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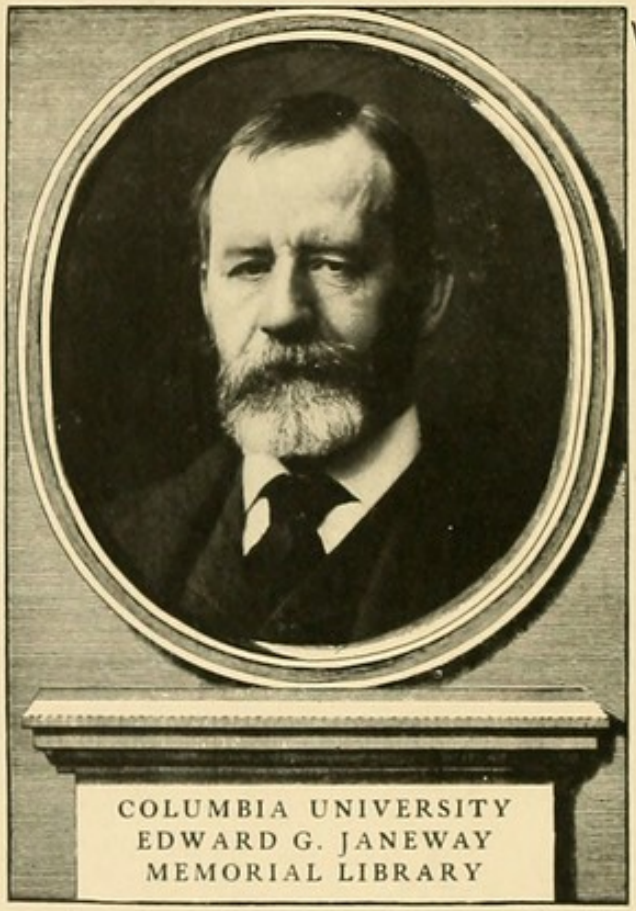
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THE PRACTICE OF MEDICINE

TYSON
AND
FUSSELL

BY DR. TYSON

A Treatise on Bright's Disease and Diabetes. With especial reference to Pathology and Therapeutics. Including a section on Ocular Changes in Bright's Disease and Diabetes. Second Edition Revised. Seven Colored Plates and 43 other Illustrations. Octavo. Cloth, *net*, $4.00.

"Dr Tyson's special interest and long experience in the observation and treatment of Bright's Disease and Diabetes cause the profession to welcome with pleasure this second edition."—*Bulletin of Johns Hopkins Hospital.*

THE PRACTICE OF MEDICINE

A TEXT-BOOK FOR PRACTITIONERS AND STUDENTS WITH SPECIAL REFERENCE TO DIAGNOSIS AND TREATMENT

BY

JAMES TYSON, M. D., LL. D.

EMERITUS PROFESSOR OF MEDICINE IN THE UNIVERSITY OF PENNSYLVANIA AND FORMERLY PHYSICIAN TO THE HOSPITAL OF THE UNIVERSITY; FORMERLY PHYSICIAN TO THE PENNSYLVANIA HOSPITAL; LATE PRESIDENT OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA; MEMBER OF THE ASSOCIATION OF AMERICAN PHYSICIANS, ETC.

AND

M. HOWARD FUSSELL, M. D.

PROFESSOR OF APPLIED THERAPEUTICS IN THE UNIVERSITY OF PENNSYLVANIA AND PHYSICIAN TO THE HOSPITAL OF THE UNIVERSITY; EPISCOPAL HOSPITAL; ST. TIMOTHY'S HOSPITAL, CHESTNUT HILL HOSPITAL; MEMBER OF THE ASSOCIATION OF AMERICAN PHYSICIANS; MEMBER OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA, ETC., ETC.

SIXTH EDITION, REVISED AND REWRITTEN

WITH SIX PLATES
AND 179 OTHER ILLUSTRATIONS

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1914

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TO THE MEMORY OF

PROFESSOR J. M. DA COSTA, M.D., LL.D.

LATE PROFESSOR OF PRACTICE OF MEDICINE AND OF CLINICAL MEDICINE

IN THE JEFFERSON MEDICAL COLLEGE OF PHILADELPHIA

PREFACE TO THE SIXTH EDITION.

It was with a desire to do all possible to bring my book thoroughly up to date that I associated with me in the preparation of this edition, Dr. M. Howard Fussell, Professor of Applied Therapeutics in the University of Pennsylvania, whose broad training and large experience fit him eminently for this service.

As intimated in the title page the book has been rewritten to a considerable extent. This may be said particularly of the sections on Pellagra, Acute Anterior Poliomyelitis, and Caisson Disease. To other sections much has been added. This is true of Typhoid and Typhus Fevers, of Tuberculosis and Diseases of the Heart and Kidneys. Quite a number of new subjects have been introduced and while some are of minor importance, it is thought that all demand a place in a book which aims to cover more or less the whole field of internal medicine. Among these are Diseases of the Pituitary Gland, Trypanosomiasis, Rocky Mountain Spotted Fever, Diverticulitis, Bacteriuria, Melanuria, Oxaluria, Phosphaturia, Indicanuria, Cystinuria, Erythremia, Diseases of the Thymus Gland, Hypothyroidism and Hyperthyroidism, Hypertrophic Pulmonary Arthropathy, Osteitis Deformans, Leontiasis Ossea, Osteogenesis Imperfecta, Osteopsathyrosis, and Oxycephaly.

The section on Pellagra has been written by Dr. Edward Jenner Wood of of Wilmington, North Carolina, the accomplished author of the new Treatise on Pellagra, 1913, and that on the Phenosulphonephthalein Test by Dr. Alexander Randall, whose experience with it has been very large. Dr. Spiller has continued his supervision of the section on Nervous Diseases. For these services I am greatly indebted.

The paragraphs heretofore devoted to the history of the development of our knowledge as far as obtainable of the various diseases considered have been omitted, because more space had to be secured for new matter without increasing the size of the book. Readers will have to consult previous editions for these interesting histories. For the same reason the valuable chapter on Parasites by Dr. Allen J. Smith had to be curtailed.

JAMES TYSON.

1506 Spruce St., Philadelphia.

PREFACE TO THE FIRST EDITION.

I HAVE no apology to make for preparing this book. I have long contemplated it, and have finished it after several years' labor. It has taken some time, because it represents almost purely personal work, which has been frequently interrupted. It does not pretend to be based on my personal practice only. In these days of specialized work this would be impossible, though with most of even the rare forms of disease in every section I have had some experience. To fill in the gaps of my own knowledge, I have used that of others, but have always sought to make suitable acknowledgment to the proper source, and if this has not been done in any case, it has been a matter of oversight.

