of weight and a dragging discomfort are common. Anorexia, nausea and vomiting, eructations, and irregularity of bowel-action are associated sometimes. In large hydronephrotic cysts a continuous dull, aching pain only may be felt, or, as is not infrequently the case, the tumor may be absolutely painless. Obstinate constipation may result from compression of the colon, or in moderate enlargements diarrhea may occur from the pressure-irritation.

Usually a swelling is detected in the renal region. It gradually increases in size, and in marked enlargements distinct bulging may be visible in the hypochondriac and lumbar regions. *Palpation* reveals a rounded, firm, yet somewhat elastic and sometimes fluctuating tumor. There may be slight tenderness. Dulness on percussion is found over the mass, except where the colon overlies it, when tympany is elicited; this is a characteristic sign of kidney tumors. Moderate enlargements generally do not descend during inspiration.

The *intermittent* form of hydronephrosis (Landau) is interesting from the variations that occur in the size of the tumors. A *marked diminution is coincident with a more or less sudden increase in the quantity of urine passed;* and, on the other hand, as the tumor gradually enlarges the flow of urine decreases. These cases are in most instances due to movable kidney. *Colicky pains* often usher in the periods of greatest distention preceding the sudden increase in the flow of clear urine. This variety of the affection occurs most frequently in women that have borne children. The *general symptoms* scarcely amount to more than a certain loss of flesh incident to the associated worry and anxiety. The filling of the nephrydrotic cyst, the distention, and the pain and discharge, with subsidence of the tumor, recur with variable frequency. Violent exercise inflicting a sudden jar may precipitate the attacks. The tumor may continue to develop in size for several days after the pain has disappeared. The latter may last from several hours to a day. During the intervals, and after the urine has increased in quantity, gradually or quickly, the patient may feel tolerably comfortable for weeks or months. For obvious reasons the tumor is rather mobile in intermittent hydronephrosis.

The occurrence of chills, fever and sweats, nausea and vomiting, abdominal distention, and rapid pulse usually indicates suppuration and pyonephrosis. The urine will then be cloudy and reveal pus, following both discharge and aspiration. A lowered specific gravity and the presence of albumin will be noted when a chronic nephritis has been set up. The functional kidney test which is most practical is that by the employment of indigocarmin.[1] Increased arterial tension and symptoms of acute febrile or chronic afebrile uremia may be added.

Hydronephrosis paraplegica is a form of the disease in which paraplegia develops as a complication.

The *course* of nephrydrosis is usually chronic, with variations and exacerbations depending upon the cause of the affection.

Diagnosis.—This is obviously very difficult in cases in which the

[1] *Therapeutic Gazette*, February, 1911.

accumulation of liquid is small. Characteristic signs are the gradual development of a tumor in either flank, as described above, with diminution in the urinary flow, followed by a more or less sudden free discharge and the subsidence of the tumor, with recurrences (as in the intermittent variety). When these do not occur and the tumor continuously enlarges, aspiration may be practised to determine whether the mass is solid or liquid; the nature of the latter may also thus be ascertained, whether urinary or not. Ureteral catheterization is of great value as a diagnostic criterion.

Differential Diagnosis.—The nephrydrotic sac must be distinguished by exclusion from an *ovarian cyst*, *cystic kidney*, and *tumors of the spleen*, *liver*, and *gall-bladder*. Very large cysts may be mistaken for *ascites*. Assurance of the presence of the colon over the tumor is diagnostic, and a chemical examination of the fluid obtained by the use of the exploring needle will suffice in most cases. It should be remembered, however, that a slight amount of urea is sometimes found in ovarian cystic fluid. The presence of pus-cells in abundance in the aspirated fluid, with symptoms of suppuration, is significant of pyonephrosis. Segregation and catheterization of the ureters may elicit decisive evidence during the existence of the tumor.

Prognosis.—This is generally unfavorable, though in unilateral hydronephrosis evidences of compensation on the part of the unaffected kidney should render the case guardedly favorable, particularly if the cause be a movable kidney. The bilateral affection is always grave, owing to the danger of uremia. Infection of the cyst with pus-organisms is usually a fatal complication. Recovery may ensue in rare instances in which a spontaneous discharge of the liquid takes place. Rupture of the sac is unlikely.

Treatment.—The removal of the cause is seldom feasible. Symptomatic treatment only is required in mild cases, though sometimes gentle massage over the sac, properly directed and cautiously applied (to avoid rupture), may cause a reduction in the size of the tumor. Most often surgical measures only are of use. These embrace puncture and aspiration, incision (nephrotomy) and drainage, nephrorrhaphy, nephrectomy, and the formation of a renal fistula. These procedures, however, are undertaken only when successive reaccumulations of the fluid follow those measures first mentioned.

PERINEPHRIC ABSCESS.

(*Perinephritis.*)

Definition.—Suppurative inflammation of the connective tissue surrounding the kidney.

Pathology.—The suppuration attacks the lax adipose tissue or the fatty capsule in which the kidney is imbedded and the adjacent retroperitoneal tissue. The starting-point of suppuration is usually behind the kidney. There may be several small abscesses at first, but more often a single large abscess is found. The walls may be soft and shreddy, or in more chronic cases thickened and fibrous. A bulging externally over the affected lumbar region is not infrequent, particularly

in large and extensive accumulations of pus. The latter has a tendency at a given point to burrow into the surrounding tissues, and especially downward toward the iliac fossa, pointing in the groin near Poupart's ligament. It may extend backward and open upon the skin-surface. Sometimes the pus perforates the diaphragm and discharges through the pleural cavity and lungs, or the colon, vagina, bladder, or peritoneum may be perforated. The pus is occasionally quite offensive, and may be ichorous from an admixture of infiltrated urine. Perirenal abscess due to calculous pyonephrosis may contain calculi that have ulcerated through pelvic or renal walls. Thickening of the adjacent peritoneum is often found. In certain cases of perinephritis, which usually gave no symptoms during life, the *postmortem* examination has revealed fibrous adhesions and a firm and thickened and fatty capsule, stripped with difficulty from the true capsule of the kidney.

Etiology.—Perirenal abscesses, when not traumatic in origin, develop most frequently as a result of purulent pyelo-nephritis or pyonephrosis. Hence they are *usually secondary.* Other primary conditions that may cause perirenal suppuration are the following: extension of inflammation from the ureter or pelvis of the kidney, pelvic abscess, appendiceal or hepatic abscesses, spinal caries (psoas abscess), and empyema. Sometimes tuberculous processes in the kidney and suppurating new growths, as carcinoma and cysts (including the echinococcus), are complicated by perirenal abscess. More rarely such severe infectious diseases as typhus fever, small-pox, and pyemia lead to purulent perinephritis. Finally, there are cases for which no cause is discoverable.

Symptoms.—Subjectively, there is noted a *dull, throbbing pain* over the affected region that is increased by motion; sometimes, when the abscess is large and presses on the large nerve-trunks, the pains may become shooting in character and be felt in the leg on the same side. *Numbness* may also be felt. Pain and tenderness on palpation are common. The patient is prostrated, weak, and often quite emaciated, and flexure of the thigh on the affected side is frequent. The characteristic fever of suppuration is present in the deeply remitting or intermitting type, with alternating chills and debilitating sweats. Pus is found in the urine only when the kidney is involved. Sooner or later evidences of a *tumor* are seen; the areas can be palpated, and a gradual bulging in the lumbar area, increasing slowly, with smoothness and glistening of the skin and pitting (edema), may be observed. *Fluctuation* is frequently apparent in advanced cases, and in favorable cases signs of "pointing" appear.

Diagnosis.—Should the abscess tend to burrow downward, the condition may be somewhat obscure on account of the absence of distinct local symptoms. Indeed, involvement of the psoas may give rise to symptoms of coxitis, as pain referred to the knee-joint. The diagnosis is usually easy, and when in doubt as to whether the tumor is an abscess or an hydronephrosis or solid mass, the exploring needle should be used.

Differential Diagnosis.—An important point in differentiating perinephric abscess from suppurative pyelitis or pyelo-nephritis alone is the fact that in the latter the quantity of urine is usually diminished, whilst in the former there is less apt to be any interference with the

renal secretion. Again, whilst in the latter the urine usually contains blood and pus, in the former the urine is free from blood, though not necessarily from pus, and casts are apt to be absent here.

Prognosis.—This is guardedly favorable if the abscess points externally in the lumbar area. Of course rupture into the peritoneal cavity, bladder, bowel, and groin is always a serious occurrence.

The **treatment** is surgical, consisting in free incision and drainage.

CYSTIC KIDNEY.

(*Renal Cyst.*)

Pathology.—Congenital cystic kidneys are in reality collections of cysts, varying in size from a pea to a marble, and separated from each other by septa of compressed renal or fibrous tissue. Either one, or frequently both, kidneys may be affected with what is sometimes termed *congenital cystic degeneration of the kidneys.* There is considerable enlargement of the organs, and during intra-uterine life they may attain an enormous size. In mild cases the affection may be tolerated for some years after birth. The cystic fluid may be either clear, turbid, reddish-yellow, or dark-brown in color, acid in reaction, and holds in solution urinary salts, blood, cholesterin, and sometimes uric acid and urea. A single layer of flattened epithelial cells lines the cyst-walls. The cysts themselves seem to be dilatations of the renal tubules and of Bowman's capsules, due, in some instances, to an obliteration of the tubules of the papillæ or to stenosis of some portion of the urinary tract.

The cystic kidneys usually met with in adult life (acquired) are of several varieties: (1) One or perhaps a few cysts may be present, larger usually than those in the congenital cystic kidney, which seem to cause no interference with the normal renal functions. Sometimes a reddish-brown colloid material is contained in these cysts.

(2) Small and often quite minute cysts frequently accompany the chronic nephritic kidney that is small, contracted, and cirrhotic. These result from dilated tubules and capsules when the former are narrowed by the hyperplasia of fibrous tissue.

(3) Cystic kidneys in adults may have the pathologic characteristics of the congenital variety—a mere aggregation of cysts containing clear or colored serum or a cloudy, dark, thick, colloid liquid. This condition is sometimes associated with similar cystic disease of the liver and spleen. It may be a late manifestation of mild congenital defects. The kidneys have been found converted into cysts in cases in which the presence of calculi (uric acid) in the tubules has probably started the cystic degeneration.

(4) Solitary cystic adenoma rarely occurs. It is in the form of a globular tumor projecting from the surface (usually the anterior) of the kidney. It may be as large as an orange, and may be enclosed in a distinct capsule. On section the mass is found to be composed of various-sized cysts separated by septa of fibrous tissue lined with cuboid or columnar epithelium. The remainder of the kidney appears to be quite healthy.

Symptoms.—These may be absent in adults until the sudden development of uremia. Ordinarily, the clinical picture is similar to that of chronic interstitial nephritis. There is an increase in the quantity of urine, which is of low specific gravity; the normal solids are diminished in quantity: and aceto-soluble albumin may be present (Clifford Mitchell).[1] Slight albuminuria may be present. On *palpation* a large, rounded, and *sponge-like* mass may be felt in either hypochondrium or on both sides. Cardiac hypertrophy and increased arterial tension, as in chronic cirrhosis, are also frequently met with in cystic degeneration of the kidneys. Parker[2] reports a case which was followed by exfoliative dermatitis. Cystic disease of the liver may be associated.

The **diagnosis** can only be made upon the presence of the above symptoms and the discovery of the clear physical signs of the tumor. It should be pointed out that a possible complication of perinephric abscess, due to rupture of one or more of the cysts (as has occurred—Osler), would of course render a diagnosis wellnigh impossible.

Prognosis.—Bilateral cystic disease of the kidney must eventually prove fatal, due to uremia or cardiac failure. Solitary cysts give a tolerably favorable outlook under proper surgical interference.

Treatment.—The unilocular cysts just referred to above may be removed, capsule and all, and the kidney sutured. Bilateral disease cannot be operated upon for obvious reasons; unilateral cystic degeneration may be treated by nephrectomy, with narrow chances of success.

NEW GROWTHS OF THE KIDNEY.

THE most common tumors of the kidney are those belonging to the class of adenomata (benign) and those that are either sarcomatous or carcinomatous (malignant).

Adenomata may be congenital or acquired. They grow in the cortex of the kidney in the form of small nodular masses, which in some cases may increase to a considerable size before any symptoms are produced. A cystic growth may be combined with adenoma (*cystic adenoma*), and *lymphadenoma* is also occasionally seen as a secondary growth. Other benign tumors that may affect the kidney are *angioma*, *fibroma*, and *lipoma*. Very large vascular adenomata may become malignant. Grawitz, Lubarsch, Kelly, and others have described a variety of tumor (*hypernephroma*) derived from aberrant adrenal tissue misplaced in the kidney.

Symptoms.—The important points in the diagnosis of hypernephroma are hematuria at long intervals, pain and tumor, the latter giving rise to pressure symptoms. *X*-ray plates are of value in differentiating the hematuria of stone (Moffitt).

Sarcoma and **carcinoma** may be either primary or secondary. *Sarcoma* is frequently congenital in origin, and may have an admixture of striped muscular tissue. The presence of the latter in the kidney points to developmental disturbances during embryonic life as the cause of a variety of tumor known as *rhabdomyoma*. Alveolar sarcoma is also met with. Renal sarcoma is not uncommon in children.

Renal carcinoma is probably of less frequent occurrence than sar-

[1] *Phila. Med. Jour.*, Aug. 19, 1899. [2] *Amer. Jour. Med. Sci.*, Sept., 1899.

coma; it may, however, be found in children as well as in aged persons, the two extremes of life. Carcinoma of the kidney is usually of the soft medullary or encephaloid variety. As a primary affection it probably originates in the renal tubules. Secondary carcinoma of the kidney, although probably more frequent than the primary form, is seldom of clinical importance. Renal carcinoma may occur as a diffuse infiltration or in nodular masses, one kidney usually being affected in primary carcinoma. The tumor sometimes reaches an enormous size, and instances are recorded in which nearly the whole abdomen has been filled, and in which the growth weighed as much as 31 lbs. (14 kgms., Roberts). Rhabdomyomata do not, as a rule, attain a very large size, though sarcomata may grow quite large. Softening and hemorrhage within these malignant growths may occur. The pelvis of the kidney may be invaded, and metastatic areas may form in the liver or the lungs, though this occurs in the case of primary renal carcinoma less readily than from carcinoma in other organs. The renal parenchyma is either partially or wholly destroyed, the pyramids being attacked later than the cortex.

Symptoms.—*Lumbar pain* on the affected side is often an early symptom, and may persist throughout the course of the disease. It may be paroxysmal, and be felt extending down the thigh, or it may be dull, dragging, and limited in character. Pain is not, however, a constant symptom in a certain proportion of the cases.

Hematuria may occur early or late, and often appears before any tumor is palpable. The blood may be in a fluid state or in clots, the latter not seldom taking the form of pelvic or urethral casts, the passage of which may give rise to colicky pains. Casts of the ureter sometimes resemble lumbricoid worms. The hemorrhage may be excessive and cause marked weakness and a symptomatic anemia, superadded to the cancerous anemia that is usually present; on the other hand, it may be so slight as to be discoverable only microscopically. It recurs at irregular intervals of days or weeks. Large clots may accumulate in the bladder and cause vesical irritability. The urine from the healthy kidney may be quite normal, and may be secured for observation by ureteral catheterization. *Anorexia, nausea and vomiting, progressive loss of flesh* and *strength*, increasing pallor, and the concomitant symptoms of the cancerous cachexia are seen to develop.

Physical Signs.—These may not be sufficient to reveal the presence of the tumor for some time after the above symptoms have been observed. The appearance of a palpable tumor in either flank is a definite aid to diagnosis. It is felt between the ribs and pelvis latero-anteriorly, and at first, when small and on the right side, it may be movable. Both sarcoma and carcinoma of the kidney may assume enormous sizes. The tumor feels dense and hard (except rapidly-growing tumors, as encephaloid), either smooth or lobulated, and, when not too large, may retain the natural position and form of the kidney. The growth extends downward and inward, and in the very large malignant renal tumors of childhood the abdomen shows considerable enlargement, along with an abnormal pulsation and a prominence of the veins. Usually the tumor does not move with respiration. Percussion gives dulness over the mass, although in small and moderately large tumors the overlying colon may cause a tympanitic note to be heard.

Diagnosis.—The presence of a tumor, when not too large and distinctly occupying the lumbar and lower laterel abdominal region, together with hematuria, pain of a local nature, and progressive failure of nutrition, may be looked upon as diagnostic of a malignant type of renal tumor. The relation of the colon to the tumor and immovability of the latter during respiration are also diagnostic.

Differential Diagnosis.—This is a very difficult subject. Affections such as hydronephrosis, paranephritic cyst,[1] pyonephrosis, cystic kidney, hydatids, ovarian, splenic, and hepatic tumors and (particularly in children) retroperitoneal sarcoma must be differentiated from renal growths. Careful bimanual palpation will aid in the diagnosis, but the exclusion of other lumbar enlargements must be made by close attention to the history and to the development and course of the symptoms. Hematuria alone, in aged persons, is suggestive of carcinoma when no tangible cause for the presence of the blood is at hand. Hepatic and splenic tumors are usually movable during deep breathing, whilst renal tumors are not so. In cases of hepatic growths also the area of dulness extends higher, whilst in renal growths on the right side a tympanitic area generally lies between the liver and the tumor. The characteristic notch and edge of the spleen, and the absence of the overlying colon-tympany, are points that distinguish splenic enlargements from those of the left kidney. Pelvic growths (ovarian and uterine) enlarge from below upward, and are readily detected by vaginal examination. In children Löbstein's cancer (retroperitoneal sarcoma), if very large, is easily mistaken for a renal tumor, except that it is usually more centrally situated and more firmly fixed.

Prognosis and Treatment.—The termination in cases of renal carcinoma is inevitably fatal, and children succumb more quickly than adults. The disease may last from a few months to sometimes a year or two.

If the kidney be removed while the growth is still small, the prognosis is fairly good; but if large or if metastatic tumors have formed, the prognosis is always bad. Bloch warmly advocates in some cases the removal of small sections of kidney-substance, to avert the necessity of a nephrectomy by proving the non-malignancy of the growth. The *treatment*, aside from early surgical measures, is entirely symptomatic and supportive, and obviously it is unsuccessful. Renal colic, excessive hematuria, and a gradually lowered vitality may be met by the use of palliatives, tonics, and by nutritious and easily digestible diet. Nuclein may be tried hypodermically or by the mouth.

II. DISEASES OF THE BLADDER.

CYSTITIS.

Definition.—Inflammation of the mucous membrane of the bladder. It may be either *acute* or *chronic*, the latter being clinically the much more frequent condition.

[1] *Jour. Amer. Med. Assoc.*, June 27, 1903, p. 1775.

ACUTE CYSTITIS.

Pathology.—Cystoscopic examination performed according to Pawlik's or Kelly's method, hereafter to be described, reveals an intensely hyperemic condition of the vesical mucosa, which is puffy, edematous, and of a bright-red color; this may be more intense at points, especially in the vicinity of the trigone. The membrane is bathed in a thick, tenacious muco-pus, and here and there may be noted denuded areas, and the exfoliated epithelium often hanging in shreds from the bladder-wall. Hemorrhagic effusions may be observed. In the severer grades of the disease the intense general hyperemia causes a disappearance of the blood-vessels that are to be seen in the normal condition. Occasionally small patches of ulceration, due to abscess formation (*phlegmonous cystitis*), may be observed, and in rare and fatal instances the entire bladder-wall is involved in a necrotic process.

Etiology.—Cases of acute cystitis may be grouped according to their origin into four main classes, as follows:

(1) **Catarrhal.**—Like other mucosæ, the vesical epithelium is very responsive to systemic circulatory disturbances. Thus, sudden exposure to extremes of cold or heat or violent atmospheric changes, thereby abruptly suppressing the action of the skin, may be potent influences in the etiology of the disease. An intense acute catarrhal inflammation may follow retention of the urine in the bladder, with or without its subsequent decomposition; it may also be the result of pressure from an enlarged prostate or other tumor, and may follow cystocele, urethral stricture, or paresis of the bladder-wall. In overdistention of the bladder, with the accumulation of a gallon (4 liters) or more of urine, the so-called acute *exfoliative cystitis* may result, in which the entire mucous membrane of the bladder may be shed, and the symptoms of grave uremic intoxication supervene. The prolonged retention of urine is followed by decomposition of the fluid, and this, by its irritant action, always excites a cystitis that soon assumes the chronic type.

(2) **Septic.**—This may result either from the direct introduction of pus-producing germs into the bladder or from the systemic transmission of these micro-organisms to the organ. This is known as the *bacterial origin* of cystitis. Under the first class may be mentioned the passage of an unclean catheter or sound; this is a cause of cystitis in puerperal women, and in men who are the subjects of minor grades of urethral stricture, and who have been subjected to gradual dilatation by means of bougies. *Gonorrheal cystitis* is also to be included under this heading. There is a condition known as *febrile cystitis*, which constitutes the second class of septic cases. This comprises the vesical inflammation that is present in the various febrile conditions, and which is probably a direct result of the presence in the urine of the causal bacilli or their toxins (Fitz). Thus, in all of the infectious diseases and fevers (typhoid and the other exanthemata, rheumatism, diphtheria, tuberculosis) there is noted a cystitis of varying degrees of severity that can be accounted for only by the local irritant action of the specific germ of the associated disease, or its eliminating toxins. The so-called *gouty cystitis*, which occurs in lithemic individuals, and which is due to the irritating, concentrated urine, may also be here included.

(3) **Toxic.**—Certain drugs when introduced into the system manifest

an irritant action upon the vesical mucosa, and promptly excite a severe grade of acute cystitis. Prominent among these may be mentioned cantharides and other irritants of the urinary tract—cubebs, copaiba, and sinapis. Workers in coal-tar dye-stuffs are sometimes affected with acute cystitis.

(4) **Traumatic.**—Traumatic inflammation of the bladder follows the improper and careless use of the catheter, sound, or other instrument; the presence in the bladder of calculi or other foreign bodies; and the pressure of the fetus in parturition, or of large masses of impacted feces.

(5) **From Adjacent Inflammation.**—Irritation with consecutive inflammation may result from the extension of an inflammatory process from surrounding structures either by continuity or contiguity of tissue. Thus, a cystitis may follow a urethritis—gonorrheal or otherwise; it may result from an extension downward of a ureteritis, or it may be consequent upon a vaginitis, a malignant neoplasm of an adjacent viscus, a salpingitis, pelvic peritonitis, or pelvic abscess in the immediate vicinity of the bladder, as in the vesico-uterine pouch.

Symptoms.—The symptoms of acute cystitis are very marked. *Pain*, *vesical irritability*, *vesical* and *rectal tenesmus*, *frequency of micturition*, *fever*, and *urinary changes* are all pronounced. Prominent among these is *pain*, which may be most intense and is the earliest and most persistent manifestation of the disease. Its seat is the suprapubic region, whence it may radiate to the sacral region, the perineum, the end of the penis, or the upper portion of the thighs; it is most constant, but is worst just before micturition, by which it may be alleviated. It is considerably relieved by the recumbent posture, and is aggravated by pressure over the bladder.

With the pain, and probably ranking second in severity, is the *rectal* and *vesical tenesmus*, or *strangury*. There is an almost constant desire to urinate. The *urine* may be opaque or highly colored. It is often bloody (in very acute cases the vesical contents may consist of a small quantity of pure blood only), is of a specific gravity varying from 1005 to 1030 (in the febrile cases), and contains pus-corpuscles, mucous flakes, shreds of disintegrated and exfoliated epithelium (bladder), and micro-organisms. Thomas R. Brown,[1] in a bacteriological study of 26 cases, found the exciting causes as follows: bacillus coli communis, 57.7 per cent.; staphylococcus pyogenes albus, 19.2 per cent.; staphylococcus pyogenes aureus, 7.7 per cent.; and B. pyocyaneus, B. typhosus, and B. proteus vulgaris (Haiser), each 3.8 per cent.

Gonorrheal infection may invade the vesicle mucosæ when mixed or pure cultures of this organism are recoverable from the urine; fungous mycelial threads and yeast-cells have even been found in certain cases (*mycotic cystitis*). The urine is commonly acid in reaction, though Brown found it alkaline where the excitant was the B. proteus vulgaris. It may become less acid or alkaline should the condition become modified. More or less albumin will be noted, and on standing a dense sediment forms in the bottom of the flask, which is composed of all the foregoing substances, as shown by chemical and microscopic examination. The total quantity of urine voided in the twenty-four hours may be normal

[1] *Johns Hopkins Hospital Bulletin*, January, 1901, p. 4.

in amount or even slightly in excess of the normal. On the other hand, if exfoliation of the mucous membrane takes place, there may occur partial or even total suppression of the urine.

Fever, with or without an initial rigor, persists throughout the attack, but is not of a severe type, save in the septic and malignant (diphtheritic) forms of the disease, when it may reach 103°–105° F. (39.4°–40.5° C.).

Abscesses may form, and betray themselves by localized pain, tenderness, and, in some cases, by a circumscribed induration requiring surgical treatment.

In the variety associated with extreme exfoliation of the vesical mucosa *grave uremic manifestations* follow. These include all the features of the typhoid state (dry, brown tongue, mild delirium, nervous and muscular twitching; headache; gastric disturbances; and coma). There is also some degree of malaise and anorexia.

It must not be forgotten that acute cystitis may represent an acute exacerbation in the chronic form, and at times may assume a severe type of the disease.

Diagnosis.—Cystitis should be readily recognized from the history of the case and the frequency of the two almost pathognomonic symptoms—suprapubic pain and vesical tenesmus. An examination of the urine will reveal the characteristic clinical features. The percentage of albumin is usually much larger in nephritis than in irritability of the bladder. The differentiation between cystitis and vesical irritability will be noted under the latter condition. Urethritis may be excluded by means of the two-glass test. For example, if urination into two glasses reveals pus in both, after carefully washing out the urethra as far as the compressor urethræ muscle, it is "very positive proof that cystitis or some inflammation further up the canal is present" (Greene and Brooks).

The **prognosis** of the milder grades of cystitis is good; the septic and malignant cases offer a much graver outlook. Extension of the process upward toward the kidneys is always serious.

Treatment.—The treatment of acute cystitis includes prophylactic, hygienic, and medicinal measures.

Prophylactic.—Most important is the prevention of the disease, and this includes, in addition to the usual care of the body, the observance of thorough asepsis.

Hygienic.—The cause of the disease, if evident (calculus, external pressure), should be sought and removed. The patient should at once be placed absolutely at rest in the recumbent posture. The *diet* must be regulated, and all irritating, highly seasoned articles of food must be interdicted. Alcohol in any form is prohibited. An absolute milk diet will be most beneficial. The patient should be instructed to drink freely of water and other diluent drinks. The free action of the skin may be secured by friction and warm bathing.

Medicinal.—The drugs to be employed are the saline laxatives and the various mild diuretics and urinary alterants. The reaction of the urine will indicate the variety of alterant to be employed. If it be acid, alkaline waters are serviceable, as the soda-preparations, Vichy, or the potassium salts. In alkaline conditions of the urine probably the most valuable drugs are benzoic and boracic acid and salol. Benzoic acid is

best administered in the form of ammonium benzoate, which may be given in 10-grain (0.648) doses thrice daily in the compound infusion of buchu, or in uva ursi. Hot applications and hot local bathing (sitz-baths) will do much to relieve the pain and tenesmus; if these be severe, a rectal suppository of opium and belladonna or an enema of chloral hydrate will generally give prompt relief. Tincture of cannabis indica, administered internally, may answer if opium be contraindicated. Under such a course as the preceding a cure may be expected within eight or ten days. It is prudent to advise the patients to wear flannel or silk binders over the abdomen, to avoid chilling of the surface and subsequent acute attacks.

CHRONIC CYSTITIS.

Pathology.—The vesical mucosa is not so hyperemic as in the acute variety, but is of a peculiar muddy or grayish-blue (slate) color, dotted here and there with patches of erosion or of actual ulceration. Slight hemorrhages may and do occur. Owing to the slow course of the disease there follows an immense thickening of the bladder-wall from hyperplasia, conjoined with more or less edema, of the tissues. The result is a contraction of the wall with a proportionate diminution in the vesical capacity. The mucosa may become polypoid in spots, and there rarely follows obstruction of the ureteral orifices, with consequent dilatation of the ureters and renal pelves from a damming back of the secretion. In the majority of cases, however, the changes will be found on cystoscopic examination to be limited to the lower portion of the bladder. The urinary changes are about as in the acute form, save that the reaction is alkaline and the amount of mucus and pus is proportionately greater.

Etiology.—Chronic inflammation of the bladder may be the result of a neglected or oft-repeated acute attack. It may occur from the persistent action of an exciting cause, as the presence of some irritating substance (calculus) in the bladder, or of some excitant external to that viscus, as a localized inflammation or a displaced uterus. The tuberculous variety and that due to neoplasmata are insidious in development.

The **symptoms** and **diagnosis** differ but slightly from those of acute cystitis, although the pain and tenesmus are less intense. Oppositely, the *amount of albumin* in the urine is comparatively large. The same remark applies to the *quantity of mucus* and *pus* (*vide* Pathology); indeed, the last-named ingredient often forms a thick gelatinous mass in the standing urine that tends to adhere to the receptacle. According to Brown's researches bacterial flora contribute liberally toward chronic cystitis: B. coli communis was present in the urine in 55.2 per cent. (50 per cent. in pure culture, and once combined with B. tuberculosis); staphylococcus pyogenes aureus, 10.3 per cent.; staphylococcus albus, 6.9 per cent.; B. proteus vulgaris, 3.4 per cent. The reaction of the urine is often neutral or alkaline where infection is due to the three last-named organisms. An alkaline reaction exists in 80 to 90 per cent. of cases. The cystoscope is an invaluable aid to the recognition of chronic cystitis. Chronic cystitis is accompanied by debility and emaciation, which, however, are of slow development.

The **prognosis** is always serious, and the course of the disease is at the best protracted.

Treatment.—Very generally, the treatment set down for the acute disease will not answer in the chronic form. Undoubtedly, there will follow more or less amelioration of the symptoms, but the tendency is toward a prolonged chronicity. In such cases, after the removal of the ascertainable causes so far as practicable, we are compelled to resort to local treatment of the bladder. This includes—(1) Vesical irrigation; (2) Topical applications; (3) Permanent drainage of the bladder.

Vesical irrigation is secured by means of an aseptic soft-rubber catheter which is connected with a graduated glass funnel: a siphonage is produced by the alternate elevation and depression of the funnel, which contains the irrigating fluid. The latter may consist of plain sterilized (boiled) water, sterile normal salt-solution (40–60 gr. to the pint—2.59–4.0 per ½ liter), or a weak solution of mercuric chlorid (1: 50,000–100,000). The irrigation should be done slowly, and not more than twice or thrice daily in severe cases, and much less frequently in ordinary cases, according to the exigencies of the condition.

Vesical medication may be secured by means of the funnel after irrigation, the medicating substances being dissolved in a pint of water and allowed to flow slowly in and out of the bladder. The drugs that may be used in this manner are silver nitrate or zinc sulphate (1–5 gr. to the ounce—0.0648–0.324 to 32.0) or a saturated solution of boric acid. If the salts of zinc or silver are used, not more than an ounce of the solution should be allowed to enter the bladder, and much less than this amount will generally suffice. In cases in which there exist patches of ulceration the application must be made directly to these areas through the endoscope or cystoscope. Stronger solutions may now be employed, as silver nitrate, 20–30 gr. (1.29–1.94) to the ounce. This application should be followed by a slight irrigation of the bladder.

When this local medication fails to effect a cure, permanent drainage of the bladder must be secured—in the male by a suprapubic or perineal incision, and in the female by the establishment of a vesico-vaginal fistula. This places the bladder absolutely at rest, and gives the inflamed mucosa a chance to heal under proper medication.

As to internal remedies, various agents that possess a local stimulating effect upon the genito-urinary tract are advised by most authors, but I think little is to be gained from their employment as compared with the results achievable from topical treatment. Most efficacious among internal remedies are—oil of sandalwood, terebene, urotropin, pichi, buchu (fluid extract), and the oil of copaiba. If disinfection of the bladder *in loco* is not practicable, antiseptics should be given internally, combined with those stated above. Salol and potassium chlorate are excellent for this purpose.

NEOPLASMS OF THE BLADDER.

PRIMARY new-growths of the bladder are exceedingly rare, occurring, however, with greater frequency in males in about the proportion of 3 to 1; they may be either benign or malignant. On the other hand, secondary neoplasmata, particularly carcinomata, are relatively common.

The most frequent variety of new-growth encountered is carcinoma, particularly the so-called villous or papillomatous carcinoma, Williams[1] finding in 20 women affected with bladder-tumor, carcinoma in 16. Other growths are sarcomatous, fibromatous, cystic, and papillomatous in nature.

The **symptoms** are the same for all varieties, and include, first and most commonly, *hemorrhage* (which is both persistent and free), together with pain, frequency of micturition, and occasionally the discharge of detached fragments of the growth. In carcinomatous cases of advanced standing cachexia will be marked. By means of the cystoscope the nature of the complaint is disclosed. In the case of secondary growths the primary tumor may often be detected.

The **prognosis,** of course, will depend upon the nature of the growth.

The **treatment** is purely surgical.

VESICAL HEMORRHAGE.

(*Vesical Hemorrhoids.*)

HEMORRHAGE of the bladder has been mentioned as a symptom of various affections, both general and local, among the former being leukemia and malarial hematuria, and among the latter nephrolithiasis and tuberculosis and carcinoma of the bladder. It is also a prominent manifestation in stone in the bladder, and not infrequently appears in pregnancy (late). Independently of the operation of all of the above-mentioned etiologic factors, hemorrhage has been known to occur from the bladder, and recent precise methods of exploring the viscus (endoscopic examination) have shown it to be due to a hemorrhoidal state of the vessels. The hemorrhage may be profuse, and, rarely, even fatal in its effects.

The **diagnosis** is based in part upon the absence of the more obvious causes of hematuria and the presence of free bleedings, but chiefly upon the result of a careful cystoscopic exploration of the bladder.

The **prognosis,** so far as my experience extends, is eminently favorable, though a few fatal cases have been reported.

Treatment.—This is mainly local. The bladder may be irrigated with an astringent solution (1 per cent. tannic acid, ½ per cent. alum), and this may be alternated with an antiseptic solution (3 per cent. boric acid, 1 per cent. salicylic acid). I have recently observed a case in which recovery followed the internal admission of the extract. hamamelis fluid. (ʒj–4.0), *t. i. d.*

[1] *Brit. Med. Jour.*, 1889.

NEUROSES OF THE BLADDER.

IRRITABILITY OF THE BLADDER.

Definition.—By this term is meant a condition of the bladder in which there exists an hyperesthesia of the organ, especially of the neck—that portion surrounding the urethral and ureteral orifices (*vesical trigone*)—without the presence of any tangible cause therefor. This must be distinguished from the irritability that is associated with true organic disease of the bladder itself, as in the presence of calculi, tumors, or fissure of the neck, or with disease of the surrounding structures.

Pathology.—Cystoscopic examination of the bladder may reveal a slight increase in the vascularity of the mucous membrane. The condition of irritable bladder in women, which has previously been held to be a purely functional derangement, is now regarded by Dacheux and Zuckerkandl as a localized hyperemia, especially at the *bas fond*, and less often at the beginning of the urethra.[1]

Etiology.—While in many instances no well-defined causal relations can be determined, it is very generally true that the patients who are the subjects of vesical irritability are individuals of a neurotic temperament, very often manifesting strong hysteric tendencies. They are generally ill-nourished, fretful, irritable, peevish, suffering almost constantly from vague neuralgic attacks in different portions of the body (cephalalgia, tic douloureux, lumbo-sacral pain), and in a chronic condition of physical prostration. Frequently they eventually develop a true hypochondriasis or melancholia. In others there may be found a history of extreme mental and physical tire, overwork, business anxiety, over-indulgence in venery, menstrual irregularity, dysmenorrhea, ovarian or uterine disorders, long-continued gastro-intestinal disturbance (dyspepsia), improper hygienic surroundings, improper regimen, indulgence in late hours, and a general lack of will-power. It must, however, be remembered that subjects of chronic malarial intoxication very often manifest all the symptoms of vesical irritability, marked, it may be, by a feature of more or less periodicity. Lithemic individuals also are very prone to develop a pronounced vesical irritability, the affection in them probably resulting from the local action of the highly concentrated and irritating urine. The condition must commonly, however, be regarded as belonging essentially to the large group of neuroses.

In a certain percentage of cases the bladder-trouble is a reflex manifestation of some disease of an adjacent organ, as the urethra, ureter, vagina, rectum, anus, or the internal organs of generation. These are not, however, to be looked upon as cases of true neurotic vesical irritability.

Symptoms.—The symptoms of irritable bladder are mainly extreme painfulness and *frequency of micturition*, associated with marked *vesical* and *rectal tenesmus*. The dysuria is not always or altogether relieved by micturition; indeed, the pain may be just as severe, or even worse after, than before, the voiding of the urine. Especially is this true when there coexists a more or less spasmodic muscular action of the bladder-walls,

[1] *The American Year-Book of Medicine and Surgery*, 1897, p. 576.

the hypersensitive mucosa then being squeezed, and the patient suffering at times to such an extent as to be thrown almost into a state of collapse. There is usually a sense of weight or pressure in the pubic region, which is largely relieved when the patient assumes the recumbent posture. Urination is often performed spasmodically, or there may be a *spasm* of the urethra and neck of the bladder resulting in an utter inability to perform the act. The *urine* may be normal in appearance and amount. Very often it is increased in quantity (*hysteric polyuria*), and at times the opposite may be true and more or less suppression be noted. In lithemic cases the urinary characteristics already mentioned under that condition will be present (*vide* p. 440).

Diagnosis.—Very frequently will simple vesical irritability be confounded with true cystitis. The points of differentiation, however, are as follows:

Irritable Bladder.	Cystitis.
The patient is of a neurotic temperament, and generally gives no history of organic bladder-disease nor of operations upon the bladder.	May occur in any individual, irrespective of temperament. It frequently follows catherization, sounding, or other traumatism.
Pain is severe, and often worse after micturition.	The pain is usually much relieved by micturition.
The constitutional symptoms are those of nervous depression.	The constitutional symptoms are not marked, save in grave cases.
Never results fatally.	May result fatally.
The urine does not present any marked alteration in its physical or chemical qualities. It may show hyperacidity, or extreme concentration, or dilution.	There are always present marked and characteristic alterations in the physical and chemical qualities of the urine.
The appearance of the mucosa is negative in true neurosis.	Cystoscopic exploration reveals the angry and diseased mucosa, and may show the cause (calculus, tumor).
The duration is always protracted.	The duration of acute attacks may be short.

Prognosis.—Good as regards life; doubtful as regards the ultimate cure of the patient.

Treatment.—Since the condition is largely one of neurotic origin, the attention of the physician must be directed mainly toward a betterment of the state of the nervous system. Absolute rest, physical and mental, must be insisted upon, and the patient must be subjected to a course of strict moral suasion whenever this may be deemed necessary. Any cause of reflex irritation must be removed, and a careful search should be instituted for some such condition as cervical stenosis, uterine displacements, anal fissure, hemorrhoids, stricture of the rectum, vaginitis, urethritis, tuberculous infection of Skene's glands of the urethra, chronic gastro-intestinal catarrh, and the like. The habits of the patient must be inquired into, and late hours, the eating of improper and unwholesome articles of food, masturbation, or the reading of sensational and trashy literature corrected. In many instances the pronounced neurasthenic condition demands a course, more or less protracted, of the Weir Mitchell rest-treatment (*vide* Neurasthenia, p. 1234). The urine should be carefully examined for lithemic and other pathologic features, and by an ap-

propriate course of treatment it should be rendered as bland and unirritating as possible. Large draughts of diluent drinks may be of benefit, and if these be combined with the prolonged administration of nerve-sedatives and antispasmodics, a marked amelioration of the patient's condition may be secured. In cases associated with spasmodic muscular contraction it may become necessary to employ an occasional suppository of opium and belladonna, or an enema of chloral hydrate. Change of air and scene, regulation of the diet, the institution of a proper course of gymnastics, mental and physical, and the observance of a happy and cheerful atmosphere will generally do much to improve the patient's condition. The administration of tonics (strychnin, iron) and the prevention of constipation are very essential. Especially must it be remembered that in all these cases of simple vesical irritability physical exploration of the bladder is absolutely contraindicated. The patient's mind must be directed away from the bladder in order to secure good results.

NEUROSES OF MICTURITION.

1. **Incontinence of Urine** (*Enuresis*).—An inability to retain the urine. This may arise from a number of causes. Frequently it is the result of some lesion of the spinal cord involving the sphincteric center of the bladder; this is known as *paralytic incontinence*, and is to be recognized by a constant dribbling, alternating with spurts of urine when voluntary or involuntary muscular action is brought into play, as in the act of coughing, sneezing, or bending forward of the body. It may be the result of a general bodily weakness or after prostrating diseases (typhoid, late stages of pulmonary tuberculosis). Again, it may result from some local condition in the bladder or urethra. Here may be mentioned paralysis of the urethra from over-dilatation or from traumatism, or that due to pressure of the fetal head in a prolonged labor; imperfect vesical innervation; over-distention of the bladder, producing a paresis of its walls; or from some temporary obstruction at the urethra or base of the bladder, such as a tumor or a sharply retroflexed uterus. It may be a result of over-distention of the bladder, with partial paralysis of the sphincter, the bladder remaining overfilled, while there is a constant escape of a few drops of urine (*incontinence of retention*). It may follow some local causes of irritation, as the presence of vesical calculi, pressure from an anteflexed uterus upon the fundus of the bladder, cystitis, and parasites. The condition known as *spasmodic incontinence* is that due to an over-action of the compressor muscle of the bladder, as a consequence of which there is a diminution of the vesical capacity, the urine being forcibly and involuntarily ejected at irregular intervals. Finally, *nocturnal enuresis* is that variety which is so common in young, delicate, and often neurotic children: this is usually noticed in the early hours of sleep, and is often the result of some local irritation acting upon a hypersensitive organism, such as the presence of oxyurias, an elongated prepuce, contraction of the urethral meatus, or masturbation. Bierhoff[1] is of the opinion that the essential or ultimate condition is hyperesthesia of the deep urethra or sphincter from hyperemia or inflammation. Nocturnal incontinence may be a manifestation of nocturnal epilepsy or

[1] *Phila. Med. Jour.*, May 26, 1900.

of incipient cerebral or spinal disease (Fitz). Adenoid vegetations may bear an indirect causative relation to the condition, and it may be a symptom of thyroid hypoplasia. In the female, urethral papillomata and caruncles have been assigned as causes. The hyperacidity of the urine associated with podagra may also excite enuresis. The constant escape of urine in the paretic cases is apt to result in extensive excoriation of the parts.

The *treatment* varies according to the cause. The enuresis of children, if left alone, will eventually cure itself as the age and strength of the patient increases, though obvious exciting causes, if present, should be removed if not impracticable. Good hygiene, systematic evacuation of the bladder, elevation of the hips on a pillow in bed, plenty of out-of-door exercise, a change to the seashore or mountains, an abundance of suitable and strengthening food, mainly vegetable, so that a large proportion of the fluid output occurs by way of the intestines, with a minimum of water, and milk, especially late in the day, and the administration of tonics (iron, cod-liver or olive oil, and strychnin), will generally effect a cure. The fluid extract of rhus aromatica in 5- to 15-drop doses, thrice daily, has been very beneficial in children. Excellent results often follow the administration of minute doses of atropin or tincture of belladonna. A favorite formula of my own in cases possessing a hypersensitive nervous organization has long been as follows:

℞. Tr. belladonnæ, ʒss–j (2.0–4.0);
Sodii brom., ʒij (8.0);
Ac. hydrobrom. dil., ʒijss (10.0);
Ext. ergotæ fl., ʒij (8.0);
Glycerini, ʒj (4.0);
Elix. simplicis, q. s. ad ʒiv (128.0).
M. et Sig. ʒj (4.0) three or four times a day for a child of five years.

In very delicate or feeble children suffering from enuresis I substitute a motor tonic and stimulant (tr. nucis vom.) for the bromids or nerve-sedatives. In cases showing marked hyperacidity the alkalies or alkaline mineral waters, with careful rearrangement of the diet, are indicated. Klotz advises to raise the foot of the bed and make the patient sleep on his side. Again, the little sufferer may be wakened prior to the hour for the occurrence of the incontinence.

Spasmodic action of the vesical compressor may be relieved by the cautious use of the motor depressants, while its converse, paresis, demands the exhibition of full doses of strychnin or tincture of nux vomica. The judicious and careful use of the catheter, followed by the administration of strychnin, will promptly effect a cure in the incontinence of retention. Any local cause of vesical irritation must be removed. Galvanism in the paretic cases, applied both to the bladder and to the urethra, may be of service. Forchheimer uses the faradic current; in girls one pole is introduced into the vagina, in boys into the rectum, while the other pole is placed over the region of the bladder. The current must not be of too great strength, and he begins with the weakest induction current, which is gradually increased. In the female Sänger suggests massage of the urethra. Vibratory massage has proved successful in a

few cases. Should excoriation occur, bland ointments, as of zinc oxid and lanolin, should be used. Removal of adenoid vegetations has been recommended in cases in which they produce conditions of malnutrition. For cases caused by thyroid insufficiency, the use of thyroid extract will relieve the enuresis and also bring about marked improvement in the general physical and mental condition.

2. **Retention.**—Nervous retention of the urine is occasionally encountered in hysteric and highly neurotic individuals. Its most common manifestation is an inability to urinate in the presence of others. It is also occasionally noted after childbirth, when it may be due to nervous reaction, to edema and tortuosity of the urethra, or to a temporary inability of the bladder-walls to contract upon their contents, thereby permitting a longer retention of the vesical contents, and even favoring over-distention of the organ. If the urine be allowed to remain for too long a period in the bladder, fermentative changes follow and a secondary cystitis will result. Under these circumstances an exfoliation of a portion or even of the entire bladder-epithelium may be noted.

The *treatment* consists in the administration of strychnin and other nerve-tonics, in building up the general constitution, and in affording a change of air and recreation. In that variety following childbirth the patient should be urged to make voluntary efforts at micturition, and these may be seconded by the firm application of an abdominal binder and compress, or of hot, moist flannel cloths, kept up for twenty minutes or a half hour. The sound of running water, as when pouring water from a pitcher into the basin, often causes a contraction of the bladder and excites the flow of urine. It may become necessary, the foregoing methods failing, to resort to catheterization, the usual antiseptic precautions being observed.

PART IX.

DISEASES OF THE NERVOUS SYSTEM.

THE central nervous system is generally divided into two parts—the brain and the cord. The *brain* consists of the cerebral hemispheres, the basal ganglia, the pons, the cerebellum, and the medulla. The cerebral hemispheres are joined together by the corpus callosum and the anterior and posterior commissures. They are united to the pons by the crura cerebri, and the pons is continuous with the medulla, which in turn is continuous with the spinal cord. The surface of the cerebral hemispheres is divided by sulci or fissures into various regions, known as the frontal, parietal, temporo-sphenoidal, and occipital lobes. The superior longitudinal fissure separates the two hemispheres; the fissure of Sylvius is between the frontal and parietal lobes above and the temporo-sphenoidal lobe below. The fissure of Rolando divides the frontal from the parietal lobe, and the parieto-occipital fissure the latter from the occipital lobe. The continuation of the last-named fissure upon the median surface forms the upper boundary of the cuneus, the lower boundary of which is the calcarine fissure. The hippocampal fissure separates the fascia dentata from the hippocampal gyrus, and by its extension inward produces an elevation in the lateral ventricle known as the hippocampus major. Each lobe is subdivided by secondary fissures into a number of lobules. The topography of the hemispheres is important because it is now possible to map out with considerable accuracy the regions in which various motor impulses originate, and with less accuracy the regions in which various sensory phenomena are perceived. The accompanying diagrams illustrate, more satisfactorily than could any description, the regions that have been hitherto determined (Figs. 68, 69, and 70). There is some discussion in regard to the degree of individuality of these centers, but the weight of evidence inclines to the belief that they are not sharply delimited. Ordinarily speaking, one side of the brain innervates the opposite side of the body; but certain parts, as the muscles of the trunk, appear to receive impulses simultaneously from both hemispheres, and other functions seem to be accomplished exclusively upon one side; thus motor speech is ordinarily disturbed only when the lesion is in the left hemisphere.

The central nervous system is composed practically of two elements—the neuroglia, or supporting substance, and the neurons. The neuroglia consists of round cells with radiating processes, lying in the

midst of a tangled network of fibers. Its function appears to be exactly similar to that of connective tissue. The neuron, or nerve-unit, consists of a ganglion-cell, the protoplasmic processes springing from it, and the neuraxon, or axis-cylinder. The cell-body consists of protoplasm and nucleus. The latter contains a nucleolus and a small amount of chromatin; the former is composed of a reticulum of fibrillar ground-mass, in which are found, in certain cells, peculiar bodies, that take the basic stain, are irregularly spindle-shaped, and are often arranged concentrically to the nucleus; they also extend a short distance into the protoplasmic processes. The protoplasmic processes branch irregularly, and along the sides of the finer ramifications are placed short lateral offshoots, the buds or gemmules. The axis-cylinder is a single process, of uniform thickness, usually single, but sometimes

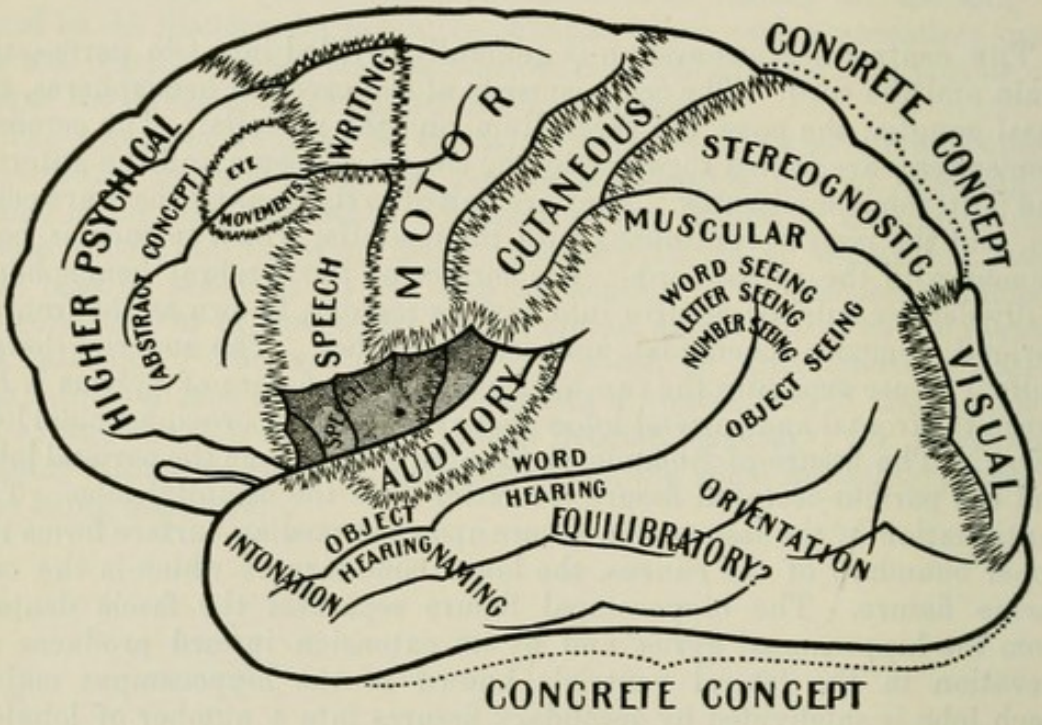


FIG. 68.—Side view of human brain, showing localization of functions (Charles K. Mills).

branched, and giving off at regular intervals fine, long branches, the collaterals; it terminates either as a tuft of fine fibers surrounding a ganglion-cell, or in a motor plate in the muscles, or in a special sense-corpuscle in the skin. It cannot be too frequently reiterated that each neuron constitutes an individual unit that is entirely independent of all other neurons and has no anatomical connection with them whatever.[1] A physiological communication must, of course, exist, that perhaps is analogous to electric induction; and it has been suggested, by Dercum among others, that during life the protoplasmic processes move about and make contact with the surrounding nervous structures. The functions of the various elements of the neuron are as yet imperfectly understood. The cell-body appears to exercise a trophic action over the

[1] Apáthy, and more recently Bethe, have claimed that delicate neuro-fibrils pass from one neuron to another. This has not yet been confirmed.

other parts, especially the axis-cylinder. It probably also generates the motor impulses. The protoplasmic process may have nutritive func-

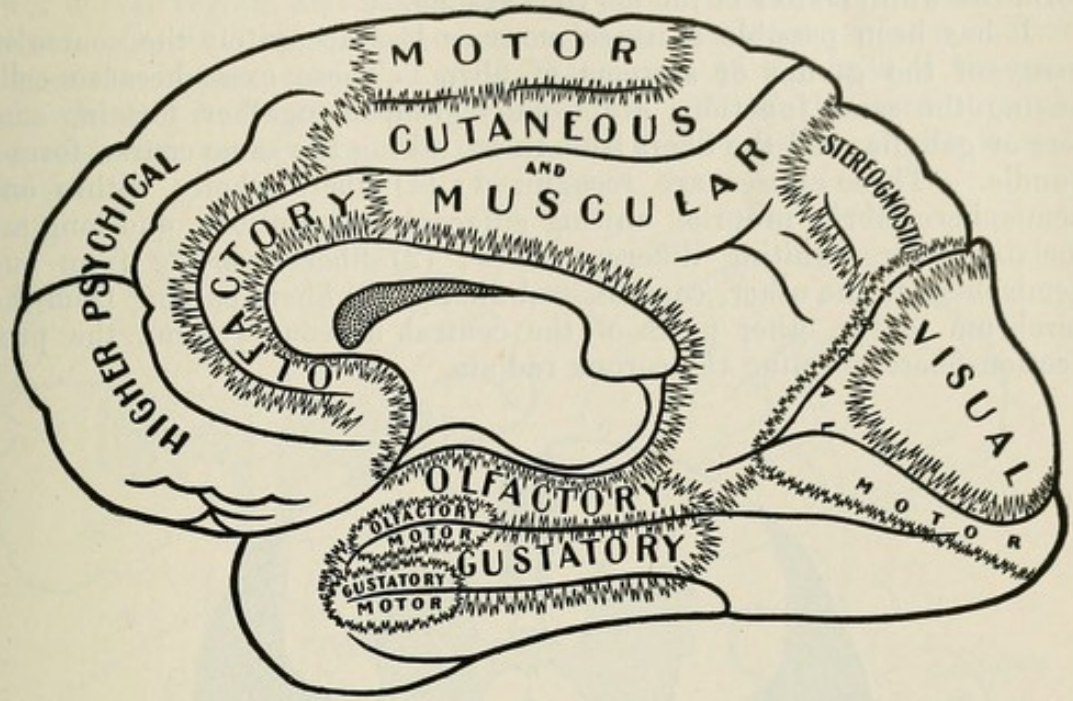


FIG. 69.—View of the mesial surface of human brain, showing localization of functions (Charles K. Mills).

tions, or serve to conduct impulses to the cells (cellipetal). The axis-cylinder conducts impulses from the cells (cellifugal), except in the case

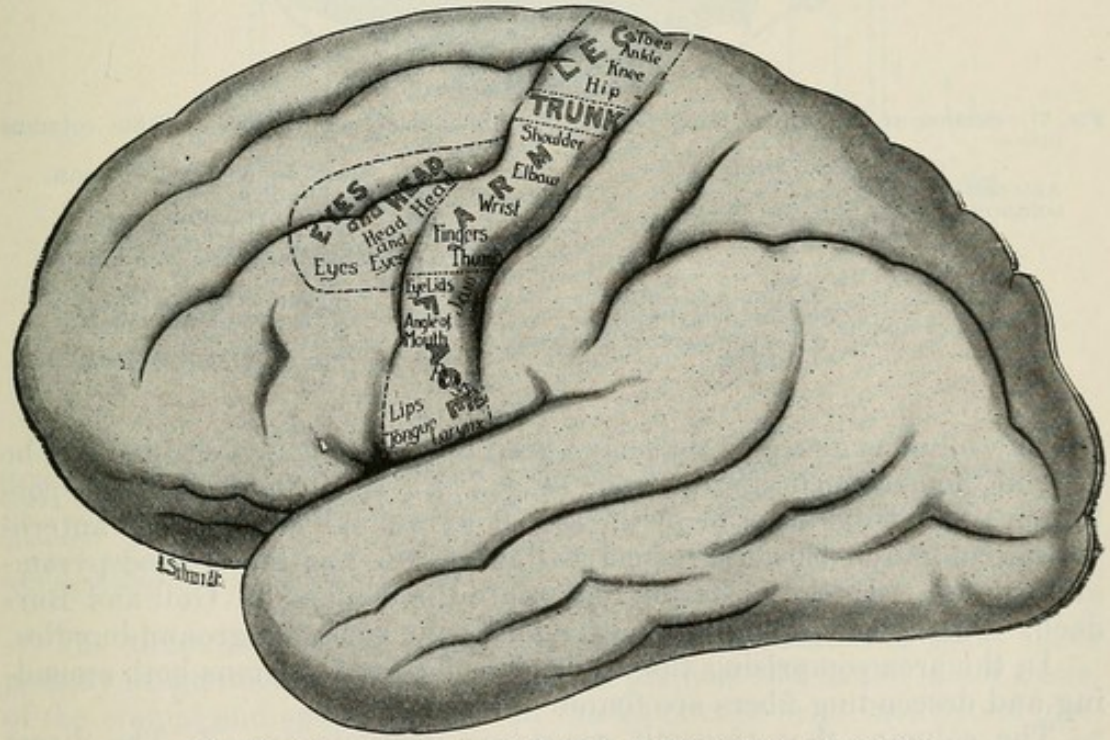


FIG. 70.—The subdivisions of the motor cortex (Mills and Frazier).

of the peripheral process of the cells of the spinal ganglion.[1] A short

[1] Lenhossek has suggested that this is a modified protoplasmic process.

distance from the cell the axis-cylinder is enveloped by the myelin-sheath, giving rise to the nerve-fiber, and when aggregated together these fibers form the white matter of the nervous system.

It has been possible to trace more or less accurately the course of many of the groups or systems of fibers. These exist because cells having the same functions are usually grouped together, forming centers or ganglia, and the fibers from these, taking the same course, form a bundle. Three classes are recognized: (1) fibers wholly within one hemisphere, fibriæ propriæ, uniting adjacent convolutions, and long association-fibers, uniting different lobes; (2) fibers passing from one hemisphere to the other, commissural fibers; (3) fibers passing from the cerebrum to the other parts of the central nervous system, the projection-fibers, forming the corona radiata.

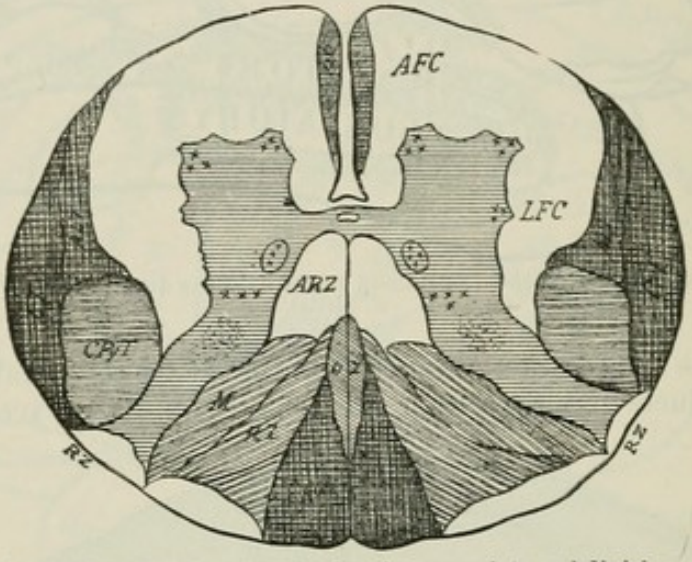


FIG. 71.—Section of spinal cord (after Dana), showing complete subdivision of white columns into—

Anterior columns.
- *DPy*, direct pyramidal tract.
- *AFC*, anterior fundamental column.

Lateral columns.
- *LFC*, lateral fundamental column.
- *LL*, lateral limiting layer.
- *CPyT*, crossed pyramidal tract.
- *CT*, direct cerebellar tract.
- *ALT*, antero-lateral ascending tract.

Posterior columns.
- Column of Goll.
- Column of Burdach.
- *RZ*, rim-zone, or Lissauer's column.

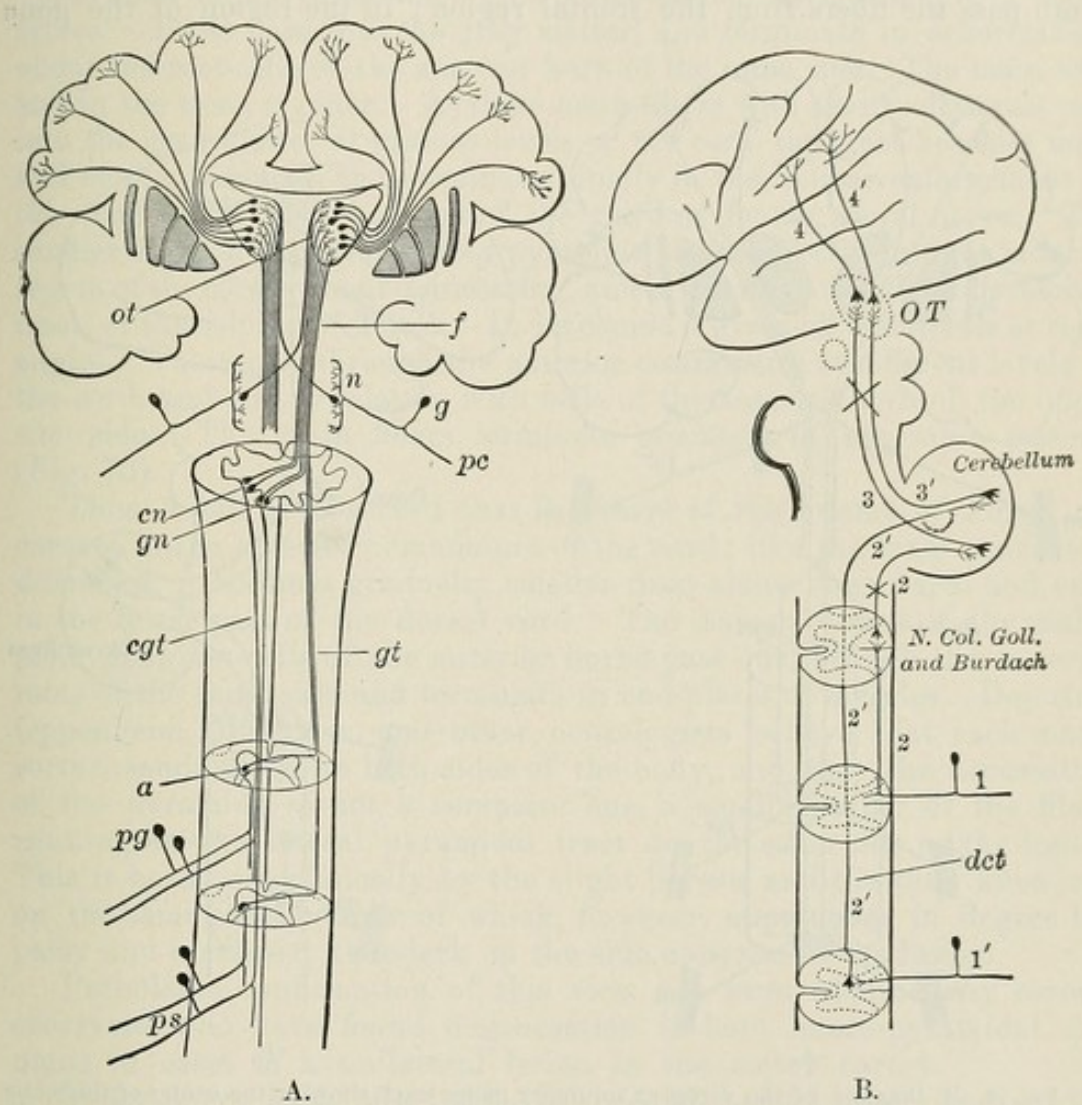
- *ARZ*, anterior root-zone.
- *MRZ*, middle root-zone.
- *OZ*, oval zone.
- *PRZ*, posterior root-zone.

The columns or tracts that have been mapped out in the cord may be seen in the accompanying diagram (Fig. 71). In the antero-lateral portion are found the anterior or uncrossed pyramidal column, the antero-lateral column of Gowers, the cerebellar column, and the crossed pyramidal column. In the posterior region are the columns of Goll and Burdach. The rest of the white matter forms the so-called ground-bundles.

In the area comprising the anterior and lateral columns both ascending and descending fibers are found.

The columns that transmit *ascending impulses* are—1. The direct lateral cerebellar column. 2. The antero-lateral ascending column of Gowers. 3. The antero-lateral ground-bundle or fundamental column. 4. The columns of Goll and Burdach. *Descending impulses* are transmitted chiefly by the direct and crossed pyramidal tracts and the antero-

lateral descending tract. The direct lateral cerebellar tract of Flechsig takes origin in the cells of the column of Clarke, and first appears in the lower dorsal region, and passes through the restiform body to the cerebellum. Gowers' tract, or the antero-lateral ascending column, is first seen in the lumbar cord, and arises from some of the cells of the posterior horn. It then crosses to the other side of the cord through the posterior commissure and terminates in the region of the lateral nucleus.



A. B.

FIG. 72.—A, the direct sensory tract: *ps*, Peripheral spinal nerves; *pg*, ganglion on posterior roots of spinal nerves; *gt*, Gower's tract; *cgt*, columns of Goll and Burdach; *cn*, nucleus cuneatus; *gn*, nucleus gracilis; *a*, cells in posterior horn; *pc*, peripheral cranial nerve; *g*, ganglion on cranial sensory nerve; *n*, cells of cranial sensory nerves in medulla; *f*, fillet; *ot*, optic thalamus.

B, indirect sensory tracts (Van Gehuchten): *dct*, Direct cerebellar tract. The numbers represent the different series of neurons.

The direct and crossed pyramidal columns constitute the great motor path by which fibers descend from the cortex and end in the motor nuclei of the cranial and spinal nerves—in the latter case in the multipolar ganglion-cells of the anterior horns. Their origin is in the motor region of the cerebral cortex—*i. e.*, the ascending frontal, the paracentral lobule, and part of the second frontal convolution (Fig. 70); they then approach one another, as do the fibers from all parts of the cerebral cortex (known collectively as the *corona radiata*), to enter the internal capsule. This

may be described as a wedge, bounded in front and to the inner side by the caudate nucleus and the optic thalamus, and on the outer side by the lenticular nucleus.

All of the fibers of the corona radiata do not pass through the internal capsule, some being lost in the gray matter of the basal ganglia, while others take origin in the ganglia. The angle of the internal capsule is known as the genu or knee, the part anterior to it as the anterior limb, and the part posterior as the posterior limb. Through the anterior limb pass the fibers from the frontal region; in the region of the genu

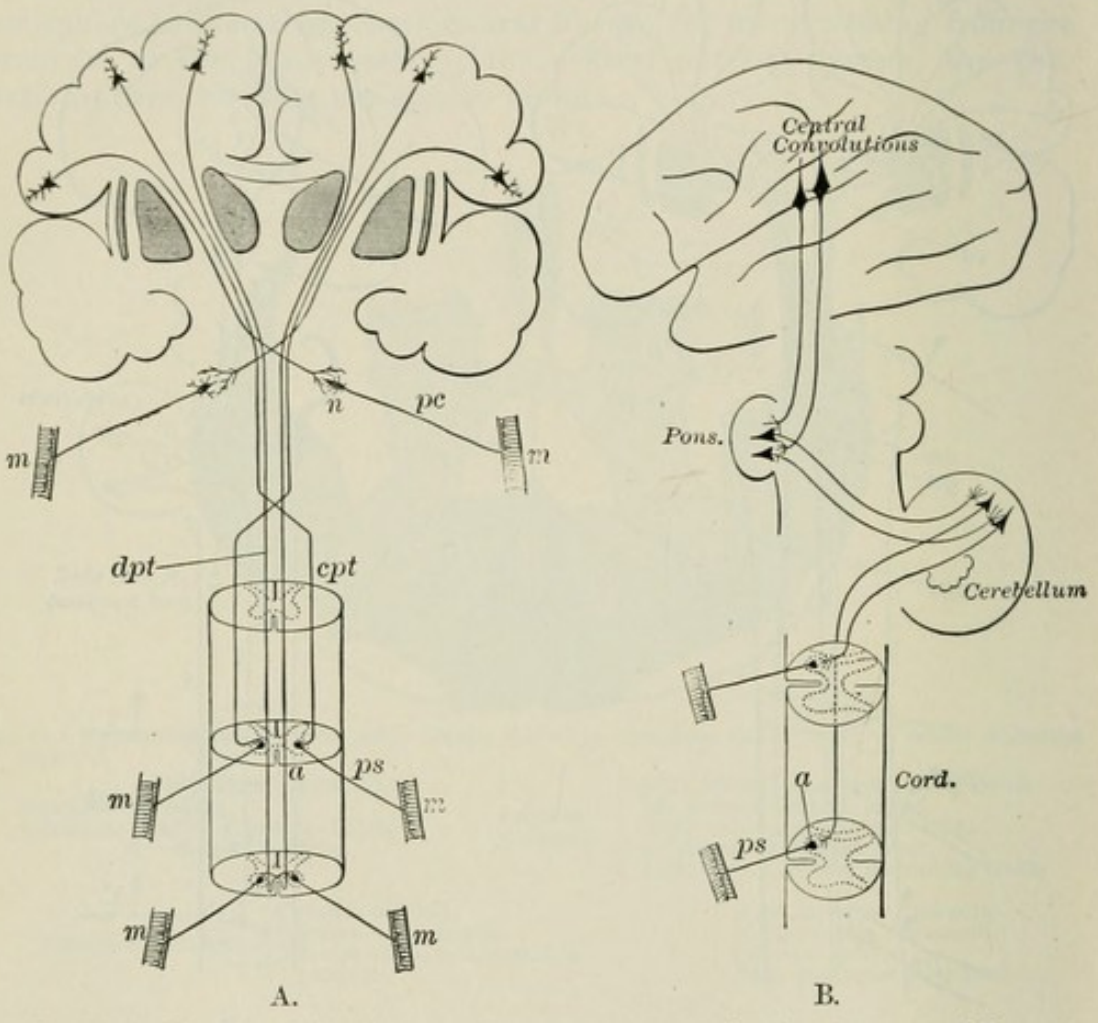


FIG. 73.—A, Diagram of the direct or voluntary motor tract, showing the center of the motor impulses from the cerebral cortex of the voluntary muscles (Van Gehuchten): *m*, Muscles; *n*, cells of nuclei of motor cranial nerves in pons and medulla; *a*, motor cells in anterior horns of spinal cord; *dpt*, direct pyramidal tract; *cpt*, crossed pyramidal tract; *pc*, peripheral cranial nerve; *ps*, peripheral spinal nerve.

B, Diagram of the indirect or involuntary motor tract.

are the fibers for the muscles of the face and tongue; and in the posterior limb, the motor fibers to the extremities, also the sensory or tegmental fibers, and at its posterior end the fibers of the optic radiation.

The crusta consists of fibers that pass through the pons and enter the medulla, constituting its pyramidal tracts.

The tegmental fibers are continuous through the longitudinal fibers of the pons with those derived from the *formatio reticularis* of the medulla. This is formed by fibers from the superior cerebellar peduncles, the olivary body, and the posterior and lateral columns of the cord, which are rein-

forced in their upward course by fibers derived from the quadrigeminal and geniculate bodies.

Tracing the pyramidal fibers through the medulla, they will be found to divide into two unequal portions at its lower part. The larger decussates at this point (the region of the first and second cervical nerves), constituting the decussation of the pyramids; it then crosses to the posterior part of the lateral column of the opposite side, in which it runs as the crossed pyramidal tract.

In their course these fibers give off collaterals at right angles to themselves. These pass into the gray matter, and terminate in arborizations about the root-cells of the anterior horn of the same side. The main axes end in the same manner. As these main fibers with their collaterals pass into the gray matter at various levels of the cord, the tract becomes more and more attenuated, and terminates finally in the lumbar enlargement of the cord in the neighborhood of the third or fourth sacral nerve. The smaller division of the medullary pyramids passes directly into the anterior region of the cord without decussating, and is known as the direct pyramidal tract, or the column of Türck. In its course it gives off collaterals at right angles. These pass through the anterior commissure at different levels of the cord, and end in relation with cells of the anterior horn of the opposite side. The main fibers terminate precisely in the same manner (Fig. 73).

Thus it will be observed that the fibers of the column of Türck decussate in the anterior commissure of the cord; like the tract previously described, it becomes gradually smaller from above downward, and ends in the lower part of the dorsal cord. The axis-cylinders of the multipolar ganglion-cells of the anterior horns pass out through the anterior roots of the same side and terminate in end-plates of muscles. Dejerine, Oppenheim, Monakow, and other neurologists believe that each motor cortex sends fibers to both sides of the body, and that the decussation of the pyramids is not a complete one, a small number of the fibers running in the lateral pyramidal tract on the same side as the lesion. This is borne out clinically by the slight paresis and the plus knee-jerk on the same side, neither of which, however, approaches in degree the palsy and increased knee-jerk on the side opposite to the lesion.

Pathologic confirmation of this view has been obtained by several observers, who have found degeneration in both latero-pyramidal columns in cases of a unilateral lesion in the motor cortex.

Motor-fibers from the nuclei of cranial nerves after decussating join with motor fibers of the internal capsule. The exact course of these fibers, however, has not been demonstrated anatomically. Since many of the muscles supplied by the cranial nerves functionate bilaterally—*e. g.* the eye-muscles and the muscles of mastication—the supposition is that in addition to fibers from its own nucleus each motor cranial nerve receives fibers from the corresponding nucleus of the opposite side. It was Broadbent who first pointed out that parts that functionate bilaterally are supplied from both sides of the brain.

The course of the fibers of the posterior column is as follows:

The ganglion-cells on the posterior roots give rise to two fibers, fused for a short distance from the cell, but soon bifurcating. The longer of the two, the centrifugal fiber, extends to the surface

and terminates in pointed or bulbous endings in the epidermis, or in special sensory nerve-endings in tactile cells, tactile corpuscles, or end-bulbs. The centripetal fibers or axons penetrate the cord, and divide in the white matter into ascending and descending fibers. The former may be either long or short. The short fibers are vertical at first, but finally bend into the gray matter, and end in relation with certain cells of the anterior cornua, forming perhaps a part of the reflex arc. Their collaterals end in a similar manner. The long fibers extend up the cord to the medulla, ending in the usual manner in the gray nuclei of the columns of Goll and Burdach; these are known as the *nucleus gracilis* and *nucleus cuneatus*, respectively. They also give off collaterals in their course. The descending fibers, on the other hand, are all short, and probably constitute the so-called *comma tract* of Schultze.

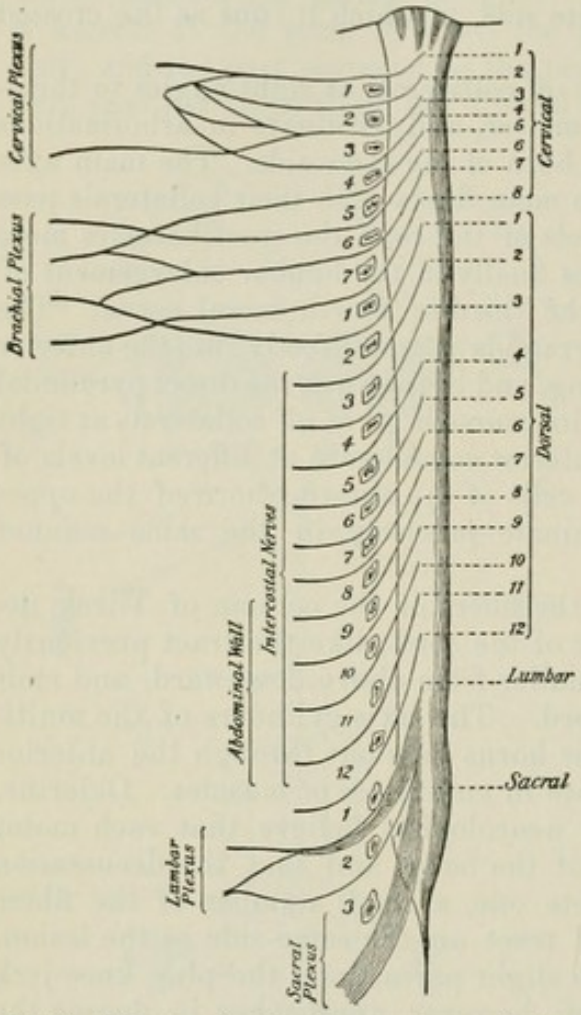


FIG. 74.—Diagram showing the groupings and plexuses of the spinal nerves (redrawn after Baker).

Since fibers continue to enter the cord at different levels, those that have entered below are pushed more and more toward the median line. It will thus be seen that the column of Goll is made up almost entirely of long fibers, and that the column of Burdach also contains long fibers, although it is probable that the short ones predominate. The long fibers are concerned in muscular coördination and equilibrium. It is likely that the fibers of pain and temperature sense, although entering by the posterior roots, do not pass up through the posterior columns, but rather through the tract of Gowers.

The skin areas innervated by the different segments of the cord are shown in Fig. 75.

Since the post-natal growth of the vertebræ is more rapid than that of the cord, it follows that the spinal nerves assume a more and more oblique position, until finally the spinal segments, each of which consists of an anterior and posterior nerve-bundle with a transverse plane of white substance, lie considerably above the vertebræ after which they are named (see Fig. 74). The following table (Starr, modified by Mills and Dana from the experimental and clinical studies of Thorburn and others) shows the localization of function (not organs) in the different segments of the cord:

Localization of the Functions of the Segments of the Spinal Cord.

Segment.	Muscles.	Reflex and Centers.	Sensation.
First cervical.	Rectus laterales. Rectus capitis. Anticus and posticus. Sterno-hyoid. Sterno-thyroid.		
Second and third cervical.	Sterno-mastoid. Trapezius. Scaleni and neck. Omo-hyoid. Diaphragm.	*Hypochondrium* (?). Sudden inspiration produced by sudden pressure beneath the lower border of the ribs.	Back of head to vertex and neck. (Occipitalis major, occipitalis minor, auricularis magnus, superficialis colli, and supraclavicular.)
Fourth cervical.	Diaphragm. Deltoid. Biceps. Coraco-brachialis. Supinator longus. Rhomboid. Supra- and infra-spinatus.	*Pupillary* (fourth cervical to second dorsal). Dilatation of the pupil produced by irritation of the neck.	Neck. Shoulder, anterior surface. Outer arm. (Supraclavicular, circumflex, external musculo-cutaneous, cutaneous.)
Fifth cervical.	Deltoid. Biceps. Coraco-brachialis. Brachialis anticus. Supinator longus. Supinator brevis. Deep muscles of shoulder-blade. Rhomboid. Teres minor. Pectoralis (clavicular part). Serratus magnus.	*Scapular* (fifth cervical to first dorsal). Irritation of skin over the scapula produces contraction of the scapular muscles. *Supinator longus.* Tapping the tendon of the supinator longus produces flexion of forearm.	Back of shoulder and arm. Outer side of arm and forearm to the wrist. (Supraclavicular, circumflex, external cutaneous, internal cutaneous, posterior spinal branches.)
Sixth cervical.	Deltoid. Biceps. Brachialis anticus. Subscapular. Pectoralis (clavicular part). Serratus magnus. Triceps. Pronators. Rhomboid. Latissimus dorsi.	*Triceps* (fifth to sixth cervical). Tapping elbow tendon produces extension of forearm. *Posterior wrist* (sixth to eighth cervical). Tapping tendons causes extension of the hand.	Outer side and front of forearm. Back of hand, radial distribution. (Chiefly external cutaneous, internal cutaneous, radial.)
Seventh cervical.	Triceps (long head). Extensors of wrist and fingers. Pronators of wrist. Flexors of wrist. Subscapular. Pectoralis (costal part). Serratus magnus. Latissimus dorsi. Teres major.	*Anterior wrist* (seventh to eighth cervical). Tapping anterior tendons causes flexion of wrist. *Palmar* (seventh cervical to first dorsal). Stroking the palm causes closure of the fingers.	Radial distribution in the hand. Median distribution in the palm, thumb, index, and one half of the middle finger. (External cutaneous, internal cutaneous, radial, median, posterior spinal branches.)
Eighth cervical.	Triceps (long head). Flexors of wrist and fingers. Intrinsic hand-muscles.	. . . . .	Ulnar area of hand, back, and palm, inner border of forearm. (Internal cutaneous, ulnar.)
First dorsal.	Extensors of thumb. Intrinsic hand-muscles. Thenar and hypothenar muscles.	. . . . .	Chiefly inner side of forearm and arm to near the axilla. (Chiefly internal cutaneous and nerve of Wrisberg or lesser internal cutaneous.)
Second dorsal.	. . . . .	. . . . .	Inner side of arm near to and in the axilla. (Intercosto-humeral.)
Second to twelfth dorsal.	Muscles of back and abdomen. Erectores spinæ.	*Epigastric* (fourth to seventh dorsal). Tickling mammary region causes retraction of the epigastrium.	Skin of the chest and abdomen, in bands running around and downward, corresponding to spinal nerves.
		Abdominal (seventh to eleventh dorsal). Stroking side of abdomen causes retraction of belly.	Upper gluteal region. (Intercostals and dorsal posterior nerves.)
		Vaso-motor centers. Second dorsal to second lumbar.	Eleventh and twelfth dorsal testicle.

Segment.	Muscles.	Reflex and Centers.	Sensation.
First lumbar.	None.	*Cremasteric* (first to third lumbar). Stroking inner thigh causes retraction of scrotum.	Skin over groin and front of scrotum. (Ilio-hypogastric, ilio-inguinal.) Testicle.
Second lumbar.	Vastus internus.	*Patellar.* Striking patellar tendon causes extension of the leg.	Outer side and upper front of thigh. Lumbar region. (Genito-crural, external cutaneous.)
Third lumbar.	Sartorius; adductors of thigh. Flexors of thigh.	. . . . .	Front and outer side of thigh. Inner side of leg and foot.
Fourth lumbar.	Extensors of knee. Abductors of thigh.	*Gluteal* (fourth to fifth lumbar). Stroking buttock causes dimpling in fold of buttock.	Inner side of thigh, leg, and foot. (Internal cutaneous, long saphenous, obturator.)
Fifth lumbar.	Outward rotators. Flexors of knee. Flexors of ankle. Peronei. Extensors of toes.	*Achilles tendon.* Over-extension causes rapid flexion of ankle, called *ankle-clonus.*	Back of thigh and outer side of leg and ankle; sole; dorsum of foot. (External popliteal, external saphenous, musculo-cutaneous, plantar.)
First and second sacral.	Calf-muscles. Glutei. Peronei. Extensors of ankle. Small muscles of foot.	*Plantar* (fifth lumbar to second sacral). Tickling sole of foot causes flexion of toes and retraction of leg.	Back of buttock and thigh, side of leg and ankle: sole; dorsum of foot.
Third, fourth, and fifth sacral.	Perineal. Muscles of bladder, rectum, and external genitals.	Genital center. Vesical center. Anal center.	Circumanal region, anus, rectum, penis, urethra, vagina, perineum. (Small sciatic, pudic, inferior hemorrhoidal, inferior pudendal.)

To the foregoing table, which illustrates spinal localization, should be added another, showing what functions reside in the pons and medulla, as follows:

Nuclei.

III. Sphincter iris. Ciliary muscles. Levator palpebræ superioris. Rectus internus (in convergence). Rectus superior. Rectus inferior.

IV. Obliquus inferior. Obliquus superior. (Upper facial group.)

V. (Associated movement of levator palpebræ.) Muscles of lower jaw.

VI. Rectus externus. Rectus inter. of opposite side in lateral movements.

VII.—Facial muscles.

IX. Muscles of pharynx.

X. Muscles of esophagus.

XI. Muscles of larynx. (Motor cortical area, see p. 1067.)

XII. (Lower facial group.) Muscles of tongue.

Sensory Cortical Area.—Owing to the extensive compensation of sensory fibers, by means of which each side of the brain sends fibers to both sides of the body, it is impossible to map out the center with precision.

It is generally believed, for reasons already stated, that the ascending parietal convolution and parietal lobe contain muscular and tactile sensory functions. It is possible that the sensory zone extends to the mesial surface of the hemisphere, as does the motor area. That this is the chief sensory center, as claimed by some observers, is, however, very questionable (Figs. 68 and 69).

From the cuneus, fibers pass to the pulvinar, forming the optic radiation of Gratiolet. From the pulvinar they apparently pass to the external geniculate bodies, and thence to the anterior corpus quadrigeminum. The optic tracts arise by two roots that curve round the crusta on either side and unite immediately in front of the tuber cinereum. Fibers from the two tracts pass to the homologous sides of both retinæ; therefore the lesions posterior to the chiasm give rise to blindness of half of each retina on the same side, although the blind fields are on the opposite side—lateral homonymous hemianopsia (Fig. 77).