I had not, at the outset, expected to illustrate the work, but, as it progressed, a certain number of illustrations seemed necessary, not only to explain the text, but also, in a few instances, to render clearer the treatment described. Thus the number of charts and other drawings has grown to nearly a hundred, all of which, it is hoped, will be found useful. In expectation of the ultimate adoption of the metric system for the measuring of doses, these have been indicated throughout the book in the metric and English measures.

Acknowledgment is due to Dr. Joseph P. Walsh and Mr. M. A. Morin for suggestions after reading the text, to Dr. William Schleif for material assistance in Section XV, and to my son, Dr. T. Mellor Tyson, for assistance throughout the work and especially in preparing the index.

1506 SPRUCE ST., PHILADELPHIA.

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PRACTICE OF MEDICINE.

SECTION I.

INFECTIOUS DISEASES.

TYPHOID FEVER.

SYNONYMS.—*Typhus abdominalis; Enteric Fever; Pythogenic Fever; Gastro-enteric Fever; Nervous Fever.*

Definition.—Typhoid fever is an acute infectious disease due to the typhoid bacillus—the bacillus of Eberth. It is especially characterized pathologically by hyperplastic and ulcerative lesions of the lymph follicles of the intestine, of the mesenteric glands, and by enlargement of the spleen.

Etiology.—The *bacillus typhosus*, which is the cause of typhoid fever, was discovered by Eberth in 1880 in the intestine. The bacillus is found in the lymphatic system, including the mesenteric glands and spleen, in the liver and the kidneys, the blood and bone-marrow, and in bile and urine, as well as in the rose-colored spots.

The bacillus is actively motile, is short, rod-like, its length is one to three micromillimeters, breadth .5 to .8 of a micromillimeter.[1] Its size and shape vary somewhat with the culture-medium and the age of the bacillus. It is actively motile due to the posession of from two to twelve flagellæ attached to ends and sides. Its ends are rounded, and sometimes there can be seen toward them, glistening, clear spaces; it does not produce spores.[2] They closely resemble the bacilli coli from which they can be differentiated by special cultural methods, or agglutinating action of specific immune sera.

The bacillus stains readily in a saturated watery solution of methyl-blue, but not by Gram's method. Cultures may be made from the fecal discharges on the tenth day of the disease or later, but with difficulty, and are often negative. Cultures are now readily made from the blood.

The resisting powers of the typhoid bacillus are very great. It thrives at room-temperature. The thermal death-point is given by Sternberg at 156° F. (69° C.). According to Klemperer and Levy, the bacilli remain vital for three months in distilled water, though in ordinary water the commoner and more vigorous saprophytes consume them. When buried in the upper layers of the soil, they retain their vitality for nearly six months. Cold has no effect upon them, for repeated freezing and thawing fail to kill them. They have lived upon linen for from 60 to 72 days, and on buck-

[1] A micron or micromillimeter is 1/1000 of a millimeter = 1/2500 inch.
[2] Sternberg, "Jour. of Am. Med. Assoc.," August 22, 1891, p. 390.

skin from 80 to 85 days. One-tenth to 0.2 of one per cent. carbolic acid added to a culture-medium is without effect upon the growth of the bacillus; 0.5 of one per cent. strength of carbolic acid and 0.05 of one per cent. corrosive sublimate solutions are, however, fatal to it. Of all agents except high heat, sunlight seems to be among the most powerful to destroy it. The experiments of Billings and Peckham,[1] go to show that insolation for two hours destroys 98 per cent. of the germs, and in three to six hours kills all.

These bacilli yield an endotoxin, and not a soluble toxin as was at first supposed.

The bacillus itself most frequently enters the blood through the stomach in drinking-water or milk, in both of which it has been found during epidemics. There is reason to believe also that it may be inhaled. It has been found in water-filters by Harold C. Ernest and T. M. Prudden. It is quite well settled that the bacilli find their way into food and drink through the careless disposition of alvine discharges from typhoid fever patients, and that food may be contaminated by contagion conveyed from these discharges by the common house-fly. An oyster bed may be infected by sewage; green vegetables, by polluted water sprinkled upon them. Direct contagion from the patient to a second person may occur by means of fecal matter or urine soiling the hands of an attendant and being thus conveyed to the mouth and swallowed. The hands of a nurse may be contaminated by the water in which the patients are tubbed.

Whether the bacilli multiply outside the body in the water of wells or rivers to which they have obtained access is not well settled. A most noteworthy instance of an epidemic caused by contamination of drinking-water occurred in 1885 at Plymouth, Penna., U. S. A., where 1200 persons were attacked and 130 died, all the cases starting from a single subject, whose discharges contaminated the water-supply. The epidemic (1897) at Maidstone, England, furnishes another illustration of the effect of contaminated water-supply. Within two weeks after the outbreak, about the middle of September, 509 cases were reported; by October 27, 1748 cases; November 17, 1848 cases; in all, about 1900 in a population of 35,000. *The bacilli develop rapidly in milk and in the soil.* Many epidemics have been directly traced to contaminated milk supply, the milk having been infected either by the patient, a typhoid carrier, contaminated water or flies. Persons who have been ill with typhoid fever and who have entirely recovered have been found to transmit the disease long after apparent cure. The source of contagion in these "typhoid carriers" is the stool which is found to contain myriads of virulent typhoid bacilli. The relatively infrequent communication of typhoid fever to physicians, nurses, and others in close communication with the disease is explained by the fact that the contagion escapes from the patient in the stools and urine, and as these are commonly promptly disposed of, the chances for the dissemination of the poison from these sources are correspondingly few. Carelessness in the disposition of these discharges, as the result of which they are allowed to dry on linen, whence the bacilli pass into the air of the room, does some-

[1] "Influences of Certain Agents in Destroying the Vitality of the Typhoid and Colon Bacillus," "Sciene," February 15, 1895.

times occasion the infection of nurses and physicians and others attending on typhoid cases. Bacilli are said to have been found in sputum.[1]

Predisposing Causes.—Experience fails to establish definite predisposing causes of typhoid fever, but new-comers to an infected area are more likely to be attacked than old residents, as early shown by the French physicians in Paris. It certainly often attacks the strong and healthy as fiercely as the feeble and delicate, while allowance must be made for the more frequent exposure of the healthy. Typhoid fever is unlimited in its distribution by climate or civilization, but it may be complicated by disease peculiar to certain localities, pre-eminently malaria.

Typhoid fever is a disease of adolescents and adults under 30, although it may occur at any age. Less common in children, the disease has been found in a child five days old, while not a few cases have been reported in sucklings. Infection *in utero* is claimed as possible because of successful cultures of bacilli from the fetus. In the young the duration of the disease is short and the prognosis singularly favorable. It has occurred at the age of 75, 86, and even 90. More men than women have typhoid fever (71 per cent. of 444 cases collected by Reginald H. Fitz), probably because of their more frequent exposure. The assertion that the pregnant state seems to protect against typhoid fever is not substantiated by experience in Philadelphia, in evidence of which may be stated that within two months there were received in the wards at the Hospital of the University of Pennsylvania three pregnant women with typhoid fever.

Typhoid fever is more common in the late summer and autumn months than at any other time of the year, whence one of the names, "autumnal fever." It has been observed that hot and dry summers are followed by more cases than hot and moist summers. Liebermeister explains the relation of typhoid to the hot and dry season by the fact that at this season the quantity of solid matter in springs is relatively larger; that the poison, in other words, is more concentrated, and therefore more virulent. Special epidemics may occur at any season. Thus the epidemic of typhoid fever at Plymouth, Penna., alluded to, began April 10, and raged with greatest fury during May and June. Other epidemics illustrate the same truth.

Morbid Anatomy.—The characteristic lesions of typhoid fever include the changes in the lymphoid structures of the intestine, the solitary glands and Peyer's patches, though properly speaking the disease affects the body as a whole. Histologically according to Adami there is proliferation of the lymphoid elements and perifollicular infiltration of leukocytes and intense congestion of all the vessels. The Peyer's patches are prominent, plaque-like, and intensely red, while the solitary glands are red, enlarged and sometimes polypoid. In the second week the central portions of the intestinal glands undergo coagulation necrosis, slough away and leave a ragged ulcer. In a few days well-defined ulcers are caused by more sloughing. The ulcers have a smooth base and indurated edges. This, when it represents a single follicle, is small and circular, not more than from three to six millimeters (1/8 to 1/4 inch) in diameter; large and elliptical when an entire *Peyer's patch* is involved. Such a patch is usually opposite the mesenteric attach-

[1] "Jehle, Wien. klin. Wochenschrift," 1902. "Glaser, Deutsch. med. Wochenschr.," 1902, No. 43, pages 772 and 793.

ment, having its longest diameter parallel with the length of the bowel and its shorter transverse, thus reversing the relations of the tubercular ulcer. Much larger ulcers are sometimes formed by the union of others, especially toward the lower end of the bowel. The floor of the ulcer is usually the submucosa, or the muscular coat of the bowel, but it may be the peritoneum, and even this is sometimes sphacelated, appearing as an opaque white membrane that sooner or later breaks and the bowel is perforated. More commonly, the ulcer heals, and the patient recovers, but the normal glandular structure of the gut at the seat of the ulcer is not restored. Necropsy frequently discovers ulcers in different stages of healing. The large intestine is also invaded in probably one-third of the cases, and the process may terminate here also in perforation. Ulceration may extend to the appendix, where, too, perforation sometimes takes place.

Similar infiltration of the lymph nodules and lymph cords of the *mesenteric glands* and of the *spleen* may occur, contributing to the enlargement of these organs. In the spleen it is associated with an active hyperemia that contributes to further enlargement, generally recognizable during life. The organ may reach twice or three times its normal size—*i. e.*, 435 to 650 gm. (14 to 20 ounces). There has even been rupture of this organ; see a case recently reported by Conner of N. Y. Hemorrhagic infarcts have been found in the spleen in from four to seven per cent. of cases coming to autopsy. Abscess of the spleen has been found.

Perforation has been noted at necropsy in 5.7 per cent. of cases—that is, 114 out of 2000 autopsies in Munich; by Osler, in 2.48 per cent. of 685 cases; and J. Alison Scott, in 3.6 per cent. of 9713 cases. It occurred in only one of 105 soldiers treated at the University Hospital in the fall of 1898.

As to the location of perforation, Hawkins found it in 61 of 72 cases in the ileum, three in the cecum, three in the appendix, and five in the colon, most of the latter being in the sigmoid flexure. In 167 cases collected by Fitz the ileum was perforated in 136, the large intestine in 20, the appendix in five, Meckel's diverticulum in four, the jejunum in two. The number of perforations may vary from one to several. The accident is most frequent in the third week, or close to the third week. It is more frequent in men.

The *liver*, among organs more rarely affected, shows cloudy swelling, granular and fatty degeneration of its cells, lymphatic nodular areas, and even liver abscess with pylephlebitis, and acute yellow atrophy. Abscess of the liver was found 12 times in the Munich necropsies, and acute yellow atrophy three times. Pylephlebitis has followed abscess of the mesentery and perforation of the appendix. Typhoid bacilli are often found in the gall-bladder in fatal cases; in Chiari's reports[1] 19 out of 22; in Simon Flexner's, seven out of 14. Perforation of the gall-bladder is sometimes met, and Keen has collected 30 cases in his book on the "Surgical Complications and Sequels of Typhoid Fever," 1898.

In the *kidneys* there may be cloudy swelling and granular degeneration of renal cells, more rarely acute nephritis, which may even be hemorrhagic; also miliary abscesses in which typhoid bacilli have been found. Diphther-

[1] "Prager medicinische Wochenschrift," 1893, No. 22.

itic and catarrhal inflammation of the pelvis of the kidney and catarrhal inflammation of the bladder are occasionally present.

Changes in the *respiratory organs* are often found. Among the rarer of these are edema of the glottis, ulceration of the larynx, and even necrosis of the laryngeal cartilages. This occurs frequently. Keen has collected 221 cases, many of which gave rise to serious symptoms. Hypostatic congestion of the lungs is quite common; pneumonia is more infrequent. The pneumonia may be of pneumococcic origin or it may be the result of implantation of the typhoid bacillus in the lung, a true typhoid pneumonia. Even gangrene of the lungs was found in 40 of the Munich cases; abscess, in 14; and hemorrhagic infarction, in 129. Pleurisy and empyema are rare events.

In the *circulatory system* there may be thrombosis of veins, especially of the femoral, causing the not very rare symptom of milk-leg; more rarely there is thrombosis of the femoral artery, which may be preceded by embolism. Thrombosis of the cerebral veins may give rise to hemiplegia. Endocarditis, pericarditis and myocarditis may be present. The latter condition is attested by a yellow, soft, and flabby muscle seen after death.