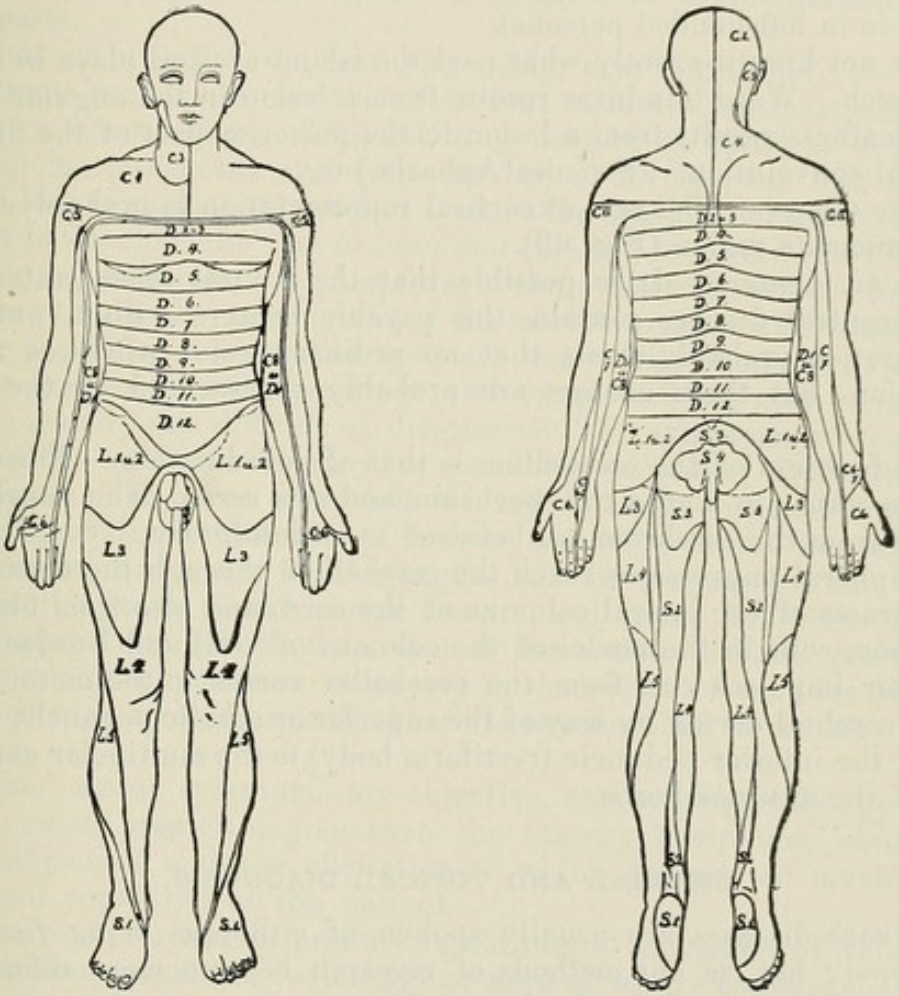


FIG. 75.—Showing the regions innervated by the different spinal roots or the corresponding segments of the cord. It should be remembered that the limits are not in reality so sharply defined, but extend into one another (Kocher).

Visual Centers.—The exact center for ordinary vision is in the cortex of the occipital lobe of the inner surface in the region of the calcarine fissure. A higher center exists, probably located in the angular gyrus, a lesion of which produces mind-blindness; this is a condition in which vision is not lost, but the objects seen are not recognized by the individual. Ferrier says that a lesion in this region sometimes gives rise to crossed amblyopia. The eye opposite to the lesions is chiefly affected, though vision is also restricted in the eye on the same side of the lesion (visual tract).

Olfactory Center.—This is located in the anterior part of the uncinate convolution, on the inner surface of the temporal lobe. It is possible,

too, that fibers pass from this region through the anterior commissure to the cortex of the opposite hemisphere.

Auditory Center.—A lesion in the posterior part of the first temporal convolution produces a deafness in the opposite ear that is transient in character, owing to compensation. Bilateral lesions produce complete deafness. Mind-deafness, or an inability to understand spoken words, has resulted from a lesion in the first temporal convolution of the left side.

Speech Center.—The articulate speech center is located in the posterior part of the left third or inferior frontal convolution, and in the adjacent part of the ascending frontal in right-handed people (but on the right side in left-handed persons).

It is not known exactly what part the island of Reil plays in articulate speech. Word-blindness results from a lesion in the angular gyrus. Word-deafness results from a lesion in the posterior part of the first left temporal convolution. (See also Aphasia.)

Taste Center.—The area of cortical representation is probably located in the uncinate region (Fig. 69).

Psychic Centers.—It is possible that the frontal lobes, anterior to the precentral fissure, contain the psychic centers. Such extensive compensation probably exists that no ordinary lesion produces mental aberration, but these centers are probably represented by the whole cortex.

The function of the cerebellum is that of coördination. Fibers pass from its cortex to that of the cerebrum, and *vice versa*. The impressions derived from the cerebrum are believed to be inhibitory.

Peripheral impressions reach the cerebellum through the direct cerebellar tracts of the lateral columns of the cord, and also from fibers derived from cells in the nuclei of the columns of Goll and Burdach.

Motor impulses run from the cerebellar cortex to the motor region of the cerebral cortex by way of the superior or middle peduncle, and by way of the inferior peduncle (restiform body) to the multipolar ganglion-cells of the anterior horns.

GENERAL AND TOPICAL DIAGNOSIS.

Nervous diseases are usually spoken of either as being *functional or organic;* but, as our methods of research become more refined and our technic more perfect, the breach between these two groups is being gradually but steadily lessened.

Organic nervous diseases may be produced by two types of lesions:

1. *Irritative*, causing an increase of function, continuous or intermittent. 2. *Destructive*, resulting in paralysis of motion or sensation, or both.

Irritative lesions are prone to become destructive in course of time. They may be operative in the upper segment, which includes the brain and fibers leading to or from it as far as the ganglion-cells of the cord; or in the lower segment, including the multipolar ganglion-cells of the anterior horn, together with the peripheral motor nerve-fibers.

When a complete pathway is involved a systemic disease is said to be produced. When two or more paths or neuron complexes are simultaneously involved combined systemic disease results.

Brain-lesions may be (*a*) focal or (*b*) diffuse. Cord-lesions are either (*a*) transverse, (*b*) focal, or (*c*) insular (a series of foci).

Cord-lesions result in ascending or descending degeneration, the destructive process travelling, as a rule, in the direction in which impulses are normally transmitted. In the fillet degeneration may extend up or down.

The theory has been advanced that the vulnerability of the tracts of the spinal axis is in direct proportion to the degree of their functional activity; hence the reflex (sensory and pyramidal) tracts are more likely to degenerate under nutritional disturbances or toxic processes than other parts.

It has been supposed that the tardy myelination of the pyramidal tracts predisposes to various nervous maladies, and particularly to those of a spastic type. The following may be accepted as a general rule: the motor-nervous system is the last to develop, the first to lose, and the last to regain, its function; while the sensory nervous system is the first to develop, the last to lose, and the first to regain, its function. In making a diagnosis it is, therefore, of the utmost importance to try to determine the locality and extent of the morbid process, and to ascertain whether the lesion is a focal or systemic one. The symptomatology of systemic diseases is pretty constant, and, except in their very incipiency, they are usually not difficult of diagnosis. The symptoms of focal diseases, on the other hand, vary, of necessity, according to the location of the focus. They are often difficult and at times impossible to diagnose. Especially is this true of lesions occurring in the frontal lobes of the cerebrum, in the basal ganglia, and in the cerebellum.

Since the study of the motor centers and tracts has been pursued with so much more success than that of the sensory system, positive or negative motor phenomena occurring in the course of nervous diseases furnish us with much more valuable information than do sensory manifestations.

Further, motor symptoms are objective, and consequently appeal to us in a much greater degree than the sensory symptoms, which are purely subjective, and the elicitation of which depends so much upon the mental capability of the patient.

Irritative motor-lesions produce, according to the degree of irritation, either fibrillary muscular twitchings or mild or severe convulsions, tonic or clonic in character.

Destructive motor-lesions, according to their extent, produce mere muscular weakness, paresis, or actual paralysis of a single muscle, groups of muscles, or of the entire musculature of one or more limbs.

Irritative sensory lesions give rise to neuralgia, hyperesthesia, or hyperalgesia.

Destructive sensory lesions cause a more or less complete absence of sensation, as analgesia, anesthesia, or loss of temperature-sense.

Upper-segment or Upper-system Diseases.—A lesion occurring in the motor pathway anywhere between the cortex and the multipolar cells of the anterior horns (but not including the latter) gives rise to the following symptom-complex: Loss of motion, both automatic and volitional, and chiefly on the side of the body opposite to the lesion. The paralysis is usually spastic in type. The muscles resist passive move-

ments, showing that their tone is increased. This is relative, and is due to the removal of cerebral inhibition, which allows the lower centers free play. They also tend to undergo shortening, and contractures result. Reflexes below the seat of the lesion are increased. If it is cerebral, this is chiefly on the side opposite the lesion, but also on the same side, the increase being the result of the removal of cerebral influences.

Owing to inactivity, the muscles of the paralyzed members may undergo some atrophy, though there are no degenerative changes, since the peripheral neuron bodies are intact. Usually there is very little if any, excepting when the disease has been acquired in childhood, in which case there is found to be a general lack of growth, bone as well as muscle. For the same reason the response to electric stimulation is not interfered with.

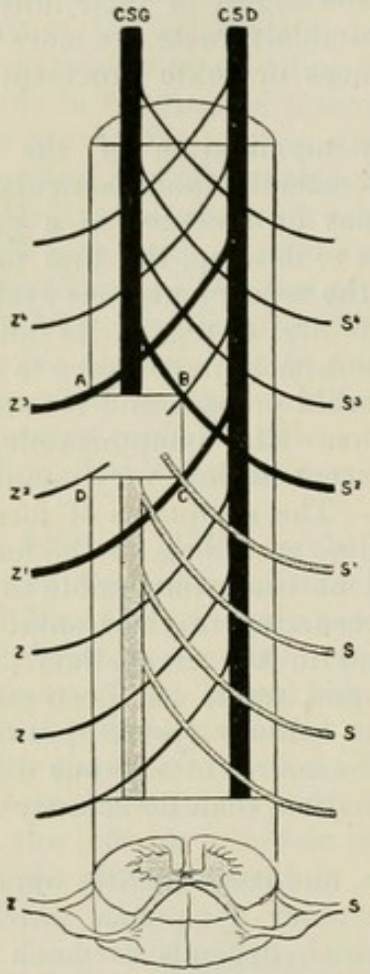


FIG. 76.—Scheme representing cord-lesion and effects in Brown-Séquard paralysis (after Brissaud): *CSG*, Left sensitivetract; *CSD*, right sensitive tract; *A*, *B*, *C*, *D*, lesion involving the left half of the cord; *S*, *S*, *S*, sensory roots from right side of body; *Z*, *Z*, *Z*, sensory roots from left side of body; Z^1, Z^3, and S^2 are irritated only at the points *A*, *B*, *C*, and their peripheral area is hyperesthetic; Z^2 is divided and its skin area is anesthetic on the same side as the lesion. Corresponding to S^1 and all the roots below arising from the right side of the body, there is anesthesia.

An irritative lesion of this upper system, particularly when operative in or upon the cortical region, gives rise to tonic or clonic convulsive movements. When the lesion is localized to a single center, focal or so-called Jacksonian epilepsy results. The cortex is wonderfully tolerant, when the lesion is of gradual onset, and the parts accommodate themselves to the slowly increasing pressure. However, a local irritative lesion may at first cause widespread symptoms, due, as Nothnagel pointed out, to pressure, vascular disturbances, or irritative inhibition.

Lower-segment or Lower-system Diseases.—This includes the peripheral neuron system. Since there is no crossing of the fibers, the lesion and resulting paralysis are on the same side of the body. The paralysis, however, is of the flaccid, flail-like variety, hypotonus being present. The muscles offer no resistance whatever to passive movement, contractures do not occur, excepting when there occurs the unopposed action of healthy antagonistic muscles, and reflexes are lost. Extreme degrees of wasting occur in this type of paralysis, owing partly to disuse, but chiefly to the fact that the neuron body, the neutritional or trophic center for the fiber, is injured. Pathologic changes, therefore, take place in the muscles themselves, and form a true degenerative atrophy. The protoplasm first becomes granular and then fatty; it then breaks down and is absorbed. Its place is

taken by the connective tissue, which is both relatively and absolutely increased, so that in the course of time fibrous masses alone remain. Electric changes also occur. The muscles first cease to respond to the faradic current, and soon respond in an abnormal manner to the galvanic. Instead of short, sharp contractions, they react in a slow, wavy manner, ACC being equal to or stronger than KCC. Irritative lesions of this system may cause fibrillary muscular contractions and peripheral convulsions, of which laryngismus stridulus is a type.

Owing to the arrangement of the various tracts of the spinal cord a lesion involving its lateral half causes a peculiar combination of symptoms, frequently termed Brown-Séquard's paralysis.

It is met with particularly as a result of injuries (knife-thrusts and the like), though it may also be due to tumor or caries of the cord, to syphilis, or to any process causing compression of one-half of the cord. Such lesions intercept the motor impulses of the same side; the fibers having crossed in the medulla, the sensory fibers, conducting pain and temperature impressions, cross in the cord soon after entering, and hence these forms of sensation will be absent on the side opposite to the lesion; tactile sensation may also be abolished, but in some cases is not (*vide* Fig. 76). A lesion in the cervical cord above the arm-nuclei causes motor paralysis of both arm and leg of the same side (spinal hemiplegia) and sensory paralysis on the opposite side. If in the dorsal or lumbar cord, the leg on the corresponding side is paralyzed, while that of the other is anesthetic. Lesions are seldom strictly confined to one side of the cord, but overlap a trifle, so that there is apt to be some loss of power on the anesthetic side; this, however, may be due to the redecussation of a few motor-fibers at a lower level. The side of the lesion is hyperesthetic—a fact for which no satisfactory explanation has ever been advanced. Muscular sense is diminished or lost on the same side. Above the hyperesthetic region an area of anesthesia commonly exists, and above this, again, an area of hyperesthesia. The reflexes are increased on the side of the lesion (inhibition being removed), and the temperature of that side is usually higher. On the anesthetic side the motor power, reflexes, muscle sense, and temperature are all normal.

I. DISEASES OF THE PERIPHERAL NERVES.

NEURALGIA.

Definition.—Neuralgia (*nerve-pain*) is a term used to denote painful sensations that have the following characteristics: 1st. In their distribution they follow the course of the nerve-trunks or their branches. 2d. They show a tendency to shift from place to place. 3d. There are painful points (*points douloureux*) along the course of the nerve-trunks. 4th. Intermission and remission of the pain. 5th. There are no constant objective signs and no constant morbid changes in the nerves. True neuralgia is, therefore, a functional condition, and a symptom produced by a number of different causes.

Any nerve in the body may be affected. Quite often one can find no

definite cause of the neuralgia. A number of cases classified as such are due either to a mild perineuritis or to disease of the root ganglion, as in herpes zoster. In many of these it may be difficult to make a differential diagnosis if the process is not severe enough to interfere with the function of the nerve, and such cases may be classified with the neuralgias.

Etiology.—Anemia from any cause; toxemia, which may be of exogenous origin, as from lead, arsenic, mercury, copper, alcohol, tobacco, tea, coffee, any of the infectious diseases, or endogenous, as diabetes and nephritis; the deprivation of either morphin or cocain in a habitué; reflex, as the referred pains of visceral disease, thus a sciatica may be due to prostatic disease, neuralgic pain in the distribution of the sixth dorsal nerve may be due to heart disease, and so on;[1] heredity, neuropathic, gouty, and scrofulous individuals being especially liable; exposure to cold and wet; chronic vascular disease, especially arteriosclerosis. The early stages of involvement of nerve trunks from pressure of tumors, especially neuromata, aneurism, fractures, exostoses, displacements of bones, etc.; irritation, as from decayed teeth, etc.; general impairment of health, as in neurasthenia.

General Symptomatology.—The neuralgic attack may be of sudden or slow onset, with or without prodromata. When the latter exist they consist of a sense of uneasiness, perverted sensations, chilliness, and stinging or slight burning pains. The pain is usually of a paroxysmal, darting, boring character, radiating into the distribution of the affected nerves. In the intervals there may be either dull pain or freedom from it. In the case of the reflex neuralgias the area supplied by the affected nerve-roots is the seat of the pain. It is apt to be increased by movements of the affected parts, draughts, or excitement. Tenderness may be found over certain points, especially where the nerve emerges from a bony canal (points of Valleix); these are not always present. The affected part is usually hyperesthetic; occasionally, however, it is anesthetic, and may continue so for some time after an attack.

Reflex muscular contraction may be present in proportion to the intensity of sensory irritation. Vasomotor symptoms manifest themselves in the flushing or blanching of the affected part and in increased secretions, as sweating.

Trophic disturbances may result in temporary or permanent changes. To the former belong the herpetic and urticarial eruptions, while the latter groups include change of color in, loss of, or overgrowth of the hair, various changes in the skin (as pigmentation and morphea, and even ulceration, though in the latter instance there is probably a more profound pathologic change than that which we regard as the cause of neuralgia). Unless the attacks are severe or prolonged, however, the general system seldom suffers.

Diagnosis.—Neuralgia must be distinguished from *neuritis*, a mild attack of which, as has been said, it may resemble very closely. So much so, in fact, that in some cases the distinction cannot be made. Whenever the function of the nerve is interfered with, as shown by motor weakness,

[1] These relations have been studied and classified by Henry Head, *Brain*, 1893, p. 1; 1894, p. 23; 1896, p. 153.

constant diminution of sensibility, absent or diminished reflexes, tenderness along the nerve-trunk, and wasting of the muscles, neuritis is present. Headache is distinguished by the fact that the pain is more or less diffuse, and is not paroxysmal and shooting in type. It must be remembered that pain of a neuralgic type may occur in diseases of the cranial or spinal bones, tumor of the cord or its membranes, tabes dorsalis, multiple sclerosis, syringomyelia, meningomyelitis, basal meningitis, cervico-pachymeningitis, tumor of the cerebello-pontile angle, of the Gasserian ganglion, and inflammation of the sensory root ganglia, either cranial or spinal. The diagnostic points will be detailed under their respective headings.

Prognosis.—This depends upon the cause, whether removable or not; some forms, especially those of the fifth and sciatic nerves, are very intractable (pp. 1079, 1082). There is always the danger in chronic cases of a drug habit being formed.

Certain types require special mention. Treatment is given on p. 1083.

TIC DOULOUREUX.

This is a neuralgia involving one or more of the branches of the fifth nerve. It varies greatly in character and intensity in different cases, and in its severest forms is one of the most terrible of all the diseases of the nervous system.

The *pathology* is doubtful. In those cases that have been subjected to surgical operation, excised portions of the nerves sometimes were normal and sometimes contained a moderate number of degenerated fibers. In other cases in which the Gasserian ganglion has been removed and examined, considerable sclerosis of the blood-vessels has been detected, alterations in the axis-cylinders of the nerves, and occasionally moderate changes in the ganglion cells. It is not known exactly how these lesions produce the symptoms, but it is probable that vascular alterations are exceedingly important.

The *etiology* is various. Neuropathic heredity appears to play an important part. It is more frequently a disease of late than of early life. Peripheral irritation is frequently found, and when removed often results in complete cure. Among the structures disease of which is a frequent cause of tic douloureux are the nose and the cavities entering into it, and the mouth. Lesions of the former structures comprise chronic irritations, spurs, occlusion of the nasal openings, and suppuration. In the latter, abscesses at the roots of the teeth, irritated pulp, and occasionally, either non-eruption or malpositions of the teeth, are among the exciting factors. It is possible that eye-strain may also be an exciting cause.

The *symptoms* may be variable in extent, duration, and intensity. In the mild form there is only an occasional paroxysm, limited to one of the branches of the nerve, such as the type that occurs in acute coryza. In the more severe form there may be repeated paroxysms, the intervals varying from a few minutes to several days, involving the whole side of the face, and causing, for the time being, complete prostration on the part of the patient. The pain is often radiating, or of a rending or boring character, and sometimes so severe as to cause nausea. It is often accompanied by certain vasomotor or secretory phenomena, such

as flushing, perspiration, or excessive tear-production, and even in some cases more or less persistent edema of the skin. In some instances there may be more or less twitching of the facial muscles. The duration of the attacks varies greatly. The paroxysms may succeed each other frequently for long periods of time, or until the patient becomes insane or commits suicide; in other cases, after a few paroxysms, the attack passes off and may not return for months. In some instances the pain is persistent, and although in these cases it is rarely severe, the discomfort of the patient is sometimes greater. The painful points are: for the first branch, the supraorbital foramen; for the second, the infraorbital foramen; for the third, the mental foramen. Often the surrounding portions of the skin, particularly those where the periosteum is near the surface, are tender. If the disease continues for some time there may be trophic changes, such as the formation of ulcers, drying of the skin, and the appearance of gray hairs.

The *diagnosis* may have to be made from tumor involving the Gasserian ganglion or one in the cerebello-pontile angle. The former can be distinguished by the existence of anesthesia in the fifth-nerve distribution. The diagnostic points of the latter are given on p. 1188. Bulbar tabes (p. 1148) may also cause pain in the course of the fifth nerve. Neuralgic pain in front of the auricle and within the meatus may be due to disease of the geniculate ganglion or intumescentia gangliformis (p. 1103). A skiagram will show the existence of non-erupted teeth.

The *prognosis* depends largely upon the cause. In the so-called idiopathic cases it is exceedingly unfavorable.

The *treatment* consists first in the removal of the cause, if it can be found. The eyes, nose, and mouth should be carefully examined, and any source of irritation thoroughly removed. For the treatment of the paroxysms, if they are mild, the coal-tar analgesics may be employed. If severe, the only drug that is at all effective is morphin, which is preferably given hypodermically. Of course, in nearly all instances, if the disease is chronic, the patient becomes addicted to the use of this drug. Occasionally, the external application of a mixture of the ointments of opium and belladonna has proved of service. Strychnin, in full doses, combined with complete rest and liquid diet, aconitin, nitroglycerin in old people in full doses, have sometimes rendered the attacks milder and less frequent. They should be given hypodermically. Salicylates may also prove useful. The general health of the patient should be improved if possible, and the disease treated expectantly for some time. The galvanic current, the anode being placed over the painful areas and given without interruption, may sometimes prove useful. Formerly, section of the painful nerve branch was employed, and even resection of a portion of the nerve, but the results were either so trifling or so transient that in recent years the tendency has been to resort more frequently to the excision of the Gasserian ganglion. This is a serious operation, and the mortality is considerable; nevertheless, it is often wiser to employ it early rather than to delay until the patient is exhausted by long suffering. Spiller has suggested the section of the sensory root of the ganglion, and this operation gives the same results as excision of the ganglion. The most satisfactory method of treatment, if medical methods fail, is the injection of alcohol into the region of the foramina rotundum or ovale,

according to the branch affected.[1] By this plan relief for several years at least is obtained.

NEURALGIA OF THE NECK AND TRUNK.

The cervical branches of the dorsal and lumbar nerves are involved in this group.

1. *Cervico-occipital neuralgia*, occurring in the occipital and posterior parietal region, is apt to be quite severe, but when not due to spondylitis (the result of caries) or neoplasms the prognosis is fair. It is sometimes the result of direct pressure, as in carrying heavy loads on the neck and shoulders. The painful spot is found between the mastoid process and upper cervical vertebræ. Falling of the hair may also occur. This is much more apt to take place, however, when the occipitalis minor is involved, as it is said that the latter is generally a syphilitic neuralgia.

2. *Phrenic neuralgia* has been described, but is a rare condition. The pain is in the lower anterior thoracic region, at the points of insertion of the diaphragm.

3. *Intercostal Neuralgia.*—The middle intercostal nerves are most liable to be affected, and generally on the left side. The posterior dorsal branches are seldom involved. When specially severe and persistent, intercostal neuralgia may be a symptom of disease of the cord or its membranes, aneurysm of the aorta, neoplasms, or disease of the vertebræ or ribs. Traumatism and cold also give rise to it. This form of neuralgia is most common in women, the painful spots being at the extremity and at the middle of the ribs. The pain is of a sharp, lancinating character and radiates along the nerve. It is intensified by all movements of the chest; hence the affected side is more or less fixed. Herpes may develop, but in such cases it is probable that an inflammation of the root ganglion exists (p. 1132).

4. *Mastodynia* is really a variety of intercostal neuralgia, and occurs almost solely among women. It is very painful and gives rise to the development of tender "lumps" in the breast, simulating malignant disease. The paroxysms are often accompanied by vomiting.

5. *Lumbo-abdominal neuralgia* is not a common form. The pain is chiefly in the lumbar region, though the hypogastrium, genitals, and buttocks may also be involved.

NEURALGIA OF THE EXTREMITIES.

Cervico-brachial neuralgia occurs in the distribution of the four lower cervical nerves. When the condition is bilateral we should look for disease of the cord, especially tabes or membranes, for new growths, or for disease of the vertebræ. When unilateral, any of the causes already enumerated may be operative. The radial and ulnar nerves are more frequently affected than the median. The pain is most apt to be distributed along the whole course of the nerve, but painful points are found in the following situations: in the axilla; over the brachial plexus; on the shoulder, where the cutaneous branches of the circumflex nerve emerge through the deltoid muscle; about the middle of the outer surface of the upper arm; over the ulnar nerve; in the sulcus between the olecranon

[1] *Jour. of the Amer. Med. Assoc.*, Jan. 20, 1912, p. 155.

and epitrochlea; also near the wrist and at the bend of the elbow over the musculo-spiral nerve.

Femoral or *crural neuralgia* is a somewhat rare type that attacks the anterior surface of the thigh, the knee-joint, and the inner surface of the leg and foot.

Obturator neuralgia is distributed along the inner side of the thigh down to, and including, the knee-joint. This form is common in women subject to ovarian diseases, and may be mistaken for the pain of hip-joint disease.

Sciatica is such a common condition that a more extended description is necessary. The term is applied to pain in the course of the sciatic nerve, whether due to a pure neuralgia or a neuritis. It is probable that the majority of the cases are due to a perineuritis in which the presence of the exudation is not sufficient to cause marked interference with the nerve functions. It may be caused by any of the causes of neuralgia (p. 1078), but most cases are due to exposure, especially marked and sudden changes in temperature. Traumatism and prolonged pressure is also a frequent cause of the neuritic form. That due to pressure most frequently occurs after childbirth. It occurs most commonly in men during middle life. Some cases are due to chronic constipation. The painful points are in the gluteal region and the popliteal space or malleolar region, though tenderness may be elicited along the whole course of the nerve. The pain is sharp and shooting, or more often of a tearing variety. It may be localized to the region either of the sciatic notch or calf. The anterior crural nerve may also be involved. It is increased by putting the nerve on the stretch, which can be done by forcibly flexing the thigh on the body (Laseque's sign), and by motion after a period of rest. Rarely both nerves are involved, especially if due to toxemia. Pain may also be felt in the lumbar region. Fine or coarse tremors or spasms may be present. Herpes occasionally develops along the course of the nerve. In cases due to neuritis of any severity the Achilles jerk is absent, wasting and weakness of the muscles occur, and sensation in the foot and leg may be diminished or absent.

In making the *diagnosis*, it must be borne in mind that tabetic pains and neuritis, due to diabetes, may simulate *sciatica*. The pain of hip-joint disease and either inflammation or relaxation of the sacro-iliac synchondrosis may also simulate it. A careful examination will reveal the true condition. The same may be said of tumor involving the cord and its membranes (p. 1157). Lesions of the cauda equina also cause sciatic pain which is usually bilateral, and, in addition, there will be atrophy and paralysis and involvement of the sphincters (p. 1159). Intra-pelvic growths should also be borne in mind. The pain of *intermittent claudication* occurs only after exercise, is not limited to the course of the nerves, and the posterior tibial and dorsalis pedis arteries will not be palpable. In making a *prognosis* it must be borne in mind that it is often rebellious to treatment, but most cases ultimately recover. Relapses are apt to occur.

The most useful plan of treatment is absolute rest in bed, with the limb kept perfectly still by means of sand-bags or a long splint, with the application of heat along the course of the nerve, and the galvanic current, the anode over the sciatic notch and the other at the foot, applied daily for ten

minutes without interruption. High-frequency and the static-wave current are useful in many cases. Full doses of the salicylates should be given internally. Local applications of one of the salicylic acid preparations, as a 25 per cent. ointment of mesotan, may also be of service. In old people with arterosclerosis full doses of nitroglycerin and potassium iodid may give relief. In severe chronic cases electricity, as mentioned above, and counter-irritation, preferably by means of a succession of small fly-blisters along the course of the nerve, is of great value. When all else fails, stretching the nerve after exposing it often cures, but sometimes aggravates, the symptoms. The bowels should always be kept free. Deep injections of cocain, eucain, thein, ether, or chloroform are sometimes used, and even distilled water may give relief when injected into the nerve. The use of guaiacol (♏i–ij—0.066–0.1332) in association with chloroform (♏x—0.666) by this method has yielded very encouraging results in my hands.[1] Excellent results have recently been reported from deep perineural injections of salt solution.[2]

Neuralgia of the Genitalia and Rectum.—These varieties are not met with frequently. The former is sometimes a symptom of stone, prostatic disease, or stricture, and in women ovarian and uterine neuralgias are generally hysteric manifestations. Coccygodynia, unless of traumatic origin, is almost solely found in women. The pain in the region of the coccyx is excruciating at times, and may even call for operation.

Visceral Neuralgia.—As implied by the name, these forms are neuralgias resident in the various viscera. They most frequently attack the stomach or bowel, and are recognized as colic. Other viscera may also be involved (liver, kidney). Such pains may be simulated by tabetic crises (p. 1147).

Treatment of Neuralgia.—The first requisite in the treatment of neuralgia is to ascertain whether it is due to local or general causes. That of the former class may be caused by a cicatrix, neuroma, aneurysm, neoplasm, or by caries or traumatism; and the treatment must necessarily be directed toward the removal of the cause when possible. When the fault is a general one, the neuralgia may occur either as the immediate result of the systemic disease, or remotely, as the result of the altered blood-state (anemia). This is particularly well illustrated by an attack of malaria, in which it is obvious that success can only be obtained by attention to the underlying cause. It is sometimes necessary to use an analgesic, of which morphin is certainly the best. Its therapeutic value is most decided when the drug is given hypodermically, and if injected directly over the track of the painful nerve (*e. g.*, supraorbital branch of the fifth), it not only affords immediate relief, but also obviates recurrences of the painful paroxysms in many instances. It is, however, scarcely necessary to urge the exercise of caution, for the morphin-habit is readily formed in these cases. The following may also be used: antipyrin, phenacetin, codein, veratrum viride, aconite, also counter-irritants and vesicants, including the galvanic current, which is applied by placing the anode over the tender spots if they exist, otherwise over the seat of the pain. A rapidly interrupted faradic current applied over this area and the high-frequency current may also prove valuable. The general

[1] "The External and Internal Use of Guaiacol," *Therapeutic Gazette*, Mar. 15, 1895.
[2] D'Orsay Hecht, *Jour. Amer. Med. Assoc.*, Feb. 6, 1909, p. 444.

tone of the system must be attended to, bad habits prohibited, the state of the bowels regulated, and the eyes examined and corrected for errors of refraction. Rest is a valuable adjunct to any form of treatment. In severe neuralgia of either the brachial or lumbosacral plexus division of the posterior roots has been practised with varied success.[1]

NEURITIS.

Definition.—An inflammation of a nerve or of its fibrous envelope. It may be confined to a single nerve, termed *local neuritis*, or a number of nerves may be affected, when it is termed *multiple* or *poly-neuritis*.

Pathology.—The inflammation may be chiefly confined to the sheath of the nerve (perineural) or may, in addition, involve the deeper portions of the sheath (endoneurium), in which an accumulation of lymphoid elements will be found between the nerve-bundles. This form is known as *interstitial neuritis*, and is the condition usually found in the localized form. The nerve will be found to be swollen and red in color, but the nerve-fibers do not appear involved. Eventually, however, changes resembling those found in Wallerian degeneration may occur, the myelin becoming fragmented, the nuclei in the sheath of Schwann increasing in number, the nuclei of the internodal cells becoming swollen, and the nerve-fibers undergoing granular degeneration. In *parenchymatous neuritis*, the condition found in multiple neuritis, the nerve-fibers are primarily and principally affected. Changes like those met with in Wallerian degeneration described above are met with, but the sheath shows little evidence of inflammation.

Etiology.—(*a*) *Local neuritis* may be due to—(1) Exposure or cold (the so-called *rheumatic neuritis*). (2) Extension of inflammation from neighboring parts. (3) Traumatism—wounds, compression, excessive stretching resulting from fractures or dislocation, electrical shock. (4) Microbic and autogenetic poisons. (5) Arterio-sclerosis. (6) Stoop shoulders, which cause compression of the axillary structures between the humerus and ribs, causing either brachial or ulnar neuritis.[2]

(*b*) *Multiple neuritis* may be due to—(1) Poisons of extrinsic origin—carbon monoxid, alcohol, carbon bisulphid, lead, arsenic, mercury, ether. (2) Poisons resulting from the infectious fevers (typhoid, diphtheria, variola, typhus, leprosy, beri-beri, measles, syphilis, tuberculosis, septicemia, malaria, influenza. (3) Poisons produced within the body, as from gout, rheumatism, diabetes, and pregnancy. (4) Cachexias, anemia, carcinoma, arteriosclerosis. (5) Cases arise in which no definite cause can be ascertained; these are the so-called *idiopathic* or spontaneous cases.

Symptoms.—(*a*) **Focal Neuritis.**—In localized neuritis the symptoms vary according to the function of the nerve involved. In the case of a sensory nerve there is pain, usually of a boring or shooting character, along its course and distribution. There is also tenderness on pressure along the nerve. The skin is generally hyperalgesic (though tactile sen-

[1] *New York Medical Jour.*, Aug. 3, 1907, p. 192, and *Jour. Nerv. and Ment. Dis.*, Sept., 1907, p. 589.

[2] Goldthwait, *Jour. Amer. Med. Assoc.*, Sept. 11, 1909, p. 852.

sation is often lowered), may be reddened, sometimes edematous, and local sweatings may occur. In the more chronic cases trophic symptoms eventually arise, as glossiness of the skin and an impaired growth of the nails. When a motor nerve bears the brunt of the attack there is more or less impairment of motion, even amounting to paralysis; and ultimately wasting of the muscles and even reactions of degeneration take place. When both motor and sensory nerves are simultaneously involved the symptoms will necessarily partake of a mixed character. Many cases of a mild type occur in which the symptoms consist of pain, tenderness on pressure over the affected nerves, some impairment of motion, slight atrophy, and a diminished contractility to the faradic current. The constitutional symptoms are, as a rule, of little moment. The symptoms of neuritis affecting special nerves are detailed on pp. 1090–1117.

(*b*) **Multiple neuritis** is an involvement of the peripheral nerves in various parts of the body, affected simultaneously or in quick succession, and due to endogenous or exogenous poisons.

Among cases due to poisons of extrinsic origin is *alcoholic neuritis.* This is the most common type of multiple neuritis. It results from spirit-drinking in moderate amounts and continued over a long time. The onset is generally slow, being preceded by gastric catarrh, insomnia, and particularly numbness and tingling of the extremities. A rapid, weak heart and a tendency to sweating on exertion may also be present. Weakness, especially of the extensor muscles of the wrists and dorsal flexors of the feet; pain and muscular tenderness, the latter being most prominent in the muscles of the calf, where it is usually an early symptom, are soon noticed. As a rule, the legs are first affected, and in mild cases the arms may escape. As a rule, however, all of the nerves supplying the extremities ultimately become more or less affected, and, in extreme cases, cranial nerves may also suffer. The reflexes are lost (rarely the knee-jerks may be increased in the early stages), muscular atrophy becomes marked, and pain and tenderness very severe. Rarely loss of control of the bladder and rectum take place. Fever is seldom noticed. More or less impairment of pain, tactile, and muscle sense may also be present. The early loss of power in the extensor muscles soon causes double wrist- and foot-drop, and the gait, owing to the effort to make the toes clear the ground, is of a peculiar high-stepping variety, known as "steppage gait."

The cutaneous reflexes are preserved unless the anesthesia is marked. In less severe cases a certain amount of incoördination may be present. When this is the case, the absence of the knee-jerk, the loss of muscular sense, occurrence of ataxia, and the pains in the extremities simulate locomotor ataxia, and the term *pseudo-tabes* has been applied to the condition. Vasomotor and trophic symptoms appear, and in some cases the special senses are involved (impairment of vision, amblyopia, limitation of the color-field). The mental symptoms are important. They may be so slight as to consist merely of loss of memory, irritability, perhaps an hallucination or illusion (particularly after nightfall, and especially if the patient has had insomnia), or they may be very severe, consisting of marked mental impairment, hallucinations, delusions, disorientation, etc., a symptom group known as Korsakow's psychosis. The duration of an attack varies from a few weeks to a year or more.

Arsenic neuritis differs from the above in that the mental symptoms are generally absent. The onset may be much more abrupt and the course is usually shorter.

Carbon bisulfid neuritis occurs chiefly in workers in rubber factories and imitation silks. There are noted intense frontal headache, giddiness, marked excitability, muscular cramps, and possibly convulsions. *Saturnine neuritis* is confined to motor nerves, and especially to those of the upper extremities, the posterior interosseous branch of the musculospirals being especially liable to be involved, causing double wrist-drop. Any or all nerves may, however, become affected. Peculiar features are the usual absence of pain and tenderness, and the escape of the supinator longus and extensor ossis metacarpi pollicis muscles. Lesions of the anterior cornua are more likely to occur in saturnine multiple neuritis than in any of the other varieties. Delirium (lead encephalopathy), optic neuritis, and convulsions may occur, but are not common symptoms.

Cases due to an attack of some infectious disease may be local or multiple, and generally present the same symptoms of neuritis due to any other cause. *Recurring Multiple Neuritis.*—A few cases have been reported in which attacks of more or less widespread paralysis, due to neuritis, have recurred. *Senile neuritis* occurs in old age, and is probably a degeneration due to arteriosclerosis. The symptoms develop gradually and consist of weakness and numbness of the limbs, especially the lower; absent knee-jerks, sometimes slight atrophy and diminished response to the faradic current. Cranial nerves may also be affected.

Spontaneous or the so-called *idiopathic neuritis* does not differ from the general type of the disease, except that no cause can be discovered to account for it.

Beri-Beri.—This is a form of multiple neuritis, occurring endemically, chiefly in the islands of the Pacific Ocean and in Asia. It is especially prevalent in Japan and the Philippines. Sporadic cases are met with in increasing frequency in Europe and America, brought on ships from the Orient. Its exact nature is not known. It is probably an infectious malady, which, to a certain extent, may be communicated either by direct contact or by living in rooms where a case has previously been.[1] It is especially apt to occur where many are crowded into a limited space, as jails, barracks, ships, etc. Moisture and heat favor its development. An exclusive rice diet may act as a predisposing factor.

The essential feature of the pathology is the changes in the nerves; these are inflammatory and degenerative. Degeneration in the muscles also occurs, and not infrequently serous effusion. A variety of clinical types have been recognized. Of these the most important are the wasting and the wet forms. The onset may be rapid or more gradual. In the first type there is loss of power in the limbs, wasting of the muscles, and more or less emaciation. Subjectively, there are pain and paresthesiæ in the limbs, tenderness in the muscles and over the nerve trunks. The patients also complain of weakness, dyspnea, and palpitation. The wet form is characterized by the earlier or later occurrence of general anasarca, with effusion into the serous cavities. The swelling may be enormous and obscure the muscular wasting. Sometimes the

[1] *Brain*, 1903, p. 488.

dyspnea and palpitation of the heart predominate. The prognosis is usually favorable, but the course is prolonged and recurrence is not unusual. In the cardiac form death may occur in a few days.

Diagnosis.—This does not present any difficulty, as a rule. In the early stages, *acute anterior poliomyelitis* and *acute ascending paralysis* may be mistaken. In the former constitutional symptoms usually precede by several days the development of the paralysis, which, when it occurs, is usually more or less general, to be followed by a rapid improvement in most of the affected limbs. The paralysis in multiple neuritis develops progressively. If pain and tenderness occur in poliomyelitis, they consist of a general hyperesthesia, and are not confined to the affected nerve-trunks, as in neuritis.

In *ascending paralysis* there are no sensory symptoms, there is neither muscular atrophy nor electric change, and the order in which the paralyses supervene differs from that of peripheral neuritis.

Cases of pseudo-tabes are sometimes confounded with *locomotor ataxia*. The main points of differentiation are included in the following table:

Pseudo-tabes.	Locomotor Ataxia.
The course is shorter, and often results in recovery.	The course is progressive from bad to worse, and chronic in nature.
Pain is never of the fulgurant type.	Fulgurant pains often are present. Pain-crises are almost diagnostic.
There is tenderness over the nerve-trunks.	There is no tenderness over the nerves.
Sensory disturbances are more marked (tingling and numbness).	Sensory disturbances are less marked.
Argyll-Robertson pupil is absent.	Argyll-Robertson pupil is present.
There is a "foot-drop," with the typical "steppage" gait.	No "foot-drop." The toes are raised, and the foot is brought down flatly, with the heel first.
Paralysis is often present.	There is no actual loss of power.

The distinguishing symptoms from progressive neural atrophy are given on p. 1088.

Prognosis.—Peripheral neuritis may terminate in one of the following ways, according to Drs. Gibson and Fleming[1]: 1. In complete recovery; 2. With damaged peripheral nerves; 3. With injury to the central nervous system, especially of the cells in the anterior horns; 4. In death from failure of the organic centers, especially that of respiration. The prognosis is generally good, though in the acute variety (from any cause) it should be guarded, and occasionally is grave. Exposure and chill, alcohol, diphtheria, and beri-beri give rise to the most serious types, and often cause death by failure of the heart or respiration or by coagula in the vessels. Mild cases may entirely recover in a few weeks, while severe ones often require a year or two.

Treatment.—First ascertain the cause and, if possible, remove it. It may be unwise in alcoholic cases to stop the alcohol suddenly, but each case must be judged on its merits. Rest is very important, and all sources of worry should be stopped. Locally, anodynes may be employed and the part wrapped in cotton wool. Ointments of either ichthyol and belladonna, or some of the salicylic acid preparations for external use, as mesotan, are often of service. The pain can often be relieved for several hours by the application of the galvanic current,

[1] *Edinburgh Hospital Reports*, vol. iii.

applied without interruption down the limb. In acute cases, especially in the earlier stages, the salicylates are valuable. The general health should be toned up by strychnin and tonics, and by nourishing but easily digestible food. Further medication will depend upon the etiology, quinin being demanded in malarial, iodids and other measures to eliminate the lead in lead cases. As soon as the acute cases have subsided, massage and passive movements should be begun, galvanism applied to the muscles, and warm-water or sulphur baths administered. Care should be taken to prevent deformity due to the unopposed action of antagonistic muscles, as when foot- or wrist-drop is present.

PROGRESSIVE NEURAL MUSCULAR ATROPHY.

(*Hoffman*).

(*Progressive Neurotic Muscular Atrophy; Charcot-Marie-Tooth-Type of Progressive Muscular Atrophy; Peroneal Type, Gowers.*)

Definition.—A degenerative process, apparently commencing in the nerves, and characterized by muscular degeneration, with subsequent contractures, sensory disturbences, and a loss of the reflexes.

Pathology.—Sclerosis of the posterior columns of the cord, slight degeneration of the pyramidal tracts, alteration of the columns of Clarke, atrophy of the cells in the anterior horns of the cord, degeneration of the peripheral nerve-fibers and of the intramuscular branches, atrophy of the muscle-fibers, and chronic spinal meningitis have been found by different observers in cases of this disease.

Etiology.—Heredity seems to play an important part in the causation of the disease, which may either occur in successive generations of a family or affect several members of the same generation. Sporadic cases occasionally occur for which it is impossible to trace any ancestral influence, though, as the disease has been known to skip a generation, it is not impossible that such cases are still hereditary. Males are much more frequently affected than females, and the disease almost invariably commences between the ages of ten and twenty years.

Symptoms.—As the name implies, muscular wasting usually begins in the muscles of the feet or hands, either the peronei, the common extensors of the toes, or the small muscles of the foot itself, or else in the muscles of the thenar and hypothenar eminences and the interossei. Usually the atrophy is symmetric. In the feet it leads to an early development of club-foot, which is most pronounced when the extremity is at rest. Very early the atrophy of the small muscles causes the toes to assume the claw position, and the atrophy of the peroneals causes foot-drop, so that in walking the foot is dragged along the ground. In the later stages the foot becomes permanently fixed in a position of equino-varus or valgus. The hands have the characteristic appearance given by a flattening of the ball of the thumb and middle finger. The interosseal grooves also become deeper and the fingers gradually assume the claw-like position ("*main en griffe*"). The disease extends slowly upward, involving the muscles of the calf and forearm; the muscles of

the thighs, upper arms, and trunk usually escape. The affected muscles usually show distinct fibrillary twitchings. When electrically examined, the muscles either show a marked diminution in reaction to the galvanic and faradic currents, or distinct reaction of degeneration can be elicited. Similar electric changes are also found in the nerves. Mechanic excitability of the muscles is considerably diminished, these changes being found also in the muscles that are apparently healthy. The tendon-reflexes are usually absent, although in the early stages, when the muscles of the thigh are still unaltered, the knee-jerk may be merely diminished. Sensation is sometimes unaltered, but ordinarily there is some diminution of sensibility in the peripheral parts of the limbs. Often there are paresthesiæ and, occasionally, pains of considerable intensity. The general condition of the patient, however, remains excellent. The vegetative organs are unaffected and nutrition is, therefore, intact.

The **diagnosis** can be made from other forms of progressive muscular atrophy (particularly the type "Aran-Duchenne," p. 1142) by the sensory disturbances, the fact that the proximal muscles escape, and the early age at which the symptoms appear; from the *muscular dystrophies* by the presence of fibrillary tremors, sensory symptoms, and changes in the electrical reactions and the escape of the proximal muscles; from multiple neuritis by the absence of tenderness over the nerve-trunks; from acute poliomyelitis by the mode of development.

The **prognosis** is good as regards life, but unfavorable as regards cure or even improvement. The course of the disease is extremely slow.

The **treatment** employed in the other forms of amyotrophy may be tried, but so far nothing has succeeded in staying the course of the disease.

A type of disease closely allied to the preceding has been described by Déjerine under the name of "*infantile hypertrophic and progressive interstitial neuritis.*" The muscular symptoms were the same, but there were in addition ataxia, lancinating pains in the limbs, considerable sensory disturbances, Romberg's sign, myosis, with slow or absent pupillary reflexes and nystagmus. In addition to these a peculiar symptom in his case was the enormous hypertrophy of the nerve-trunks, which could be felt under the skin as large, firm cords. Pathologically the muscles showed degenerative changes and the nerves a pseudo-hypertrophy due to the enormous proliferation of the connective tissue and degeneration in the posterior columns of the spinal cord. The disease appears also to be due to old hereditary influence, the first 2 cases described being a brother and sister.

NEUROMATA.

NEUROMATA, or tumors of nerves, have been described as (*a*) true and (*b*) false.

(*a*) *True neuromata* consist of medullated or non-medullated nerve-fibers (the myelinic and amyelinic varieties—Virchow), and rarely of ganglion-cells also.

(*b*) *False neuromata* contain no nerve-elements. The growth is situated on the nerve-trunk itself, and consists of either fibrous, myxomatous, gliomatous, or sarcomatous tissue.

Neuromata have also been classified according to their situation as (1) Stump neuromata, or bulbous nerves; (2) Subcutaneous neuromata, or *tubercula dolorosa;* (3) Nerve-trunk neuromata; (4) Plexiform neuromata.

(1) *Stump neuromata* develop on stumps or on the ends of divided nerves as the result of traumatism. They may consist of fibrous tissue, but are usually myelinic.

(2) *Subcutaneous tumors*, or *tubercula dolorosa*, are painful, as the latter word implies, and are apt to be multiple. In individuals so afflicted nerve-trunk neuromata may coexist.

(3) *Nerve-trunk neuromata* are usually multiple. In one case quoted by Gowers as many as 3020 were found. They may be true or false. In the former case the nerve-fibers are less apt to be interfered with than in the heterologous growth.

(4) *Plexiform neuromata* consist of beaded and tortuous, interlacing neural cords. They are usually congenital.

Etiology.—Neuromata may be due to traumatism. When multiple, however, they are usually hereditary, occurring in families of a neurotic or strumous diathesis. They are most commonly found in men.

Symptoms.—There may be none. When present their character necessarily depends on the nature of the nerve involved and whether the lesion is an irritative or destructive one. More or less pain, numbness or tingling, paresthesia, and palsy are among the most common. Various reflex manifestations have been described, and epileptiform convulsions have been attributed to their presence, probably unjustly.

Treatment.—Apart from anodynes, operative measures are alone of value, except when the tumors are the result of syphilis, as occasionally happens; in such cases specific treatment must be employed.

It must not be forgotten, however, that stump neuromata may occur in those hereditarily predisposed, in which case, as Bowlby has pointed out, their removal will almost surely be followed by a return.

DISEASES OF THE CRANIAL NERVES.

OLFACTORY NERVE.

THE following morbid conditions have been described in connection with the sense of smell:

(*a*) *Hyperosmia* or *Olfactory Hyperesthesia.*—The sense of smell is abnormally acute, so that objects, and even persons, can be recognized by this means. It occurs in hysteria and insanity.

(*b*) *Parosmia* (perverted sense of smell) may occur for one or for many odors and is often associated with an obtunding of the normal sense.

(*c*) *Subjective sensations* of smell are due to the same causes as the above. An olfactory aura may precede an attack of epilepsy. Olfactory hallucinations occur occasionally in the insane, and in irritative lesions of the uncinate gyrus (Fig. 69).

(*d*) *Anosmia* or *olfactory anesthesia* (loss of the sense of smell) may be caused by—(1) injury to the peripheral filament by local disease of the nasal mucous membrane. (2) Injury to the nerve-trunk or bulb, by blows on the head, basal fractures of the skull, bone-disease, and

meningitis. Anosmia may occur during locomotor ataxia. Pungent and powerful odors have been said to cause loss of the sense of smell, due to excessive stimulation. There may be a congenital absence of the olfactory nerves. (3) Centric lesions, as tumors in the region of the uncinate gyrus (Fig. 69). Unilateral anosmia has been described as part of a hemianesthesia, due to a lesion in the posterior part of the internal capsule and in hysteria.

In testing the sense of smell it is advisable to use aromatic oils, as they only stimulate the olfactory nerve, while ammonia and such strong substances also stimulate the fifth nerve. It is obviously necessary to make a rhinoscopic examination.

Treatment is generally unsatisfactory, though the cause must be removed when possible. When the disturbance is due to some general condition, as hysteria, it may of course be disregarded, as it will improve with the disease.

DISEASES OF THE RETINA, OPTIC NERVE AND TRACT.

The Retina.—Hemorrhage into the retina may be venous or arterial, single or multiple, monocular or binocular. It may be part of a general vascular change; occasionally it occurs during parturition, but more often at the menopause; it may be an indication of renal trouble or of some primary or symptomatic anemia, as in leukocythemia, pernicious anemia, or malaria. Hemorrhage is prone to occur also in depraved nutritional conditions, in purpura, and in scurvy.

More or less complete loss of vision develops in these cases, either suddenly or gradually; an ophthalmoscopic examination is necessary to make the diagnosis. If the hemorrhage is superficial, the eye-ground is red and swollen; if deeper, the blood escapes between the fibers of the retina, spreads them out, and assumes a flame-shaped appearance.

Retinitis.—Three forms of this condition are commonly described—(1) albuminuric, (2) syphilitic, and (3) pigmentary.

(1) *Albuminuric retinitis* is probably not a distinct affection, but part of a general fibro-vascular change associated with nephritis. The failure of vision may precede the advent of albuminuria, but more often the two conditions are coincident. It occurs in chronic nephritis, especially in the interstitial variety.

The retinal changes, according to Gowers, are either *hemorrhagic* or *degenerative*. In the former the arterial blood occupying the interstices between the fibers assumes a striated or feathery aspect, while in the degenerative form white patches of fatty degeneration or deposits of cholesterin are dotted over the fundus; they may also be grouped about the macula lutea, or around the disk. Occasionally the latter appears swollen, owing to the effusion of serum into the fiber-layer.

(2) *Syphilitic retinitis* generally occurs in the later stages of acquired syphilis, and particularly in neglected cases. Failure of vision directs attention to the eye-ground, which is found to have either scattered or uniformly distributed whitish or slightly opalescent filmy patches upon it. The vitreous may be turbid also. Retinitis is far less common than choroiditis or chorio-retinitis.

(3) *Pigmentary retinitis* is essentially a chronic process, usually attacking young adults, and, as a rule, more than one member of a family. It may also occur in inherited syphilis and in low grades of vitality.

The affected parts receive a deposit of pigment which specially follows the course of the main arteries. At the same time a circumferential annulus of pigment forms. This gradually encroaches more and more upon the disk, until finally atrophy ensues.

Among retinal affections occur also—

(*a*) **Toxic Amblyopia.**—This is due, as a rule, to tobacco or alcohol, and more rarely to certain drugs and lead-poisoning. Failure of vision is gradual and progressive, though it rarely reaches absolute blindness. The center of the field is chiefly affected, and a central scotoma for red and green exists; this is said to be caused by a chronic neuritis beginning in the fibers that are distributed to the macula lutea. It is believed to be due to a retrobulbar neuritis.

(*b*) **Hemeralopia,** or *day-blindness*, may either be functional or a symptom of some retinal affection—*e. g.*, hyperesthesia or albinism, or the result of central cataract. Objects can either not be seen at all or only indistinctly during the day or in a strong artificial light; but at night vision is excellent.

(*c*) **Nyctalopia,** or *night-blindness*. In this condition vision may be normal during the day or in a strong artificial light, but after nightfall or in a darkened room objects can be seen only with difficulty or not at all. It is usually associated with syphilitic retinitis.

Optic Nerve.—The important pathologic conditions of the optic nerve, especially with reference to diseases of the nervous system, are: (1) *neuritis* and (2) *atrophy*. (1) Neuritis is met with in two forms: first, where the lesion is not visible at the intra-ocular end of the nerve (orbital optic neuritis or retrobulbar neuritis, see Toxic Amblyopia), and, second, where the lesions are visible at the intra-ocular nerve-ending. The latter is the more important, and is also known as papillitis. Optic neuritis may be caused by the acute infectious diseases, syphilis, lead, alcohol, uremia, anemia, menstrual disorders, exposure to cold, rheumatism, injuries, disease of the orbital region, and possibly intranasal lesions. Rarely it is congenital, and mild forms may be caused by refractive errors. When the nerve head projects markedly into the interior of the eye, it is known as "choked disk," or from the condition present, papilledema. This is not a true neuritis, but is due to mechanical causes. The early injection with stasis of the retinal vessels, edema, and elevation of the nerve-head, and final cellular infiltration, with new tissue formation leading to atrophy, are due to distention of the sheath of Schwalbe by obstructed cerebrospinal fluid.[1] Neuritis at times may be associated. Kidney disease may cause a similar condition. Intracranial lesions are the most frequent causes. Of these, brain tumor ranks first. Others are meningitis, cerebral abscess, cerebral and meningeal hemorrhage, thrombosis of the cavernous sinus, chronic hydrocephalus, serous meningitis, and aneurysm of the internal carotid. Vision may not be lost for some time. If the process is not arrested, consecutive atrophy occurs.

(2) *Optic Atrophy.*—This may be *primary*, when it is usually associated with disease of the spinal cord and brain, as tabes dorsalis, paretic dementia, and multiple sclerosis, *secondary*, when it results from pressure more or less directly applied to the optic chiasm or tracts, and *consecutive*, when it follows a previous neuritis or "choked disk." There is also

[1] Bordley and Cushing, *Jour. Amer. Med. Assoc.*, 1909, lii., p. 353.

an hereditary form known as Leber's disease, and that which occurs in amaurotic family idiocy (p. 1248).

In any case there is alteration of the field of vision, color perception is abnormal, and there is more or less dimness of sight. In the hereditary form the disk is less white than in the other, and the vessels are almost normal in appearance.

The Optic Tract.—The lesions of the optic tract are important rather on account of their situation than their nature. They may exist without corresponding changes in the retina, although when they have lasted for a long time there is often some secondary atrophy resulting from a descending degeneration of the optic nerves. Lesions of the chiasm usually affect the decussating fibers, causing blindness of the nasal halves of the retina, and, in consequence, temporal hemianopsia. This condition occurs in basal tumors especially of the hypophysis, and has therefore been observed in acromegaly, in tuberculous basal meningitis, and in hydrocephalus. Lesions of either optic tract, if complete, causes homonymous bilateral hemianopsia; if incomplete, there is irregular disturbance of the visual field, sometimes bilateral, sometimes unilateral. It may be involved in hemorrhage, tumors, softening or basilar meningitis; ordinarily other structures are also involved, giving rise to symptoms of focal disease. Lesions anterior to the anterior corpora quadrigemina usually cause more or less destruction of some of the other cranial nerves, with the production of ocular palsies, or disturbances of the other special senses, or anesthesiæ or neuralgias of the face. A very valuable sign, that, however, cannot always be elicited, is the failure of the pupil to contract when light is thrown upon the blind half of the retina. This is explained by supposing that the pupillary reflex center is situated in the anterior corpus quadrigeminus, lateral geniculate body, and pulvinar. If the lesions affect the optic thalamus or the internal capsule, hemiplegia and hemianesthesia are also often present or may form the most important symptoms. Lesions posterior to the anterior corpora quadrigemina produce hemianopsia without disturbance of the pupillary reflex. These lesions are divided into two groups, the cortical and the subcortical, and they may be of two varieties, either irritative or paralytic. The irritative lesions give rise to hallucinations of sight, which may vary from the scotomata of migraine to most complex visions. Paralytic lesions ordinarily lead to hemianopsia. Occasionally curious symptoms are produced, the visual field being sometimes irregular, while at others only certain elements of sight are affected, cases having been reported in which the hemianopsia only involved the recognition of colors, not of form. In all these cases the pupillary reflexes are not affected. Bilateral lesions do not always lead to total blindness; sometimes the macula lutea escapes and the patient is able to see only by direct fixation. Occasionally a single lesion will produce total blindness in one eye, but this is rare, and no satisfactory explanation has been found to account for it. Cortical lesions are those involving the occipital lobe. The center of visual perception appears to be in the cuneus and calcarine fissure; if this is destroyed on both sides, blindness occurs. If on one side, lateral homonymous hemianopsia (Fig. 77). The center for the recognition of the object seen is apparently upon the convex surface of the occipital lobe, probably in the second and third convolutions, but it may extend also into the temporal

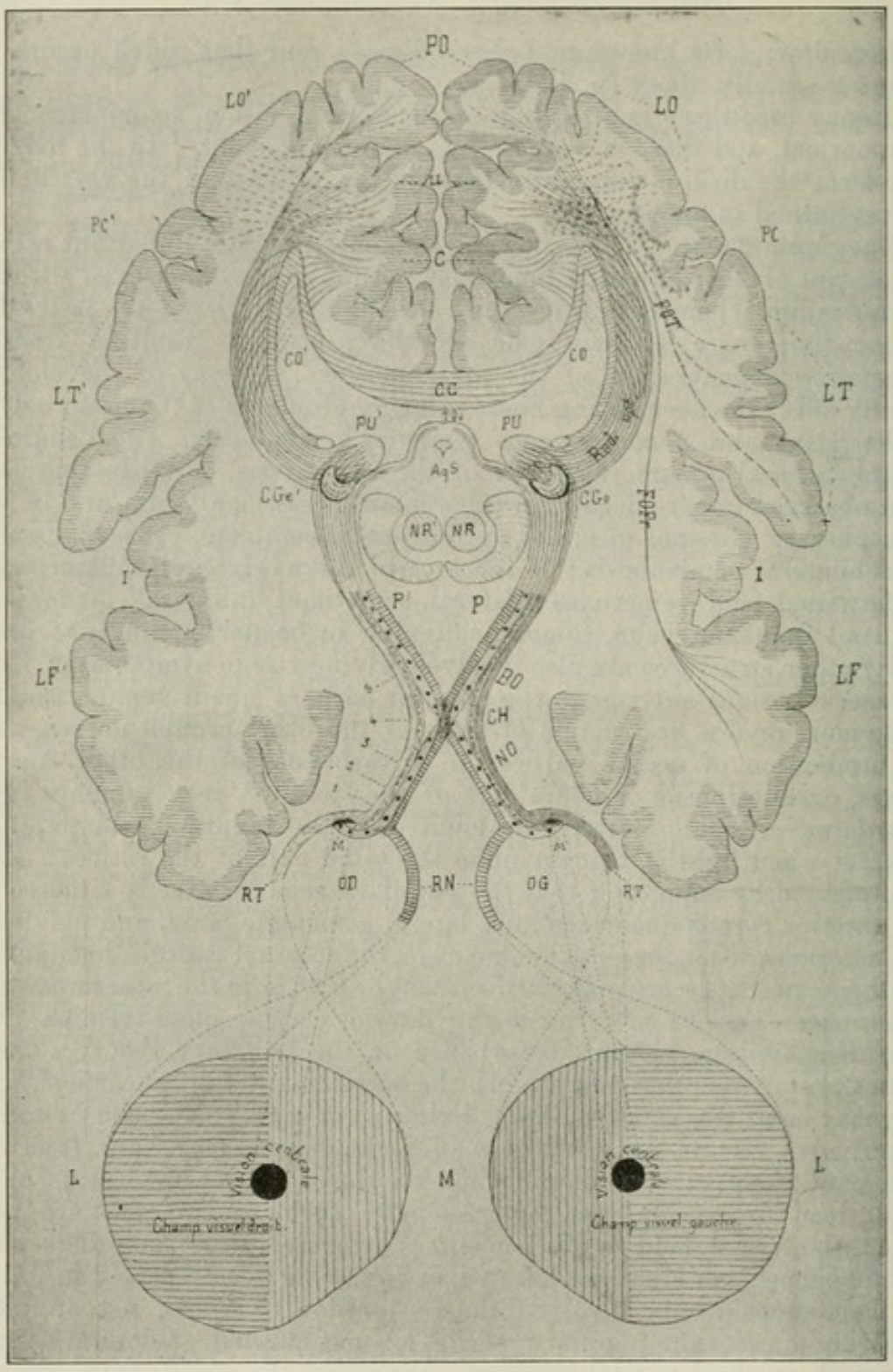


FIG. 77.—Diagram of the visual apparatus (after Vialet): *LO, LO'*, occipital lobes; *C*, cuneus; *Rad. opt.*, optic radiation; *TQa*, anterior corpus quadrigeminus; *PU, PU'*, pulvinar; *CGe*, external geniculate ganglion; *BO*, optic tract; *CH*, optic chiasm; *NO*, optic nerve; *OD*, right eye; *OG*, left eye; *RN*, nasal half of retina (supplied by the opposite hemisphere); *RT*, temporal half of the retina (supplied by the homolateral hemisphere); *M*, macula lutea. A total transverse lesion at 1, 2, or 3 would cause total blindness of the right eye. A lesion at 4, destroying the central part of the chiasm, would cause blindness of the nasal halves of the retinæ, and therefore bitemporal hemianopsia. A lesion at 5 would cause blindness of the right halves of the retinæ, and therefore left homonymous hemianopsia. The pupillary reflex would be lost in the affected half of the eye in all these cases. A lesion in the optic radiation would cause symptoms similar to those of the corresponding optic tract, excepting that the pupillary reflex would be preserved. Lesions of the cortex cause various disturbances of vision according to the part affected.

lobe. When this is destroyed the patient can see either objects, words, letters, or symbols, as the case may be, with which he was once familiar, but fails to recognize them; this is called *mind-blindness* or visual agnosia. Hemianopsia is very frequently merely a temporary symptom, and as such it may occur in uremia, apoplexy, migraine, and certain intoxications, especially that of lead. It may also occur in brain tumor, and disappear if the pressure is relieved, as by trephining. It is a permanent symptom only when the visual tract has been involved by some destructive lesion. If the patient is perfectly conscious and intelligent, it is not difficult to recognize it; nevertheless, its presence can often be detected in young children and in those who are only partially conscious or unable to speak. This can be accomplished by taking a bright object, placing it behind the head, and then bringing it forward slowly, first on one side and then on the other. It will then be noted that the patient perceives it on the hemianopsic side only when it has been brought to the middle line, whilst when moved on the other side the eyes will turn toward it when it is still a considerable distance from this point. Another method is to bring a blunt object (a wisp of cotton) very nearly in cantact with the cornea, first on the one and then on the other side of the median line. The palpebral reflex will occur upon the normal side whilst the object is still some distance away; on the blind side only when it has come in contact with the conjunctiva (see Fig. 77).

DISEASES OF THE MOTOR NERVES OF THE EYEBALL (THIRD, FOURTH, AND SIXTH).

The extrinsic ocular muscles are supplied by these three nerves, while the intrinsic are supplied by the third and the sympathetic.

I. **The motor oculi,** or **third nerve,** is purely motor, and supplies all the muscles of the eye except the superior oblique and external rectus, and controls in part also the ciliary muscle and the sphincter of the iris. Its apparent origin is from the inner side of the crus cerebri just anterior to the pons. It can be traced through the crus, however, to its deep origin in a nucleus beneath the corpora quadrigemina, situated in the floor of the aqueduct of Sylvius. Above the crus it pierces the dura, passes between the two clinoid processes of the sphenoid bone, along the outer wall of the cavernous sinus, where it receives some filaments from the cavernous plexus of the sympathetic; it then divides into two branches that enter the orbit through the sphenoid fissure. The superior and smaller division supplies the superior rectus and levator palpebræ superioris, while the inferior and larger branch subdivides into three portions, one going to the internal rectus, another to the inferior rectus, and the third to the inferior oblique.

Lesions of the third nerve result in (1) spasm or (2) paralysis.

Spasm rarely if ever occurs in all the muscles simultaneously. Any muscle may be affected, but the internal rectus and levator palpebræ are specially liable. It is met with in meningitis, hypermetropia, and hysteria; also as nystagmus, in which the spasm is clonic and bilateral; it also occurs in albinism, occasionally in coal-miners, or it may be congenital.

Irritation of the center or nerve may cause contraction of the pupil (*myosis*), as occurs in locomotor ataxia. The same result is brought about by paralysis of the sympathetic.

Paralysis.—The nerve may be involved in any part of its course by

inflammatory deposits or tumors, or the nucleus may be diseased. In the latter case there is usually ophthalmoplegia.

Relapsing and recurring palsy are two varieties. The first occurs chiefly in syphilitic subjects. One nerve becomes affected and partially recovers; the other one then becomes paralyzed, and partially recovers, relapses, and so on. The internal muscles may be involved.

Recurring or periodic palsy, the *migraine ophthalmique* of Charcot, is a rare form (p. 1209). It occurs in both sexes, but women are especially susceptible. It may begin in infancy and recur at intervals for years, the attacks being periodic, lasting a few days to six or eight weeks, and ending in complete recovery. They may be precipitated by some emotional disturbance, by menstruation, or by exhaustion. Their exact nature is not understood, but they resemble migraine in that there is severe headache or pain, usually over one eye, and in their association with vomiting.

Generally paralysis of the extra-ocular muscles is partial, and the symptoms will vary according to the muscles affected. When they are all involved there are ptosis, divergent strabismus, diplopia, and dilated pupil, with loss of the light-reflex and accommodation.

Intra-ocular Paralysis.—(*a*) **Cycloplegia,** or ciliary muscle-paralysis, gives rise to a loss of the power of accommodation, so that "far-sight" is good, while "near-sight" is blurred and indistinct. This can be corrected by a convex glass. Bilateral cycloplegia is usually due to a nuclear lesion. It occurs most frequently as a symptom of neuritis following diphtheria.

(*b*) **Iridoplegia.**—The pupil may be dilated (*mydriasis*) from palsy of the sphincter or spasm of the dilator, or it may be contracted (*myosis*) from the antithesis of the above.

The iris has three actions—two reflex and one associated: First, a reflex contraction of the sphincter on exposure of the eye to the light; second, a reflex dilatation of the radiating fibers on stimulation of some cutaneous nerve; and, third, a contraction on accommodation, usually, but not necessarily, associated with convergence (Gowers).

First, light-reflex iridoplegia. The iris reflex is lost in locomotor ataxia, in general paresis, and occasionally in disease of the peripheral portion of the third nerve, and sometimes also in syphilis. Accommodation and convergence are, however, usually preserved (*Argyll-Robertson pupil*). When these also are lost the condition is termed *ophthalmoplegia interna.*

In testing this reflex care must be taken to avoid the contraction of accommodation. The patient should look at a remote part of the room; then a light is brought suddenly in front of, and three or four feet distant from, the eye. One eye should be examined at a time, the other being covered, but not closed.

Second, skin-reflex iridoplegia. Normally, painful stimulation of the skin of the neck causes reflex dilatation of the pupil (pupillary skin-reflex), the afferent impulse being carried along the sympathetic. In locomotor ataxia myosis often exists. In such cases Erb showed that the skin-reflex was lost (*spinal myosis*).

Third, accommodation iridoplegia, in which the power of accommodation is lost. The pupil does not become smaller when looking at near objects. Westphal and Piltz have recently discovered independently that in certain pathological conditions the pupil contracts strongly upon closure, or attempted closure against resistance, of the eyelids. This reflex occurs most constantly in general paresis. Its exact significance is not known.

II. The **fourth nerve,** or **patheticus,** the smallest cranial nerve, supplies the superior oblique muscle. Its superficial origin is to the outer side of the crus cerebri, just in front of the pons. The fibers can be traced backward to the valve of Vieussens, in the substance of which it decussates with its fellow. Its deep origin is in a nucleus in the floor of the aqueduct of Sylvius, immediately behind and in close connection with the third-nerve nucleus. After piercing the dura mater the nerve runs along the outer wall of the cavernous sinus and enters the orbit through the sphenoid fissure. Since the superior oblique muscle directs the eyeball downward and rotates it, paralysis causes defective downward and inward movements, and consequent diplopia with inclination of the head forward and to the sound side. When occurring alone it is probably due to a nuclear lesion.

III. **The sixth nerve,** or **abducens,** has its deep origin in the floor of the fourth ventricle in close proximity to the seventh-nerve nucleus. Its superficial origin is from the lower part of the pons, in the groove between it and the medulla. Emerging, it pierces the dura, runs along the cavernous sinus, and enters the orbit through the sphenoid fissure to supply the external rectus. Owing to its long course, this nerve is specially liable to injury, usually from pressure due to tumors or from syphilitic or other forms of meningitis. Paralysis of the muscle causes convergent strabismus and consequent diplopia, owing to an inability to rotate the eye outward. In nuclear lesions the external rectus of the same side and the internal rectus of the opposite side are paralyzed, loss of associated lateral movements of the eyes toward the side of the lesion resulting. Conjugate deviation, the eyes being directed away from the side of the lesion, may also be observed. This is due to the fact that the sixth nerve nucleus and that part of the third nerve nucleus governing the internal rectus are connected through the posterior longitudinal fasciculus.

The internal rectus is not wholly controlled by the sixth nerve nucleus, however, for when the eye with the paralyzed external rectus is covered, the opposite internal rectus will act, though less readily than normally. Independent contraction of the internal rectus is controlled alone by the third nerve nucleus. Conjugate deviation also occurs in supranuclear lesions, as in apoplexy (p. 1165); if irritative, the deviation is away from the side of the lesion; if paralytic, toward it.

General Symptomatology of Paralysis of the Eye-muscles.—Loss of power in the ocular muscles is indicated by five kinds of symptoms (Gowers): (1) *Limitation of Movement.*—The amount of limitation in the movement of the eyeball is in direct ratio to the degree of palsy. In complete palsy the globe is ultimately fixed, owing to contraction of the unopposed muscle. In partial paralysis, as the limit of movement is approached the motion is often jerky (*paralytic nystagmus*).

(2) *Strabismus.*—Owing to defective movement the axes of the eyes do not correspond. "The deviation of the axis of the paralyzed eye from parallelism with that of the sound eye is termed the primary deviation."

(3) *Secondary Deviation.*—"If the sound eye is prevented from seeing the object, and the patient looks at this (is made to 'fix' it) only with the affected eye, the sound eye is moved still farther in that direction, and hence the deviation of the visual axes is increased. This is called the 'secondary deviation,' and depends on the fact that two muscles normally acting in unison are equally stimulated (innervated) for

any given movement. When one is weak, the amount of nerve-force employed to move the sound eye acts equally on the impaired eye, and hence the overaction. In paralytic strabismus fixation with the sound eye shows the primary deviation, while fixation with the affected eye reveals secondary deviation. In ordinary strabismus due to spasm this does not hold good; it matters not which eye is used, deviation remains the same."

(4) *Erroneous Projection.*—We judge of our relation to surrounding objects by the position of the eyeball as indicated to us by the degree of stimulation necessarily brought to bear on the ocular muscles. When one of these muscles is weak, the additional stimulation (innervation) necessary to move it in fixing an object impresses us with the idea that it is really farther away than is actually the case, and in attempting to touch it the finger goes beyond. This erroneous projection, or interference of visual sense-impressions, causes a disturbance of equilibrium and gives rise to vertigo, which has been named "ocular vertigo."

(5) *Double Vision.*—This is not due alone to a difference in the axis of vision, causing images on non-corresponding portions of the retina, but also to the erroneous projection. "If the patient looks with both eyes, the field of the unaffected eye, being normally projected, does not correspond with the field of the affected eye; the images formed in the two eyes are mentally referred to different positions; objects are seen double" (Gowers). The "true image" is that one formed in the sound eye, while the retina of the affected eye receives the "false image." The symptom is known as *diplopia*.

Homonymous or *simple diplopia* is that in which the false image appears on the "same side of the other as the eye by which it is seen." This is due to paralysis of an abductor muscle—*convergent strabismus*. *Crossed diplopia* occurs in divergent strabismus, the result of paralysis of an adductor. The false image appears to be on the other side of the real object—*i. e.* toward the sound eye.

Gowers' mnemonic is, "When the visual lines (prolonged ocular axes) cross, the diplopia is not crossed."

Ophthalmoplegia, a paralytic condition of the eye-muscles, may be partial or complete. Either the internal or the external muscles may be involved, constituting *ophthalmoplegia interna* or *externa*, and, when both are affected, total ophthalmoplegia. The lesions may be due to disease either of the nerve trunks or nuclei. The former may be due to some infectious disease, as diphtheria; excessive use of alcohol, arteriosclerosis; traumatism, causing either fracture at the base of the skull or hemorrhage into the region of the nerves; pressure of an aneurysm of a cerebral blood-vessel, basal meningitis, especially if syphilitic, and tumor. The latter may also be due to infectious diseases and excessive use of alcohol. Chronic lead-poisoning may also cause it, and it may be an early manifestation of either tabes, paresis, disseminated sclerosis, or cerebral syphilis. Tumors, inflammation within the orbit, or fractures involving it, and thrombosis of the cavernous sinus may also be causes of a more or less developed ophthalmoplegia. If acute, the condition may be due to a hemorrhage in the region of the nuclei, or an embolus or thrombosis in a branch of the basilar artery, or an inflammation of the nuclei, due either to infection or intoxication, as mentioned above (polio-encephalitis superior of Wernicke). Symptoms of bulbar palsy may coexist in chronic nuclear degenerations.

The **symptoms** vary necessarily according to the muscles involved. The eyes fail to follow objects and the face acquires a peculiar expression ("Hutchinson face").

The **treatment** consists in the removal of the cause when possible. As a large number of cases are due to some of the manifestations of syphilis, iodids and mercury should be used in all cases where another cause is not definitely known. In inflammatory cases counterirritation is employed by blisters placed on the temples, behind the ears, or at the occiput, or by leeches. Internally, the salicylates, mercury, iodids, and general tonics are useful. Rarely a case will recover spontaneously. Electricity is probably of little value. The diplopia, unless it can be obviated by a suitable lens, should be met by means of an opaque glass.

DISEASES OF THE FIFTH NERVE.

The trigeminus nerve has an extensive origin from the floor of the fourth ventricle. It supplies with sensation the whole region innervated by all the other cranial nerves except the first and second. It resembles a spinal nerve in that it has two roots, a motor and sensory, and on the latter a ganglion (*Gasserian*). From the latter arise three sensory branches—viz. the ophthalmic, superior maxillary, and inferior maxillary. A motor root joins the last named, the largest branch of the fifth nerve.

Morbid conditions of the fifth nerve cause sensory, motor, or gustatory symptoms. The lesion may be—(1) Pontine hemorrhage, softening, sclerosis, or tumor. (2) Disease or injury at the base of the brain—*e. g.* meningitis, gumma or other tumor, caries of bone. (3) Disease or injury of the branches, as neuritis, pressure due to aneurysm of the internal carotid or to a tumor in the cerebello-pontile angle or sphenomaxillary region, orbital cellulitis, and punctured wounds of the mouth and nose. (4) Rarely fracture of the skull. (5) Diseases of the Gasserian ganglion.

Symptoms.—**Sensory Portion.**—In the irritative stage the chief feature is pain; this may be shooting, boring, or burning in character. Tenderness along the course of the nerve and hyperesthesia may also exist. Later, anesthesia develops in the distribution of some or all of the branches in the skin of the face and in the mucous membrane of the nose, mouth, lips, tongue, and, in some cases, of the hard and soft palate also. The occurrence of such anesthesia, associated with pain, indicates an organic lesion, usually of the ganglion, as distinguished from a functional neuralgia (p. 1077).

The secretions are often increased, though at first they are lessened; hence the anosmia, due to dryness of the nasal mucosa. Loss of sense of taste may also occur. Other trophic changes are—inflammation and ulceration of the gums, looseness of the teeth, and inflammation of the eye. Corneal opacities, ulceration, sometimes perforation, and finally complete destruction of the eye—neuro-paralytic ophthalmia—are noted. This is especially apt to occur when the Gasserian ganglion is involved. Painful and intractable herpes may develop. Hemifacial atrophy may result from disease of the fifth nerve (Mendel).

Motor Portion.—*Paralysis.*—Partial or complete inhibition of the movement of the muscles in the region supplied—*i. e.*, those of the jaw, the masseter, temporal, pterygoid, mylo-hyoid, and the posterior

belly of the digastric. The degree of palsy can be ascertained by placing a finger on each masseter or temporal muscle while the patient alternately opens and forcibly closes the mouth. In external pterygoid paralysis movement toward the sound side is impossible, and on depression of the lower jaw it deviates toward the affected side. Ultimately wasting of the muscles, with deformity, takes place.

Spasm (the so-called "masticatory spasm" of Romberg) may be tonic or clonic. In tonic spasm—trismus or lockjaw—the jaw is firmly set and the muscles are hard, rigid, and sometimes painful. This occurs in tetanus, in certain cases of tetany and hysteria, in caries of the teeth, occasionally after exposure, and in irritative centric or peripheral lesions. Clonic spasm is more or less continuous or intermittent. The former consists of short, quick, vertical or rarely lateral movements (*e. g.*, gnashing of the teeth), usually associated with some other condition, as paralysis agitans, general convulsions, and the like, or it may exist alone, especially in women late in life. The intermittent form is rare and occasionally occurs in chorea. Contractions are single, forcible, and are separated by some little time. The tongue and cheeks may be bitten in the attack. (See Tic, p. 1216.)

Gustatory Portion.—*Symptoms* referable to this portion are not always present in disease of the fifth nerve. Many neurologists do not believe that gustatory sensations are transmitted by it, the glossopharyngeal (p. 1110) being believed by them to be the nerve of taste. If the fifth does take part in this function, it does it for the anterior two-thirds of the tongue. There may be a loss of taste without sensory disturbance, or *vice versâ*, or both may exist contemporaneously. Lesions of the nerve-root or middle-ear disease may cause it, but pontine lesions, as a rule, do not. It occurs in paralysis of the seventh nerve, if the lesion is in the Fallopian canal, due to involvement of the chorda tympani. A perverted sense of taste—*parageusia*—may be present in hysteria and insanity. Increased sensitiveness—*hypergeusia*—and subjective sensations of taste may result from irritative lesions, and the latter may precede an attack of epilepsy (as an aura).

The **diagnosis** is not difficult as a rule. Anesthesia in the area supplied by the nerve, with pain, is in favor of organic disease, the nature of which must be determined by the accompanying symptoms. Spasm may be simulated in cases of rheumatism or rheumatoid arthritis involving the temporo-maxillary articulation.

Treatment.—The underlying cause should be attacked when possible, and mercury, the iodids, and the salicylates should be administered in specific cases and in those due to exposure. Analgesics, and even opiates, may be necessary. Sometimes vigorous counter-irritation is of value. Attention must be paid to the condition of the general system. The battery may be tried, preferably with the faradic current, or by means of electricity short and extremely rapid blows may be made over the nerve.

DISEASES OF THE SEVENTH OR FACIAL NERVE.

The nucleus of this nerve in the floor of the fourth ventricle is in relation with those of the sixth, eighth, and twelfth nerves. Like the spinal nerves, it has an upper and a lower neuron or motor segment, the former

extending from the cortical center in the lower Rolandic region to the nucleus, while the latter runs from the nucleus to the periphery. Lesions may involve any part of the tract, producing either spasm or paralysis.

Spasm.—This may be either general or partial, affecting only the orbicularis palpebrarum (*blepharospasm*). It is sometimes called *tic facialis* or mimic spasm (p. 1217).

Etiology.—The commonest causes are peripheral irritations, and particularly those that involve the trigeminus, as carious teeth, conjunctivitis, or some nasal irritation. Less frequently irritation in some other part of the body, as intestinal parasites or uterine disease, may be the exciting cause. Finally, there may be lesions in any part of the motor tract supplying the face, either in the cortex (meningeal tumor, exostoses, or focal softening), when it becomes one of the manifestations of the Jacksonian convulsion; in the facial nucleus in the lower part of the pons; along the course of the facial nerve (aneurism or atheroma of the vertebral artery); and as a sequela of peripheral paralysis of the nerve (Bell's Palsy, p. 1101). Morbid changes in the nerve itself or in the muscles have not been observed.

The **symptoms** of the disease include, first, the *spasm:* this is usually a sudden clonic convulsion of the muscles of one side of the face, with closure of the eyelids and retraction of the angle of the mouth. Rarely there are associated movements of the palate and eyeballs. The spasms may be single or they may occur in groups frequently repeated, or recur constantly at more or less irregular intervals. Less frequently the contraction may be tonic in character, lasting several seconds or even minutes. These forms are frequently associated with clonic spasms. Ordinarily the spasm is painless. Sometimes there is also *tinnitus aurium.* Occasionally edema of the face, especially in the orbital region, occurs. The immediate exciting cause of an attack may be fatigue or excitement, or it may occur as an associated movement, as in a case that I observed, in which spasm always accompanied the beginning of speaking.

The **diagnosis** must be made from tic (p. 1216). It may be occasionally confounded with *chorea*, especially when the latter is chiefly localized in the face, or with *athetosis* due to infantile brain-lesions. In the former the movements are not so quick nor confined exclusively to the anatomical distribution of a certain nerve; in the latter hemiplegia will usually coexist (p. 1174). In fact, athetosis is a spasm. Recognition of the cause is often very difficult, and a careful examination of the whole body should be made for any possible source of irritation.

The **prognosis** is extremely unfavorable for cure, since only in cases of recent occurrence, and with a distinct source of peripheral irritation, is permanent recovery likely.

The **treatment** consists in the removal of any source of irritation and the application of electricity, particularly the mild galvanic currents, with the anode over the sensitive points. Patrick[1] has injected alcohol into the region of the nerve at the stylomastoid foramen with success. The use of antispasmodics, as conium, gelsemium, morphin, and the bromids, may give temporary, but rarely permanent, relief.

Paralysis (Bell's Palsy).—Depending on the seat of the lesion, we have—(*a*) supra-nuclear, (*b*) nuclear, and (*c*) infra-nuclear palsy. The

[1] *Jour. Nerv. and Ment. Dis.*, Jan., 1909.

following table presents the general differences between upper and lower neuron palsy:

Supra-nuclear Paralysis.	Nuclear and Infra-nuclear Paralysis.
The upper part of the face is not affected, the muscles of the angle of the mouth being chiefly concerned.	All parts of the face involved, including the orbicularis and frontalis. Nuclear palsies are sometimes incomplete.
Voluntary movements are more impaired than the emotional.	Voluntary and emotional movements equally affected.
All reflex movements are normal.	All reflex movements are lost.
Electric reaction is normal, or only slightly impaired to both galvanic and faradic currents.	Reactions of degeneration are present.
There is no wasting.	Wasting is present.

(*a*) *Supra-nuclear paralysis* is generally associated with hemiplegia, the palsy of face and limbs being on the same side—*i. e.* opposite the lesion, which may consist of a hemorrhage, tumor, abscess, softening. It may be the result of injury, and may be situated in the cortex, corona radiata, or the internal capsule. When the cortical face-center is alone involved, the limbs escape (*monoplegia facialis*). This form is rare.

(*b*) *Nuclear paralysis* is due to hemorrhage, tumor, or softening at the site of the nucleus in the pons, in which case paralysis of the arm and leg of the opposite side frequently coexists. It may also result from an attack of diphtheria, and very rarely occurs in cases of anteropoliomyelitis (polioencephalitis). It most commonly occurs in connection with the involvement of the motor nuclei of the ninth and tenth nerves and the nucleus of the twelfth in the disease known as glosso-labiolaryngeal paralysis or chronic bulbar palsy. As already noted, the symptoms are similar to those of infranuclear paralysis, but the affection is usually bilateral (p. 1129).

(*c*) *Infranuclear paralysis* is caused by pressure on the nerve at the base of the brain by tumors, meningitis, aneurysm, or hemorrhage. In the Fallopian canal the nerve may be damaged by bone-disease or some form of otitis. This is the seat, too, of the so-called "rheumatic neuritis," the result of exposure or infection (Bell's Palsy).

Fracture of the base of the skull or injury to the nerve as it emerges from the stylo-mastoid foramen may result in facial palsy. *Diplegia facialis* is rare, but may be caused by a single lesion in the pons, where the facial paths cross, or by two lesions, one on either side. The causes enumerated above, when bilateral, beget double facial paralysis.

Lesions in the lower part of the pons may result in crossed hemiplegia, the fibers being involved in their course between the nucleus and the point of emergence of the nerve, the side of the pons. The face will be paralyzed on the same side as the lesion, since this latter is below the decussation of the facial tracts, and involves the outgoing nerve, together with opposite hemiplegia. In alternate or crossed hemiplegia the facial palsy is of the infra-nuclear type, while in ordinary hemiplegia the supra-nuclear type is met with. Certain symptoms of nerve-irritation may precede the actual palsy or may be concomitant, such as slight pain and tenderness, some swelling in front of the ear, muscular twitching, and occasionally vertigo.

Symptoms.—The affected side is immobile and expressionless, and the

normal lines are diminished or abolished. This is seen most markedly in those above middle life. The eye cannot be closed, owing to weakness of the orbicularis palpebrarum, and, as the tears are not directed into their proper channel, the eye waters. Voluntary and emotional movements are lost. Whistling and smoking are performed with difficulty, if at all; if the cheeks are puffed out, air escapes upon the paralyzed side; food collects between the teeth and cheek, owing to paralysis of the buccinator; in drinking the patient inclines the head to the sound side to prevent escape of the liquid from the corner of the mouth. The dilator naris is paralyzed; hence sniffing is interfered with, and the sense of smell is diminished on that side.

When the tongue is protruded it seems to be drawn toward the palsied side. This is not the case, however, the effect being due to contraction of the unopposed muscles on the sound side of the face. All reflex movements are lost. The palate is not affected and sensation is not impaired. When the nerve is involved between the *intumescentia gangliformis* and the origin of the chorda tympani—*i. e.*, within the Fallopian canal—taste is lost in the anterior part of the tongue, and there is some diminution in the secretion of saliva. When other parts of the nerve are diseased, taste is not interfered with. Hearing may be increased, owing to paralysis of the stapedius, with consequent unopposed action of the tensor tympani. When due to middle-ear disease and in disease of the base of the brain, involving both facial and auditory nerves, hearing is lessened. In the latter, however, bone conduction will be either diminished or lost. Some degree of wasting takes place in the affected muscles, and both quantitative and qualitative electric changes quickly follow the palsy. If the intumescentia gangliformis is involved, we may have herpes of the auricle and neuralgic pains in the ear in addition to paralysis. This may also occur without paralysis.