As to the *nervous system*, notwithstanding the intensity of the nervous symptoms at times, meningitis is a rare event, though both serous and purulent forms have been met, typhoid bacilli being found *in loco* as the apparent cause; also thrombosis of cortical veins and parenchymatous changes in nerve-trunks, even when there have been no symptoms of neuritis. Abscess of the brain has also been found with the bacillus typhosus *in loco*.

In the *muscular system* granular and hyaline transformation of voluntary muscle may occur, as in other fever processes.

Osseous System.—Periostitis or ostitis in various bones of the body is not rare. Warfield T. Longcope[1] has studied the *marrow of bone in typhoid fever*. He found congestion, edema, focal necrosis and many large phagocytic cells which last he regards as typical. A striking feature was a mild general hyperplasia of all the blood-forming cells, with also some and often a very decided increase of the non-granular cells and swelling of the lymphoid follicles. There was a marked scarcity of eosinophiles. Polymorphonuclear leukocytes were numerous in the uncomplicated cases; in cases complicated by acute infections they were less numerous, practically absent. The lesions were more marked the more prolonged the disease before death.

Abscesses in the parotid gland are a familiar lesion; more rarely, abscesses in the intermuscular tissue.

General invasion of all organs and of the blood (bacillæmia) by the typhoid bacillus are now not infrequently found.

Typhoid fever without enteric lesions has been reported by Sidney Philips, J. W. Moore, Simon Flexner, and others, without these lesions. In doubtful cases the Widal reaction and the presence of bacilli as determined by cultures must be appealed to.

Symptoms and Course.—A certain *period of incubation* is necessary after

[1] Longcope, "Bulletin of the Ayer Clinical Laboratory of the Pennsylvania Hospital," No. 2, January, 1905.

the successful implantation of the bacillus before typhoid fever arises. This varies from a week to two weeks and even longer. The period of incubation is usually without symptoms, but there may be a sense of weariness and indisposition to exertion, the latter often overcome by force of will; also a want of appetite and a slight coating of the tongue. These symptoms, more strictly speaking, belong to the prodrome, and are in turn not sharply separated from those of the disease itself, which usually sets in very gradually and is often quite advanced before suspected, indeed, sometimes well advanced, constituting the "walking" or "ambulatory" typhoid. In *children the onset is less gradual, frequently abrupt.* There may be headache, anorexia, a furred tongue, nausea, chilliness, but only rarely a decided rigor. The disease may be ushered in by muscular *pain* in the back or legs. *Nosebleed* has always been considered characteristic, and yet it is less frequent than might be expected from the text-book statements. More common is *looseness* of the *bowels*. Or if not looseness an aperient acts more severely than it does in a healthy person. All this time there is slight fever, and the patient feels wretched, the fever and the discomfort increase, and finally he goes to bed. The tendency to looseness of the bowels and epistaxis, more than any other symptom of this group, justify strong suspicion of the existence of typhoid fever. Yet one or both are quite often absent. Certain epidemics are more apt to be attended by diarrhea than others. The *abdomen* soon becomes slightly distended and tympanitic, and pressure in the right iliac fossa will usually elicit *tenderness* with *gurgling*. At times there is *colicky pain* of varying severity independent of pressure; at others the *gastric* symptoms are marked and *nausea* and *vomiting* are present.

Usually about the eighth day, rarely later and sometimes a little earlier, *rose-colored spots* make their appearance on the skin of the abdomen and chest, more rarely elsewhere on the body. These call for further description. They are usually bright red in color, and are well compared to a fleabite. They are very slightly raised above the surface and disappear on pressure, to return instantly after its removal. Their number varies greatly. Sometimes they are very numerous, covering the entire trunk; oftener there are four or five to ten, again one or two, most rarely none. When numerous, they occur in successive crops, each crop lasting from two to four days. Histologically, they are circumscribed, actively hyperemic areas, the hyperemia being excited by some irritant, which may be the typhoid bacillus itself, since it has been found in the spots. Only in the most malignant cases is there any blood found outside of the vessels, and when this occurs, the spots can be made to disappear but partially on pressure. The association of roseolar spots is so intimate with the disease that they have been regarded as pathognomonic. Rose-colored spots are much more uncommon in children.

In addition to rose-colored spots, *sudamina* are often present in large numbers on the skin, especially when the disease is associated with much sweating, but their occurrence is by no means constant, and their association with other diseases in which there is perspiration is well known. More rarely, *petechiæ* and *vibices* are noted in adynamic forms of the disease. An *erythema* is quite often found on the skin of the chest and abdomen. Peli-

omatous patches—the *tâche bleuâtre*—sometimes are found on the skin of the thorax, abdomen, and thighs; also the *tâche cérébrale*—a red line, produced on drawing the finger-nail over the skin—but neither have any symptomatic significance. *Herpes* is rare but occurs according to McCrae and to Phillips in about one per cent. of the cases. *Jaundice* is occasionally seen, and may be the result of an obstructive cholangitis excited by the bacillus.

Enlargement of the spleen is an almost constant clinical feature of typhoid fever. If the vertical dulness exceeds the depth of two ribs and an interspace, enlargement is present. Not only may this be recognized by percussion, but by palpation as well. Clinicians generally lay great stress on palpation, and enlargement may sometimes be detected by it when the organ eludes percussion by reason of tympany. At times the outline is indistinct, at others both the tip and anterior edge of the organ can be distinctly located. Lay the patient on his back. Palpate the abdomen below the margin of the ribs on the left side with the left hand, forcing the tips of the fingers very gently under the ribs. Have the patient take a deep inspiration, at the same time pushing the spleen forward with the right hand placed posteriorly over the eleventh and twelfth ribs. Enlargement can generally be detected at the end of the first week or in the first half of the second week, when the organ may reach twice or three times its normal size. *By the end of the third week in uncomplicated cases it begins to diminish in size.* The enlarged spleen may also be tender. Enlargement is less frequent in cases occurring late in life. Recently, desiring to know in what proportion of cases of typhoid fever the spleen is palpable, the record of 1000 cases at the Pennsylvania Hospital was examined and it was found that in 32.8 per cent. the organ was palpable on admission. This can only be approximate, as considerable variation was found in the observations by different resident physicians.

Early, too, in the disease the patient may have a slight *cough*, unassociated with physical signs, or at most those of a mild bronchial catarrh.

The *fever* is at once the most important and characteristic symptom, and from the temperature alone a diagnosis can be suspected. During the *increment* of the disease it exhibits a peculiar, tide-like evening rise and morning fall, while the temperature of each morning and evening is from one and a half to three degrees higher than that of the previous morning and evening. The patient is rarely seen at the very beginning of this first stage; it lasts commonly a week. Frequently it is succeeded at the end of four or five days by the acme or *fastigium*, in which are continued the evening rise and the morning fall, but the evening and morning difference is less marked, the tidal character is no longer present, and the temperature is high throughout. The average duration of the fastigium is five to eight and ten days, being longer in severe cases and shorter in milder ones. In protracted cases the period of febrile elevation may be still longer. It is during it that we meet the maximum temperature, quite often 105° F. (40.5° C.) or a little above, more rarely 106° F. (41.1° C.). A temperature of 106° F. is not infrequently followed by recovery, but while 107° F. (41.6° C.) and 108° F. (42.2° C.) and even 109° F. (42.7° C.) are met, such cases have invariably, in our experience, terminated fatally.

The fastigium is succeeded by the third stage, or *period of decrement* or decline, in which the reverse of the initial stage is shown by an evening temperature lower than that of the previous evening, and the morning temperature lower than that of the previous morning, but with the evening temperature still higher than that of the morning of the same day. This decline continues until the normal is reached, and from one to two weeks are consumed before that is attained. The whole is much better shown and more easily understood from a chart than from a description in words. Such a chart of the temperature uninfluenced by treatment is seen in figure 1, although the rise and fall are not always as regular as indicated. In a

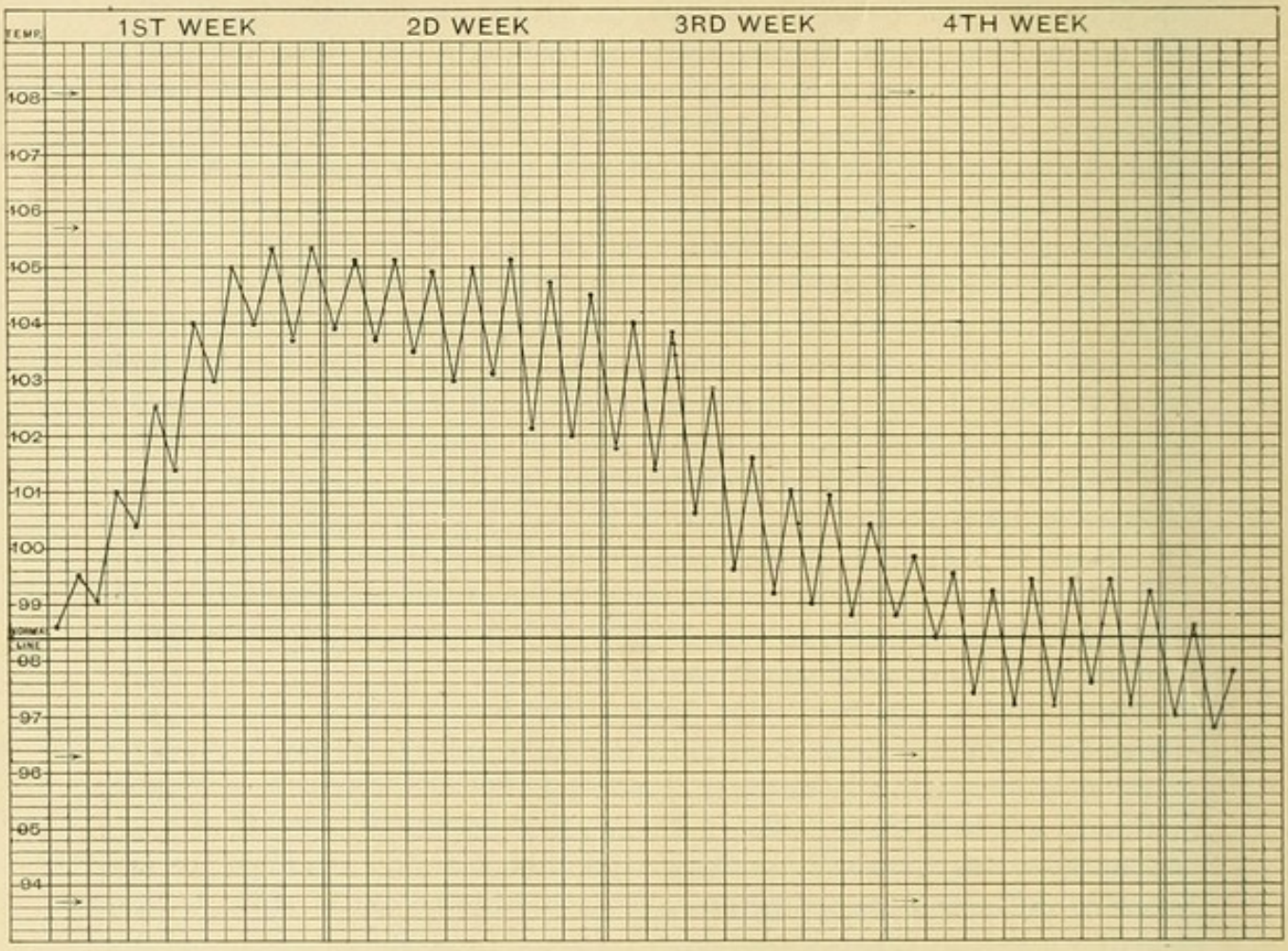


FIG. 1.—Temperature Chart of a Typical Case of Typhoid Fever Uninfluenced by Treatment.

typical case one might safely place the first stage at four days to a week; the second, or fastigium, as seven to ten; and the third, about as long as the second, the shorter period corresponding to a mild case and the longer to a severe one. The fever does not always reach the higher temperature shown in the chart, and sometimes the maximum never reaches 102° F. (38.9° C.). On the other hand, there is sometimes a difference of three or four degrees in the morning and evening temperature, and the latter may drop to normal. In ordinary cases the evening temperature falls to the normal in the course of the fourth week, but in severe cases the temperature keeps up during the fifth and even sixth week, these cases having almost invariably extensive ulceration with great tenderness of the abdomen and meteorism.

Many of them terminate unfavorably by hemorrhage or perforation. In some cases the temperature is quite irregular throughout the entire course of the disease. Frequently after the morning temperature has reached normal, the fever rises abruptly in the evening, to fall again in the morning, a true remittent or intermittent type of fever, lasting several days. One case on record at the Episcopal Hospital had this type for fifteen days with nothing discoverable to account for it.