The duration of an attack varies from a few days to several months or a year, and in rare cases it is permanent. The onset is usually acute, and the acme of the attack may be reached in from a few hours to a couple of days.

Diagnosis.—From the table previously given it will be easy to differentiate supra-nuclear from infra-nuclear palsy. When contractures have taken place, owing to the furrows thus produced the affected side may be taken for the sound side, but on getting the patient to whistle the true state of affairs will manifest itself.

Prgonosis.—In the rheumatic cases and those due to middle-ear disease recovery usually occurs in from six weeks to three months. Permanent contractures and deformity, sometimes associated with clonic spasm may result.

Treatment.—Search for the cause. If ear-disease is present, make provision for free drainage; if syphilis, give iodid of potash, mercury, or both. In cases due to cold, the so-called rheumatic palsies, counter-irritation is especially called for, and cantharidal collodion, fly-blisters, or the actual cautery behind the ear or over the occiput are very useful. The bowels should be freely opened, and diaphoretics or hot baths, alkaline diuretics, and salicylates administered; in the inflammatory stage small doses of mercury are of value, and later mercuric iodid or general tonics. After the acute symptoms have subsided (in about ten days), galvanism should be employed to stimulate the nerves and to help in maintaining the tone

of the muscles. When contractures threaten in late cases the use of electricity should be dispensed with. When the paralysis has become permanent benefit can sometimes be rendered by transplanting parts of either the hypoglossal or spinal accessory motor nerves into the trunk of the facial peripheral to the lesion. For severe and chronic pain due to disease of the geniculate ganglion or intumescentia gangliformis cure has been obtained by its removal.[1]

DISEASES OF THE AUDITORY NERVE.

The eighth nerve has its deep origin in the medulla. It consists of two parts: the cochlear, which has to do with hearing, and the vestibular, which has to do with maintaining of our relation to space, or, in other words, our equilibrium. The auditory fibers decussate in the region of the nuclei, passing in the posterior extremity of the internal capsule to the opposite hemisphere. The cortical center is in the temporosphenoidal lobe (first and second convolutions, Fig. 68). It is also connected with the medial geniculate body and posterior corpora quadrigemina; the vestibular branch, in addition, is connected with the cerebellum. Destruction of that of the left side results in *word-deafness*: thus, spoken words may be heard, but are not recognized as such. This is not a common condition. Rarely the auditory tract may be involved between the cortex and the nucleus. The nerve may be implicated at the base of the brain by tumors of the cerebello-pontile angle, aneurysms, hemorrhage, meningitis, and traumatism. Erb has described a primary nerve degeneration in tabes dorsalis. Disease may attack the labyrinth, either primarily or secondarily to middle-ear disease, which, if confined to the cochlear division, causes deafness, and, if to the vestibular branch in the semicircular canals, vertigo. If both branches are involved, deafness and vertigo coexist. Drugs—quinin, apiol, salicylates—may cause deafness similar to the labyrinthine variety. In anemia and in other conditions in which the general health is below par, also in hysteria, hearing may be affected. The lesions give rise either to an increased or diminished sense of hearing:

(*a*) *Hyperacusis*, in which certain or all sounds are intensified. Paralysis of the stapedius muscle causes low notes to be heard with great intensity. Auditory hyperesthesia may also occur in hysteria or during the course of cerebral or general disease.

(*b*) *Dysacusis*—difficult hearing—may be due to middle-ear disease, or it may exist as a "nervous deafness," the result of labyrinthine or nerve-disease. These may be differentiated by means of the tuning-fork. Normally, air-conduction is better than bone-conduction, and if in a deaf person a tuning-fork can be heard vibrating longer when held against the skull-vault or temporal bone than in front of the ear, there is some impairment of conduction in the meatus or middle ear. When the patient is deaf, and yet the normal relation is maintained between air- and bone-conduction, the labyrinth or the nerve is at fault.

(*c*) *Tinnitus aurium*—irritation of the auditory nerve—a condition in which subjective sounds occur, such as whirring, buzzing, ticking, or ringing in character. In certain subjects they are worse at night than during the day, and at times they are paroxysmal; as a rule, in any case they are intensified when the general system is below par.

Tinnitus may be caused by anemic or depraved nutritional states,

[1] *Jour. Amer. Med. Assoc.*, Dec. 25, 1909, p. 2144.

high blood-pressure, intracranial aneurysm, pressure on the cervical sympathetic by enlarged glands, tumor, or aneurysm, impacted cerumen, otitis media, labyrinthine disturbance, blows upon the head, excessive auditory stimulation, loud noises, or it may occur during an attack of migraine or as an epileptic aura. In a neurasthenic individual the subjective noise, no matter what the cause, will be accentuated. The more complex and elaborate the sound, the greater the probability of its being of central origin. (See Ménière's Disease.)

Treatment.—Careful search must be made for the cause of any of these morbid conditions just described, and, when practicable, they should be removed. The system should be brought into as good a condition as possible. In hyperesthesia bromids occasionally avail. In dysacusis little can be done when the cause is labyrinthine. The same is true when the nerve or its centers are involved. For tinnitus, counter-irritation and electricity may be tried externally, and iodids internally, but with little hope of relief; in addition, sedatives, as the bromids, are generally called for, and even morphin may be necessary in paroxysmal attacks. Occasionally a single large dose of pilocarpin (gr. $\frac{1}{10}$) may give relief for some time. Operation has been resorted to (p. 1106).

MÉNIÈRE'S DISEASE.

Definition.—An aural or labyrinthine vertigo—originally described by Ménière in 1861; the cardinal symptoms are vertigo, deafness, noises in the ear, and sometimes vomiting.

Pathology.—There may be an inflammation or atrophy of the nerve-endings. There are also changes in the labyrinthine membrane from any cause or from hemorrhage.

Etiology.—Ménière's disease is most common after thirty, and is rarely met with before that age. It is twice as common in men as in women. The precise lesion is labyrinthine, and is the result of exposure, gout, syphilis, senile change, congestion, and, more rarely, hemorrhage. Any cerebral disturbance or gastric or other irritation is apt to induce an attack.

Symptoms.—Vertigo is present, and varies from an extremely slight transient attack, and one that is entirely subjective, to one of almost explosive violence. The patient may have a sensation of having been struck, and then of falling heavily to the ground. The slight form may be continuous with more or less frequent severe attacks, or a complete intermission of days, weeks, or months may transpire. The attacks may arise without apparent cause, or as a result of a blow or even a sudden movement, and occur during both working and sleeping hours. The giddiness, when severe, causes nausea and vomiting, and, if prolonged, bile is vomited as in ordinary bilious attacks. When the attack is very acute momentary unconsciousness supervenes. Nystagmus and diplopia may occur during an attack. Tinnitus and deafness usually exist together, the former may be either mild or very severe. It is usually constant, and possibly worse during an attack; it may be entirely absent between the attacks. The latter (nervous deafness) is constant and of varying severity in different individuals.

Diagnosis.—The occurrence of vertigo and tinnitus in a person with more or less nervous deafness, with or without gastric symptoms,

establishes the diagnosis. The tinnitus and the character of the deafness usually suffice to distinguish this from other forms of vertigo. Vertigo and deafness may also be caused by middle-ear disease, but in such a case examination (p. 1104) will show that the deafness is not of nerve origin. Similar symptoms may also be caused either by a growth or patch of meningitis situated in the cerebellopontile angle. In such a case other symptoms of brain tumor or meningitis will be present, and the seventh nerve is also usually affected. In epilepsy with auditory auræ the period of unconsciousness is generally much longer, and on regaining consciousness the patient is dull and drowsy for some time. It is possible also, as a rule, to elicit a history of convulsions.

Prognosis.—In some cases the condition grows progressively worse until deafness supervenes, when it ceases. Often, however, arrest or improvement, or even complete recovery, may be secured. In heart-disease the shock may prove fatal, and in the very acute but, fortunately, rare cases the prognosis is always bad.

Treatment.—Counter-irritation over the mastoid process and the internal use of bromids to lessen the morbid sensibility will prove valuable. The emunctories must be gotten in good condition, and any underlying disease, as syphilis or gout, must be treated. Charcot suggested the use of drugs that produce tinnitus—quinin, for instance. The cases were worse at the time, but some of them seemed to improve subsequently. Gowers employs sodium salicylate in 5-gr. (0.324) doses, thrice daily, believing that more good arises when such drugs are given in moderation. Small doses of pilocarpin sometimes do good. Apiol might be tried in this connection. Nitroglycerin and the nitrites are sometimes of value in cases associated with arteriosclerosis. Division of the auditory nerve has cured some cases.[1]

DISEASES OF THE GLOSSO-PHARYNGEAL NERVE.

The ninth cranial nerve has its origin in the posterior part of the floor of the fourth ventricle, in close relation with the pneumogastric nerve. Our knowledge as to its function is not exact, both because it is seldom if ever involved alone, and also, on account of its many connections (with the trigeminus, the facial, the pneumogastric, and the sympathetic nerves), it is difficult to say whether the terminal fibers involved represent the functions of its roots or of one of its connections (Gowers).

Its fibers are distributed to the tonsils, the back of the tongue, the soft palate, the pharynx, the Eustachian tubes, the tympanitic cavity. It supplies both motor and sensory fibers, it is also the nerve of taste, certainly for the posterior portion of the tongue, and possibly, by means of connections with the fifth nerve, for the anterior as well. This nerve is involved in the nuclear degenerations that are spoken of as bulbar palsies. It may be also affected by meningitis or new growths.

DISEASES OF THE PNEUMOGASTRIC NERVE.

As already stated, the origin of the tenth cranial nerve is in intimate relation with that of the ninth. It is also continuous below with that of the eleventh, and all three are associated with the center for the hypoglossal nerve. The nerve proper arises from the side of the medulla, and runs on either side of the neck in the sheath of the carotid

[1] Ballance, *The Lancet*, 1908, vol. ii.

artery, lying behind that vessel. It enters the thorax in front of the subclavian artery on the right side, and between the subclavian and the carotid on the left; then it courses beside the esophagus, and is distributed to the pharynx, larynx, lungs, heart, esophagus, and stomach, and sends fibers to the intestines and spleen.

The esophageal fibers are both motor and sensory, gastric fibers being chiefly sensory. The vagus is in part the motor nerve of the intestines. It also contains both accelerator and inhibitory fibers for the respiratory center, is the cardiac inhibitory nerve and a vasodilator, and is said to contain trophic fibers for the heart and lungs.

Etiology.—The nerve may be involved at its nucleus either by hemorrhage or softening. The nuclei of the ninth, eleventh, and twelfth nerves, and frequently the seventh, are simultaneously attacked, either wholly or in part, giving rise to a group of symptoms known as *bulbar palsy*. The tenth nerve at its superficial origin may be compressed by neoplasms, aneurysms, and the products of *meningitis;* in its course down the neck it may suffer pressure, or may either be tied in ligating the carotid artery or cut in the removal of a tumor or enlarged glands. Very rarely it may be injured by incised or punctured wounds, or be the seat of neuritis due to exposure or to some toxemia. The morbid conditions of the pneumogastric are best studied by considering the branches of distribution separately.

(*a*) **Pharyngeal Branches.**—The muscles and mucous membrane of the pharynx are supplied by branches of the pneumogastric and glossopharyngeal nerves, constituting the pharyngeal plexus. The pharynx may be the seat of spasm or paralysis: this is purely a "functional" condition, and usually occurs in hysteric (*globulus hystericus*) or in nervous individuals.

Paralysis of the pharynx causes difficulty in swallowing, so that food remains in the mouth instead of being passed into the esophagus. Particles often enter the larynx and give rise to paroxysms of coughing, and at times cause choking. When the soft palate is also paralyzed, the food is regurgitated into the nose. The lesion is generally nuclear. The root of the nerve may be involved as it leaves the side of the medulla by meningitis or by pressure from a neoplasm or an aneurysm. It may also be caused by a toxic neuritis, as in diphtheria.

(*b*) **Laryngeal Branches.**—The superior laryngeal nerve furnishes sensory fibers to the mucous membrane of the larynx above the vocal cords, and supplies also the crico-thyroid and epiglottidean muscles. The inferior or recurrent laryngeal nerve, which takes its origin in the superior thoracic region, winds around the arch of the aorta on the left side and around the subclavian artery on the right, reaching the larynx by running up between the trachea and esophagus. It is the sensory nerve of the larynx below the vocal cords, also of the entire trachea, and supplies all the muscles of the larynx except those named above. It has been shown that the motor fibers of the larynx come from the glosso-pharyngeal nucleus, the pneumogastric fibers being sensory.

Spasm of the larynx is due to over-action of the glottis-closers (the adductors), though some cases described in this category are probably instances of abductor paralysis. The condition is rather rare in adults, but quite common in children (laryngismus stridulus), and particularly in rachitic subjects. An attack may also be induced in those predisposed by any form of nerve-irritation or catarrhal condition of the respiratory

tract. It may be part of a general neurosis; it is sometimes seen in tabes dorsalis (*laryngeal crisis*); and Liveing reports that he has seen it take the place of an attack of migraine. *Spastic aphoria* consists of a spasm induced whenever an attempt to speak is made. Laryngeal spasms occur most frequently at night. Dyspnea is the most striking symptom, and is so intense in some cases that suffocation seems imminent. The patient may be cyanotic. Soon the retained carbonic acid gas causes relaxation, but, as the cords open slowly, the inspiration is accompanied by a crowing sound, and the expiratory sound is harsher than normal.

Paralysis of the larynx may be the result of a nuclear degeneration (glosso-pharyngeal), as in chronic bulbar paralysis; this form may occur in disseminated sclerosis, tabes dorsalis, general paralysis of the insane, and in certain toxemias. The paralysis is generally bilateral; rarely it is unilateral.

A cerebral lesion in the laryngeal cortical center may cause pseudo-bulbar paralysis. Since the two centers are compensatory, the lesion must be bilateral. This may also be caused by capsular lesions (p. 1166).

The nerve may be involved at its root or in any part of the trunk, and such lesions are usually unilateral. The recurrent laryngeal nerve, especially the left, is more apt to be diseased than the superior, on account of its position. Thus, the arch of the aorta is more frequently the seat of an aneurysm than the subclavian; enlarged thoracic glands, neoplasms, and an enlarged thyroid can also damage these nerves. The peripheral filaments may be attacked as part of a multiple neuritis.

In certain cases the muscles become weakened without being paralyzed, this possibly being due to a local neuritis, or to a congestion and inflammation of the mucous membrane from over-use (*clergymen's sore throat*), or as the result of exposure.

The following are the chief forms of paralysis:

(1) *Complete Paralysis.*—By this is generally understood paralysis of all except the crico-thyroid and epiglottidean muscles, though occasionally these may also be involved. Since the cords are paralyzed, phonation is impossible. As a rule, there is no interference with respiration, though the pressure of the in-going air may bring the cords nearer together, and thus produce a certain amount of inspiratory harshness.

As the cords cannot be closed, coughing is impossible, as the air escapes through the glottis, and no expulsive force can be given to it. When the paralysis is unilateral these symptoms will of necessity be modified, and some degree of phonation may be possible. The most common cause of this condition is an involvement of the recurrent laryngeal nerve; the lesion may, however, be nuclear or in the course of the nerve-trunk.

(2) *Paralysis of the Abductors.*—The only special abductor muscles are the posterior crico-arytenoids. When they are involved the glottis fails to open in inspiration, and the unopposed adductors bring the vocal cords together. They are still more closely approximated during inspiration by the column of air, and hence the prolonged, stridulous inspiratory sound. Phonation and expiration are practically unchanged. It is quite likely that many cases supposed to be instances of hysteric spasm of the glottis are really cases of abductor paralysis.

In unilateral paralysis the normal movements of the unaffected vocal cord prevent any marked degree of dyspnea and stridor: phonation is usually hoarse and of a low pitch. In cases of long duration the symptoms become more marked as the unopposed adductors undergo secondary contracture and still further narrow the glottis.

This condition may be due either to central disease or to some local change. The abductor muscles may be degenerated, while all the other laryngeal muscles are healthy, or one or both recurrent nerves may be affected. These nerves innervate both the abductors and adductors, and it is not clearly understood why the abductors alone should suffer when the parent nerve-trunk is involved. At any time it might be a very grave condition, for should any swelling of the cords supervene nothing but a prompt laryngotomy could prevent suffocation.

(3) *Adductor Paralysis.*—The cords move normally during respiration, and hence there is no stridor; as they cannot be approximated, however, phonation is impossible. This condition is met with in hysteria, producing hysteric aphonia, in public speakers who overtax their voices, and also in laryngitis.

The following table, from Gowers' text-book on *Diseases of the Nervous System*, enables one to get a comprehensive idea of the subject:

Symptoms.	Signs.	Lesions.
No voice; no cough; stridor only on deep inspiration.	Both cords moderately abducted and motionless.	Total bilateral palsy.
Voice low-pitched and hoarse; no cough; stridor absent or slight on deep breathing.	One cord moderately abducted and motionless, the other moving freely, and even beyond the middle line in phonation.	Total unilateral palsy.
Voice little changed; cough normal: inspiration difficult and long, with loud stridor.	Both cords near together, and, during inspiration, not separated, but even drawn nearer together.	Total abductor palsy.
Symptoms inconclusive; little affection of voice or cough.	One cord near the middle line, not moving during inspiration; the other normal.	Unilateral abductor palsy.
No voice; perfect cough; no stridor or dyspnea.	Cords normal in position, and moving normally in respiration, but not brought together on an attempt at phonation.	Adductor palsy.

Sensory disturbances of the larynx are rare, and especially hyperesthesia. Anesthesia may be due to hysteria, or to bulbar paralysis, or to disease of the superior laryngeal nerve. It is dangerous, as food may enter the windpipe.

(*c*) **Cardiac Branches.**—These with branches from the sympathetic form the cardiac plexus. The vagus contains both accelerator and inhibitory fibers, but the latter predominate; therefore irritation of the nerve, either centric or peripheral, will slow the heart's action. Czermak was able to slow the action of his heart by pressing a small tumor in his neck against the vagus nerve. When the function of the nerve is lowered, inhibition is removed and the heart's action becomes rapid.

This may be brought about by a toxemic neuritis, by pressure, accidental ligature, or by incised or punctured wounds. Various emotions and nervous states may bring about the same result.

(*d*) **Pulmonary Branches.**—Both accelerator and inhibitory fibers exist, but in this case the accelerator influence predominates, so that irritation results in increased respiratory movements or even in bronchial spasm, since the bronchial muscles are also supplied by this nerve. It is this nerve that is supposed to be concerned in the production of asthmatic paroxysms. Therefore, when the nerve-function is lowered the respirations become much slower. The nerve is supposed to contain trophic fibers for the lungs.

(*e*) **Esophageal,** (*f*) **Gastric, and** (*g*) **Intestinal Branches.**—The esophageal branches are rarely damaged, and irritation (spasm) occurs more frequently than paralysis. The pneumogastric gives the sensory, and in part the motor, nerve-supply to the stomach, and irritation gives rise to increased contractions with some pain.

The sensation of hunger is supposed to be associated with the vagus nerve, and vomiting may result from direct or reflex irritation. Paralysis causes some diminution of the gastric contractions. Normally, the vagi accelerate intestinal peristalsis.

Treatment.—It is almost always impossible to remove the cause of the above conditions. Syphilitic lesions are probably the most amenable, and in the various laryngeal palsies electricity may be employed, though it is of somewhat doubtful utility, and in abductor palsy may possibly exert a harmful influence by stimulating the adductors. Strychnin and general tonics should be administered. Massage of the larynx may be tried, and in spasmodic conditions attention should be directed to the general physical state. All sources of nerve-irritation should be removed if possible, and bromids, or even chloral, should be given.

DISEASES OF THE SPINAL ACCESSORY NERVE.

This nerve consists of two parts—an external or spinal, and an internal or accessory, portion. The latter has already been described in connection with the pneumogastric nerve. It forms the motor portion of that nerve, and is distributed to the laryngeal and pharyngeal muscles. The spinal element arises from the multipolar ganglion-cells in the anterior gray horns of the cervical cord, ascends and enters the cranium through the foramen magnum, and leaves it, after joining with the accessory part, through the jugular foramen. It supplies the sternomastoid muscle and in part the trapezius.

Injury or disease of the nerve may result in spasm or paralysis. Only the spinal part is considered in this section.

TORTICOLLIS.

(*Wry-neck.*)

This may be a congenital or an acquired condition.

Congenital torticollis, or "fixed wry-neck," is the result of an atrophy and shortening of the sterno-mastoid muscle, brought about by some intra-uterine condition or, possibly, by an injury at birth. The

right muscle is most commonly affected. The head turns slightly toward the sound side; the eye may deviate, and curvature of the cervical spine may develop.

Facial asymmetry is a usual concomitant of this condition. The face on the same side as the lesion develops less rapidly than the other side, and in time secondary contracture of the unopposed muscles takes place. The torticollis can be cured by tenotomy, but the facial asymmetry persists. Fixation is necessary for a while when contracture exists.

Spasmodic wry-neck may be tonic or clonic. These forms may coexist, alternate, or occur independently in different individuals. The condition is met with almost exclusively in adults, and occurs most frequently in middle-aged men.

Pathology.—Usually no macroscopic or microscopic evidence of any lesion has been discovered, and the condition is probably dependent upon an overactivity of the neurons in the various centers that control the muscles of the affected part. Some cases belong to the Tics (p. 1216).

Etiology.—The influence of sex and age has been mentioned; a neurotic heredity may also predispose. Torticollis may follow habit-spasm, or some injury to the head or neck, or exposure to cold, the latter constituting the "rheumatic" type. It may be due to an irritative lesion either in the spinal cord above the fifth cervical segment, or to tumor, hemorrhage, meningitis, or bone disease in the upper part of the vertebral canal. Most cases are apparently functional, and may be due to reflex irritation, as eye-strain, or occur without apparent cause.

Symptoms.—The occiput is drawn toward the shoulder of the affected side, the chin is elevated, and the face rotated more or less toward the sound side. The sternomastoid may alone be affected, but the upper fibers of the trapezius are usually also involved. In addition, the superior obliquus and complexus of the same side and the splenius capitis and inferior obliquus of the opposite may be involved. Affection of the deep muscles causes greater retraction of the head than when the sternomastoid and trapezius are alone the seat of spasm. Spinal curvature may ensue, the convexity being toward the sound side. This only takes place in cases that have existed for some time. Clonic spasm is infinitely more distressing and more apt to be permanent.

Some pain and muscular twitching may precede the onset of the attack, though, as a rule, muscular contractions are the first indication. These are mild at first, and rarely abruptly, more commonly slowly, they increase in severity. As the case progresses other muscles, and even those of the arm, become involved. Cases have been described in which certain muscles or groups of muscles in the hand or arm have been primarily affected, the condition gradually spreading from them. The spasm usually ceases during sleep. An attack may cause pain, but, as a rule, it induces merely a feeling of fatigue in the muscles; it is worse if the patient is excited or emotional. Bilateral spasm may occur, the muscles of both sides being equally affected (*retro-collic spasm*). Gowers speaks of a case in which the backward displacement of the head was so great that the face was horizontal and looked directly upward.

Diagnosis.—As a rule this is not difficult. When spasm is induced by enlarged and painful glands beneath the sterno-mastoid the

age of the patient will be of value in determining the true condition. This usually occurs in children; true wry-neck, on the other hand, very rarely commences before the thirtieth year. Hysteric spasm may also simulate spasmodic torticollis, but it generally occurs in young women, and usually other evidences of hysteria are also present. The *rheumatic type* and the rigidity induced by *caries of the spine* must be differentiated from one another and from spasmodic wry-neck. If the rigidity comes on suddenly, following exposure to cold or wet, and the pain is not increased at night or by depressing the head upon the spine, and is relieved by hot applications, the condition is probably rheumatic. When the rigidity and pain are of slow onset, without history of exposure, and the pain is both worse at night and is increased by depressing the head upon the spine, but is relieved by elevating the head, the condition is very probably one of caries of the spine. In irritative lesions within the spinal canal, either intra- or extramedullary, the spasm is usually bilateral and tonic, and other symptoms of involvement of the cord are likely to be found.

Prognosis.—Very rarely the torticollis may diminish or even cease after an existence of months or years. Usually, however, it is persistent, either being stationary or slowly increasing in severity and widening in range. The prognosis must always be guarded, and in severe cases grave as to recovery, though the disease does not shorten life.

Treatment.—Generally very little can be expected from medication. Bromids, morphin, chloral, hyoscyamus, or cannabis indica may be tried, as may the various forms of counterirritation. Atropin, in increasing doses, administered hypodermically into the muscles, has been effectual in some cases. Massage of the affected muscles and rest in bed may also at times be of service. Galvanism should be tried, the negative pole being placed over the occipital region and the positive over the affected muscles. Nerve-stretching and tenotomy of the affected muscles is of very little value. The only surgical procedure that has proved of any distinct value is neurectomy of the spinal accessory and posterior branches of the upper four cervical nerves, with excision of a part of the nerves to prevent reunion. This necessarily causes paralysis and atrophy of the muscles supplied; but, since it often abolishes the spasm, the slight loss of power and the interference with the movement of the head are comparatively infinitesimal. The results, however, are not uniform, even so far as the spasm is concerned.

PARALYSIS OF THE SPINAL ACCESSORY NERVE.

The accessory portion has been previously considered in describing the laryngeal branches of the pneumogastric.

In the spinal portion the nuclei may be involved in degenerative lesions of the motor region of the spinal gray matter. The nerve-trunk may be damaged by pressure from exudative products (meningitis), tumors, or caries, with resulting paralysis and wasting of the sternomastoid and, in part, of the trapezius. This latter muscle is also supplied by the cervical nerves. The patient has difficulty in rotating the head to the side opposite that on which the paralysis exists, and the affected muscle does not stand out in movements of the head. Unless secondary contraction of the unopposed muscle sets in, no deviation oc-

curs when the head is at rest. The only portion of the trapezius that is involved in paralysis of the external part of the eleventh nerve stretches from the occipital bone to the acromion. The normal contour of the neck is lost in such cases, and the ability to raise the arm is interfered with because the trapezius cannot fix the scapula, the fulcrum of the deltoid. Bilateral paralysis may occur as in progressive muscular atrophy; if both sterno-mastoids are involved, the head falls backward; if both trapezii, it falls forward.

The **treatment** is that of the underlying cause. If the lesion is nuclear, practically nothing can be done. If the condition is due to pressure, in some cases relief may be obtained. Electricity and massage should be employed during the recovery of the nerve.

DISEASES OF THE HYPOGLOSSAL NERVE.

The nucleus of the twelfth cranial nerve is in the most posterior portion of the floor of the fourth ventricle. It is said by some observers that the nuclei of the fibers for the palate and vocal cords that run in the spinal accessory nerve may be in the lower part of the twelfth-nerve nucleus.

The cortical center for this nerve is in the lower part of the ascending frontal convolution, in the neighborhood of the cortical facial center. This propinquity probably explains the simultaneous involvement of the facial and lingual muscles in some cases. The hypoglossal is the motor nerve for the tongue and for most of the muscles attached to the hyoid bone. Spasm or paralysis may follow disease of the nerve.

Spasm may be either unilateral or bilateral. It is probably met with most commonly in hysteria, or as a part of some general convulsive condition, as epilepsy or chorea. It may also be associated with facial spasm, as mentioned above. Irritation of the fifth nerve (dental caries, ulceration of the gums) seems to be responsible for some cases. "Paroxysmal clonic spasm" is a form in which the tongue is rapidly thrust in and out (p. 1216). Various sensations in the affected region may precede the attack. A rare form—*aphthongia*—is induced when an attempt to speak is made. The prognosis in this condition is good, and a general tonic treatment is indicated.

Paralysis may result from supra-nuclear, nuclear, or infra-nuclear lesions.

Supra-nuclear.—The lesion may be anywhere between the cortex (lower part of the ascending frontal gyrus) and the medulla, and causes paralysis on the opposite side. In this condition the affected muscles do not atrophy nor do they show any electric change.

Nuclear.—The lesion is usually degenerative. It may either be of sudden onset (vascular), less rapid, but still acute (inflammatory), or it may be chronic, as in bulbar palsy or tabes dorsalis. The nuclei are so close together that the condition is almost invariably bilateral.

Infra-nuclear.—The fibers may be injured by the pressure of neoplasms or by the products of meningitis or of syphilis. Disease of the bone may also involve the nerve in its passage through the foramen. More rarely, some traumatism or disease of the upper cervical vertebræ may simultaneously injure the eleventh and twelfth nerves.

Symptoms.—Paralysis and atrophy of one or both sides of the tongue and fibrillary twitchings may be noted, and if the condition be unilateral, the tongue when protruded deviates toward the affected side. Articulation, mastication, and swallowing are but very slightly interfered with. In the bilateral form, however, these are very much impaired; the tongue cannot be protruded and lies motionless on the floor of the mouth. The atrophy is muscular. This throws the mucous membrane into deep folds. Sensation and taste are unaltered.

Diagnosis.—If the lesion is supranuclear, there is usually hemiplegia on the same side as the lingual paralysis, without atrophy of the tongue muscles. When nuclear it is, as has been said, generally bilateral and forms part of a bulbar paralysis. There is also wasting of the lingual muscles. When the fibers are involved in the medulla there is paralysis of the tongue on one side, of the limbs on the other, and the tongue deviates from the paralyzed side of the body. Outside the medulla the condition is, as a rule, unilateral, and the spinal accessory fibers are frequently involved. In the nuclear and intranuclear varieties there is wasting of the muscles.

The **prognosis** is usually unfavorable, and the **treatment** consists of a course of general tonics and of mercury and the iodids, with counter-irritation.

DISEASES OF THE SPINAL NERVES.

DISEASES OF THE CERVICAL PLEXUS.

Phrenic Nerve.—This nerve is usually involved as a result of some lesion of the ganglion-cells in the anterior gray horns at the level of the third or fourth cervical segments. The trunk may be damaged by pressure, as by aneurysm or neoplasms, or by traumatism, or it may be the seat of neuritis. More or less immobility of the diaphragm follows, amounting in some cases to complete paralysis. This is not readily seen with the patient at rest, and in women it is specially hard to observe, as their breathing is chiefly of the costal type. The abdomen moves in during inspiration, and out during expiration, forming the reverse of the normal movements. Exertion readily causes dyspnea, and pulmonary diseases are apt to be exaggerated as the products of secretion accumulate. If the lesion is bilateral, death occurs in a very short time after distressing dyspnea. Neuritis of one phrenic nerve has been observed, and leads to high position of the diaphragm on the affected side, with collapse of the corresponding lung. The *x*-ray may assist in the diagnosis.

DISEASES OF THE BRACHIAL PLEXUS.

This may either be involved *in toto*, or any of its branches may be affected separately, or the nerve-roots that unite to form the brachial plexus. Isolated disease of any of the roots may be produced by injury, caries of the vertebræ, or meningeal disease. The symptoms will be almost exactly the same as those produced by disease of the corresponding segment of the cord, but are more likely to be unilateral, and to be either purely motor or sensory, unless the lesion is extensive.

The posterior thoracic supplies the serratus magnus muscle. It may be injured directly by pressure, as in the carrying of heavy loads on the shoulder or by a fall or other traumatism. Rarely, it follows exposure to cold. Its involvement may be a part of an anterior polio-myelitis or of a progressive muscular atrophy. When the muscle is paralyzed the posterior edge of the scapula stands out prominently, and particularly when the arm is moved forward. Neuralgic pains in the neck generally precede the neuritis. The course of the disease is always slow. During the early stage counter-irritation, the iodids and mercury internally, and later electric stimulation to keep up the tone of the muscles, constitute the treatment.

Combined Paralysis.—Two or more nerves, or even the entire plexus, may be involved at one time by new growths in the cervical region, neuritis, stretching or rupture of the nerves by wounds, fractures, or dislocations, particularly by subcoracoid dislocation and stoop shoulders, which cause compression of the axillary structures between the humerus and the ribs.[1] Duchenne has described a form of palsy produced in infants during birth, due to laceration of and hemorrnage about the nerve-fibers by severe traction on either the head when the shoulders are obstructed or on the shoulders in breech presentations. The roots involved are usually the fifth and sixth, frequently just at their junction. The muscles involved are the deltoid, biceps, brachialis anticus, supinator longus, infra- and supra-spinati. Other roots and muscles may also be affected. This condition is known as "obstetric paralysis." Brachial neuritis may follow some injury to one of the nerve branches (ascending neuritis) or it may be primary. The latter variety is rare and usually occurs after middle life, especially in cases with a gouty history. Paroxysmal or continuous pain, increased by any movement of the arm and tenderness on pressure over the affected nerves, is the chief symptom. If on the left side, it simulates angina pectoris.

Individual Nerves of the Arm.—These may be damaged by pressure due to a tumor, an aneurysm, or to callus. *Sleep-palsy* and *crutch-palsy* are both pressure-palsies. The nerves may also be contused or torn in fractures or dislocations, and palsy may follow a fall or blow upon the shoulder; I have seen it occur in a heavy man after a fall upon the hand. Primary or secondary neuritis may develop, and, very rarely, neuromata appear.

The *supra-scapular* nerve supplies the supra- and infra-spinati muscles. Paralysis causes imperfect outward rotation of the humerus and rotation of the scapula, with elevation and inversion of the lower angle. Various movements of the arm are thereby interfered with, and the limb tires very readily. More work is thrown on the deltoid, and in time it hypertrophies, causing it to stand out more prominently against the infra-spinatus. The skin over the scapula is usually anesthetic.

The *circumflex* nerve supplies the deltoid and teres minor and the skin over the deltoid and the shoulder-joint. Paralysis results in inability to raise the arm and in wasting of the muscles, with or without anesthesia. Adhesions may form in the joint (p. 1263).

The *musculo-spiral* nerve is more often paralyzed than any other nerve of the arm, its position rendering it particularly liable to pressure.

[1] Goldthwaite, *Jour. Amer. Med. Assoc.*, Sept. 11, 1909, p. 852.

It supplies the triceps and supinator muscles, and is the extensor nerve of the arm. It also supplies the skin on the radial side of the forearm, dorsal surface of the thumb and hand, corresponding to the index and middle fingers. A lesion high up results in paralysis of the extensors of the elbow, wrist and hand, and of the supinators. Probably the point most commonly attacked is about the middle of the humerus. In such cases the triceps escapes. The characteristic symptoms, however, are wrist-drop and finger-drop, consisting of an inability to extend the hand on the forearm, also the first phalanges of the fingers and thumb. In pressure-palsies, usually due to sleeping with the head upon the arm, particularly after the excessive use of alcohol, the power of supination is usually lost also. Sensory symptoms vary and are seldom pronounced. There may be slight impairment or tingling or burning sensations.

This condition can usually be differentiated from lead-palsy by the rapidity of onset—by the fact that pressure-palsies are almost invariably unilateral, and that the supinators are involved. Lead-palsy has a slow onset and is bilateral, generally without supinator involvement. Loss of sensation precedes the pressure-palsy. The history too will generally throw some light on the case. I have seen a case of right-sided unilateral wrist-drop in a man who worked in lead with his right hand only. Bilateral wrist-drop may occur in any form of toxic neuritis, but the involvement of other nerves, the manner of attack, and the history of the case will serve to simplify the diagnosis.

Recovery follows in almost all cases of musculo-spiral nerve-involvement, though in cases in which qualitative nerve-changes have taken place it is necessarily delayed.

The *treatment* is that of neuritis.

The *median nerve* supplies the pronators, digital flexors, except the ulnar half of the deep flexor, the radial flexor of the wrist, the abductor and flexor muscles of the thumb, and the two radial lumbricales. It furnishes sensation to the radial side of the palm and front of the thumb, and to the front and back of the first and second and half of the third fingers. This nerve may be the seat of an injury or of neuritis, but is seldom involved alone. A form described by Hunt[1] is due to pressure upon the nerve at the base of the thenar eminence, the principal symptoms being atrophy and paralysis of the muscles forming it. Localization in this group distinguishes it from a beginning progressive muscular atrophy (p. 1142). The most striking symptoms (median nerve palsy) are wasting of the thenar eminence and an inability to oppose the thumb to the tips of the fingers. Loss of pronation of the forearm. Ulnar flexion of the wrist alone remains. Flexion of the second phalanges upon the first is interfered with. Sensation may or may not be lost.

The *ulnar nerve* supplies the ulnar flexor of the wrist, the ulnar half of the deep flexor of the fingers, the muscles of the little finger, the adductor and inner head of the short flexor of the thumb, the interossei, and some of the lumbricales. It supplies with sensation the front of one and a half and the back of two and a half fingers on the ulnar side. Paralysis causes radial deviation of the hand in flexion of the wrist, loss of adduction of the thumb, and inability to move the little finger. The hypothenar prominence disappears.

The first phalanges cannot be flexed, and the second and third can-

[1] *Journal Nervous and Mental Diseases*, January, 1910, p. 46.

not be extended. This is exaggerated in old cases, though still it is not so marked as the "claw hand" of progressive muscular atrophy, since the first two lumbricales escape, being supplied by the median nerve. Sensory symptoms vary. If the deep palmar branch is alone affected, as it may be by pressure, as it passes between the tendinous origins of the abductor minimi digiti and flexor brevis minimi digiti, the sensory symptoms are absent.[1] Care must be taken not to mistake this condition for the beginning of a progressive spinal muscular atrophy (p. 1142).

The *diagnosis* is usually easy. It is well to remember that, since this nerve is the lowest in its point of origin of any considered in this group, ascending cord-diseases will involve it before any of the other brachial nerves. It may also be damaged by disease limited to the lowest part of the cervical enlargement of the cord.

DISEASES OF THE LUMBAR AND SACRAL PLEXUSES.

The **lumbar plexus or its branches** may be involved by abdominal growths, enlarged glands, psoas abscess, disease of the vertebræ, neuritis, and rarely by wounds or dislocation of the hip or during parturition.

The Obturator Nerve.—When the power of adduction of the thigh is lost and the affected leg cannot be crossed over the other, outward rotation is somewhat impaired.

Anterior crural nerve paralysis causes loss of power and wasting of the extensors of the knee, loss of knee-jerk, and anesthesia of most of the thigh and the inner side of the leg and foot.

The *superior gluteal nerve* supplies the gluteus minimus and medius muscles. When it is involved adduction and circumduction of the thigh are lost.

The **sacral plexus and its branches** may be damaged by pelvic neoplasms or inflammation, neuritis (generally secondary to sciatic nerve-involvement), pressure during labor, wounds, dislocations, aneurysms, and diseases of the bone.

The *small sciatic nerve* supplies the gluteus maximus muscle. It is seldom involved alone. Lesions cause difficulty in rising from the sitting posture and anesthesia of the back of the thigh and of the upper part of the leg posteriorly.

The *great sciatic nerve* supplies the flexors of the leg and the muscles below the knee, and also sensation to the outer half of the leg, the sole, and part of the dorsum of the foot. Paralysis causes more or less interference with the act of walking, anesthesia in the part supplied, and wasting of the muscles. More or less weakness of them may sometimes be discovered in sciatica. (See Sciatica, p. 1082).

The *external popliteal* or *peroneal nerve* supplies the tibialis anticus, the peronei, the long extensor of the toes, and the extensor brevis digitorum; it also supplies sensation to the outer half of the front of the leg and to the dorsum of the foot. Paralysis causes foot-drop and toe-drop, rendering it necessary to lift the leg high in walking, so that the foot will clear the ground; this constitutes the *steppage gait* referred

[1] Hunt, "Occupation Neuritis of Deep Palmar Branch of Ulnar Nerve," *Jour. Nerv. and Ment. Dis.*, Nov., 1908, p. 673.

to in the section on Neuritis. If sensory impairment is present it will be found on the outer half of the front of the leg and the dorsum of the foot.

The *internal popliteal nerve* supplies the popliteus, tibialis posticus, the calf muscles, the long flexors of the toes, and the muscles of the sole. When paralyzed, flexion of the foot and toes is impossible, and sensation is lost over the back of the leg in its lower part and over the sole. In old cases talipes calcaneus results. The plantar nerves are rarely, if ever, involved alone. Disease of the plexuses outside the canal must be distinguished from lesions inside involving the cauda equina (p. 1159), most commonly these are either a fracture dislocation of one or more lumbar vertebræ below the first; hemorrhage, or tumor. If the first, the diagnosis is easy, as the fractured vertebræ can be easily recognized by inspection, and in doubtful cases, the skiagram. A history of traumatism is important in both fracture and hemorrhage, and the symptoms are usually bilateral, but not always strictly symmetrical; the pain is severe. Tumor would be indicated by a slow but progressive development of atrophic paralysis, absence of reflexes, sensory paralysis, intense sacral pain of a radiating character, and often tenderness in the same region. There is also usually sphincter paralysis.

Solution of continuity in an *intercostal nerve*, as in a fracture of a rib, rarely gives rise to any symptom except a small area of anesthesia at the sternal end of the corresponding interspace.

II. INFLAMMATION OF THE MENINGES.

MENINGITIS is very rarely a primary condition. Both the dura and pia may be involved. In the former case the inflammation is usually due to some morbid condition of the vertebræ, while in the latter it is secondary to some infection, as in pyemia, sepsis, pneumonia, typhoid, or the acute exanthemata. It may be part of a tuberculous condition (*vide* Tuberculosis, p. 249) or of epidemic cerebrospinal meningitis (p. 95). Injuries also lead to inflammation of the meninges of the cord.

INFLAMMATION OF THE DURA MATER.

CEREBRAL PACHYMENINGITIS.

Inflammation.—This may be met with on the outer or inner surface (*pachymeningitis externa* or *interna*). Of the external variety the chief causes are (*a*) traumatism, (*b*) disease of the bone, (*c*) syphilis, and (*d*) middle-ear disease. That due to traumatism is often seen, and in the mildest form is of little moment. When severe and accompanied by fracture with or without displacement, infection of the membranes may either take place at once or later from diseased bone. That form due to caries or any other form of osteitis is always dangerous, owing to the possibility of infection of the diploë. The brain-sinuses will then become affected, and infected emboli may pass into the circulation, with the development of pyemia. In the syphilitic variety the inner table of the skull is thickened and roughened, and more or less pus and granular

material is found between it and the dura (see also Syphilis of the Nervous System). Sinuses may communicate with the exterior.

The **symptoms** are indefinite in mild cases, and may consist only of *headache.* In the severe forms there are *headache, malaise, chills, fever, drowsiness*, and later *stupor*, and rarely *convulsions, paralysis*, or other symptoms of compression. The ophthalmoscope will reveal more or less evidence of *choked disc.* Rigors are suggestive of the onset of pyemia.

The **treatment** varies with the cause. Antiphlogistic measures and counter-irritation are of value, and in the severe grades operative interference may be necessary. The internal variety either occurs as a simple inflammation or may be so acute as to cause extravasation of blood. This may organize, and, together with the products of inflammation, cause a pseudo-membrane. Rarely is pus found.

Internal hemorrhagic pachymeningitis, or hematoma of the dura mater, is characterized by the formation of a fibrous exudate upon the inner surface of the dura, into which capillaries extend that subsequently rupture. It is found most commonly among alcoholics, the insane, and epileptic. It is rare in childhood.

The **symptoms** are variable. The entire course may be without symptoms, or they may be marked by the existence of other conditions. More frequently there are *headache* and *convulsions*, followed later by *paralyses*, coma, and death. The location of the lesion causes considerable modification of the symptomatology. In the milder form recovery frequently occurs, or the case may become chronic. If the onset is sudden, the symptoms may resemble those of hemorrhage.

The **diagnosis** is always difficult. In children muscular contractions and convulsions are frequently met with; in adults the slow onset may be the only difference between this condition and an attack of *grand mal.* Of course, there is a greater periodicity in epilepsy; but a repetition of the attacks occurs in hematoma, and, as already stated, the repeated hemorrhages are believed by some to be the cause of the lamination of the false membrane.

The **prognosis** is extremely unfavorable in children, but is much less so in adults.

The **treatment** calls for the use of leeches behind the ears and over the temples, the ice-cap, and counter-irritation. Free movement of the bowels should be promptly secured, and later the iodids or mercurials should be administered.

SPINAL PACHYMENINGITIS.

Definition.—Inflammation of the dura mater. The dura may be involved on its outer or inner surface (*pachymeningitis externa* or *interna*), or the loose connective tissue between the dura and bony canal may be the seat of a peripachymeningitis.

Pachymeningitis externa is always secondary, and usually results from disease of the vertebræ, due to syphilis, tuberculosis (Pott's disease), or malignant disease, or from pressure due to tumors or to traumatism. It may either be acute or chronic. Of the latter type, those cases due to Pott's disease are most common. The membrane is involved to a greater or less extent. The internal surface may escape entirely, or it may be slightly roughened and adherent to the arachnoid; externally, however, the dura is usually thickened, rough, and covered with a cheesy material.

Pachymeningitis interna was first described by Charcot in 1871, and named "*pachymeningitis cervicalis hypertrophica.*" It is of obscure origin, but traumatism, alcoholism, and syphilis have been given as causes. The dura is generally much thickened, and gives the impression of being made up of a number of concentric layers. Hemorrhages may occur within the dura or within the newly formed tissue. The pia is only involved to a slight degree, as a rule, but becomes adherent to the dura. Areas of degeneration may occur in the cord, as may also dilatation of its central canal. As implied by the name, this variety of pachymeningitis is found chiefly in the cervical region, and the clinical symptoms result from involvement of the nerve-roots and compression of the cord. It is a chronic process, and has been divided into three periods, as follows: (*a*) *The painful period*, lasting, as a rule, two or three months, in which severe neuralgic pains exist, their location being determined by the roots involved. They are mostly in the occiput and upper extremities, however. Early there may be hyperesthesia, numbness, tingling, and, rarely, an herpetic eruption. (*b*) *The Paralytic Period.*—As a result of compression of the motor roots an atrophic paralysis of the upper extremities develops. A peculiar selective tendency is manifested, the distribution of the median and ulnar nerves being principally involved. This results in a modified "claw-hand" deformity and in an overextension of the wrists, with flexion of the fingers. Anesthesia may be noted. (*c*) *Spastic Paraplegia.*—This results when the compression has produced degeneration of the cord. Generally, there are paresis of the lower extremities and increased reflexes, but no muscular wasting, since the trophic centers are intact. Occasionally, however, anesthesia and paralysis of the legs and bladder develop, bed-sores following, and finally death from exhaustion.

The *prognosis* is unfavorable, practically all cases terminate in death, but the duration is variable.

The *diagnosis* must be made from amyotrophic lateral sclerosis, vertebral caries, syringomyelia, and from pressure by tumors. Amyotrophic lateral sclerosis does not give rise to sensory disturbances; bulbar symptoms are often present, the lower extremities may atrophy, and the bladder functions are preserved. In vertebral caries tenderness on jarring will be present, and a skiagram will probably show evidence of bone disease. Syringomyelia induces characteristic symptoms of loss of temperature and pain sense with the preservation of tactile sense (dissociation of sensation), although this may rarely be present in pachymeningitis, but severe neuralgic or radiating pains are rare in syringomyelia. From tumor the diagnosis may be difficult, the symptoms in this, however, usually develop more gradually and at first are unilateral.

Treatment is not of much avail. Potassium iodid and mercury are the chief measures. In cases otherwise hopeless an exploratory operation is sometimes justifiable.

Pachymeningitis hæmorrhagica interna, or *hematoma of the dura mater*, may occur in any part of the cord, and is usually associated with a similar condition in the cerebral dura. Cysts may be found in the inner surface of the dura, containing broken-down blood-cells and hematoidin crystals, and in their neighborhood an increase of fibrous tissue may be noted. The condition occurs most frequently in alcoholic or general paralytics.

LEPTOMENINGITIS.

Definition.—Inflammation of the pia mater. This may be either acute or chronic.

CEREBRAL LEPTOMENINGITIS.

CEREBRAL leptomeningitis is an inflammatory condition of the pia arachnoid; it occurs in various forms, that may be classified either according to the distribution of the process, into meningitis of the convexity, of the base, or cerebrospinal meningitis, or according to the cause.

Etiology.—As it is infectious, this is always micro-organismal. It is customary to distinguish between the forms produced by the pyogenic micro-organisms and by the tubercle bacilli. Among the former the most important are the pneumococcus, the meningococcus, the staphylococcus, and the bacillus of influenza, but a great variety of other bacteria have been found, such as the colon bacillus, the typhoid bacillus, and others in rare or isolated instances. The method of access to the meninges varies, either along the blood or lymph channels from some focus of infection, as the lungs, the nasal cavities, or in the course of an infectious process that gives rise to bacteremia, as pyemia; or by direct extension, as in middle-ear disease, or disease of the sinuses of the face. Meningitis, particularly the tuberculous variety, may follow injuries to the head. It may be due to syphilis. Purulent meningitis may occur at any age. Tuberculous meningitis is more common in childhood.

Pathology.—In the extent and degree of the inflammation, great variations exist. It may be either (1) limited to the convexity, with or without involvement of the sides; (2) limited to the base; or (3) general, involving both convexity and base. In the early stages and in the mild forms there may be no more than an injection of the part. Later, inflammatory products are met with, usually following the course of the meningeal vessels, but sometimes covering considerable areas. This form of leptomeningitis, unlike the tuberculous variety, is prone to attack the convexity of the brain.

Symptoms.—These are very varied, and naturally depend on the *seat* and *extent* of the inflammation. Those cases in which symptoms pointing to involvement of the base occur need not be discussed here, since they are considered in detail under the tuberculous variety. In any case *headache*, localized or general, is usually present. In children too young to talk its presence is often indicated by crying or putting the hand to the head. *Delirium*, *insomnia*, and *coma* are also met with in different cases. There is more or less *fever*. *Constipation*, a *coated tongue*, *vomiting*, a *rapid pulse*, are usual, and the *tâche cérébrale* may be elicited. Spasmodic movements may occur, or even general *convulsions*. Of course, in cases of inflammation of the base, the cranial nerves become affected, and we have *ptosis* or *strabismus*, *facial spasm* or *palsy*, and, if the fifth nerve is involved, *sensory* and *trophic* changes. The head is usually retracted until it seems to bore into the pillow; the muscles of the back of the neck are tense; the spine is often rigid; the abdomen retracted, and the limbs flexed. The tendon reflexes are exaggerated and cutaneous irritability greatly increased. *Kernig's sign* consists in

the inability of the patient to straighten the leg, when the thigh is flexed to a position of 90 degrees to the axis of the body. It is nearly always present in acute non-tuberculous meningitis, but often absent in the tuberculous form; occasionally it may be found in focal encephalitis, either acute or chronic, and even in acute infectious disease—typhoid fever. It is, therefore, valuable as a suggestive sign of meningitis, but can no longer be considered pathognomonic. A type frequently found in young children, and thought by many to be a form of sporadic cerebrospinal meningitis (p. 95), consists of an inflammation confined to the meninges of the posterior part of the base of the brain from the optic commissure to the medulla. From its location hydrocephalus frequently develops; blindness, due to pressure on the optic chiasm, also is common. The intense retraction of the head is a characteristic symptom. It is known as *posterior basic meningitis*.

Diagnosis.—Where no etiologic hint can be obtained the diagnosis is generally in doubt for two or three days. There may be nothing more than a reflex irritation (dental or gastro-intestinal), or possibly one of the infectious fevers. The symptoms should be studied in their entirety; one or two supposedly pathognomonic signs should not be allowed to cloud our vision. In some cases it may be necessary to distinguish meningitis from the so-called *serous meningitis* (p. 1123). Meningitic symptoms, associated with marked delirium, may occur in the course of acute articular rheumatism. In this condition the cerebrospinal fluid will be clear (p. 1123). The condition is known as cerebral rheumatism. It must also be borne in mind that meningeal symptoms are simulated by the infectious diseases (p. 1123). Having made the diagnosis of meningitis, it becomes important to **differentiate** the *tuberculous* from the *non-tuberculous* variety. The family history is of importance. In *tuberculous meningitis* the focal symptoms usually appear early, and are due to involvement of the cranial nerves at the base of the brain, chiefly those controlling the eye. The eye-grounds often show a slight perineuritis without choked discs, and perhaps one or more miliary tubercles. There is sometimes a mild form of confusional delirium, often preceding the appearance of focal symptoms. The leukocytes are slightly, if at all, increased. There is rarely rigidity of the neck. In other forms of meningitis this appears early; the optic nerve shows intense inflammation and there is usually pronounced leukocytosis. Examination of the fluid withdrawn by lumbar puncture (p. 1124) is important. A differential count of the leukocytes in the spinal fluid should always be made (Cytodiagnosis, see p. 1124).

Prognosis.—This is always grave. A percentage of cases of epidemic cerebrospinal meningitis, varying with the severity of the epidemic, may recover. In all other forms any termination, except in death, is exceedingly exceptional. Remissions frequently occur in the symptoms, and the course may be very prolonged.

Treatment.—We have no specific, and all that can be done is to meet the symptomatic indications. Absolute quiet in a darkened room, an ice-cap to the head, and the internal use of full doses of hexamethylenamin (urotropin) may be of service. Opium may have to be given for pain. If of syphilitic origin, either mercury should be used by inunction or injection or salvarsan (606) employed. In certain cases—*e. g.*, those secondary to middle-ear disease—either opening the skull above

and below the tentorium and draining,[1] or the operation of Haynes,[2] which consists in draining the cisterna magna in the posterior fossa, may seem justifiable. When in doubt the physician should not delay action until too late, but should call in a surgeon while there is still hope for some benefit.

The treatment of the epidemic and tubercular forms is detailed on pages 95 and 249.

SEROUS MENINGITIS.

(*Meningitis Serosa; Wet Brain.*)

THIS condition, first described by Quincke, which in the acute form may arise spontaneously or follow various infective processes. Chronic alcoholism is a frequent cause. The symptoms resemble those of acute cerebral meningitis, and lumbar puncture may be necessary to establish the diagnosis, in serous meningitis the fluid being clear, not containing organisms, and escaping under great pressure. The more chronic type closely simulates tumor of the brain, especially a subtentorial growth, as paralysis of cranial nerves, choked disc, convulsions, and ataxia may all be symptoms of this condition.

The *symptoms* are apt to fluctuate, and if they develop acutely after an infection of some sort it would be in favor of serous meningitis. In some cases an inflammation of the ependyma exists, and internal hydrocephalus (p. 1190) may follow.

The *prognosis* is doubtful, but recovery takes place in a fair proportion of cases. In the alcoholic type the greater the rigidity and retraction of the head the worse the outlook.

Quincke advises mercurial inunctions in all cases. Counter-irritation to the back of the neck and cold to the head may also be used. Lumbar puncture is of great service. The bowels should be kept free and the diet liquid, but liberal in quantity (hot milk, eggs, broths). In the alcoholic cases the inunctions should be omitted and strychnin in full doses (gr. $\frac{1}{60}$ every three hours) given.

ACUTE SPINAL LEPTOMENINGITIS.

(*Acute Spinal Meningitis.*)

Etiology.—This is always microörganismal, and a great variety of bacteria have been discovered. The most common is the pneumococcus, in which case the disease may or may not be associated with pneumonia; next in frequency is the meningococcus; and then the various pyogenic cocci, the influenza bacillus, the typhoid bacillus, etc. It may be due to syphilis; rarely it may be tubercular.

Pathology.—The vessels are injected, the membrane becomes cloudy, a sero-fibrinous or purulent exudate either surrounds the cord or may only exist in patches, and in the more severe cases the cord itself is involved (*meningomyelitis*). The spinal meninges alone may be involved to a greater or less extent, but, as a rule, the cerebral meninges

[1] Day, *Annals Otology, Rhinology, and Laryngology*, June, 1911.

[2] *Trans. Amer. Laryngol., Rhinolog., and Otolog. Society*, 1912, and *Progressive Medicine*, Mar., 1913, p. 43.

are similarly involved. Tubercles will be found in the tubercular form. It should be remembered that many cases presenting clinically the picture of meningitis show absolutely no gross *postmortem* lesions of the cerebral or spinal membranes. This may occur from any toxemia, but especially in pneumonia, typhoid fever, influenza, and rheumatism no lesions, not even microscopic, are found. These are spoken of as *meningismus*.

Symptoms.—These are chiefly pain in the back, often excruciating, with fixation, retraction of the head, tenderness on pressure along the spine, tremors or spasm of the muscles, and various sensory disturbances. Reflexes are early increased, and later diminished or absent. Should the cord be involved, paralysis, incontinence of urine and feces, and even bed-sores, may develop (p. 1135). The symptoms are more fully discussed in speaking of the tuberculous and epidemic varieties.

Diagnosis.—It is often very difficult to differentiate the several varieties of spinal meningitis, and equally so to decide whether the case is actually meningeal when some other disease is present. The tuberculous form is readily diagnosticated, especially if any collateral evidence of tuberculosis exists. It is a point of some value in the diagnosis to note the absence of marked leukocytosis in the cerebrospinal fluid obtained by lumbar puncture in tuberculous and its presence in purulent meningitis. The presence of *Kernig's sign* is in favor of cerebrospinal meningitis.

Spinal paracentesis or *lumbar puncture*, first introduced by Quincke of Kiel in 1891, is a most valuable diagnostic measure and simple of application. It is performed as follows: The patient either sits up or lies preferably upon the left side, with the back arched and the knees flexed against the abdomen. The spine of the fourth lumbar vertebra should be located (a line drawn from one posterior superior spine of the ilium to the other passes across it), and the puncture made half an inch to one side, at the level of its lower end. The needle should be inclined at an angle of about 45 degrees to the surface of the skin, and should be thrust in a distance of from 2½ to 3 inches. The most scrupulous asepsis must be observed. The spinal fluid flows readily, either in a stream when the pressure is high, or drop by drop if it is normal. In purulent meningitis it is cloudy, contains pus-cells, and does not reduce Fehling's solution; in tuberculous meningitis it is usually clear and does reduce Fehling's solution; in cerebral hemorrhage it may be bloody, but as admixed blood may be due to the injury of a vessel by the needle, this sign should be used with caution. The quantity obtained varies from 2 or 3 to 80 or 90 c.cm. After centrifugation a differential count of white cells should be made. An excess of lymphocytes indicates a tubercular infection; an excess of polymorphonuclear cells, a pyogenic infection. Cultures should be made and the sediment or coagulum stained for bacteria. In meningismus the fluid is practically normal. Often there is great relief from the puncture, and occasionally, in serous meningitis, the patient appears to be permanently benefited.

The **prognosis** is unfavorable as a rule, particularly in the tuberculous form.

The **treatment** is the same as that of cerebro-spinal meningitis (*vide* p. 103).

CHRONIC LEPTOMENINGITIS.

This disease may follow the acute form or be due to chronic alcoholism, syphilis, trauma, or disease of the cord.

Pathology.—The pia is cloudy and swollen, and often adherent to the arachnoid, or all three membranes may be glued together. They are usually injected. Usually there is considerable proliferation of fibrous tissue. The periphery of the cord is also occasionally affected. A condition known as *circumscribed serous spinal meningitis* may be here mentioned. It may follow either traumatism or infectious diseases. When the dura is slit, its opening is filled by a more or less opaque pia that bulges forth under tension of the contained fluid which is clear.[1]

Symptoms.—These are not well marked. Unless the nerve-roots are involved the symptoms are slight or none at all exist; however, pains of a radiating character, stiffness, tremors, hyperesthesia, herpes, and even paralyses, may occur. The course is slow, and may extend over many years. Idiopathic circumscribed spinal serous meningitis is described in connection with spinal tumors, as the symptoms of the two are practically the same (p. 1157).

The *prognosis* is unfavorable ultimately.

The *treatment* consists in the use of iodids and mercury internally, and the application of baths, and counter-irritation along the spine.

III. DISEASES OF SPINAL CORD.

HEMORRHAGE INTO THE SPINAL MENINGES.

(*Meningeal Apoplexy; Hematorrachis.*)

(*a*) **Extrameningeal hemorrhage** occurs when the blood is between the dura and spinal canal.

(*b*) **Intrameningeal hemorrhage** is that in which the bleeding takes place beneath the dura.

Large hemorrhages are more common in the extrameningeal form; they result from trauma or rupture of an aneurysm. The peridural space will accommodate a large amount of blood without giving rise to pressure-symptoms. Caries of the vertebræ or carcinoma may cause hemorrhage by erosion and rupture of a blood-vessel. The intra-meningeal form is common, and may result from meningitis, from trauma, or may occur as a complication of any of the infectious or hemorrhagic diseases. In such cases the hemorrhages are small and scattered. It may also occur in convulsive disorders or in strychnin-poisoning. Rupture of an aneurysm at the base of the brain may give rise to extensive hemorrhage; blood may also pass into the spinal cerebrospinal fluid from either a ventricular or subdural cerebral hemorrhage.

Symptoms.—When the hemorrhage is large enough to cause pressure the symptoms are very acute, apoplectiform indeed, but consciousness is preserved. Generally, however, they are quite indefinite. In any case they depend upon the degree and location of the compression. At first they are irritative—viz., hyperesthesia, paresthesia, neuralgic pains that

[1] Weisenburg, *Amer. Jour. Med. Sci.*, Nov., 1910, p. 719.

are radiating in character, muscular irritability, tremors, or contractions. If subdural, the fluid obtained by lumbar puncture will probably contain blood. Later, paralytic symptoms may develop, as anesthesia and bladder- and bowel-symptoms, girdle pains, or, when the lesion is high up, interference with respiration and pupillary changes.

The **diagnosis** is often difficult, unless the onset is sudden and explosive.

The **prognosis** depends on the cause and extent of the hemorrhage. If small in amount, absorption is usually prompt, with little or no disturbance of function remaining.

The **treatment** consists of rest, ice to the spine, and morphin to relieve pain; later mercury and the iodids may be given to hasten absorption. Local measures, such as leeches, cupping, etc., or general styptics, such as ergot and calcium chlorid, are of very doubtful value; although the latter may be used if the hemorrhagic diathesis is present. In certain cases operative procedures, with a view to removing the clot, may be justifiable.

HYPEREMIA AND ANEMIA OF THE CORD.

These may be due to qualitative and quantitative changes in the blood, and morbid conditions of the vessel-walls. They give rise to no characteristic symptoms, unless softening or degeneration occurs.

The blood-vessels may be the seat of peri- or endarteritis, and rarely miliary aneurysms may develop. Embolism and thrombosis also occur, the former much less frequently than the latter, which is prone to follow sclerotic changes in the vessels, giving rise to ischemia and ultimately to softening (p. 1138). Degenerative changes in the cord may be caused by anemia (p. 1157).

HEMORRHAGE INTO THE SPINAL CORD.

(*Hematomyelia; Spinal Apoplexy.*)

This is of very much less frequent occurrence than cerebral hemorrhage. It is usually due to traumatism, but may possibly follow some severe strain or overexertion, probably only when the vessels are atheromatous. Hemorrhage may occur in cases of myelitis, epidemic cerebro-spinal meningitis, syringomyelia, tumors of the cord, convulsive disorders, and infectious diseases; it is, however, usually small. If the hemorrhage is extensive, disruption of more or less cord-substance necessarily follows. An area may exist large enough to cause distention of the cord without rupture, and from this extravasations may take place in the cord-substance above and below. Unilateral hemorrhage may occur, the gray matter being chiefly involved. If of recent origin, fresh blood will be found *postmortem;* but if of long standing, a brown or brownish-yellow area will be noted, consisting of disintegrated blood-corpuscles, cell-detritus, and hematoidin crystals.

The *symptoms* necessarily vary according to the region involved, the gray matter of the cervical region being that most frequently affected. The onset is always sudden, the symptoms rarely requiring as long as a half-hour to develop. They consist of a flaccid paralysis of the limbs

below the seat of the lesion, loss of reflexes, and probably of sensibility. The urine and feces will also be retained. Consciousness is not lost. If in the cervical region, contraction of the pupils and narrowing of the palpebral tissues will be observed, owing to involvement of the oculo-pupillary fibers at the eighth cervical and first dorsal segments (cervical sympathetic nerve). If death does not occur, the symptoms gradually more or less subside, and ultimately resemble those of acute myelitis (p. 1135). As the hemorrhage is most often in the gray matter, dissociation of sensation (preservation of tactile and loss of pain and temperature sense) will result; atrophy of the muscles supplied by the affected segments (p. 1071) will also result; below the seat of the lesion the paralysis becomes spastic with increased tendon reflexes. If one side of the cord is principally involved, the Brown-Séquard syndrome (p. 1077) results.

The *diagnosis* must be made in acute cases from meningeal hemorrhage and, if traumatic, fracture of the vertebra. The presence of severe lancinating pain, muscular twitchings, and a less degree of paralysis distinguishes the former. The latter can usually be determined by the characteristic deformity, as determined by inspection and the skiagram—hemorrhage, of course, can coexist with fracture. The residual symptoms, if the patient survive, may be mistaken for syringomyelia and myelitis; the history of traumatism and sudden onset will distinguish it from the former. In the latter it may be difficult, as hemorrhage frequently precedes the development of myelitis in traumatic cases. A history of apoplectiform onset is evidence that the primary condition at least was hemorrhagic.

The *prognosis* during the acute stage is doubtful, death may occur from exhaustion or septic infection due to bed-sores or cystitis. If this stage is survived, a considerable degree of power may return and the patient get about with a more or less spastic paraplegia, and if in the cervical region, atrophic paralysis of the arms and hands.

Treatment.—Rest, ice locally, attention to bladder and bowels, and the internal use of calcium salts and opium make up the treatment of the acute stage; afterward the treatment is similar to that of myelitis (p. 1137).

CAISSON DISEASE.

(*Diver's Paralysis.*)

Definition.—A paralytic condition caused by sudden transference from an abnormally great atmospheric pressure to one of normal intensity.

The **etiology** of the disease is very clear, and certain predisposing factors are worthy of note. Divers are more apt to suffer if they have been working at extreme depths, particularly if the period of exposure to great pressure has been prolonged; every moderate pressure will sometimes produce symptoms if continued for a sufficient length of time, and short periods of rest do not prevent the development of the disease. Ordinarily, it can be said that unless the pressure exceeds two and one-half or three atmospheres no danger may be apprehended. Alcoholism is a predisposing cause.

Pathology.—Two theories have been advanced to account for the condition. One is that under the high pressure the blood becomes overcharged with nitrogen gas; when the pressure is relieved this is liberated, causing emboli, which block up the spinal vessels. The other is that the blood is driven from the surface, causing distention of the vessels with paralysis of their walls; when the air pressure diminishes they are unable to accommodate themselves to the changed condition, and stasis with congestion and hemorrhages result. Both factors are probably accountable. Small hemorrhages and laceration of nerve-fibers have been found in the cord.

The **symptoms** vary greatly in intensity. In the mildest form they consist of *neuralgic pains in the joints*, sometimes with slight articular swelling, *headache*, *giddiness*, and a little *tinnitus*. These pains may become more violent, particularly in the loins, and be followed by a gradual *loss of power* and by *anesthesia* in the limbs; these symptoms may disappear in a few hours or become more severe, with the development of *complete paralysis* and interference with the action of the sphincters. This paralysis usually assumes the form of *paraplegia; monoplegia* and *hemiplegia* also occur, and sometimes there are complete paralysis and anesthesia of all four extremities and of the trunk. In the most severe cases *cerebral symptoms* are also present, consisting of sudden loss of consciousness, profound coma, irregular respiratory action, and finally, after a short time, death from cardiac failure.

The **diagnosis** is very easy. It is possible, however, that an attack of *apoplexy* should occur in a man who has been under water, and the patient should always be examined for the presence of this or some other organic lesion.

The **prognosis** varies with the intensity of the symptoms. The lighter forms consist merely of joint-pains and slight dizziness that usually pass away in the course of a few hours. Paraplegias or hemiplegias, developing slowly and not assuming a severe form, are also transient in character. A more severe paraplegia is usually permanent, although some improvement may be expected. The apoplectic forms are almost invariably fatal in the course of a few hours.

The **treatment** consists, firstly, of prophylactic measures. In all places where caisson-work is carried on one or more locks should be provided in which the pressure can be gradually reduced until it is approximately that of the atmosphere. Divers should be instructed to come slowly to the surface. If the pressure exceeds three atmospheres, the maximum length of the working-period should not be more than one hour, and several hours should be permitted between the descents. A chamber should also be provided in which a man who exhibits symptoms of the disease can be once more subjected to a pressure greater than that of the atmosphere, as this usually causes an arrest of the process. When, however, the condition resembles that of acute myelitis, the treatment is purely symptomatic. It consists of rest, careful hygiene, and a stimulating diet. Stimulating liniments and the rapidly interrupted faradic current may be used for the pain. If the heart is not weak, phenacetin and similar drugs may be used. If it is, strychnin and caffein may prove useful. In the comatose cases enemas of hot coffee should be used and artificial respiration and inhalation of oxygen may be necessary. For the resulting paralysis the treatment is that employed for chronic myelitis.

BULBAR PARALYSIS.

(*Glosso-labio-laryngeal Paralysis.*)

Definition.—Bulbar paralysis is usually termed a disease of the brain, but as the pons and medulla are anatomically and physiologically parts of the cord, it seems more logical to discuss it here. It is an acute or chronic disease, due to involvement of the motor nuclei of the medulla oblongata, and is characterized chiefly by a difficulty of speech or of deglutition. Three varieties have been described:

1. **Sudden** or **apoplectiform**, this being due to hemorrhage, embolism, or thrombosis either of a branch of the basilar or inferior posterior cerebellar arteries. In the latter case anesthesia of the face, ataxia, and other symptoms also occur (p. 1172). The onset is always sudden, often with vertigo, and without loss of consciousness. The power to articulate is impaired or lost. The lips and tongue are involved, and hence the pendulous lower lip, the dribbling of saliva, and the atrophy of the lingual muscles. There are dysphagia and generally frequent attacks of choking.

The *symptoms* are less characteristic than those of the degenerative form. They are less regular in type, and usually are widespread at first; later, some improvement takes place. In other cases, after more or less of a respite, degeneration sets in and they grow progressively worse. Death, however, usually occurs speedily.

The *diagnosis* of this type is not usually difficult. "Pseudo-bulbar paralysis" must be borne in mind, however, and is a condition due to a bilateral lesion of the motor cerebral cortex in the lower frontal parietal region or of the motor fibers in the course. There is great danger to life for some little while in these sudden cases. Later the prognosis is rather more favorable than in the other forms.

2. **Acute Inflammatory.**—Here the onset is less abrupt, requiring a few days to a week to develop, and follows either one of the infectious diseases, the excessive use of alcohol, or lead-poisoning (polio-encephalitis inferior). But for this fact the symptoms are much the same as in the preceding form. It may be associated with acute anterior poliomyelitis (p. 1130).

3. **Chronic Bulbar Paralysis.**—This condition occurs chiefly in males beyond middle life. The cause can seldom be discovered, though certain cases seem to be of toxic origin. It may develop in the course of progressive muscular atrophy, amyotrophic lateral sclerosis, and insular sclerosis.

The *symptoms* are bilateral, the tongue being usually the first to suffer. The patient may notice that he cannot speak for any length of time without fatigue, and that he will then articulate indistinctly. Soon he observes that there is a marked and progressive *impairment of speech*. The muscles of the lips and other muscles of the lower part of the face atrophy. He can no longer whistle. Speech is rendered still more defective, owing to paralysis of the lips. The lower lip drops, and the saliva constantly dribbles from the mouth and may be greatly increased in amount. *Difficulty in swallowing* is always present to a greater or less degree. Owing to the lingual paralysis, the tongue can neither be protruded nor can it be used to manipulate the food and make a bolus. It is atrophied and the mucous membrane is wrinkled. *Fibrillar tremors* are present and reactions of degeneration may occur. The *larynx* is

involved, so that phonation is imperfect, but it is not so marked as the implication of other parts. Particles that enter the larynx cannot be ejected, owing to motor paralysis. There are no sensory symptoms, and the power of taste is normal. The mind generally remains clear, though the patient is often emotional, and cries or laughs without apparent cause. This type of bulbar palsy is particularly liable to develop in the course of either progressive muscular atrophy or myotrophic lateral sclerosis, or may precede them. The *course* of the disease is slow, and death is usually due either to inspiration-pneumonia or to interference with respiration or circulation.

The **diagnosis** is not difficult, as a rule, the bilateral character of the symptoms rendering them distinctive. In the *pseudo-bulbar form* the limbs are often paralyzed also (double hemiplegia). The tongue is not atrophied, the muscles of the face do not show changes in their electrical reaction, and there is usually a history of successive apoplectic attacks (p. 1166). *Tumors* rarely, if ever, give rise to such regular bilateral symptoms. It may also have to be distinguished from myasthenia gravis (see p. 1167).

Treatment.—The disease is incurable. Hypodermics of strychnin, or of strychnin, morphin, and atropin, are of value in controlling the salivary flow. Electricity is of no value. Semisolid food is probably the most readily taken, and it is often necessary either to use an esophageal tube or to employ rectal alimentation.

ACUTE ANTERIOR POLIOMYELITIS.

ESSENTIAL PARALYSIS OF CHILDREN.

(*Atrophic Spinal Paralysis; Infantile Paralysis.*)

Definition.—An infectious febrile disease of more or less rapid onset, associated with muscular paralysis and atrophy, occurring chiefly in children, and most frequently in those under three years of age.

Etiology.—The precise cause is not known, but it is evidently due to a specific infection of unknown nature. The disease has been produced in monkeys by intracerebral inoculation with an emulsion of an affected spinal cord.[1] Monkeys have also been inoculated with the secretions from the nasopharynx of those suffering from the disease. The virus will live in the intestines and pass from the body in the feces.[2] While probably the disease is communicable to some extent by direct contact, the stable fly[3] (Stomoxys Calcitrans) plays a large part in its transmission. The disease may occur at any age, but by far the greatest number of cases occur before the third year of life; they are about equally distributed between the two sexes. Later in life the condition is more common in males, chiefly between the ages of ten and twenty-five. It is rare after this period. Epidemics have been described, and, notably, one occurring during the summer of 1894. Dr. Caverly, of Rutland, Vt.,

[1] Flexner and Lewis, *Jour. Amer. Med. Assoc.*, Jan. 1, 1910, p. 45.
[2] Flexner, Clark, and Dochez, *Jour. Amer. Med. Assoc.*, July 27, 1912, p. 273.
[3] *Jour. Amer. Med. Assoc.*, Nov. 2, 1912, p. 1627.

then reported 126 cases occurring in Otter Creek Valley, a limestone region of Vermont. Similar epidemics have been observed in Norway and Sweden, and in various parts of Europe and the United States. It is especially apt to occur in warm weather. Traumatism may be a predisposing factor. The acute infectious diseases may cause a symptom group resembling the specific disease.

Pathology.—The parenchyma of the heart, liver, and kidneys are the seat of cloudy swelling, and the lymph-nodes and spleen show hyperplasia and proliferated endothelial cells.

Macroscopically the cord is congested and softened with hemorrhages into the anterior horns.

Microscopically the perivascular lymph-spaces are found filled with cells, first polymorphonuclear leukocytes, later endothelial cells and lymphocytes. The vessels are congested, their walls degenerated, with rupture of and hemorrhage from the capillaries. Degeneration and destruction of the ganglion cells and of the nerve-fibers in the anterior roots. A round-cell infiltration of the pia-arachnoid. These changes are most pronounced in the anterior gray matter of the lumbar and cervical enlargements, but the white matter may also be affected. The pons and medulla may also show similar changes.

Symptoms.—The onset is generally acute, and may be sudden. Constitutional symptoms are present as a rule. Generally, the sequence is as follows: Fever (usually slight), malaise, possibly vomiting (especially in children), diarrhea, headache, and restlessness. In a few hours or after one or two days paralysis supervenes and quickly spreads, involving a greater or less area; it then remains stationary for from two to four days to from five to eight weeks, when improvement takes place, beginning in the part last affected. In some cases, after a most trifling indisposition over night, paresis is met with in the morning. In a few weeks only that portion remains paralyzed that is to be permanently damaged. Wasting of the muscles will be noticed a week or two after the onset of paralysis; these become flaccid and give the reactions of degeneration. Sensory symptoms are very rarely present. There may, however, be general hyperesthesia during the early stages, and evidences of meningeal irritation, as rigidity, retraction of the head, etc. The reflexes are lost, both superficial and deep, and later contractures, due to the overaction of unopposed muscles, may develop and result in various deformities. The growth of bone is seriously impaired in some cases.

Diagnosis.—Usually this is not difficult, except, possibly, for the first few days in some cases. Before the occurrence of paralysis in suspected cases examination of the cerebrospinal fluid, obtained by lumbar puncture, is of service. In this stage it may be slightly turbid, but is usually clear, contains an increase of lymphocytes and globulin and sugar, as shown by testing with Fehling's solution. This latter reaction does not occur in the fluid from any form of meningitis except the tubercular. Bacteria are absent, and when paralysis ensues all of these changes disappear.[1] Close scrutiny will enable one to differentiate between this disease and a pseudo-palsy, the result of pain on active or passive motion, as seen in rickets, scurvy, and in hip-joint disease. From multiple neuritis it is distinguished by the absence of tenderness over the nerve-trunks and the fact that in neuritis the symptoms

[1] Flexner and Clark, *Jour. Amer. Medical Assoc.*, Feb. 25, 1911, p. 586.

are progressive and not retrogressive, as they are in poliomyelitis. The symptoms of the cerebral palsies of childhood are given on p. 1174, and of transverse myelitis on p. 1135.

Prognosis.—Some impairment of motion and more or less wasting of the muscles almost invariably remain. Danger to life is usually not great, although death may occur from either involvement of the bulbar nuclei or violence of the toxemia. Marked improvement in power may result several years after the oncoming of the disease if proper treatment is persisted in.

Treatment.—Owing to the possibility of the disease being communicable during the acute stage, the patient should be isolated and the discharges disinfected, as in other similar diseases. The mouth especially should be kept as clean as possible with washes containing formalin. If fever is excessive, cool sponging or an ice-bag to the head may be employed. If pain and headache are severe, lumbar puncture may relieve. If the respiratory muscles are affected, oxygen inhalations are of service. During the acute stage a brisk calomel purge, followed by a saline, is of benefit; and it is necessary to support the general condition. For this reason absolute rest should be enjoined; the diet should be liquid and nourishing, and stimulants should be given freely if necessary. Hexamethylenamin probably exerts an influence in destroying the disease germ. After the first few weeks the affected parts must be kept warm by means of cotton wool or extra clothing or artificial heat. As soon as possible the child is to be taken into the fresh air. It is of vital importance to keep up the general systemic tone, and hence the necessity for fresh air, change of scene, and for nourishing but easily digestible food. During this period massage and electricity should be employed, together with the administration of strychnin. In the later stages, when contractures have set in, mechanical appliances may be necessary to correct deformity and to give support. In suitable cases either nerve anastomoses or the transplantation of tendons have given good results.

CHRONIC POLIOMYELITIS IN ADULTS.

That **chronic poliomyelitis** exists has been proved by Oppenheim and other observers. The symptoms resemble very much those of progressive spinal muscular atrophy (p. 1142). In this affection, however, the intrinsic muscles of the hands are usually first affected, while in chronic poliomyelitis any group may be the first to suffer.

Treatment.—Mercury or the iodids may be tried, especially if there is a history of syphilis. Electricity and massage are of the greatest value.

ACUTE POSTERIOR POLIOMYELITIS.

(Herpes Zoster.)

THE posterior root ganglia of the spinal nerves, and those found in connection with the sensory cranial nerves, are also subject to inflammation due to some infective agent. The symptoms are neuralgic pain in the course of the affected nerves, possibly anesthesia in their distribution, and herpetic eruptions. Constitutional symptoms may also be present. Herpes zoster is a type of this disease. See also p. 1103.

While most cases recover, it may be a serious condition. Sight may be lost if the vesicles involve the eye, and unsightly scarring is not unusual.

The treatment consists in protecting the vesicles from rupture and infection by anodyne powders or salves, covered with a dressing. Anodynes internally, if the pain is severe. Drugs seem to have no effect in shortening the disease, but the salicylates, quinin, and general tonics may be tried.

ACUTE ASCENDING PARALYSIS.

(*Landry's Paralysis.*)

Definition.—An acute paralysis, beginning in the legs and ascending by way of the trunk and upper extremities, and ultimately involving the *medullary* centers. It usually runs a short course, and, as a rule, terminates in death.

Pathology.—Although in many cases neither gross nor microscopic lesions have been found, either in the cells, peripheral fibers, or muscles, a number of different anatomical changes have been found in cases believed to have this disease, viz., multiple neuritis, acute diffuse myelitis, and poliomyelitis. A fluid exudation in the central canal of the cord and hyaline change in the central arteries have also been found. The symptoms are evidently due to a severe type of infection, involving the peripheral motor neuron, which when very malignant, causes death before visible changes occur, while in less acute cases the characteristic evidences of neuritis, myelitis, or both, can be discovered.

Etiology.—No definite cause is known. It has followed cold and exposure, traumatism, and the infectious fevers, including influenza. It occurs in males chiefly between twenty and forty years.

Symptoms.—In the most acute cases there are practically no prodromal symptoms other than malaise and possibly chilly sensations. Weakness, followed in a few hours or a day or two by paralysis, develops in the lower extremities. One may be involved a few hours earlier than the other. It spreads toward, and soon involves, the trunk also, and in quick succession the arms. The third and usually fatal stage is reached when bulbar symptoms develop. Very rarely the upper extremities may be first attacked. Death may occur in forty-eight hours. The paralysis is a flaccid one; the muscles can be passively moved without offering any resistance. Wasting rarely occurs and there are no electrical changes. In less acute cases a decided febrile stage precedes the onset of paralysis, chills, fever, malaise, and possibly formication or even sharp pain. In any case the later symptoms are preëminently or solely motor. Sensory symptoms when present are very slight. The deep reflexes are absent. The bladder and rectum are not implicated nor do bed-sores develop. As stated, when the bulb is attacked death generally follows, due to cardiac or respiratory failure or to interference with deglutition. There are no cerebral symptoms.

Course.—Death may occur in from forty-eight hours to a few weeks. A few cases of recovery have been reported (Sinkler), in some of which paralysis had been widespread, even reaching the bulb, judging from the

labored respiration. When improvement takes place, it does so in the reverse order to the onset, so that the last part affected is the first to recover. It is much slower than the invasion.

Diagnosis.—The rapid onset of a paralysis that usually ascends, the relaxation of the muscles, slight wasting, if any, and the absence of electric changes and of sensory symptoms, with or without fever, serve to make the diagnosis and to distinguish Landry's disease from poliomyelitis, neuritis, and spinal hemorrhage. For the differential diagnosis between Landry's paralysis and acute myelitis, see page 1136.

Prognosis.—Always grave, particularly if bulbar symptoms occur, and especially if they appear early.

The **treatment** is essentially the same as that for any acute disease of the cord or nerves—*i. e.*, rest, freedom from all excitement or worry, warm baths and packs, moderate purgation, and diaphoresis; hexamethylenamin, ergot, belladonna, salicylates, and iodids internally. Should the patient survive, electricity and massage should be employed.

ACUTE MYELITIS.

(*Myelitis; Acute Diffuse Myelitis; Transverse Myelitis; Myelomalacia.*)

Definition and Varieties.—An inflammation, with softening of the cord, giving rise to various groups of symptoms depending upon the region or regions involved, and not, therefore, as constant in its symptomatology as the systemic nervous diseases (tabes dorsalis, lateral sclerosis). It may be acute, subacute, or chronic. If the gray matter only is involved, it is termed *poliomyelitis* (p. 1130); if a small vertical extent (several segments) of both white and gray matter, *transverse myelitis;* if an extensive area of both white and gray matter, *diffuse myelitis;* if a large area of gray matter, *central myelitis;* if in scattered areas, *disseminated myelitis.* If it follows a previous hemorrhage, *hemorrhagic myelitis*, and if it is caused by pressure, as a tumor or bone disease, *compression myelitis.*

Etiology.—Myelitis may follow exposure (especially in alcoholics), the infectious fevers, and it may be due to traumatism or disease of the vertebræ (caries, malignant disease). Syphilis precedes it in nearly one-half of all cases. It has also been described as following peripheral neuritis, ascending neuritis, and we meet with some cases in which pregnancy seems to act as the predisposing cause. In many of the cases occurring in those who have arterial disease the cause is thrombosis in a spinal artery, causing softening. This is known as *myelomalacia.* It may be difficult clinically to distinguish it from true myelitis. It is most common in males, generally from fifteen to thirty years of age.

Pathology.—The cord may present little or no change to the naked eye, or in the most acute cases it may be diffluent. Between these extremes many grades exist in which the pia will be found congested and adherent, the cord being more or less ingested and areas of softening, and even cavities, being found. Three forms of softening are spoken of by some writers—the red, yellow, and gray—depending upon the predominance of blood, fat, or connective tissue respectively. The *postmor-*

tem finding depends upon the duration of the disease; the more chronic the course, the greater the amount of nervous connective tissue (neuroglia), and in consequence sclerosis will be the predominant feature. The nerve-cells and fibers are found in various stages of disintegration, the former being swollen, vacuolated, granular, and their processes broken and in many cases missing; while the latter swell, the myelin breaks up, undergoes fatty change, and is removed, and the axis-cylinders finally break up and disappear. A single area of degeneration may exist centrally, in one half of the cord, transversely, or many localized or widely-disseminated areas may be found; but above and below all of them will be found degenerated fibers—ascending and descending degeneration—due to a solution of continuity between the cell-body and its axis-cylinder process. Transverse myelitis, or when the extent of several segments of the cord is affected, is the most common.

Symptoms.—These will vary according to the seat and extent of the lesion. In the most acute form the course of the disease is quite rapid, reminding one of hemorrhage into the cord or membranes; the onset, however, is not so explosive, and, though rapid, it is not sudden. It is most apt to follow cold or exposure. There may be chills and fever, malaise, backache, pains in the limbs; quite often, however, there is no warning. Motor weakness develops, and is rapidly followed by paralysis. Some irritative sensory symptoms appear, as hyperesthesia and paresthesia, and then more or less complete anesthesia supervenes. The reflexes are generally lost; there is incontinence of urine and feces, and bed-sores and cystitis develop with frightful rapidity. The temperature now rises to 105° F. (40.5° C.) or even higher, and typhoid symptoms, exhaustion, and death close the scene. I have seen a case that developed in a woman a few days after delivery and proved fatal in six days. In the majority of instances constitutional symptoms are not so marked, in fact they are often absent, and the paralysis develops more slowly.