When the disease begins with a *chill—a rare event*—the temperature rises more rapidly in the beginning. Sudden falls of a decided character may occur in consequence of hemorrhage from the bowels, or the nose or from collapse after perforation of the bowels. Sudden rises are produced by indiscretion in diet and overexertion or the supervention of some acute inflammatory affection, as pneumonia, or phlebitis. In a few cases the temperature is not at all characteristic. Rarely there is a reversal of temperature the higher being found in the morning.

Copious *sweating* characterizes some cases of typhoid fever, though the skin is more commonly dry. Sometimes, during the reaction after a cold bath, there is perspiration. The profuse sweats first alluded to are not attended by a reduction of temperature, being sometimes present when the temperature is highest. Cases of recurring paroxysms of chill, fever, and sweat are reported, which simulate intermittent fever, and may reasonably be mistaken for it.

The *pulse* is only moderately frequent, 90 to 120 being the usual range, while a proximity to 100 is quite frequently maintained. In grave cases it becomes more frequent, 140 or more; when, if maintained, it is a rather unfavorable symptom, due to high temperature or complications. Temperature and pulse do not always increase *pari passu*. *Dicrotism* may occur with frequent pulse, but dicrotism also occurs in the early stage, when it is regarded by some as diagnostic. According to Curschmann, dicrotism is more common in typhoid fever than all the other infectious diseases taken together. During convalescence the pulse gradually resumes its normal character, and sometimes becomes abnormally slow, falling to 30 or less. we have had a case in which the pulse fell as low as 18, and continued for one day between 20 and 36. Typhoid fever is characterized by low blood pressure the fall beginning toward the end of the first week. The pressure remains low until convalescence is established.

The *breathing rate* commonly advances with the rate of the pulse, but is sometimes increased in frequency by temporary causes and rarely is disproportionately slow. In a very striking case at the University Hospital the rate fell to twelve in a minute, and continued thus for an hour.

The *heart-sounds*, at first natural, grow less loud as adynamia progresses, and the first sound may even disappear in grave cases. Sometimes a soft systolic murmur develops at the apex, usually at the end of the second week. Sometimes it acquires greater intensity. It has been especially studied by M. G. Hayem,[1] who ascribes it not to an endocarditis, but to a relaxation of the muscle which results in imperfect apposition of the valves

[1] M. G. Hayem, "Des manifestations cardiaques de la fièvre typhoid," "Le Progrès Médical," 17 Juillet ,1875. p. 401 *et seq.*

and a consequent regurgitation. This murmur disappears as recovery takes place, and the heart-muscle grows strong.

In the beginning the typical *tongue* is covered with white fur in the back and center, is rather dry, is somewhat pointed, and the tip and edges are bright red. As the disease advances, the tongue, previously furred, tends to become dry and brown, clearing, however, at the edges and tip as the case improves. In severe cases, especially if the mouth is not kept clean, *stomatitis* with fissures and bleeding may occur, and sordes may collect on the teeth, while the lips become covered with black crusts, constituting the "fuliginous coating." These phenomena are almost unknown with the bath treatment. Mild grades of *pharyngitis* producing painful swallowing, sometimes usher in the attack, more particularly in certain epidemics.

The *diarrhea* of typhoid fever has been alluded to. It is said to be present in 20 to 30 per cent. of cases. Usually corresponding in severity with the extent of the local lesion, it is seldom troublesome or difficult to control, and is sometimes absent throughout. The stools have no characteristic qualities. They may be grayish-yellow and are usually fetid. Persistent severe diarrhea points to extensive ulceration.

Meteorism in moderate degree is an almost constant symptom. The distention by gas is commonly ascribed to atony of the bowels. Its presence in high degrees adds to the seriousness of the case, since it corresponds usually with the extent of bowel lesion, and soon succeeds perforation.

Hemorrhage from the bowels, also a consequence of intestinal ulceration and the separation of sloughs, is a serious symptom, but by no means always fatal, though large quantities of blood are sometimes discharged *per anum*. The occurrence of such hemorrhage is followed by a rapid reduction of the temperature, as shown in chart Fig. 2, and a pallor and faintness such as are common to large hemorrhages elsewhere. As stated, very profuse hemorrhages may be followed by recovery, and it is barely possible that a favorable influence may sometimes be exerted by them. Very rarely a patient will bleed to death. Hemorrhage was a cause in 11 out of 56 deaths in Osler's 685 cases. It occurred 99 times in 2000 cases in Munich, and eight times in 105 soldiers under our care after the Spanish-American war. Severe and occasionally fatal hemorrhage may occur from the nose.

Delirium is less constantly present in typhoid fever than in typhus, and may be absent throughout. It may, however, be very active, requiring the patient to be carefully watched to prevent him from leaving his bed and seriously endangering his life. More than one victim has leaped from a window with fatal results under such circumstances. In certain cases, especially when the initial headache is very intense, this symptom continues and to it are added fever and delirium so extreme that meningitis is simulated, though the true form of this disease rarely occurs. Such cases illustrate the nervous "form" of the disease. A tendency to drowsiness, and even to stupor, suggested the common name "typhoid," but it is less characteristic than in typhus. Rarely *convulsion* occurs in the course of the disease, in Murchison's experience in six out of 2690 cases.[1]

Muscular tremor is a symptom in severe cases, when it would seem to

[1] See a paper by Thomas Claytor in "Philadelphia Medical Journal," March 3, 1900.

indicate a muscular weakness or exhaustion, which may be an effect of high temperature or of the specific poison of the disease. Carphologia, or "picking at the bedclothes," is a symptom of which the unfavorable import has been somewhat exaggerated, probably because of the popular fa-

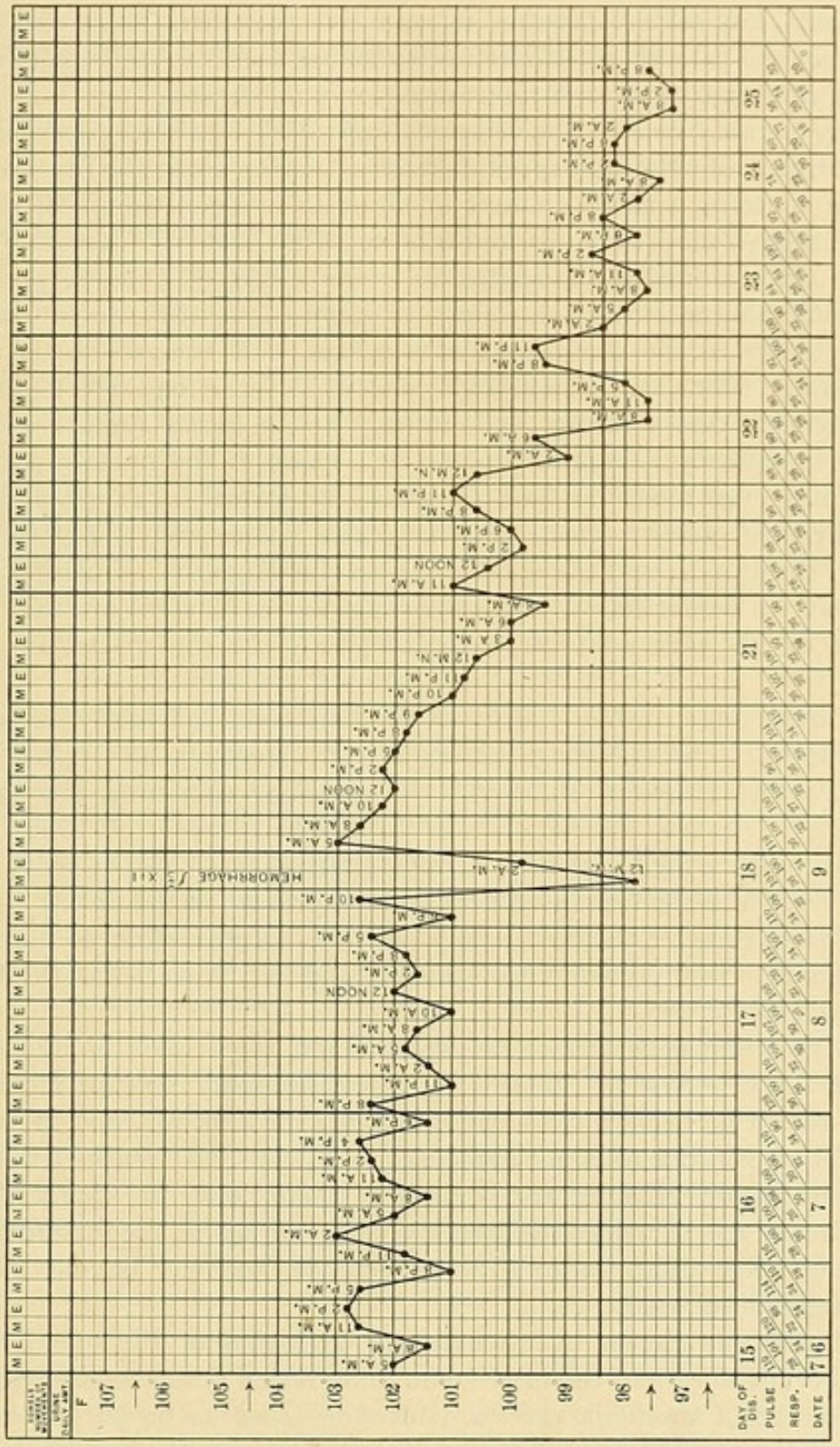


FIG. 2.—Showing Drop in Temperature Incident to Intestinal Hemorrhage.

miliarity with Dame Quickly's interpretation in Falstaff's illness. Concurrently with these "typhoid" symptoms, the tongue reaches its maximum dryness, and may be dark and leathery in appearance, while sordes may collect on the teeth.

Hiccough is an infrequent, but sometimes obstinate symptom.

Apart from an initial *bronchial catarrh*, which sometimes ushers in the disease, the typhoid patient sooner or later acquires a *slight cough*, due to hypostatic congestion of the lungs, but it is easily kept within bounds by frequent changes in the position of the patient. Occasionally, the cough is quite severe, but seldom requires more active treatment than this. The initial bronchial catarrh, too, sometimes assumes severity, while more rarely the symptoms and signs of *pneumonia usher in the disease.*

Changes in the Urine.—The urine is always dark-hued and concentrated, with a correspondingly high specific gravity. Often when the fever is high the urine contains a small amount of albumin. When complicated with *nephritis*, there is more albumin, and tube-casts are present. Recent French statistics place albuminuria, regardless of its cause, at over 20 per cent. While such albuminurias are found in grave cases, they do not appear to add greatly to the seriousness of the case, and recovery is the usual termination. More rarely, nephritis in a mild form may develop during convalescence. Most rarely, still, it may be an initial symptom of the disease, constituting a nephro-typhoid analogous to the pneumo-typhoid, when it may even mask the true disease by its severity. It is well named by the French—*fièvre typhoide â forme renale.* Only the Widal test, the intestinal symptoms, and the spots clear up the diagnosis. Such nephritis may rarely be hemorrhagic. The toxic properties of urine are said to be increased during typhoid fever, especially while the cold baths are being used.

The urine may contain bacilli of typhoid fever, generally associated with albumin. The following summary from Norman B. Gwyn's paper in the "Johns Hopkins Bulletin," June, 1899, condenses our present knowledge:

"1. In quite a high percentage, perhaps from 20 to 30 per cent., of all cases of typhoid fever typhoid bacilli may be present in the urine.

"2. When present, they are usually in pure culture, often so numerous as to make the freshly voided urine turbid, and may then be detected by a cover-slip examination.

"3. Appearing generally in the second and third week of illness, the organisms may persist for months or years, probably multiplying in the bladder, the urine being apparently a suitable medium for their growth.

"4. Though often showing evidences of cystitis and marked renal involvement, the urine containing bacilli has usually only the characteristics of an ordinary febrile urine; the presence of bacilli has no prognostic importance, and their disappearance or persistence, without having induced local change, is the rule.

"5. Lastly, as shown by Richardson, irrigation of the bladder with bichlorid of mercury and the internal administration of hexamethylenamine —a compound of ammonia and formaldehyde—seem to be safe methods of removing the bacilli; 30 to 60 grains of the latter quickly removing all bacilli in six cases."

More recent studies, especially by Hiss (*loc. citate*), go to show that the urine, in consequence of the more prolonged presence of the bacilli, may be a more frequent source of infection to the community at large than the feces.

The so-called *diazo reaction* of urine, to which attention was first called by Ehrlich in 1882, is so constant in this disease as to be deservedly regarded as a symptom. It was found by John Hewetson in 136 out of 196 cases, and by Arthur R. Edwards in 128 out of 130 cases, and by Simon in 22 out of 26 cases. I have never found it absent when the test was made sufficiently early.