Acute transverse myelitis is the type most frequently met with, the lesion being generally situated in the dorsal cord. The motor symptoms generally appear before the sensory symptoms, though they may be contemporaneous. In any event, they are apt at first to be irritative. The limbs will feel tired and heavy and drag in walking, and tremors or twitching occur, even cramps, and later paralysis, partial or complete, in the region involved. The lower limbs may alone be involved, or when the lesion is in the cervical region paralysis and atrophy of the upper with a spastic condition of the lower extremities may develop. The breathing is generally diaphragmatic in cases in which the intercostal muscles are involved. If the lesion is still nigher up, death will quickly take place from failure of respiration. Such cases, however, are more apt to occur in the type known as *disseminated myelitis*, in which bulbar symptoms are prone to appear. The sensory symptoms at first are those of a tingling or burning character, or formication. Later, certain or all forms of sensation may be lost, and, roughly speaking, the upper level of anesthesia corresponds to the level of the cord involved. This "boundary region" is apt to be hyperesthetic, and in it the "girdle-feeling" is experienced. The reflexes are usually lost at first; they may remain permanently absent or they may return, and become exaggerated below the

lesion. The condition of the tendon-reflexes may enable one to locate the position of the cord-lesion, they being lost in parts supplied by the affected segments, but increased below the seat of the lesion. Whether or not there is wasting of the muscles depends on the location of the lesion; if in the dorsal cord, as is usually the case, none will be found. When the cervical region is involved, the muscles supplied by the segments involved will atrophy and the reaction of degeneration will develop. The same thing occurs if in the lumbar cord (p. 1071). Below the seat of the lesion there is paralysis, but not atrophy. Loss of control of the bowel and bladder may be among the earliest symptoms, though this is not the rule. While superficial ulceration may occur in any neglected case, the most marked trophic changes take place in those in which the lumbar cord is involved, either directly or by extension. In such cases, despite the most assiduous attention, extensive bed-sores develop. The course of the disease depends on the cause and extent of the lesions. Death may occur in a few weeks from exhaustion, heart or respiratory failure, or from kidney diseases secondary to cystitis. Recovery is the rule, though with more or less permanent damage due to degeneration of some of the paths of conduction.

Diagnosis.—The distinction from hemorrhage into the cord or membranes has already been mentioned. From Landry's paralysis it can be separated by a reference to the subjoined table:

Acute Myelitis.	Landry's Disease.
Paralysis is sudden and generally becomes complete.	Paralysis begins in the feet and rapidly spreads to the muscles of respiration and deglutition.
Wasting and bed-sores are marked.	Trophic disturbances are absent.
If atrophy occurs, reaction of degeneration is present.	No reactions of degeneration.
Early involvement of the sphincters.	Bladder and rectum are not involved.
Girdle-pains sometimes mark the height of the lesion.	Girdle-pains are absent.
Sensory paralysis.	No loss of sensation.

Anterior poliomyelitis is not accompanied by sensory paralysis. Bed-sores and disturbances of the sphincters do not occur. In *peripheral neuritis* pain of a shooting character and tenderness over the affected nerves are present, and is almost invariably the first symptom to appear. Motor symptoms may not appear for some days. This is not the case in myelitis. In *compression of the cord* sufficient collateral evidence, as evidence of bone disease, can usually be obtained to differentiate it from myelitis. *Hysteric paraplegia* is occasionally misleading. The character of the patient and the previous history should be thoroughly considered; moreover, in this form there are no trophic changes, and, as a rule, no bladder-symptoms; at any rate, there is no cystitis. Retention of urine may occur, but not incontinence, and the Babinski reflex is absent. The diagnosis of myelitis can usually be made without great difficulty from the motor and sensory symptoms, the vesical, rectal, and trophic symptoms, and often from the presence of the girdle-sensation in addition. Myelomalacia can usually not be distinguished clinically from myelitis. The symptoms occurring in an old person, without any of the causes of the latter having been operative and the existence of a previous history of syphilis, is rather in favor of the former.

Prognosis.—The most acute cases are fatal in from three days to a week. Less acute cases generally recover with more or less loss of motor power. Improvement may continue for several years.

Treatment.—Very little can be done to arrest the process in acute myelitis. Absolute rest should be enjoined, and the patient given a nutritious liquid diet with free stimulation. The patient should be placed on an air- or water-bed. Trophic changes should be looked for daily, and at the first sign of their appearance alcohol or some stimulating liniment should be employed. If the skin is broken, absolute cleanliness must be observed, and the wounds dressed antiseptically. It is well, also, to change the patient's position from time to time to avoid too long-continued pressure in any one spot. Either the salicylates or hexamethylenamin should be given in infectious cases, and in specific cases, mercury and potassium iodid in full doses. A general tonic and supportive treatment is indicated, and later massage, electricity, and baths.

CHRONIC MYELITIS.

THAT there are both a subacute and a chronic form of myelitis is generally conceded, though these types are not sharply circumscribed. As has been previously mentioned, it is quite likely that many cases exist in which the clinical symptoms do not seem to warrant the diagnosis of myelitis, and yet extensive areas of degeneration may be found *post-mortem*.

Etiology.—This is not clearly known; an acute attack may terminate and the tissue of the cord become sclerosed with persistence of the symptoms; or the disease may commence insidiously as the result of the existence of some chronic infectious process, such as syphilis, it may be the sequel of an acute infection, such as typhoid fever, or follow a fall or blow upon the back.

Pathology.—Histologically, the chief differences from the acute variety consist in the greater amount of sclerosis, the thickened blood-vessels with contracted lumen, and an entire absence of recent hemorrhage. In some cases also the pia is much thickened in patches and firmly adherent. The nerve-cells are either seen to be in advanced stages of degeneration or they have actually disappeared. Secondary degenerations, above and below, proceed from the primary foci.

Symptoms.—Any symptom occurring in the acute may be duplicated in the chronic form, though the onset of the latter is gradual. The symptoms are more or less obtrusive, according to the region of the cord that is affected, and it may be several years before they are fully developed. In those cases which do not follow the acute, the first symptoms complained of are usually numbness of the legs and a feeling of weakness, which gradually progresses until in some cases the legs may become useless. If the meninges are involved, as they frequently are (meningomyelitis), shooting pains in the extremities and a girdle sensation are complained of. The symptoms differ somewhat, according to the nature of the lesion. This is usually of the transverse variety, which, if in the dorsal region, as it usually is, causes a spastic paraplegia, with increased deep reflexes, Babinski reflex, some sphincter disturbance, and

more or less complete loss of sensation to the level of the affected segments. If in the cervical enlargement, which is rare, more or less atrophic paralysis of the arms, owing to the involvement of the gray matter, with a spastic paralysis of the legs and loss of sensation to the affected segments, will be present. If in the lumbar region, the symptoms will differ somewhat, according to the segments involved. There will, however, be atrophy in certain muscles and spasticity and absence of atrophy in others (p. 1135). Owing to involving of the posterior columns there may be more or less ataxia, so that the gait is a mixture of a spastic and ataxic type (ataxic paraplegia). The disseminated type resembles multiple sclerosis.

Diagnosis.—The gradual, and in many cases the irregular, onset characterize this disease. In its various phases it may simulate almost any spinal-cord disease, and it is most apt to be confounded with tumor-pressure (carious or malignant), primary lateral sclerosis, amyotrophic lateral sclerosis, and syringomyelia. *Pressure*, whether due to a tumor, to caries, or to malignant disease, is apt to cause pain radiating in character, and the last two usually present collateral evidences in the deformity and cachexia (p. 1139). The symptoms, too, in the case of tumor may, at first, be unilateral and confined principally to the muscles and skin areas supplied by the affected segments (p. 1158). Amyotrophic lateral sclerosis is distinguished by the fibrillary tremors in the atrophied muscles, absence of sphincter involvement, and sensory symptoms. Syringomyelia is characterized by the loss of pain and temperature sense with preservation of tactile sense in certain areas.

The **prognosis** is necessarily grave. Recovery may be possible, but it is extremely rare. The process, however, may be arrested and the patient live for years more or less helpless.

Treatment.—More can be expected from general hygienic measures than from the use of drugs. In the early stages rest is indicated, but it is well also to employ passive exercise to prevent, if possible, a too great contraction of the muscles. As soon as expedient—each case being judged on its merits—the patient should be taken out of doors. Change of air and of scene is advisable, as are also baths and massage. Mild counter-irritation may be applied to the spine, but care should be taken to avoid the areas of anesthesia. General tonics—iron, quinin, arsenic, and strychnin—should be given, also mercury and the iodids. The greatest possible care of the bladder should be taken in order to avoid cystitis. Good results have been obtained by Bailey and Elsberg[1] from the performance of what they term "spinal decompression." This consists in opening the spinal dura over the seat of the lesion and then closing the wound in the usual manner.

COMPRESSION OF THE SPINAL CORD.

(*Compression Myelitis.*)

It is of importance to be able to recognize this condition. To be sure, it is not always possible to diagnose it with certainty, but when there is a reasonable surety the question of operation may arise. Since

[1] *Jour. Amer. Med. Assoc.*, Mar. 9, 1912, p. 675.

it has so many features in common with myelitis, the necessity for reserve and caution in arriving at a conclusion is manifest, because the latter condition would not be benefited by any operative procedure.

Etiology.—We may classify the causes of compression under three headings—(*a*) traumatism (fracture), (*b*) inflammatory disease (caries of the spine, due to tuberculosis or syphilis), and (*c*) neoplasms (including various tumors, gummata, and aneurism); but these will receive separate consideration (*infra*).

Pathology.—The postmortem findings will depend upon the degree and duration of the pressure. More or less meningitis is often associated, especially in the cases due to vertebral caries. The cord will be more or less flattened and distorted at the seat of pressure, and in the early stages hyperemic and possibly softened. Later it is hard, sclerosed, and of a grayish color, and above and below the compressed region degenerated areas will be seen on sectioning the cord. Microscopic examination reveals various stages of degeneration of the nerve elements at the point of pressure and secondary degeneration of the various tracts. The nerve-roots will be more or less damaged by compression.

Symptoms.—These will vary according to the site of the lesion and the extent of involvement—*i. e.*, the vertical extent, the degree of pressure exerted, and the amount of inflammation present. Two groups of symptoms are present in typical cases—first, those due to involvement of the nerve-roots, and, second, those dependent upon involvement of the cord itself—*ascending* and *descending* degeneration. Pressure upon the *posterior roots* gives rise to *pain*, neuralgic in character and radiating along the course of the nerves. The parts supplied are usually *tender*, and there may be *paresthesia* and *formication*. These irritative symptoms are followed sooner or later by destructive changes, and hence the anesthesia. There may be spontaneous pain in the anesthetic areas (*anæsthesia dolorosa*), which areas are of the segmental type and depend in location upon the cord segment and corresponding nerve-roots involved (Fig. 75). Pressure upon the *anterior roots* also causes irritative and paralytic symptoms, and hence the early twitching, or even spastic condition, and later the loss of power or paralysis. The muscles supplied by nerves from the affected segments waste, and qualitative and quantitative electric changes can be elicited.

The *second group* of symptoms, due to secondary degenerations, then develops, and may set in either rapidly or slowly. If myelitis promptly supervenes and is extensive, *cord-symptoms* of a pronounced type develop quickly. The parts below the lesion will become weak, there will be girdle pains, and a sense of constriction or pain in the legs. Sensory paralysis is usually not so marked in this region as the motor, as the sensory tracts are less vulnerable to pressure than the motor, but hyperesthesia and hyperalgesia are present in most cases. They may, however, be absent. The reflexes are usually increased. If the cause of compression ceases to act for some time, some improvement takes place, due possibly to the subsidence of the myelitis. If the pressure is of *slow onset*, great tolerance is manifested. Usually sensation is recovered before motion. In certain cases, however, motor power is regained, while the muscular and tactile senses do not return. In such instances, in which the posterior columns bear the brunt of the trouble, incoördination results and there is secondary ataxia.

Diagnosis.—If the combined symptoms of peripheral and central origin develop slowly in the order named, compression is likely. It has been asserted[1] that when the paralysis is due to pressure, actual degenerative changes not yet having taken place, that the tendon reflexes are not increased but the skin reflexes (plantar) are very much so. *Myelitis* gives rise first to cord-, and only later to peripheral symptoms; hence the difficulty in cases in which myelitis develops quickly. Extensive root-symptoms are suggestive of *meningeal involvement*. In any event, too much stress should not be placed on the nervous symptoms alone. The spine should be carefully examined and palpated for points of tenderness. Careful note should also be taken as to whether there is any limitation of movement or deformity (kyphosis). The family history may suggest *tuberculosis* (caries of the spine). A skiagraph of the spine is often valuable, as it must be borne in mind Pott's disease may occur without spinal deformity. Tumor is discussed on p. 1156. The history will indicate if due to vertebral fracture.

The **prognosis** depends entirely upon the cause. Having ascertained this, it then depends upon the possibility of its removal.

Treatment.—In general the treatment is that of myelitis. When due to tubercular disease of the vertebra, the treatment indicated is for that condition, and a surgeon should be consulted, though operative cases are the exception rather than the rule. It is well to impress upon the patient and relatives the chronicity of the condition, but faithful and persistent efforts will yield good results. Rest is of vital importance, particularly when the disease is active. The patient should be kept in bed in a recumbent position until consolidation has taken place. Extension may be necessary. Good and easily assimilable food and cod-liver oil and alteratives should be given. The nutrition of the muscles may be improved by general friction (massage). As soon as possible a plaster jacket should be put on the patient, and he should be taken into the open air and sunlight. If a history of syphilis is obtained, that condition should be vigorously treated. In vertebral fracture the question of operation to remove the fragments of bone pressing on the cord may arise. When due to tumor, see page 1159.

PRIMARY LATERAL SCLEROSIS.

By this is meant a primary degeneration of the central motor neurones. It is a rare condition, most cases so called being due to a secondary degeneration caused by a lesion cutting the tracts somewhere. A few cases have been reported in which no such lesion could be found. The *hereditary type*[2] is probably the most common. The only pathologic change observed is in the pyramidal tracts of the anterior and lateral regions.

Etiology.—It is most apt to occur when there is a *neuropathic family tendency*. *Age*, generally between twenty-five and forty, exerts an etiologic influence. *Exposure*, *acute disease*, and *traumatism* are all

[1] *Jour. Amer. Med. Assoc.*, Jan. 25, 1913, p. 269.
[2] Spiller, *Phila. Med. Jour.*, June 21, 1902.

predisposing causes. *Syphilis* has been said to predispose to the condition, but if so, it is rather rare. Most cases presenting this symptom-complex are due to a secondary degeneration of the pyramidal tracts, caused by some lesion, as a mild myelitis higher up. Vertebral disease may also cause similar symptoms. A number of cases have been reported occurring in a number of generations of a family (*hereditary spastic spinal paralysis*). In these cases the symptoms may appear in childhood.

Symptoms.—In typical cases the onset is slow. The patient complains of feeling tired, and is less capable of exertion than formerly. Weakness of the legs develops, and with it increasing difficulty in walking. Even at an early stage some rigidity of the muscles will be present when the limb is extended; later this becomes a prominent symptom. The spasm is at first of little moment. It may only be noticed in the morning. When the disease has advanced, however, it becomes pronounced, so that it may not be possible to flex the limb, or, if flexed and an effort is made to extend it, it will often spring forward like a knife-blade in clasp-like rapidity. This spasticity is often so marked that in walking, so long as the ball of the foot touches the ground, clonic contractions occur; these also appear when the individual is in a sitting posture unless his legs are extended. The gait is characteristic; the legs are stiff, and move with an evident effort, while the toes scrape the ground. In some cases the adductor spasm is so great that the legs not only cannot be separated, but are actually overlapped in walking (*cross-leg progression*). In course of time the power of walking may be lost. The flexor muscles are usually weakened. The knee-jerk is very much exaggerated, a mere tap causing a sharp, quick response. Ankle-clonus can always be elicited. The Babinski reflex is present (extension of the toes when the sole of the foot is irritated). Pains and other sensory manifestations are often absent, though dull and fleeting pains in the back and limbs may be complained of. The arms are frequently unaffected. The sphincters are rarely involved, and ocular symptoms do not occur. Seguin states that the ability to retain the urine is lessened and precipitate micturition results.

The **diagnosis** is not difficult. Certain hysteric cases may occasionally simulate it very closely, but these do not present the characteristic spasticity of the true form, nor is the knee-jerk increased quite as much, ankle-clonus is either slight or absent, and the Babinski reflex is not present. Then, too, in hysteria spots of anesthesia are commonly met with. In myelitis there is usually more or less sensory paralysis and involvement of the sphincters; if, however, it is very mild in type, the diagnosis is most difficult. The possibility of caries of the vertebræ must be borne in mind when the symptoms are developing (p. 1138). The congenital type, due to cerebral lesions, is described on p. 1163. Hydrocephalus may also be mistaken (p. 1190).

Treatment consists of maintaining the general health, warm baths for the spasticity and antisyphilitic medication if there is a history of that disease. In the hereditary form benefit has been obtained by tenotomies, followed by electricity. Cutting the posterior nerve-roots may also be considered, if spasticity and not weakness is the dominant condition.[1]

[1] *University of Penna. Med. Bull.*, Jan., 1910, 314; *New York Medical Journal*, Jan. 29, 1910, 215.

PROGRESSIVE SPINAL MUSCULAR ATROPHY.

(*Amyotrophia Spinalis Progressiva; Type of Duchenne-Aran.*)

Definition.—A disease of the peripheral motor neurons and the muscles they supply, usually beginning in the cervical region.

Pathology.—There is atrophy of the anterior cornua of the cord, affecting chiefly the ganglion-cells, degeneration of the nerve-fibers and of the muscles. Occasionally there are small areas of sclerosis that may involve the pyramidal columns for a short distance.

Etiology.—The disease appears to be hereditary in a few cases, and in these may develop in childhood. A commonly accepted predisposing cause is prolonged severe muscular exertion. Syphilis may also be a factor. It is most common in males, and most frequently appears during the third decade of life.

Symptomatology.—The first changes usually appear in the *thenar and hypothenar eminences* of the hands, but may begin in other muscles. These become flat and soft; there are loss of power, some stiffness, and inability to perform delicate coördinated movements; the thumb assumes a position parallel to the other fingers (*ape-hand*); the interossei muscles waste and grooves appear between the metacarpal bones. The degenerative changes do not ascend by continuity, the deltoid usually being affected immediately after the muscles of the hand. If the two hands have not been affected simultaneously, the other now begins to show characteristic changes. In the lower limbs the quadriceps femoris is usually the first muscle attacked. The disease gradually involves one group of muscles after another until a large part of the muscular system is affected. All the affected muscles exhibit the fibrillary twitchings and the wasting. Hypertrophy never occurs and the *paralysis* is always flaccid. The fibrillary twitchings are characteristic, but not pathognomonic. They are not always constant, but may be developed by slightly irritating the muscle. At first there is usually quantitative diminution of the response to the faradic and galvanic currents, but as the disease progresses the reaction of degeneration becomes completely developed. The *reflexes* diminish in proportion to the atrophy of the muscles, and ultimately disappear completely; the patients gradually become almost incapable of voluntary motion; but for a time they learn to overcome their disabilities by the compensatory use of other groups of muscles. In the late stage the diaphragm becomes paralyzed and bulbar symptoms appear (p. 1129); usually the patients die from inspiration-pneumonia. Rare and probably accidental symptoms are disturbances of the pupillary reflexes and increase in the secretion of sweat.

Differential Diagnosis.—In *chronic anteropoliomyelitis* groups of muscles are affected without any particular order, certain groups of muscles becoming paralyzed suddenly, followed by the gradual involvement of other muscles; in *amyotrophic lateral sclerosis* the spastic symptoms are present; in *syringomyelia* and *pachymeningitis cervicalis hypertrophica* disturbance of sensation, pain, and trophic lesions occur; in *Pott's disease* affecting the lower cervical region there are tenderness over the spine and sensory disturbances; in *peripheral neuritis* pain and tenderness over the nerve-trunks are present; in *arthritic atrophy* joint-symptoms are present; and in the peculiar *muscle-atrophies* following

excessive use of certain groups of muscles, rapid improvement occurs when the cause is removed and the symptoms are confined to the muscles originally affected (either median or ulnar distribution) (pp. 1116 and 1117). The muscular dystrophies are described on page 1260.

Prognosis.—This is unfavorable as to cure. The course is exceedingly slow, and the patients often live for a number of years after the first symptoms have appeared. They are, however, exceedingly liable to pulmonary complications, particularly a fatal form of bronchitis.

Treatment.—Prophylactic measures, such as the avoidance of prolonged excessive work, are rarely possible. Retardation may possibly be obtained by the systematic use of electricity, massage, and gymnastics. Gowers advocates the hypodermic injection of strychnin nitrate in ascending doses, commencing with $\frac{1}{100}$ gr. and rapidly increasing to $\frac{1}{40}$; one injection should be given daily. The general nutrition should be kept at the highest possible point.

AMYOTROPHIC LATERAL SCLEROSIS.

(*Charcot's Disease.*)

Definition.—A disease of both central and peripheral motor neurons, effecting, therefore, the entire motor tract from the cerebral cortex to the muscles, characterized by loss of power, spastic symptoms, and muscular atrophy. The first clear and thorough description of the clinical symptoms and pathological anatomy was given by Charcot in 1872.

Etiology.—The disease is more frequent in males and usually begins in early adult life. Exposure has sometimes been noted in the previous history, but neuropathic heredity does not appear to have any influence. Syphilis may be a cause.

Pathology.—The pyramidal tracts are degenerated, the process commencing either in the cortex, crura, or medulla, and extending to the termination of the neurons in the cord. The ganglion-cells of the anterior cornua are atrophic, there is degeneration of the anterior roots and of the muscle-fibers, the blood-vessels in the affected parts are dilated, and in the early stages granular cells are present.

Symptoms.—Three stages are generally recognized: (1) The involvement of the upper extremities. (2) The participation of the lower extremities. (3) The appearance of bulbar symptoms. At first there are weakness of the upper arms, atrophy of the muscles, and moderate exaggeration of the reflexes; in the course of a few months the symptoms of spastic paraplegia develop, all the reflexes are greatly increased, and there are chin- and ankle-clonus, the Babinski reflex, and dragging of the feet. The wasted muscles show fibrillary twitchings and give the reactions of degeneration. Contractures then occur, the forearms are flexed on the arms, the hands are held in pronation, and the proximal phalanges of the fingers bent backward, giving rise to the so-called *claw-hand*. From time to time there are tonic spasms in the muscles, particularly in the calves. Sensation is not disturbed, excepting for the occurrence of occasional slight paresthesia, and the sphincters continue to functionate normally.

Finally, the bulbar symptoms appear, and there is paralysis of the lower part of the face, which becomes rigid and expressionless, with the mouth partly open and saliva dribbling from the angles. Deglutition and articulation become difficult or impossible, and death finally occurs from exhaustion or inspiration-pneumonia. During the course of the disease the intellect is slightly involved. Memory is impaired, the conduct becomes childish, and there is a tendency to weep or laugh without cause. Atypical cases occur, in which either the lower extremities are first involved or the paralytic symptoms are more prominent than the spastic symptoms, or the bulbar symptoms appear very early. The symptoms at first may be unilateral (p. 1144). The course is steadily progressive, and death usually occurs within two years.

The **differential diagnosis** is to be made from *multiple sclerosis* by the absence of nystagmus, of the intention-tremor, and of sensory disturbances, and by the degenerative changes in the muscles; from *transverse myelitis* by the absence of sphincter disturbance, sensory paralysis, and of pain, from *progressive spinal muscular atrophy* by the presence of spastic symptoms; from *syringomyelia* by the absence of sensory disturbances, trophic lesions of the skin and joints, and the greater regularity of the course; from *pressure upon the spinal cord* by the absence of pain, sensory paralysis, and sphincter disturbance. It must be remembered that amyotrophic lateral sclerosis may be associated with multiple sclerosis or infantile spinal paralysis.

Prognosis.—It will be understood from the foregoing description that death is the invariable termination. The course is progressive, although sometimes very deliberate, and even temporary amelioration rarely occurs.

Treatment.—The patient should be rendered as comfortable as possible, excessive physical exercise avoided, and the general nutrition increased. Arsenic and mercury are useless.

UNILATERAL ASCENDING AND UNILATERAL DESCENDING PARALYSIS.

Unilateral ascending paralysis, first described as an entity by Mills, is a form of possibly various pathology which is characterized by paresis commencing in one leg and extending to the arm on the same side. The paresis may be flaccid (Patrick), with loss of the reflexes, or spastic (Mills, Potts, etc.), with increased knee-jerks, ankle-clonus, and the Babinski phenomenon. In time complete paralysis may develop. Degeneration of the motor tract has been found. According to Mills[1] it may be produced: (1) By primary degeneration of the pyramidal tracts, to which may be added other degenerative lesions; (2) as the early stage of multiple sclerosis; (3) as the form assumed by unilateral amyotrophic lateral sclerosis; (4) as the order of progression in unilateral paralysis agitans; (5) as the expression of a focal lesion either cerebral or spinal; (6) as a clinical type in cerebrospinal syphilis; (7) as a peripheral or hysterical affection. There is no treatment.

[1] *Journal Nervous and Mental Diseases*, April, 1900, and Proceedings Neurological Section of *Amer. Med. Assoc.*, 1906, p. 166.

INTERMITTENT PARAPLEGIA.

(*Intermittent Claudication.*)

Three forms of this affection have been described, *i. e.*, (1) Those due to an arteritis of the vessels supplying the peripheral nerves; (2) those caused by a spasm of these arteries; (3) those due to similar phenomena occurring in spinal arteries. The symptoms consist of muscular cramps and weakness, usually but not always in one or both legs, occurring after moderate exertion. In the first type there is often loss of pulsation in the posterior tibial and dorsalis pedis arteries, in the third the sphincters are affected and cramps do not occur (p. 1163). The treatment is that of arteriosclerosis.

TABES DORSALIS.

(*Locomotor Ataxia; Posterior Sclerosis.*)

Definition.—A disease, primarily of the posterior nerve-roots, with consequent secondary degeneration of the posterior columns, also degeneration of peripheral nerves and those of special sense, particularly the optic. It is characterized by more or less incoördination of movement, various sensory and trophic disturbances, and impairment of the special senses.

Etiology.—Syphilis precedes such a large proportion of all the cases (60 to 90 per cent.) that it is reasonable to assume that it is the commonest and perhaps the exclusive cause. Whether the tabes occurs because the individual is predisposed or because the syphilitic virus in these cases has some peculiar predilection for the central nervous system is not determined. Some interesting evidence has been collected in favor of the latter view. There is often a history of injury, severe prolonged muscular exertion, dissipation, or sexual excess. Race appears to be of some importance, but an increasing proportion of cases is found among negroes and Jews, who were formerly considered partially immune. Males are more liable to the disease than females in the proportion of 10 to 1. About 75 per cent. of all cases commence between the ages of thirty and fifty.

Pathology.—Macroscopically, it may be observed—1. That the posterior roots are more or less atrophied and grayish in color.

2. There is a thickening and adhesion of the spinal membranes, with some degree of congestion, particularly noticeable in the posterior region (not a constant change).

3. There is a slight change in the shape of the cord, and the affected regions assume a grayish tint. Change of color is well seen after the cord is hardened. Microscopically, the first changes are found in the posterior root, usually the lumbar, followed by sclerosis in the column of Burdach and zone of Lissauer, also most marked in the lumbar region and localized at the point of entrance of the root-fibers. Higher up the columns of Goll will be found involved. The fibers coming from the posterior roots to join the column of Clark are also sclerosed, but as the

cells, as a rule, are not destroyed, the direct cerebellar tract is rarely involved. In advanced cases sclerosis of Gower's tract may also be observed. While the disease usually first affects the lumbar nerve-roots, either the sacral, upper thoracic, cervical, or bulbar nerve-roots may be first involved, in which event the cord changes above noted will be found more marked in one of these respective areas. In addition to the cord changes degeneration of peripheral spinal nerves and of cranial nerves and their nuclei, especially the ocular, may be found. Less marked changes may also be found in the anterior nerve-roots.

Nageotte's views as to the pathogenesis of the disease are now those most generally accepted. In brief, he believes that tabes is the result of a local affection of the spinal roots at the height of the "nerfs radiculaires"—*i. e.*, that part of the root from its entrance into the dura

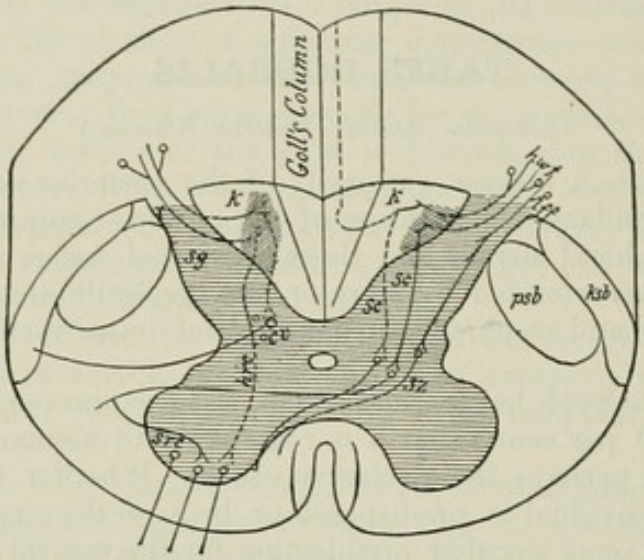


FIG. 78.—Diagram of primary degeneration-areas and secondary degeneration of the fibers in the beginning stage of tabes (Leube): *psb*, pyramidal tract; *ksb*, cerebellar tract; *hwf*, posterior root-fibers; *lff*, lateral entrance of delicate root-fibers; *k*, area of earliest degeneration; *r*, marginal zone; *sg*, substantia gelatinosa; *cv*, Clark's columns; *i*, anterior zones (remaining free); *sc*, sensory collateral fibers; *hrc*, collateral reflex of posterior column; *src*, collateral reflex of the lateral column; ——, healthy fibers; ------, degenerated fibers.

mater to the spinal ganglion. It consists of an endo- and perineuritis with interstitial and parenchymatous changes, due to a mild but chronic syphilitic meningitis. The "nerf radiculaire," being a channel for the lymph circulation in the central nervous system, is believed to be a spot more vulnerable to toxic and irritating material circulating in the cerebrospinal fluid. This is further intensified by the fact that the nerve-fibers lose their neurilemma sheaths as they pass through the pia, and, hence, when damaged by the meningeal exudation, have no power of regeneration. The degeneration found in the cord is, hence, secondary, the nerve-fibers being separated from their trophic centers, the posterior ganglion.

In addition to changes in the nervous system, certain cases present some morbid condition of the osseous system, consisting of erosion of the intra-articular cartilages and atrophy and absorption of the bony articulating surfaces.

Symptoms.—These may be grouped into various stages: the prodromal, preataxic, ataxic, and paralytic. The *prodromal stage* may extend over a number of years, the symptoms are slight, and often make very

little impression upon the patient. They consist of occasional pains, usually in the legs, of transient disturbances of the ocular muscles leading to ptosis, diplopia, etc., occasionally of slight diminution of vision, most noticeable at night, of more or less pronounced impairment, very rarely exaltation, of sexual power, and neurasthenic symptoms. The symptoms become characteristic only in the *preataxic stage*. The pains in the legs become more frequent and assume the typical fulgurant or lancinating type; that is, a stabbing or boring sensation, shooting along the limbs and lasting for a brief interval of time. There is often numbness or anesthesia of the extremities and the patient feels as if walking on cotton. The pupils give the Argyll-Robertson phenomenon (failure to contract to light, but do to convergence), there is permanent myosis, nyctalopia, and the paresis of the eye-muscles may be still present or may have disappeared; primary atrophy of the optic nerve will also frequently be found. There is usually some disturbance of motion, chiefly manifested at night, and ataxia may be revealed by the finer tests (having the patient hop backward on one leg). The patella and Achilles jerks are diminished or absent. There is now distinct impairment of sexual power and difficulty in urination. Martin has described a peculiar loss of tone and muscle sense of the rectal sphincters. The sensory symptoms belong to this and the following stage.

The dominant symptom of the *ataxic stage* is the incoördination of movement. This gives rise to the *ataxic gait*. The legs are kept far apart and are lifted higher than is necessary from the ground, they are brought down violently, and the gait is of a peculiar stamping, irregular, slightly staggering character. Walking without the aid of a cane soon becomes impossible, and the feet are carefully watched. Ataxia of the arms occurs later and is manifested by difficulty in grasping objects or in accomplishing finer coördinated movements. Ataxia of the lower extremities may be tested by directing the patient to touch with his toe an object held above it, or, when lying down, to place the heel of one foot upon the knee of the other; of the upper limbs by directing him to touch rapidly the tip of the nose with the forefinger, or to spread the arms apart and bring the forefingers rapidly together. Loss of station, or *Romberg's symptom*, is tested by directing him to stand with the feet close together and to close the eyes. The swaying of the body will vary from several inches in either direction to falling over. The reflexes are now completely abolished, and there may be some wasting of the muscles, and marked muscular hypotonia, causing relaxation of the joints, will frequently be found. The sphincters are involved, there is often difficulty in voiding the urine, associated with incontinence, and, as a result of careless catheterization, cystitis is often acquired. The facial expression is peculiar, the pallor, drooping lids, small pupils, and deep lines give an impression of weariness, dulness, and apathy that is quite characteristic.

The *sensory symptoms* are various: in addition to the fulgurant pains, there may be visceral crises, characterized by sudden severe pain and disturbance of function. The most common seat is the stomach, and the crises are associated with vomiting of acid material. Crises may also involve the larynx, liver, kidneys, clitoris, and bladder. Diminished sensation affects the organs, nerves, and areas of the skin. There is loss of sensation in the testicles or breasts, and severe blows in the pit of the

stomach cause no distress. *Biernacki's symptom*, loss of sensation in the ulnar nerve when pressed upon at the elbow is present. The same phenomenon can be observed in the peroneal nerve, where it winds around the head of the fibula. These are early symptoms. Areas of anesthesia or hyperesthesia, usually segmental in type, can be detected upon the trunk or less frequently upon the extremities. Astereognosis, or the loss of the ability to recognize objects, may be present on one or both sides, or the stereognostic sense may not be impaired. The *girdle pain* is a feeling of constriction about some part of the trunk that may be very uncomfortable. *Trophic changes* are of various kinds, painless loosening of the teeth; *arthropathies*, characterized by enlargement and erosion of the joints, which are painless; fragility of the bones leading to spontaneous fracture; herpes and perforating ulcer of the foot. The *paralytic stage* inaugurates the termination of the disease. Locomotion becomes impossible, or can only be accomplished with the aid of two canes, loss of control over the bladder is complete, the patient is querulent or even demented, and muscular wasting and bed-sores may appear. In either this or the ataxic stage the optic nerve may atrophy, and this is often associated with a remarkable improvement in the ataxia that is at present inexplicable. Death usually occurs as a result of infection, either through the bladder or lungs, more rarely as the result, apparently, of exhaustion.

Atypical cases are not rare. In *cervical tabes* the ataxia may appear first in the upper extremities and may be more severe in them, and the fulgurant pains may be limited to them. Laryngeal crises are more common in this form, and neuralgic pain in the course of the fifth nerve may be present. Certain classical symptoms may not appear in the entire course of the disease. In sacral tabes the knee-jerks may be present, while the Achilles jerks are lost.

Course.—The earliest symptoms are usually observed from 5 to 20 years after the syphilitic infection. Rarely the disease runs a very rapid course. The preataxic symptoms—pain, loss of knee-jerk, Argyll-Robertson pupil, with or without ptosis and diplopia—may only exist a few weeks before incoördination develops. The latter will then reach its acme in twenty to thirty days. This is very unusual, however. As a rule, the first or preataxic stage extends over a period varying from months to even as long as twenty-five years. Dr. Wm. Egbert Robertson has related to me the case of a man aged fifty-eight who for fifteen years has had fulgurant pains and an absence of the knee-jerk, but neither ocular nor any other symptoms. In some cases the first stage may be unnoticed. The second or ataxic stage—that of incoördination—is generally slowly progressive, finally reaching a point at which it remains; rarely, more or less improvement may follow. When optic atrophy develops, ataxia either does not appear, or, having done so, fails to advance. The final stage in a few cases is only reached when the patient has become paralyzed and bedridden.

Diagnosis.—This is readily made when we have a combination of the absent knee-jerk, fulgurant pains, and the Argyll-Robertson pupil.

Differential Diagnosis.—*Peripheral Neuritis.*—The symmetric distribution of symptoms, tenderness in the muscles and over the nerve trunks, more weakness and wasting, pain (not fulgurant in type), absence

of the Argyll-Robertson pupil, and the history of the case are sufficient. *Alcoholic* and, more rarely, *arsenical neuritis* give rise to a condition closely resembling true tabes, in that there is the loss of knee-jerk, often sharp pain, and incoördination, though the latter symptom is never as marked as in advanced tabes. The gait, however, is totally different, and consists of the high "steppage" gait described in the discussion of Peripheral Neuritis.

General paralysis of the insane (p. 1198) may present much difficulty. Spinal symptoms may occur in general paresis, and conversely in certain cases of tabes symptoms of general paresis develop. Such cases are really combinations.

Ataxic Paraplegia.—Apart from the absence of pain and anesthesia, incoördination is followed by a spastic condition. The knee-jerk is much exaggerated and ankle-clonus develops.

Cerebellar Disease.—The incoördination does not resemble that of ataxia; optic neuritis is present; also headache and vomiting appear in well-marked cases. The knee-jerk is usually present.

Ataxia may be present in *combined sclerosis*, due to anemia and infections (p. 1153), as lesions of the posterior columns are present. In this disease the Argyll-Robertson pupil is absent. While paresthesia is pronounced, the characteristic pains of tabes are not complained of. The Babinski reflex will also usually be found. The history of the onset and course of the disease also differs.

The crises may be mistaken for *disease of the various organs involved.* Repeated attacks of acute pain, tabetic in character, and particularly in adult males, should, however, excite suspicion, and an absence of the knee-jerk and other characteristic evidences will always be present in ataxia.

When the chief lesion is in the dorsal region the pain may be mistaken for that of *spinal caries* or even *neuralgia* or *rheumatism.* From caries it may be differentiated by the fact that in vertebral disease the pain is more or less localized, and that it is much increased by movements. Moreover, the other symptoms of ataxia are wanting—*e. g.* ocular troubles, incoördination, and absence of the knee-jerk. The latter point also holds good in cases of rheumatism and intercostal neuralgia. For the diagnosis from hereditary ataxia, *vide* p. 1152. Help may be afforded in doubtful cases by an examination of the fluid obtained by lumbar puncture (p. 1124); in tabes an increased number of lymphocytes being found. It is proper to state that these may also be found in paresis and other syphilitic affections of the cord. The Wassermann test may also help in doubtful cases, although this may be negative in some cases of genuine tabes.

Prognosis.—The outlook is not particularly bright. While, as already stated, the disease does not cause death, recovery does not directly occur. Even improvement, excepting of the most temporary nature, is rare, but the disease sometimes seems to be arrested for comparatively long periods. The possibility of arresting the progress of the disease is greater when proper treatment is begun early.

Treatment.—Rest (first suggested by Weir Mitchell) is imperative when the patient commences treatment, and especially when pain is an early symptom, massage and electricity being employed meanwhile to

keep up the tone of the muscles. In my opinion the rest treatment retards the progress of ataxia more effectively than any other measure, but it cannot be used with the expectation of producing a cure. The bowels should be moved daily, and the urinary functions especially looked to. In certain cases catheterization is necessary. The patient should then be taught, first, what surgical cleanliness means; and secondly, how to use the instrument. Urotropin in doses of gr. v—0.3, three or four times daily, is a valuable prophylactic against cystitis. Counter-irritation along the spine and suspension are useless. The diet should not be heavy, and if gastric crises occur special care should be taken in this direction. As some cases of syphilitic leptomeningitis may simulate tabes so closely that the differential diagnosis is difficult or impossible, some prescribe antisyphilitic medication as a routine treatment. In old cases of tabes this is apt to be harmful. In those, however, in which the cellular content of the cerebrospinal fluid is great and a Wassermann reaction present it should be tried. Mott has stated that in true tabes the cells do not diminish with such treatment, but in syphilitic pseudo-tabes they will. In such cases, therefore, if not otherwise contra-indicated, salvarsan, mercury, and the iodids should be given a trial with reason to hope for improvement. Salvarsan has been found to relieve the pains even when no other benefit resulted. It should be remembered in using this agent that meningo-encephalitis and myelitis, sometimes fatal, may be caused by large or frequent doses.[1] Arsenic, chlorid of gold and soda, and nitrate of silver seem to have some influence upon the course of the disease.

The fulgurant pains, or those of the various crises, are occasionally so severe as to require codein, or even morphin, though the use of the latter agent is always to be postponed until other means are exhausted. Antipyrin or salol and phenacetin may also be tried. Heroin is often almost a specific. In some cases the crises are so severe that even morphin fails to give relief. The cutting of the posterior roots (seventh to tenth) of the dorsal nerves (rhizotomy) has been efficacious in the relief of intractable gastric crises.[2] In any case the patient should live a simple, regular life, avoiding excesses of all kinds, and particularly sexual and alcoholic indulgences.

Electricity is of service in relieving paresthesia and pain. For this purpose either a rapidly interrupted faradic, static spark or high-frequency current applied to the extremities may be tried. Hydrotherapy is a serviceable measure if judiciously employed. Neither cold nor hot baths are free from deleterious effects, but tepid baths (80°–90° F.—26.6°–32.2° C.), combined with gentle friction of the body-surface, are signally useful.

Recently it has been discovered (Frenkel) that the *ataxia* can be greatly improved by systematic exercises designed to train the muscles in coördinated movements. The important points are to avoid fatigue and irritation, and to increase gradually the complexity of the tasks.

[1] Newmark, *Amer. Jour. Med. Sci.*, Dec., 1912, p. 848.
[2] Frazier, *Amer. Jour. Med. Sci.*, Jan., 1913, 116.

HEREDITARY ATAXIA.

This may be divided into two types, viz., the spinal type or Friedreich's disease and the cerebellar.

FRIEDREICH'S DISEASE.

(*Friedreich's Ataxia.*)

Definition.—An hereditary disease, first described in 1861 by Friedreich. The symptoms are primarily manifested in early life, and the disease is characterized by ataxia, defective speech, nystagmus, absence of the knee-jerk, and more or less secondary deformity, as spinal curvature or talipes.

Etiology.—1. Family tendency (heredity) has a strong influence. A single case, however, may develop in a family. It is due to an inherited and inherent lack of vitality in certain parts of the nervous system (abiotrophy).

Age.—Most commonly the disease appears between the third and twelfth years, though it may appear earlier or later.

Infectious fevers (in particular) and other acute diseases frequently precede the evolution of this complaint. Trauma and many other conditions have been described as exciting causes.

Pathology.—The cord frequently is smaller than normal, and at times there is some thickening of the membranes over the posterior aspect. Microscopically are found degeneration of the posterior columns, more marked in the column of Goll, of the crossed and direct pyramidal tracts, the direct cerebellar tract, and that of Gowers. In the columns of Clarke are found atrophy of the cells with loss of fibers. In some cases the posterior roots may also be found degenerated, also the cells in the anterior horns and the peripheral nerves. The cerebellum is frequently smaller than normal, and degeneration of the cells composing the dentate nucleus occurs. The cells of Purkinjie are also atrophied in some cases. Changes have also been found in the cerebrum consisting of atrophy of the gyri, and changes in the cerebral cortical cells. Atrophy and disappearance of the cells of the posterior root ganglia have also at times been observed. Changes in the muscles similar to those found in the dystrophies (p. 1260) may occur. The degeneration in the posterior columns is more marked usually than in other parts, the pyramidal tracts being next in severity.

Symptoms.—The earliest evidence of the disease is impaired coördination, first in the legs, and, later, in the arms; it is most marked when the eyes are closed. Attention is often called to this symptom by the fact that the child stumbles, ambles, and staggers, and cannot walk properly. The gait, however, lacks the pronounced stamp of true ataxia. Rutimeyer has pointed out that in many cases the great toes are turned upward. Some affected children never learn to walk. Romberg's symptom is generally present. Movements of the arms, when these are ataxic, are irregular and jerky, and jerky movements of the head may also be observed. Bilateral nystagmus develops and the speech becomes affected. At first there is a mere impediment (a stuttering), but later syllables or even whole words are omitted and an unintelligible jargon results. The knee-jerks are almost always absent. There is no optic atrophy,[1] nor are

[1] See Hereditary Cerebellar Ataxia, p. 1152.

any sensory symptoms present as a rule. The sphincters are not involved. There are no trophic changes in the skin or the joints, and no visceral crises. Vasomotor symptoms—flushing, sweating—are sometimes observed. There is usually no mental change.

Talipes and spinal curvature are generally met with after the disease has existed for some time. In old cases muscular weakness and wasting are present, but the muscles do not give the reactions of degeneration.

The **course** is always slow. It may last for many years, thirty or even more.

Diagnosis.—Usually this is not difficult, and especially when more than one case exists in a family. The age, incoördination, shambling gait, nystagmus, scanning speech, and deformity are strikingly characteristic.

Differential Diagnosis.—*Tabes dorsalis* appears later in life, and the preataxic stage (pain, absent knee-jerk, and ocular symptoms) is generally well marked. It is absent in hereditary ataxia, nor does the latter present the sensory and visceral symptoms met with in the former. The gait is very different and the Argyll-Robertson pupillary changes are never present.

Ataxic paraplegia shows an exaggerated knee-jerk, the presence of ankle-clonus, and an absence of the ocular symptoms, nystagmus, and the scanning speech.

Disseminated Sclerosis.—In this disease the tendon jerks are usually increased, optic atrophy, indicated by a pallor of the temporal halves of the discs, is present; the speech is more likely to be sing-song instead of thick and indistinct, and epileptiform and apoplectiform attacks are liable to occur, and the disease usually comes on later in life (twenty to thirty years).

The **prognosis** is necessarily bad. The disease is progressive, though it does not kill directly. It may last thirty years or more.

Treatment.—Little or nothing can be accomplished. The same general treatment should be pursued as for locomotor ataxia.

HEREDITARY CEREBELLAR ATAXIA.

(*Marie and Nonne.*)

In this type the most pronounced pathological change in most cases is an atrophy of the cerebellum, in others defective development of the tracts in the cord leading to the cerebellum has been found. The etiology and symptoms are similar to those of Friedreich's disease, except that it usually develops somewhat later in life, the knee-jerks are present or increased, and optic nerve atrophy, diplopia, and Argyll-Robertson pupil may be found.

ATAXIC PARAPLEGIA.

(*Posterolateral Sclerosis.*)

This name was given by Gowers to a condition in which spastic paraplegia and ataxia coexist, owing to simultaneous involvement of the lateral and posterior columns. A similar condition may follow an acute

myelitis, representing the chronic type of that disease (p. 1137). Disseminated sclerosis may possibly present the same symptoms. The type Gowers describes occurs chiefly in males of middle age. Traumatism and exposure seem to predispose to the disease, as does syphilis very rarely.

Symptoms.—These develop insidiously. The patient tires rapidly. and some impairment of the power of walking is observed. In turning quickly he stumbles, and there is difficulty in walking in the dark, or even in standing when the feet are close together. The reflexes are increased at an early date, and spasticity supervenes and is progressive, though it never becomes as marked as in uncombined lateral sclerosis. The gait is somewhat similar to that met with in locomotor ataxia, but it lacks the forcible stamp present in *that disease.* When the arms are involved the same ataxia, with weakness, spasticity, and increased reflexes, is met with. Sensory symptoms are generally absent and fulgurant pains are never present. When pain occurs at all, it is of a dull character and often in the sacral region. Optic atrophy does not occur. Nystagmus is often seen, though other eye-symptoms very rarely appear. Sexual power is lost. The sphincters are not usually involved, though retention of urine may occur. Ultimately, the case generally partakes more of the nature of a lateral sclerosis, but the features of a posterior sclerosis may rarely predominate. Mental symptoms often develop in the late stages. The so-called Erb's type of syphilis of the cord produces a very similar group of symptoms. In this, however, there is incontinence of urine and sometimes of feces.

The *diagnosis* is easy in typical cases. The ataxia, with myotatic irritability and spasticity in the absence of sensory and ocular symptoms, is characteristic.

The *treatment* consists in maintaining the general health by proper hygiene, food, etc. If due to syphilis, a vigorous antisyphilitic treatment is necessary.

COMBINED SYSTEM SCLEROSIS.

(*Subacute Combined Sclerosis of the Spinal Cord; Diffuse Degeneration of Spinal Cord.*)

THIS affection, while described by many as a true combined sclerosis or system disease, is more correctly classified as a diffuse process, as has been done by Putnam and Taylor.[1] It was first described by Lichtheim in 1887, in cases due to pernicious anemia. Putnam, in 1891, described cases due to other causes, viz., influenza, chronic diarrhea, lead-poisoning, and malaria. It occurs most frequently between thirty and sixty, and always follows some chronic wasting disease or toxic condition. It has been described in pellagra.[2]

The posterior columns are usually first and most involved. The lateral columns, especially the crossed pyramidal tracts, are also soon affected. Other tracts may become affected later, as do also the cells

[1] *Journal Nervous and Mental Diseases*, Jan., 1901, p. 1.
[2] *Amer. Jour. Med. Sci.*, Jan., 1911, p. 94.

in the anterior horns and, in some cases, the anterior nerve-roots. The blood-vessels in the affected areas are engorged, their walls thickened.

The initial symptom is usually a persistent paresthesia, usually of the feet; with this some weakness, rigidity, and possibly ataxia will be found. Later the arms become similarly affected. At this time the tendon jerks will usually be found increased. There may also be pain in the back and limbs. Later the muscles become flaccid and the deep reflexes lost. Late in the course of the disease loss of sensation and muscular atrophy may also occur.

The *diagnosis* is based upon the presence of the above symptoms occurring in one suffering from any of the causes mentioned.

The *prognosis* is bad, death usually occurring in from six months to three years.

The *treatment* consists of general measures to combat the cause if known and improve the general health. If anemia exists, iron and arsenic should be given in full doses.

SYRINGOMYELIA.

Definition.—A neurogliar overgrowth of more or less vertical extent, and situated in the gray matter of the cord in the neighborhood of the central canal. Its symptomatology is not constant, but the following have come to be looked upon as typical of most cases, viz., progressive muscular atrophy and dissociation of sensation (*i. e.*, impairment or loss of temperature—and pain-sense, with retention of the tactile and muscular sense and trophic and vasomotor disturbances).

Etiology.—The symptoms usually develop during the second or third decades. The exciting cause is uncertain. Traumatism by producing hemorrhage into the gray matter possibly may cause some cases. Many sufferers from this disease have congenital anomalies of various sorts, as abnormal smallness, disproportionately large hands and feet, varieties of club-feet, etc. Syphilis plays no direct part, but may have some influence by its causation of diseased blood-vessels.

Pathology.—The usual seat of the process is in the cervico-dorsal region, but it may be in other regions or extend throughout the length of the cord, even into the medulla. It consists of an overgrowth of neurogliar tissue (gliomatosis) in the region of the central canal of the cord. This breaks down and forms a cavity, which usually extends irregularly in a transverse direction backward into the posterior horns, or may extend into the anterior. The cavity is lined with a varying thickness of gliomatous tissue. Secondary degenerations occur in the white matter, either ascending or descending, according to the tracts cut off. In most cases the condition results from a congenital anomaly of the central embryonal tissue, resulting during the early years of adult life in a slow-growing hyperplasia, having some of the characteristics of a benign neoplasm and a marked tendency toward the formation of cavities.

Symptoms.—Owing to the fact that different levels of the cord are involved, and that the extent claimed by the process varies in different

cases, it will readily be understood that no account, however concise, will fit every case. The disease is of *slow onset.* The commonest situation of the cavity is the lower cervical region; when this is the case the earliest symptoms appear in the hands, there is a numbness, loss of the pain and temperature senses, usually in an area bounded by a horizontal line surrounding the limb (glove anesthesia), and preservation of the touch sense. This constitutes the *dissociation of sensation*, perhaps the most characteristic symptom of the disease. The first symptom usually noticed is weakness and atrophy of the muscles of the hands, which show changes in the electrical reactions and fibrillary tremors, as in progressive spinal muscular atrophy. The atrophy may begin in other groups, according to the location of the lesion. Vasomotor and usually trophic changes occur in the ends of the fingers, especially multiple painless whitlows. Neuralgic pains and often exaggeration of the tendon reflexes are present in the arms. At the same time the syndrome of the transverse lesion of the spinal cord develops there is spastic paresis of the legs and disturbance of the functions of the bladder and rectum. As the disease progresses the symptoms become more general. The pain and temperature sensations are lost over large areas, the tactile and muscular sensations are preserved; there may, however, be areas in which all forms of sensation are lost. The trophic lesions are various; Charcot's joint or a dry arthritis may occur, there may be extensive bed-sores, or slight injuries may lead to chronic sores. Vasomotor disturbances are common, especially in the secretion of sweat. As a result of unequal involvement of the muscles of the back, lateral curvature of the spine occurs. Not infrequently, as a result of the involvement of the posterior columns, incoordination, with loss of knee-jerks, similar to that observed in tabes dorsalis, may develop.

As the morbid process extends upward, the centers in the medulla become involved, giving rise to bulbar symptoms, such as paralyses of the cranial nerves and disturbances of the urinary secretion. These are usually terminal signs. The symptoms, of course, vary with the position of the lesion in the cord, and in rare cases they may commence in the legs or indicate primary involvement of the medulla.

The disease originally described by Morvan of Brittany in 1883 should be included here. He had observed many cases prior to that time, but his attention was specially called to the matter by a case of whitlow which he incised, but to his surprise no pain whatever was experienced. He described *the disease as affecting the upper extremities*, with neuralgia, progressive paresis and wasting, dissociated anesthesia, and, later, painless whitlows and necrosis of the phalanges. Joffroy and Achard have made three autopsies upon cases dying of this disease, and in each syringomyelia was found. In Gombault's case neuritis was present. The current view is that *Morvan's disease* is a variety of syringomyelia.

Diagnosis.—The loss of pain and thermic sense, with preservation of the muscular and tactile senses, in association with the muscular wasting, which is most marked in the upper extremities; and with the spasticity of the lower extremities, and the trophic changes, especially in the fingers, constitute a group of symptoms that has come to be regarded as typical.

Differential Diagnosis.—*Hypertrophic cervical pachymeningitis* may be mistaken for this disease, and *vice versâ*. In this case, however, the pain is usually greater, the tactile sense is apt to be lost, and possibly the other senses also; but there is not the dissociation met with in syringomyelia. *Amyotrophic lateral sclerosis* presents neither sensory nor trophic symptoms, other than the muscular wasting. *Disseminated sclerosis*, apart from the tremor that is usually present, presents less trophic disturbance. Hemorrhage into the gray matter of the cord may cause a similar symptom-complex; in this, however, the onset is acute, and usually follows traumatism. The neural form of *leprosy* may present a clinical picture that cannot be differentiated. There are dissociation of sensation, trophic changes in the fingers, and muscular degeneration. Even spasticity of the lower limbs may occur, although this is rare.

The **prognosis** is always unfavorable, though the disease runs a very chronic course, lasting even fifteen or twenty years.

Treatment.—Nothing can be done, except by attention to hygienic and dietetic details.

TUMORS OF THE SPINAL CORD AND ITS MEMBRANES.

Under this heading are included the granulomata, parasitic cysts, and those due to other causes, as circumscribed spinal serous meningitis as well as neoplasms proper. The classification of Bruns is a convenient one:

I. Tumors which, arising in its envelopes, secondarily affect the spinal cord.

(*a*) Vertebral tumors arising from the spinal column or the soft tissues immediately surrounding it.

(*b*) Intravertebral tumors, which may be divided into two classes, in accordance with their relation to the dura mater.

1. Extradural tumors originating in the periosteum of the vertebra, the outer layer of the dura mater, or the fatty areolar tissue of the epidural space.

2. Intradural tumors originating from the inner layers of the dura, the arachnoid, the ligamentum denticulatum, the spinal roots, or the pia mater.

II. Intramedullary tumors of intrinsic spinal origin. Those arising from the vertebra are frequently malignant, either carcinoma or sarcoma, and are usually metastatic. Myelomata also occur. Benign growths, as osteomata, exostoses, chondromata, etc., are rare.

Extradural growths comprise sarcomata, lipomata, fibromata, myxomata, and chondromata. The first two are the most common.

Intradural tumors may be either diffuse or localized. Sarcomata, which may or may not be metastatic; endotheliomata, cylindromata, fibromata, and lymphangiomata. Fibromyxomata and fibrosarcomata are frequently found in connection with the nerve-roots. Cysts are also found within the dura. Intradural growths are usually found in the lateral or posterolateral surfaces of the cord, a fact which facilitates their removal.

Intramedullary neoplasms comprise gliomata, sarcomata, angiosarcomata, gummata, and tubercles. Gliomata usually give rise to the symptom-complex known as syringomyelia (p. 1154), but may be circumscribed.

Etiology.—As has been said, malignant and tubercular growths are often secondary to similar conditions elsewhere. Trauma seems to be a cause in some cases, especially non-parasitic cysts. They most frequently occur after middle life (forty to sixty). Extramedullary growths are more common than intramedullary. The former occur most frequently in the dorsal region; the latter in either the cervical or lumbar enlargement.

Symptoms.—The symptoms of extramedullary growths are due to irritation of nerve-roots, especially the posterior and compression of the cord (p. 1138). These are, therefore, pain in course of the roots arising from the affected region and a gradually developing paraplegia. If the anterior roots of either enlargement are affected, clonic spasms may occur. The pain is usually shooting in character, but in between the paroxysms it may be constant. Hyperesthesia may also be present in the skin area supplied by the affected nerves. According to Starr, the order in which the symptoms arise is commonly: (1) Peculiar pains of limited distribution; (2) Increase of reflexes below the lesion; (3) Paraplegia; (4) Loss of sensibility; (5) Loss of all subjacent reflexes. The pain, as well as evidences of compression, may at first be unilateral and the Brown-Séquard syndrome (p. 1077) may be present. As the growth enlarges they become bilateral. In some cases ataxic symptoms with increased reflexes may be more prominent than paralysis.[1] Segmental areas of anesthesia usually soon develop.

The symptoms of intramedullary tumors depend on their location; if within the gray matter, the symptoms are those of syringomyelia (p. 1154), otherwise they resemble those of a slowly developing myelitis, motion being lost before sensation. Pain is not apt to be a prominent symptom until the periphery is reached. The Brown-Séquard syndrome frequently occurs. The functions of the different segments of the cord are given on page 1071. Interference with these functions points to the particular part of the cord involved.

Circumscribed spinal serous meningitis[2] causes symptoms so much resembling tumor that it may be mentioned here. The dura is usually found very tense and bluish in color, but no macroscopic lesion is found. When opened the fluid escapes under marked pressure. The symptoms are usually sensory, especially intense pain of a segmental distribution first appearing before other symptoms develop, which are those of pressure, as in tumor. A peculiarity is that the symptoms are apt to vary from time to time, according as the pressure of the fluid increases or diminishes.

Course.—Tumors usually grow slowly, and therefore the symptoms are gradual in their development. Ordinarily there are periods of arrest or even improvement that are followed subsequently by further advance. The *duration* of spinal tumors is variable. Those of malignant nature or rapid growth may produce death in a short time; those that simply exert pressure and enlarge very slowly may not produce total disability

[1] Potts, *Journal Nervous and Mental Diseases*, Oct., 1910, p. 621.
[2] *Amer. Jour. Med. Sci.*, Nov., 1910, p. 719.

for several years. In general it may be said that from five to ten years is the ordinary limit after the first appearance of motor disturbance. Some tumors, however, particularly lipomata, produce only slight disturbances throughout life, or else no symptoms at all, remaining entirely latent.

The **diagnosis** involves three points: first, the recognition of the presence of the tumor; second, of its site; and third, of its nature. The prodromal symptoms of spinal tumor are often confounded with *neuralgia* or *lumbago*. It is sometimes possible to make a **differential diagnosis** by means of the presence, in neuralgic conditions, especially of intercostal nature, of the sensitive points along the course of the ribs, and of the existence, in the case of tumor, of exaggerated knee-jerks and sensitiveness over certain portions of the vertebral column. In the paraplegic condition it may be confounded with a *neuritis*, but in this there is tenderness over the nerve-trunks, absence of reflexes in the paralyzed parts. Sensory paralysis, if it exists, is not confined to the distribution of individual nerves, but is of the segmental type (Fig. 75), and the sphincters are not disturbed. Lesions of the cauda equina may be difficult to differentiate (p. 1159).

The *intrinsic diseases* of the spinal canal give rise to much greater difficulty, especially *myelitis* and *pachymeningitis cervicalis*. From the former the correct diagnosis may sometimes be suspected, because in tumor there are severe radiating pains and the symptoms are more pronounced on one side than on the other, and are apt to be more gradual in their development. Moreover, the symptoms of segmentary involvement are sharper and the root-symptoms more characteristic. From pachymeningitis cervicalis a tumor in the cervical region can be usually distinguished by the fact that the radiating pains are less severe and the symptoms not so distinctly bilateral. It may be impossible to distinguish a central tumor from *syringomyelia* unless the symptoms of root-pressure are quite distinct. *Pott's disease*, in its early stage, may also give rise to some difficulty. However, the rapid development of the kyphosis, and particularly the pain that is elicited by sudden pressure upon the head, renders it possible, after a reasonable period of observation, to recognize the true nature of the case (p. 1138).

The *diagnosis of the position of the tumor* has been largely discussed in the Symptomatology. In general, this is determined by determining the existence of symptoms dependent upon interference with the functions of certain segments of the cord (p. 1071, Fig. 75) plus disturbance of the functions of tracts of the cord, causing symptoms in parts innovated by the segments below those affected (*vide* Compression of Spinal Cord, p. 1138). The symptom-complex may, however, be considerably disturbed by the presence of multiple tumors. In these cases the majority ordinarily remain latent. It may also be said that the presence of root-pains suggests a meningeal seat, while pronounced paraplegia, dissociation of sensation, or the Brown-Séquard symptom-complex, points to the presence of a tumor in the substance of the cord itself. (See table on p. 1071.)

Finally, the recognition of the *nature of the growth* can often be made from the history of the existence of the tumor or an infectious process in other parts of the body; the rapidity of the growth; the age of the

patient; and occasionally from the results of an exploratory operation. It must be remembered, however, that it does not always follow that a tumor in the spinal canal is similar to that found elsewhere.

The **prognosis** depends upon the severity of the symptoms, the rapidity of their development, and the nature of the growth, if this should be known. Complete subsidence of all the symptoms may occur, even after a spastic paraplegia has existed. Of course this is only likely in those cases in which the tumor can be removed by operation or absorbed through the action of drugs.

The **treatment** depends wholly upon the recognition of the nature of the tumor; if this be syphilitic, mercury and potassium iodid should be given in full doses. If, on the other hand, it is not specific, and especially if extradural, operation would seem to offer a possibility of cure. This has been done successfully a number of times. Early operation in cases of circumscribed spinal serous meningitis has given most excellent results. As the prognosis is, in general, unfavorable as to cure and often gloomy as to life, the clinician should not hesitate to recommend surgical interference.

LESIONS OF THE CONUS MEDULLARIS, EPICONUS, AND THE CAUDA EQUINA.

As symptoms produced by lesions of these regions frequently resemble each other, they are described together.

The conus medullaris comprises that portion of the cord extending from the filum terminale to and including the third sacral segment. Lesions in this region are characterized by the absence of paralysis of the limbs and by paralysis of the sphincters of the bladder and rectum with loss of sexual power. There is also a saddle-shaped area of anesthesia involving the skin about the anus, perineum, scrotum, penis, and the mucous membrane of the urethra and anus. The testicle is sensitive, its nerve-supply originating higher up (Fig. 75, p. 1071).

That part of the cord comprised between the fourth and fifth lumbar as the upper and the second and third sacral segments as the lower limit, is known as the *epiconus*. A lesion here causes loss of Achilles jerks, preservation of knee-jerks, intact sphincters, motor paralysis, most marked in the peroneal muscles, with atrophy and reactions of degeneration and a steppage-gait. Sensory paralysis will be found in the distribution of the affected segments (Fig. 75).

As the spinal cord terminates at the second lumbar vertebra, tumors or injuries below this point produce symptoms only in so far as they compress or destroy the lumbar and sacral roots (cauda equina). This destruction may be partial or complete. If partial, there are *paralyses* of various groups of muscles and circumscribed areas of anesthesia, with radiating pain in the course of the affected roots. This, especially in the sciatic nerves, may precede by a considerable period the development of other symptoms. There may or may not be a disturbance of the functions of the bladder and sphincters. If all of the nerve-roots are involved, there are complete

anesthesia, complete paraplegia, flaccid in character, with reactions of degeneration in the muscles, loss of the knee-jerk and Achilles jerk, absence of the Babinski jerk, and rectal and vesical incontinence. In some cases there may be isolated paralysis of the bladder and rectum. These lesions may consist of tumors, such as are found in the membranes of the cord or on the nerve-roots, and it should be noted that, probably on account of greater space for their development, tumors in this situation are apt to be larger than those in other parts of the spinal canal. They may also consist of fractures or lesions occurring as a result of congenital anomalies, such as spina bifida. If tumor is suspected, the treatment is similar to that of spinal tumors (p. 1159). In conus lesions the incontinence of urine may be relieved by anastomosing the first lumbar and third and fourth sacral roots.[1]

IV. DISEASES OF THE BRAIN.

DISTURBANCES OF CIRCULATION OF THE BRAIN AND MENINGES.

Meningeal Hemorrhage.—Hemorrhage may be (1) extradural—(*a*) traumatic and (*b*) due to rupture of a vessel by erosion, the result of caries; or (2) intradural—into the so-called arachnoid sac—(*a*) traumatic; (*b*) due to injuries at birth (p. 1173); (*c*) due to pachymeningitis interna; (*d*) met with in general paralysis of the insane; (*e*) occurring in the course of anemia, scurvy, or some other profoundly altered blood condition; (*f*) in cardiac, renal, or pulmonary disease; (*g*) the result of strain —*e. g.*, whooping-cough.

The **symptoms** will depend upon the circumstances, whether the amount of blood is small or large, whether the onset is *gradual* or *abrupt;* they may be further obscured by the primary disease or by *shock*, if the cause is some trauma. In the slight forms absolutely nothing characteristic exists. In others there are *headache*, *vertigo*, *vomiting*, and possibly *mental confusion*, *convulsions*, or *coma;* in fact, the ordinary symptoms of apoplexy. The blood-pressure is increased. Cases due to traumatism are of most importance, both from a diagnostic and therapeutic point of view. If extradural, the hemorrhage is usually from a branch of the middle meningeal. When such is the case the symptoms are characteristic. They consist of a varying period, in extreme cases a day or more, in which, with the exception of a brief period of evidences of concussion, there are no symptoms, the patient possibly going about his business. Then he gradually becomes more and more stupid, muscular twitching, and some degree of paralysis upon the side opposite the seat of hemorrhage, and if the posterior branch is the one affected, sensory symptoms appear. A choked disk and Babinski reflex may also be found on this side and a dilated pupil upon the side of the hemorrhage (Hutchinson's pupil). When the hemorrhage is subdural,

[1] Frazier and Mills, *Jour. Amer. Med. Assoc.*, Dec. 21, 1912, p. 2202.

the symptoms usually appear more quickly and the paralysis is more profound. In these cases blood will be found in the cerebrospinal fluid.

The **treatment** is that of cerebral hemorrhage, except in those due to traumatism, when opening the skull over the seat of hemorrhage should at once be done. Cushing has recently done this in infantile cases with success.

HYPEREMIA.

Definition.—An abnormal increase in the amount of blood in the cerebral capillaries. The condition is not in any way associated with the primary phenomena of inflammation.

What has already been mentioned in the case of hyperemia of the cord is equally true in this case—viz., that while congestion undoubtedly may take place, there is nothing symptomatically pathognomonic in the fact, and hence we do not recognize it as a definite clinical entity. The transient apoplectiform seizures, which may occur during the course of paresis, brain tumors, and multiple sclerosis, have been ascribed to a sudden congestion; in other words, a localized active hyperemia. They should be treated by slight elevation of and cold applications to the head, mild purgation, and bromids internally.

Passive congestion is met with in cases of obstruction of the cerebral sinuses and veins, and is due to pressure on the superior cava or the innominate or jugular veins by tumors or aneurysms; also in suffocation and strangling, in cases of excessive strain, and in tricuspid insufficiency.

In passive congestion the veins and sinuses are engorged and more or less edema may be present. It may be suspected if in cases of mitral and tricuspid valvular disease of the heart chronic headache and hebetude occurs.

The **treatment** in such cases will consist in endeavoring to restore the circulation to as near the normal condition as possible (p. 666).

ANEMIA.

Definition.—A condition in which an insufficient amount of blood circulates in the cerebral capillaries.

It is due to exhausting discharges (diarrhea), an abnormally slow pulse or weak heart, to hemorrhage, obstructive endarteritis of the vessels supplying the brain, to syncopal attacks and dilatation of the intestinal vessels, owing to the too rapid withdrawal of ascitic fluid.

Disease of the blood itself may also cause the symptoms attributed to anemia of the brain.

Symptoms.—The most exaggerated type is met with after a profuse hemorrhage. There are *pallor*, *weakness*, *vertigo*, *headache*, *flashes of light*, *subjective noises*, *rapid respiration*, *cool skin*, possibly profuse sweating, and in extreme cases *coma*, *convulsions*, and *death*. We are more familiar with the ordinary fainting attack. When cerebral anemia is brought about more slowly, "irritable weakness" results. The patient is either *somnolent*, *dull*, and *apathetic;* or he may be a victim of *insomnia*. *Headache*, *vertigo*, *tinnitus aurium*, *muscæ volitantes*, and lowered muscular power are present. The patient becomes irritable on the slightest provocation. Marshall Hall has described a group of symptoms as "hydrocephaloid," from their resemblance to hydrocephalus; they occur especially in young children after diarrhea. There are *pal-*

lor, hebetude, contracted pupils, and *depressed fontanels*. The somnolence may deepen into a coma that often becomes more profound, until death results.

The transient attacks of paralysis and loss of consciousness which occur in those suffering from arterial sclerosis are due probably to a localized anemia caused by spasm of the vessels supplying the particular part of the brain affected.

The **treatment** varies with the cause. The recumbent posture is always indicated, and in some cases it is necessary to depress the head, administer stimulants, and even transfuse or inject a normal saline solution. Ordinarily it consists of improving the tone of the circulation and quality of the blood. In the transient apoplexies caused by arteriosclerosis, nitroglycerin in full doses is of service. A light and easily assimilable diet should be given during convalescence.

EDEMA OF THE BRAIN.

Definition.—An infiltration of serum into the subarachnoid space and a greater or less increase of ventricular fluid, with or without infiltration into the brain-substance.

Pathology.—The fluid is chiefly in the meshes and beneath the membrane. The ventricular fluid is increased in amount; the brain-substance is pale, and in some cases infiltrated and softened. Microscopically, lacunæ may be seen in the cerebral tissue, the perivascular spaces are dilated, and some slight degree of nerve-cell degeneration is often present.

Etiology.—Edema is met with in Bright's disease, in senile cerebral atrophy, and as a result of passive hyperemia.

Symptoms.—In general the symptoms are those of *anemia*, though nothing definite is known of them. Since the condition is always secondary, it may be that symptoms directly referable to the edema are masked by the primary condition. Cases of apoplexy are seen occasionally, in which the only postmortem finding is an effusion of fluid into the pia and ventricles. This has been termed "serous apoplexy." (See also Serous Meningitis, p. 1123.)

The **treatment** is that of the primary condition. Lumbar puncture may be employed.

VASCULAR DEGENERATION.

Arterial.—The cerebral arteries undergo a more or less decided degenerative change in the majority of people past middle life (Bichat said seven-tenths). It is met with much earlier, however, as a result of disease. Bright's disease, rheumatism, gout, alcoholism—in fact, any irritation of the vessel-wall, whether autogenous, the result of faulty metabolism, or whether introduced from without, as alcohol—is capable of bringing about a change of the inner seat of the vessel, to which Virchow gave the name "*endarteritis deformans*." The circle of Willis and its branches are the most frequent seats. Various stages may be met with in different vessels or even in the same vessel—viz. hyaline degeneration, fatty degeneration, liquefaction-necrosis, atheromatous ulcers, and calcification.

Syphilitic arteritis is not a true degenerative process. It is rather a proliferative process in which both intima and adventitia are involved. Arterial degeneration is the cause of many diseases of the nervous system; for instance, cerebral apoplexy, myelomalacia (p. 1134), neuritis (p. 1084). A condition resembling multiple sclerosis, due to disseminated areas of softening, may also occur. A symptom-group, characteristic of arteriosclerosis of the cerebrospinal vessels, consists of headache, vertigo, inability to stand well with the eyes closed, a gait consisting of short, shuffling steps, laughing and crying without cause, increased knee-jerks, and mental failure. Senile dementia is also so caused (p. 1194). Degeneration of the vessels of the limbs and cord may cause pseudoparalysis, as intermittent claudication (p. 1145). Spasms of degenerated vessels are liable to occur in the brain, also causing transient apoplectic attacks (p. 1161). Also such symptomatic conditions as headache, neuralgias, vertigo, tremor, and epileptiform convulsions.

Venous.—The veins are less liable to disease than the arteries, possibly because they are more yielding, yet the same pathologic changes may be met with in them. They are more commonly damaged by extension of inflammation from neighboring tissues or by pressure.

Aneurysm.—Dilatation of a vessel results from any of the causes above mentioned. The aneurysms may be very small—miliary—or often as large as a filbert-nut, and rarely as large as a hen's egg. They occur more commonly in males than in females. The middle cerebrals and basilar are most frequently attacked, and next come the internal carotid, the vertebral, and the anterior and posterior cerebrals. Miliary aneurysms are frequently found in enormous numbers upon the basilar branches of the cerebral arteries.

Symptoms of Aneurysm.—There may be none; but in any case they are due to pressure exerted by the mass, and are therefore comparable to tumors of the brain. In many cases the first evidence of any trouble is an *apoplectic attack*, and it is scarcely necessary to add that this is usually fatal. In other cases *headache*, *vertigo*, and *optic neuritis* are present, and more rarely a *subjective murmur*. Still more rarely an *objective murmur* may exist.

APOPLEXY.

Definition.—As defined by Dana, "apoplexy is a clinical term used to indicate a condition characterized by sudden paralysis, usually attended with loss of consciousness, and due either to the breaking or blocking up of a blood-vessel in the brain." Thus we have hemorrhagic apoplexy, due to the rupture of a blood-vessel (intracranial hemorrhage), and embolic or thrombotic apoplexy, due to either an embolus lodging in or a thrombus forming in a cerebral vessel (acute cerebral softening).

CEREBRAL HEMORRHAGE.

Definition.—Hemorrhage into the brain-substance: bleeding into the meninges is generally embraced in the definition (p. 1160).

Pathology and Etiology.—At the time of birth and during childhood there is some tendency to cerebral hemorrhage (see p. 1173). From this period to the age of forty the liability is small; after this, it progressively increases. The predisposing causes are alcoholism, syphilis,

and gout. Hereditary influence may also be a factor, as may also the infectious fevers. Rarely it complicates scurvy and purpura hæmorrhagica. The exciting causes are lifting heavy weights, straining at stool, coitus, and mental excitement; but hemorrhages occur in which no exciting cause can be determined. These causes are usually only operative in those predisposed. Transient apoplectiform attacks, due to sudden congestion, may occur in multiple sclerosis, brain tumor, and paresis. In intracerebral hemorrhage the blood will be found to have infiltrated the brain-substance, and, if extensive, it may have penetrated into the ventricle. In such cases the white matter is torn asunder, leaving a ragged space that is more or less filled with recent clot and fragmentary gray matter; if the ventricles have been entered, blood may escape from the lowest into the subarachnoid space. In less severe cases the territory involved is less extensive, and the blood may occupy a single space or several small spaces, forming mere separations of the nerve-fibers. Other changes take place according to the duration of the case. The blood changes color and gradually grows lighter, while reactive inflammation about the lesion results in the formation of a wall. The cyst—for such it has become through fatty degeneration of its contents—may remain as such or, when the lesion is a small one, connective tissue may form within and a scar result. The larger arteries are generally atheromatous, and an aneurysm is occasionally met with. The actual cause of the hemorrhage in most cases is the rupture of a miliary aneurism, which is a tiny dilatation upon a small vessel. Many of these can usually be seen on the degenerated vessels of a brain in which such rupture has occurred. The vessels otherwise present the changes of arteriosclerosis. A vessel may, however, rupture when miliary aneurisms are not present. Hyaline degeneration may in some cases be the condition present. Rupture may also occur in an area of softening due to extension to the vessel-wall of some neighboring form of inflammation. Such cases appear often to be due to injury to the head, the hemorrhage occurring some little time after the reception of the injury (delayed apoplexy).[1] It is very seldom that the actual source of the hemorrhage can be discovered.

Secondary degeneration follows a lesion occurring in the motor region (the cortex or internal capsule), so that sclerotic changes can be traced from the cortex through the corona radiata, internal capsule, crura, pons, and medulla, to the termination of the fibers in the cord.

Andral states that varicose veins occur in the pia, and that they occasionally rupture. Capillary hemorrhage may follow the plugging of a large vein, and of the larger vessels any one or more may be involved, but it has been observed that hemorrhage tends to take place at particular places. In more than one-half of all cases the lenticulo-striate artery (Charcot's artery of cerebral hemorrhage) gives way, and damages the lenticular nucleus and internal capsule. Other regions in the order in which hemorrhage occurs are as follows: centrum ovale, cortex, pons, peduncle, cerebellum, optic thalamus, and the posterior and anterior parts of the hemispheres. Hemorrhage into the cerebrum occurs twenty times more often than hemorrhage into the cerebellum; it may take place into the brain-substance, into the ventricles, or into the meninges, the latter form having already been considered. Ventricular hemorrhage in a great number of cases is caused by a more or less extensive lacera-

[1] Allen, *Journal Nervous and Mental Diseases*, October, 1908, 763.

tion of brain-matter, thus permitting the blood to escape into the ventricles. Not only the lateral ventricles, but the third and fourth also, may contain blood.

Symptoms.—As in the great majority of cases the motor tract is damaged, the following description is of a hemorrhage in that region. It must he remembered that other parts of the brain may be the seat of the lesion (p. 1166). Generally, the patient is seized without any warning, but in other cases *headache*, *depression*, and more or less *paresthesia* precede an attack. The loss of consciousness is usually the first manifestation, though for a few moments before, motor weakness, with or without spasmodic movements,[1] may exist. In very slight attacks consciousness may be preserved throughout or there may be a feeling of vertigo or mental confusion. The symptoms are in direct proportion to the extent and position of the hemorrhage. The patient falls, the face is usually congested, one side often expressionless, and the cheek flaps during respiration. Breathing is stertorous and, in grave cases, of the Cheyne-Stokes type; the pulse is generally feeble for a few moments, but soon becomes full and bounding in character. The blood-pressure in most cases is high, and a choked disk may be present on the side of the hemorrhage. The pupils vary, but are usually contracted. There is frequently a relaxation of the sphincters, and on raising the limbs it will be found that those of one side offer absolutely no resistance. The *temperature*, especially on the paralyzed side, is slightly lowered at first, but after a few hours rises to, or just above, normal. In grave cases it will either remain low or will mount up to 106° F. (41.1° C.) or even higher. Such cases are usually fatal. *Conjugate deviation* of the head and eyes takes place in marked cases; the deviation during the early stages may be toward the paralyzed side, as irritation causes a spasm of the muscles; for the same reason there may be early rigidity of all the muscles of the paralyzed side, but after the irritation subsides (a few hours to a day or two), the deviation is toward the lesion and away from the paralyzed side; in pontine hemorrhage the opposite to this occurs, as it is here due to involvement of the sixth nucleus, after decussation has occurred (p. 1097). As a rule, the symptoms that we group under the term apoplexy—viz., loss of consciousness, motor power, and sensation, with or without relaxation of the sphincters—pass off in twelve to twenty-four hours. In fatal cases the coma deepens, but death rarely ensues under twelve hours. In hemorrhage into the medulla or ventricles it may be more rapid.

During the first few days (stage of irritation) after the onset *febrile reaction* sets in, with irritative symptoms due to the inflammatory changes occurring about the original lesion. There are fever, sometimes delirium, twitchings or spasmodic movements of a more pronounced type, and sometimes rigidity in the affected limbs. The temperature of the paralyzed side is often from one-half to two degrees higher than the temperature on the sound side. At first all reflexes may be lost, but the tendon reflexes usually soon return, and the Babinski phenomenon (extension of the toes when the sole of the foot is irritated) very soon appears. The corneal and abdominal reflexes may remain permanently absent. Difficulty in swallowing and thickness and indistinctness of speech, due to muscular

[1] Convulsions at the onset of hemorrhage are rare except in children. When they do occur, they indicate that it is probably cortical.

paralysis, is usually present at first, but, as a rule, disappears. This must be distinguished from aphasia (p. 1175), which may result if the lesion is in the left side of the brain. Death may take place during this stage. Cases are generally fatal also in which a second "stroke" follows closely upon the first, indicating a fresh hemorrhage. After the reactionary period a stationary period follows; sooner or later control of the damaged members is then gradually, but not perfectly, regained. The degree of recovery is dependent upon the resumption of function of slightly damaged tissue or upon the compensatory activity of the other side of the brain. In certain cases the structural damage has been too great, and permanent paralysis remains, with rigidity, slight wasting, secondary contractures, and increased deep reflexes.

Ingravescent Apoplexy.—In certain cases the onset is slow, consciousness being lost gradually. Coma deepens, and the case, as a rule, terminates fatally.

Ventricular Hemorrhage.—This may be primary or secondary. The symptoms are very severe and death soon occurs. Blood may be found in the cerebrospinal fluid obtained by lumbar puncture.

Hemiplegia.—When this is complete, one side of the face and the arm and leg of one side, generally the same, are all involved (see Pontine Hemorrhage). The facial palsy is not complete, the frontalis and orbicularis oculi escaping. The tongue when protruded deviates toward the paralyzed side. As a rule, the arm is affected to a greater extent than the leg. The trunk muscles and muscles of swallowing and speech nearly always escape, possibly owing, as Broadbent suggested, to the functional unison of the spinal nuclei of the *two sides* that preside over them, and, since they habitually act together, he supposed that they might be stimulated from either hemisphere. If, however, the patient has a second attack affecting the other side of the brain, these functions are interfered with and symptoms simulating bulbar palsy result (p. 1129). This is known as *pseudo-bulbar palsy*, and may be distinguished from the true form by the absence of atrophy of the tongue. The paralysis is usually spastic, and, therefore, the tendon reflexes on the paralyzed side are increased, there is patellar and ankle clonus, and the Babinski phenomenon. The tricipital and bicipital reflexes and the scapulo-humeral reflex are easily elicited. The reflexes upon the unaffected side are also exaggerated, but the pathologic forms are rarely present. The abdominal, cremasteric, and other skin reflexes are lost on the affected side. This is important as a distinction from hysterical hemiplegia.

Sensation is, of course, absent during the period of unconsciousness. Subsequent sensory disturbances are not constant for all cases. In some cases permanent anesthesia for all forms of sensation upon the affected side persists, with loss of the skin reflexes. This indicates a lesion in the posterior part of posterior limb of the capsule, and lateral homonymous hemianopsia is usually associated. Occasionally only dissociation of sensation is present, tactile sensation being preserved, whilst muscular and thermal sensation are lost or diminished. The stereognostic sense is often seriously disturbed in these cases.

The special senses may be temporarily perverted or their functions in abeyance, but rarely do permanent disturbances occur.

Pontine Hemorrhage.—This is indicated by marked contraction of the pupils, high temperature, and paralysis of cranial nerves upon the side

of the hemorrhage and of the arm and leg upon the other (crossed paralysis). Bulbar symptoms may remain permanently.

Crossed Hemiplegia.—When a lesion occurs in the lower part of the pons, the fibers of the facial nerve that are involved have already decussated; hence facial palsy occurs on the same side as the lesion. The fibers coming from the cortex are implicated before their decussation, so that paralysis of the limbs occurs on the side opposite to the lesion. Lesion of the crus may lead to oculomotor palsy of the same side, and palsy of the face, arm, and leg of the opposite side.

Cerebellar Hemorrhage.—This is difficult to recognize. Paralysis of the limbs is usually absent, but of cranial nerves is common. Bulbar symptoms are marked, and death usually occurs.

Serous Apoplexy.—The cases present clinical evidences of apoplexy, but the only *postmortem* finding is an excess of serum, and this is in no way responsible for the apoplexy. These cases probably belong in the same category as those just mentioned, but occur in old persons whose brains have atrophied.

Course and Terminations.—As previously intimated, the course depends on the position and extent of the lesion. In the most extensive cases death rarely takes place under several hours. Hemorrhage into the medulla may prove fatal more quickly. In the milder cases, perfect recovery may take place in a few days or weeks. Generally, however, when little or no improvement occurs in two or three months, permanent changes result. The facial muscles soon recover, and next the leg. At first the patient is able merely to move the toes. Daily improvement then follows until he can support his weight; dragging of the feet rarely disappears absolutely. In the meantime a less pronounced change for the better has been taking place in the arm. This member very rarely recovers to the same extent as the leg, and secondary contractures develop in time, the hand and arm becoming flexed, while the leg is extended. The hand is usually bluish and cold, and swells if kept in a dependent position. Some ataxia may be noticed if motion is possible. Pain in the affected limbs sometimes occurs, in the majority of such cases a lesion has been found in or near the optic thalamus. Other later manifestations that are only occasionally met with are athetosis, posthemiplegic chorea (p. 1174), and tremors. Varying degrees of mental deterioration may develop and epileptiform convulsions occur.

There is no degeneration of the affected muscles as a rule; nor are there electric changes, except during the irritative period, when the response to stimulation is heightened. Occasionally marked atrophy occurs, and is due in some cases, as Charcot has shown, to changes in the cells of the anterior horns. In others no such change is found, and we are forced to regard the wasting as cerebral.

Differential Diagnosis.—Apoplexy is to be distinguished from other conditions causing unconsciousness, such as traumatism to the head, cardiac syncope, epilepsy, alcohol- or opium-poisoning, insolation, and uremia. If some previous history can be obtained, the difficulty of the diagnosis is lessened, though it may still be great. If there is evidence of a blow upon the head, the possibility of meningeal hemorrhage must be considered (p. 1160). In simple concussion there are evidences of shock without any paralysis; in *syncopal attacks* the pulse is very feeble and the face is pale, respiration being shallow and often suspended. The

sphincters are hardly ever relaxed; the reflexes are usually preserved and the skin is often moist. In *epilepsy* scarring of the tongue may be present, and there is a history of previous attacks, or, failing to obtain this, one can usually learn that a convulsion has immediately preceded the coma. With *alcoholism* the case is more difficult. The odor of alcohol on the breath is of no value, as spirits may have been given by a bystander; moreover, hemorrhage is common in alcoholics (*vide* table of differential diagnosis. In *opium-poisoning* the coma comes on gradually, and when not too profound the patient can be aroused when shaken or shouted at. The respirations, which are very slow and deep at other times, become somewhat quicker and shallower when he is aroused. In *insolation* the temperature suffices, as a rule, though, as stated, high temperature may occur in apoplexy. The presence of albumin is not conclusive evidence of *uremic poisoning* unless the centrifuge and the microscope reveal the presence of casts or other indications of renal change; even then the case may be one of apoplexy in a subject of nephritis. It is important to remember also that uremia may cause a hemiplegia, which, as a rule, is not persistent. In the case of *diabetic coma* the presence of sugar in the urine serves to make the diagnosis. When we meet with a comatose case in which there is absolutely no resistance when the limbs of one side are raised, while those of the other still exhibit some tonicity, particularly if the deep reflexes are exaggerated on the flaccid side, and a Babinski reflex and conjugate deviation of the head and eyes present, the probability is that it is an apoplectic attack. It is of great importance to tell whether the condition is due to hemorrhage, embolism, or thrombosis, although at times this may be impossible. The tabulated points of distinction given below may afford aid:

Embolism.	Hemorrhage.
Early adult life.	Late adult life; in early life rare.
Previous development of cardiac disease following acute rheumatism, sepsis, chronic valvular disease, aneurism, pregnancy.	Cardiac hypertrophy, arteriosclerosis, increased arterial tension. In children, previous infectious disease.
	History that the patient up to the time of attack was well; also the finding of casts in urine and other symptoms of chronic nephritis.
During the attack there is an absence of congestion of the face; the pulse is normal; in cardiac affections it is accelerated and irregular.	During the attack there are noted flushes (reddish) of the face, pulsating carotids, and slow pulse.
Temperature normal or but slightly disturbed.	Temperature during the attack is subnormal, followed by a rise, especially on paralyzed side.
The attack, as a rule, is short; if there is a protracted embolic infarction, the duration is long; usually the circulation adjusts itself promptly.	The duration is, as a rule, longer. Coma of long duration (about two days) gives a very unfavorable prognosis.
Hemiplegia is right-sided usually. Paralysis may occur first, followed by convulsions and coma.	Remote effects quite frequent; alteration in the urine—albuminuria, polyuria.
Ophthalmoscopic Examination.	
At times the ophthalmoscope reveals either a recent or an old embolus in the arteria centralis retinæ.	The retinal arteries may show various stages of arterio-sclerosis; as a result there may be a hemorrhagic retinitis or there may be a thrombus of the central vein of the retinæ. A mild degree of choked disk may be present.

Thrombosis.	Hemorrhage.
Prodromes, as transient attacks of weakness, numbness, vertigo and headache, frequent.	Prodromes not very frequent.
Consciousness frequently preserved.	Usually lost.
Age of patient greater (after 50), except in syphilitic cases, when it may occur in early adult life.	More apt to occur between 40 and 50.
Paralysis may develop slowly, sometimes taking several hours to become complete.	Develops at once.
Temperature changes not so marked (initial fall followed by rise).	Temperature changes marked.
Attack occurs while patient is at rest (during sleep).	Attack occurs during physical exertion.
Pulse weak, breathing quiet. Face not flushed. Vessels atheromatous.	Pulse slow and full, blood-pressure increased, breathing stertorous, face flushed.
Pupillary disturbances not marked.	Pupils unequal or contracted.

It is not an uncommon occurrence to have patients brought to a hospital dazed and smelling of liquor. These should always be carefully watched, for mistakes readily occur, and many such cases have been condemned to a prison-cell when they were really suffering from cerebral hemorrhage.

Prognosis.—This is serious, and even if death does not occur, more or less disability is sure to result. Even if the brain injury does not cause death, severe bed-sores are likely to develop upon the affected side, and the patient succumbs either to infection or exhaustion. If, after the primary attack, the blood-pressure remains high, other hemorrhages are likely to occur, and the prognosis is, therefore, graver. Coma persisting longer than two days and high temperature are also bad prognostic signs.

Treatment.—If a diagnosis of hemorrhage cannot be positively made, care must be taken not to do harm; therefore the treatment should be expectant. The patient should be kept as quiet as possible and in the recumbent position, with the head somewhat elevated, and preferably on the side, to prevent the paralyzed tongue from falling back into the throat. The clothing about the neck should be loosened to prevent constriction. An ice-bag may be put to the head and hot bricks or a hot-water bottle to the feet, while sinapisms may be placed on the back of the neck or on other parts of the body. The bowels should be made to move freely; a cathartic may be exhibited by the mouth (croton oil, gtt. j or ij), and at the same time an enema may be given. If the patient can swallow, calcium salts may be given to increase the coagulability of the blood. Nitroglycerin or veratrum viride may be given to reduce the blood-pressure. When the pulse is very slow and the blood-pressure either very high (280 mm. or over) or progressively increasing, Cushing[1] has advised making an osteoplastic flap on the side of the hemorrhage. In this connection it is well to remember that a moderate increase of blood-pressure is beneficial, as it is due to the increased effort of nature to get blood to the vital centers in the medulla which otherwise becomes anemic, due to its compression against the foramen magnum by the increased intracranial pressure. When consciousness returns the patient should be kept absolutely quiet for several days and only liquid food permitted. Later an endeavor should be made to keep up the tone of the affected muscles by massage and electricity. The general arterial disease should also be treated by appropriate hygiene, the use of the iodids, etc.

[1] *Amer. Jour. Med. Sci.*, June, 1903.

EMBOLISM AND THROMBOSIS.

(*Acute Cerebral Softening.*)

Embolism.—Definition and Etiology.—The obstruction of arteries or capillaries by material brought to the spot from some other part by the blood-current. The material, generally fibrin, usually comes from the heart, and is either a vegetation of a recent endocarditis or, more commonly, of chronic valvular disease; it may possibly be a fragment of the valve plus the fibrin in ulcerative endocarditis. In the latter case the plug is generally septic, giving rise to suppurative processes. An embolus may be washed from the auricular recesses, from an aneurysm of the aorta or carotid, or from atheromatous patches: rarely from the pulmonary veins.

In puerperal women, and in certain febrile processes (diphtheria and pneumonia) the coagulability of the blood is increased. Heart-clots form, and fragments may be washed into the cerebral vessels. Owing to the direction of the vessels the embolus most frequently enters the left carotid, whence it usually passes to the left middle cerebral. Almost any cerebral artery may be obstructed, but the cerebellar very rarely. Embolism occurs most frequently between the tenth and fortieth years of life. The middle cerebrals are most frequently involved, and next in order the internal carotid and anterior cerebrals.

Pathology.—The region of the brain that is cut off from its blood-supply by the embolus undergoes softening. The cortical changes are less marked than those of the central ganglia, since in the former case more or less anastomosis exists, and none in the latter. When the embolus is septic one or more metastatic abscesses result. The degree of softening varies in different cases within wide limits. There may be nothing more than a slight diminution in the consistence, the affected area being somewhat paler than normal, or absolute dissolution may occur, the myelin breaking up into granules, while the tissue becomes infiltrated with serum, and the vessels undergo hyaline or more often fatty change. The color of the part varies with the amount of blood. In recent cases it is red. As the hemoglobin is absorbed a yellow color appears, and soon predominates. Red and yellow softening are found chiefly in the cortex. The so-called white softening is met with particularly in the white matter. A variety of red softening in which numerous small hemorrhages exist has been termed capillary apoplexy, while *plaques jaunes* is the term given by the French to a form of yellow softening often seen in the cortex of old people. The ultimate changes depend in a great measure upon the extent of the lesion. If this is small, the granular débris is absorbed, and the proliferation of connective tissue results in the formation of a scar. On the other hand, if large the solid elements are removed, and the cavity that remains contains more or less fluid (a cyst). In many instances fibers, trabeculæ, and even vessels that have escaped destruction, pass through the cyst.

Thrombosis.—Definition.—Obstruction of a vessel due to clotting *in situ*. This may occur (*a*) in the arteries or (*b*) in the veins and sinuses.

In the Arteries.—**Etiology.**—Thrombosis results from disease of the vessel-wall, atheroma, endarteritis, or syphilitic arteritis, extension

from surrounding diseased areas, traumatism, in aneurysms, in depraved blood-states, and at the seat of lodgement of an embolus. Thrombosis of a cerebral vessel may rarely follow ligation of the carotid. In general we may say thrombosis results from (1) changes in the vessel-wall, (2) retardation of the blood-current, and (3) hypercoagulability of the blood. It occurs most frequently in the middle cerebral, basilar, internal carotid, and vertebral arteries.

Pathology.—The changes in the brain-tissue are precisely those described under Embolism. Within the vessel a clot is found adherent to the vessel-wall, and extending from the nearest large branch on the proximal side to the contracted branches on the distal side. If of recent and rapid formation, it is always of a red color. The slower the formation the paler the color. Such clots are often laminated. The ultimate changes are contraction and atrophy, or, more rarely, calcification, or even softening and removal, the vessel again becoming patulous.

In the Veins and Sinuses.—**Etiology.**—Thrombosis may be (1) primary, due to general causes, or (2) the result of local changes.

Primary thrombosis is less common than the secondary variety. It is met with in marasmic children (one of the causes of infantile hemiplegia—Gowers), in which the clot is called marantic thrombosis, cachexia, phthisis, carcinoma, and in blood-dyscrasiæ (anemia, chlorosis).

Secondary thrombosis usually results from an extension of neighboring forms of inflammation, caries of the bone, middle-ear disease, or meningitis. It may also be due to fracture of the skull or compression of a sinus by a tumor.

Pathology.—In primary thrombosis the most common seat is the superior longitudinal sinus. From this it spreads into the veins of both sides, and frequently also into the lateral sinuses of one or both sides. In secondary thrombosis the sinus nearest the local disease suffers. The veins emptying into the sinus involved become distended, often rupture, and in consequence the brain-tissue and pia become infiltrated. When the veins of Galen are blocked serum escapes into the ventricles. Red, yellow, and white softening is met with as a final result of the extravasation. Secondary thrombi are usually septic, and give rise to abscess formation.

Symptoms.—Following Embolism or Thrombosis of Arteries.—The symptoms necessarily depend upon the position and extent of the lesion. Often it is discovered *postmortem*, not having been suspected during life. We meet with many such cases occurring in late adult life. Then, too, extensive lesions may occur in those portions of the brain that never yield any localizing symptoms—the frontal region, for instance. Apart from the etiologic differences, the clinical pictures of embolism and thrombosis differ as follows: In the former the *onset* is sudden, without premonitory signs, and is in many cases accompanied by loss of consciousness. In addition to symptoms arising directly through implication of the particular part involved, there are those of *shock*. In the less severe cases consciousness soon returns and the apoplectic symptoms pass off. When more severe, *coma* supervenes and may prove fatal. When hyperemia occurs in or about the motor region the irritation may give rise to *convulsions*. In other cases *delirium* is a promi-

nent feature; hence three varieties of softening are described by some writers—the *apoplectic*, *convulsive*, and *delirious*, from the prevailing feature. Thrombosis may commence abruptly, but, as a rule, the onset is slow, the patient meanwhile complaining of vague pains, numbness, tingling, headache, and vertigo. It is observed that a gradually increasing impairment of the mind is going on, and that motor weakness, slight at first, increases until the function is lost. The special symptoms are, as stated, dependent upon the location of the obstruction. If this is in the middle cerebral artery, the most common seat, there will be *hemiplegia*. The trunk may be spared and one of its branches stopped. The latter run to the third frontal, ascending parietal, supramarginal, angular, and temporal gyri. Thus, then, we can account for the aphasia so often met with in these cases by the plugging of the branch that supplies the third frontal convolution of the left side. If both middle cerebrals are plugged, symptoms develop that are indistinguishable from hemorrhage into the ventricles. This condition is generally fatal. Thrombotic obstruction of the anterior and posterior cerebral arteries rarely causes symptoms, owing to compensatory circulation. "Hebetude and dulness of intellect may occur" (Osler), with obstruction of the anterior cerebral. Hemianopsia may arise from a lesion of the posterior cerebral, since it supplies the cuneus. Plugging of a vertebral artery causes symptoms of acute bulbar palsy, as does also that of the posterior inferior cerebellar artery, which is a branch of the vertebral,[1] in this the symptoms develop without loss of consciousness. There may or may not be slight weakness of the limbs of the side opposite the lesion which, if present, soon disappears. Pain and temperature sensations are diminished in these limbs and the opposite side of the face, and there may also be pain and paresthesia in this area. Ataxia may be present in the limbs on the side of the lesion. The muscles of deglutition, larynx, and soft palate are paralyzed on this side (p. 1129).

Cerebellar softening is rare. There may be no symptoms if only one hemisphere is involved; otherwise they are similar to those of cerebellar disease due to other lesions.

Thrombosis in veins and sinuses cause variable symptoms. Those directly due to the vascular disturbance are severe headache, optic neuritis, delirium or convulsions, and, later, great depression. Hemiplegia may result. When the superior longitudinal sinus is affected, epistaxis is common, while in lateral-sinus involvement post-auricular edema occurs. If the cavernous sinus is affected, there are exophthalmus and ophthalmoplegia on the affected side, with edema of the orbital and frontal regions. In secondary cases, moreover, we have to reckon with the cause. Since this is so often septic, septicemic symptoms are the rule.

Diagnosis.—This must be made, if possible, from hemorrhage; the points are given on p. 1169. If it cannot be made with certainty, treatment should be very guarded, as treatment for hemorrhage would do harm in thrombosis, and *vice versâ*.

Prognosis.—This is somewhat better than in hemorrhage, death not being so liable to occur and the resultant disability less.

Treatment.—*Of Embolism and Thrombosis of Arteries.*—Very little can be done in brain-softening. In the early stages, however,

[1] Spiller, *Jour. Nerv. and Ment. Dis.*, June, 1908.

while it is absolutely impossible to repair the tissue already damaged, an effort should be made to prevent the spread of the process. Rest in bed with the head low should be insisted on. When shock is present, it must be met by gentle stimulation, ammonium carbonate, and even by alcohol and digitalis in some cases; hot-water bottles may be applied to the body. Citric acid, in the form of lemon-juice, may be given to prevent further coagulation. Venesection is contraindicated. The bowels should be made to move gently and purgation should be avoided. Nitroglycerin and small doses of the iodids are useful in thrombosis. Later, as stated, symptoms of irritation often appear. In such cases the bromids should be given, and also a diaphoretic mixture, or ice should be placed to the head. In any case in which syphilis, rheumatism, gout, chorea, or other malady capable of causing or adding to the trouble exists, the original disease should be treated promptly and thoroughly. In the meantime efforts should be made to improve the patient's general tone by the strict observance of hygienic and dietetic rules.

Of Thrombosis of Veins and Sinuses.—Treatment in these cases depends largely on the cause. In the primary form it is that of the systemic disease. Good, wholesome, and easily assimilable food should be given, together with a tonic treatment. In secondary thrombosis careful search should be made for pent-up inflammatory products, which should be liberated at the earliest possible moment. The brilliant results of operative interference in some apparently hopeless cases should suggest its employment whenever there is good reason for suspecting septic sinus-thrombosis. The emunctories must act freely. Counter-irritation applied to the neck is of questionable value, but internally quinin, iron, and strychnin, and, if stimulation is necessary, ammonia and alcohol, will all be useful.

CEREBRAL PALSIES OF CHILDREN.

THE paralysis may involve all four extremities (diplegia), or be paraplegic or hemiplegic in its distribution.

Etiology.—They may be congenital or develop during the first few years of life, usually within the first two. The former, in the great majority of instances, are due to meningeal hemorrhage, sometimes venous, occurring during birth. In such cases there is often a history of difficult or forceps delivery; it may occur, however, during easy labors. When due to this cause, the symptoms are frequently diplegic, but may be either hemiplegic or paraplegic. Some are possibly due to a fetal meningo-encephalitis; another cause may be lack of development of the motor tracts, and in such cases a history of premature labor may be obtained. Either neurotic taints, alcoholism, or syphilis in the parents may have some influence in the causation. The congenital cases have been known as *Little's disease* and *birth palsy*.

The hemiplegic form is most frequent in the latter group. These cases usually follow the infectious diseases, and are due in some instances to either hemorrhage, thrombosis, or embolism in a branch of the sylvian artery; others may be due to a cortical polio-encephalitis (Strümpell) (p. 1183).

Pathology.—If the patient live for a number of years, the following lesions may be found: atrophy and sclerosis, either of a group of convo-

lutions, an entire lobe or the hemisphere is most frequent. The affected parts are firm and hard, and the convolutions smaller than the normal. The sclerosis may be diffuse, and there may be nodular projections (hypertrophic sclerosis).

Next most frequently is found *porencephalus*, by which is meant loss of substance, forming cavities and cysts on the surface of the brain which may extend into the ventricle. Porencephalus may be due either to hemorrhage occurring at birth, lack of development, or the lesions of apoplexy (embolism, thrombosis, hemorrhage), which may occur after birth. The primary lesion in the cases of atrophy and sclerosis is doubtful. Striimpell attributed them to a cortical polio-encephalitis.

Symptoms.—It is important if possible to recognize the occurrence of meningeal hemorrhage during birth, as treatment then may be of service. These are convulsions, asphyxia, tense and non-pulsating fontanelles, slowing of the pulse, inequality of the pupils, an intense choked disc, and blood in the cerebrospinal fluid. Usually the symptoms are not noticed until the child is several months old, when it will be observed that he is unable to sit up, and that the head rolls about, owing to weakness of the neck muscles. When it is time for him to walk, he does not attempt to do so, and examination will show more or less rigidity of the limbs. Later, when the child does walk, the gait will be more or less spastic, sometimes so much so that the knees rub against each other, and in extreme cases one leg may be crossed over the other (cross-legged progression). The deep reflexes are increased, unless the spasticity is so great as to prevent contraction of the muscles, and the Babinski phenomenon is present. The arms are also rigid, but usually do not suffer so much as the legs. The face is rarely affected. In the hemiplegic cases the affected side does not develop as well as the normal one, and the limbs are often shorter and the muscles smaller. They are firm, however, and the presence of the reflexes and normal electric reactions will distinguish the condition from a true atrophy, due to peripheral neuron disease.

When hemiplegia follows an infectious disease, there are usually convulsions, with or without loss of consciousness, followed by paralysis of a similar type to that above described.

Many of these cases show mental impairment, and suffer from epileptiform convulsions. They are specially prone to develop spasmodic conditions, such as *posthemiplegic chorea* and *athetosis*. The former consists of choreiform movements developing in the paralyzed limbs. There is also a condition consisting of intermittent tonic spasms affecting groups of muscles called hemihypertonia postapoplectica.[1] Athetosis consists of peculiar slow, worm-like movements, in some cases only of the fingers and toes; in others the arms and legs are also affected, and more rarely the muscles of the face. There is a marked tendency in the movements to overextension, and they are increased by attempts to move the limbs. When the face is affected various grimaces and contortions occur. In some cases these movements are the most prominent feature of the case, the motor paralysis being slight.

Diagnosis.—This should not be difficult if attention is paid to the history and symptoms. Paralysis due to neuritis or anterior poliomyelitis is distinguished by muscular atrophy, absent reflexes, etc. The paraplegic

[1] Spiller, *Phila Med. Jour.*, Dec. 16, 1899.

type might be mistaken for the hereditary spinal form (p. 1143). The history of the development of this condition and absence of cerebral symptoms easily distinguish the two. Athetosis may be mistaken for chorea; the history and presence of evidence of disease of the pyramidal tracts and rhythmic character of the movements are sufficient to make the distinction.

Prognosis as regards duration of life is good, as regards disability and cure, is bad. The symptoms may be relieved. If the diagnosis of hemorrhage during labor can be made, the skull may be opened and the clots removed, as has been successfully done by Cushing.[1] If spasticity is much greater than paralysis, relief may be obtained by cutting some of the posterior nerve-roots, as recommended for lateral sclerosis (p. 1141). Usually the treatment consists in passive movements and electricity applied to the extensor muscles to overcome the spasticity. Tenotomies and tendon transplantations may also be necessary. The failure of mental power, if it exists, must be treated, if not too extreme, by proper educational measures, and the epileptiform convulsions, if they occur, by the measures recommended for that disease (p. 1208).

APHASIA.

Definition.—By aphasia is meant either a partial or complete loss of the power of either expression or comprehension (or of both combined) of any of the usual signs of language, not dependent upon lesions of the peripheral nerves or organs, but upon lesions of the cortical centers concerned in speech, or the tracts connecting them.

Etiology.—Aphasia, in most cases, and practically always, if permanent, is due to a destructive lesion of either the centers or tracts above mentioned. Usually this is either caused by a cerebral hemorrhage or acute softening (*vide* apoplexy), and is, therefore, frequently associated with hemiplegia. It may, however, be caused by any organic lesion, as tumor, encephalitis, or abscess. Transient aphasia may be caused by so-called functional disturbances. It may follow severe fright, anger, hemorrhage, and exhaustion; occur as a symptom of migraine, or be caused by toxemias, as uremia, gout, vegetable and mineral poisons, and infectious diseases.

The Genesis of Speech and Location of Centers.—To properly understand the development of the symptoms of aphasia, some knowledge of the evolution of language is essential. The child learns to understand language before he can utter it. Through the different senses he perceives the different characteristics and appearance of an object. These percepts are stored away in the brain, and gradually the child learns to associate the name that he hears applied to a certain object with that object. The memory of the sound of this word is stored in the center for word memories, which is in the first temporal convolution of the left side in right-handed persons, and *vice versâ* in those who are left handed. Whenever this word is used, the various characteristics of the object are

[1] *Amer. Jour. Med. Sci.*, Oct., 1905, p. 563.

at once brought into consciousness by a stimulation of the different centers where the memories of these characteristics are stored. Eventually the child learns to make the various coördinated muscular movements necessary to pronounce the word. The memory of necessary movements of the lips (tongue, larynx) required to pronounce a word are also stored in a center, which in right-handed persons is the foot of the third left frontal convolution (Broca's convolution psychomotor center, glossokinesthetic center of Bastian). The exciting of one of these centers excites the others; thus, if we hear the ringing of a bell, a mental image of the other characteristics of the bell is formed, and the proper word designating that image comes into consciousness by stimulation of the center for word memories. If it is desired to pronounce the word, an impulse is sent from this center to Broca's convolution, which starts in motion the various movements of the organs of speech necessary to pronounce the word.

Even if a word is not audibly pronounced, mental images of words enter into thought processes, as do also the muscular movements necessary to pronounce the word. This is constantly being done in silent thinking, when the sounds of words are mentally recalled without visible movements of the muscles necessary to pronounce the word being made. This has been termed the *internal language.*

We learn to read by associating the visual appearance of certain symbols with the sound previously acquired of the respective letters and words. These so-called visual memories are also stored in the cerebral cortex, probably in the angular gyrus and its vicinity. When one reads aloud, the words are first recognized by the visual center, which calls up the corresponding sound in the auditory center, from which the glossokinesthetic center is stimulated, and the memory of the required muscle-movements necessary to articulate the word is called up. Then through the centers for these muscles in the foot of the precentral convolution (Fig. 79) the word is pronounced. When we read silently, the same process takes place by means of the internal language.

In learning to write the visual perception of the letters is associated with certain muscular movements of the fingers and arms necessary to make them. These memories are kept in the second frontal convolution of the left side (in right-handed people). This center has been termed the writing or cheirokinesthetic center. As in writing, each letter is self-dictated by means of either the spoken or internal language, the other centers involved in these processes are first excited. Reference to Fig. 79 will show the location of the centers mentioned and their probable connections.

From the foregoing it will be seen that while each center has certain functions, these are more or less dependent upon each other. It has been observed that a lesion developing suddenly in one center causes more or less interference in the function of the others; this is especially the case soon after the onset; later, permanent symptoms more closely related to the affected center remain. These early symptoms have been explained by von Monakow[1] to be due to a lowering of functional activity in a more or less distant part of the speech mechanism, due to the upsetting of the balance between the several parts of this mechanism produced by the destruction of one of the integral parts by the lesion. He termed this diaschisis.

[1] *Neurol. Centralblatt*, November, 1906, p. 1026.

Classification.—Based on whether the receptive or emissive function is affected, aphasia has been divided into sensory and motor. Sensory aphasia (Wernicke) is present when the peripheral apparatus being intact, the patient is either unable to understand the language which he has been accustomed to use or is unable to recognize the meaning of the

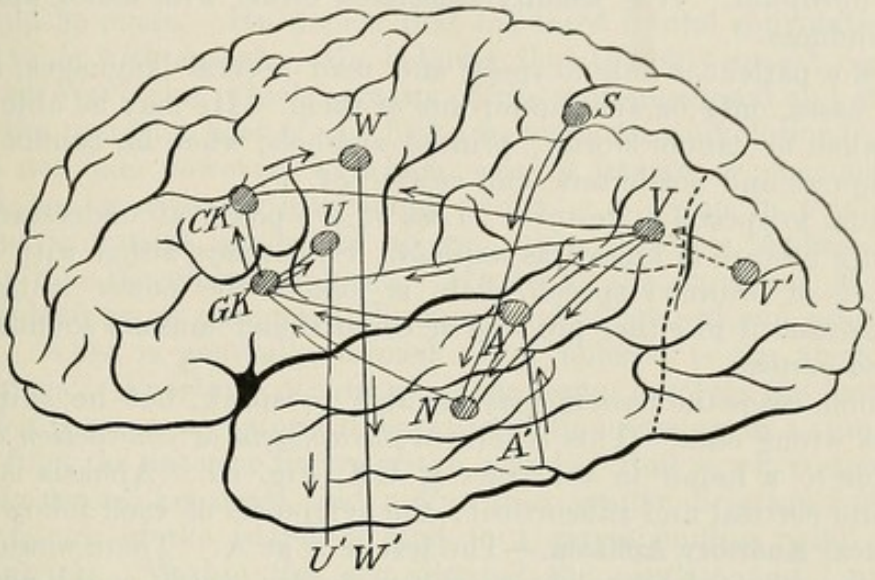


FIG. 79.—Diagram showing probable pathways of nervous impulses concerned in speech and writing:[1] A, Center for auditory word memories in first temporal convolution; V, center for visual word memories in angular gyrus; GK, glossokinesthetic center (Bastian), or psychomotor center, at foot of third temporal convolution; U, centers for muscles involved in articulation at foot of central convolution; CK, probable centers for memory of muscular movements involved in writing (cheirokinesthetic center of Bastian); W, centers for controlling muscles of arm and hand in central convolutions; A-A', tract from cortex of temporal lobe (auditory centers) to center for word memories; V-V', tract from cuneus to center for visual memories; W-W', tract from arm and hand centers to cells in anterior horns of cord and peripheral nerves controlling these muscles (pyramidal tract); U-U', tract from centers for muscles of articulation to centers in pons and medulla and nerves controlling those muscles (pyramidal tract). In speaking, impulses travel from A-GK-U-U'; in reading aloud, from V-A-GK-U-U'; in silent reading, from V-A-GK; in writing spontaneously, V-A-GK-CK-W-W'; in writing from dictation, A-V-GK-CK-W-W'; in copying, V-CK-W-W'; N, naming center (?); S, center for stereognostic sense in parietal lobe (Potts).

symbols, either written or printed, with which he was once familiar. In other words, he hears, but the once familiar sounds are to the patient as a foreign language; he sees them, but they look to him as those of a foreign language, and he cannot read understandingly. The former is known as *word-deafness* or *auditory aphasia*, the latter as *word-blindness*, *alexia*, or *visual aphasia*.

Closely related to this is a form termed by Freund *optic aphasia*, in which the patient when shown an object with which he was once familiar is unable to call up its name. He is, however, able to pronounce the name when he hears it. This symptom is termed *anomia*, and is probably due to a lesion in the center N (Fig. 79) or one of the tracts, SN, VN, or AN, for the reason that naming an object depends upon the recognition of its visual, auditory, or tactile characteristics. Another is the form known as mind or psychic blindness, in which, in addition to loss of the power of reading, there also is inability to distinguish between objects and persons and to know the proper use of things. This last is known

[1] In right-handed persons these centers and tracts are situated in the left side of the brain; in left-handed persons they are situated in the right side of the brain.

as apraxia (p. 1179). Mind-blindness, therefore, is word-blindness plus apraxia.

Motor aphasia (Broca's), or aphemia, is present when the patient, the peripheral speech apparatus (lips, tongue, larynx) being intact, is unable either partially or completely to give utterance to his thoughts. Loss of the power of writing, not dependent upon paralysis of the muscles, is termed *agraphia*. It is usually associated either with motor aphasia or word-blindness.

When a patient is able to speak and read several languages, he may, in some cases, only be aphasic for one of them. He may be able to read letters when he cannot words; printed symbols, when he cannot written ones; figures and not letters, and *vice versâ*.

Related to speech is gesture. Loss of the power of understanding or employing gesture is known as *amimia*. Sometimes, either with or without defects of ordinary speech, there is loss of the power in those who once possessed it to either produce or comprehend musical sounds. This is termed *amusia*.

In some cases the patient may be able to speak, but he skips words and uses wrong ones. This is termed *paraphasia* or *conduction aphasia*, being due to a lesion in the tract A–GK, Fig. 79. Aphasia is also divided into cortical and subcortical; the symptoms of each follow:

Cortical Auditory Aphasia.—The lesion is at A. There would be loss of the power of understanding spoken words; words could not be repeated or written from dictation. Stimulation of this center being necessary to activate GK, spoken speech would be defective, wrong words used, and paraphasia result. The internal language also being interfered with, the power of reading and writing is defective (tracts V–A–GK, Fig. 79).

Subcortical Auditory Aphasia.—The lesion is in the tract A–A′, the center A being intact. The patient is unable to understand spoken words. Reading and writing from dictation are not well performed. Some paraphasia is present, as the patient, when he pronounces a word, is unable by hearing to recognize its correctness. The internal language is intact and word memories can be recalled (A); hence silent reading and writing are not interfered with (tract V–A–GK, Fig. 79).

Cortical Visual Aphasia.—Lesion in center V. There is inability to read (alexia) aloud or silently, to write spontaneously from dictation, or to copy understandingly. Speech is not interfered with.

Subcortical Visual Aphasia.—Lesion in tract V′–V, loss of ability to read and copy understandingly. Through the tract V′–CK he can copy mechanically. The center V being intact, visual memories can be recalled; hence he can write imperfectly, as one with his eyes closed.

Cortical Motor Aphasia.—Lesion in center GK. Spontaneous speech, repeating words, and reading aloud are either completely or partially lost. Owing to the loss of the internal language, the power of silent reading and writing is also lost (V–A–GK, Fig. 79). Language is understood.

Subcortical Motor Aphasia.—Lesion in tract GK–U. Spontaneous speech, repeating words, and reading aloud are lost. The internal language being intact (A and GK), silent reading and writing are not interfered with. Language is understood.

Two or more of these centers or their connecting tracts may be diseased, and then a combined motor and sensory aphasia will result. See also p. 1176.

Marie's Theory of Aphasia.—The above is a presentation of the commonly accepted views upon this subject. In 1906 Marie[1] denied the truth of these views and advanced those entirely different. While these in their entirety have not been generally accepted, a brief mention of them should be made. He denies that the third frontal convolution has anything to do with speech. He believes that the only speech center is the zone of Wernicke, which consists of the supramarginal and angular gyri and the posterior part of the first two temporal convolutions. This region is not one, however, in which sensory images or memories are stored up, but is a purely intellectual center. A lesion in this region causes the symptoms described above, under Sensory Aphasia, and termed by him Wernicke's aphasia. He denies the existence of either pure word-deafness or pure word-blindness, and of cortical and subcortical varieties. What is generally termed motor aphasia is the above form plus anarthria. Anarthria occurs when the lesion involves the lenticular zone, which is an area comprised between a line passing in a transverse direction from the anterior fissure of the island of Reil to a corresponding point in the lateral ventricle, and a line in a similar direction from the posterior fissure of the island of Reil to a corresponding point of the lateral ventricle. Within this are situated the caudate and lenticular nuclei, the external capsule, the cortex of the island of Reil, and the internal capsule. The anarthria or aphemia of Marie is characterized by loss of speech, with preservation of the understanding of words, of reading and writing. It is an interference with the coördination of movements required for phonation, without motor paralysis. As has been previously stated, what is commonly termed motor or Broca's aphasia is this, plus a lesion of Wernicke's zone, which produces the symptoms usually known as sensory aphasia.

Closely related to aphasia are *apraxia* and the *stereognostic sense*.

Apraxia has been divided into sensory and motor or dyspraxia.[2] In the former, also known as *agnosia*, there is inability to recognize a heretofore familiar object by any one of the senses, and hence there is inability to name it. Thus, if an object is not recognized by sight, there is visual apraxia, or agnosia, by hearing, auditory apraxia, or agnosia, and so on. Visual agnosia is also known as mind-blindness (p. 1095). In the latter the object is recognized, but the patient has forgotten how to use it in the proper way; thus a pencil is recognized and named, but the patient does not know what to do with it. These symptoms are usually due to lesions of the left hemisphere in right-handed people. Motor apraxia has been frequently observed when the lesion was either in the first and second frontal convolutions of the left side or in the corpus callosum. *Stereognostic sense* is the name given to memories preserved in the cerebral cortex (parietal lobe, Fig. 79) of the characteristics of objects by which we are enabled to recognize them without seeing them. Thus, when a person who is blindfolded recognizes that an object placed in his hand is

[1] *La Semaine Médicale*, May 23, 1906, p. 241; also Dercum, *New York Med. Jour.*, Jan. 5, 1907, p. 7.

[2] Wilson, *Brain*, 1908, p. 164.

a dollar, he does so by his memory of the peculiar shape, hardness, weight, etc., of previous dollars. The sense depends upon a correlation of tactile, pressure, muscle, and temperature perceptions, and its interpretation by the higher psychic centers (parietal lobe, Fig. 79). Loss or diminution of any one of the above forms of sensation causes loss of this sense, which has been termed *astereognosis*, or tactile agnosia. Lately it has been proposed to restrict this term to inability to recognize the shape and consistence of objects, while inability to recognize them has been termed *asymbolia*.

Diagnosis of Aphasia.—This depends upon the presence of the symptoms detailed above, the peripheral apparatus being intact. This will serve to differentiate it from speech defects due to paralysis of the muscles concerned in speech, such as occurs in bulbar and pseudo-bulbar palsy. Motor aphasia is more common than sensory. The condition is usually associated with a right-sided hemiplegia, due to a lesion (thrombosis) in the middle cerebral artery, but may be due to any destructive lesion, as tumor, encephalitis, etc.

Prognosis.—The younger the patient, the better the outlook for some return of speech. Improvement may continue for a considerable time, and eventually fair power return. Many, however, never improve.

Treatment.—This consists of reëducation by the same methods as in teaching children to speak, read, and write.

PROGRESSIVE LENTICULAR DEGENERATION.

This is a rare condition described by Wilson.[1] It may attack several members of a family, but is not hereditary. It always occurs in young people, and may be either acute or chronic. The symptoms consist of bilateral tremor of the limbs which is increased by volitional movement (sometimes the head also is affected); spasticity of the limbs and face; dysphagia and dysarthria and finally anarthria; sometimes causeless laughter and emotionalism; difficulty in maintaining the equilibrium owing to the spasticity; sometimes transitory mental symptoms. There is no real motor weakness, and the reflexes are normal. Cirrhosis of the liver is always associated, but no evidence of disease of the liver is present during life. Death occurs in from six months to five years, and in all the cases examined a bilateral symmetrical softening of the lenticular nucleus was found. Wilson believes it to be due to a toxin which probably has some relation to the cirrhosis of the liver. In this connection the softening of the lenticular nuclei, due to carbon monoxid poisoning, is of interest.[2] These cases, the symptoms of which may appear several days after the patient has apparently recovered, have spasticity of the limbs and stupor, the reflexes, however, may be increased and the Babinski phenomenon present.[3]

[1] *Brain*, 1912, p. 295.
[2] Dana, *Jour. Nerv. and Ment. Dis.*, 1908, p. 65.
[3] McConnell and Spiller, *Jour. Amer. Med. Assoc.*, Dec. 14, 1912, p. 2122.

INFLAMMATION OF THE BRAIN.

(*Encephalitis.*)

Definition.—Encephalitis, strictly speaking, is an inflammation of the brain-substance, and does not include inflammation of the meninges, though in many instances the two conditions coexist as the result of a common cause, or one may precede and give rise to the other. Encephalitis is met with in two forms—(*a*) Suppurative and (*b*) Hemorrhagic.

SUPPURATIVE ENCEPHALITIS.

(*Abscess.*)

Pathology.—In very acute cases no time is given for encapsulation; when of longer duration, however, the abscess is well circumscribed, having a well-defined wall, within which there are cell-detritus, pus, and sometimes more or less altered blood. It may be offensive. About it the brain-substance is generally softened and edematous. The abscess is generally single, except in pyemic cases, and varies greatly in size in different instances.

Etiology.—Abscess of the brain is a more or less circumscribed process, due to (1) *Injury.*—In the majority of cases of abscess following head-injuries either a compound fracture of the skull exists, with or without *hernia cerebri* (fungus cerebri), or a punctured wound has been made. Less commonly it may follow a simple fracture, and rarely it is said to occur when neither a fracture nor even an abrasion of the scalp has been produced. Meningitis is an almost invariable concomitant. (2) *Extension from some neighboring inflammatory focus*, as from orbital, nasal, or aural disease, in which the bones have usually become affected. (3) *Pyemia*, in which case the abscesses are apt to be small and multiple. It is also met with occasionally in gangrene of the lung, bronchiectasis, ulcerative endocarditis, suppurative hepatitis, or bone-disease, and rarely in chronic septic processes. (4) *Congenital Heart-disease.*—Little is known of this condition. Within the past two or three years Northrup, Packard, Sir Dyce Duckworth, and Osler have reported such cases. (5) *Obstruction of an artery, vein, or sinus*, whether of spontaneous origin or the result of ligature, may give rise to abscess. Generally, however, the cerebral change is that of softening, and not of true suppuration. (6) *Intracranial tumors.* (7) *Infectious fevers.* Inflammation of the middle and internal ear is the most common cause, especially if the tympanum and mastoid cells are affected. Most cases occur between the ages of ten and thirty.

Symptoms.—These vary greatly according to the nature, situation, and size of the abscess, and are frequently masked or confused by the coexistence of various complications, such as injury to the head, meningitis, septicemia, or an infectious disease. In acute abscess there are the symptoms of acute septic infection, to which are added those of focal disease of the brain. As the abscess is secondary, the septic manifestations usually appear first. They are, of course, chills, fever, leukocytosis, etc. Often the primary focus can be recognized as endocarditis or pneumonia. The febrile process continues, there is usually severe delirium, and finally the symptoms of brain disease develop, either slowly or abruptly.

The general symptoms are headache, vertigo, vomiting, and convulsions, all of which are very constant and develop early; later, depending upon the situation of the abscess, motor and sensory disturbances appear. The most common are hemiplegia, clonic spasms, irregular involuntary movements, aphasia, hemiparesis, and hemianopsia. Kernig's sign may be present if the motor region is involved, and the reflexes are usually greatly exaggerated. Congestion of the eye-grounds is common, but choked disks are rare. The course is rapid and severe.

Chronic abscess develops, as a rule, insidiously; of the general symptoms, fever of a hectic type is most important, and there is usually a moderate leukocytosis. The pulse may be very slow. Headache, often severe and localized, is common, and there may be frequent vomiting. There is often vertigo, occasionally convulsions, and sometimes choked disks. Cachexia may also occur. The focal symptoms are of the utmost importance. The commonest is hemiparesis of the opposite side, but hemianopsia, hemihypesthesia, or unilateral loss of the muscle sense, and astereognosis may also be present. A peculiar feature of chronic abscess is the slowly progressive character of the symptoms, indicating extension forward or backward in the line of least resistance, and not increasing pressure in one place, as in the case of tumor. Occasionally the abscess ruptures spontaneously into one of the cavities of the head (nasal, aural), and temporary relief may be experienced. An abscess may be "latent" in almost any region, these latent abscesses being typified in certain cases of congenital heart-disease. I do not think they were suspected during life in any of the cases reported thus far, and therefore optic neuritis has not been looked for. When the abscess is due to ear disease, phlebitis of the lateral and superior petrosal sinuses frequently coexists; in such cases there will be edema about the ear and neck and hardness of the jugular veins.

Diagnosis.—In the acute cases following injury little difficulty presents as a rule, though even in this group a latent period may exist. With such a history, however, the onset of headache, fever, delirium, and convulsive movements is decidedly suspicious, and, should optic neuritis also exist, practically no doubt can remain. When aural or nasal disease exists the head-symptoms should be carefully studied, since they are prone to develop in ear disease soon after a cessation in the discharge. A slow pulse associated with fever is very characteristic of abscess.

Differential Diagnosis.—Brain-tumor usually runs a more chronic course, and is seldom accompanied by fever, at least not until its final stage. The causes of abscess are absent, excepting in the case of tubercular tumors, when abscess may be associated. It may be impossible to differentiate cerebral abscess from meningitis, and the two conditions often coexist, as already stated.

The **prognosis** is always grave.

Treatment.—When an abscess is diagnosed immediate operation is indicated. Suspected cases may be treated symptomatically unless focal symptoms develop. It must be remembered, however, that in a great many cases no localizing symptoms appear, and, since we know that most abscesses occur either in the temporo-sphenoidal lobe or in the cerebellum, when we have reason to suspect the presence of one, these regions should be explored.

ACUTE HEMORRHAGIC ENCEPHALITIS.

Definition and Varieties.—This is a condition characterized by foci of inflammation scattered throughout the gray matter of the brain that are not accompanied by suppuration. The cortex alone may be affected (encephalitis of Strümpell), and a certain number of cases of cerebral palsy in children (p. 1173) are due to the process being localized in the motor region. Adults may be also attacked. The gray matter about the aqueduct of Sylvius, with involvement of the nuclei of motor nerves of the eye, is a frequent seat (polioencephalitis superior of Wernicke). The nuclei of other motor cranial nerves may be affected (acute bulbar palsy or polioencephalitis inferior). The cerebellum may also be involved. These forms may occur either separately or combined.

Etiology.—These are chiefly chronic alcoholism and the acute infectious diseases, especially influenza. Lead-, ptomain-poisoning, and trauma may also be causes. It is more common in children and young adults.

Morbid Anatomy.—This consists of hyperemia, hemorrhage, round-cell infiltration, degenerated blood-vessels and nerve cells in the affected areas. Poliomyelitis may be associated, they being similar affections.

Symptoms.—General symptoms, as headache, convulsions, vertigo, stupor, delirium, rigidity of the neck, more or less elevation of temperature, sometimes preceded by chills, may be present. The focal symptoms depend on the areas involved. The symptoms of acute polioencephalitis superior and inferior are given on pages 1098 and 1129. If the motor cortex is involved, there may be convulsions of the Jacksonian type, paralysis, either monoplegic, hemiplegic, or diplegic, with increased reflexes and the Babinski phenomenon. Ataxia, hemianesthesia, aphasia, and optic neuritis may also be present. Either the symptoms of poliomyelitis or a general diffuse myelitis may co-exist.

Diagnosis. Any combination of the above symptoms following one of the causes given, especially if any of the general symptoms are also present, would be suggestive. Meningitis might be confounded, especially in the early stages. Lumbar puncture might be of service in distinguishing the two.

Prognosis.—This is grave, but recovery may occur. In those that do, some permanent paralysis or epilepsy may remain.

Treatment.—This consists of ice-bags to the head, purgation with calomel, hexamethylenamin in full doses, and bromids and other sedatives if there are restlessness and delirium. Otherwise the treatment is symptomatic.

INTRACRANIAL GROWTHS.

(*Brain Tumors.*)

Owing to their close relationship, new growths, both of the brain and membranes, are here considered.

Pathology.—Rindfleisch has classified intra-cranial tumors according to the tissue from which they spring, thus:

1. Having their origin in the *membranes*, either extra-cerebral or

intra-ventricular; these include tubercle, gumma, carcinoma, sarcoma, myxoma, lipoma, cholesteatoma, and psammoma; small fibroids have also been described. Enchondroma and osteoma may arise from the falx or from the bones of the skull.

2. From *blood-vessels:* to this group belong aneurysms, tubercles, and gummata.

3. Originating in the *neurogliar tissue:* glioma.

4. Originating in the *connective tissue:* sarcoma.

I will here consider these new growths in the order of frequency with which they are met.

1. **Tubercle** is most common in children and young adults, and is generally multiple (see Tuberculosis, p. 286).

2. **Sarcoma** is usually of the round- or spindle-celled variety; there may also be melanotic lympho- or fibro-sarcomata. Sarcomata are apt to diffuse themselves through the brain-substance quite rapidly.

3. **Glioma.**—Infiltrating tumors, generally single, and showing no definite line of demarcation from the surrounding brain-structure. They may be soft, even telangiectatic, or quite firm. They often run a very chronic course.

4. **Gummata** are generally small and often multiple. They spring from the membranes or the adventitia of blood-vessels, or from connective-tissue septa. Frequently they are attached to the periosteum of the skull.

5. **Carcinomata** are secondary growths, and are generally small and round, but in some cases they perforate the bones of the skull, producing a fungus hematoides.

6. **Fibromata** are not common. They either grow in the membranes or aid in the formation of a mixed tumor, as fibro-sarcoma. Other tumors met with less frequently are as follows: 7. **Osteoma**; 8. **Enchondroma**; 9. **Myxoma**; 10. **Lipoma**; 11. **Angioma**; 12. **Cholesteatoma.**

13. **Hydatids** are rare, especially in America. They may develop in any part of the brain or its membranes, and are said to occur most frequently in children. 14. **Cysticerci** may also occur in the brain or its membranes.

15. **Brain-cysts** are probably most often due to absorption of areas of softening from any cause, but they also occur between the dura and skull, as has been described. The lack of cerebral substance, due either to imperfect development or to atrophy following vascular obstruction or injury at birth, has been termed porencephalia by Hesche.

Etiology.—Age and sex are the chief factors; tuberculosis is far more common in children than in adults, while gummata when found appear almost invariably in adults, as do malignant growths. As a whole, new growths are more common between the twentieth and fortieth years, and males are more often affected than females. Traumatism seems to be an exciting cause in some instances.

Symptoms.—These are (1) General, and (2) Focal.

General Symptoms.—*Headache* varies in degree and character; it is not of any value as a localizing symptom, nor is tenderness on pressure. Tenderness upon percussion, however, is often detected in the neighborhood of the tumor.

Vertigo in a mild form is quite a common symptom. In cerebellar cases it is often very marked.

Vomiting occurs in most cases, and generally bears no relation to the time of taking food; this constitutes an important point in the diagnosis. The vomiting is apt to be exaggerated in cerebellar tumor.

Papilledema or choked disk (p. 1092) is present, according to Gowers, in four-fifths of all cases; in 82 per cent. according to Oppenheim, and in two-thirds according to Knapp. It occurs most frequently and early in tumors beneath the tentorium. It may develop rapidly and lead to complete blindness by the development of consecutive atrophy, or, more slowly, and even show very little if any loss of sight for a long time. It is usually bilateral, but often more pronounced on one side than the other. In many cases this is on the side of the growth. In rare cases there is progressive atrophy of the nerves without swelling. Headache, vomiting, and *choked disk* are "classical symptoms" of brain-tumor, and when met with simultaneously are quite characteristic. Reversal and interlacing of the color fields may also be met with (p. 1231).

Mental disturbance is very common. Dulness and stupor are most reliable evidences of intracranial growth, and especially when occurring with any of the above symptoms. The patient may be emotional or hysteric. Pseudo-apoplexy may occur as the result either of the growth or of hemorrhage taking place about it.

Convulsions are focal, either Jacksonian (p. 1207) or general, depending on the location of the lesion.

Constitutional and other symptoms may include progressive weakness, loss of appetite and of flesh, amenorrhea, infantilism, pupillary changes, and changes in the pulse, respiration, etc., and possibly slight fever. In certain tumors in the basal ganglia hyperpyrexia occurs. High fever is often significant of meningeal inflammation, as in syphilitic cases.

The **focal symptoms** are of two kinds: first, those due to direct local action (irritation or compression), and second, those due to changes occurring about the growth—indirect irritation, hemorrhage or softening, or merely congestion; thus can intermission or remission in symptoms be explained. The chief regional symptoms are as follows in—

(*a*) Tumors in the *prefrontal region*. *Headache*, not limited to the frontal region, with more or less mental impairment and drowsiness (though this is not constant by any means); and perhaps a disturbance of the sense of smell. No motor or sensory symptoms are present, as a rule, although vertigo and ataxia of the cerebellar type have been observed. The tumor may, however, grow backward, and either encroach on the motor region or cause motor symptoms indirectly. Downward growth would result in aphasia. A tendency to punning or joking has been noticed in some cases.

(*b*) Tumors in the *motor region*. The early symptoms are irritative and, later, paralytic. The former give rise to *spasm*, which is often very localized at first, possibly in a few muscles (Jacksonian epilepsy). The point of origin and direction of spread of the spasm are valuable localizing symptoms. Sooner or later destruction of the area causes *paralysis*. We may have spasm in one limb and monoplegia of the other on the same side. It may be necessary at times to decide if a growth involved the cortex primarily or is subcortical. In the former case muscular

spasm usually occurs before paralysis, while in the latter paralysis appears first, the Jacksonian attack not occurring until the tumor has extended to the cortex. Involvement of the left third frontal region causes motor aphasia.

(*c*) Tumors in the *parietal lobes*. There is usually ataxia of the limbs of the opposite side and astereognosis; later on, homonymous hemianopia, and if the ascending parietal convolution (Fig. 68) is involved, diminution of tactile sensibility may be present. When the posterior part of the left side is involved (angular or supramarginal gyri) we may meet with word-blindness or mind-blindness.

(*d*) Tumors in the *temporal lobes* may be latent, or there may be disturbances of taste and smell. If the posterior part of the first convolution of the left side is involved, we have word-deafness or other psychical disturbance of hearing, giving rise to auditory aphasia.

(*e*) Tumors in the *occipital lobes*. A unilateral tumor produces lateral homonymous hemianopsia, in which the Wernicke hemianopic pupillary inaction sign is absent, while a bilateral lesion may cause blindness. In certain cases, too, mind-blindness results, or "soul-blindness," as it was at one time called (p. 1178). Visual hallucinations, as flashes of light, may also occur.

(*f*) Tumors of the *corpus callosum* are often latent; they may, however, cause unilateral or bilateral motor symptoms. Often some mental aberration is noted. Motor apraxia may also be present (p. 1179).

(*g*) Tumors of the *corpora quadrigemina*, owing to their relations to the cerebellum, often cause disturbances of gait similar to that caused by disease of that organ. There is also more or less paralysis of the motor nerves of the eye and loss of the power of associated movements of the eyes upward. There may also be lateral homonymous hemianopsia (Wernicke hemianopic pupillary inaction sign present) and deafness. There may also be weakness of the opposite side.

(*h*) Tumors of the crus often cause a peculiar type of crossed hemiplegia, in which the face, arm, and leg are involved on the opposite, and the muscles supplied by the third nerve (eye-muscles) on the same side. There may also be hemianesthesia (syndrome of Weber).

(*i*) Tumors involving the *base*, when growing in the anterior fossa, give rise to exophthalmos, disturbances of smell and vision, and possibly to mental impairment. When in the middle fossa the symptoms are chiefly those of involvement of the third and fifth nerve, consisting of ptosis and other oculomotor symptoms and facial neuralgia, with anesthesia in the distribution of the fifth nerve. When such anesthesia associated with pain is alone present, it indicates tumor or other destructive disease of the Gasserian ganglion. When the tumor involves the pituitary gland, temporal hemianopsia, amblyopia or amaurosis, optic atrophy, and frontal headache occur. If the functions of the gland are diminished, the syndrome of Fröhlich (*dystrophia adiposogenitalis*, *adiposis cerebralis*) develops. In this we may find adiposity, lack of sexual development, abnormal tolerance for sugar, lack of hair, subnormal temperature, polyuria, polydipsia, dry skin, and sometimes epileptiform convulsions (see Acromegaly). Tumors have been found in this region in cases of acromegaly (*q. v.*). When in the posterior fossa, facial neuralgia,

neuroparalytic ophthalmia, or seventh or eighth nerve involvement and crossed hemiplegia are met with.

(*j*) Tumors in or about the *basal ganglia*, if quite small, cannot be diagnosed. When of larger size those involving the thalamus may cause hemiplegia and hemianesthesia by pressure upon the internal capsule, and lateral homonymous hemianopsia by pressure on the optic radiation. They may also cause obstruction and consequent distention of the ventricles (internal hydrocephalus). They may also give rise to amimia (contralateral paresis of the face only during laughing or weeping) and hemichorea or athetosis. The functions of the corpus striatum are not well understood, but lesions of the lenticular nucleus are believed to cause either anarthria or dysarthria (pp. 1179 and 1180). Weakness of a monoplegic or hemiplegic type may also be caused.

(*k*) Tumors in the *cerebellum* are comparatively frequent, both in children and adults. When toward the outer surface of the lateral lobes the localizing symptoms may not be marked. By pressure upon either the aqueduct of Sylvius or foramen of Magendie they often cause internal hydrocephalus (p. 1190), and the symptoms due to that condition may complicate those due to the tumor. The symptoms of growths in this region depend upon their situation, whether in either the middle or one of the lateral lobes. Tumors in the space between the cerebellum, pons, and medulla, known as the *cerebellopontile angle*, cause symptoms similar to growths in the lateral cerebellar lobes and may be here also considered.

If the *middle lobe* is alone affected, the usual symptoms are rapidly developing—choked disk, severe headache, marked ataxia of the cerebellar type, vertigo, nystagmus, the Romberg symptom, weakness of the muscles of the back, and sometimes of those of the legs. Occasionally, rigidity of the muscles, retraction of the head, and tetanic-like seizures have been observed. The knee-jerks may be either increased, normal, or absent, and may vary to this extent in the same patient. Palsies of cranial nerves, especially the ocular, due to pressure and usually bilateral, may occur.

Growths involving one of the lateral lobes do not cause localizing symptoms unless they press upon the middle lobe, when those described above occur. They are apt to be more marked upon the side of the lesion, and the patient usually has a tendency to fall toward this side (the opposite may occur, however). When vertigo occurs, external objects move from the side of the lesion to the opposite side, the rotation of the body being in the same direction.[1] The sixth and seventh cranial nerves are those usually affected in lesion of the lateral lobes, the paralysis being unilateral and on the homolateral side. Others may be affected. Inability to rapidly pronate and supinate the forearm upon the side of the lesion may be present (diadococinesia of Babinski). Hemiasynergia, or the straightening of the leg, after the thigh has been flexed on the body and the leg on the thigh, in a jerky incoördinate manner, may also be noticed on the side of the lesion. The head is also sometimes held inclined to the shoulder of the opposite side.[2]

[1] Stewart and Holmes, *Brain*, 1904, p. 525.
[2] Batten, *Brain*, 1903, p. 71.

Tumors of the *cerebellopontile angle* are usually encapsulated fibromata attached to either the auditory or trigeminal nerves, usually the former. The early symptoms depend upon the cranial nerve from which the growth arises. If the auditory, there are attacks resembling those of Ménière's syndrome (p. 1105); if the trigeminal, there are neuralgic pains in the course of that nerve, with possibly sensory paralysis in its distribution. Other cranial nerves, especially the seventh, soon become affected, and the symptoms of tumor of the lateral lobe become more or less marked. The differential points between growths involving the lateral lobe, cerebellopontile angle, and pons are well given in the table of Stewart and Holmes:[1]

Symptoms and Signs.	Lateral Cerebellar Tumors.	Extracerebellar Tumors.	Intrapontine Tumors.
Optic neuritis.	Early and intense.	Variable.	Often absent or late.
Vertigo.	Subjective rotation of self from the side of the lesion.	Subjective rotation of self to the side of the lesion.	Indefinite.
Cranial nerves— V.	Rarely affected.	Often affected.	Affection of these nerves often bilateral. Paresis may be supranuclear or nuclear, and grouped according to nuclear arrangement.
" VI.	Weakness of conjugate deviation to side of lesion. Weakness of external rectus on side of lesion. Slow deliberate nystagmus to side of lesion.	Same as in unilateral cerebellar tumors.	
" VII.	Paresis slight if present.	Paresis more marked.	
" VIII.	Deafness on side of lesion incomplete and variable. Tinnitus general.	Deafness on side of lesion marked—generally complete. Tinnitus referred to ear on side of lesion.	Paralysis of a nerve on one side and of an adjacent or distant nerve on the opposite side.
" IX.	Never affected.	Occasional paresis on side of lesion.	
" X.	"	" "	
" XI.	"	" "	Permanent paralysis of conjugate deviation of the eyes.
" XII.	"	Supranuclear paresis on contralateral side.	
Motor system.	Homolateral paresis, ataxia, and atonia.	Homolateral paresis and ataxia; contralateral spastic paresis common—occasionally bilateral.	Paresis often bilateral, with spasticity. Ataxia general.
Sensory system.	No change.	No change.	Occasionally hemianesthesia.
Reflexes—Tendon.	Variable, often diminished.	Generally increased, especially on contralateral side.	Increased often unequally.
Superficial.	Normal.	Often diminished on contralateral side.	Diminished often unequally.
Plantar.	Flexor.	Flexor or extensor. Extensor on contralateral or both sides.	Extensor on one or both sides.
Sphincters.	Not affected.	Rarely affected.	Generally affected.

(*l*) Tumors in the *pons* produce symptoms according to their size and location. If high up, a crossed paralysis, similar to that caused by a growth in the crus, will result. When a little lower down a motor and

[1] *Brain*, 1904, p. 549.

sensory paralysis of the arm and leg of the opposite side with paralysis of the sensory portion of the cranial nerve on the same side may result. If the lower portion is affected, paralysis of the arm and leg of the opposite side with paralysis of the sixth, seventh, and eighth nerves will occur. More or less sensory paralysis may also be present. If the cerebellar peduncles are involved, forced movements and ataxia (cerebellar type) will result. If either the nucleus of the sixth nerve or the fibers of the posterior longitudinal fasciculus is involved, there will be loss of associated lateral movements of the eyeballs toward the side of the lesion, while the power of convergence remains.

Tumors of the medulla cause symptoms of progressive bulbar palsy (p. 1129) plus more or less hemiplegia and hemianesthesia. If the growth is small, the symptoms may be more marked on one side.

Course.—Many cases run a very chronic course. Others may have existed months or years without symptoms, and then develop suddenly, owing to hemorrhage, thrombosis, or acute softening about the tumor. Either improvement may take place or the case may speedily progress to a fatal termination.

Diagnosis.—The general symptoms are usually sufficient to warrant a diagnosis. The gradual onset and progressive character without fever, in the apparent absence of any etiologic factor, are, as a rule, enough to indicate that a tumor is present, while its location can only be determined by the focal symptoms. Recently the *x*-ray has been used with some success for the localization of tumors that have undergone calcareous degeneration.

The **prognosis** is always grave. Syphilitic growths are the only ones amenable to medical treatment. Cortical growths, especially if in the motor region or its neighborhood, if encapsulated, are amenable to surgical treatment. Growths in both the lateral cerebellar region and cerebellopontile angle and pituitary gland have also been removed, but the operation is more serious; tubercle may recover by the growth becoming encapsulated and calcified. Nothing can be said as to the possible duration of life. Several years may elapse between the appearance of the symptoms and their fatal termination, or death may occur suddenly.

Treatment.—In any case recourse should be had to mercury and the iodids, and this treatment should be pushed, since it will certainly benefit syphilitic cases, and it is believed to be of some value even in the non-syphilitic. Other symptoms should be met as they arise. The question of operation must be considered where medical measures have proved of no avail. If the situation of the growth is favorable and the nature of the tumor is not malignant, an operation is likely to be successful. The percentage of recoveries is increasing as the technic becomes more perfect (see Bruns, *Geschwulste des Gehirns*, etc.). When the growth cannot be localized, or is in a position unfavorable for operation, much benefit may be obtained by relieving pressure by the so-called operation of decompression. Blindness, which will surely result if choked disk is allowed to persist any length of time, and the severe headache may thus be prevented.[1]

[1] Frazier and Spiller, *Univer. of Penna. Med. Bull.*, Sept., 1906; Cushing, "Principles of Cerebral Surgery," *Jour. Amer. Med. Assoc.*, Jan. 16, 1909, p. 184.

CHRONIC HYDROCEPHALUS.

This affection is divided into external and internal hydrocephalus.

EXTERNAL HYDROCEPHALUS.

Etiology.—External hydrocephalus may depend upon a congenital smallness of the brain or upon a congenital enlargement of the skull. The space between the brain and the bone is filled by an excess of subarachnoid fluid (*vacuum dropsy*), or there may be a wasting of the brain, such as occurs in old age or in chronic cachectic conditions.

Pathology.—When the skull is opened, the bone is usually found to be thin; the dura is normal; the arachnoid is lifted from the surface of the cortex by a considerable accumulation of clear fluid of low specific gravity; the convolutions may be somewhat flattened and the cortex slightly thinned. Upon microscopic examination no changes are found in the brain-substance. Sometimes the effusion is general; sometimes it is sacculated.

The **symptoms** depend upon the form. In cases in which there is hypoplasia of the brain or in which the brain has wasted, no pressure-symptoms are present. All the manifestations are purely psychic in nature, and similar to those of *microcephaly* or *senile dementia*. In cases, however, in which the cranium cavity is abnormally large, it is probable that the real cause resides in a congenital excess of subarachnoid fluid.

The **prognosis** is gloomy; nevertheless, it is possible that the disease may undergo spontaneous cure as a result of rupture into the nasal fossa.

The **treatment** is the same as for the internal variety (*vide infra*).

INTERNAL HYDROCEPHALUS.

This is a condition in which one or more of the ventricular cavities of the brain are distended by the cerebro-spinal fluid. In the *congenital form* and in that occurring in early childhood, this is associated with more or less enlargement of the skull. In the later *acquired forms* the cranium does not yield so readily, and the enlargement does not exist or is slight.

The **etiology** of the *congenital form* is unknown, though the fact that it frequently occurs in several children of the same family has led to the supposition that it is dependent upon some hereditary influence. In some cases it has been referred to emotional disturbances suffered by the mother during pregnancy, and in still other cases an anatomic foundation has been discovered, such as enlargement of the pineal gland. It is generally supposed that the immediate cause is chronic ependymitis.

The acquired form is usually secondary to inflammatory conditions (particularly meningitis) or to brain-tumor. Some cases, however, occur in childhood that are apparently not due to either of these causes.

The **pathology** of the condition varies with its nature. In the congenital forms, upon opening the head the skull is found to be thin. The fontanels and sutures are either still open and connected only by a membrane, or closed by Wormian bones. The dura may be thickened, but usually is normal; the substance of the brain is slightly softened—although this is not invariably the case—and very much thinned. This thinning is, as a rule, particularly noticeable in the corpus callosum and commissures,

which may, indeed, either be torn apart or completely atrophied. The enlargement ordinarily affects the two lateral ventricles, the third ventricle, and the aqueduct as far as its entrance into the fourth ventricle, which is commonly less involved than the other cavities. The ependyma is sometimes smooth, but more often shows small projections, which, according to Virchow, are composed of brain-substance, but in some cases are due to proliferation of the glia tissue beneath the ependyma. The enlargement may not be uniform. If due to obstruction of the foramen of Monro, one or both lateral ventricles are usually enlarged, whilst the third ventricle either remains of normal size or is diminished. If due to enlargement of the pineal gland, the aqueduct does not show the funnel-shaped distention. Another cause upon which considerable weight has been laid is the closure of the transverse fissure between the cerebellum and medulla. The quantity of fluid may be enormous, as much as 4 or 5 liters (5 or 6 quarts) having been recorded. The thinning of the brain-substance is also remarkable when one considers that a cerebrum 5 mm. ($\frac{1}{5}$ in.) in thickness is apparently able to perform a large proportion of its ordinary psychic functions. The atrophy seems to affect particularly the white substance, especially the myelin-sheaths.

In cases of the *acquired form*, unless they occur early in life, the enlargement of the skull is not very noticeable; the substance of the brain shows considerable softening; the ventricles are moderately enlarged, and, particularly in the chronic forms due. to tuberculosis, are considerably roughened. The most pronounced cases are those that occur when there is a tumor in the occipital fossa which compresses the veins of Galen. Basal meningitis causing an obliteration of the foramen of Majendie is also a cause. In these cases the accumulation of liquid is slower, the brain yields more gradually to pressure, and the dilatation is more pronounced. Ordinarily, there is considerable flattening of the convolutions. In a few of these cases inflammatory changes in the ependyma have led to partial obliteration of the ventricles, particularly in the anterior horns or the lateral ventricles. Occasionally also bands of organized lymph may cross the ventricles in various directions; the liquid is of higher specific gravity and contains more albumin than in the non-inflammatory varieties.

Symptoms.—The most characteristic appearance in congenital hydrocephalus is the *globular enlargement of the head.* Upon palpation the fontanels are found to be still patulous and usually bulging, and the sutures are open. The head is usually so heavy that it cannot be held upright, but falls backward or to one side. The face appears proportionately very small. *Motility* is usually disturbed, the legs are spastic, and the child either does not learn to walk at all or only long after the usual time. There are sometimes choreic movements of the upper extremities. The *eyes* frequently show nystagmus and conjugate deviation, and often there is either choked disk or atrophy of the optic nerve. Fischer has described a *systolic murmur* that can be heard if the stethoscope is placed over the anterior fontanel. Its cause is unknown *Convulsive attacks* are common; they are epileptic in type, and, as a rule, ultimately cause death. *Intelligence* is usually considerably impaired. and sometimes the children are idiots; more often they merely show retardation of intellectual development. Occasionally—and this even in

the most pronounced cases—the intelligence is well preserved. Henoch records the case of a boy three years of age whose head was 75 cm. (29.6 in.) in circumference, and who could speak both French and German. Ordinarily, the children are quiet and apathetic, but they may be querulous. *Nutrition* is commonly seriously disturbed, the children sometimes exhibiting pronounced cachexia. They may, however, be well nourished and, to a certain degree, vigorous. The symptoms of the chronic form in adults are those of brain-tumor without focal symptoms.

The **diagnosis** is ordinarily very easy. Careless observation may lead to confusion with *rachitis* but the square shape of the head and the presence of other rachitic deformities in the skeleton should lead to a prompt recognition of the true nature of the case.

The **prognosis** is extremely unfavorable, the majority of the children dying about the fifth year. A few cases, however, may live until they reach young adult life, and still fewer apparently recover entirely.

Treatment is of course difficult. Potassium iodid and mercury have been employed without much beneficial effect. Cod-liver oil may be given to stimulate nutrition, and purgatives occasionally relieve pressure-symptoms temporarily. Among the mechanical procedures constant pressure upon the head seems the most valuable. This can be obtained by means of strips of adhesive plaster or by the application of an elastic band. Drainage of the ventricles has given good results in some cases.[1] If convulsions develop, they should be combated by bromids and purgatives. At times there may be difficulty in making the diagnosis from brain-tumor; *x*-rays may prove of service in distinguishing between the two.[2]

ACUTE DELIRIUM.

(*Acute Delirious Mania; Typho-mania; Acute Periencephalitis; Bell's Mania.*)

Definition.—An acute maniacal delirium associated with hallucinations, with a febrile course, of limited duration and of grave prognosis.

Pathology.—Visible changes are usually absent, there may be found minute pericapillary hemorrhages and degenerative changes in the ganglion-cells. Sometimes injection of the pia and minute hemorrhages into the gray matter may be observed with the naked eye. Cramer has reported a case in which the pericapillary spaces of the brain were filled with mononuclear leukocytes, surrounding which were recent hemorrhages; he also noted the fact that the ganglion-cells, instead of exhibiting normally formed chromophilic bodies, were filled apparently with fine dust.

Etiology.—The disease occurs in either sex with about equal frequency. Predisposing conditions are neuropathic heredity, nervous disposition, the presence of other nervous diseases, particularly neurasthenia and epilepsy, alcoholic or sexual excesses, and severe prolonged anxiety.

[1] *Rev. Neurol. and Psychiat.*, Jan., 1911, p. 1.
[2] Spiller, *Review of Neurology and Psychiatry*, Jan., 1911, p. 8.

It frequently occurs apparently as the immediate result of menstruation, parturition, injuries to the head, sunstroke, acute infectious diseases, particularly pneumonia and typhoid fever, and it may develop in the course of chronic mental diseases. Occasionally, however, it appears to arise without any definite cause.

Symptoms.—The disease usually commences with certain indefinite prodromes. These consist of *restlessness*, associated either with melancholia, preoccupation, or anxiety. The *intelligence* becomes distinctly decreased; the patient loses appetite, is constipated, and commences to emaciate. During sleep unpleasant dreams or nightmares almost invariably occur. Sometimes there is a sense of impending mental disorder. This period gradually changes to one of defiance, which perhaps, even in the prodromal stage, may lead to violence and injury to those in the neighborhood. The prodromal stage rapidly passes to *acute delirium*, in which two steps may be recognized—excitation and collapse. The excited stage commences suddenly; there is great confusion; the patients ejaculate disconnected sentences or words or even syllables. There is great anxiety, and even fear, and the patients exhibit intense excitement, suffering very often with delusions of persecution by their environment, and nearly always having hallucinations, either of sight or sound. Often their minds are occupied by some subject that had previously caused them great anxiety—either disgrace, business, or other misfortune. The mania is often dangerous; indeed, it is likely that the disease known as "running amuck" in the Malay Peninsula is simply one of the forms of acute delirium. The patient soon becomes restless, throws himself from one side of the bed to the other, and makes efforts to rise and escape from the room. The tongue is dry, the pulse rapid and weak. Petechiæ may appear upon the skin, and there is nearly always more or less fever, not rarely rising to 105° (40.5° C.) or even more. Rapid emaciation supervenes. There are all the objective symptoms of irritation of the brain—myosis and increased reflexes, and often hyperesthesia, although the patients pay little attention to any injury they may inflict upon themselves. Sometimes there seem to be curious imperative movements; at others, imperative ideas. In a case that I observed the patient rhymed, very imperfectly it is true, each two successive sentences. This stage of excitation soon passes into one of *stupor* and *collapse;* fever may become even higher, and the pulse still more rapid and weaker. The patient lies in a condition of muttering delirium, with carphologia. All the symptoms are those of profound exhaustion: the eyes are hollow, the lips and teeth covered with sordes, and the emaciation extreme. The skin becomes dryer, and finally cyanotic, the pupils dilate, and there may be marked anesthesia. Death ordinarily occurs at the end of two or three days after the commencement of this condition. Occasionally the course of the disease is interrupted by intervals in which the patients exhibit more or less lucidity. The disease is related to an asthenic condition known as confusional insanity, which is due to the same causes, but in it the patients exhibit, in place of excitement, depression, with fear of poisoning and positive refusal of all food, mental confusion, disorientation, failure of memory, slight elevation of temperature, or, indeed, a subnormal temperature, and very rapid emaciation. It is most apt to occur in patients previously debilitated. In the paralytic form there is vaso-

motor paralysis with cyanosis, depression, and often stupor. From these the patient passes into an algid state, in which death occurs.

The **differential diagnosis** is frequently difficult. In many infectious diseases, particularly *pneumonia* and *typhoid*, hallucinatory delirium may develop. This, of course, must be suspected in these diseases, and it is advisable, if possible, to examine the blood in all cases of acute delirium by Widal's method. In *acute mania* fever is rare, emaciation is not so rapid, and the mental symptoms are more purely psychical. In *general paralysis*, toward the end maniacal attacks may develop, but the history of the previous existence of the disease, the presence of the Argyll-Robertson pupil, and the absence of fever lead one to suspect the true diagnosis. Finally, in *delirium tremens* the fine tremor of the hands and tongue, and, if possible to obtain it, a history of recent debauch should clear up the diagnosis. The course of the disease is variable; it may vary from three or four days to as many weeks. Those cases are most rapid in which excitation is most profound.

The **prognosis** in acute delirium is most unfavorable, and is more so for men (according to Krafft-Ebing) than for women. Those cases that were previously debilitated, either as a result of chronic alcoholism, or chronic exhaustive diseases, or childbirth, are the most serious. Those that develop suddenly, and from the beginning are very severe, are also nearly always fatal; if there are no lucid intervals, or if those that occur are short and imperfect, the prognosis is graver; and the same is true of those who suffer from obstinate insomnia. In confusional insanity it is better although serious.

The **treatment** is, of course, unsatisfactory. Calomel should be administered in the earlier stages of the disease. At the same time the temperature should be combated by cool baths or packs and an ice-bag should be applied to the head. Sleep should be obtained by the use of chloral, bromid, and the more modern hypnotics, which are to be preferred to morphin. Hyoscin seems to be particularly indicated. In the later stages of the disease stimulants should be administered freely. Excellent results have been obtained (Solivetti) by the hypodermic administration of Bonjean's ergotin. Nutrition must be maintained by forced feeding with milk, eggs, broths, etc.

SENILE DEMENTIA.

This is a condition symptomatic of sclerosis of the cerebral arteries. Some of the manifestations, indeed, are those of general arteriosclerosis. It usually comes on after fifty years of age, and is slightly more common —at least more noticeable—in the male than in the female sex. The first **symptoms** are loss of memory, especially for recent events, failure to keep engagements, and slight querulousness. These steadily progress. The patient forgets not only facts, but words, and the speech may, in consequence, resemble—to a certain extent—some of the manifestations of aphasia. At the same time judgment is impaired; the patient is irri-

table, occasionally ridiculous; becomes suspicious, particularly of his immediate family and friends, and is apt, at times, to become violent toward them. He becomes careless about his person and clothing, spills food while eating; often sleeps during the day, especially after eating, and heavily at night. Finally, the dementia may become complete, and the patient become entirely unable to care for himself. The objective signs are usually the hardened arteries, tremor of the hands and lips, the arcus senilis, the wrinkled, dry skin with prominent veins, and the progressive emaciation. Often the urine is of low specific gravity, and contains a slight amount of albumin. Death usually occurs from some complication, such as cerebral hemorrhage or uremia, or from some intercurrent condition, such as broncho-pneumonia. The **treatment** is that for arteric-sclerosis.

V. DISEASES OF BRAIN AND CORD.

MULTIPLE SCLEROSIS.

(*Insular or Disseminated Sclerosis.*)

Definition.—A disease due to the development of sclerotic patches, occurring in an irregular manner throughout either or both the brain and spinal cord. It is characterized by paresis, intention-tremors, scanning speech, nystagmus, and mental disturbances.

Etiology.—It is not certain that there is a single cause for all cases of multiple sclerosis. Most frequently it follows some infectious condition, particularly the exanthemata, typhoid, malaria, pneumonia, and perhaps influenza and sunstroke. The metallic poisons, as lead, also seem to have an etiological significance. A history of trauma, of exposure, or profound emotional shock is often obtained. Neuropathic heredity often exists. The majority of cases apparently begin between the ages of twenty and thirty, but children may be affected. Sex is not an important factor. The disease is far more common than was formerly believed (Taylor), as the atypical forms are often not recognized.

Pathology.—The sclerotic tissue occurs especially in the white matter, though any part of the cerebrospinal axis may suffer. The cortex is rarely implicated. The spots are usually well circumscribed, gray or grayish-red in color, and on section may be level with, raised from, or depressed beneath the normal line of section according as to whether it is in the early, hypertrophic, or cirrhotic stage. The cranial nerves may be involved at their origin, the first, second, and tenth being particularly vulnerable. The medullary sheath of nerve-fibers in the affected region degenerates early, but the axons are markedly resistant. Since they are not cut off from their trophic center, secondary degeneration is rarely met with. The blood-vessels show more or less proliferation of the adventitia, and endarteritis is not an uncommon condition. Whether this vascular change is primary or secondary is unknown. Microscopically, the sclerotic areas are made up of an overgrowth of neuroglia-cells and fibers and of the ordinary connective tissue. In certain cases these

patches exhibit some tendency to involve special parts of the nervous system, as the lateral or posterior columns.

Symptoms.—These may be described under two headings: first, the general symptoms, or those common to all cases of the disease, and not explicable from the position of the sclerosis; and, secondly, those dependent on the locality of the lesions. The disease is always chronic, and either remissions, or one or more intermissions occur, and in some cases may extend over several years. The first evidence of the disease is *loss of power*, first in one, then in the other, lower extremity. Later, paresis develops in the upper extremity. Sooner or later other general symptoms appear—viz., tremors, nystagmus, scanning speech, increased reflexes, and optic-nerve atrophy. The *tremor* is volitional (*intention-tremor*), and when the patient is at rest no abnormal movement is manifest, as a rule. On attempting to use the hands, or in walking, more or less coarse tremor is observed. This may be well brought out by the finger-to-nose test. The head may be similarly involved, and some incoördination is commonly associated therewith. As the paresis is spastic, the tendon reflexes are increased, and even ankle-clonus and the Babinski phenomenon may be present. The abdominal reflex is often absent. In addition, a certain degree of ataxia or incoördination of motion is present, which is independent of the tremor. Strümpell has studied this especially. The *nystagmus* is sometimes only noticeable when the eyes are moved, but usually it is constant. It is more marked in lateral than in vertical movements. *Speech* is at first slow and drawling, and of a peculiar monotonous character; later it becomes even more deliberate, and is then spoken of as scanning, each syllable being pronounced separately with a slight rising and falling cadence. Optic-nerve atrophy is of frequent occurrence. It begins with pallor of the temporal edges of the discs, a valuable sign (Müller). Other cranial nerves, particularly the motor nerves of the eyes, may be affected, and at times are early symptoms. The sensory disturbances are less important than the motor phenomena. They consist of areas of hyperesthesia, particularly in the extremities, that are usually transient, and occasional tingling or numbness in the limbs. There is usually no wasting of, nor electric change in, the muscles, nor do bed-sores occur. Vertigo is usually present. The patients are usually emotional, and laugh or cry upon slight provocation; often the outbursts of laughter are wholly causeless. In other cases dementia, or even acute maniacal outbursts, are met with, but these are rare. During this stage epileptiform or apoplectiform attacks may occur. The symptoms directly resulting from the local lesions cannot be given in detail. Certain types result, however, that depend upon the tendency of the sclerotic areas to involve certain tracts, and these are—first, a form resembling lateral sclerosis, either bilateral or unilateral (p. 1140), due to implication of the lateral tract; and, secondly, a form similar to locomotor ataxia, in which the posterior columns especially suffer. In some of these cases the general symptoms described above are not very apparent, or only one or two of them may be present. Such are difficult of diagnosis ("formes frustes" of Charcot).

The **diagnosis** is generally easy after the disease has lasted some time. The intention-tremor and the gradual and progressive loss of power, with increased reflexes, scanning speech, and mental deterioration,

are sufficient. The following table gives the differential points between this disease and *paralysis agitans*, *tabes dorsalis*, and *hereditary ataxia*:

Disseminated Sclerosis.	Paralysis Agitans.	Tabes Dorsalis.	Hereditary Ataxia.
Rarely occurs in children. Generally between the twentieth and thirtieth years.	Occurs in persons over forty years of age.	Rarely before the twentieth year.	Usually before the twentieth year. Generally affects several in the same family.
No sensory symptoms, as a rule. Sight may be impaired, the hearing less frequently. The Argyll-Robertson pupil is absent.	No sensory or special-sense symptoms of any importance. Argyll-Robertson pupil is absent.	Fulgurant pains an early symptom. Sight and hearing are commonly affected. Often diplopia and Argyll-Robertson pupil are present.	Sensory symptoms are rarely present. Diplopia and Argyll-Robertson pupil are absent.
Nystagmus is present, as a rule.	No nystagmus.	No nystagmus.	Nystagmus is frequent.
Reflexes are exaggerated; ankle-clonus is present. There may be muscular rigidity.	Reflexes are normal; very rarely they may be plus. Permanent muscular rigidity.	The knee-jerk, ankle-clonus, and rigidity are all absent.	The knee-jerk is lost in the course of the disease; it is rarely increased. No rigidity.
Scanning speech.	Speech is slow and deliberate on commencing a sentence, but soon it becomes hurried.	No speech-defects.	Speech is slow and irregularly scanning.
A tremor is generally present on voluntary movements only. If the tremor occurs during rest, it is fine. Oscillations of the head are frequent; of the trunk, less so.	Tremor when at rest. Voluntary movement may make it cease temporarily. The head may shake, with rather a vertical than an oscillatory movement.	No tremor. Incoördination is marked. No oscillations of the head or trunk. Romberg's symptom is present. Trophic disturbances are common.	Incoördination is present, is increased by closing the eyes. Static ataxia may be noted.
Mental disturbance may occur.	No mental phenomena.	Mental disturbance is rare.	No mental disturbance.
Gait is usually spastic and paretic, and often uncertain.	The face is immobile and mask-like. The gait is propulsion, festination, retropulsion, or lateropulsion.	The gait is stamping in character; the legs are moved stiffly. There is difficulty in urination.	The gait is swaying and irregular, like that of a drunken man. The legs are not kept wide apart as in locomotor ataxia.

The most difficult differential diagnosis is from syphilis of the central nervous system. This is characterized, as a rule, by the more rapid development, the presence either of the Argyll-Robertson pupil or complete pupillary immobility, the absence of the typical group of symptoms, and the response to antisyphilitic treatment. It should not be forgotten that any of the symptoms of disseminated sclerosis may be present in cerebrospinal syphilis, and that cases of the former disease may lack one or more of the fundamental symptoms, and cases will sometimes occur in which the differential diagnosis cannot be made. Examination of the cerebro-

spinal fluid may be of assistance, as in syphilis a marked pleocytosis, an increase of globulin, as shown by Noguchi's butyric acid or other test, and possibly the Wassermann reaction will be found. The latter will also be found in the blood if the patient has not had antisyphilitic treatment within six months, in which event it may not. Arterio-sclerosis may produce multiple areas of softening, causing a symptom group resembling multiple sclerosis. This, however, is more apt to occur in old people, while multiple sclerosis is more common in early middle life. Disseminated myelo-encephalitis (p. 1135) may also be mistaken. In this there may be fever and a history of previous infection.

The **course** usually extends over five to ten or even fifteen years, and death is generally the result of some intercurrent affection, though it may occur during an apoplectiform or convulsive attack. Rarely it is due to failure of the heart or respiration. Remissions of considerable length of time may occur.

The **prognosis** is favorable as far as life is concerned, and some improvement may even occur, but entire recovery cannot be expected.

Treatment.—Silver nitrate, mercury, the iodids, quinin, and arsenic may be tried. Rest and easily assimilable food are of prime importance.

PSEUDOSCLEROSIS AND DIFFUSE SCLEROSIS.

In 1883 Westphal described a case characterized by disturbance of speech, slowness of the movements, decrease of intelligence, increased irritability, apoplectiform attacks, pronounced tremor, spasticity and increased reflexes, slight disturbance of sensation, and no involvement of the sphincters. The autopsy was entirely negative. Since then similar cases have been reported, especially by Strümpell. Later investigations seem to show that these cases are really due to a diffuse sclerosis, such a condition having been found, those cases heretofore characterized as pseudosclerosis being a mild degree of it.[1]

The *diagnosis* cannot be made from multiple sclerosis during life.

Treatment is without avail.

CEREBROSPINAL SYPHILIS.

Syphilis also affects both the brain and spinal cord. The symptoms are detailed on p. 394. (See also Apoplexy, Tabes, and Paresis.)

GENERAL PARALYSIS OF THE INSANE.

(*General Paresis; Parezis; Chronic Diffuse Meningo-encephalitis; Dementia Paralytica.*)

Definition.—A chronic disease involving the cerebrum, spinal cord, and the meninges, and characterized by a gradual loss of power, tremors, and progressive mental decay.

[1] *Jour. Amer. Med. Assoc.*, Nov. 11, 1905.

Pathology.—The intima and adventitia of the blood-vessels undergo proliferative changes, and the perivascular spaces are dilated and contain an excessive quantity of fluid, also cellular elements. Obliterative endarteritis occurs also. Atrophy and degeneration of the cerebrum are met with, chiefly involving the cortex, particularly that of the frontal or parietal regions and the anterior basal region. The ventricles are dilated and the ependyma is granular. The membranes are thickened and opaque, and adherent to the surface of the convolutions, so that the cortex is torn upon their removal. Hemorrhage may take place into the subdural spaces, and may vary in amount from a mere stain to the formation of a pseudo-membrane.

Secondary sclerotic and degenerative changes are found in the posterior and pyramidal tracts of the cord in most cases.

Etiology.—As in locomotor ataxia, a history of syphilitic infection is obtained in a large majority of all cases. Noguchi and Moore have demonstrated the spirochæta pallida in all the layers of the brain cortex except the outer one in 12 cases out of 70 examined.[1] The condition occurs more frequently in men than in women, and usually between the thirtieth and fiftieth years. Business or domestic troubles, and, in fact, great anxiety of mind from any cause, also venereal or alcoholic excesses, serve more or less directly to induce the disease. Trauma and heredity play but a minor part, if any. The white races, Hebrew and Caucasian, seem to be especially predisposed, the yellow and black races less so, although it is occasionally observed among the negroes of America. It is undoubtedly increasing in frequency.

Symptoms.—The **prodromal stage** may last for months. The symptoms are both mental and physical, either of which may appear first and exist alone for some time, or they may be contemporaneous in point of onset. As a rule, some *alteration of the character* and demeanor of the patient is the first evidence of the trouble. The patient suffers from insomnia and is generally restless, as well as incapable of sustained effort. He will be forgetful and perhaps careless where he was formerly careful and attentive. The sexual desire may be excessive. The *ego* will figure prominently in his sayings and doings. Sometimes he is hypochondriacal, sometimes exalted, and feels strong and competent. Among the physical signs are frequent twitchings and *tremors* of the facial muscles, particularly of those about the mouth and the tongue. Tremors of the hand and arm seriously interfere with writing; tremor of the lips and tongue renders the *speech* thick, blurred, and hesitating, and syllables are omitted from words, or even whole words lost from sentences; and the *pupils* are frequently unequal and fail to react to light, but do in convergence and accommodation (Argyll-Robertson pupil), primary optic atrophy may also be present. The tendon reflexes may be either diminished, lost, or exaggerated. These symptoms extend over a variable period, with one or more remissions as a rule, and sometimes with a complete intermission and an apparent cure. Sooner or later, however, the next stage develops.

Stage of Excitement or Depression.—The symptoms of this stage are superadded to those of the first, which by this time have grown gradually more pronounced; *loss of power* usually is already a prominent feature.

[1] *Jour. Amer. Med. Assoc.*, Mar. 29, 1913, 1002.

A state of excitement is most commonly met with, and is characterized by a most remarkable prodigality of thought and speech. The patient believes himself to be possessed of enormous wealth or of great rank and power. Extravagance, unsafe business ventures, and irrational generosity are common. He is boisterous, sleepless, and constantly and actively engaged in pursuing his extravagant ideas. Women are apt to believe themselves pregnant. In many cases, however, this state is characterized by nothing more than a feeling of well-being and satisfaction with one's self and freedom from care and worry when such would be justified. In other cases this delusion of grandeur (*expansive delirium*) is absent and the patient is melancholic with delusions of persecution. This is especially apt to be the case if his physical condition is lowered by some intercurrent disease. Remissions of all these symptoms are not rarely met with. These states may alternate. *Epileptiform* or *apoplectiform attacks* may occur, followed by paralysis in this stage and also as early symptoms. In the large majority of cases the mental decay is progressive, until finally complete dementia is reached; the patient then becomes bedridden, bladder and rectal symptoms develop, and possibly bed-sores. Death results from exhaustion or from some intercurrent disease.

Diagnosis.—This is sometimes difficult in the earliest stages, particularly when the mental phenomena alone exist. The slight change of character and the occasional outbursts of temper or unrestrained jollity may be regarded as mere moods more or less directly dependent upon the daily routine. When mental depression exists it may be mistaken for neurasthenia. When to these symptoms are added the tremor, the defects of speech, the inequality of the pupils, and paresis, the clinical picture gradually assumes definite shape, and ofttimes, long before expansive delirium or melancholia develops, a positive diagnosis is made. The tabetic type of the disease presents many points of resemblance to tabes dorsalis. There are ataxia, loss of knee-jerks, disturbance of micturition, fulgurant pains, visceral anesthesia, and Biernäcki's symptom (absence of tenderness over the ulnar nerve). To these are added tremor of the lips, disturbance of speech, and the peculiar mental symptoms. In the cerebrospinal fluid obtained by lumbar puncture will be found the globulin reaction, an increased number of lymphocytes, and plasma cells, and in many cases the Wassermann reaction with both the blood-serum and cerebrospinal fluid.

Differential Diagnosis.—The diseases with which it is most likely to be confounded are—(1) *Disseminated sclerosis*, (2) *Paralysis agitans*, (3) *Cerebral syphilis*, (4) *Neurasthenia*, (5) *Chronic alcoholism*, (6) *Chronic lead-poisoning with cerebral symptoms*, (7) *Bulbar palsy*, (8) *Chronic mania*, (9) *Dementia from any cause, as senile or terminal dementia.*

(1) In *disseminated sclerosis* the mental symptoms are even less obtrusive in the earlier stages, the first evidence of the disease being paresis in the lower extremities. The tremor, too, is volitional, the speech is scanning, and nystagmus is present. Mental phenomena develop late if at all, and are not expansive in nature.

(2) In *paralysis agitans* there are frequently no mental changes, and in any case they consist of nothing more than dulness. The characteristic attitude and gait; the tremor when at rest, which sometimes ceases on

movement; the speech, which is hesitating at first, then hurried; the high-pitched voice; the absence of pupillary changes—all mark paralysis agitans. Remissions are uncommon.

(3) *Cerebral syphilis* may also simulate paretic dementia. In cerebral syphilis the tremor may or may not be present, but no speech-defect occurs; and attacks of severe headache are frequent and usually severe. Palsies of cranial nerves and complete immobility of the pupils are more liable to be due to syphilis. The condition often passes into dementia, but the peculiar expensive delusions of paresis rarely, if ever, occur. Mott states that in syphilitic pseudo-paresis the lymphocytes in the cerebrospinal fluid disappear under antisyphilitic treatment, but in true paresis they do not. Plasma cells are not present in the fluid in syphilis, and the lymphocytes are usually much greater in number (95–451 per c.mm.) than in true paresis (8–78 per c.mm.).

(4) The characteristic physical symptoms will distinguish the two.

(5 and 6) Both of these poisons may cause symptoms resembling those of paresis. The history and recurrence of hallucinations, which are very uncommon in paresis, will often serve to differentiate the two conditions. Often, however, the diagnosis can only certainly be made when the patient recovers, which he will not do if he has true paresis.

(7) The absence of mental symptoms and the occurrence of atrophy of the tongue, paralysis of the vocal cords, etc. (p. 1129), distinguishes *bulbar palsy*.

(8) Patients with *chronic mania* do not have the peculiar physical symptoms of paresis, and there will usually be a history of a previous attack of acute mania. Spells of acute excitement may, however, occur during the course of paresis.

The presence of an increase of the lymphocytes in the cerebral spinal fluid and the Wassermann test or some of its modifications will distinguish paresis from any of the above, excepting syphilis (*vide supra*). There is also a possibility that a patient having alcoholic pseudo-paresis may have had syphilis, which might render these tests fallacious.

The **prognosis** is gloomy and recovery never occurs. The tendency is toward a fatal termination in from two to three years. In rare cases the progress may be slow or remissions may delay the termination for several years longer.

Treatment.—Drugs are of no value in a curative sense, except in those cases that are due to syphilis, when mercury and the iodids must be pushed. The use of salvarsan ("606") may be of service in incipient cases, if none of the contra-indications to its use are present, otherwise it has been of very little value. Bromids, morphin, chloral, or, still better, sulfonal, trional, or hyoscin, may be used in combating the insomnia and attacks of delirium. These cases cannot be properly cared for at home; indeed, their removal to an asylum is generally imperative. The tendency to bed-sores must not be forgotten, and continuous rest in bed must, therefore, be postponed as long as possible.

VI. GENERAL AND FUNCTIONAL DISEASES.

INFANTILE CONVULSIONS.

(*Eclampsia Infantilis.*)

UNDER this term are grouped a number of conditions, with convulsive attacks as the common symptoms.

The **causes** are: 1. Organic brain lesions (pp. 1163 and 1183). 2. Neuropathic tendency, that is manifested later as hysteria or epilepsy. 3. Emotional disturbances, as fright. 4. Rickets, in about 30 per cent. of all cases. 5. Acute infectious disease, especially as an initial symptom of pneumonia, and more rarely of scarlet fever, small-pox, and pernicious malarial infection. 6. Inflammation of the serous membranes, as meningitis, where the relation is direct, or pleuritis or peritonitis. 7. Kidney disease, in which they are uremic. 8. Peripheral irritation; dentition has long been supposed to be a chief factor in their causation, but it is now believed that the chief cause is the presence of rickets. Intestinal parasites have also been found, particularly the ascaris lumbricoides, and the convulsions have ceased after their expulsion. 9. Debility, especially that resulting from gastro-intestinal disorders.

Pathology.—The pathologic changes may be divided into two groups: (1) those bearing an etiologic relation to the convulsive attacks, and (2) those that are merely consecutive. Among the former are meningeal bleeding, tumor, gliosis (either hypertrophic or atrophic), and hydrocephalus. Then there are general conditions that seem to predispose to this condition or, at any rate, are frequently associated with it, such as rachitis. The consecutive lesions are hemorrhages into the meninges or into the substance of the brain and the spinal cord, an increase in the amount of cerebro-spinal fluid, and congestion of the pia or the substance of the brain.

The **symptoms** of the attack vary according to its intensity. In the most severe form they resemble in all respects those of an epileptic seizure. At first the eyes deviate upward or to one or the other side, and the gaze becomes fixed and staring; next there are *twitchings of the muscles* of the face, sometimes slight and limited to one side, and sometimes general, often involving the muscles of mastication and giving rise to trismus or gnashing of the teeth. Next there are *tetanic contractions* of the extremities, the fingers being strongly flexed, the hands flexed upon the arms, and the feet in the position of pes equinus or sometimes in the dorsal flexion, and both legs and arms rigidly extended. Often the muscles of the trunk are involved, and there is either opisthotonos or respiratory cramp, with excessive hardness of the abdominal muscles. This rigid condition is interrupted at brief intervals by sudden twitchings, or occasionally the convulsion becomes clonic instead of tonic, and there are repeated extensions and contractions of the extremities, shaking of the head, and quivering of the whole body. As a result of the respiratory cramp, *cyanosis* rapidly develops and may reach an extreme degree. The forced respirations give rise to a foam that collects about the lips, and is often mixed with blood from the bitten tongue. Urine is often, and feces occasionally, passed involuntarily. In nearly all cases unconsciousness is

complete. Many of the slight attacks are accompanied by a cry or by an attack of screaming. The tetanic state usually lasts for a minute or two; then there are a few clonic movements, relaxation becoming rapidly complete, and the spasm is ended by a few deep respirations. The child may return to consciousness, although it is usually drowsy or stupid, or it may pass into a deep sleep from which it cannot be aroused. Often in the latter condition attacks will recur at irregular intervals, and sometimes a single attack may continue for some time, although from time to time there are slight twitchings followed by partial relaxation (*status eclampticus*—Lewis). The attack may come on suddenly, or, as is more frequently the case, it may be preceded by a period of restlessness and irritability. A milder form of the spasm consists of sudden fixation of the eyes, slight twitching of the body, and a peculiar dusky pallor that passes away in a few moments. In other rare cases consciousness may persist, although the patient is aphasic. Laryngismus stridulus is an analogous condition (*vide* Diseases of the Larynx, p. 517).

The **diagnosis** of the condition is very easy. The recognition of the cause, however, is very important and often difficult. Every case should be first examined for rickets, and then the gums should be investigated; also the condition of the child's nutrition and the presence of symptoms of gastric or enteric irritations. If fever exists, it is important to discover its cause. The character of the convulsion is often of value in distinguishing between the idiopathic or reflex type and that due to organic brain-disease. Convulsions beginning immediately after birth, or an injury, either persisting or else disappearing gradually, are probably caused by meningeal hemorrhage (p. 1173). An attack of a Jacksonian type would, of course, indicate the presence of a focal lesion; and if this be a tumor, there will probably be bulging of the anterior fontanel, severe headache, and the ophthalmoscope will reveal a neuro-retinitis. If, after the attack, pareses or paralyses are present, a focal lesion is still more likely. Hydrocephalus is usually recognized with ease. Some cases exist, however, in which it is impossible to discover any adequate cause.

The **prognosis** varies according to the etiology. In cases with organic brain-disease it is unfavorable as regards cure. In those forms that precede epilepsy or functional nervous diseases the spasms usually disappear after the first dentition, and the patients appear to have recovered for a time. In those, however, in whom the symptoms are due to some peripheral irritation or to rachitis, the outlook is fair, although even these now and then develop into permanent epilepsy. The convulsions themselves are either often immediately fatal, or so exhausting to the patient that he succumbs readily to the disease that produced them. In these cases the prognosis depends upon the frequency and severity of the attacks, death usually terminating those in which the status eclampticus has been established. The prognosis for ultimate cure depends also in part upon the length of time that the condition has existed; if but for a short time before an arrest has been established, recurrence is much less likely. Gowers, however, says that even after a year's duration permanent cure may sometimes be obtained.

The **treatment** naturally falls into two parts—that of the attack and that of the interval. Unquestionably, the most efficacious antispasmodic

that we possess for this condition is chloroform. A few drops may be put upon a handkerchief and held carefully over the nose and mouth of the little patient. A very small quantity usually suffices, and the effect is almost instantaneous. In addition to this, chloral and the bromids may be given by the rectum, and it is often useful to add to these one of the coal-tar antipyretics, particularly antipyrin. Formerly hot mustard-baths were much in favor, but unless they do good at once they are not likely to be of any use. In a very obstinate case under my care they were absolutely valueless, and were replaced by momentary immersion in ice-cold baths and vigorous friction, which seemed to act very favorably. If any known source of irritation is present, as an overloaded stomach, it should be relieved at once, if possible, by the stomach-tube or an emetic. An enteritis may be temporarily benefited by an enema or by a moderate dose of calomel. The treatment during the interval depends upon the nature of the cause. If rachitis exists, it should be treated according to the principles laid down in my discussion of this disease. If dentition is suspected, the gums may be lanced, but this should only be done when they present distinct signs of irritation. Gastro-intestinal disorders of any kind should be relieved as soon as possible, and intestinal parasites must be expelled. In infectious diseases the convulsions usually disappear after the initial stages, and require no further attention. In organic brain-disease, providing it be not syphilitic in nature, very little can be done. Finally, in those cases in which no cause can be discovered bromids are the only resource, and should be given in sufficient doses: from gr. iij–v (0.194 to 0.324) per day to children of six months, and from gr. v to x (0.324 to 0.648) to those between six and sixteen months.

EPILEPSY.

Definition.—A condition characterized by attacks of unconsciousness, with or without convulsions. We are scarcely justified in speaking of epilepsy as a disease. It seems, in reality, to be a symptom, though in many cases (the so-called idiopathic cases) we do not know the underlying cause. The type of cases in which the unconscious period is very brief (momentary), with no convulsion following or at most but a slight rigidity, is termed *petit mal*. The more pronounced type, with prolonged unconsciousness and severe general convulsions, constitutes *grand mal*. That form first described by Hughlings Jackson in which the convulsion is localized, and in which unconsciousness may or may not occur, is called *Jacksonian*, *focal*, or *cortical epilepsy*.

Pathology.—Epilepsy is a symptom, and the inevitable question must be, "Of what?" In certain cases this can be answered (in the organic cases), since the lesion is demonstrable; but in others (functional or idiopathic) there is no demonstrable lesion. Among the causes of the former are brain-tumors, meningitis, traumatism inflicted either at birth or subsequently, atrophy and sclerosis, vascular disturbances, syphilis, and toxemia, both autogenous and exogenous. Peripheral lesions too

may give rise to it. Little can be said about the idiopathic variety, we can only enumerate causes; we do not know in any case how these act, and we do not know the ultimate pathology. Many writers apply the name "epilepsy" only to the idiopathic form, while others include all apyretic affections characterized by the occurrence of fits, whether of centric or peripheral origin. Brown-Séquard believes that the distinction between the various kinds of convulsions is artificial, and that the correct classification should be based on the knowledge of the cause.

Etiology.—The causes are (1) *predisposing*, (2) *exciting* or *determining*. Among the former, which refer particularly to the idiopathic form, are—

(*a*) *Age.*—The following tables show the early onset in a large majority of cases analyzed by Gowers, Hesse, and Osler:

Age at onset.	Proportion affected. Observers. Gowers	Hesse	Per cent.
Before 10	422	393	33.3
From 11 to 20	665	364	42.
" 21 " 30	224	111	13.7
" 31 " 40	87	59	6.
" 41 " 50	31	51	3.4
" 51 " 60	16	13	1.2
" 61 " 70	4	4	0.3
71	1	0	
Total,	1450	995	

Age at onset (Osler).	Number.	Age at onset (Osler).	Number.
1	74	9	17
2	62	10	27
3	51	11	17
4	24	12	18
5	17	13	15
6	18	14	21
7	19	15	34
8	23	Total,	437

(*b*) *Heredity.*—Family neuroses, such as insanity, hysteria, chorea, etc., are common, but it is decidedly more the exception than the rule to find either parent epileptic.

(*c*) *Alcohol.*—The causal relationship between an abuse of alcohol by the parents and epilepsy seems rather pronounced. Féré says that of 594 epileptics examined by him, 258 had parents who were hard drinkers. Echeverria refers to 572, 257 of which he believed could be traced directly to the abuse of alcohol.

(*d*) *Syphilis* does not predispose. When it gives rise to changes in the brain and cord, which in turn cause epilepsy, it is in reality a determining cause.

(*e*) *Eye-strain* is no longer regarded seriously as a predisposing influence.

The **exciting** or **determining causes** are traumatism to the head with or without actual lesion of the skull or brain, mental shock, various morbid conditions of the membranes of the brain or of the brain proper (*e. g.*, after hemiplegia), or peripheral irritation (dentition, worms, a cicatrix, an adherent prepuce, etc.). Not a few cases are dependent upon toxic substances in the blood, as in uremia and lead-poisoning. Excessive indul-

gence in alcohol or over-eating often precipitates the attack. Great emotion and nervous shock (fright) seem to be exciting causes in some cases. There are cases of bradycardia in which epileptiform attacks occur (Stokes-Adams disease). And cerebral arteriosclerosis may cause the epileptiform attacks that occur in old people.

Symptoms.—Petit Mal.—In this condition the majority of cases belong to the following type: The attack begins suddenly; perhaps while talking to the patient his expression suddenly becomes blank, the face pales, the pupils dilate, and he is evidently not conscious. In a moment or two he gathers his scattered senses and picks up the thread of the conversation. Very often he is not cognizant of any lapse of time or has but a vague idea that something has occurred. If carefully observed, fine clonic movements may be detected in many cases, it may be of the facial muscles or of the hands. Convulsions never occur, the dominant feature being the unconsciousness. On regaining consciousness the patient may act strangely and appear dazed; it is seldom, however, that he falls in attacks of this kind. Occasionally a peculiar dreamy state takes the place of an ordinary attack, or the individual may be the victim of imperative ideas. Falret has described a condition (*epilepsie larvé*) known as masked epilepsy, in which maniacal outbursts or explosions of passion occur.

Grand Mal or Haut Mal.—In many cases some subjective symptom precedes the actual attack. In its most specialized form it is termed an *aura*, and includes any phenomenon, motor and sensory, that ushers in an attack. While the aura differs in different cases, it is almost invariably constant in the same case, so that one will have a subjective sensation of sound, another of light, either flashes or colors, etc. There are other signs that occasionally antedate an attack, and which may or may not precede each attack (headache, drowsiness, change of disposition, palpitation, perverted appetite, sexual or other, etc.). Many attacks begin precipitately with absolutely no previous warning. In such cases the patient may or may not utter a piercing sound (*epileptic cry*), falling at the same time, no matter where or in what position he may be. Hence the danger to which epileptics are always subjected. A peculiar onset occurs in the so-called "*procursive epilepsy*," in which the patient suddenly starts off and runs some distance before the paroxysm begins.

Paroxysmal Period.—In many cases, whether preceded by an aura or not, this stage is ushered in by a *spasm* that is tonic in character. The patient falls, perhaps because of the loss of consciousness, though in those cases in which he drops precipitately he is probably thrown by the violence of the spasm. The head is usually extended, the muscles of the larynx and trunk contracted, and hence the epileptic cry and the dyspnea, while the lower limbs are generally extended, the upper semiflexed, and the fingers tightly clenched. This period of rigidity lasts but a few seconds before *clonic convulsions* appear.

Intercurrent contractions vary in different cases from very mild movements to those so severe as to toss the individual about. The face, pale at first, becomes congested, and the jaw works in churning the saliva into a froth; this is blood-tinged when the tongue is bitten. The respiration is jerky, gasping, and there may be a loss of control of the bladder and bowels. In idiopathic cases this stage lasts from one to five or six min-

utes. The spasms gradually diminish, and without regaining consciousness the patient passes into a deep sleep, immediately preceded in some cases, however, by coma in which the breathing is stertorous. During the sleep, which lasts about an hour, the patient is completely relaxed. On waking he usually appears confused and complains of feeling tired. His limbs may ache for several days.

Occasionally attacks follow one another in quick succession, with no period of consciousness intervening (*status epilepticus*)—a very dangerous condition.

Post-epileptic phenomena are variable. The patient may become maniacal, homicidal, or may simply be mentally deficient for a few days, with perhaps some slight speech-disturbance. A condition known as epileptic automatism may follow or take the place of the convulsion. In this state the patient may go about, converse, and perform apparently purposive acts of which he has no recollection afterward. In the course of time every epileptic's brain-power deteriorates. Paralysis sometimes occurs, is usually transient, and may be unilateral or bilateral.

Nocturnal Epilepsy.—In this condition the attacks occur at night, and may be entirely unknown either to the patient or his friends. He complains from time to time of feeling tired on rising in the morning, his limbs and head ache, and he is generally duller than usual; he may even be confused. Such a history is suggestive, and the suspicion is strengthened if in addition he has urinated involuntarily or if blood-spots are found on his pillow.

Jacksonian epilepsy is characterized by spasm that is generally local in character; in fact, it is always so in the beginning, though occasionally it may spread and become general. Consciousness is preserved in the milder forms. Tingling or other subjective sensations may precede an attack. They are usually due to some irritation of the motor cortex (tumors, meningitis, softening, trauma, etc.). Subcortical lesions and certain toxemic conditions can also give rise to it.

Myoclonus epilepsy is characterized by epileptic seizures of the ordinary type, while in the interval between the attacks the patient suffers from clonic spasms of various muscles. The spasms vary in intensity from fibrillary tremors (myokymia) to violent spasms of the large muscles (myoclonus, p. 1214). It may be a family disease.[1]

Diagnosis.—When a definite history is obtainable the difficulty of the diagnosis is less, particularly if an aura occurs. The attack can be frequently diagnosed from other epileptoid conditions at the time by the explosive onset, the brief tonic and somewhat longer clonic spasm, profound unconsciousness followed by a deep sleep, and when these are present by an involuntary passage of urine, frothing at the mouth, and biting of the tongue.

Differential Diagnosis.—In *uremia* the state of the urine (catheterize if necessary), and often the odor, serve to differentiate it. It may be impossible to detect fraud, so perfectly is the disease simulated by those anxious to excite pity, judicial or otherwise, or by those whose accomplices rifle the pockets of sympathetic bystanders. Hysteria may also resemble it very closely. Gowers has tabulated the chief differences as follows:

[1] Clark, *Rev. Neurol. and Psychit.*, July, 1907, p. 532.

	Epilepsy.	Hysteroid.
Apparent cause	None.	Emotion.
Warning	Any, but especially unilateral or epigastric aura.	Palpitation, malaise, choking, bilateral foot-aura.
Onset	Always sudden.	Often gradual.
Scream	At onset.	During course.
Convulsion	Rigidity followed by "jerking;" rarely rigidity alone.	Rigidity or "struggling," throwing about of limbs or head, arching of back.
Pupils	Dilated and immobile.	Mobile and active.
Biting	Tongue.	Lips, hands, or other people or things. Very rare.
Micturition	Frequent.	Never.
Defecation	Occasional.	Never.
Talking	Never.	Frequent.
Duration	A few minutes.	More than ten minutes, often much longer.
Restraint necessary	To prevent accident.	To control violence.
Termination	Spontaneous.	Spontaneous or induced (water, etc.).

Epileptiform seizures may occur in the course of multiple sclerosis, paresis, and dementia præcox.

Prognosis.—Idiopathic epilepsy very rarely is cured. In most cases it will be found that an apparent recovery is merely a prolonged intermission. Cases that are evidently symptomatic are sometimes curable if the cause can be removed. Death is seldom due directly to an attack. Fatal accidents may, however, be caused by an attack.

Treatment.—When an aura occurs, advantage may indirectly be taken of it to aid in aborting the attack. The only efficient remedy is nitrite of amyl inhaled as in angina pectoris. In Jacksonian epilepsy, constriction of the limb in which the aura occurs may sometimes be sufficient. Salt, a popular remedy, is useless. Every effort should be made to lessen the liability of danger to the patient—first from falling, and secondly, from the violence of the spasms. One may at times be justified in using ether or chloroform by inhalation to control the severity of the convulsions. After loosening the clothing, and putting a cork or something between the teeth to prevent biting the tongue, nothing more can be done at the time. Between the attacks special care should be taken to put the system in good condition, and all sources of worry and irritation should be removed so far as possible. Particular attention should be given to the stomach and bowels and the removal of all sources of reflex irritations, as eye-strain, adenoids, intranasal obstructions, bad teeth, adherent prepuce, etc. The food should be light and easily digestible, and systematic colonic flushing is often advantageous.

As to *medicinal measures*, the bromids are of the greatest value. The sodium and potassium salts are most commonly employed, the former, as a rule, being better borne by the stomach. They may be given in milk or in one of the medicated waters. Strontium bromid has been used rather extensively of late, and has yielded excellent results. While idiosyncrasies are met with, it may generally be given in from 10- to 20-gr. doses (0.972–1.944) three or four times a day, and preferably after meals. Each case must be treated according to its special indications. Symptoms of bromism (acne, sore throat, drowsiness, and gastric disturbance) should be carefully guarded against. Should they develop, the

dose of bromid must be reduced, and Fowler's solution administered for a few days. H. C. Wood recommends that the latter should be given continuously with the bromids, thereby preventing or, at all events, lessening the liability to bromism. Other remedies sometimes employed are nitroglycerin (hypodermically), cannabis indica, silver nitrate, zinc, borax, solanum or horse-nettle, chloral, antipyrin, veronal, and chloretone. Surgical measures occasionally yield good results, this being particularly true in focal epilepsy—*i. e.*, when the cortical centers are the seat of an irritating lesion, as a tumor or depressed fracture which can be removed. Even in these cases, if the convulsions have continued for two years or over, the outlook is not good. In idiopathic epilepsy removal of the motor cortex has been tried in those cases in which an aura suggested a local origin—*e. g.*, in a center for a particular group of muscles. The results have been discouraging, in all cases the attacks recurring with increased severity after an interval of remission. It is a curious fact that almost any surgical operation will diminish or check the epileptic attacks for a time, and I have known as simple a procedure as venesection to afford complete relief in a severe case for several months. The results ascribed to various operations may be explained in large part by this fact.

MIGRAINE.

(*Hemicrania; Sick Headache.*)

Definition.—A neurosis characterized by severe attacks of headache, often paroxysmal and more or less periodic, with disturbances of vision and with or without nausea and vomiting.

Etiology.—The condition is frequently hereditary, and in the large majority of the cases that I have seen it has been transmitted by or through the mother. It usually appears early in life. Various other neuroses are common in families subject to this condition. Females are more frequently affected than males, and migraine seems to be associated with diseases peculiar to women, especially menstrual disorders. Among the exciting causes may be mentioned gastric disturbances, dental irritation, naso-pharyngeal diseases (adenoids, etc.), eye-strain, grief, emotion—in short, anything that tends to lower the physical or mental tone occurring in those hereditarily predisposed. Recently attention has been called to auto-intoxication (leucomainic poisoning) as a cause of certain cases. A gouty diathesis seems to be operative in many cases.

Pathology.—This is profoundly obscure, since no lesion has ever been discovered. By some it is thought to be a vasomotor disturbance, and the transient paralytic symptoms that may occur are believed to be due to arterial spasm. Very rarely the disease has been observed in some subjects to replace an attack of epilepsy or even to alternate with true epileptic attacks.

Symptoms.—As a rule, the patient can prognosticate an attack. In the cases of slow onset he may feel indisposed for some hours before, being languid, drowsy, with general discomfort and perhaps nausea. In other cases various *subjective sensations* occur, lasting from a few minutes to several hours. Of these, disturbances of vision are most common, such as flashes of light, spectra, visions of animals or weird forms, or scotoma, etc. Lateral homonymous hemianopsia has also been observed. Audi-

tory sensations are rare, as are those of the other special senses. Transient palsies and aphasia also may appear, the latter occurring when the pain is on the right side. The palsies are often hemiplegic, being present on the side opposite to that in which the pain is. Numbness and tingling may also be symptoms. Complete oculomotor palsy, lasting several days, may occur. After these phenomena have existed for some time *headache* supervenes, when, as a rule, they cease. The pain, at least in the beginning, is usually unilateral, as the name suggests, though later it may and often does involve the entire cranium, spreading from a single point of origin—over one eye, for instance. The affected region may be tender to the touch or it may be the seat of numbness or tingling. *Nausea* and *vomiting* are common symptoms, with or without vertigo. Vasomotor symptoms are frequent, usually the face is pale (angiospastic type), but it may be flushed (angioparalytic type). A brief period of unconsciousness occurs in some cases, and spasmodic movements may also be observed occasionally. This fact is of particular interest, since it serves to support the view that migraine is in some way related to epilepsy, and, as has been stated, attacks of migraine and epilepsy may alternate. Unlike epilepsy, migraine does not tend to impair the mental faculties, no matter how long the patient has been afflicted. During an attack, however, he may have melancholia or be incapacitated mentally and physically for two or three days.

Course.—The disease generally begins in early life, and in nearly half of the cases before the fifteenth year, recurring with a certain degree of periodicity until old age, when it often passes away. It may cease in women at the menopause, and in men between the fortieth and fiftieth years.

The **prognosis** is good as far as life is concerned. This disease is incurable, though the condition of the patient may be alleviated.

Treatment.—The management of the disease may be considered under two heads: (1) treatment of the attack, and (2) the treatment between the attacks, which necessarily includes prevention. The patient should be put to bed in a slightly darkened room, and all sources of noise and confusion should be removed as far as possible. The attack may be so severe as to justify the use of morphin hypodermically. The coal-tar derivatives have met with most favor, however, as remedial measures, and preferably antipyrin and phenacetin, though their occasional depressing effect should be borne in mind. Caffein may be given with phenacetin with advantage, and aromatic spirits of ammonia is a useful adjuvant to antipyrin.

Acetanilid may often be substituted for phenacetin with apparent advantage. In twenty-four hours this may be discontinued, and potassium bromid should be given in liq. ammon. acetatis in doses of gr. x to xv (0.648–0.972). Local applications of menthol, or fly-blisters may be employed, or even superficial points may be made with the actual cautery. The rapidly interrupted faradic current applied with a dry wire brush over the painful side of the head may give temporary relief. Washing out the lower bowel has been recommended.

In the angio-spastic type full doses of nitroglycerin may be of service.

Between the attacks the general health should be carefully looked after. The so-called uric-acid diathesis is common in subjects of migraine.

Haig staunchly advocates the use of salicylates in such cases in addition to the dietetic measures. Anemia should be treated by iron in some form, dialyzed or Blaud's pill. The bowels should be kept normal by means of some saline (Hunyadi, etc.), or by the fluid extract of cascara sagrada. An examination of the stomach-contents often shows the existence of anacidity; when this is corrected there is usually marked improvement in the migraine. The extract of cannabis indica is employed by some over a long period of time, just as the bromids are in epilepsy. It is given in doses of gr. $\frac{1}{4}$ to $\frac{1}{2}$ (0.0162–0.0324), two or three times a day, after meals. While exercise and fresh air are admirable adjuvants to any form of treatment, it must not be forgotten that fatigue invites an attack. Proper rest, care and regularity in dieting, and the avoidance of excitement are the chief points to be observed.

ACUTE CHOREA.

(*Sydenham's Chorea; St. Vitus' or St. Anthony's Dance.*)

Definition.—The type of chorea described by Sydenham is a more or less acute disease, due to an infectious agent or its toxin. It has a special predilection for children, and is characterized by involuntary, irregular, non-purposive muscular movements, more or less marked mental change, by a great liability to endocarditis and a tendency to recurrence, particularly during the spring and fall. It has a close relation to acute articular rheumatism.

Pathology.—No definite lesion can be ascribed to the disease, though, as is usual in such cases, a great variety of lesions have been described. Chief among these is the embolic theory, advanced by Kirkes and subscribed to by Bastian and Hughlings Jackson. Since emboli are not found in every case, however, they cannot be the sole cause. The disease is, however, probably due to the action of a bacterial poison upon the brain cortex. In about 25 per cent. of the cases there is an association of chorea and acute articular rheumatism.

Etiology.—(1) *Age.*—By far the greatest number of cases occur before the twentieth year, but it may develop at any age. Most cases occur between the tenth and fifteenth years.

Sex.—Females are most frequently attacked, and probably in two-thirds of all cases.

Race.—Acute chorea is rarely met with except among the white races.

Heredity.—A history of other neuroses (hysteria, epilepsy, etc.) in the ancestors may be obtained, and an unstable nervous system obtained from such stock may predispose to the disease. Children who develop chorea are especially apt to be of a nervous, excitable temperament, and may be hysterical as well as choreic. It is in cases of this type particularly that *fright* acts as an exciting cause.

Infectious Diseases.—There seems to be a relationship between some cases of chorea and other infectious diseases. This, however, is only at all marked in acute articular rheumatism and scarlatina. It is not yet proved, however, as has been claimed, that chorea and rheumatism are due to the same cause.

Pregnancy is a frequent cause of chorea in adult life. When so caused, it is apt to be severe. It is most prone to develop during the earlier months, and especially in primiparæ. It often assumes the maniacal type.

The influence of *reflex irritation* is probably much overrated, whether intestinal, genital, or from ocular defects, but these may aggravate the disease.

Symptoms.—The common or ordinary form is frequently met with, especially during the spring and fall. Primary attacks may occur at any time, but recurrent outbreaks are most prone to develop in the spring. There is a slight affection manifested by restlessness, disturbed rest at night, and by irregular and purposeless *muscular movements*, that are most marked in, or entirely confined to, the upper extremities, the head, or the facial muscles; or the condition may be unilateral. They usually cease during sleep. *Endocarditis* may not give rise to characteristic signs. More or less *muscular weakness* is present and the patient tires very readily. The child's *disposition* is changed, outbursts of temper being quite common on the slightest provocation. *Fever* is absent in this form unless complications exist, though when severe joint-troubles or endocarditis are present, the temperature will rise. *Anemia* is often present, and with it headaches, irritable heart, and hemic murmurs. Indeed, in some instances not even a murmur can be heard, though *postmortem* records of cases with a history of chorea show that in most of them the valves are affected. The mitral valve is most commonly involved, the endocarditis usually being of the verrucose type. There are rarely any sensory symptoms, severe pain, at all events, being extremely rare. Little dependence can be placed on complaints of tingling or burning pain unless they are voluntary, for the mental make-up of choreic patients is such that they are apt to dwell upon slight ailments suggested to them through leading questions. The reflexes do not differ from those of normal children.

There is a more severe type of chorea, in which the movements are extremely violent, causing severe injuries, even fractures, and leading in some cases to death from exhaustion. The *psychic symptoms* are often marked, and speech may be impaired to such an extent as to render articulation unintelligible. Such a condition may occur in the first, but it is probably met with more often after one or more mild attacks. The gravest form of chorea is *chorea insaniens*, in which the movements are violent and constant. Speech is much affected, insomnia is marked, and fever and maniacal delirium develop, followed in some cases by exhaustion and death. Various forms of skin eruptions may be seen.

The **course** is from six to twelve weeks, though the most trifling cases may recover in a month or less. Others persist six months or even more. In about two-fifths of all cases there is more than one attack, while Gowers has reported one case in which there were nine recurrences. In one of my own cases two attacks occurred annually—spring and autumn—for two years. A fatal issue is very rare in children, and absolute recovery is the rule. The maniacal form, which usually develops in adults, especially in pregnant women, as previously stated, is more often fatal, though recovery is also the rule in such cases.

Diagnosis.—As a rule, this is quite simple. The age of the patient, the mode of onset, and the character of the movements suffice to prevent

mistakes. It may be mistaken for hereditary chorea (p. 1213), and for the various spasmodic movements due to hysteria (p. 1228), tics (p. 1216), and myoclonus (p. 1214), and attention has been called (p. 1174) to the possibility of mistaking athetosis and other spasmodic movements associated sometimes with cerebral palsies for it. Tremor from any cause is characterized by the rhythmic, regular character of the movements.

Treatment.—This is largely hygienic—a fact that must be strongly dwelt upon in discussing with the parent the management of the patient. The avoidance of all forms of nerve-strain is of the utmost importance, and the amount of school-work and home-study should be carefully inquired into, and all excess absolutely prohibited. In the milder forms rest in bed is not imperative, but active exercise must be forbidden, since it invites cardiac troubles, the tendency to heart-involvement already being great in chorea. In the more severe forms rest in bed is a *sine quâ non*. In any case an abundance of rest is called for, and when insomnia is present it should be promptly handled. An important element of the treatment that is sometimes indispensable is the change of environment, associated with rest. As a rule, the patients do best in a warm climate and at the seashore. In severe cases the bromids should first be tried internally, and a warm bath administered just before bedtime. Hyoscin hydrobromate may also prove useful to lessen the severity of the movements. In other cases morphin or chloral may be required, though it must not be forgotten that the latter is a cardiac depressant; veronal or trional should, therefore, be given the preference. The bowels must be regulated and the diet should be light and wholesome, with an abundance of fruit and fresh vegetables. Cod-liver oil is usually indicated. When anemia is present, it is to be met by the use of some one of the preparations of iron. Should reflex irritation be found to exist, it should be corrected at once. Of the therapeutics of the disease not much can be said. We have no specific, but the two most useful drugs are arsenic and cimicifuga, the former as Fowler's solution and the latter as the fluidextract. Fowler's solution should be given in 3- to 5-drop doses three times a day for a few days, and then increased 1 drop *per diem* until the point of tolerance is reached or the physiologic action is manifest. The remedy is now to be continued, but in reduced dosage. The late Hiram Corson first warmly recommended cimicifuga in the treatment of chorea, and I have found the combined use of this drug and arsenic to be more prompt and efficient than the latter alone, particularly in protracted cases. If rheumatic symptoms appear, the salicylates should be administered in full doses, aspirin being particularly well taken. Between the attacks of chorea, syrup of the iodid of iron may be given with advantage.

HUNTINGDON'S CHOREA.

(*Chronic Progressive Chorea; Hereditary Chorea.*)

Definition.—An hereditary disease affecting many members of a family, developing in early adult life, and characterized by irregular muscular contractions, incoördination, and progressive dementia. The disease was first definitely described by Huntingdon in 1872, but other

writers had already alluded to it. Sporadic cases are reported from time to time.

Etiology.—The disease is strictly hereditary, and has been traced through five generations. The offspring of parents that escape are forever immune. It occasionally alternates with idiocy, epilepsy, and various degenerative conditions. It appears to be endemic in certain localities, and still exists upon the southern shore of Long Island, where Huntingdon first observed it.

Pathology.—The changes found consist of chronic pachy- and leptomeningitis, chronic hemorrhagic encephalitis, characterized by round-cell infiltration of the cortex, degeneration of the ganglion-cells, proliferation of the neuroglia, sclerosis of the blood-vessels with dilatation of the perivascular and lymph-spaces, and numerous hemorrhagic foci (Facklam). There are also atrophy and thinning of the cortex, slight irregular changes in the spinal cord, and multiplication of the nuclei in the muscles.

Symptomatology.—The *choreiform movements* commence insidiously and only in the rarest cases become pronounced. They are usually susceptible to voluntary inhibition and disappear during sleep. They usually appear first in one extremity and then gradually invade the other parts of the body. There is considerable *incoördination* of movement. The *mental symptoms* consist of progressive dementia, irritability often attaining maniacal violence, delusions of persecution, and rapid diminution of intelligence. In some cases the mental changes are very slight.

The **course** is steadily progressive, but the patient may live to an advanced age.

Diagnosis.—The only condition likely to cause confusion is senile chorea with dementia. In this the mental symptoms are usually slight and the motor symptoms more violent. The family character of the disease is also lacking. By many, however, this is supposed to be a type of Huntingdon's chorea.

Treatment is entirely symptomatic.

PARAMYOCLONUS MULTIPLEX.

(*Myoclonus Multiplex.*)

Definition.—This is a disease of unknown pathology, first described by Friedreich, and, as its name implies, characterized by clonic contractions in various groups of muscles.

Its **etiology** is obscure. Heredity unquestionably plays an important part, nearly all the patients having among their ancestry cases of one or more forms of nervous disease. Emotional disturbance, as fright, is often the exciting cause. It usually develops in early adult life, and is probably more common in males. In one case that I observed it was associated with idiocy.

The **symptoms** of the disease consist of *clonic contractions* of individual muscles, which cause either no or very little movement of the parts controlled by them, occurring chiefly in the muscles of the extrem-

ities and the trunk, and only occasionally involving the muscles of the face. These contractions are very sudden; so much so that they have been described as lightning-like. Voluntary movement diminishes them somewhat, emotional disturbance increases them considerably, while during sleep they disappear. The power of the muscles, their size and nutrition, remain unimpaired. The *electric reactions* are normal, but electric stimuli and any cutaneous irritation are apt to precipitate an attack. The *tendon-reflexes* are increased. Sometimes the patient gives vent to a peculiar *grunt*, which is probably due to involvement of the larynx and diaphragm. In some of the cases sensitive points have been found over the spinal column, and not a few have presented other stigmata of hysteria.

Varieties of this disease are myokymia or persistent quivering of the muscles (fibrillary chorea of Morvan), and fibrillary or fascicular twitchings (myoclonus fibrillaris multiplex of Kny). In these cases organic disease of the peripheral motor neuron, tuberculosis, and neurasthenia must be excluded (pp. 1142, 1144, 1235). All of these symptoms may coexist in the same case.

Pathology.—Friedreich believed that it was due to irritation of the anterior horn cells. In the case studied by Hunt nothing was found but hypertrophy of the muscle-fibers.

Diagnosis.—The essential features are sudden, quick, spontaneous, multiple contractions of individual muscles, which do not cause movement of the parts supplied by the affected muscles. This is sufficient to distinguish it from chorea. The so-called *electric choreas* of Bergeron and Henoch are probably varieties of the same disease. A similar type of movement may also be due to hysteria; when so caused, other manifestations of this disease will be found (p. 1227). It may be mistaken for one of the forms of tic. These are discussed below, where the differential points are given. Myoclonus may also occur in association with other diseases, as tuberculosis, epilepsy, and the muscular dystrophies (p. 1270). A family type has also been described by Unverricht.

In the non-hysteric form the **prognosis** is serious, very few of the cases ever showing permanent improvement.

The **treatment** consists of rest, isolation, full feeding, hypodermic injections of hyoscine, arsenic, bromids and valerian internally, and the application of electricity. The latter seems most effective when applied to the spinal column, a constant galvanic stream being employed and the anode being placed over the sensitive vertebræ. It is not unlikely, however, that these cases are of an hysteric nature.

ELECTRIC CHOREA OF DUBINI.

A disease closely allied by its symptoms with the preceding, but probably of very different etiology, is the *chorea electrica of Dubini*, a disease endemic in Northern Italy. It occurs at all ages, affects both sexes, and appears to be of an infectious nature. Occasionally congestion of the meninges has been found; in other cases there are inflammatory lesions in other parts of the body, and particularly in the lungs.

Symptoms.—The disease commences with severe pains in the head, the neck, and the lumbar region. After a brief interval contractions

occur in the muscles, usually appearing first in the upper extremities, but rapidly becoming general. They are almost continuous, and are separated by approximately equal intervals, so that they are distinctly rhythmic in character. From time to time there are attacks of general convulsions, that may occur as often as four times per day, and are usually followed by paresis of the limbs. There is slight hyperesthesia of the skin, and usually more or less fever.

The *prognosis* is extremely unfavorable, death occurring in 90 per cent. of the cases. The duration of the disease varies from two or three days to four or five months, death usually occurring from heart-failure while the patient is comatose.

No satisfactory *treatment* has been suggested.

DYSBASIA LORDOTICA PROGRESSIVA.

THIS affection, also termed *dystonia musculorum deformans* and *torti-pelvis*, has been recently described by Oppenheim.[1] It occurs in children and young adults of the Jewish race. The symptoms consist of tonic and clonic spasms of the muscles around the pelvis, associated sometimes with similar twitchings of other muscles. These cause a deformity around the pelvis. Excepting the deformity the symptoms disappear when the patient is recumbent. Attempts to either stand or walk develop them. There is marked lordosis in the lower dorsal and upper lumbar regions, with prominence of the buttocks and a peculiar gait which has been termed "the monkey or dromedary gait." Symptoms of organic disease of the nervous system are absent.

TIC.

(*Habit Chorea; Habit Spasm; Tic Convulsif; Motor Tic; Palmus.*)

Definition.—Meige[2] has defined a tic to be "a coördinated purposive act, provoked in the first instance by some external cause or by an idea; repetition leads to its becoming habitual, and, finally, to its involuntary production without cause and for no purpose, at the same time as its form, intensity, and frequency are exaggerated; it thus assumes the characters of a convulsive movement, inopportune and excessive; its execution is often preceded by an irresistible impulse, its suppression associated with discomfort. The effect of distraction or of volitional effort is to diminish its activity; in sleep it disappears. It occurs in predisposed individuals, who usually show other indications of mental instability."

[1] Fraenkel, *Jour. Nerv. and Ment. Dis.*, June, 1912, p. 361.

[2] Tics and their Treatment, Meige and Feindel, translated by Wilson, p. 260

Etiology and Symptoms.—These have been practically described in the definition given above. A few points should be emphasized. The disease is especially apt to develop in those who possess a neuropathic ancestry, and who themselves show other evidences of a neurotic diathesis, as neurasthenia, hysteria, etc. Most cases are originally excited by an effort to relieve some peripheral irritation. Thus, a man who had a sore spot on the upper lip which he was constantly moistening with his tongue found that after the sore had healed he continued to protrude the tongue involuntarily. This protrusion has occurred more or less frequently since, until it has become an unconscious act, and at the same time the tongue is protruded much further than would occur in a normal protrusion. By concentrating his attention upon himself he could prevent this, but a marked feeling of discomfort was caused by so doing. He had had several attacks of "nervous breakdown."

Tics may develop at any age. Any group of muscles may be involved. Thus, we have facial or mimic tics, tics of the nose or sniffing tics, of the lips or sucking tics, of the jaws or biting ticks, of the tongue or licking tics, of the neck or nodding tics, of the trunk-muscles, of the arms, of the hands, or scratching tics, of the legs or leaping tics, of respiration or snoring, sniffing, blowing, whistling, coughing, and sobbing tics, etc. Any voluntary and purposive act may be so simulated. The movements are usually quick, and, as has been said, are imitations and exaggerations of normal acts. The predisposed may acquire the condition by imitation of one who has the disease.

Diagnosis.—In the first place, this must be made from a true spasm. A spasm, as defined by Meige (*loc. cit.*), is the motor reaction consequent on stimulation of some point in a reflex spinal or bulbospinal arc. In other words, it is due to some irritative lesion. Patrick[1] has well summarized the points of difference, part of which are here given: Tic is more common than spasm, and invariably develops in a nervous or neuropathic individual. Disposition or temperament appears to have nothing to do with spasm.

Spasm is absolutely devoid of voluntary or involuntary control, while tic, to some extent, is always under the control of the will, and always subject to involuntary control by strong emotional or intellectual pre-occupation. Spasm is an anatomical, tic a physiological, disorder. Voluntary simulation of a true spasm is practically impossible. The patient himself can always repeat or imitate his tic movement, and another person can nearly always do so. A spasm in its incipiency may be confined to part of a muscle or of the muscles taking part in a movement (fascicular contractions). A tic always involves all of the muscles taking part in any one physiological movement. From chorea tic is readily distinguished by the coördinate and purposive character of the movements; from tic douloureux (p. 1079), by the absence of pain. In the so-called general tic, or Tourette's disease, there are mental symptoms (p. 1218) which do not occur in the form just described.

Prognosis and Treatment.—The prognosis is doubtful as regards cure. Of course, it does not cause death. The treatment is educational—that is, training the patient to perform movements the opposite to those involved in the tic; this he can do before a mirror several times daily.

[1] *Journal Nervous and Mental Diseases*, January, 1909, p. 1.

In addition, measures to improve the neuropathic state—viz., fresh air, easily digested, plain food, etc.

GENERAL TIC.

(*Maladie des Tics convulsif; Maladie de Gilles de la Tourette.*)

Definition.—A disease apparently psychic in nature and characterized by coördinated spasmodic movements, explosive sounds or words, and imperative ideas, without intellectual disturbance.

The **pathology** of the disease is unknown. It occurs in those suffering from neuropathic heredity, and usually indirectly. It most frequently commences in childhood—that is, before puberty—and affects either sex.

Symptoms.—The disease generally commences in the *orbicularis palpebrarum*, the first movement being an uncontrollable winking. This, as a rule, is rapidly associated with movements of the muscles of the face, causing the patient to exhibit various *grimaces;* finally, other muscles of the body may be involved, and the patient is compelled to repeat many times some apparently purposeful and coördinated movement, as the brushing away of insects or the stroking of the beard. From time to time he emits sounds that may be either inarticulate cries or imitation of some animal, as the crowing of the cock or the barking of the dog, or the repetition of some obscene word (*coprolalia*). These movements are partially under the control of the will, and are diminished by occupation, but increased by emotion. At other times the patient is compelled to imitate sounds that he has just heard, no matter how unusual or unexpected (*echolalia*). A more curious symptom is the imitation of movements that he has observed (*echokinesis*), which may lead to most absurd or painful results. Still another psychic symptom is the occasional development of imperative ideas. These usually take the form of a desire to recall some unimportant word or syllable (*onomatomania*) or the performance of difficult problems in mental arithmetic (*arithmomania*). One of my patients, a boy of fourteen years, before undertaking a definite act, would repeat the words "ten, ten, ten," three times, followed by a rapid count of figures from one to ten. If riding in a public conveyance, he would do the same, endeavoring to finish before reaching a definite place, as a street-corner, or before hearing the sound of the voice or whistle of the conductor if in a trolley-car. A failure to accomplish the task was cause for intense mental worry. These patients are usually affected at the same time with a certain degree of melancholia or anxiety that interferes to some extent with their normal life. The disease is, as a rule, very obstinate, and ordinarily continues throughout life.

The **differential diagnosis** is not difficult, the presence of motor tic, associated with the peculiar mental symptoms, being characteristic.

The **prognosis** is unfavorable for cure; death, however, almost never occurs as the result of the disease.

The **treatment** is symptomatic, and consists in putting the patient in the most favorable physical condition possible; also hydrotherapy, change of climate, tonics, and the correction of any atonic condition, are all useful measures. Potassium bromid to a certain extent controls the paroxysms when they become very severe.

SALTATORIC SPASM.

(*Jumpers; Latah.*)

Definition.—This is a term applied to a peculiar clonic contraction occurring in the lower legs of a patient on attempting to stand upright. The disease was first described by Bamberger. It appears to occur more frequently in men than in women, and usually in individuals who have suffered from other functional diseases. Occasionally it appears in those who exhibit hysteric phenomena. The condition may develop after severe exertion, and sometimes appears during convalescence from an acute disease. In one of my own cases the condition suddenly arose in the course of habit-chorea. Saltatoric spasm is not a clinical variety of true chorea.

Symptoms.—This condition is probably related to the tics (p. 1216). When the patient attempts to stand, violent clonic convulsions take place in the muscles of the legs, particularly of the calves. These may cause the patient simply to rise on his toes, or they may be so severe as to cause him to spring from the ground, in which case he usually falls. As soon as he lies down the spasms disappear, but they may be produced in patients lying in bed by pressing against the feet.

The **prognosis** is generally favorable. The attacks usually last for a period of from two days (Gowers) to a few weeks, but a few cases have been recorded that persisted throughout life. Gowers recommends diaphoretic treatment. Antispasmodics may also be employed, and in those cases with hysteric stigmata suggestion is useful.

PARALYSIS AGITANS.

(*Shaking Palsy; Parkinson's Disease.*)

Definition.—A chronic disease characterized by a tremor; by rigidity of the muscles; by the peculiar character of the speech and gait, and by a progressive, but very seldom complete, loss of power.

Pathology.—Lesions that are probably only senile in type have been frequently described. There are peri- and endarteritis, irregular degenerations in the posterior columns, and numerous amyloid bodies. Other observers have noted changes in the motor cells of the cerebral cortex. Camp has called attention to the constancy of changes in the muscles, and disturbance of the function of the parathyroids has been thought by some to be the cause of the trouble.

Etiology.—Paralysis agitans is a disease of adult life, developing in the large majority of cases between the fortieth and forty-fifth years; it is met with more often in men than in women. No definite etiologic factor is known, though, as with most, if not all, nervous diseases it is predisposed to by mental strain, worry, or trouble of any kind. Some cases seem to be excited by traumatism.

Symptoms.—Usually the first evidence of the disease is *tremor*, slight at first, and in the extremities, the hand usually being the first to betray it. The movement is very characteristic, the thumb and forefinger being approximated as in the act of making a pill. At the same time the

hand is semi-rotated and the forearm trembles more or less as a whole. The upper arm is either but slightly or not at all affected. The legs are also but slightly implicated. The tremor is most noticeable when the patient is sitting with one leg crossed over the other, the foot then being sure to be in more or less constant motion. When the head is involved (rather the exception than the rule) the motion is a nodding one. The tremors cease when the patient sleeps, but are continuous during waking hours, though it is not rare to meet with cases in which, during purposeful acts necessitating the use of the affected parts, the tremors diminish or even cease temporarily, to return as soon as the voluntary motion is completed. The latter movements, it will be noticed, are awkward, and as the disease advances they become more and more stiff. This *rigidity*, with its consequent impairment of activity, is another cardinal feature of the disease. The patient's movements are slow and apparently measured. There is some impairment of power also, but it is slight, and may be rather from disease than from a direct nerve- or muscle-involvement. Turning in bed unaided is difficult or impossible. The skin is often thickened, and to this change the rigidity has been in part ascribed, but I believe incorrectly, because, particularly in young patients, it may be delicate in texture.

Two of the most striking symptoms of the disorder are the *gait* and *attitude* of the patient. He walks with head and body bent forward, eyes directed toward the ground a short distance ahead, and takes short, mincing, and somewhat hurried steps (*festination*), giving one the impression that he is about to fall, which he would do but for each successive step, which, as it were, gives him a fresh center of gravity. His station is equally striking. The head and back are bent forward, the feet are kept some little distance apart, and one in front of the other, while the arms are slightly flexed and pendulous. From time to time the patient will make a slight forward movement (*propulsion*), or else, if walking, bend or fall backward (*retropulsion*). The facial muscles are set, the eyebrows arched, and the whole expression is "mask-like." The general slow character of all movements, except walking, which is necessarily quicker, is imparted to the speech, though after a sentence is begun the balance may be rendered normally or even hurriedly. The voice may be high-pitched. The pulse is usually rapid; the skin flushes easily, and the patients are sensitive to cold. There are no trophic or sensory symptoms, and the reflexes are normal. Apart from the diffidence, amounting in some cases to a positive dislike for meeting people, and the melancholia occasionally induced by brooding over the affliction, there are no mental changes.

Cases occur in which the tremor is very slight or absent, the diagnosis then being based on the rigidity, attitude, and facial expression.

Course.—The disease is almost always of slow onset and of insidious progress. Often one side is involved before the other, or the two sides are unequally affected. Very rarely the earlier symptoms may develop somewhat rapidly, but in every case their further progress is slow. Disappearance of tremor, usually transient, has been observed in the side affected by a subsequent hemiplegia. The course may be interrupted from time to time; even seeming improvement may take place, but it is not maintained. The disease lasts for years, and the patient usually dies of some intercurrent disease.

The **diagnosis** is not at all difficult when the tremor, attitude, gait, and rigidity have developed. During the earlier stages it may be confounded with *multiple sclerosis*, though this condition develops earlier in life, and the volitional character of the tremor, the nystagmus, and the scanning speech should serve to differentiate it. The muscular rigidity, peculiar gait, and temporary cessation of the tremor after some muscular effort distinguishes it from senile tremor. That the rigidity is not due to disease of the central motor neuron is shown by the absence of the Babinski reflex, and, in most cases, absence of increased tendon reflexes.

Treatment.—The medical management of the disease is unsatisfactory. Graduated exercise, tepid baths, and massage should be employed to keep up the tone of the muscles. The patient should avoid excitement and overfatigue, both mental and physical. The long-continued use of arsenic may be of service, and hyoscine hydrobromate (gr. $\frac{1}{200}$—$\frac{1}{100}$ t. i. d.) often relieves the symptoms. Electricity in the form of dipolar faradic baths has been recommended. Parathyroid gland (gr. $\frac{1}{20}$ of the powdered gland) three or four times daily has given good results.

OTHER FORMS OF TREMOR.

1. **Hereditary tremor** has been described by C. L. Dana, who has also reported interesting cases. It may commence in infancy and continue till old age, unaccompanied by detectable lesions.

2. **Simple tremor**, lasting a longer or shorter period (oftener it is comparatively brief), is a rare condition and without serious possibilities. Its etiology is unknown, though it is sometimes aggravated by nervous shocks and other debilitating conditions.

3. **Senile Tremor.**—This is common in old persons, and rarely appears before three-score-and-ten years. It is excited by muscular motions, is always fine, and affects chiefly the hands and arms; more rarely the neck is also involved, and the head may then be seen to tremble.

4. **Toxic Tremor.**—This results from the action of alcohol, lead, mercury, tobacco, and other poisonous substances. It is usually fine and irregular (*vide* The Intoxications).

5. **Hysteric tremor** (*vide* Hysteria).

6. Tremor may also be a symptom of neurasthenia, exophthalmic goiter, and multiple sclerosis.

TETANY.

Definition.—A disease of unknown cause, characterized by paroxysms of tonic cramp that usually affect the flexor muscles of the extremities, by sensory disturbances, and by a peculiar alteration of the electric reaction of the muscles.

Etiology.—Tetany may occur in epidemics, and has, therefore, been supposed to be infectious. There is some doubt, however, whether these epidemics are cases of true tetany or are hysteric in nature. There is also some evidence that it is due to an intoxication occurring in the course of some other morbid condition. Tetany is frequently associated with infectious diseases; it also occurs in connection with gastro-intestinal dis-

orders, especially dilatation of the stomach, diarrhea, and intestinal parasites, during pregnancy and lactation, and it is associated with the myxedema that develops after the removal of the thyroid gland, in which cases it is due to the parathyroids also being removed. Exposure to cold has often preceded the disease. Occupation seems to exert a remarkable influence upon the predisposition to it, the great majority of those affected being shoemakers or tailors. In childhood males are far more frequently attacked than females, and the victims are frequently rachitic, but in adult life this proportion is reversed. Heredity may have some influence, since tetany often occurs in several members of the same family. It is much more common in the spring months, and, curiously enough, it appears to be endemic in certain localities, particularly Leipsic and Vienna. It is rare in the United States.

Pathology.—Distinct morbid lesions of the nervous system have not been found in all cases. Slight vascular changes in the brain and cord and vacuolization of the ganglion-cells have been described, but these are not peculiar to this disease. It has been supposed that changes ought to be found in the motor nerves, but the most careful observers have failed to detect them. It is evidently occasioned by a toxemia which, in some cases, is due to absence or disease of the parathyroid glands. Excessive excretion of calcium salts is found in the urine.

The **symptoms** fall naturally into two groups: (1) Those of the paroxysm, and (2) Those of the interval. (1) The **first symptoms** of an attack usually consist of peculiar sensory disturbances in the limbs, either tingling, formication, pain, or numbness, and these may precede the attack for some hours or even days. Stiffness of the muscles usually begins in the fingers. There may be slight clonic movements at first, but this is not frequent. The limbs are symmetrically involved. The *spasm* commences first in the hand, the fingers being straightened and flexed upon the hand, and bunched so that the hand has been likened to that of the scrivener or obstetrician. The spasm then extends to the muscles of the forearm and arm, and usually also to the feet and lower limbs. If the cramp is slight, the pain may be insignificant, but ordinarily it is severe, and is increased by attempts to overcome the contractures. The muscles are hard, painful upon pressure, and occasionally fibrillary twitchings may be observed. There is sometimes a slight edema, and often sweating of the limbs. The paroxysms may last for several minutes or for several hours or even days, and may even persist during sleep. If, however, the period is very long, remissions are usually observed. Sometimes a series of paroxysms may occur with considerable regularity. Attacks are more likely to occur at night, and they may also be brought on by prolonged and severe muscular effort, or by emotional shocks. Besides the paresthesiæ in the affected extremities, the patient may suffer from severe headache or pain in the back and neck.

(2) The **symptoms of the interval** are—*Trousseau's sign—i. e.* the possibility of causing an attack by prolonged pressure upon the main nerve-trunks or vessels of the limbs. Frankl-Hochwart has shown that pressure upon the nerves is essential; pressure upon the vessels acting secondarily if at all. *Chvostek's sign* is a peculiar excitability of the muscles of the face, so that spasms are produced if the trunks of the facial nerve are lightly percussed by a hammer. This occurs in other conditions, particularly the cachexiæ, but in most cases of tetany the spasm

occurs if the skin of the face is lightly stroked; and this reaction appears to be pathognomonic. *Erb's sign* consists of a greatly increased electric excitability of the muscles, and, occasionally, of an alteration of the electric reaction, so that AClC may be greater than KClC. Moreover, AOTe is often obtained, and, in at least 2 cases, KOTe has also been noted. The last two reactions occur in no other condition. *Hoffman's sign* consists of an increased reaction of the sensory nerves to electric stimuli. The facies of the patient is peculiar and characteristic. The face is slightly swollen, dusky, and expressionless, but if carefully examined usually no edema can be detected. Often this swelling is also found in the hands and feet, and may be associated with distinct enlargement of the veins. Even during the interval the feet when at rest have a tendency, particularly in children, to assume a slightly inverted and extended position.

The *sensory disturbances* consist of cramp-like pains during the attack, some diminution of sensation in the affected parts, and increased sensibility of the sensory nerves. The contractures are not invariably limited to the extremities. Sometimes the muscles of the neck, back, and larynx are involved; sometimes also the diaphragm, and occasionally the compressor urethra. Involvement of the larynx gives rise to stridulous respiration; involvement of the diaphragm to severe dyspnea; when the urethra is compressed there is retention of the urine. *Fever* occurs in about one-half the cases; it is slight and generally limited to the paroxysm. The urine usually contains a large amount of phosphates, and less frequently indican is present in excess. Partial tetany—that is, with the absence of one or more of the cardinal signs—occurs not infrequently, especially in gastro-intestinal conditions. Pain, cramp, and Trousseau's sign are usually present.

The **differential diagnosis** is very easy, as a rule, if the patient is seen during a paroxysm. The severer forms may, however, be confounded with *tetanus* or *meningitis*. Tetany can be distinguished from the former by the fact that the spasm begins at the periphery and rarely affects the muscles of the jaw. From the latter it may be diagnosed by the absence of coma and the slighter degree of fever. The diagnosis from certain forms of *ergotism* is more difficult, and can often be made only by careful attention to the etiology. The *hysteric forms* can be differentiated by finding various hysteric stigmata. An epidemic occurring among young women should give rise to a suspicion of the true nature of the malady.

Prognosis.—The duration varies from a few days to many months, the most obstinate forms being those due to thyroidectomy, in which the parathyroids have also been removed, and chronic diarrhea. The disease cannot be said to have disappeared until the characteristic symptoms of the interval (Trousseau's, Erb's, and Chvostek's signs) can no longer be elicited. The prognosis is usually favorable, nearly all cases tending to spontaneous cure. Death, however, may occur from chronic diarrhea, from respiratory failure when the diaphragm is involved, and from cachexia strumipriva.

The **treatment** is purely symptomatic. The patient should be placed in the most favorable hygienic conditions and given plenty of nourishing food, especially that containing calcium salts. Full doses of these salts, as calcium chlorid or lactophosphate, should also be given. During the spasm bromids or chloroform-inhalations seem to give the best

results. The most important therapeutic measure is the correction of the underlying cause. Thus, in children rachitis is almost invariably associated with tetany, and the most efficient remedies are iron and cod-liver oil. Intestinal disorders should be treated according to the principles laid down in the discussion of these diseases. The form due to removal of the thyroid gland usually disappears under a course of parathyroid medication, while that occurring during pregnancy usually persists until delivery.

PERIODIC PARALYSIS.

(*Family Periodic Paralysis.*)

Definition.—A disease characterized by paroxysmal attacks of complete paralysis, and alteration in the electrical reactions, occurring in many members of a family.

Pathology.—In excised fragments of muscle Goldflam and Oppenheim found hypertrophy of the fibers and slight vacuolation, without multiplication of the nuclei or proliferation of the connective tissue. In most cases no changes have been found, and the condition has been supposed to be an auto-intoxication, associated with a lowered condition of metabolism. In a case observed by Mailhouse there was a diminished excretion of calcium and magnesium salts in the urine, and he mentions the possibility of the paralysis being due to their inhibitory influence upon nerve and muscle when retained within the body.

Etiology.—The disease is purely hereditary. Both sexes are affected. The attacks appear to be more frequent in summer, and often seem to occur after overfilling the stomach.

Symptoms.—The attacks are preceded by *prodromes* in the form of vague discomfort or paresthesia. The patient then usually falls asleep and awakens completely paralyzed. Speech, deglutition, and the sphincters are unaffected. During the attack there is often transient *albuminuria*, with blood-cells in the urine. The *reflexes* are abolished, and the muscles either do not react well or not at all to the electric current. The *paralysis* lasts from twelve hours to three days, and then there is an outbreak of perspiration, with gradual recovery, the muscles of the head first regaining power. During the interval the muscles react to electricity and the reflexes return. Dilatation of the heart has existed during an attack, to disappear during the interval. A few cases have been associated with migraine.

Prognosis.—The disease does not usually kill; but there appears to be no tendency to recover, and a few cases have died during the attack.

Treatment.—This involves only caring for the children during the attack. Large doses of potassium citrate have been beneficial.

HYSTERIA.

Definition.—A condition of the general nervous system partaking of the natures of both a neurosis and a psychosis, and characterized by a vast multiplicity of clinical manifestations, all indicative of a loss of voluntary control over inhibitory and active nervous influence.

Pathology.—Hysteria is to be regarded as essentially a morbid entity, without, however, any tangible pathologic features. The most careful *postmortem* examinations of subjects who have while in life manifested pronounced hysteric symptoms have failed to reveal any organic nervous alterations, however slight. The occurrence of the affection in men as well as in women excludes the former theory of a uterine pathology, which, though an idle fancy, held sway for so many centuries and gave origin to the name by which the condition is generally recognized.

Etiology.—There are a large number of predisposing and exciting factors, all of which, however, may be grouped under a few dominant heads. Thus among the former must be mentioned, pre-eminently, *heredity*. The investigations of many neurologists and alienists of divers lands have gone far to demonstrate that at the foundation of the vast majority, if not of all, of the hysterias is to be discovered an inherited neurotic tendency or temperament. The family histories of these patients generally reveal a large number of consanguineous, neurotic, or hysteric individuals. It is apparently in close relationship with the various psychoses and major neuroses (epilepsy, chorea, tetany); and with the so-called rheumatic diathesis.

In the process of transmission one generation may entirely escape the pernicious influence, and successive generations may manifest strikingly different evidences of the disease, in one the neurotic and in another the psychic element predominating. A curious phenomenon that is worthy of mention is the apparent *contagiousness* of hysteria; moreover, the baleful influence one neurotic individual exerts over the unfortunates of this temperament explains the so-called "hysteric epidemics" that have swept over communities, and even over vast tracts of land or entire countries, at different periods of the world's history. Similar, though limited, outbreaks may still be seen in the nervous wards of hospitals or in religious and political conventions, and these depend largely upon the general prevalence of the neurotic disposition untempered by a virile will-power.

The hysteric temperament may be, and often is, fostered by improper and pernicious modes of life, especially by luxurious and sensuous living and by the habit of gratifying every desire of the will during early life. It is manifested at this early stage of the individual's existence by hypersensitiveness, brilliancy, undue enthusiasm, and a more or less erratic turn of mind.

Contrary to the prevailing opinion, hysteria is not limited to the female sex, although they are the chief sufferers from the more dramatic forms. Instances of a most rebellious nature not infrequently occur in the opposite sex.

Age.—The condition is generally encountered between the ages of fifteen and thirty years, although it is often enough observed in young children. After the latter age the frequency of the disease rapidly diminishes.

A very influential factor in the production of the disease is the *lack* of *proper mental development.* It stands to reason that those who are coarse and illiterate, and who have not been taught the lessons of self-control, and who are subject to the various and multiplex superstitions that are ever prevalent among the masses, will respond more quickly and

more generally to the causes that tend to destroy mental equilibrium. Hence, hysteria or insanity shows its rankest development among those whose education and culture are defective. This is, however, by no means an inevitable law, for over-stimulation of the faculties may be just as deleterious as under-stimulation, and some of the brightest lights of the world have manifested at various periods of their lives decided hysteric symptoms.

Improper hygienic surroundings, tending as they do to enervation and physical depression, are influential predisposing factors in the evolution of hysteria. In addition to poor and insufficient food, lack of proper ventilation, overcrowding in foul habitations, and insufficient bathing, must be mentioned the enervating influence of hot and moist climates. It is generally conceded that more cases of hysteria occur in the warm than in the colder portions of the temperate zones, and that this proportion increases *pari passu* with the height of the temperature.

Finally, the causal influence of the chronic toxemias (alcoholism, morphinism, absinthism, saturnism and intoxications by other metals) is to be mentioned. In systemic poisoning the depraved condition of the physical reacts upon the mental organism, and sooner or later hysteric manifestations may be found to coexist with the original toxic phenomena.

The *exciting causes* of hysteria may be grouped as follows:

(1) Most commonly psycho-neurosis follows some profound emotion or mental or moral shock. Thus, in neurotic males it may be excited by excessive and protracted business-worry or excitement, or by active competition in certain lines of occupation, or by some heavy and unexpected monetary reversal. In females it is not uncommon as a sequel to the establishment of puberty and the menstrual function, or to the physiologic arrest of menstruation at the period of the climacteric. Especially is it prone to develop in young and illegitimately pregnant women, or during the first pregnancy in newly-married women of a neurotic temperament. Great religious excitement during the progress of a revival-wave and profound political upheavals have been most potent in establishing the disease in numerous instances; and other profound mental impressions, of fear, grief, or great and unexpected joy, have assumed the exciting rôle. In this connection the theories of Breur and Freud, now receiving considerable attention, but only accepted by comparatively few, should be mentioned,[1] but in the space at hand it is impossible to give them in detail. They teach that hysteria is always due to a physical or psychical trauma, which may have occurred some time before the symptoms develop. According to Freud, the trauma practically always is sexual in nature. Thus there develop in the period before puberty definite sexual activities, which are mostly of a perverse nature. These activities do not, as a rule, lead to a definite neurosis up to the time of puberty, which, in the psychic sphere, appears earlier than in the physical; but sexual fantasy maintains a perverse direction by reason of the infantile sexual activities. On constitutional (affect) grounds the increased fantasy of the hysteric leads to the formation of complexes which are not taken up by the personality, and, by reason of shame or disgust, remain buried in the subconscious mind. There

[1] *Psychanalysis*, Brill, W. B. Saunders Co.

therefore results a conflict between the characteristic normal libido and the sexual repressions of these buried infantile perversions. These conflicts give rise to the hysterical symptoms.[1]

(2) Extreme physical prostration, the result of some very acute or much protracted chronic disease, may exert an etiologic effect. Thus, some of the most marked and intractable forms of the disease have resulted from the specific fevers (typhoid, typhus, and the other exanthemata), while it is not rare in a varying degree in the final stages of tuberculosis, chronic nephritis, and other grave constitutional affections of long standing.

(3) The so-called "traumatic hysteria" has come to occupy a prominent place in the etiologic category of the disease. Especially do we find the incurable varieties of hysteria resulting from a slight or, it may be, a more severe traumatism. It must be remembered that a considerable period of time may intervene between the date of the injury and the appearance of the initial hysteric symptoms, so that in all cases it becomes of the utmost importance to make a careful study of the patient's history for signs of traumatism, however remote. It has also been noted that oft-repeated minor traumatism may finally result in some hysteric manifestations.

(4) Finally, in a limited number of cases sexual excesses and masturbation are the influential factors in the production of hysteria. These sexual cases, though few in number, do exist, and are especially to be found among the class of so-called sexual perverts.

Clinical History.—Clinically, hysteria presents three well-marked stages, known respectively as the *prodromal*, the *convulsive*, and the *latent*. The latter is also designated as the *interconvulsive stage* or the *period of the stigmata*, and during this period the number of the symptoms and their complexity almost baffle attempts at classification; they can, however, best be portrayed by presenting them under the heads of the various systems (*vide infra*).

(1) **The Prodromal Stage.**—The prodromes are invariably present, and at times they are more marked than at others. They are evident alike to both patient and physician, and are largely psychic in nature. There may be noted a marked mental depression associated with introspection, and, it may be, with a form of mild mania or of melancholia. A condition of aprosexia develops, and the patient becomes irritable, restless, and discontented. The mental derangement may manifest itself in the form of delusions or nightmare, and there is a characteristic neglect of the toilet and attire. There may also be disturbances of the gastro-intestinal tract—viz., anorexia, nausea, vomiting, constipation, and perversions of taste. These phenomena persist for several days and are followed by emotional disturbances—spasms of hysteric laughing and crying—that immediately precede the *aura*, which is as marked a feature in hysteria as in epilepsy. It may assume one of a number of forms, but more commonly it has an ovarian, a cervical, a cerebral, or a surface or cutaneous origin (unilateral). Very frequently the convulsion is preceded by a condition of extreme sensitiveness and pain in one or both ovarian regions, so that the lightest touch at a point on the abdominal surface one inch above Poupart's ligament, and midway between the pubis and the anterior superior iliac spine, will elicit ex-

[1] Jelliffe, Osler's Modern Medicine, vol. vii., p. 816.

quisite tenderness. This is so constant and characteristic that many patients can invariably predict the onset of the convulsion. Not infrequently the aura begins in the neck, the patient experiencing a sensation as of a ball lodging in the throat (*globus hystericus*): this is due to a spasmodic contraction of the muscles of the pharynx and esophagus, and is accompanied by tachycardia and a sense of suffocation. If the aura originate above the scalp, it is characterized by the sudden appearance, generally in the top of the head, of a severe neuralgic pain, as if produced by the entrance of a nail (*clavus hystericus*); this is frequently associated with vertigo and tinnitus aurium. The aura, finally, may appear in the form of spots of cutaneous tenderness, mainly localized upon the trunk, to which areas has been given the name of *hysterogenous zones.*

(2) **The Hysteric Convulsion.**—Closely following upon the footsteps of these prodromes, and immediately following the aura, the hysteric convulsion may appear. Most commonly this is epileptoid in nature; rarely it assumes a less common type. Hence it becomes necessary to describe several of the forms of the convulsions—viz. (*a*) the *epileptoid* (hystero-epilepsy); (*b*) the *gymnastic* (clownism); (*c*) the *emotional cataleptic*, or *dramatic*; and (*d*) the *delirious*. All of these forms may be present in the same attack, the one passing quickly into the other, or, as in the abortive cases, one or the other form will predominate. Briefly described, the characteristic features are as follows:

(*a*) *Epileptoid* (*Hystero-epilepsy*).—Immediately upon the appearance of the aura the patient commonly emits a shriek and falls upon the floor or in some convenient place, taking special care to do herself no injury: this is in strong contradistinction to the true epileptic spasm. The head and limbs are thrown about by more or less violent clonic muscular spasms, and at times a condition of opisthotonos or other trunkal contortion (emprosthotonos, pleurosthotonos) may be noticed; these muscular movements, however, are more or less volitional, and are not the aimless movements of the true epileptic. In some cases there is merely a tonic spasm or muscular rigidity. The patient may or may not foam at the mouth. There is a constant twitching of the eyelids and the eyes are rolled about, but apparently retain a more or less observant expression. Consciousness, as a rule, is not fully lost. The facial muscles are distorted, rapid changes of expression being noted (*hysteric trismus*), and respiration is somewhat impeded. As the convulsion passes off the movements gradually subside, and the patient generally sinks into a state of quiescence or, it may be, into a light sleep. This may be followed by complete temporary recovery, or the epileptoid may pass into one of the other forms of the convulsive seizure. The duration of the spasm as described is usually longer than that of a true epileptic seizure. This form, more or less severe, is the one usually seen in this country.

(*b*) *The Gymnastic Form* (*Clownish*).—This stage is characterized by violent and grotesque muscular movements. Here are to be grouped all of the more curious manifestations of the disease recorded in the history of medicine. The most difficult feats of the contortionist are performed with apparent ease; the patient may suddenly begin to dance or jump at a most astonishing rate, persisting in the movements until she drops from pure physical exhaustion. The so-called religious ceremonies of the

Shakers of Lebanon, Pennsylvania, and of the Jumpers of the Middle Ages are manifestations of this form of hysteria. In children the attack may appear as the so-called *beast-mimicry*, in which the movements or sounds of the lower animals may be simulated; such is also the explanation of the condition known as *spurious hydrophobia*. Consciousness is never lost during this period.

(*c*) *The Emotional Cataleptic, or Dramatic Form.*—In this form the patient seems to suffer from delusions or hallucinations that are apparently the outcome of the preceding condition. The emotion that is most developed in the patient's moral constitution now dominates his spasmodic actions. As Lloyd aptly expresses it: "The third period of the hysteric convulsion is one of dramatic representation of emotional images, and these are of countless varieties, according to time and person." All of the manifestations of the cataleptic state are present. Sensation is largely abolished, consciousness is retained, and the patient is usually able to recall events that have transpired during the period. Especially common now is the assumption of dramatic and passionate attitudes, which, as described by Richer, include "the attitude of the cross, of defence, of menace, of appeal, of lubricity, of ecstasy, of dread of animals (as rats), of scorn," and the like. The body of the patient retains, at times for indefinite periods, whatever position is first assumed (*hysteric catalepsy*). In some cases the patient falls into a condition of apparent sleep or narcolepsy (*hysteric sleep, hysteric somnolence, hysteric trance*) of varying degrees of intensity; this may persist for any period of time, from a hour or two up to weeks, months, or even years. In these extreme cases, while the patient at first appears to be in a normal sleep, sooner or later the body assumes a corpse-like appearance, with pale, waxy skin, almost imperceptible respiration and cardiac action, and a subnormal temperature.

(*d*) *The Stage of Delirium.*—The final stage of the hysteric convulsion is but a continuation of the preceding period, with, however, a cessation of the muscular movement to a great extent. The tendency now is to delirium of a mild type, tinged with more or less melancholia. Consciousness is maintained throughout this stage, and there now appear some curious motor phenomena that may persist for days or weeks. These may consist in the abolishment of muscular power in various portions of the body. Very often associated with these motor phenomena is noted a condition of mutism that lasts for indefinite periods of time.

Hysteric paralyses occur, and may simulate any form of the organic paralyses (monoplegia, hemiplegia, paraplegia). In many cases the patient is left with a more or less permanent spasm of a single set of muscles or of associated sets. These so-called *hysteric contractures* may affect any portion of the body. One arm may be bent at the elbow or one leg at the knee; in the former case the fingers are rigidly contracted and embrace the thumb, which is crossed upon the palm, while in the latter the toes are strongly flexed upon the plantar surface and the foot is inverted. The ankle- and knee-jerk persist. In other cases a curious spastic gait is produced that closely simulates that of spinal sclerosis. The muscles of the hips, shoulder, back, and neck (*hysteric torticollis*) may share in the process. In women the muscles of the diaphragm and abdominal walls may be involved (*hysteric pseudo-cyesis*). *Hysteric rotary spasm, hysteric athetosis*, and *hysteric tremor* are all dependent upon a spasmodic

action of the muscles affected. The convulsive seizure generally is of *short duration*, lasting but fifteen to thirty minutes. Occasionally, however, there is developed a prolonged convulsive status, during which time the patient continually falls from one convulsion into another, until one hundred or more may be recorded and the excess of nervous power is exhausted.

(3) **The Latent or Interconvulsive Stage, or Period of the Stigmata.**—After the convulsive attack the patient enters upon a more or less prolonged interval of comparative quiet; this is characterized, however, by numberless and varied phenomena—the *hysteric stigmata*. The whole course of the affection may be comprised in this period, convulsions being absent. As I have already stated, these can best be described under the heads of the various systems:

(*a*) **The Nervous System.**—This presents the most characteristic hysteric stigmata. They are generally grouped into the three classes of *motor*, *sensory*, and *psychic*.

The *motor symptoms* have already been referred to in part in the description of the hysteric convulsion. They embrace every variety of muscular pathology, from obdurate paralysis to and including tremor, which may be either fine or coarse, incoördination, and tonic spasm or contraction. The hysteric paralyses, as stated, may be absolute or partial, and either general or limited to groups or to individual muscles, and may simulate any variety of true paralysis of organic origin. There is usually noted an exaggeration of the reflexes of the affected side; muscular wasting, if present, is very slight and due to disuse; usually it is absent. It is not at all uncommon to find associated contractures and sensory phenomena. The paralyzed limb or limbs show evidences of circulatory disturbances, as edema and bluish discoloration. In the paraplegic cases it is unusual for trophic disturbances (bed-sores) to appear. Paralysis of muscles supplied by motor cranial nerves, especially those of the larynx and pharynx, may occur, with these exceptions it is usually a pseudo-paralysis due to spasm. Hysteric tremors are not infrequent, and are usually well marked and persistent. They are generally associated with contractures and other hysteric stigmata. Choreiform movements may be simulated, but they are usually more quick and rhythmical than true chorea. It is important to remember that hysteria may co-exist with chorea; also, that apparently true choreic movements may arise from imitation, in which event it is justifiable to term them hysterical.

Hysteric incoördination (*hysteric ataxia*) has also been termed *astasia-abasia;* it is one of the rarest of the motor phenomena of hysteria. The name implies an inability to stand or walk, although muscular power in the legs and trunk is retained, and they can be moved perfectly well when the patient is at rest. *Hysteric contractures* may occur as distinct phenomena or may be associated with some form of hysteric paralysis. Usually the contractures occur with startling abruptness, and are most intense and persistent. They may persist during sleep, but disappear under the influence of an anesthetic. There may be associated sensory phenomena. The toes and the fingers are most frequently the seat of contracture, but the muscles of the face and neck may likewise share in the affection.

Sensory Symptoms.—The anesthetic, hysteric, and paresthetic varieties are noted. The anesthesia may be general or it may involve but half of

the body or scattered areas of the cutaneous surface. *Segmental anesthesia* is the term applied to that condition in which a limb or a portion of a limb is involved. Not only is the skin affected, but often the deeper tissues as well, and there is generally some vasomotor involvement, as is shown by the fact that punctures by a needle are not followed by bleeding. There is often associated an anesthesia of one or more of the special senses (*hysteric amaurosis* or *blindness*, *hysteric deafness*, and *hysteric anosmia*). The anesthesia is severe, as a rule, immediately after an hysteric convulsion, but it may be entirely absent throughout a given case of hysteria. There is often contraction of the field of vision or inversion of the color fields, the red being more extensive than the blue. This may also be due to brain tumor.

Hysteric hyperesthesia is also a frequent clinical manifestation, and is generally confined to limited areas, as the ovarian, mammary, or spinal regions, or to one of the larger joints (*hysteric joint*), simulating organic disease of the part. Pressure upon these areas may precipitate paroxysmal attacks, and they have been termed hysterogenic zones. These conditions can be recognized by etherizing the patient, when perfect mobility of the affected joint is noted. When one of the mammæ is involved, the organ becomes exceedingly painful to the touch and slightly edematous (*hysteric breast*). *Hysteric paresthesiæ* include the common varieties of formication, dead fingers, and the like.

Psychic Symptoms.—These form some of the most interesting and remarkable of the manifestations of the disease. Lethargy or a tendency to sleep may exist, the periods of which may follow or alternate with the crises. The sleep in this condition is peculiar because complete muscular relaxation does not exist, as is the case in ordinary sleep. There may also be mental depression and unrest, melancholia, and a notable lack of volitional power whereby the patient becomes especially open to the suggestions of the hypnotist. Double consciousness or somnambulism is a peculiar state, often following a grand crisis, but occasionally arising independently. The morbid period may last for a few minutes or hours or may extend for days or months. During its continuance the patient may be excited and more or less abnormal, or an apparently normal person of altered character. The most remarkable feature is the loss of memory for the normal state, and the recollection of what transpired during the preceding attacks, and loss of memory for all that happened during the attacks in the normal state, so that the subject may actually live two lives. Analogous to these are the states of ambulatory automatism, in which, as a result of an irresistible impulse, the subjects may wander considerable distances from home, appearing more or less normal during the journey, but preserving an imperfect recollection of what had taken place. Somewhat similar attacks occur as substitutes for the epileptic attack.

(*b*) **The Digestive System.**—Among the usual clinical manifestations of this group may be mentioned *anorexia* (which may be complete), a strange and persistent perversion of taste, occasional uncontrollable vomiting without nausea (*hysteric vomiting*, *anorexia nervosa*), marked dyspepsia, and at times extreme emaciation with dryness and a parchment-like feel of the skin. Excessive flatulence and the peristaltic unrest of Küssmaul may be marked symptoms, as may also either diarrhea or constipa-

tion. *Hysteric hematemesis* is the result of swallowing blood; this is usually drawn from the gums or tonsils, or it may be taken secretly by the patient from other external sources.

(*c*) **The Respiratory System.**—Difficulty of respiration (*hysteric dyspnea*) is not uncommon, and is characterized by an extreme rapidity and shallowness of the respiratory movements. These are much out of proportion to the heart-beats, and are unassociated with cyanosis. In other cases the disturbance assumes the form of uncontrollable yawning, sneezing, or hiccoughing, due probably to a spasmodic action of the involuntary muscles of the bronchial tubes and diaphragm. *Hysteric cough* is a troublesome, and very often a stubborn symptom, occurring especially in young females. It is dry and barking, and, as a rule, unaccompanied by expectoration. At times it may be followed by *hysteric hemoptysis*, in which there is an escape of light-red fluid from the pharyngeal mucosa. *Hysteric aphonia* is also frequently noted; in this condition the patient speaks in a scarcely audible whisper. In such cases restoration of the voice is as of sudden occurrence as is its loss. In one of my own cases aphonia manifested almost true intermittence for a period of five years, while during the last two years or over it has stubbornly persisted even without remission (p. 1109).

(*d*) **The Vascular System.**—*Hysteric tachycardia* is often noted, and much less frequently *hysteric bradycardia* appears. A variety of *pseudo-angina* is not of rare occurrence (*vide* Angina Pectoris, p. 713). Very frequently the patient exhibits a localized flushing of the skin (*hysteric erythema*), and especially of the face and neck, or, as has already been noted, there may be an apparent bloodlessness of a part. Profuse general or localized sweating is not uncommon, and may at times be bloody.

Hysteric fever may be mentioned here as a rare manifestation, the bodily temperature usually being normal in hysteria. The elevation of temperature may be moderate or there may be an extreme hyperpyrexia (110°–120° F.—43.3°–48.8° C.), without grave results. If this be associated with localized neuralgia, it becomes a difficult matter to diagnose between the neurotic condition and organic disease of the apparently affected part.

(*e*) **The Urinary System.**—An excessive flow of urine (*hysteric polyuria*) is of very common occurrence, while the opposite condition (*anuria*) is much rarer.

Diagnosis.—The diagnosis of hysteria depends entirely upon the discovery and recognition of the hysteric stigmata; for one or more of them is always present. Of these the most frequent are areas of anesthesia, concentric narrowing of the visual field and inversion of the color fields, and hysterical aphonia, although any of the others that have been described may occur. If, in addition, hysterical crises are present or have been observed, the diagnosis becomes certain. A valuable feature is the inability to explain the symptoms by reference to the anatomy of the nervous system. It must not be forgotten that hysteria and organic disease may coexist.

Differential Diagnosis.—Very important is it to distinguish between hysteric and true paralyses, and between hysteric and organic abdominal tumors. In the following tables the most striking points of difference between these conditions have been set down:

Hysteric Palsies.	Organic Palsies.
Occur without a previous history of organic disease, but with a neurotic history. Traumatism may be the cause.	Are always secondary to organic disease of the neuromuscular system.
Are accompanied by other hysteric stigmata or perversions of sensation.	Hysteric stigmata are absent.
Are not accompanied by wasting of the muscles involved.	If due to a lesion of the peripheral neuron, atrophy is present. In central neuron lesions it is usually not marked.
Reactions of degeneration are absent.	Reactions of degeneration are more or less marked in peripheral palsies. In central, the electrical reactions are normal.
The power of motion returns before sensation.	Sensation if absent first reappears.
In hysteric hemiplegia the facial muscles are not involved.	The facial muscles of the same or opposite side are often involved in true hemiplegia.
Anesthesia generally causes relaxation of hysteric contractions.	Organic paralytic contractions are not affected by anesthesia.
The sphincters are never involved.	Often are in paraplegias.
Babinski reflex not present.	In central palsies (pyramidal tract) it is present.

Hysteric Abdominal Tumors (Pseudocyesis).	Organic Abdominal Tumors.
Almost invariably occur in neurotic women near the menopause.	Occur irrespective of sex.
The percussion-note is invariably tympanitic.	The percussion-note over the swelling is dull, or a dull tympany.
Anesthesia causes a disappearance of the tumor.	Anesthesia has no effect upon the tumor.
Is variable as to size and tonicity.	Slowly but steadily progresses in size.
Is accompanied by tympany and flatulence.	The bowels are not always distended by gas.

Hysterical hemianesthesia differs from that due to organic disease in that the special senses and mucous membranes are affected. The line of demarcation is sharp and in the middle line. Tickling the anesthetic mucous membrane of the nose will cause tears to flow, which will not happen if of organic origin, and lateral homonymous hemianopsia is never present. If either the segmented form or scattered areas of sensory paralysis are present, they often have no connection with any known area of nerve distribution. They all may be transient, reappearing and disappearing, and changing their location.

The differential diagnosis between hysteria and true neurasthenia, psychasthenia, and epilepsy will be found in the discussion of these affections.

Prognosis.—As regards death, the prognosis in hysteria is good; true hysteric patients never die of the disease, nor does the hysteric spasm ever result fatally. As to an ultimate cure, however, the prognosis is very doubtful. If the disease occur early in life and if there is a marked congenital neurotic tendency manifested in the patient, there is almost no hope of effecting a permanent cure. In the acquired cases, under proper moral and hygienic control great benefit may be effected or even an absolute cure recorded.

Treatment.—Of the Temperament.—Accurately speaking, the treatment of hysteria should be begun before birth. Neurotic women bearing

children should be subjected to a course of rest-cure and mental and moral suasion, and the condition of their nervous systems should receive the careful attention of the attending physician. Neurotic children require the greatest care during the developmental period. A strong physique must be secured by proper attention to out-of-door exercise, and, for the time being, even at the expense of mental culture. Such children should not be subjected to the "cramming" process so common in our modern courses of education, but should be trained, if possible, at home, where the element of competition may be eliminated. Systematic hours of study and of recreation (with absolute rest from study during the summer months), and opportunities of travel and change of air and scene, will work wonders in these hyperesthetic little individuals. Especially at the time of puberty is the greatest of care required in order to avoid an additional strain upon the already seriously taxed nervous system. In addition to the foregoing a strict watch must be kept over the moral nature of the child. The satisfaction of every whim and the lack of moral suasion are the surest ways to develop the hysteric temperament. When possible the child should be taken away from the enervating influences of city life. The diet should be plain, but nutritious, and all over-indulgence is to be absolutely prohibited. Frequent bathing and friction of the skin are very beneficial, as well as careful regulation of the emunctories generally.

The Hysteric Convulsion.—As hysteric patients almost never injure themselves during a paroxysm, protective measures are not necessary. Indeed, the attack is usually prolonged by attention and observation. Extreme measures to cut short an attack are only justifiable if the friends and relatives become unduly anxious. Cold plunge-bathing, dashing cold water into the face, or the hypodermic injection of apormorphin, thereby producing a profound mental shock, may have a beneficial effect. Pressure over the ovary or upon one of the large vessels (as the carotid) will sometimes promptly induce a termination of the attack.

Internal Treatment.—In the latent period of the disease it is probable that most can be done to improve the condition of the patient. In addition to the general laws of mental and physical regimen already advanced, she should be taught, so far as possible, the undignified condition into which she is sinking, and advised and encouraged to exert powerful efforts to control her nervous organism. All harsh methods are to be deprecated, nor should she, after the first admonition, be reminded too constantly of her condition. Full doses of the nerve-sedatives and antispasmodics (valerian, asafetida, sumbul, musk, and camphor), together with the general tonics (iron, arsenic, strychnin), are often useful. Change of environment, and particularly of associates, is often of the greatest value. I have repeatedly found the rest-cure of Weir Mitchell especially beneficial at this time; it is fully described under Neurasthenia (*vide* p. 1239).

Hypnotism has commanded considerable attention during this stage of the disease, and it is claimed that under the suggestion of the hypnotist an absolute cure very frequently follows. This is not altogether true, however, for while many patients are undoubtedly benefited by this procedure, the good result must be attributed not alone to the suggestion of the operator, but also to the profound mental effect produced upon the patient by the mysterious process. Hysterical symptoms can be relieved,

however, by suggestion and persuasion, *without* putting the patient in the hypnotic state, and this constitutes a valuable procedure.

In the treatment of the organic manifestations, which, it must be remembered, are dependent entirely upon the general nervous condition, the physician is called upon to exercise the greatest amount of tact. As far as is possible the mind of the patient must be directed away from the affected part. The *irritable bladder* must be treated by internal remedies, as boric or benzoic acid, salol, or the compound infusion of buchu, and not by local irrigation and catheterization.

Hysteric vomiting may not require any special medication. Occasionally, however, it may be relieved by rectal alimentation or gastric lavage. Cocain hydrochlorate in the form of a 10 per cent. solution (3 to 5 drops internally), and the application of mild counter-irritation or of a small fly-blister over the epigastrium will be useful. Cannabis indica, acetanilid, phenacetin, and antipyrin, in small doses and only when absolutely needful, will relieve *hysteric neuralgias*, especially the cephalalgia. For the pseudo-angina pectoris, digitalis, strophanthus, caffein, amyl nitrite, or nitroglycerin, or a combination of these drugs in suitable doses, may be exhibited.

For the *pelvic hyperesthesia* of hysteric females local applications (tincture of iodin, croton oil, or a small fly-blister) over the ovarian region may prove very beneficial.

Hysteric palsies, either general or local, and hysteric disturbances of the special senses, must be treated on general principles. As far as is possible the patient's attention must be directed from the affected part or parts, and an occasional local blistering, the use of galvanism and massage, with daily friction, will be of service, especially when they are supplemented by an appropriate course of internal medication.

Electricity is a very valuable adjunct. The static current is most effective, and it may be applied in various forms. Perhaps the most useful of these is the spark, which should be drawn from the anesthetic area or the paralyzed limb, thus producing a profound mental effect.

Analytical or Cathartic Method.—Based upon the theories of Breur and Freud (p. 1226), this method has been evolved. Briefly, it consists in getting the patient "to tell the story of his life." In other words, while in a relaxed condition (reclining on a couch) he is encouraged to make a confession of all the disagreeable happenings of his life. This may require some time and a number of séances. Clues to these may be obtained from the description of the patient's dreams. Gaps may have to be filled in by the physician by some method of psycho-analysis. After the so-called mental catharsis has occurred, the cause of the trouble being so determined, the patient usually recovers.[1]

NEURASTHENIA.

Definition.—Functional exhaustion and irritability of the nerve-centers. Neurasthenia is the expression of an abnormal sensitiveness (irritability) in response to stimuli, and of weakness of the nerve-centers presiding over the organic functions. Several varieties—cerebral, spinal, cardiac, and gastric—have been distinguished, owing to the fact that the predominating features may be manifested by single organs or systems of

[1] *Psychanalysis*, Brill, W. B. Saunders Co.

the body. That the disease is essentially generalized in all instances, however, I do not doubt. It is not a psychosis.

Pathology.—A variable degree of weakness of the sympathetic centers, permitting congestions on trivial provocation, is obvious, but there are no discoverable lesions (coarse) in the nerve-centers that are peculiar to the affection. C. Y. Hodge[1] has invited attention to certain changes in nerve-cells during the active exercise of their function, and something of pathologic importance has been added to our previous knowledge by his observations. There are many causes and associated affections that present a variety of morbid lesions, but they are purely incidental. It should be pointed out here that neurasthenia is often found in association with other functional nervous disorders—a fact that has not only caused mental confusion among certain authors, but has also led to the belief among others that as a distinct affection it does not exist. Glénard, in 1888, called attention to the frequent association of profound neurasthenic symptoms with splanchnoptosis.

Etiology.—The causes are divisible into—1, predisposing; and 2, exciting. Among the **former** (*a*) *heredity* heads the list. A clear history of nervousness or morbid irritability in one or both parents (oftener the father) is at times obtainable. Ancestors that were sufferers from gout, rheumatism, syphilis, tuberculosis, and chronic alcoholism, all diseases that exhaust vitality, may have transmitted to their offspring a strong neurasthenic disposition. The latter have inherited a small stock of nervous energy with which to begin life's unceasing struggle.

Other predisposing factors are—(*b*) improper training, mental and physical, (*c*) the character of the mental pursuits, those entailing strains being especially deleterious. (*d*) *Age* and *sex* are not without appreciable effect, most cases occurring between the twentieth and fiftieth years, when the work and worry of life are maximal; they are more frequent in men than in women, and (*e*) disturbances of metabolism accompanied by an abnormally low output of endogenous uric acid (Peck and Thompson).

Exciting Causes.—According to my own observations, *traumatism* has an active potency, though it is probably not the most frequent cause. *Overwork*, at least in America, is responsible for a greater number of cases than any other single factor, and in estimating its effects the relativity of individual nerve-capital must be carefully considered. Associated causes are to be observed in unpleasurable emotional excitement, mental worriment, particularly if dependent upon love-affairs, and sexual excesses. *Abuse of the sexual organs*, excessive venery, masturbation, and the like are powerful in producing neurasthenia. Finally, as stated under Pathology, the condition may be induced by other functional and organic affections (symptomatic neurasthenia).

Symptoms.—The subjective symptoms are protean and varied, and are usually described with great detail, for the patients are, as a rule, exceedingly voluble. Among the more prominent features entering into the symptom-complex of neurasthenia are great *irritability*, *physical fatigue* without adequate reason, even to a feeling of utter exhaustion on rising in the morning, *disturbed sleep*, *headache*, with a sense of weight and constriction, *impairment of memory*, *anorexia*, and

[1] *Journal of Morphology*, vol. v., No. 11, p. 95.

constipation; the patient is very irritable, dispirited, is fearful, and frequently sinks into a state of absolute dejection. Female sufferers—and less frequently males also—may manifest strong emotions, and in such cases the condition presents many points of resemblance to the milder forms of hysteria. The external appearances may be indicative of sound, vigorous health; oftener, however, the physiognomy is worn and anxious.

The **motor phenomena** include, besides readily oncoming exhaustion of the muscular strength under exercise, a variable condition of the tendon-reactions. On the whole, however, they are increased. Muscular *tremors* (fine) are sometimes present, when neurasthenia is the result of trauma or fright (Dercum), and spasmodic contractions of small isolated groups of muscular fibers of the face, trunk, or extremities are observed.

The **sensory** disturbances are varied and sometimes striking. The patient makes constant complaint of feeling "tired" or "never rested," and indeed sometimes betakes himself to bed for this reason. A feeling of "lightness," giddiness, and even true vertigo, may occur and recur, and rarely the latter symptom is wellnigh continuous. The *headache* (previously mentioned) is often wholly dependent upon mental work, since it disappears with the cessation of the latter. Another form of *pain* is a dull aching that may be generalized, though more commonly it is confined to the small of the back and limbs. *Spinal tenderness,* when sought for, may often be elicited over certain circumscribed areas or mere points, and it may be combined with a deep-seated ache or an exacerbating pain ("spinal irritation"). Cutaneous *hyperesthesia* is common, but anesthesia is not found in uncomplicated neurasthenia. Numbness, either spontaneous or as the result of slight pressure, is a conspicuous feature for a variable period upon or near the nerve-trunks, and linked with it there may be a generalized or localized feeling of coolness of the body-surface, or of pricking sensations (formications) and circumscribed subjective sensations of heat and burning.

The *psychic* symptoms grow out of the same fundamental conditions as do the *physical* symptoms—*i. e.* fatigue of the nerve-centers. As would be expected, then, the capacity for sustained mental work is generally lessened, and the power to concentrate or rivet the attention upon any subject as well. The patient is self-centered, sensitive to a degree, easily angered, and is morbidly suspicious. His emotional nature is unstable, and the mental depression (before mentioned) deepens until it approaches true hypochondria.

Insomnia is one of the most constant and troublesome of all the symptoms of neurasthenia. It occurs in various forms. Usually the patient goes to sleep readily, but awakens in a few hours and remains awake either for the rest of the night or until morning is approaching; sometimes there is difficulty in falling asleep; sometimes rest is frequently disturbed. Agrypnia, total inability to sleep, occurs only in the most severe forms of the disease. Disturbances of the organs of special sense are not wanting. The eye presents the most important fatigue-symptoms. *Vision* may be imperfect (blurred) and continuous close use of the eyes may be impossible. There is a lack of power of accommodation and retinal hyperesthesia may supervene. The pupils may be unnaturally large. All forms of *tinnitus* constantly arise in neurasthenia, and may lend so vivid a coloring to the clinical picture that the real nature of the attack is liable to be overlooked. I have recently seen a case of the

sort occurring in a clergyman in whom aural disease had previously been diagnosticated. This symptom, like all others due to neurasthenia, may, however, be associated with genuine organic diseases of the ear (*otoneurasthenia*). Disturbances of taste sometimes appear, but they are of minor importance. *Vasomotor* disorders, such as hot flushes and profuse sweats, commonly arise in consequence of the diminished tone of the arteries; these form quite distressing fatigue-symptoms. Visible throbbing of the superficial vessels and of the abdominal aorta, and rarely also of the veins and the capillary pulse, occur. The *urinary phenomena* may excite particular attention owing to their prominence, and this remark applies especially to the frequent combination of neurasthenia and lithemia (*lithemic neurasthenia*). Oxaluria and transient glycosuria and albuminuria may also be present. The daily amount of urine is often small, and less frequently it is large. The sexual apparatus is weak and irritable, as shown by seminal emissions and incomplete erections, and by premature ejaculation. The fear of becoming impotent often renders the mental attitude of those really potent such as to excite the keenest compassion. The orgasm in the female and the emission in the male are followed by a sense of prostration and mental depression.

The somatic disturbances referable to the heart (palpitation, precordial pain) have been considered under Neuroses of the Heart, and the various gastro-intestinal features in the discussion of Neuroses of the Stomach. Reference has already been made to several clinical varieties based upon the predominance of special and localized groups of symptoms—*e. g.*, when the reigning features are spinal the variety is termed *spinal neurasthenia;* when these are presented by the sexual apparatus, *sexual neurasthenia*, and so on. A further subdivision has recently been made in which the predominant symptoms are various morbid fears, imperative ideas, impulsive acts, and the so-called doubting mania. This has been termed *psychasthenia* (p. 1242). The most obstinate type of neurasthenia is that associated with congenital defects of structure, particularly splanchnoptosis, the so-called Glénard's disease. It does not differ essentially from the ordinary forms, but the gastro-intestinal symptoms predominate.

Diagnosis.—That cases of neurasthenia are misdiagnosed as other conditions, and the reverse, I feel convinced. An important matter at the outset is to avoid confounding the neurasthenic symptoms (secondary) of various local and general organic diseases with the primary form by a careful exclusion of the latter. From *hysteria* the diagnosis is as follows:

Hysteria.	Neurasthenia.
By nature a psycho-neurosis.	A neurosis; often with a pronounced psychical element.
Occurs in individuals presenting a marked hereditary taint.	Occurs as the result of nerve-tire, overwork, and the like in individuals not necessarily presenting hereditary taint.
The onset is frequently abrupt.	The onset is always gradual.
The clinical features are dependent upon an excess of nervous energy.	Is characterized by a notable lack or insufficiency of nerve-force.
Presents the characteristic stigmata, as paralysis and anesthesia in most cases.	These are absent.
Is sometimes accompanied by violent convulsive seizures.	Convulsive seizures never occur.
Neuralgic attacks infrequent and absent.	Neuralgic attacks are very common.
Insomnia is not marked.	Insomnia is very common.

Hysteria, it is to be remembered, may be a complication of neurasthenia, and this association must be distinguished from simple hysteria. Neurasthenia must also be distinguished from psychasthenia (p. 1242). It should also be remembered that neurasthenic symptoms may mark the commencement of various grave physical and mental disorders. Tuberculosis, diseases of the blood, dilated stomach, gastric cancer, gastroptosis, movable kidney, chronic uterine and ovarian disease, paresis, dementia præcox, hypochondria, pellagra[1] (p. 1286), and melancholia should all be considered before the diagnosis of pure neurasthenia is made.

Prognosis.—Neurasthenia is a curable disease if appropriate treatment be commenced before secondary structural changes set in and render the use of the most approved measures of no avail. In long-standing cases deleterious habits (morphinism, chloralism, alcoholism) are sometimes developed and prevent the possibility of a cure. Hysteria (the complication) tends to delay, but does not preclude, recovery.

Treatment.—The first step should be, after locating the major cause or causes, to remove them, or, if this be impossible, to minimize their baneful influence so far as may be. For example, if the conditions have been induced by overwork of the brain, rest for the organ must be procured; if sexual excesses have been the obvious responsible factor, rest for the sexual apparatus is imperatively demanded. In the next place, the mental and moral environment must be conducive to contentment and to wholesome forms of exercise of the mind. In this way the exhausted stock of nervous energy can be often increased by the natural recuperative forces alone. Indeed, successful removal of the essential etiologic influences is in the milder forms followed by prompt recovery. In not a few instances the symptoms disappear as the result of a prolonged sojourn in a suitable climate or by travel for a considerable period with its ever-accompanying change of scene, though it is well in doing so to avoid the din and excitement of large cities. The compulsory rest and complete isolation, combined with the purity of atmosphere, afforded by a sea-voyage sometimes work admirable results. Being occupied by easy and agreeable employment under supervision, as embroidery, basket-making, etc., has been successful. Unfortunately, many subjects suffering with neurasthenia, and particularly males, are either unable or unwilling to arrest the loss of nervous function by ceasing their excessive activities. In the majority of instances, for the reasons above stated, certain other measures —hygienic and medicinal—are to be advised.

To Dr. S. Weir Mitchell belongs the credit of having systematized the "rest-cure" in the management of this disease. This mode of treatment involves (1) physical and psychic rest. The former is obtained by strict confinement to bed, the latter by isolation from all business, professional, household, and family affairs; in severe cases, to the complete exclusion of the family. (2) Hypernutrition. This requires the administration of a quantity of food in excess of the amount required merely to maintain life and repair waste, and is usually secured by feeding at frequent intervals and using nutritious food. (3) The stimulation of the metabolic processes. This is accomplished by massage, passive movements, and electricity. (4) The encouragement and education of the patient. This depends largely upon the tact and authority of the physician and nurse, although graduated and increasing voluntary mus-

[1] Tucker, *Amer. Jour. Med. Sci.*, March, 1912, p. 332.

cular and mental exercises are of some value. In long-standing cases rest should be made absolute if possible, while in the milder forms merely lengthening the hours for sleep or rest in bed often suffices. The amount of rest must be accurately proportioned to the necessity of each case.

The patient is to be put in charge of a properly selected nurse, who will afford agreeable entertainment by suitable conversation and reading under the direction of the physician. In desperate cases the patient should not be allowed to feed himself, must not rise to void the urine or feces, nor even turn in bed without the help of the nurse.

Upon the careful regulation of the *diet* depends, to a large extent, the success of the treatment. This must be modified to suit each individual patient, and, when it is possible, it is desirable to first make a careful quantitative and qualitative examination of the stomach contents.

The two commonest derangements are excess of hydrochloric acid, with retention of stomach-contents and anacidity.

In the former condition the diet must consist of the lighter meats, the more readily digested vegetables, particularly the legumes, the cereals, light desserts, toasted bread, crackers, etc., a liberal amount of fat and milk, and eggs in moderation. I have rarely found it necessary to commence with a very restricted diet of, say milk, but as a rule, the patient can begin on three full meals a day, with a luncheon consisting of milk and toast or crackers, malted milk, chocolate, or cocoa, etc., in the mid-morning and mid-afternoon, before going to bed, and, in severe cases, also once during the night. These cases are also benefited by the administration of moderate quantities of soda after each meal.

In the achylic form the total quantity in each meal should be somewhat less, but from six to eight meals may be given in the course of twenty-four hours. The diet should consist of meats, eggs, the legumes, the acid vegetables, the cereals, bread, fruit, light desserts, a moderate amount of coffee, and a liberal amount of milk. Hydrochloric acid should be given before each meal. In either case the caloric value of the diet should exceed 3000 in the course of twenty-four hours.

In cases in which the gastric analysis is not possible, it is of advantage to commence with small quantities of food, and to increase them rapidly until a very liberal diet is being taken. As a general rule the red meats should not be given too frequently, the coarser vegetables should be excluded entirely, and sweet food should be taken sparingly. Most cases do well on cream taken in quantities of four to eight ounces at a time, sipped slowly, while, at the same time, crackers or toast are eaten.

It is, of course, understood that the patient should never be consulted about the diet. The nurse brings the food at the proper time, served in the most attractive manner possible, and at meal-time always in courses, so that only moderate amounts of food are placed before the patient at one time. The patient should lie perfectly flat for at least an hour after eating.

Passive exercise, *massage*, and *electricity* form an essential part of the "rest cure." Massage should not be commenced until the second or third day. At first it should be continued for a few minutes only, and consist of gentle rubbing or light strokes. As tolerance becomes established, it should be practised for a longer period (about an hour).

Deeper rolling, kneading, and spiral manipulations are then allowable. The direction of the venous blood-current—toward the center of the body from the periphery—is to be borne in mind, and all massage-motions are to be made in the same direction. This measure is to be carried out by the nurse, who should be a well-trained masseuse and thoroughly acquainted with the details of her work. Electricity, like massage, compensates for the lack of exercise. The slowly-interrupted faradic current is to be selected, and the aim should always be to induce satisfactory contractions with the least amount of pain. The current should be applied to the individual muscles, one of the extremities being selected, and the poles applied over the motor points, passing from muscle to muscle until all have been faradized. The time of each sitting should not exceed half an hour. The entire body should also receive the faradic current (rapidly interrupted). A large sponge moistened with salt water is applied at the nape of the neck, and another to the soles of the feet, and the strongest current tolerable is thus used. This process should be continued from fifteen to twenty minutes, and, like the faradization of the single muscles, it is to be repeated at intervals of twenty-four hours. Passive movements should be employed systematically, slowly increasing in duration and extent. They promote circulation and nutrition and are soothing.

Hydrotherapy may be employed in the form of the shower, spray, bath, or pack, and is most efficacious when quickly applied for a few moments and followed by vigorous toweling to reinforce the action of the cold. In insomnia with difficulty in going to sleep, the dry cold pack applied to the spine for an hour is often of benefit. Extreme caution is necessary at the beginning of the application of cold to the surface, since there are neurasthenic subjects who not only fail to receive benefit, but are rendered worse in consequence of a highly sensitive organization.

Although the administration of *drugs* plays a minor part in the management of the rest-cure, in certain cases they are essential, and must not be excluded. Musser lauds very highly ascending doses of the tincture of nux vomica, taken before meals. It is his custom to begin with 10 or 15 minims before each meal, increasing the dose every second day until as much as a dram of the tincture is taken three times a day before meals, providing no untoward symptoms arise. This method of administration, while helpful, has not, in my hands, yielded the brilliant results that are claimed for it. Alkalies or acids are indicated, according to the condition of the stomach-contents. If there is excess of acid the alkalies may be taken after meals; if there is a deficiency of acid, hydrochloric acid should always be given before meals, and in ample doses—from 10 to 20 minims of the dilute acid.

The most important drugs are the laxatives. In the beginning calomel and the salines are often of great value in flushing out the intestines, but during the course of the disease it is the invariable rule never to purge. Of the vegetable laxatives cascara is probably the most useful, but aloin and rhubarb are often of service. Of the saline laxatives the most efficient are certain forms of magnesia and phosphate of soda. In cases where there is considerable alkalinity from 5 to 10 grains of oxide of magnesia in powder form may advantageously replace the soda after meals. Or, 1 or 2 drams of the milk of magnesia may be given in

milk, two or three times a day. Small doses of phosphate of soda in hot water should be given just before breakfast in the morning, being carefully regulated so that only a laxative effect is produced.

If there is pronounced anemia, moderate doses of iron, perhaps combined with arsenic and the bichloride of mercury, are often of service, but must be discontinued if they interfere with the appetite. There is considerable dispute regarding the value of phosphoric acid, or its salts. According to my experience, it is of comparatively little value.

The rest-cure in all of its details should be continued for a period ranging from four to eight weeks. The patient should leave his bed in the most gradual manner, and should sit up for a few minutes only at first, the time being gradually lengthened; soon exercise may be commenced in a like manner and be cautiously increased. During this period of convalescence it is my custom to omit the electric treatment, while the massage is continued at intervals of two or three days for some weeks. After the patient has made some improvement, as evidenced by a large appetite, the disappearance of the most pronounced subjective symptoms, and especially by a substantial gain of weight (twenty to twenty-five pounds—11.3 kgms.), he should be advised to make a change of residence, preferably to the country, the mountains, or the seashore, being guided by the season and the wishes of the patient.

We must increase the activity of the metabolic processes in cases in which the endogenous uric acid output is lowered (400 gr. or less daily). Peck and Thompson[1] advocate the use of the electric-light baths. Cases which are not able to undergo these methods (they being somewhat expensive) must be treated symptomatically, small doses of the bromids, combined with arsenic, if irritability is excessive. Cold douches or baths, with vigorous rubbing in the mornings; tonics, as the glycerophosphates, iron, etc., attention to the gastro-intestinal tract, encouragement, regulation of the employment, as much fresh air as possible, electricity in the form of high-frequency currents, etc.

TRAUMATIC NEUROSES.

Owing to the marked influence of trauma in causing both neurasthenia and hysteria, such cases are often specially classified under the above title. The symptoms are, however, essentially those of either hysteria or neurasthenia, or both combined. The general rules of diagnosis and prognosis in these conditions here apply. It should be remembered that the strain incident to litigation, to which these cases are often subjected, may retard recovery.

PSYCHASTHENIA.

A GROUP of symptoms, until recently classified under neurasthenia, has been given the above title. They consist of obsessions, fears, doubts, undue anxiety, uncontrollable movements, deficient will-power, combined with more or less of the physical symptoms of neurasthenia. As examples of these mental symptoms may be mentioned dread of impending danger, either to family or self, fear of open spaces (agoraphobia), fear of closed places (claustrophobia), of being alone (monophobia), fear of

[1] *Jour. Amer. Med. Assoc.*, Feb. 29, 1908.

crowds, abnormal fear of storms, of wind, etc.; fear of personal defilement (mysophobia); the doubting mania, in which the patient is never certain that he has performed an action correctly; irresistible impulse to touch certain objects (delire du toucher); irresistible tendencies to repeat continually certain words (onomatomania), to count a certain number of times before performing an action (arithmomania), etc. The patient is conscious of the absurdity of these actions and feelings, but cannot resist them. Epileptiform convulsions may also occur. The prognosis is doubtful, but treatment similar to that recommended for hysteria may achieve good results.

OCCUPATION-NEUROSES.

Definition.—Conditions in which the performance of certain habitual coördinated movements is prevented by the development of cramp, tremor, paralysis, or pain.

The **pathology** of this condition is unknown. It is probably purely functional, and the discovery of appreciable lesions is not to be expected, though nodular thickening of the peripheral nerves has been described in a few cases.

The **etiology** is various. Those following any occupation requiring the continuous repetition of fine, coördinated, muscular movements, as sewing, type-writing, playing musical instruments, telegraphing, and writing, may be affected. Writing is the most common cause, and is known as scrivener's palsy, or writer's cramp. It is the form here particularly described, although the symptoms due to other causes are similar. Males are far more frequently affected than females, the condition usually occurring in early adult life, although children are not exempt. Gowers lays great stress upon improper methods of holding the pen, particularly those in which most of the writing is done from the wrist; that is, with the muscles of the forearm and hand. As scrivener's palsy occurs sometimes in those that write properly, and as a similar condition is not uncommon in other occupations, it seems unlikely that this is the most important cause. A person with a neurotic temperament is far more apt to be affected by the disease than one with a normal nervous system; we, therefore, frequently find it associated with hysteria, neurasthenia, or great bashfulness, and not infrequently it is possible to elicit a neuropathic heredity in the family history. It is also met with in certain other nervous diseases (epilepsy, locomotor ataxia—in the early stage). Often the patients admit that at the time the disease developed they were suffering from severe anxiety.

Symptoms.—**Motor.**—When the patient attempts to write there is usually a cramp of the flexor muscles of the forearm, so that the pen is held more or less rigidly, and it is almost impossible to control its motions. Less frequently there is a cramp of the extensor muscles, so that the fingers are spread and it is impossible to hold the pen at all. Sometimes there is a sudden twitching, and the pen may be thrown altogether out of the hand. The spasm is nearly always tonic in character, but often it is associated with a fine tremor, and at times there are clonic movements. In some cases, and particularly those occurring in patients showing hysteric stigmata, there is a coarse, irregular tremor, most

marked when the patient is under observation. Paresis is frequently associated with the cramp, so that the arm soon becomes tired and it is almost impossible to write. This fatigue may in a few moments progress to almost complete paralysis of the arm, but, curiously enough, both fatigue and paralysis disappear as soon as some coördinated movement other than writing is undertaken.

Sensory.—Pain is very common, and is neuralgic or cramp-like in character, being referred either to the muscles, bones, or joints. In intensity it varies from a dull ache to the most excruciating burning, and may form the only symptom, the muscles performing their work perfectly. At times it is sharply localized to one particular joint, affecting either the metacarpal bones or the fingers. Quite often the patient complains of a tingling or burning sensation in the limb, or it may be numb and the hand feels, when writing, as if a heavy weight were attached to it. Often there is tenderness either of the muscles or the nerves, which may be localized in certain points. In very severe cases *vasomotor disturbances* occasionally occur. The disease ordinarily commences slowly. At first the subject notices that the handwriting is not quite as perfect as before, a stroke occasionally going astray; later distinct spasms appear, and these are finally associated with pain.

The **diagnosis** is usually easy. Care must, however, be taken not to call every disturbance of writing writer's cramp; thus in paralysis agitans, in slowly developing hemiplegia, in multiple sclerosis, paresis, and in locomotor ataxia disturbances of writing frequently—in fact, almost invariably—occur. Moreover, those cases in which hysteria or neurasthenia seems to be at the bottom of the trouble should be carefully differentiated from those that are apparently idiopathic.

The **prognosis** is rather unfavorable, though complete cure is sometimes attained.

The **treatment** consists first in a total cessation of writing; if this is impossible, various mechanical devices may be employed to use another set of muscles or the old ones rather differently, such as a thick penholder or one constructed with supports for the fingers. Local treatment of the arm in the form of electricity should be advised; the anode of a constant galvanic current of medium intensity should be placed over the sensitive points on the nerves and over the bodies of the muscles. The wire brush employed, with the rapidly interrupted faradic current, to stroke the painful nerves and muscles, affords great relief. Massage, and particularly careful and systematic exercises, are also of great value. At the same time, the general condition of the patient must not be neglected. In those associated with neurasthenia a treatment appropriate to this condition should be employed.

ACROMEGALY.

(*Giantism.*)

Definition.—A disease first recognized and described by Marie, and characterized by a progressive and peculiar enlargement of the face and extremities.

Pathology.—Those cases that have been examined *postmortem* have shown, as the most constant change, an enlargement of the pituitary body,

with a corresponding dilatation of the sella turcica, and a persistence of the thymus gland. Alterations may be found in other ductless glands, especially the thyroid, which may be either goitrous or atrophied. In some cases the pituitary may not be enlarged macroscopically. The lips, tongue, and trachea are usually considerably enlarged, and the sexual organs may either be hypertrophied or atrophied, the latter condition being more common in the uterus and testicles. The bones of the extremities and face are thickened, apparently chiefly as a result of hyperplasia of the spongy portion, and Klebs has shown that the peripheral vessels, particularly those in the affected bones, are also larger. Occasionally there are hypertrophy of the heart and enlargement of the spleen and liver.

Acromegaly is due to an increased activity of the anterior lobes of the pituitary body occurring after epiphyseal union has taken place. If it occurs before this (in childhood) *gigantism* is the result. In this changes in the pituitary will be found similar to those occurring in acromegaly (p. 1244).

Cushing[1] has described cases in which with overgrowth are associated symptoms of posterior lobe insufficiency, *i. e.*, adiposis, increased sugar tolerance, polyuria, polydipsia, subnormal temperature, dry skin, loss of hair, epileptiform disturbances, etc. He believes such cases are due to anterior lobe hyperplasia associated with posterior lobe hypoplasia.

Both sexes are about equally affected, and the disease ordinarily commences in adolescence.

The earliest **symptom** is usually an increase in the thickness of the fingers and toes, so that rings, gloves, and shoes are too small and can no longer be worn. This *enlargement* is chiefly in thickness, although there is also a certain amount of increase in length. Both the soft and hard parts are affected. The nails are flattened, longitudinally ridged, and more friable (*spade-like hand*). The *face* becomes considerably enlarged; the supraorbital ridges project, giving rise to a rather simian aspect; the nose becomes broader and longer; the cheek-bones project; but the most positive characteristic is the enormous enlargement of the lower jaw, so that it becomes broader and prognathous, and the lower teeth can no longer be brought in apposition with the upper. The *spinal column* is ordinarily kyphotic, the change affecting the upper dorsal and cervical regions. Frequently there is also an associated scoliosis. The rest of the skeleton remains unaffected for a long time; finally, changes may be observed in the clavicles, sternum, ribs, pelvis, and particularly in the patellæ. The *skin* sometimes shows slight pigmentation; the hair is rough and may become thinner; the *muscles* occasionally exhibit increased electric excitability, and less frequently there is muscular atrophy with reactions of degeneration. The lips, tongue, and tonsils are usually enlarged, and the larynx is increased in dimensions, so that the *voice* becomes deep and rough; this is a very characteristic symptom in women. Ordinarily, an area of dulness can be detected in the upper part of the sternum that has been ascribed to the persistence of the thymus gland. The *tendon-reflexes* may either be normal, diminished, or abolished. They are never exaggerated. The *urine* is increased in amount, and glycosuria is often present. The secretion of *sweat* is also greatly increased. The subjective symptoms consist of severe intermittent or continuous *head-*

[1] *Amer. Jour. Med. Sci.*, Mar., 1913, p. 313.

ache and of a *diminution of the visual power*. There may be paresis of the third nerve, giving rise to external strabismus, and sometimes to *temporal hemianopsia* as a result of pressure upon the central part of the chiasm by an enlarged pituitary body. Sometimes late in the disease there are occasional momentary general tremors. The patients often present polyphagia and polydipsia. Neuro-retinitis and subsequent atrophy of the optic nerve may also occur. The mental condition is affected, and there are usually great apathy and diffidence (perhaps explicable by their changed appearance), loss of memory, and somnolence. Symptoms of either myxedema, exophthalmic goiter, syringomyelia, and epilepsy may co-exist.

Diagnosis.—In the later stages the appearance is characteristic, and acromegaly can then hardly be confounded with other diseases. The peculiar enlargement of the extremities, the oval, prognathous, and distorted face, the deep, rough voice, the more or less pronounced pigmentation of the skin, the wasting of the muscles, and the profound cachexia give a perfect clinical picture. In those cases in which the cachexia has become extreme there are from time to time peculiar tremors or spasms of the body.

Differential Diagnosis.—In the earlier stages the disease is most easily confounded with the *hypertrophic pulmonary osteo-arthropathy of Marie*. In this both hands and feet are greatly enlarged; but the fingers are club-shaped, the face is not involved, and there usually exists some chronic pulmonary complication. In a case that I observed there were bronchiectasis and bronchorrhea. From *osteitis deformans* it may be distinguished by the fact that in this condition chiefly the long bones of the limbs and the flat bones of the skull are hypertrophied and very painful. *Elephantiasis* may be distinguished by the fact that it attacks the lower limbs, does not involve the bones, and the skin presents a granular or a nodular appearance. From *arthritis deformans* acromegaly may be distinguished by the fact that the disease is painful, and is associated with great deformity of the joints, the face ordinarily escaping. The following table (after Dercum) will serve to distinguish two diseases that are apt to be confounded with one another:

Acromegaly.	Myxedema.
Occurs most commonly in early adult life.	A disease of mature life—forty to fifty years.
In males and females equally.	Five times as frequent in females as in males.
Enlargement of the bones characteristic.	No enlargement of the bones.
Marked prognathism of jaw and flattening of cheeks.	Face full-moon-shaped.
Skin brownish-yellow; hair coarse and unwieldy; nails short and striated.	Skin pale, waxy, shiny, and boggy; hair falls out; nails not affected.
Fingers symmetric and sausage-shaped.	Fingers clubbed at the end.
Administration of thyroid extract is of the smallest benefit.	Thyroid treatment of the greatest benefit.

A skiagraphic examination is of great value in doubtful cases, as enlargement of the sella turcica will usually be found.

The **prognosis** is hopeless for cure and doubtful for duration. The disease is progressive, although it remains stationary for a longer or shorter period. Retrogression never occurs. Ordinarily, the patient

dies of some intercurrent condition; although death may be due to the cachexia of acromegaly itself. Life, however, may last for twenty years after the appearance of the first symptoms.

Medical treatment of the condition has proved unavailing. Extracts of both thyroid and pituitary glands have been used with not encouraging results. Removal of the pituitary gland, if the seat of tumor, has been done with good results in some cases. The cephalalgia can be more or less completely controlled by antipyrin or caffein. Phosphorus, mercury, the iodids, and arsenic have been useful in some cases.

ADIPOSIS DOLOROSA.

THIS disease was first described by Dercum, of Philadelphia, in 1888. It may be defined as a condition in which masses of fat are deposited irregularly in the subcutaneous tissue of the body, with tenderness and spontaneous pain in these masses, and derangement of the menstrual functions. Several cases, including the one first described by Dercum, have been examined postmortem, and a variety of changes have been found. The fat is usually denser than ordinary fat, due to the presence of a considerable amount of fibrous connective-tissue trabeculæ. The thyroid glands are sometimes small and sclerotic, and, in the case recorded by Burr, there was a tumor of the pituitary body. The cutaneous nerves show a moderate amount of degeneration, sometimes associated with interstitial neuritis. The main nerve-trunks are usually normal. Hemolymph-glands have been found in the fatty tissue.

The **etiology** of the condition is unknown. It has been ascribed to an early climacteric, and to the changes in the thyroid gland, but it is not understood how either condition could give rise to the clinical feature of adiposis dolorosa. It occurs almost exclusively in women.

Symptomatology.—Some time in adult life the patient begins to grow stout. This condition gradually progresses, and the patient notices that the fat is more or less irregularly distributed, appearing first in one and then in another part of the body, and that in the places in which it appears there are severe pains of a burning, shooting character. Finally, the masses of fat become huge; as a result of their weight they become pendulous; they are elastic, give an indistinct sense of fluctuation, but do not pit on pressure. The skin remains soft and flexible as normal. There are no distinct evidences of muscular degeneration, but the patient becomes weak and indisposed to physical exertion. There is no disturbance of the psychic functions, but the mental processes are sluggish. The cutaneous sensibility may be slightly altered, areas of anesthesia, or particularly of hypesthesia, being found in various parts of the body. The knee-jerks are usually lost, but Romberg's symptom is not present. Death occurs as a result of some intercurrent affection.

The **differential diagnosis** is to be made from simple obesity and from myxedema. From simple obesity it differs by the fact that the fat is firmer; it is irregularly distributed; nodules appear and disappear in the skin; and particularly by the sharp pains in the fatty masses. From myxedema, by the absence of mental changes, and of tetany, and by the presence of the pains in the fatty masses. The distinguishing test is the failure to respond to thyroid medication. (See also pp. 1186 and 1245.)

The **prognosis** is hopeless for cure, but the duration of the disease is often greatly prolonged. Dercum's original case was under observation for eleven years, and then died of fatty degeneration of the heart.

Treatment is unavailing. The administration of thyroid substance appears to be of no benefit. The pains must be controlled with anodynes, employing at first the coal-tar analgesics, particularly phenacetin, which must be used in small doses on account of the chronic nature of the case, and if this is insufficient, morphin must be administered.

AMAUROTIC FAMILY IDIOCY.

TAY and Sachs have described, independently, a most extraordinary disease of the central nervous system which is characterized by the occurrence, a few months or a few years after birth, of marked impairment of intelligence, and gradually progressive loss of vision. The pathology of the disease is not known. Degeneration of the cells, perivascular accumulation of round cells, and some degeneration of the fibers in the central nervous system have been found. The etiology of the disease is not understood. It is usually hereditary or familiar, that is to say, several children in one family are sure to be affected. As it occurs in early life direct inheritance is, of course, impossible, but children of the ancestors have sometimes suffered from the same condition. It is also racial; all the cases hitherto recorded, with one doubtful exception, having occurred among Jews.

The **symptomatology** is as follows: The child at first develops normally, appears healthy and intelligent. Usually in the latter portion of the first year or in the early part of the second, its mother observes that it does not notice as well as formerly; that it appears to be weaker and less intelligent. It gradually becomes more and more idiotic until it is a complete imbecile, uncleanly in its habits, and at the same time the blindness progressively increases. This blindness appears to be due to a degeneration of the retina, the earliest sign being a bluish discoloration or spot in the region of the macula. The reflexes are usually greatly increased and sensation becomes generally blunted.

The **differential diagnosis** is to be made from other forms of idiocy occurring early in life. The race, the familiar type of the disease, and particularly the progressive blindness, with the peculiar changes in the eye-ground, usually suffice to determine the character of the disease.

The **prognosis** is hopeless. The children die in the course of from three to five years.

Treatment is of no avail. Prophylaxis has been attempted, particularly by keeping the mother in good condition before and during pregnancy, and by careful attention to the health of the child during early infancy. As only a certain number of children in each family are affected, it is impossible to determine how effective these measures are. They should at least be employed in all cases in which one member of the family has had the disease. Antisyphilitic remedies are injurious.

VII. VASOMOTOR AND TROPHIC DISORDERS.

ANGIONEUROTIC EDEMA.

(*Acute Circumscribed Edema of the Skin; Intermittent Angioneurotic Edema; Giant Urticaria.*)

Definition.—A disease characterized by the appearance of an edematous swelling of the skin or mucous membranes. In general it is not accompanied by constitutional symptoms.

The **pathology** of the disease is obscure. It is supposed to be due either to venous stasis or to some nervous influence upon the lymph-channels, causing them to exude liquid. No lesions have as yet been described.

Etiology.—Neuropathic heredity appears to have some influence upon the disease, but nervous manifestations in the patient himself are more important. Occasionally the condition follows infectious diseases or severe hemorrhage. The most important exciting causes are cold and emotional disturbances. The disease occurs most frequently in males, and almost exclusively in early adult life.

Symptoms.—The *edema* usually appears suddenly, is sharply circumscribed, and the skin of the affected area is slightly elevated and reddened, or else somewhat paler than the surrounding tissue. It does not pit on pressure. Ordinarily, subjective symptoms are absent; occasionally there are slight *paresthesiæ*. The edema may appear in any part of the body, but usually it is most common on the backs of the hands or legs and in the face, especially the eyelid. Occasionally it may appear upon the mucous membranes either of the lips, tongue, or glottis; in the latter situation it sometimes produces severe dyspnea, and at least in one case it has caused death. Its presence has also been suspected in the mucous membrane of the gastro-intestinal tract. Ordinarily the patient has no symptoms whatever of disease; occasionally, however, there are severe colicky pains and sometimes vomiting. In one case hematuria was observed, and in another hemorrhage from the swollen gums; of course, in the latter case the diagnosis was doubtful. Certain writers have noted eosinophilia with, or without, leukocytosis. The patient may exhibit a certain degree of anxiety during the attack. Ordinarily the swelling persists a few days, and then disappears, but relapses are exceedingly common, and may recur very frequently for many years.

The **differential diagnosis** has to be made from urticaria, to which it bears a great similarity. According to Osler, giant urticaria is the same disease.

The **prognosis** is of course favorable for life; for cure it is more doubtful, as the disease is sometimes exceedingly obstinate.

The **treatment** consists of rest, the use of tonics particularly directed to the nervous system, and the correction of any gastro-intestinal disorder. Strychnin has proved very valuable. Atropin during the attack is also of service. If the larynx is affected, scarification of the edematous areas and even tracheotomy may be required.

RAYNAUD'S DISEASE.

(*Symmetric Gangrene.*)

Definition.—A condition apparently of vasomotor nature, affecting symmetric parts of the body, and chiefly the tips of the extremities.

Pathology.—Clinical and pathologic studies seem to show that this condition, as well as others to be mentioned under diagnosis and which are closely related, are dependent upon disease of the peripheral blood-vessels, causing deficiency of the blood-supply. This may be a vasomotor spasm or some form of endarteritis. They may also be associated with organic disease of the spinal cord, especially tabes dorsalis and peripheral neuritis.[1]

The **etiology** of the condition is obscure and complex, largely, no doubt, because a number of different conditions have been confounded under this designation. The disease occurs in children and in neurotic women, less often in men. A neuropathic heredity seems to predispose to it, and occasionally it exists in connection with other nervous diseases, as epilepsy, migrain, hysteria, and mental disorders. The occurrence of paroxysmal hemoglobinuria has led to the suspicion that malaria is an etiologic factor. I am not aware, however, that plasmodia have been found in any case, and the asserted good results following the administration of quinin are insufficient to establish the contention. Syphilis and various other infectious diseases have also been mentioned as etiologic factors. The most important exciting cause is exposure to cold, although attacks may also be brought on by severe emotional disturbances.

Symptoms.—The disease presents three grades of severity: first, anemia or local syncope; second, cyanosis or local asphyxia; and third, gangrene. *Local syncope* consists in a vasomotor spasm in one or more extremities, the fingers being most frequently affected, and rarely more than one at a time. They become white, almost waxy in appearance, cold, and hard to the touch, and they may be either dry or covered with a cold perspiration. The finger is perfectly numb, but severe neuralgic pains may be felt in the arm; if the skin be pricked with a pin, no blood flows. Ordinarily this syncope disappears gradually, the reaction being accompanied by tingling and formication in the affected digit, which ultimately returns to a normal condition. *Local asphyxia* is a further stage of this condition: in this the finger is blue and swollen, and there is a sense of discomfort that is apparently due to the stretching produced by the engorged veins. This cyanotic condition may also affect the ears, toes, and the tip of the nose, and, like the preceding stage, it may disappear without leaving any trace of its existence. Patients that have reached this stage seem to be more liable to a recurrence upon slight exposure than those who only present local syncope. The attacks are more likely to recur constantly in the same digit, and not to appear first in one and then in another. During the existence of this stage a not infrequent associated symptom is *hemoglobinuria*: this is especially apt to occur in children, and has led to the suspicion of malarial influence. In some cases, when hemoglobinuria is not found, the urine contains an excess

[1] Barker and Sladen, *Jour. Nerv. and Ment. Dis.*, Dec., 1907, p. 745; Sachs, *Amer. Jour. Med. Sci.*, Oct., 1908, p. 560.

of urates. If the attack lasts for several days, *trophic changes* take place in the finger-nail, giving rise to a transverse ridge, which persists until that portion of the nail has grown beyond the end of the finger. If local cyanosis, however, continues sufficiently long, *gangrenous* changes take place. These appear first as small black spots or vesicles filled with serum upon the end of the fingers or about the root of the nail; these gradually slough off, leaving a small ulcer that may slowly cicatrize. Often patients subject to recurrences of the disease show a number of cicatrices on the ends of the fingers, or if the ears are affected there may be slight shrivelling of their edges. The gangrene, however, may be more severe, in which case the distal phalanges of the affected fingers may become black or dark red, covered with blebs, and finally mummified. The line of demarkation then forms, and ultimately the gangrenous portion falls off, leaving an ulcerated stump that slowly cicatrizes. This form may not be limited exclusively to the hands and feet or ears, but symmetric patches sometimes appear in the skin of the breast. During the time that the gangrene is present the patients suffer from excruciating *pains* in the limbs that interfere with sleep, often causing transient melancholia, and seeming, more than the gangrene itself, to depress the general condition. Fever is rarely present; sugar is sometimes found in the urine, but not constantly.

Diagnosis must be made from erythromelalgia; acroparesthesia (p. 1253); acrocyanosis, in which condition we have cyanosis of the extremities, often associated with gangrene and ulceration, but which differs from Raynaud's disease in not being paroxysmal; and intermittent claudication, which is characterized by muscular cramps, numbness, and transient loss of motor power dependent upon exertion (p. 1145). As has been said, all of these symptom groups are closely related, and a sharp line of demarcation cannot always be maintained. The condition of the posterior tibial and dorsalis pedis arteries should always be determined, an absent or diminished pulsation being frequently found. Cases of this type are especially prevalent among the Russian Jews, and the feet are mostly affected. They are characterized by more or less constant pain, constant coldness of the feet with local syncope, tenderness of the calves, diminution of sensibility in the feet, followed by local asphyxia and gangrene. The pain is usually relieved by allowing the feet to hang down. Buerger has applied the name thrombo-angiitis obliterans to them.[1] Diabetic gangrene with neuritis may be confounded, but examination of the urine should make the diagnosis clear.

Senile gangrene differs in its mode of onset and by its occurring in old age. Tabes dorsalis, in which these symptoms may occur, can be distinguished by the occurrence of other characteristic symptoms of that disease. The same may be said of syringomyelia. Peripheral neuritis may be sometimes difficult to exclude, especially the form known as senile neuritis, due to arteriosclerosis. If tenderness over the nerve-trunks and muscular weakness and atrophy are present, neuritis undoubtedly exists.

Leprosy may also have to be considered. Scleroderma (p. 1254) is also related to the vasomotor neuroses above mentioned, and may be mistaken for them.

[1] *Amer. Jour. Med. Sci.*, Jan., 1910, p. 105.

The **prognosis** is favorable unless there is some arterial disease. Ordinarily they become in time less frequent and ultimately disappear, but in a few cases the tendency to recurrence is obstinate.

The **treatment** consists of improvement in the general condition during the intervals. During the attack the most effectual measures are a mild massage, the use of local lukewarm baths, and electricity very cautiously applied, by the application of the anode to the spine and the cathode placed in a vessel containing water into which the affected part is put. Nitroglycerin is sometimes beneficial. For the relief of the local syncope Cushing has devised a plan of treatment which has been successful. It consists in applying an elastic bandage to the limb, tight enough to stop the arterial circulation for several minutes; it is then loosened, when the circulation will usually return. In obstinate cases it may have to be repeated. Pilocarpin also has been employed with good results. If the pains are very severe, they must be combated by morphin—although gangrene may occur at the site of the injection—administered hypodermically, if necessary. Sleep should be obtained by means of narcotics. The gangrenous parts should always be carefully protected by a local dressing, and surgical intervention in the form of amputation may be required. If so, it must be done high or a return of symptoms may occur.

ERYTHROMELALGIA (*Weir Mitchell*).

(*Paralytic Vaso-motor Neurosis of the Extremities.*)

Definition.—A disease characterized by paresthesia, redness of the skin, and by pain, usually in the toes and heels, associated with more or less severe general disturbances.

The **pathology** is doubtful—arteriosclerosis of the blood-vessels in the affected limb has been found, but the disease appears to be due to some disturbance of the vasomotor centers or nerves.

Etiology.—It may occur in association with various forms of spinal cord disease (see Raynaud's disease).

Symptoms.—The earliest symptom, as a rule, is the occurrence of severe *pains in the feet*. Objectively, there are swelling and reddening of the skin, and the sensitiveness is so severe that the patient is unable to walk. The attacks occur more frequently during the summer months, and are always aggravated by exposure to heat or a vertical position of the limbs. Ulceration may occur.

The **diagnosis** is often difficult, the condition being confused with inflammation of the foot. Operations have frequently been performed upon these cases. A characteristic feature of the condition is that the redness and pain are excited by allowing the feet to hang down, and disappear when they are elevated (see also Raynaud's disease). It may occur in the course of hemiplegia and in some organic diseases of the spine, and these should be excluded.

The **prognosis** as to relief is bad; often the disease will recur at irregular periods for a number of years. The attack can usually be cut short by plunging the limb into ice-cold water.

Treatment.—This should always be tonic, and employed during the intervals; massage, hot and cold douches, and the faradic current may be used upon the affected extremities. The pain may call for anodynes. Resection of the long saphenous and musculocutaneous nerves and stretching of the plantar nerves have been done with success. It has also caused gangrene.

ACROPARESTHESIA.

(*Spastic Vasomotor Neurosis of the Extremities.*)

Definition.—A disease characterized by abnormal sensations in the hands, slight vasomotor disturbances, and slight stiffness of the fingers.

The **pathology** and **etiology** are not understood. Possibly the condition is due to some disturbance of the peripheral nervous system. It occasionally occurs after injury or as a result of prolonged exposure to cold, hence is common among laundresses. It is more frequent among women than men, and usually develops in middle life.

The **symptoms** consist in the more or less sudden development of *formication* and *tingling* or *numbness* in the fingers and finger-tips, usually bilateral, but sometimes occurring only on one side. Less frequently the toes are affected. These pains are more severe in the night and early morning, and worse in summer or after exposure to heat. The vasomotor disturbances are variable. Sometimes nothing can be observed, and sometimes the extremities are bluish and cold, sometimes pink and warm. Sensibility is rarely affected. In some cases, however, there is considerable hyperesthesia; in others moderate anesthesia. In a few cases there is stiffness of the hands. Slight *trophic disturbances* have been reported in a few cases. The attacks may last from a few minutes to several hours, and may recur frequently or only at considerable intervals. Usually during the attack the abnormal sensations are continuous, but occasionally they are intermittent in character. The condition known as *tender toes*, that occasionally occurs after an attack of typhoid fever, is probably a form of this disease. It is ascribed to the Brand treatment, but incorrectly.

The **diagnosis** is usually easy. Care should be taken, however, not to confuse these acroparesthesiæ with commencing locomotor ataxia, tetany, or hysteria. In Raynaud's disease cold increases the intensity of the symptoms.

The **prognosis** is, in general, favorable, the disease usually disappearing after some months; sometimes, however, the condition is obstinate.

The **treatment** is rather unsatisfactory. Laundresses should be advised to adopt some other vocation. Local stimulation with the faradic brush has sometimes been of value, and hydrotherapy may also be employed. At the same time, the patient should be given tonics, particularly if anemia is present. Salicylates seem to be of service in some cases. Alkaline washes are almost a specific for the tender toes. Saturated solutions of sodium bicarbonate should be employed.

MERALGIA PARÆSTHETICA.

(*Bernhardt's Disturbance of Sensation.*)

Definition.—A disease characterized by paresthesia and disturbance of sensation on the outer side of the thigh, in the region supplied by the external cutaneous femoral nerve.

Pathology.—Nawretsky has examined one case, and found chronic interstitial neuritis. There is reason to believe that this is not always present.

Etiology.—This is very various; some of the cases have been preceded by injury, excessive exercise, or infectious disease. Alcoholism, constipation, and pregnancy are also common predisposing causes; cold douches have been blamed in several instances. Sometimes the disease is hereditary. The exposed situation of the nerve is supposed to render it more liable to this peculiar disturbance.

Symptoms.—These are of two varieties: First, the *paresthesiæ*. There may be burning, tingling, or stabbing pains that are severe enough to disable the patient; or there may be only a feeling of cold or numbness. Second, the *sensory disturbances*. These vary from slight hyperesthesia to total anesthesia. The different senses are not always equally involved; pain, temperature, and electro-cutaneous sensibility being usually more profoundly affected than the others. Frequently both thighs are affected. There is often a tender point just inside the anterior superior spine of the ilium.

The **diagnosis** is easy.

The **prognosis** is doubtful. Some of the cases recover rapidly, but the majority become chronic.

Treatment.—But little can be done. Locally, the dry brush seems to do good in some cases, and the general health should be improved if possible. In aggravated cases a portion of the nerve may be excised.

SCLERODERMA DIFFUSUM.

Definition.—A peculiar hardening of the skin, with areas of pigmentation and depigmentation, associated in the more advanced stages with trophic lesions, muscular atrophies, and affections of the bones.

Pathology.—The affected skin is characterized by an increase of the connective tissue and of the elastic fibers, and by a narrowing of the vessels as a result of perivascular infiltration.

The **etiology** is not clear. Some of the cases are associated with joint-affections that resemble those of chronic rheumatism; others follow exposure to a very low temperature. The presence of trophic lesions in the skin and the development of myopathies lead to the supposition that it is properly classed with the trophic neuroses. The disease usually

occurs in middle life, although cases have been observed among children. Women are more frequently affected than men.

Symptoms.—Three stages are recognized: *First*, a rather dense edema. *Second*, a true sclerosis, in which the skin appears thicker, with an absence of the normal folds; it becomes firm and hard, so that it cannot be pinched between the fingers and lifted from the flesh. Moreover, there are always *pigmentary changes*, certain parts being darker than normal, while others become a dead white, appearing almost as if composed of alabaster. The disease, as a rule, attacks first the upper portion of the body—*i. e.* the face, neck, hands, and arms, or the surface of the thorax, and is most pronounced in those regions where the bones are subcutaneous. The *diminished elasticity* considerably interferes with the movements of the body. If the neck is affected, it is difficult to turn the head; if the skin over the joints is involved, their normal flexion and extension cannot be perfectly performed. The subjective sensations are those of tension, the patient complaining that the skin has become "too small" for him. If any forcible action is attempted, there is severe pain, accompanied by slight tears in the skin. The skin is paler and cooler than normal, and the slightest exposure to cold causes great discomfort and cyanosis. The secretion of sweat may be normal, but is usually diminished. Tactile sensibility is unimpaired. The *third stage* is that of atrophy; the skin becomes thin as paper; the other symptoms, however, remain as before, except that the secretion of sweat is abolished and *ulcerations* appear that either heal slowly or not at all. In addition, there are muscular atrophies associated with contractures. Often there is considerable *atrophy of the bones*, or there may be a development of exostoses from the periosteum (sclerodactylia). Occasionally the end-phalanges of the fingers undergo a process of gangrene that is similar, in some respects, to that of Raynaud's disease. *Chronic joint-affections* may also be observed in this stage, particularly of the fingers (see Morphea).

The **course** of the disease is variable. Usually it develops slowly and lasts for many years.

The **diagnosis** is usually easy, though occasionally it has been confused with *Addison's disease* on account of the excessive pigmentation. There is, of course, some resemblance to *Raynaud's disease*, although the condition of the skin itself is very different. In the atrophic stages it may be confounded with xeroderma pigmentosum.

The **prognosis** is always doubtful. In the later stages the patients become emaciated, and pass into a cachectic state, in which death may occur. Pulmonary complications may develop. Complete cure may, however, occur, and particularly in cases that have a rapid course.

The **treatment** is unsatisfactory. The unpleasant tension of the skin may be somewhat diminished by ointments and massage; warm water or steam baths may also give considerable relief. The most important thing is to maintain the general condition of the patient by tonics and a change of climate. Sodium salicylate has been recommended, but is probably valueless. Thiosinamin hypodermically may prove to be of service.

MORPHEA.

(*Scleroderma Circumscriptum.*)

THIS disease consists of the development of *small areas of sclerosis* that are distinctly related to the distribution of the nerves. These areas are round or oval, brownish or violet in color, and as they increase in size there develops in their centers more or less sclerosis. In these sclerotic areas there are often punctiform collections of pigment, the hairs fall out, and superficial ulcerations may be present Occasionally they may go on to atrophy of the skin. There are no constitutional symptoms.

The **diagnosis** is usually easy.

The **prognosis** as regards life is favorable; as regards cure it is doubtful.

The local **treatment** is the same as for the diffuse form of scleroderma.

AINHUM.

THIS is a disease characterized by an enlargement of the little toe and the formation of a line of demarkation at its base.

The **pathology** is not known, but it appears from a Röntgen-ray picture that the bones are absorbed. There is some dispute as to whether it is one of the manifestations of leprosy or not. At any rate, it does not appear that typical lepra bacilli have been found.

Etiology.—The disease may occur in childhood or early adult life, and is most common in negroes. It occurs almost exclusively in tropical regions—*e. g.* Brazil and Syria.

The **symptoms** of the condition consist in the formation of a *furrow* at the base of the little toe of one of the feet. This grows deeper and deeper until spontaneous amputation has occurred. Rarely the other toes on the same foot become progressively involved. Certain *vasomotor disturbances* may be observed; the foot is usually swollen, bluish-red, and cold; sometimes the other foot may exhibit similar changes without the formation of furrows at the base of the toes. There is some *diminution of sensation* to touch, temperature, and electricity, and ordinarily the patient complains of vague pains in the limbs.

The **diagnosis** is to be made from leprosy, with which, indeed, it may be identical, and congenital amputation: the latter only occasions difficulty when the disease commences in early life.

The **prognosis** is favorable to life, but the disease is usually slowly progressive.

No effective **treatment** has been discovered, but the parts should be protected against injury, and the patients may be given tonics and anodynes as required.

PROGRESSIVE HEMIATROPHY OF THE FACE.

(*Progressive Facial Atrophy.*)

Definition.—A rare disease, characterized, as its name would indicate, by a progressive atrophy of one-half of the face, stopping sharply at the middle line, and in the severer forms involving the skin, muscles, and bones.

The **pathology** of the condition is unknown. Rarely symptoms indicating inflammation of the cervical sympathetic, such as dilatation of the pupil or flushing, have been present, and symptoms indicating inflammation of the trigeminus have been equally infrequent. Mendel, however, has reported a case in which he found chronic interstitial neuritis of the branches of the trifacial, and other cases have been reported in which the Gasserian ganglion was diseased. Microscopic examination has shown a disappearance of the subcutaneous fatty tissue and a general atrophy of the elements of the skin itself, often associated with the presence of an abnormal quantity of pigment. As a rule, the vessels are relatively enlarged.

The **etiology** is unknown. The condition usually commences early in life and shows no predilection for either sex. An hereditary tendency does not appear to exist, but the disease occurs frequently as a complication of, or rather in connection with, other neurotic conditions. Of these the most frequent are neuralgia, migrain, epilepsy, and mental disorders; less frequently, tic convulsif and chorea, particularly if the latter affects the muscles of the jaw and tongue. Occasionally it has been recorded as occurring in patients suffering from locomotor ataxia or multiple sclerosis. It does not appear, however, that progressive facial atrophy has any anatomic connection with these conditions. In a few cases the disease has been preceded by an injury to the skull or face, and in others it has followed an acute infectious disease. Ordinarily it occurs in early life—*i. e.* between the tenth and fifteenth years—and in these cases it usually progresses to the most severe type.

The earliest **symptom** is a flattening of the skin on the affected side, constituting the lightest form of the disease, which may remain stationary at this point; if, however, it progresses, the muscles and bones also become involved, so that the affected half of the face is distinctly smaller than the healthy side. The objective changes that take place in the skin are the development of *white spots* in which the pigment has disappeared, and which have the appearance almost of scar-tissue, or, what is more commonly the case, of an increase in *pigmentation* with a formation of yellowish or brownish blotches, the skin being depressed in these areas, which usually lie along the course of the nerve-trunks, especially the infraorbital. The *hair* becomes thinner, dryer, and often falls out. The secretion of the sebaceous glands is diminished and the skin dryer. Rarer phenomena are the *disturbance of blushing*, so that the affected side of the face remains unchanged in color when, as a result of some emotional disturbance, the other is distinctly reddened. Disturbances of sensation are not common. In some cases electric and tactile sensibility have been diminished; in others the patients have complained of slight paresthesiæ. The special senses remain unaffected, and even when the atrophy extends to the tongue, taste remains perfect on the affected side. In one case there were a slight disturbance of hearing and occasional tinnitus.

The **diagnosis** of the condition is easy both when it is suspected and when it is far advanced. The only condition with which it could be confounded is congenital facial asymmetry. In facial hemiatrophy, however, the skin is shrunken and wrinkled, and the hair is dryer and thinner, contrasting markedly with the healthy side, and there is usually a history of commencement some years after birth. In congenital asymmetry the

difference between the two sides is slight, and the skin over the smaller side is normal in every respect. Commonly in this condition we also find differences in the development of the extremities. In a case that I recently observed with marked facial asymmetry, the left side being smaller, the hand and foot on the same side were distinctly smaller than the corresponding members.

The **prognosis** is unfavorable as regards cure. The disease itself is not in the least dangerous, and cases have been recorded that have been under observation for thirty years or more.

Treatment is unsatisfactory. The prolonged use of electricity has been said to arrest the process, and sometimes this arrest occurs spontaneously; it is not certain that the treatment is of any use.

An allied condition is **hemihypertrophy of the face.** This is an exceedingly rare condition, and is apparently always congenital. It involves chiefly the soft parts, the ear, skin, tongue, and tonsils being all enlarged. There is an increased secretion from the sebaceous glands, which may appear as small elevations upon the skin. Usually, as in congenital asymmetry, there is enlargement of the extremities on the same side. The only case that has come to autopsy presented no lesions.

Treatment is of course unavailing.

PART X.

DISEASES OF THE MUSCLES.

MYOSITIS.

RHEUMATIC myositis and the suppurative form observed in pyemia, and rarely in other acute infectious diseases, have been appropriately described in connection with the diseases to which they are secondary manifestations. There remain to be discussed two rare forms of the disorder.

INFECTIOUS MYOSITIS.

(*Acute Polymyositis*).

Definition.—A primary acute or a subacute inflammation of the voluntary muscles due to an unknown microbic agent.

Pathology.—The disease is a true inflammation of all the voluntary muscles, involving chiefly the muscular fibers, and to some extent, also, the interstitial connective tissue. Beginning with marked hyperemia, there next occurs an exudation of leukocytes. The muscles are hard, fragile, and later undergo fatty degeneration. Serous infiltration occurs and there is a slight hyperplasia of the intermuscular connective tissues. Hueppe records a case that showed nothing definite beyond a hyaline degeneration of the muscular fasciculi.

Etiology.—We are no less ignorant of the predisposing influences than of the specific exciting agency, though, perhaps, young males are most often the victims of this malady.

Symptoms.—As a rule, first the muscles of the extremities, and later of the trunk also, become swollen, firmer than normally, and stiff, rendering locomotion somewhat difficult and painful.

The involved parts may also be tender to the pressing finger, and a slight edema may be noticed that is at first more or less localized, but finally becomes generalized, and extends even to the face. An erythematous eruption then appears, which is irregularly disseminated over the skin-surface, and may tend to more or less pigmentation. Moderate pyrexia and splenic enlargement are among the early and constant symptoms. In the advanced stage the muscles of deglutition and of respiration become involved, rendering the act of swallowing difficult, and inducing marked dyspnea.

Among the *complications* may be enumerated bronchitis and bronchopneumonia, the latter often being a terminal condition.

Diagnosis.—Taken in the aggregate, the symptoms are of little diagnostic importance and the previous history is invariably negative.

Trichiniasis must be discriminated, since this disease produces an identical clinical picture. The distinction may rest upon the examination of an excised piece of affected muscle, which will not only discover the trichinæ, if present but also enable the microscopist to detect the positive evidences of polymyositis. *Multiple neuritis* presents neither swelling nor edema.

Course and Prognosis.—The course of the disease may either be comparatively rapid (two or three months), or it may be slow (chronic) and continue over two or three years. It usually terminates in death, which is caused, in the immense majority of cases, by paralysis of respiration. Occasionally, since the heart-muscle has been sometimes found to be implicated, the end may be preceded by cardiac failure.

The **treatment** is simply palliative and supportive.

PROGRESSIVE OSSIFYING MYOSITIS.

Definition.—Myositis, either general or local, in which the affected muscles undergo progressive ossification.

Pathology.—Following the changes that ordinarily characterize myositis (swelling, leukocytic exudation, etc.), a calcification that is often complete takes place. The process may extend to and involve the heart.

The **etiology** is obscure, though males are especially the subjects of the complaint, which usually begins about the time of puberty.

Diagnosis.—The muscles are represented by plates of bony hardness, leading to more or less complete ankylosis of the joints and vertebræ.

The **course** of myositis ossificans is very slow, and **treatment** has afforded only negative results.

MUSCULAR DYSTROPHIES.

Definition.—These are hereditary affections characterized by progressive muscular wasting beginning in certain groups of muscles, which is sometimes preceded by or associated with apparent hypertrophy of other muscles, without fibrillary tremors and marked change in the electrical reactions. They are also known as myopathies.

Etiology.—The only factor known is the influence of heredity, the disease running through a number of generations. It usually appears before puberty, but may develop later.

Morbid Anatomy.—In the early stages true hypertrophy of muscle fibers may be found. Later proliferation of the muscle nuclei and longitudinal splitting of the fibers, with an increase of connective tissue which takes the place of the degenerated muscle-fibers. A marked deposit of fat is present in the pseudohypertrophic type. The nervous system is normal.

Symptoms.—A number of clinical types have been described, depending upon the muscles first affected, occurrence or not of apparent muscular hypertrophy, and the age at onset. They may all more or less overlap. They are:

1. Pseudomuscular hypertrophy of Duchenne.
(*a*) Leyden-Moebius or hereditary type.
2. Erb's juvenile or scapulohumeral type.
3. Landouzy-Déjérine type, or infantile progressive muscular atrophy of Duchenne or the facioscapulohumeral type.

PSEUDOHYPERTROPHIC MUSCULAR PARALYSIS.

Symptoms.—This form usually appears under the age of ten. The enlargement as a rule affects the muscles of the calves of the legs, although various muscles in other parts of the body may be involved, as the infraspinatus and masseter, or the muscles of the arms and thighs, giving the patient the appearance of an unequally developed athlete. With this may be associated atrophy of the latissimus dorsi, lower part of the pectorals, and muscles of the upper arm and thigh. The electric reactions show no qualitative alteration, but are quantitatively diminished in proportion to the loss of power. This loss of power is manifested first in the gait, which is uncertain and waddling; next, by the difficulty the patient has in arising from the ground. He first gets on his hands and knees, then lifts his knees from the floor and, placing his hands first on his ankles, climbs up his legs until he assumes a more or less upright position (Fig. 80). In

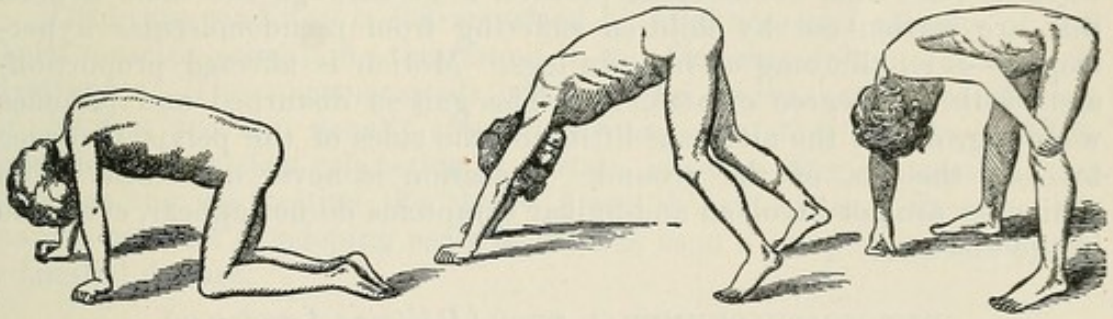


FIG. 80.—Mode of rising from the ground in pseudo-hypertrophic paralysis (Gowers).

the later stages of the disease the volume of the muscles becomes less than normal. At this period contractures may occur leading to the development of club-foot or of lateral deviation of the spine. Lordosis may also be produced by weakness of the muscles of the back, and the spinal column, being no longer properly supported, may topple to one side or the other. Ultimately the patient may lose all power in the affected limbs and pass into a cachectic state, in which he dies. Few ever reach adult life. Some of the cases, however, seem to be milder in character, and may amount to nothing more than a slight weakness, which persists throughout life but does not seriously inconvenience the patient. Often signs of intellectual disturbance are present, the patient learning more slowly and showing an impaired intellectual coördination. At other times epilepsy may be present. A peculiar variety is known by the French as *forme fruste;* this is characterized by a rapid atrophy of the hypertrophied muscles, and consequently the course of the disease is more severe.

HEREDITARY MUSCULAR PARALYSIS (*Leyden-Moebius*).

This commences in children, and usually between eight and ten years of age. It affects the muscles very much as they are affected in the

pseudohypertrophic form, except that there is no increase in size. The disease is markedly hereditary in type.

SCAPULOHUMERAL OR JUVENILE TYPE (*Erb*).

Symptoms.—This type may appear as late as twenty years of age. The muscles first affected are usually the pectorals and the latissimus dorsi. From these the process rapidly extends to the muscles in the neighborhood—*i. e.*, the serrati and the muscles of the back. The muscles of the upper arm and thigh are usually most involved. Those that are most likely to escape are the sternomastoid, the spinati, deltoid, and those of the forearm and leg below the knee. The extensors of the wrist and fingers, and the tibialis anticus and peroneal group, may, however, eventually become affected. The muscles gradually waste, and the wasting is accompanied by a corresponding loss of power, a diminution in the reflexes and of the electric reactions. Reactions of degeneration are not present. Certain peculiar appearances are produced by the atrophy of certain of the groups of muscles. As the shoulder-blades are no longer supported, they stand out from the back, giving rise to the so-called "winged" appearance, and as the result of the weakness of the muscles of the back lordosis is exceedingly common. Weakness of the muscles of the back, and particularly of the glutei, causes the patient, when he rises from the stooping posture, to go through the same actions that are carried out by children suffering from pseudomuscular hypertrophy—*i. e.*, climbing up his own legs. Motion is affected proportionately with the degree of atrophy. The gait is disturbed and becomes waddling, due to the alternate lifting of the sides of the pelvis in order to clear the foot of the ground. Sensation is never disturbed. The sphincters are not involved and bulbar symptoms do not appear, even late in the disease.

FACIOSCAPULOHUMERAL TYPE (*Déjérine-Landouzy*).

Symptoms.—This type usually develops about the third or fourth year. The disease usually begins in the muscles of the face. Of these the muscles about the angle of the mouth first undergo degeneration, giving rise to a peculiar expression, caused by the lips protruding (tapir mouth); the under lip drops forward and downward; the upper lip is wasted and expressionless; all wrinkles disappear, and the patient has a peculiar and strikingly stupid expression. The ordinary movements of the face are considerably affected. Whistling cannot be accomplished and speech is imperfect. Otherwise the course of the disease is that of the scapulohumeral type.

The **diagnosis** is to be made from the *spinal* and *neural forms* of muscular atrophy and from the *congenital absence* of certain groups of muscles. From the two first-mentioned forms it can readily be distinguished by the fact that the hand becomes involved, if at all, in the last stages of the disease; also by the absence of the reactions of degeneration and of muscular twitching. It is also diagnosed from the neural type by the absence of disturbances of sensation. From the congenital absence of certain groups of muscles the diagnosis is sometimes difficult, for, curiously enough, the groups of muscles affected are usually the same

as those affected by the myopathy. A distinction can be made partly by the history, partly by the more efficient and perfect compensatory hypertrophy of the muscles that remain.

The **course** of the disease is slowly progressive, only occasionally exhibiting a temporary arrest.

The **duration** is variable, but patients may live thirty or forty years after the first symptoms appear.

The **prognosis** is of course hopeless as regards cure or improvement. As regards existence, however, it is the most favorable of all the forms of progressive muscular atrophy—a fact that is probably due to the ability of the patients to walk until the very last stages of the disease, so that they are able to maintain a better physical condition.

The **treatment** is the same as that for other forms, and consists of electricity, massage, and especially of systematic gymnastics. Children born of dystrophic parents should be guarded carefully, their nutrition being maintained at the highest possible point and physical strain avoided. Mothers so affected should not suckle their children.

MYOTONIA ATROPHICA.

THIS rare condition is characterized by weakness and atrophy of the facial muscles resembling that found in the facioscapulohumeral type of dystrophy. The sternomastoids, vasti of the thighs and dorsal flexors of the feet are also usually affected, as may also other muscles. With this there is a slow relaxation of certain muscles after contraction, the stronger the contraction the slower being the relaxation. In some instances cataract developing early in life has been a symptom. It may be a familial disease.

ARTHRITIC MUSCULAR ATROPHY.

Pathology.—It has frequently been observed that after inflammation of a joint the muscles that move it have undergone a certain degree of atrophy. This usually occurs in the extensors, and is severe in proportion to the duration of the inflammation. Microscopic examination of the muscles shows a rather uniform diminution in the breadth of the fibers, as well as a slight proliferation of the nuclei and occasionally an indistinctness of the striation. The nerve-trunks and cord have been reported to be normal.

The **etiology** of the condition is not clearly determined. It has been supposed to be due to disuse, but if such were the case all the muscles moving the joint would be equally affected. Moreover, it sometimes occurs too rapidly to render this explanation acceptable. It has also been supposed to be due to the extension of the inflammation either to the nerves or directly to the muscles, but the other symptoms of neuritis are rarely present. Finally, Vulpian has suggested that it is of reflex origin, and this hypothesis is most generally accepted.

Symptoms.—The wasting usually occurs very rapidly after the onset of the joint-affection. The muscles show a diminished contractility to faradism and galvanism, but the reactions of degeneration do not occur. Occasionally there is fibrillary twitching. The mechanic irritability of the muscles is greatly increased, and the reflexes show a corresponding exaggeration, ankle-clonus being frequently observed when the knee- or ankle-joints are affected.

The **diagnosis** may be readily made upon the existence of the joint affection, the local character of the muscular atrophy, and the absence of degenerative reactions with increased mechanical irritability.

Prognosis.—Ordinarily, as soon as the joint has recovered, improvement commences in the muscles and progresses rapidly to complete restoration of function. In some cases, however, atrophy persists, and in a few instances secondary contractures take place.

The **treatment** consists, first, in the removal of the cause by the cure of the articular condition; secondly, in gentle massage and electric stimulation of the muscles. As a rule this should not be commenced until the joint is well.

MUSCULAR ATROPHIES.

These may also occur as a result of other conditions, such as direct injury, fracture of the bones, or prolonged work with a single group of muscles, but they scarcely demand separate description. On the other hand, *muscular hypertrophy* may occur, though rarely.

THOMSEN'S DISEASE.

(*Myotonia Congenita.*)

Definition.—An hereditary disease of the muscles in which the groups that have been contracted by a voluntary influence remain for a short time in a state of contraction, and then relax slowly.

Pathology.—Certain authors have described alterations in the terminal nerve-plates in the muscles, but it is difficult to determine whether these alterations are artificial or an actual part of the disease. The peripheral nerves are normal. The muscles themselves exhibit the following alterations: The muscle-fibers are, on the average, of an increased transverse diameter—*i. e.* the smallest are the size of ordinary muscle-fibers and the largest about twice the size. There is also a distinct and considerable increase in the number of nuclei. The protoplasm is not so clear as in normal muscles, but shows a fine granular cloudiness, rendering the striation less distinct. Occasionally, the muscle-fibers are vacuolated. The connective tissue between the muscle-fibers is normal.

Etiology.—Hereditary influence is the most important factor in the causation of the disease. Thomsen, who was himself a victim, has been able to trace the disease for five generations in his own family. Occasionally a generation is skipped. Other factors that have been supposed to act as predisposing or exciting causes are prolonged exertion (1 case having developed in a man without myotonic antecedents after two years of severe exertion) and emotional disturbance of the mother during preg-

nancy. Exposure to cold, and fright, and a neurotic temperament have also been accused of exerting a predisposing or exciting influence. The disease is somewhat more frequent in males than in females, usually develops in early life, is often associated with manifestations of mental disturbance, and occasionally occurs in those whose ancestors have exhibited lesions of the nervous system other than myotonia.

Symptoms.—The chief symptom of the disease is the so-called myotonic contraction. If the patient, after a period of rest, attempts to set a certain group of muscles in action, the first contraction is made, but is not followed by relaxation for a considerable interval—sometimes as much as a half minute; during this period the muscles remain in a state of tonic contraction. Thus, if the patient attempts to shake hands, he clasps the other hand strongly, and the clasp persists. When he lets go, it is seen that a slight degree of tonic contraction still exists, for it is impossible for him to straighten out his fingers immediately. Upon a repetition of the movement the tonic contraction recurs, but not so strongly, and if the repetition is continued, it disappears entirely, so that the muscular system of the patient behaves in all respects like that of a normal person, and long walks or other severe muscular exertion may be undertaken. In some cases practically the whole muscular system is affected, although, excepting the muscles of mastication, the muscles of the face usually escape. In others the disease is limited perhaps to the upper, perhaps to the lower, extremities. In the former condition the patient may, upon an attempt to make a vigorous motion after resting, suddenly become rigid and fall to the earth with considerable force, often injuring himself severely. He will then lie upon the ground perfectly conscious, but incapable of relaxing his muscles. When the disease, as is more frequently the case, is limited to the lower extremities, the chief disturbances observed are in walking. The first step is accomplished, whereupon the patient halts, both legs having become fixed; after a time they relax and another step is taken. The period of delay is now much shorter, and after a few more steps disappears entirely. The severity of the contraction is diminished by moderate exercise, heat, and tranquillity of the spirits, and is increased by excitement, cold, and fatigue. The muscles of deglutition and the sphincters and the muscles belonging to the nonstriated muscular system are never involved. Pain is not present, except perhaps a slight sensation of cramp, nor are there disturbances of sensation. Mental disturbances are frequent, and have been ascribed to the anxiety occasioned the patient by the disease. They consist of irritability, the avoidance of society, and sometimes of melancholia. The reflexes show various modifications; the knee-jerks may be either normal, increased, diminished, or absent. The most important pathognomonic symptoms are the alterations in the electric reactions of the muscles. The changes are as follows: Mechanical irritability of the motor nerves is normal or diminished; but of the muscles it is increased, and so modified that the contraction instead of being sudden is slow, with a long tonic after-contraction. The faradic irritability of the nerves is normal, and faradic excitation of the muscles produces a tonic contraction of long duration. The galvanic irritability is quantitatively increased and qualitatively altered; that is to say, ACC is equal to and sometimes even greater than KCC. All the contractions are slow,

tonic, and of long duration. Finally, the application of the constant galvanic stream gives rise to rhythmic contractions that pass along the body of the muscles in slowly moving waves at the rate of about one to three per second. Occasionally qualitative galvanic alterations have been observed in the nerves. Finally, the appearance of the patient is of some value. The muscles are developed almost as much as those of an athlete, without a corresponding increase of power.

The **diagnosis** is usually easy, and particularly if it be possible to examine the electric reactions. The condition might possibly be confounded with *pseudo-hypertrophic muscular paralysis*, in which the muscles are also considerably developed; but instead of being normal they manifest greatly diminished power and fail to give a myotonic reaction. From *tetany* the condition may be distinguished by the absence of Trousseau's sign, by a briefer period of tonic contracture, and an absence of severe pains. From *spastic paraplegia* and *Little's disease* it may be distinguished by the fact that in these diseases the spastic conditions are permanent and do not disappear after exercise. From *occupation-neuroses* it may be distinguished by the fact that the cramps only appear upon the performance of a certain peculiarly coördinated movement. From *hysteria* it is differentiated by the absence of stigmata and the care an hysteric patient exhibits to avoid injury to himself, and by the peculiar electric reaction. Myotonia atrophica resembles it (p. 1263).

The **prognosis** is hopeless. The disease commences in early life and continues until death, with more or less frequent remissions and exacerbations. It is possible that these remissions may be permanent, and one case has been reported of a young woman whom marriage greatly benefited. The disease is rarely dangerous to life, excepting in so far that those who suffer from it are much more liable to injury.

Treatment is exceedingly unsatisfactory. Practically nothing can be done, although in a few cases systematic stimulation of the muscles has produced some mitigation. The patients often learn methods by which they can at least diminish the unpleasant symptoms. Certain movements seem to prevent or shorten the period of tonic contraction. Of course exposure to cold or emotional disturbance should be avoided as far as possible.

MYATONIA CONGENITA.

(*Amyotonia Congenita.*)

This disease, first described by Oppenheim in 1900, occurs in early childhood, and is characterized by a more or less general hypotonia of the muscles. There is flaccidity of the limbs, especially the lower, and all of the joints are abnormally movable. Muscular power is much diminished. The deep reflexes are either diminished or lost. The electrical reactions are either quantitatively diminished or lost. The mental faculties are not impaired. The fact that it is congenital but not an hereditary disease distinguishes it from the dystrophies. It differs from amaurotic family idiocy in the absence of blindness and mental impairment. In Spiller's[1] case disease of the muscles was found, and there was

[1] *University of Penna. Med. Bull.*, Jan., 1905.

also a lesion of the thymus gland. Oppenheim believed it to be due to an arrested development of the muscle.

The disease is not necessarily fatal, and measures to improve the nutrition of the muscles (massage, electricity, etc.) may be of service.

MYASTHENIA GRAVIS.

(*Asthenic Bulbar Paralysis.*)

The exact classification of this disease is still a matter of dispute. The only definite changes that have been found are the electrical reactions in the muscles. It is characterized by progressive weakness in the muscles, an increased susceptibility to fatigue, and the occurrence of the myasthenic reaction. The **etiology** is unknown. Possibly infectious processes may have something to do with it; but it bears no definite relation to syphilis. Pathologic changes have not been found. Pemberton has suggested that it is a derangement of metabolism evidenced by an increased calcium and reduced creatinin output. The muscles of deglutition, mastication, and speech, and the group of muscles controlling the eyes are particularly affected; sometimes one group, sometimes another, being first involved. The muscles of the body also become weaker. There may be dyspnea, and even difficulty in walking. The most peculiar feature is the rapidly developing fatigue in the muscles when they have been used. Thus, if the patient attempts to lift the arm a number of times, each successive motion will be weaker than the previous, until finally complete paralysis ensues. If the eyes have been held open for any length of time the upper lid will droop until there is a transient, but complete, ptosis. The muscles also exhibit the so-called *myasthenic reaction*. Upon repeated application of the faradic current the muscles contract less and less vigorously, until finally the capacity for contraction appears to be exhausted, to return after a period of rest.

The **course** of the disease is variable. There are remissions more or less complete and prolonged, but ultimately the patient dies of progressive exhaustion or of increasing dyspnea. Occasionally patients have strangled while attempting to swallow. It has been noticed in women that the symptoms are always increased during menstruation.

The **diagnosis** is to be made from bulbar paralysis. The symptoms resemble each other very closely. In bulbar paralysis there is usually complete reaction of degeneration in the affected muscles. The myasthenic reaction is absent, and in the unaffected muscles the susceptibility to fatigue is not particularly increased. If the patient has been observed for any length of time, the remissions in the course are in favor of myasthenia. In polioencephalitis superior or acute lesion of the oculomotor nuclei, the sudden onset and permanent weakness also serve to make the distinction.

Treatment appears to be entirely without avail. Strychnin hypodermically appears to be useless; electricity is harmful. Full doses of calcium salts may be tried. The patient should be put absolutely at rest, and all sources of worry should be avoided. If there is difficulty in swallowing, the stomach-tube may be employed with advantage.

PART XI.

THE INTOXICATIONS; OBESITY; HEAT-STROKE.

THE INTOXICATIONS.

ALCOHOLISM.

(*Alcoholic Inebriety.*)

Definition.—An acute or chronic intoxication due to the abuse of alcohol. It is a general degenerative condition, particularly of the brain and nervous system, characterized by a moderate (often progressively increasing) or excessive, continuous or periodic, craving for alcohol, leading to drunkenness. Alcoholism is often simply a variety of *inebriety* or *narcomania*, a congenital or acquired brain- and nervous disease, characterized by a resistless, permanent desire for alcohol (*alcoholic inebriety*). *Mania-a-potu*, or "crazy drunkenness," is an acute maniacal condition occurring in an alcoholic drinker of a neurotic constitution. *Delirium tremens* is an hallucinatory manifestation that occurs in habitual drinkers of alcohol, either as the direct consequence of the long-continued action of alcohol on the brain, or because of its sudden withdrawal in an inebriate. *Dipsomania* is an alcoholic insanity in which an intense maniacal "drink-impulse" occurs in a periodic drinker (usually of spirits).

Pathology.—In cases of death from **acute alcoholism** the brain and kidneys are found to be greatly engorged with blood. The gastro-duodenal mucous membrane is also markedly congested, injected, and covered with a thick, sticky, blood-tinged mucus.

Chronic Alcoholism.—Since alcohol is physiologically a poison, and not a food, and essentially a drug, and not a drink, the effects of its habitual ingestion are directly to produce degeneration of nearly all of the bodily tissues, and indirectly to increase the liability to many diseases by lessening the systemic powers of resistance, thus favoring fatality from such disease. The degree of pathologic change depends upon the innate vigor of the tissues, the age at which indulgence in alcohol is commenced, and upon the kind, degree of concentration, and the quantity of alcohol habitually taken. Ethylic alcohol is less deleterious than the "fusel oil" that is sometimes used as an adulterant in spirits.

The chief effects of chronic alcohol-poisoning are seen in the nervous and digestive systems, and in the kidneys. Fatty changes are prom-

inent in the malt-liquor intemperates, while a connective-tissue overgrowth predominates in spirit-drinkers. The mucosa of the stomach presents the appearance of chronic gastric catarrh. Dilatation of the stomach is common in free drinkers of beer, ale, and porter. The liver shows the changes of chronic congestion, of fatty infiltration or degeneration, or of cirrhosis and contraction. The renal changes are analogous to those of the liver, the chronic congested ("pig-backed") and fatty kidneys occurring mostly in cases due to malt liquors, while the sclerosed and fibrous kidneys are seen in spirit habitués. The heart is often loaded with fat, and the muscular structure may reveal fatty degeneration, being pale, flabby, friable, and dilated. The blood-vessels are atheromatous, thickened, tortuous, and sometimes varicose, and sudden death has been caused in inebriates by the rupture of small aneurysms of the middle cerebral artery. In the brain the various stages of sclerosis, with shrunken, narrow, and flattened convolutions often appear. Chronic pachymeningitis, with slight hemorrhages, is not infrequent. The pia-arachnoid membrane also may be opaque and thickened, and serous effusions into the subarachnoid space and into the ventricles have been noted. The nerve-cells, nerve-centers, and nerve-fibres show degeneration, hardening, and atrophy. Alcoholic neuritis is especially prominent in many cases.

Etiology.—An impaired personal health and vigor, as well as the "personal equation" and a deficiency of will-power, self-control, conscience, and conviction, are *predisposing causes*. Drunken or inebriate parents frequently transmit to their offspring a morbid desire for alcohol, and an environment of depraved morality and of depressing and corrupting social influences are usually potent disposing influences, particularly in those who are ill prepared, by heredity or training, to resist the temptation and insidious activities of such evil surroundings. Some assert that poverty predisposes to intemperance: it is more likely to be the cause rather than the consequence of poverty. The *exciting cause* is the persistent misuse of alcohol as a beverage in the form of distilled liquors or spirits, wines, and fermented or malt liquors. "In this country there is a little appreciated but not uncommon cause of alcoholism in the use of patent medicines and nostrums as tonics and cure-alls" (Lambert).

Symptoms.—The symptoms of acute alcoholism range from mild intoxication to an acute delirium or a profound stupor and coma. It begins with the stage of *vascular relaxation* and of feelings of warmth and exhilaration, due to the depressing and paralyzing effects of the alcohol upon the vasomotor tone. The second stage is one of *partial functional paralysis of the nerve-centers*, marked disturbance of the faculties, muscular incoördination, and delirious speech. In the third stage, of "dead-drunkenness," there are acute coma, stertorous breathing, a bloated and congested face, a slow and full, but weak, pulse, a cold and clammy skin, a heavy alcoholic odor of the breath, and, sometimes, incontinence of urine and feces. It frequently happens that unconsciousness is not so profound but that the patient may be aroused, though replies to questioning are stupid and incoherent. Ordinary acute alcoholism seldom passes beyond a stage of exhilaration, ending in mild narcosis. Sometimes, however, the irritant action of the

alcohol predominates over its narcotic action, giving rise to acute alcoholic gastritis or nephritis.

Acute mental disorders ("*acute alcoholic insanity*") are not infrequently met with. *Mania-a-potu* may come on quite suddenly in debauchees, or in those who have drunk hard during a short time, as in a night's carousal. The mental excitability increases until a violent maniacal storm not unlike the mania of epilepsy possesses the drinker. While in this state of infuriated delirium homicide may be committed. Tremors are absent. Acute *alcoholic melancholia* develops suddenly in some cases, with a suicidal tendency. *Delirium tremens* is more common in alcoholic inebriates, and is also seen at times in those who drink greatly to excess, but are not habitués. Convulsive seizures have been noted in some cases, interrupting the coma ("acute alcoholic epilepsy"); these may or may not be accompanied by mania. An acute *alcoholic paralysis* from multiple neuritis (occasionally with ataxic symptoms) may attack hard drinkers, and may last for several weeks or months.

Chronic alcoholism (alcoholic inebriety) I consider a true disease. While acute alcoholism may also be an occasional manifestation of the chronic affection, it is often a vice which, if indulged in to an excessive degree, or if too frequently repeated, becomes a disease, though it is difficult to determine at what point the transition occurs. Again, it is not always easy to learn whether the early acute alcoholic excesses are really vices or morbid, diseased cravings for alcohol in hereditary narcomaniacs. The disease of inebriety (alcoholic) is a condition in which, as some one has said, it is not whether one "cannot" or "will not;" but in which one "cannot will" to resist the desire for alcohol.

The steady, so-called "moderate drinker" who saturates his blood and tissues every day for years is much more apt to suffer from chronic alcoholic poisoning with its attendant degenerations than one who goes on a "spree" once a month for a day or two, and during the intervals is free from the toxic influence of alcohol. The *symptoms* develop very gradually, and are usually marked for some time by the deceptive sensation of stimulation, warmth, and well-being, due to the vasomotor paresis and the anesthetic effects of the alcohol. Impairment of digestion is early noted. There are a coated tongue, foul breath, vomiting before breakfast, and gastric distress after eating. Constipation alternating with diarrhea is common. Muscular tremors gradually develop and often progress into an ataxic gait. Insomnia, mental impairment, and blunting of the moral sense come on. "Alcohol dims the perception, confuses the judgment, paralyzes the will, and deadens the conscience" (Kerr). In his distress and degradation the inebriate seeks to relieve himself by taking more of the alcohol, only to find, on awakening from his narcosis, that body, intellect, will, and emotion are still more depraved. In fact, the brain- and nerve-disorders are more grave, permanent, and extensive in the majority of instances than those of the viscera. This is owing to the delicacy of the nervous mechanism and to the ready degeneration under the influence of the altered blood, and the consequent impaired cellular nutrition, directly due to the toxic action and deficient normal pabulum, and indirectly to the lessened elimination of waste-products.

Dementia is often the terminal state of the chronic inebriate. Delusions of persecution are frequent, especially those of marital infidelity, in alcoholic insanity. The depurative organs manifest various symptoms due to the long-continued irritating action of alcohol. The liver is either fatty and enlarged, or cirrhotic and contracted, and jaundice, dropsy, and hemorrhoids, along with physical hepatic signs, are correspondingly observed. The watery eye, the injected conjunctivæ, the swollen eyelids, the bloated and flabby or pallid and shrunken face, the dilated capillaries of the nose (*acne rosacea*) and cheeks, may now be seen. The urinary examination will show in many cases the deranged function of the kidneys and point to the nature of structural impairment. On account of the weak and flabby heart there are palpitations, dyspnea, and precordial distress, and occasionally sharp pains. Chronic valvular endocarditis may be discovered. The pulse is soft and weak in beginning fatty degeneration of the vessels. Thickened arteries are common in old cases, and the pulsations are often increased in tension and usually rapid. Muscular capacity and endurance are greatly diminished.

Delirium tremens occurs in the majority of cases in inebriates or chronic drinkers during or after a debauch, and particularly from the use of spirituous liquors. It may occur, also, during abstinence from alcohol, on account of some mental perturbation, or fright, accidental shock, or acute inflammatory illness. It may either come on suddenly, or be preceded (often for a day) by some slight premonitory symptom, as anorexia, restlessness, or depression of spirits. The patient usually awakens at night with a tremor, becomes sleepless, wants to get out of bed to do some imaginary thing, talks constantly and incoherently, looks about uneasily and fearfully, and breaks gradually into a cool perspiration. Hallucinations of sight, hearing, and smell develop. The patient sees terrifying and loathsome reptiles, and tries to escape from them, or to clutch them in order to cast them away. The "horrors" may become so great that suicide may be attempted, as by falling out of the window. Auditory hallucinations may take the form of enemies, policemen, or the roar of wild animals. The muscular tremors increase, the pulse becomes frequent and weak, and the tongue coated with a thick white fur. There is moderate fever, which, if the delirium is prolonged, takes on a typhoid character, the tongue becoming tremulous, dry, brown, and fissured, with the onset of subsultus tendinum, carphologia, coma-vigil, and muttering delirium. In favorable cases improvement begins on the third or fourth day, from which time the symptoms gradually subside. Convalescence may be said to be established when restful sleep can be obtained; this is followed by a desire for food. In unfavorable cases the patient may pass from a typhoid state into exhaustion and death, or may die suddenly either during a paroxysm of cardiac failure or from some complication, as cerebral hemorrhage or pneumonia.

Korsakow's psychosis, or the mental symptoms that may occur in connection with alcoholic neuritis, is described on page 1085.

Diagnosis.—The condition of persons found dead-drunk is seldom mistaken for any other. The reverse more often happens, and in this way *apoplectic* and *uremic comas* may be diagnosed as alcoholic coma. Cases picked up in the street in a state of apparent unconsciousness

should be carefully tested in this regard. Instances in which, as the *postmortem* examination subsequently has shown, cerebral hemorrhage has followed a drinking-bout, render the diagnosis more difficult; in such the patient should be given the benefit of the doubt and handled as though the case were one of apoplexy. An important early step is to ascertain whether the coma is complete, or whether the patient can be roused by shouting in the ear, by applying ammonia to the nostrils, or, better still, by pressing, with gradually increasing firmness, over a sensitive spot, as the supraorbital notch; if the unconsciousness is alcoholic, he will come to his senses, if only for a moment. Abstemious apoplectics have been known to stagger and talk thickly, like drunken men (Kerr), and have been arrested and taken to a police-station instead of to a hospital. *Congestion* and *lobar pneumonia* affecting the bases of the lungs should be looked for, as they are common causes of death in drunkards. A table giving the principal points in the differential diagnosis will be found under Uremia (*vide* p. 1011).

The diagnosis of chronic alcoholism is made from the history, and from the muscular tremors (worse in the morning), vomiting, mental restlessness, "mendacity," and involuntary "lying" (Kerr). The condition may resemble general paralysis, and if the habits of the patient are kept secret it may be very difficult to differentiate these affections. A prominence of disorder of the digestive tract usually points to alcoholism. Nervous excitement, tremors, fear, wakefulness, and the distinctive physiognomy are more evident in chronic alcoholism, even when general paralysis has been caused by alcohol, which is apparently the case. *Paralysis agitans*, *locomotor ataxia*, *epilepsy*, and *nervous dyspepsia* may also be mistaken for chronic alcoholism by the unwary.

Delirium tremens is distinguished by the history, by the restlessness, delirium, hallucinations, tremors, and terrors. *Mania-a-potu* differs from the preceding mainly in its usual association with acute alcoholism in neurotics, in the muscular contractions, the furious mania, and convulsive movements. The delirium of *apical pneumonia* that obtains in some cases (as well as in meningitis) must be thought of in the diagnosis of delirium tremens. The diagnosis of *alcoholic neuritis* from other conditions simulating it will be found elsewhere (*vide* p. 1085).

Prognosis.—In acute alcoholism the prognosis is favorable in private, manageable cases. Many of the cases brought into hospitals are affected also with pneumonia, and usually die. The tissue-changes in chronic alcoholism are so profound, and they affect such delicate and vital tissues, that when the alcohol-habit thus becomes fixed permanent recovery never takes place. The treatment appropriate for the inebriate and forced abstinence from alcohol relieve many of the symptoms and some of the debility, but relapses are all too common and are almost certain to occur. Insanity and paresis are not infrequent terminations of chronic alcoholism. Many complications are apt to supervene, as Bright's disease, epilepsy, melancholia, fatty heart, pneumonia, and thrombosis. Alcoholic neuritis often clears up upon withholding alcohol and stimulating the peripheral nerves both by appropriate drugs and external remedial measures. Recovery from delirium tremens is dubious in cases of severe injury, inflammatory troubles, or infections.

Treatment.—In cases of acute drunkenness, which are only too

commonly met with, nothing special is required except to prevent the ingestion of any more alcohol and to allow the patient to sleep until the elimination of the poison is more or less complete. The effects of the intoxication, in the general depression, headache, anxious and irritable stomach, and various functional visceral and nervous disorders, may need careful corrective and sustaining treatment for a week or more. The diet should be light and nutritious. Aperient waters, hot baths, with liquor ammonii acetatis frequently repeated, and a combination of dilute mineral acid and bitter tonics (nux vomica, gentian), are also indicated.

In profound cases of alcoholic coma, convulsions, or mania-a-potu no alcohol should be given. Trite though this injunction may seem, it is important to emphasize this statement, so that the physician may be sure to counteract a popular impression that the giving of more alcohol will cause a mania to subside *permanently*, and to guard against the smuggling of liquor to the patient by his misguided friends. It is often necessary to empty the stomach at once when collapse is imminent by the use of the stomach-tube or -pump, washing out the organ with hot water, to which ginger or cinnamon has been added. To this end emetics may be used—viz. ipecac or apomorphin, hypodermically (gr. $\frac{1}{8}$ to $\frac{1}{6}$—0.008–0.0108). The external application of warmth, friction, artificial respiration, faradism to the phrenic nerve, ammonia- or amyl-nitrite-inhalations, and hypodermics of atropin, strychnin, and digitalis, may all be tried. Hot rectal enemata or a calomel purge if the stomach will tolerate the drug should be used early. The maniacal attacks may be treated by hypodermics of morphin and hyoscin, and by such sedatives as chloral, bromids in large (ʒj—4.0) doses, and rarely such hypnotics as paraldehyde, trional, chloralamid, and the like. Indeed, it is very important to secure sleep as soon as possible. An excellent formula in cases of medium severity is:

℞. Sodii bromid., ℥j (32.0);
Tr. capsici, ʒj (4.0);
Tr. digitalis, ʒss (2.0);
Elix. simplicis, q. s. ad ℥ij (64.0).—M.
Sig. ʒj (4.0) every two or three hours, in water.

As soon as some quietude and sleep have been obtained, it is in order to administer concentrated food in an easily assimilable form.

The treatment of *chronic alcoholism* is more often best conducted in "homes" for inebriates, in hospitals, and similar institutions. At the outset there must be an "unconditional surrender" in the use of alcohol. Its withdrawal should be enforced at once in many cases, and very rapidly in all others, according to the judgment of the physican as to the psychic and physical condition of the patient. Substitutes for alcohol are the strong fruit-juices, as hot lemonade or hot ginger, capsicum infusion, and cardamom tea often is useful. Coffee, milk, cocoa,and hot broths are also to be recommended. The diet should be carefully increased in nutritive strength as the gastric irritability diminishes. Sometimes such sedatives to the stomach as the bismuth preparations, effervescent alkaline drinks, and lime-water may be indicated. Peptonized food is often well borne at first in cases in which gastric distress is marked. Nutrient enemata are seldom required, but should be resorted

to in the gravest cases, particularly during the states of alcoholic dementia. The general health must be looked after by placing the patient in the best of fresh air, exercise, cold and warm bathing, by mental and social occupation, and by diversion. When the craving for alcohol is hereditary and intense, seclusion in an inebriate-house or some similar institution is often necessary for a long time to lessen the danger of lapsing into the former drink-habit.

The insomnia of chronic alcoholism may be met temporarily by the use of large doses of bromids, chloral, hyoscin, or sulfonal. Morphin may be indicated at times, but should be used with great caution in order to avoid adding the morphin-habit to that of alcohol. Perhaps the best single agent to use in counteracting the symptoms of chronic alcoholism is strychnin, either as the nitrate or sulphate, hypodermically and by the mouth; iron, arsenic, the hypophosphites, dilute phosphoric acid, quinin, gold and sodium chloride, avena sativa, and the like are often useful adjuvants in the tonic treatment. Atropin, hypodermically, may also be recommended when vascular dilatation and weakness are prominent. Sweating and purging the patient, and the administration of bromids, chloral, and gelsemium for a day or two in advance may avert a "drink-storm" or the periodic cravings for alcohol that may be expected by prodromal manifestations. Sometimes, however, as in the sudden outbursts of dipsomaniacs, there is no time to institute their treatment. It is claimed that hypnotic suggestion will abolish effectually the ardent desire for alcohol in a certain number of neurotic cases of alcoholic inebriety. Temperance revivals may be said to do permanent good only in those similar neurotic cases that are fortunately impressionable to appeals by total-abstinence orators, but, in order to maintain the reformed drunkard's pledge, it is often necessary that interested persons continue to watch, guide, and inspire him, in order that a weakened will may not precipitate a cyclic lapse into his old habits.

All the influence of culture, music, and the fine arts, of high-toned morality and pure, undefiled religion, should be enlisted to strengthen self-repect and to fortify volition and inhibition. Moral regeneration may thus in certain cases check the physical and mental degeneration, but it cannot efface the consequences of the alcoholic poisoning which it represents.

McBride recommends the following method of treatment: Hypodermic injections of strychnin three times daily, at first $\frac{1}{60}$ of a grain, increased to $\frac{1}{30}$ by the end of the first week, and at the same time hypodermic injections of atropin are given, which are rapidly increased until the patient's tongue is made dry and the pupils dilated. A bitter mixture containing cinchona, gentian, rheum, capsicum, and more atropin and strychnin is also given six times a day. During the first week, usually during the first few days, all taste for alcohol is lost. During the second week this treatment is continued, but during the third the injections of atropin are gradually diminished, and, finally, stopped, and the capsicum is withdrawn from the mixture taken by the mouth. Thus modified, the treatment is continued during the fourth week. At the end of that time the atropin is withdrawn from the mixture and the latter is given four instead of six times daily. During the sixth week the injections of strychnin are reduced and stopped. At the beginning of the week the cinchona is

also withdrawn. The author necessarily varies this treatment with the requirements of individuals.

Delirium tremens requires firm but tactful isolation and vigilant nursing. All alcohol should be withheld. If stimulation is needed, aromatic spirits of ammonia; strychnin, and atropin, with bland hot drinks and broths, may be administered. Easily digested and nutritious food should be given to support the strength. Sleep must be procured by such means as are mentioned above in the treatment for *mania-a-potu*. The dosage required, however, is usually not as great, but must be kept up longer than in the maniacal condition. Cardiac weakness may need such stimulants as digitalis and strophanthus. After the attack subsides, tonic doses of strychnin, gentian, asafetida, and iron, together with graduated exercise out-of-doors, are to be employed. Turkish baths, industrial occupations, and the like are indicated to fortify the patient against yielding to a morbid appetite.

GINGER AND COLOGNE-WATER INEBRIETY.

Habitual drinkers of alcoholic ginger, capsicum, and lavender preparations, and eau-de-Cologne are practically alcohol-habitués or inebriates. They drink these liquids for the alcohol that is in them. The so-called essence of ginger (Jamaica ginger), which contains considerable alcohol in some of its preparations, is often used primarily for relieving an attack of "cramps" or "colic," and if frequently repeated, can readily induce a morbid habit of "ginger-drinking." In other cases the craving for alcoholic indulgence (often hereditary), may have been aroused by a social glass of wine, but, from a sense of shame the desire has been kept secret, and gratified by drinking eau-de-Cologne, lavender essence, or even tincture of capsicum. Perhaps many more such cases exist, and especially among neurotic women in good circumstances, than are usually recognized.

MORPHINISM.

(*Opium-inebriety.*)

Definition.—A chronic intoxication, due to the habitual use of morphin or of opium in some other form (*opiumism*).

Pathology.—In cases of death from acute or chronic opium- or morphin-poisoning there is nothing distinctive in the pathologic appearances. In acute cases vascular congestion of the brain and membranes has been noted; but even in chronic cases the tissue-degeneration and fatty and connective-tissue proliferations that are characteristic of alcoholism, are practically absent. Decided lesions are usually traceable to associated affections. The principal anatomic changes are those due simply to malnutrition. Thus, we have the emaciation and the shrunken appearance of cerebral anemia, and pallor and atrophy of the cardiac muscle and of the vascular walls. The dried and wasted structures, due to tissue-starvation, are quite a contrast to the fat-infiltrated or degenerated, cirrhotic, and inflamed tissue of alcoholic inebriety. Direct destruction of parenchymatous cells is more evident in the later.

Etiology.—The climate, country, and nationality have a certain disposing influence in the development of opiumism and morphinism.

In the opium-growing parts of Asia, as in China, India, and Persia, where the climate is warm, enervating, and conducive to physical and moral abandonment during the greater part of the year, and in Turkey also, opium-eating-and-smoking habitués are as numerous as alcohol habitués are in Europe and America among the Caucasians.

Women are more commonly the victims of morphinism than men, except physicians and druggists as a class. Mattison has found 70 per cent. of his opiate patients to be medical practitioners. Many contracted the habit by using morphin for severe chronic neuralgia, insomnia, and the like. Indeed, pain and sleeplessness have been the principal source of this drug-habit.

Ennui and an idle spirit of irritation and adventure among the sensation-loving and luxurious sometimes sow the seeds of an indulgence in narcotics that bring forth fruitage in the form of a fixed habit.

The incautious prescribing of morphin and the too ready hypodermic use of the alkaloid by physicians in treating various cases of pain are not infrequently the cause of morphinism. Overwork of the brain, great business or social strains, prolonged worry and anxiety either with or without work, insomnia, remorse, idleness, and secret vices, are the most common predisposing agents of the morphin-habit.

Paregoric, laudanum, chlorodyne, and "soothing-syrup" are drunk to a frightful extent in large cities among the poor and miserable, and cause great disturbance of the health of the habitués.

Symptoms.—These may be in abeyance for some time, while the habit is forming and the doses are still slight. As the craving increases, the dose and its frequency increase to keep pace with the desire. Anemia gradually develops, with sallowness of the skin, wasting of the features and body, languor, weakness, functional deterioration, mental depression, anorexia, restlessness, insomnia, tremors, irritability, shyness, dilatation of the pupils (except when under the influence of the drug), and a characteristic propensity to lying. Cardialgia is often complained of by those who use opium pretty constantly. The associated vices of opiumism are less violent and inflammatory than those of alcoholism, and more secretive and speculative, such as gambling and sexual perversions. Itching is frequent, and especially after taking the opium or morphin. Attacks of chills, followed by pyrexia, with delirium and transient albuminuria (renal congestion) occur in some cases. Diarrhea and dysentery have been observed in some instances. There may be also disturbances of the visual muscular apparatus. Sufferers from painful carcinoma in whom opium or morphin is required for steady use do not become, except in rare cases, true morphinomaniacs.

The *course* of morphinism is that of a progressive asthenia, in which cardiac palpitation, dyspnea, abdominal and muscular cramps, trembling, fear, sleeplessness, mental confusion, melancholy, slovenliness, and moral obtuseness come on. Some women, known to be kleptomaniacs, have been found to be secret opiumists. Sexual impotence in the male, and amenorrhea and abortion in the female, are common results. The skin is wrinkled, dry, and harsh, and may show numerous needle-scars and abscesses in those addicted to the hypodermic use of the drug. The *termination* is the direct result of the extreme debility or marasmus or of some intercurrent affection.

The **diagnosis** must be made from the history. When the latter is wanting because of a lack of veracity or deception, *chronic alcoholism* may have to be differentiated from opiumism. The more open and often periodic habits of the alcoholic habitué, and the general aspect of the physical and mental and complicating conditions, usually show marked differences between the two drug-intoxications.

Prognosis.—The likelihood of a cure is exceedingly remote. On the other hand, under proper conditions much relief may be given and life prolonged for years. Opium smoking produces less injurious consequences, and is more readily cured than other forms of the addiction.

The **treatment** is manifestly difficult and unpromising. Institutional isolation, rest, diversion, watchful care, regular and studied feeding, baths, and graduated exercise in the open air as far as possible, but under surveillance in order to prevent the smuggling of opium, morphin, or compound preparations containing either, are the most efficient measures. As to the manner of withdrawing the narcotic, much care, judgment, and tact form a *sine quâ non* in the treatment. A sudden and absolute stoppage of the use of the drug sometimes leads to great distress, and even to collapse ("abstinence phenomena"); it is, therefore, not to be recommended, as in chronic alcoholism. On the other hand, the too gradual withdrawal is torturing. A middle course, the "rapid-gradual method" of Erlenmeyer, is usually resorted to, in which the reduction of the quantity of morphin or opium to nothing occupies but a week or ten days. Various substitutes have been recommended that generally prove not to be substitutes at all, but simply act in a symptomatic way, and may lead to another habit as bad if not worse. Such drugs as cocain, hyoscyamus, bromids, and chloral have thus been used. Hare and others have reported good results from the method of treatment suggested by Lott, namely, by the use of hyoscin hypodermically in large doses (gr. $\frac{1}{100}$ every two hours) until the patient is rendered calm or even unconscious, after which this state is to be maintained for several days and then the dose is to be gradually diminished so as to permit a return to the normal condition. Cardiac stimulants may be needed. The Towns-Lambert method of treatment is now widely adopted, and the results which have been recorded in literature are most promising.[1]

In the symptomatic treatment of the morphin-habit moderate doses of bromids, with cannabis indica and some such vegetable bitter as gentian, may prove useful in allaying the nervous irritability and restlessness at night. Sulfonal is a good hypnotic in these cases. Cathartics, stomach sedatives alternating with tonics, concentrated foods, massage, hot and cold bathing, electricity (general galvanization), and "complete control over the patient" are usually indispensable adjuncts in the treatment after the withdrawal of the opium or morphin. Cardiac stimulants, strychnin and physostigmin salicylate (gr. $\frac{1}{100}$—0.0006) hypodermically, have been recommended recently as important in counteracting the functional depression of these habitués. Industrial activity, and mental and social diversion, aid in maintaining any improvement made and in rendering the patient less liable to a relapse.

[1] For details, see *The Jour. Amer. Med. Assoc.*, Feb. 18, 1911, p. 503.

PLUMBISM.

(*Chronic Lead-poisoning; Saturnism.*)

Definition.—A chronic intoxication due to the slow absorption of lead, either industrially or accidentally.

Pathology.—The principal lesions are found in the muscles, peripheral nerves, liver, kidneys, and mucous membranes. The affected muscles are wasted, pale-yellow in color, and, in advanced cases, show a marked fibroid growth. The vessels in the muscles also reveal arteriosclerosis. The peripheral nerves are affected with a parenchymatous neuritis, and are especially involved, with degenerative changes in the nerve-endings in the muscles. The nearer we approach the spinal cord along the course of an affected motor nerve, the less marked are the changes, although in some cases a very slight involvement of the anterior nerve-root cells has been noted. The cord is usually normal.

In the brain, slight meningitis and arteriosclerosis of the cerebral blood-vessels here and there, with a corresponding connective-tissue growth and capillary hemorrhages. The liver and kidneys show parenchymatous atrophy and cirrhosis.

Etiology.—(*a*) *Personal susceptibility* to lead-poisoning is greater in some people than in others, all other things being equal. (*b*) Plumbism is more common in *adults* than in *children*, because of greater exposure. (*c*) *Sex.*—Women are more susceptible than men. (*d*) *Occupation* is the most frequent cause of lead-intoxication. Workers in white lead (plumbic carbonate), red lead, and litharge, all of which substances are used as paints, are especially to be mentioned as liable to saturnism. Among the most common industrial causes are the following: painting, plumbing, lead-mining, rolling sheet-lead, pottery-glazing, type-founding and setting, shot-making, dress-making (in which lead-dyed silk thread is used and the ends bitten off), lace-making, glass-grinding, and calico-printing. (*e*) Accidental contamination of food and drink. Men employed in the manufacture of white lead and eating lunches in dusty work-rooms suffer from plumbism. Drinking water stored in lead-lined cisterns and passed through lead pipes is frequently contaminated, especially if the water contains a slight amount of acid. Flour, bread, biscuit, candy, butter, and milk may cause poisoning by adulteration with lead chromate, used to give a rich, yellow tint to these articles; and tobacco wrapped in lead-foil has resulted in saturnism. (*f*) Workers in lead suffer more frequently during the warm season. (*g*) Previous attacks greatly increases susceptibility.

The *absorption* of the lead takes place mainly through the gastro-intestinal tract and the lungs, and much less through the skin. It may be deposited in most of the soft tissues and viscera, but especially in the nerves, muscles, and liver. *Elimination* takes place through the kidneys, and probably, though in very slight quantities, with the bile and saliva, and through the skin.

Symptoms.—Depending upon individual susceptibility, it may be months or years before the first manifestations appear. *Anemia* is an early and marked symptom. The red cells and hemoglobin are reduced correlatively. Boston, in the study of 24 cases, found the leukocytes to

number between 10,000 to 23,000 per c.mm.; and average of 12,600. The erythrocytes are pale, distorted, and show evidence of punctate basic degeneration. Grawitz and Frey regard polychromatophilia as an important blood finding (Need). The general nutrition is poor.

The characteristic *blue line* at the borders of the gums is rarely absent, especially in those who are not scrupulous in their attention to the teeth. It is, as a rule, most distinct at the roots of the lower canines and incisors, and is formed by a deposition of lead sulphid. Bluish patches may also be met with. Gowers points out that this line is black instead of blue, and is present only when the gums are slightly separated from the teeth. Slight jaundice may at times be noted.

Colic is very common and is also characteristic. The pains center around the navel, and are quite severe and griping. They are associated with retraction and rigidity of the abdominal walls, and with obstinate constipation. The pains are paroxysmal, may be referred at times to the epigastrium, and may be accompanied by vomiting. Between the paroxysms a dull pain usually exists over the whole abdomen. During the attacks the pulse-tension is increased and cardiac action lessened. The stomach contents show no HCl as a rule.

Exaggerated tendon reflexes may be present early. *Paralyses* are common symptoms, and may either be acute, subacute, or chronic in nature. Although usually localized palsies, they are sometimes generalized. The most characteristic lead-palsy is that known as *wrist-drop* (see also Multiple Neuritis, p. 1085). Both fine and coarse *tremors* occur. They usually begin in the hands and arms, are rather constant, and are aggravated by *voluntary effort* and emotional excitement.

Cramps in the affected muscles and about the joints (*lead-arthralgia*) are occasionally noted. Slight anesthesia, especially in cases of wrist-drop, is sometimes detected here and there, but may in certain instances be due to saturnine hysteria.

The *cerebral symptoms* are important. The phrase "lead encephalopathy" includes such manifestation as delirium and coma, neuro-retinitis, aphasia, convulsions, hemiplegia, amaurosis, hysteria, and insanity. The delirium and coma are the commonest brain-symptoms, and may come on suddenly with tremors and hallucination. Epileptic convulsions are often severe. Hemianopsia has been observed. Mania and melancholia occur in cases of mental unbalancing, and hysteric outbreaks are seen in girls. Intense headache is not uncommon. "Saturnine gout," so called, is described as a result of chronic plumbism. The kidneys are contracted, the heart is hypertrophied, and arteriosclerosis is marked, with a diminution in the excretion of urea and uric acid. The pulse-tension is increased. These evidences show a similarity to gout, and favor the development of uratic deposits in the joints. Lead may be discovered in the urine by laying a strip of magnesium in it and noting the deposit of metallic lead if present (Von Jaksch). Abram asserts that the addition of a solution of ammonium oxalate (1 gm. to 150 c.c. of water) facilitates the test. Hematoporphyrin may be found in the urine.

Diagnosis.—The history of exposure to lead-poisoning is usually clear in those working the metal in its various forms. *Accidental origins* of saturnism are often obscure and very difficult to trace, although if

the characteristic wrist-drop, the gingival line, colic, and cachexia be present, the diagnosis is readily made.

Alcoholic paralysis of the lower extremities may be differentiated by the history, the greater prominence of sensory symptoms, and by the absence of the blue line on the gums, and of punctate basophilia.

Prognosis.—In the absence of the graver nervous, arterial, and renal symptoms, the prognosis is good. When there is paralysis, with reactions of degeneration, and especially in primary atrophy of the muscles, the prognosis is generally bad. In encephalopathic forms, and in cases in which arteriosclerosis and renal cirrhosis are manifested, the prognosis is unfavorable, but depends upon the extent of damage done. Pulmonary tuberculosis often complicates lead intoxication.

Treatment.—The prevention of plumbism is difficult in lead-working establishments, owing to the carelessness and indifference of both employers and employees, and to the lack of any adequate antidote during exposure. Rigid cleanliness is absolutely necessary, especially of the hands and nails and before eating. Means to allay dust should be regularly and constantly employed. Milk and sulphuric-acid lemonade have been recommended for use by workers in lead, for their supposed antidotal effects. As perfect ventilation as possible should be secured, and respirators are in use in some lead-works, being worn as "snouts." Potassium iodid should be given in chronic plumbism, beginning with small doses (gr. iii–v—0.1944–0.324), given preferably in milk, after meals.

In *lead colic* hot applications to the abdomen and hypodermic injections of morphin and atropin are often indicated. Efficient doses of Epsom or Glauber's salts are used to combat the constipation. Given in combination with dilute sulphuric acid (in order to form an insoluble lead sulphate) and with belladonna, the best and speediest benefits may be obtained thereby.

Iron for the anemia, strychnin and galvanism for the paralysis, lithia-water for the renal deterioration, and nitroglycerin or sodium nitrite for the arteriosclerosis (enough to relieve increasing tension) are the symptomatic items of treatment that are usually indicated. Rarely, hopeless cases of saturnine encephalopathy need to be sent to asylums for the insane.

ARSENICISM.

(*Chronic Arsenic-poisoning.*)

Definition.—A chronic intoxication resulting from the gradual absorption of arsenic.

Pathology.—The peripheral nerves show a degenerative neuritis, and the anterior horns of the spinal cord may be similarly affected.

Etiology.—The causes of arsenicism may be habitual, industrial, medicinal or accidental, and individual predisposition varies. A neurotic diathesis usually underlies the habit of "arsenic-eating" in those who crave the drug. Not a few women suffer from chronic arsenicism as the result of the ingestion of arsenic "to improve the complexion and brilliancy of the eye." Men employed in arsenic works of various kinds often suffer from the chronic poisoning. For example, miners and smelters of arsenic pyrites, dyers and wall-paper workers using Scheele's

or Schweinfurth's green, artificial-flower makers, shot-makers, glass-workers, and taxidermists, are all liable on account of their occupations. The medicinal use of arsenic, even for a short time, may in very susceptible persons induce arsenical paralysis (Putnam; Osler). Again, "cancer cures" containing arsenic may cause poisonous effects. Accidental arsenicism may come from living in rooms where wall-paper, carpets, colored paper ornaments, toys, or curtains are contaminated with arsenic anilin dyes. Drinkers of beer may suffer, the arsenic being derived from the sulphuric acid used in manufacturing the glucose that is employed in its manufacture.

Symptoms.—There are anemia, loss of flesh and strength, dryness and irritation of the mucosæ, of the eyes, nose, throat, and upper respiratory tract. Anorexia, nausea, and diarrhea indicate the presence of a gastro-intestinal catarrh. In some cases, milder than others, the fat is well preserved. Slight puffiness of the eyelids or eyebrows may occur, and some epigastric distress may be complained of. Marked conjunctivitis, occasional dysenteric attacks, loss of the hair, and numbness and tingling in the extremities form a commonly observed symptom-group. Cutaneous symptoms may appear, as pigmentation ("arsenic-bronzing"), and eczematous, herpetic, urticarial, and pemphigoid manifestations. Albuminuria with casts and blood mark the renal irritation that sometimes occurs.

The most characteristic evidence of chronic arsenic-poisoning is seen in the gradual increasing diffuse or multiple neuritis. Differing from lead-palsy, the leg-extensors and the peroneal group of muscles are involved first, although the arms may also become affected later (*vide* Multiple Neuritis, (p. 1085). Contractions in the lower and a fine tremor of the upper extremities are apt to occur. Arsenic-poisoning may also cause headache, vertigo, melancholia, and hysteria. The drug is eliminated by the kidneys and may be found in the urine. Sometimes a great toleration of arsenic is observed in workmen and habitués, the only evidences being a clear, sallow, waxy complexion, a gloomy expression, and some dyspepsia, perhaps, as in the well-known Styrians.

Diagnosis.—This is not difficult, when once the source of the poisoning is determined. The clinical appearances are distinct from *lead-intoxication*, especially in the mode of progress of the paralysis, and in the more marked sensory symptoms combined with the motor-disturbances of arsenicism. Arsenic should be sought for in the urine.

The **prognosis** is favorable in most cases in which removal from the exposure to the influence of arsenic is possible. A few cases die from the great general debility.

Treatment.—Abstention from the use of arsenic for cosmetic purposes, avoidance of its influence in the arts, care in its medicinal administration, and prophylaxis as regards the possible or discovered sources of contamination, form the first considerations in the treatment. Elimination of the arsenic may be promoted by the use of potassium iodid and purgatives. Gastro-intestinal and other irritations must be met by appropriate sedative remedies. The neuritis and palsies require —as soon as the tenderness and pain subside—massage and electricity. Judicious and wholesome alimentation and tonics are indicated.

MERCURIALISM.

(*Chronic Mercurial Poisoning.*)

Definition.—A chronic intoxication caused by the habitual ingestion, or combined industrial absorption of mercury, in susceptible individuals.

Pathology.—No marked pathologic changes have been noted in human beings, aside from the evidences of oral, gastro-intestinal, and renal irritation and inflammation. It is not improbable that the cerebral cortical areas suffer more from metallic irritation than do the spinal or peripheral nerve-tissues.

Etiology.—Some persons are much more easily mercurialized than others. (*a*) *Salivation* and *stomatitis* from the therapeutic use of mercury form a variety that is less frequent than formerly. (*b*) *Industrial origin.* The chief cause of chronic mercurialism is the inhalation of the vapor of the metal by artisans in the industries in which it is used. Thus miners and smelters and those engaged in making mirrors, barometers, thermometers, amalgams, felt hats, vermilion-pigment, and artificial teeth sometimes suffer from chronic mercurial poisoning. It should be pointed out here that mercury is volatile at ordinary temperatures, and is absorbed into the blood through the lungs, digestive tract, and skin. Calomel vapor-baths have caused poisoning in a few cases. (*c*) Purely *accidental* mercurialization also occurs. (*d*) Women and children are more susceptible to the action of mercury than men. In all cases the mercury exists in the tissues as an albuminate.

Symptoms.—There are anemia, emaciation, gastro-intestinal disorders, stomatitis, salivation, maxillary necrosis, ulceration of the gums, loosening of the teeth, fetor of the breath, marked tremors, and paralysis. The oral symptoms are not as prominent, however, as in acute mercurial poisoning. The hair falls out, the nails become brittle, and pigmentation of the skin is seen.

The *tremor* is characteristic. It is first felt or noticed in the tongue and lips, is usually fine, later coarse and choreiform, and spreads gradually throughout the muscular system. It is aggravated by voluntary effort, and may cease during sleep in mild cases. Speech is altered. Hysteric tremors may also exist. Great irritability and restlessness are common. Aphasia, hemiplegia, hemianesthesia, and peripheral neuritis with palsies, occur. There is no atrophy, nor are the reactions of degeneration present in the paralyzed muscles. Severe pains may be present in the extremities, including the joints, and grave cerebral symptoms occasionally develop (stupidity, headache, loss of memory, insomnia, hallucinations, delirium, coma, convulsions, and confusional insanity). Albuminuria with anasarca may occur. The effects of chronic hydrargyrism in women upon their offspring are also important, the children being rachitic, weak, sickly, and prone to tuberculosis.

Diagnosis.—The history, the characteristic tremors, paresis, and mental irritability are significant. In the absence of a history of exposure to mercury, the differentiation from *progressive general paresis*, *disseminated sclerosis*, or *paralysis agitans* may be more or less difficult.

Prognosis.—Recovery is common upon the removal of the source or on removing the patient from the source of the poisoning. Fatal

terminations rarely ensue, and then in cases of mercurial encephalopathy of a grave type and with a tendency to idiocy.

Treatment.—Prevention of further poisoning is imperative, and elimination is to be promoted. Potassium chlorate, with the tincture of myrrh, and astringents are useful for the occasional stomatitis and salivation. Potassium iodid and also sulphur baths may be used to aid in the elimination of the mercury. Iron, cod-liver oil, good food and fresh air, and a free activity of the emunctories are useful. For the marked tremor, sedatives (*e. g.*, codeine, chloral, bromides, belladonna) are recommended. Electricity may be resorted to for the paresis.

FOOD-INFECTION AND PTOMAIN-POISONING.

In recent years there have been reported an increasing number of cases of serious illness that have been traced to infected and contaminated food. Undoubtedly many such instances are now brought to notice that in former times were attributed to other causes, or that were not diagnosticated because of a lack of knowledge. On the other hand, the increased consumption of canned and preserved meats has certainly augmented the liability to poisoning from these products, as the reports of cases show. Lack of care in the inspection and selection of the meats, uncleanliness, and sometimes unscrupulousness, in their handling and preparation, must result in infection, putrefaction, and toxicity. The infection of the food may be due to (1) disease of the animal or plant from which the food is derived; (2) microbic inoculation of the food after derivation and before ingestion by human beings; (3) infection by toxicogenic bacteria, and the presence of ptomains or toxalbumoses. The transmission to man of such affections in animals as tuberculosis, anthrax, glanders, and pleuro-pneumonia, by eating the infected meat, has been sufficiently proved. Again, meat and milk may become infected, before being ingested by the patient, by pathogenic microorganisms, as of typhoid fever and diphtheria, or from the production of toxins owing to the action of non-pathogenic putrefactive microorganisms. A great many instances of food-infection, particularly of meat and milk, have been shown to be due to the presence of saprophytic germs, this happening even when the articles of food have been obtained from healthy stock and have been kept free from specific pathogenic bacteria. It is not, however, the saprophytes themselves in all cases, but the poison developed in the food before it is eaten or formed in the body afterward, that produce the symptoms and sometimes death. According to Novy, some of the saprophytic bacteria with which food is infected outside of the body, under certain conditions, are capable of living in the body as parasites, especially on dead matter, and there become toxicogenic.

The chronic poisons or ptomains resulting from the action of the saprophytes in foods are called "putrefactive alkaloids;" those bacterial products of a proteid nature are called "toxalbumins" or "toxalbumoses." The latter, according to Vaughan, are more frequently present in infected foods. They are all absorbed from the digestive canal.

Poisoning by Infected Milk and Milk-products.—It is now well known that the cause of the high mortality-rate among infants in hot weather

is traceable directly or indirectly to the "summer diarrheas" in children fed artificially, wholly or partially, with milk infected by numerous varieties of saprophytic germs and thus poisoned by ptomains, such as tyrotoxicon. This special chemical poison has been isolated by Vaughan, and discovered by him in cheese. It has also been found in ice-cream, frozen custards, and cream-puffs, and has caused poisoning-symptoms mainly of acute gastro-intestinal inflammation, "constriction of the fauces," nausea and vomiting, sharp, griping intestinal pains, headache, thoracic oppression, chilliness, dizziness, and sometimes purging, followed by relief in mild cases. In the severe and long-continued forms, however, exhaustion may supervene, with subnormal temperature, coma, collapse, and death in the graver cases. No chemical or physiologic antidote is known. Elimination may be assisted, and stimulation is needed. Irrigation may be employed for the former in both stomach and bowels. Strychnin, nitroglycerin, atropin, and the aromatic spirits of ammonia are most effective as stimulants.

Meat-poisoning.—Various tainted meats, as mince-meat "warmed over," veal pie, carelessly-kept chicken salad, badly-preserved and canned meats, partially-decayed sausages (*botulismus*) have caused violent symptoms of poisoning. Diseased raw and partially-cooked meat has also been eaten with disastrous results. Prolonged cooking may fail to destroy the toxic action of certain ptomains in infected meats; also, that meat that has been cooked and kept under certain conditions may become infected with bacteria as well as when it is raw. Putrid meat, however, has been known not to cause toxic symptoms. Gärtner's bacillus (*B. enteritidis*) is probably the exciting cause of meat-poisoning.

The *symptoms* caused by the poisoning are—"(1) those due to a true infection; (2) those due to simple poisoning" (Mann). Cases of the former group run the usual course of an infectious disease, often simulating typhoid fever. Those under the second division manifest the symptoms of a violent gastro-enteritis, with vomiting, intense colicky pains, purging, fever, accelerated pulse, nervous prostration, great muscular weakness, and cramps in the calves of the legs. Often a subsequent subnormal temperature, extreme depression, convulsive movement, vertigo, dimness of vision, dyspnea, somnolence, great soreness of the mouth, collapse, and sometimes death supervene. The mortality-rate varies from 15 to 55 per cent. of all the cases.

Differential Diagnosis.—Arsenic-poisoning may have symptoms similar to those of ptomaïn-poisoning. But, as Harrington[1] points out, there are three chief points of difference: in arsenic-poisoning there is swallowing because of pain; in ptomaïn-poisoning the pupils are usually dilated and the muscular prostration is almost as extreme as a palsy.

The *treatment* is largely eliminative, symptomatic, and supportive. The prophylactic measures, private and public, are generally obvious.

Poisoning by Fish (*Ichthyismus*) **and Shell-fish.**—Many instances of this serious form of intoxication have been produced. The fish may contain certain poison-glands, ovaries, etc. Especially is this true of certain species known in Japan, one of which is believed to cause the disease called "Kakke," which prevails during the summer months in Tokio. A certain species of fish (*Clupea venenosa*) inhabiting the West

[1] *Boston Medical and Surgical Journal*, Dec. 14, 1899.

Indian waters is supposed to be always poisonous, although the source or true character of the poison is doubtful. In Russia, many cases of ichthyismus have resulted from eating both the fresh and preserved sturgeon and salmon meat that are affected with an infectious disease peculiar to the fish. In Germany and other parts of middle Europe a severe form of gastritis called "Barbencholera" follows the eating of sick barbels.

The use of tainted preserved and canned fish, eels, oysters, mussels, crabs, lobsters, and the like, is more frequently the cause of symptoms of poisoning, however. Brieger's *mytilotoxin*, the active poison formed in some mussels, and the eating of which at Wilhelmshaven caused several epidemics, is probably developed only under certain favorable conditions of saprophytic infection. Devilled crabs, lobsters, and salad have also caused severe gastro-enteritis because of contamination with germs producing ptomains. Oysters have been accused of conveying typhoid infection (*vide* p. 26). The *symptoms* of fish- and shellfish-poisoning are variable. Sometimes marked cerebro-spinal manifestations predominate, with convulsions and paralysis. Dryness and constriction of the throat, dizziness, labored respiration, disturbed vision, jerky speech or aphonia, perhaps rapid pulse, loss of coördination, numbness, coldness of the extremities, dilated pupils, paresis, collapse, and death within a few hours, may ensue.

Other cases have a pronounced gastro-intestinal or choleraic group of symptoms, with nausea and vomiting, pain, tenesmus, and mucous and bloody stools. In some of them marked cutaneous irritation is shown by erythema, great heat and itching, urticaria, and swelling. Dyspnea, lividity, and sometimes delirium, have also been noted. The *prognosis* is grave in many instances. The *treatment* is similar to the above—namely, emetics, purgatives, enemata, and lavage. The indications are to be provided for as they arise.

GRAIN- AND VEGETABLE-POISONING.

Ergotismus.—Epidemics of ergotism have resulted from the continued use of meal made from contaminated grains grown on virgin soil. The parasite (*claviceps purpurea*) is a fungus that infests rye and other grains; it does not, however, grow readily where the soil is well cultivated, and epidemics of ergot-poisoning are much less frequent than formerly, if we except certain places in Spain and Russia. According to Kobert, three poisonous substances are found in the ergot: ergotinic acid, sphacelinic acid, and cornutin. The first of these is not poisonous when taken into the stomach; the second is supposed to cause gangrene; and the last produces grave effects on the nervous system, and is found only in fresh ergot, hence the greater prevalence of nervous manifestations in sickness that breaks out soon after harvest.

The nervous symptoms are remarkable for their convulsive characteristics (*ergotismus convulsivus*). Prodromes of weakness, tingling in the extremities, and headache may exist for several weeks before the spasms come on. The formication increases, and cramps and contractures, with flexed wrists and extended feet and toes, seize the patient. In severe cases epileptoid convulsions occur and may prove fatal. Delirium and,

in very chronic cases, dementia may supervene. Recovery is slow, and the contractures may persist for some time, with muscular atrophy and anesthesia. In some interesting instances there may appear nervous symptoms resembling locomotor ataxia ("ergot tabes"), owing to posterior spinal sclerosis. Abortion results in pregnant women.

Gangrenous ergotism (*ergotismus gangrænosus*) is characterized by dry gangrene of the hands and feet, usually of the fingers and toes. Before the gradual blackening appears, there may be formication, pain, spasm, numbness, and coldness. As mortification and the line of demarkation progress, the parts drop off bit by bit, and fever may attend the sphacelation. Pneumonia (septic) may sometimes complicate this malady. The fatality has been considerable in some epidemics. The *treatment* of ergotism is entirely symptomatic.

Maidismus or Pellagra.—This is a chronic nutritional disturbance due to poisoning from eating contaminated corn-meal bread. The disease prevails extensively among the poorer classes in Italy, Roumania, Spain, France, and in the southern portions of the United States.

Pathology.—Structural changes have been found in the cord, *e. g.*, degeneration of the lateral columns in the dorsal region and of the posterior columns in the cervical and dorsal regions, and fatty degeneration and ulceration of the viscera.

Etiology.—The origin of the infection of the maize is said to be bacillary, the latter causing putrefactive or fermentative changes in the fresh, moist corn-meal, with the production of ptomains. A second theory ascribes the disease to a form of maize intoxication. Among *predisposing causes* are *age*, most cases occurring between twenty and forty years; *race*, negroes being more susceptible than whites, and *season*, the spring furnishing most instances.

The *symptoms* at the beginning are languor, debility, indigestion, anorexia, vomiting, and occasionally diarrhea. This is soon followed by erythema, pain, and roughness of the skin. Exfoliation of the latter reveals a suppurating surface. The erythema affects usually the exposed parts, beginning on the backs of the hands, the face, neck, and, sometimes, the feet. At first the involved areas resemble an ordinary sunburn, later becoming darker and desquamating, leaving behind some degree of pigmentation. In some instances bullæ and vesicles may appear. Recurrences are common and lead to puffy swelling and a thickened, indurated condition of the skin, which is now darker in color. Finally, atrophy of the skin may occur. The mucous membrane of the mouth and throat becomes inflamed with shedding of epithelium, leaving behind a raw surface so that mastication is painful. In severe cases, paresthesiæ, spasms, paraplegia (p. 1153), headache, backache, delirium, and a suicidal mania may occur. Idiocy and profound cachexia may result from numerous attacks.

The *diagnosis* rests on the association of the cutaneous, gastro-intestinal, and nervous, as well as other general, features.

Course and Prognosis.—The course of pellagra is slow and interrupted by recurrences. Generally, the prognosis is grave in the severer forms of the disease. On the other hand, if the cause be removed, and the complaint not far advanced, amelioration of the symptoms and, rarely, cure is prone to occur.

Treatment.—Prophylaxis by thorough drying and careful storing of the maize is to be aimed at. In early cases, change of food and environment may prove effective. At a later stage, the treatment should be directed to the leading symptomatic indications. No methods thus far used can be said to be curative.

Lathyrismus is an intoxication caused by the seed (used in the form of meal) of three varieties of vetch or chicken-pea, viz. *Lathyrus cicera, L. sativus*, and *L. clymenum*, or, respectively, red, German, and Spanish vetch. The meal is generally mixed with that obtained from other cereals. Its use for several hundred years has been observed to cause leg-stiffness, passing into a transverse myelitis, with sensory and motor paraplegia. Spasticity and exaggerated tendon-reflexes may remain for some time after the paralysis subsides. Slight fatty degeneration was noted by Cautain in excised bits of muscle. Very chronic cases may die in paralysis, from the toxic effects of the poison, which, thus far, has not been separated.

Mushroom-poisoning.—Though not so common as formerly, poisoning from eating non-edible mushrooms occurs now and then, owing to ignorance or carelessness in gathering, keeping, and cooking them. *Fresh morels* are poisonous, while those that have been dried and boiled are not so, because of evaporation or solution of the contained poison.

The *red agaric* (*amanita muscaria*), on account of the poisonous alkaloid muscarin that it contains, may cause very severe symptoms. These are nausea, vomiting, diarrhea, hemoglobinemia, hemoglobinuria, and jaundice (*probably hepatogenous*) in the case of fresh morel-poisoning (Strümpell). Tetanic and epileptiform convulsions give a slow pulse, dilated pupil, disturbed vision, salivation, coma, and death in the gravest cases of red-agaric intoxication, in addition to the symptoms of gastro-intestinal irritation.

The *treatment* is symptomatic. Emetics, purgatives, stimulants, and, in red-agaric poisoning, atropin, for its physiologic antidotal effect, are usually indicated.

OBESITY.

(*Polysarcia Adiposa; Lipomatosis Universalis.*)

Definition.—Corpulence, or the presence of an excessive amount of bodily fat, may be said to begin to take the form of a disease when it becomes an inconvenience or impairs the bodily functions. Many recent writers regard obesity as being symptomatic of a variety of underlying pathologic conditions rather than a disease.

Pathology.—The chief alteration is the marked and, in some instances, colossal increase in the fat deposit throughout the body. Not only is the adipose tissue greatly increased in localities where it is normally found, but the various internal organs and tissues that are normally quite or nearly free from fat may in obesity show a decided fatty infiltration. The round fat face, "double chin," broad and deep chest, large waist, thick and prominent, sometimes overhanging, abdominal *panniculus*

adiposus, and bulky, cylindric, and apparently shortened extremities, are familiar appearances *postmortem* as well as *antemortem*.

There may be differences in the number and size of the fat-globules in the histologic elements. Thus, in the plethoric form of obesity the cellular fat-globules are larger than those of the anemic or hydremic form. The *heart* is overlaid with fat. Hypertrophic dilatation is often present.

The *arteries* may show fatty changes and chronic endarteritis with sclerosis. The *veins* are often affected with varicosities. In plethoric obesity the blood shows an increase in specific gravity (1.062–1.070). The erythrocyte count may rise to 6,000,000 per c.mm. Passive congestion and edema of the *lungs*, secondary to the cardiac weakness, are common. The *liver*, *lungs*, and *kidneys* may be enlarged, owing to fatty infiltration. Chronic interstitial nephritis may form a complication.

The *stomach* may be dilated, and often shows a catarrh of the mucosa.

Pathogenesis.—Obesity is probably dependent on a disturbance of cell-activity, and this disturbance of metabolism may be transmitted through heredity (*vide infra*). The overuse of carbohydrates leads directly to fat-increase. The consumption of proteins may also result in a fat-forming non-nitrogenous residue, which if not oxidized may produce fatness (see also Etiology).

Etiology.—Among the chief *predisposing* conditions are heredity, climate, habit, occupation, temperament, age, and sex. Among 543 of my cases, in which the family history was noted, heredity was distinctly traceable in 60.7 per cent. Gout was either in association or occurred among the antecedents in 43.2 per cent. of these cases and the same was true of "rheumatism" in 35.5 per cent. The condition of 10 dates from longer or shorter periods of enforced rest, *e. g.*, following accidents and infective diseases, as typhoid fever (in 4.7 per cent. of 543 cases). The disease dated from child-birth in 16.2 per cent. of the cases and from marriage (apart from child-birth) in 4.8 per cent. among 437 females. The menopause has little if any influence. Corpulence is much more frequent among the inhabitants of hot, moist climates, and of low countries of the temperate and arctic regions. Thus it is commonly observed among Orientals, Dutchmen, South Pacific Islanders, Southern Italians, and certain African races. Sedentary habits and occupations form common predisposing factors. The sluggish, luxury- and rest-loving, phlegmatic temperament also favors an abnormal fat-deposition. As regards the age, polysarcia generally makes its appearance in persons of advanced middle life, between forty and fifty years, while hereditary obesity dates from infancy and early childhood; in women, it may appear at puberty and between thirty and forty years of age. Women, and especially Jewesses, seem to be more subject to corpulence than men. Congenital anomalies and monstrosities (idiots, cretins, acephali), also anemics and hemiplegics, are often excessively fat.

The *exciting* causes of obesity are especially the ingestion of too much fat-making food, the intemperate use of alcoholic beverages, especially beer, ale, and porter, with or without deficient exercise. The fat may be derived from an excess of albumin, fat, or carbohydrates. An excessive diet of starches and sugars acts indirectly as a fat-producer by

lessening the oxidation of the ingested fat and of the fat formed from proteids, because the carbohydrates themselves are so readily oxidized.

Symptoms.—Obesity is not accompanied by any bodily symptoms at first. Except some inconvenience, and a sense of burdensomeness during walking or working, nothing may be complained of for years. With the progressive development of the disease, however, and particularly with the involvement of the viscera, subjective manifestations increase in number and intensity. Usually the earliest troublesome symptom is *breathlessness* on exertion, due to a weak heart and to the hampering of respiration by heavy chest-walls and the upward-crowded diaphragm. In *plethoric* individuals the face is red and congested, as are also the mucous membranes (conjunctivæ, labiæ). In *anemic* subjects (usually women) the skin is pale, the muscles are flabby and weak; the pulse is small and compressible, and dyspnea, palpitation, inclination to rest often and sleep much, and dizziness (symptoms of anemia and chlorosis) are manifested. On the other hand, in plethoric, corpulent subjects (usually men) the muscles are firm and strong, and the pulse and heart-beats vigorous; later, however, the latter becomes weak and irregular. Brachycardia is not infrequent. The signs of fatty heart (*vide* p. 694) are obtained on physical examination. Muscular power may diminish and irregular fat masses (in the anemic variety) in subcutaneous tissue, are obtained on physical examination. Muscular power may diminish rapidly. Intercurrent acute infections (typhoid fever, pneumonia) are badly borne, and hyperpyrexia is usually associated with them. In the anemic form the blood-changes are of the chlorotic type, while in the plethoric both the hemoglobin percentage and erythrocytes are increased.

The *liver* may show enlargement. The passive congestion of the *respiratory mucous membrane* is often signalled by cough and distressing dyspnea and attacks of asthma. Profuse sweating is common. There may be *polyuria* or *oliguria*, according to the activity of the skin and kidneys at the same time. Uric acid and the urates are usually found to be increased.

Symptoms of *gastric catarrh* and *gastrectasia* may occur. Great thirst and bulimia are noted in some instances. Constipation may be followed by chronic diarrhea. Sexual desire is often abated, and azoospermia is not rare. Corpulent women often suffer from uterine displacement and prolapse. Amenorrhea, sterility, endometritis (congestive), leukorrhea, and an aggravated climacteric are seen in obese women also. The skin is often irritated (intertrigo) by the excessive sweating, and by the friction of cutaneous surfaces in the folds of fat, as under the breast, at the abdominal and inguinal folds, and around the scrotum and labia. This may be followed by eczema. Painful excoriations, pruritus, acne rosacea, and alopecia are also not uncommon.

There are types of adiposity with an increased assimilation limit for carbohydrates, often with dry skin, subnormal temperature and pulse, that are due to insufficiency of the posterior lobe of the pituitary body.

Complications.—Hernia, cardiac asthma, bronchitis, pulmonary congestion, edema, arteriosclerosis, albuminuria, glycosuria, anginal attacks, Cheyne-Stokes respiration, cerebral hemorrhage, and coma may manifest themselves as the precursors of the final stage.

Diagnosis.—This is not difficult in most cases. Care should

be exercised in detecting complications and sequelæ. In myxedema the skin is thick and inelastic, and the physiognomy much altered, while the lips, tongue, nostrils, and mouth are all thickened by infiltration.

The **prognosis** will depend upon the peculiar features of each individual case, the cause and its removability, and upon the variety, degree, symptoms, and prevailing complications.

Treatment.—**Prophylaxis** is important in the earlier years of those showing an hereditary predisposition to corpulence. The fat-forming (farinaceous) substances must be diminished in the dietary. The proportions of fat and proteid in the food must be regulated according to the amount of muscular activity, and the latter should be encouraged in fresh air, along with cool bathing. In those predisposed to polysarcia, all imprudences in eating and drinking should be cautioned against, and the quantities of various articles of food and the time of eating regulated. Outdoor sports and gymnastics should be also gauged accordingly.

The **dietetic** treatment of confirmed obesity is all-important. Inseparable from this is the stimulation of the bodily forces that oxidize and destroy the fat. These two means are utilized in the principal methods of treating obesity, and that method must be selected which invigorates, while at the same time it involves no weakening of the patient.

The principal systems of dietary are those known by the names of Banting, Ebstein, and Oertel.

In "Bantingism," sugars, fats, and starches are greatly reduced in the diet-list; water, however, is not restricted, and vinous and spirituous liquors are permitted. In those of a rheumatic or gouty diathesis Banting's heavy proteid and alcohol dietary is not to be recommended. It is best, I think, to exclude alcohol in most cases, owing to its effect in diminishing tissue-oxidation and in retarding cell-metabolism. This method fails to secure elimination of waste products.

In Ebstein's diet-list more than double the amount of fat and carbohydrates is permitted as compared with Banting's list, whilst the albuminous substances are diminished. Fat is freely allowed, as this does not increase stored fat (?), but tends to impair the appetite, while sugar and potatoes only are strictly forbidden.

Oertel allows more fat than Banting, but less fat and more (about double the quantity) proteids and carbohydrates than Ebstein. The amount of *free* water permitted daily is only one pint; about one pint additional in other food is allowable. This method is adapted to cases of obesity with feeble hearts and of the anemic form.

Oertel[1] writes: "The body stores up fat if more than 118 grams of albumin and 259 grams of fat, a total of 277 grams (2894 calories), are taken in. On the other hand, 110 grams of albumin and 600 grams of starch, a total of 710 grams (2944 calories), may be given without producing a deposit of fat. With a mixed diet the limit lies near 118 grams of albumin, 100 grams of fat, and 368 grams of starch, a total of 586 grams (2923 calories)." His diet-table for obesity is appended:

[1] *Twentieth Cent. Pract. of Med.*, vol. ii., pp. 698, 699.

	Albumin.	Fat.	Carbohydrates.	Calories.
Minimum	156	25	75	1180
Maximum	170	45	120	1608

Oertel gives a special diet-list in circulatory disturbances.

On the basis of Voit's laws, Strümpell recommends in the average cases 125 gm. (4 oz.) or more of albumin, 40 gm. (1¼ oz.) of fat, and 150 gm. (462 oz.) of starch. Schwenniger's rule differs from Oertel's merely in the forbidding of liquids with the meals and in permitting their use only after two hours have elapsed. Yeo's diet-list is also a useful guide. In plethoric obesity a judicious rearrangement of the food (some increase of the albuminoid substances), coupled with sufficient muscular exercise (walking, horseback-riding, bicycling, rowing, swimming, gymnastics), accomplish successful reduction, as a rule. Moritz[1] has found an exclusive milk diet extremely effectual.

Increasing weakness of the heart with an impeded circulation naturally diminishes the excretion of water by the cutaneous and renal routes. In such cases the circulatory system must receive careful attention and the consumption of fluid must be limited. If evidences of anemia be present, the amount of liquid may be much restricted and the fat-forming dishes rigidly excluded. "The hydremic form must be opposed by the ingestion of an abundance of albuminoid material, of fat-producing substance, and the hydrocarbons" (Oertel). Sabbe and Furet[2] recommend a regimen from which salt is entirely eliminated, in connection with the ingestion of fluids in abundance. The organism, in order to maintain its molecular composition, rejects the excess of fluid, which carries off excrementitious products. Under any system of dietetic treatment the patient should be weighed accurately and frequently, and the food-limit be diminished or modified according to the results. The food may be weighed and measured at first, but the patient soon learns to estimate by bulk the requisite quantity of each substance.

The following dietary illustrates what may be ordered in some cases:

Morning Meal.—Fine wheat-bread, 1¼ ounces (40.0); a soft-boiled egg; milk, 1 ounce (32.0); sugar, 77 grains (4.9); coffee, 4¼ ounces (136.0).

Noon Meal.—Soup, 3 ounces (96.0); fish, 3 ounces (96.0); roast or boiled beef, veal, or game or poultry, 6 to 8 ounces (192.0–256.0); green vegetables, 1½ ounces (48.0); bread, 1 ounce (32.0); fruit, 3 or 4 ounces (96.0–128.0); no liquid (or only 4 or 5 ounces—120.0–148.0 c.c.—of very light wine).

Afternoon Meal.—Sugar, 77 grains (4.9); coffee, 4 ounces (128.0); milk, 1 ounce (32.0); occasionally bread, 1 ounce (32.0).

Evening Meal.—Caviare, ⅓ ounce (10.6); one or two soft-boiled eggs; beefsteak, fowl, or game, 5 ounces (160.0); salad, 1 ounce (32.0); cheese, 1 dram (4.0); bread, rye or bran, ½ ounce (16.0); fruit or water, 4 to 5 ounces (120.0–148.0).

Galisch recommends the simple measure of reducing the amount of food taken after midday, so as to lessen nourishment at night, out of which to build fat.

The **mechanical treatment** (to increase oxidation), by exercise, is to be used in conjunction with the dietetic. The form of the exercise, and

[1] *Münch. med. Woch.*, 1908, xxx., p. 569.
[2] *Revue de Médicine*, 1905, No. 9, p. 674.

also the time and frequency, must be adjudged for each case (*vide supra*). When cardiac dilatation and myocardial degeneration (fatty) are the cause of symptoms of precordial distress, dyspnea, and palpitation, resort may be had to Oertel's system of graduated walking on the level or climbing along "health paths" (*vide* Fatty Overgrowth, p. 694). Or, the well-known Nauheim or Schott treatment may be used. Great care must be exercised in prescribing the mechanical treatment in obese persons who have atheromatous vessels.

Allard[1] recommends the employment of a vibrating ball controlled by an electric motor in circumscribed obesity.

The **medicinal** treatment is neither satisfactory nor successful. Causative or associated conditions—*e. g.* gout—may present special therapeutic indications. The juice of the phytolacca berry may reduce the weight, but is harmful.

Recently, the use of thyroid extract has come into favor. Leichtenstern, Wendelstadt, Ewald, and others have reported success in a number of cases, especially in those exhibiting the anemic, flabby, "myxedematoid" form of obesity. The loss of weight was from 2 to 3 pounds (1–1.5 kgms.) in one week, and as high as 20 pounds in two to four weeks. In two of my own cases belonging to this category the use of thyroid extract (desiccated) in small doses (gr. j—0.0648, t. i. d.) caused a progressive loss of weight at the rate of 4 and 6 pounds per week respectively, without injury to the general health. In cases in which dietetic measures with exercise fail, thyroid insufficiency should be suspected, and thyroid treatment instituted. Thyroidin, the active principle of the thyroid gland, as shown by Baumann and Ross, and iodothyrin give results that are perhaps as good as those of thyroid-feeding. Symptoms of thyroidism are the signal for a reduction in the dosage of thyroid extract (*vide* Myxedema, p. 502). Hematinics are indicated in the anemic variety of obesity. Finally, the treatment must be adapted to the special case, and also varied from time to time to meet indications and complications as they arise.

ADIPOSIS TUBEROSA SIMPLEX.

The writer has described a condition which resembles adiposis dolorosa (Dercum's disease) clinically, but differs from the latter in that it is dependent upon general obesity, and is, therefore, amenable to treatment.

"Circumscribed fat masses appear in the subcutaneous tissues; they form distinct, moderately dense, slightly movable, somewhat flattened tumors, ranging in size from a bean to that of a hen's egg. Their number varies all the way from one-half dozen to two dozen or more. These moderately firm fat-nodules are not distributed over the entire body, but in some cases are confined to the extremities, particularly the lower, and in others to the abdomen. The tumor masses show no tendency to fuse together, and are not elevated above the surrounding surface; they are sensitive to the touch, and may be the seat of pain, which varies in intensity within rather wide extremes, being moderately severe and distressing in rare cases and trivial or even absent in the majority of instances. The lymphatic glands are not involved, and the skin remains soft, flexible, and non-adherent. The mental processes are normally

[1] *Revue de Therapeutic*, 1905, No. 6, p. 191.

active, and also the muscles; asthenia is not present, and there is no more indisposition to physical exertion than is observed in cases of obesity, as a rule. The knee-jerks are present, and the cutaneous sensibility is unaltered, in some cases at least. The mammæ and abdominal panniculus adiposis may be overhanging or pendulous, but not in all cases. It is an uncommon condition, since it was noted in only 4 out of a total of 324 cases."[1]

HEAT-STROKE.

(*Sunstroke; Insolation; Thermic Fever; Heat-exhaustion; Heat-prostration.*)

Definition.—A diseased condition the effect of exposure to excessive heat.

Pathology.—*Rigor mortis* is marked and comes on early. The high temperature of the cadaver accelerates the putrefactive changes, which also appear early. There is considerable venous engorgement of the brain and of the cerebral and spinal membranes; also of the lungs, spleen, and conjunctiva. The blood is fluid and dark, and the corpuscles are crenated and do not tend to form rouleaux. Parenchymatous changes in the liver and kidneys may be found. Rigid contraction of the left ventricle is a notable feature, while the right ventricle is usually dilated with blood. Van Gieson's recent report of the cellular pathology of the cerebro-spinal system in 3 cases of sunstroke in New York shows an acute parenchymatous degeneration of the neurons of the whole neural axis similar to that of "a species of auto-intoxication."

Etiology.—Anything that lessens bodily resistance to external high heat predisposes to heat-stroke. Thus, privation, unsanitary surroundings, fatigue of body or mind, emotional excitement, worry, and excessive fretfulness, overeating, indulgence in alcoholics (especially), clothing suitable for cold weather, worn on hot days, and previous attacks of sunstroke, are all conducive to heat-stroke on exposure to high temperature. Males are affected more often than females, and the condition is rare in childhood. The colored race is more resistant than the white to the effect of the direct solar rays.

Sunstroke occurs in persons (on land) working hard under the direct rays of the sun, in an atmosphere that is very hot and humid, still, and sultry. Soldiers on the march and heavily accoutered, masons, bricklayers, hod-carriers, roofers, drivers, farmers, and other out-door laborers are particularly subject to insolation.

Heat-stroke and **thermic fever** are terms more appropriately applied to those similarly affected in midsummer while working in places not exposed to the sun, but yet close, confined, and excessively hot, such as glass-works, foundries, ocean steamers, stoke-holes, boiler-rooms, steam laundries, sugar-refineries, kitchens, and the like.

Heat-exhaustion (*prostratio thermica*) is caused under similar conditions as the preceding, but manifests dissimilar effects.

The majority of the cases of sunstroke occur between 2 and 5 P. M., although heat-stroke and heat-exhaustion may occur at night as late as 10 or 11 P. M., as among bakers, night engineers, and hotel cooks.

[1] *Amer. Jour. Med. Sci.*, March, 1908, by the writer.

The direct cause of the symptoms of sunstroke, heat-stroke, or heat-prostration is the action of the excessive heat upon the heat-centers, or upon the vasomotor center or nerves (H. C. Wood), the former of which, if paralyzed, produces "*thermic*" or "*heat-fever*," while the latter, if paralyzed, produces *heat-exhaustion*. Lambert and Van Gieson,[1] after a clinical and pathologic study of 805 cases, hold to the view that the immediate basis of sunstroke is autotoxic, with heat only as a contributing cause. Sambron contends for the infective nature of heat-stroke and thus explains its endemic and epidemic proclivities.

Symptoms.—Two forms of heat- or sunstroke are usually met with: (1) The *asphyxial* or *apoplectic* form; (2) the *hyperpyrexial form*. Flint believes that the majority of the cases of sunstroke are combinations of apoplexy and exhaustion. Vallin puts all cases of insolation into two classes: the first, sthenic or asphyxial, corresponding to our hyperpyrexial or congestive variety; the second, asthenic or syncopal, corresponding to our heat-exhaustion. Mixed forms may occur quite frequently, the most prominent symptoms being referable to the organs suffering the most, as the cerebro-spinal system, heart, lungs.

Heat-apoplexy (*asphyxial sunstroke*) is probably the least frequent form. There may be sudden premonitions, or dizziness, chromatopsia, throbbing headache, cessation of sweating, or dyspnea. Sometimes the patient, while at work in the sun, suddenly falls unconscious, a few convulsions may occur, and in this state he may die with symptoms of cardiac failure. More often, insensibility is not so profound as complete coma, there is much restlessness, epigastric "cramp" may be complained of, also a sense of thoracic oppression, and occasionally there are nausea and vomiting. The headache may be intense, the face is flushed, the pulse is rapid and full, the temporal and carotid arteries are bounding, the breathing may be labored and stertorous, the pupils are contracted. The skin is hot and dry, and may show petechiæ. The tongue is coated. A wild delirium has been observed in some cases. The temperature may be subnormal, and is not higher than 102° F. (38.8° C.) in many instances. In others, a mild degree of thermic fever may be associated with the apoplectic condition, the thermometer registering 104°–106° F. (40°–41.1° C.). In fatal cases the coma becomes deeper and deeper, the pulse more rapid and feeble, and Cheyne-Stokes respiration may precede the termination. A "mousey" odor about the body has been noted. In favorable cases the temperature falls to normal by lysis in three or four days, consciousness being rapidly regained at the same time.

The *hyperpyrexial* variety comprises the numerous cases of marked sunstroke that resemble the preceding type, with the addition of an intensely high temperature (*thermic fever*). The patient may suddenly become comatose and die in an asphyxiated condition, with a temperature as high as 110°–115° F. (43.3°–46.1° C.) or even higher.

Sometimes prodromes, as an anorexia, progressively increasing physical weakness, cramp-like abdominal pains, irritability and restlessness, vertigo, colored and blurred vision, lack of sweating, a "bursting" headache, and an irritable bladder may exist for several days. A subconscious (automatic) state, in which the patient may be unaware of his surroundings, although walking or even working, may be noted

[1] *Med. News*, July 24, 1897.

for hours before he is stricken down. The onset is marked by hyperpyrexia; the skin is hot, burning, dry, sometimes flushed and red, and sometimes cyanotic and clammy; the eyes are suffused or "staring and filling," with pin-point pupils. There is a full, rapid, and non-compressible pulse, and coma may be present. Clonic spasms may alternate with either muscular rigidity or flaccidity. Delirium, moaning, jactitation, and explosive expiratory sounds may occur. There is frequently incontinence of both feces and urine. The temperature is very high in most of the cases, varying from 105° to 112° F. (40.5–44.4° C.).[1] The pulse-rate varies with the temperature, from 90 to 160 beats per minute. The respirations are also increased to 24–50 per minute. Many of the alarming symptoms, including the high fever

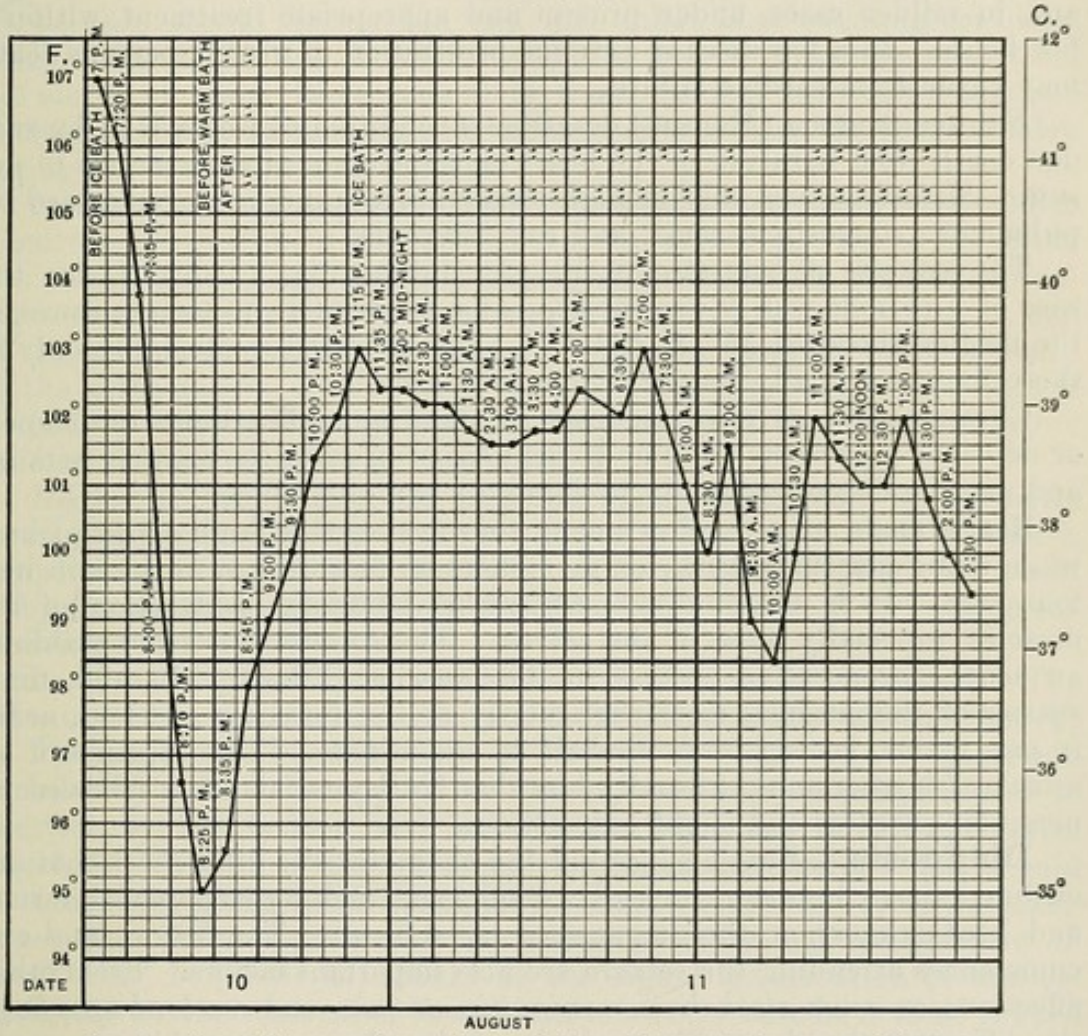


FIG. 81.—Chart of a case of sunstroke. C. B., aged twenty-nine years. Recovery.

unconsciousness, cyanosis, dyspnea, and convulsions, may greatly subside during and after the use of the cold bath. Secondary exacerbations occur for a few days before convalescence is established in the favorable cases (*vide* chart Fig. 81). Some patients never rally and die in a state of asphyxia. Retention of urine (suppression) is observed at times, and particularly in those accustomed to the use of alcohol. Leukocytosis is noted, besides the crenation of the erythrocytes.

[1] Lambert (*loc. cit.*) reports a case in the N. Y. Hosp. of 117.8° F. (47.6° C.).

Fatal *complications* of sunstroke are pneumonia, meningitis, uremia, and cardio-respiratory paralysis.

Heat-prostration or *heat-exhaustion* may come on gradually or suddenly, with prodromal symptoms (dizziness, faintness, headache, nausea, thirst, drowsiness, yawning, epigastric or lumbar pains, numbness, and tingling of the hands and feet). These are followed by coldness, clamminess, and pallor of the surface, marked muscular weakness and prostration, a small, febrile, rapid pulse, sighing breathing, syncope, and collapse in the graver cases. The temperature at first is subnormal (95° to 97° F.—35° to 36.1° C.), though mild thermic fever of from 100° to 102.5° F. (37.7°–39.1° C.) may be present. Consciousness is rarely completely absent. Recovery usually takes place within one or two days, and in milder cases, under prompt and appropriate treatment, within a few hours. In a few cases of extreme prostration in weakly persons death may ensue from cardiac failure.

Sun-traumatism (Manson) describes a condition characterized by sudden death from paralysis of the heart or respiration after exposure to the sun. *Siriasis* occurs only in high temperatures, and is characterized by pulmonary congestion, coma, and hyperpyrexia.

The sequelæ of heat-stroke are quite interesting. Osler relates the case of a patient who "was subsequently so sensitive to temperatures in the neighborhood of *75° F.* (23.8° C.) that he lived comfortably only in the cellar, and finally sought refuge in Alaska."

Chromatopsia, severe headaches, irritability and ugliness of temper, or delirium may occur in some patients as soon as warm weather sets in, and may be due occasionally to chronic meningitis (Wood).

Heat-cramps may develop among those exposed to high artificial heat while doing muscular work—*e. g.*, stokers on steamships, workers in iron foundries. It is essentially a continuous fibrillary contraction of the muscles, especially those of the calves. The condition is attributable to an acute degenerative process in the muscles (Edsall). Painful, tonic spasms of the muscles, more particularly of the forearms and legs, occur in paroxysms, lasting from one-half to one minute. The duration of an attack of heat-cramps is usually less than 24 hours, and it is followed by muscular soreness and slight exhaustion.

Diagnosis.—Bearing in mind the characteristic differences that are outlined above between sunstroke (asphyxial and hyperpyrexial forms) and heat-exhaustion, the diagnosis is not difficult. The history and circumstances attending the seizure are also important factors. From other affections, as *acute alcoholism*, *meningitis*, *uremia*, and *cerebral apoplexy*, the differentiation is readily made by noting the previous history, mode of attack, presence or absence of thermic fever, state of consciousness, urine, skin, pupils, pulse, respiration, and nervo-muscular apparatus. *Malaria* can be excluded by a blood examination.

Prognosis.—This is usually favorable in cases of heat-prostration and heat-cramps. It is less so in sunstroke, but in all cases it depends on the severity of the stroke, the previous health and habits of the patient, the complications, and the promptness and facility of the treatment. In general, cases in which unconsciousness lasts from 24 to 48 hours terminate fatally. The mortality-rate during a prolonged period of hot and humid weather may be very high, ranging from 15 to 50 per cent. In

New York City, during the week ending August 15, 1896, out of a total number of 1810 deaths, 648 were reported as due to sunstroke (Lambert).[1]

Treatment.—Prophylaxis.—This is highly imperative in hot, sultry weather, in which persons must work in the sun or in poorly-ventilated and highly-heated, closed places. Workmen should be taught and warned privately and publicly, as through the medium of the press and Health Board circulars, to take extra precautions during hot weather, to work and sleep in as well-ventilated rooms as possible, and to secure artificial ventilation, if necessary. They should live regular and temperate lives, avoiding alcohol and heavy eating; oat-meal water should be drunk, light-weight and light-colored clothing should be worn, and the direct rays of the sun should be avoided as much as possible. The condition of the skin should be watched and care taken that sweating continues freely. Shelter or rest should be sought at once if sweating stops. Cool wet cloths or green leaves should be worn inside a light straw hat, and sometimes it may be necessary for employers to shorten the hours of labor during the hottest part of the day.

Treatment of the Attack.—Cases of ordinary *heat-prostration* seldom require much treatment beyond the removal of the patient to the shade of a comparatively cool place, loosening all constricting clothing, spraying with cool water, the use of ammonia- or amyl-nitrite-inhalations, and of the aromatic spirits of ammonia or spiritus glonoïni by the mouth. If the temperature is subnormal and collapse threatens, a hot bath is advisable. Strychnin and digitalis may be used for a day or two to combat the nervo-muscular weakness.

Heat-stroke, especially the hyperpyrexial cases, must be promptly treated by the application of the ice-bath (ice floating in a tub of water), temperature about 40° F. (4.4° C.), or by rubbing, by the cold pack, or by the needle-spray with iced water for the purpose of overcoming the vasomotor depreciation.

In the *asphyxial* cases venesection is frequently indicated. The subcutaneous or intravenous injection of physiologic salt solution (F. A. Packard) may be a valuable procedure in many cases. External stimulation should be applied to the precordium by mustard and to the feet by hot bottles, and hypodermic injections of nitroglycerin, strychnin, atropin, brandy, camphor, or ether are useful. Ice should be rubbed over the head constantly. Care should, however, be taken to see that the temperature is not reduced too far. A temperature of about 102° F. (38.8° C.) should be the signal for cessation of the ice-bath, and for the removal of the patient to a cot, where he is to be rubbed dry and allowed to rest until an exacerbation of fever indicates the reapplication of the cooling measures. Ice-water enemata, with or without brandy, are often useful adjuvants. The needle-spray of cold water is an excellent nervous stimulant as well as antipyretic It is given while the patient lies on a Kibbee or netting cot, or on a cot covered with a rubber sheet so arranged as to drain into a pail or trough. Internal antipyretics are seldom well absorbed, and their depressant action is so well known as to discourage their use in place of hydrotherapy. Hutchinson, Coplin, and Bevan recommend highly the use of morphin to control the convulsions of heat-stroke. Chloroform has also been ad-

[1] *Loc. cit.*

vised. Artificial respiration in the asphyxial cases, kept up until other measures and stimulants have time to act, may be the means of saving life.

After the reduction of the hyperpyrexia the patient should be lightly covered on a cot placed in a cool place. An ice-cap should be applied to his head, and small pieces of cracked ice may be given to allay gastric irritability, with calomel to open the bowels if necessary. Albumin-water, skimmed milk, buttermilk, unfermented grape-juice, junket, and the like may be given for several days preparatory to the ingestion of heavier food. If, as sometimes happens, free diaphoresis does not come on after the reduction of most of the fever and the stimulating treatment, a hot bath may be given, and perhaps aided by the hypodermic injection of pilocarpin in urgent cases. *Sequelæ* must be treated on general principles.

The increased susceptibility to repeated attacks of insolation (after the first attack) makes it necessary to avoid exposure to heat ever after, and, if possible, to seek a cooler climate during the hot months.

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