For making the test three solutions are necessary:

1. A five per cent. solution of hydrochloric acid saturated with sulphanilic acid. This solution should be fresh.

2. A half of one per cent. solution of sodium nitrite.

3. Ammonium hydrate.

When it is desired to make the test, 40 c.c. of (1) and 1 c.c. of (2) are mixed. The hydrochloric acid, acting on the sodium nitrite, liberates nitrous acid, which in its nascent state combines with the sulphanilic acid, producing diazo-benzine-sulphonic acid. Equal parts of this mixed solution and urine are thoroughly shaken; enough of the ammonia is then allowed to flow carefully down the side of the tube to form a colorless zone above the urine mixture. At the junction of the two fluids a dark-garnet or cherry-red ring will form if the reaction takes place, and if the tube is well shaken, a uniform red color is imparted to the entire fluid, which, when allowed to stand for some hours, shows a characteristic olive-green precipitate, the upper layer of which, as a rule, has a still darker green color. The *reaction occurs about the time of the appearance of the rash* and usually *continues until the 22nd day*, but it may disappear before the end of the second week. It is, as stated, symptomatic and not pathognomonic, as it occurs in many diseases with high fever, among which measles and miliary tuberculosis are conspicuous. It may, however, be regarded as negatively pathognomonic —that is, its absence is strongly presumptive against the presence of typhoid fever.

Polyuria is a rare symptom. A remarkable case was reported by one of us.[1] In this case as much as 6750 c.c. of urine were passed in 24 hours during high fever. The cause of such excessive polyuria to problematical.

Indicanuria is claimed by Judson Daland[2] to be quite frequent in typhoid fever and is said to especially demand thorough cleansing of the oral and nasal cavities whence putrefactive substances may be carried to the stomach, as well as absorbed *ex loco*.

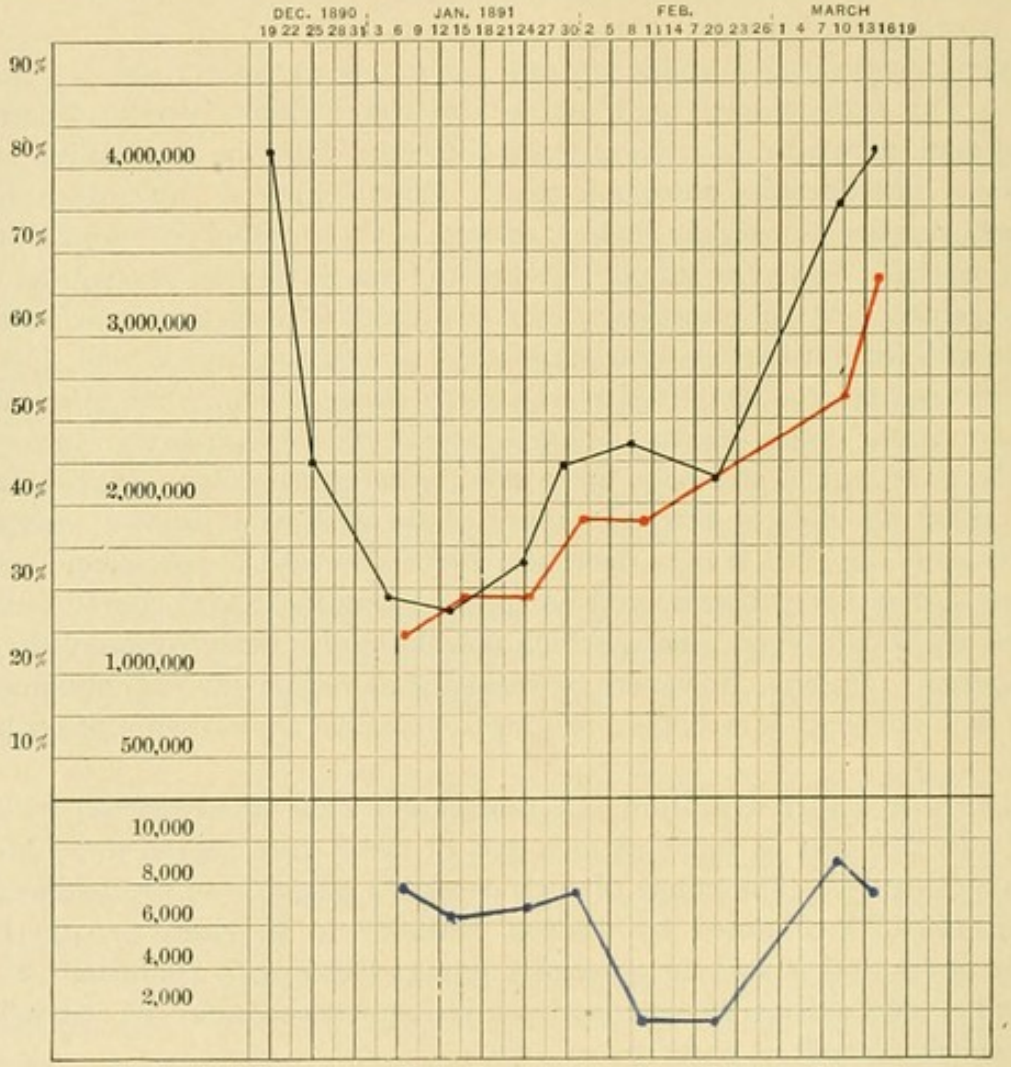
Changes in the Blood.—The state of the blood in typhoid fever early claimed attention and even the earliest observers, begininng with Le Canu in 1837, noted a diminution of *red blood-corpuscles*. This observation has been essentially confirmed by the most recent studies with modern accurate methods, among which those by Ouskow,[3] by Khetagurow,[4] and by W. S. Thayer[5] are conspicuous.

At the beginning of the fever the number of red blood-corpuscles is normal and even at the upper limit of normal, because the patients are apt to be young and strong, while in some instances the initial diarrhea or pro-

[1] Fussell, Carmany and Hudson, "Transactions Association of Amer. Physicians," 1904.
[2] Daland, "American Medicine," vol. viii., 1904, p. 764.
[3] "The Blood as a Tissue," St. Petersburg, 1890.
[4] "Pathological Changes in the Blood in Typhoid Fever," Inaug. diss., St. Petersburg, 1891.
[5] "Two Cases of Post-typhoid Anemia, with Remarks on the Value of Examination of the Blood," vol. v., "Johns Hopkins Hospital Reports," 1895.

nounced sweating may cause slight concentration of the blood. During the first two weeks the number of red corpuscles gradually falls, though but slightly. With defervescence they fall off more rapidly, reaching a minimum usually about the first week of convalescence, after which there is a gradual rise to the normal, followed again by a possible slight fall when the patient gets up. The fall in the number of red corpuscles, while relatively slight, usually bears a direct relation to the severity of the case.

The *hemoglobin* is always reduced and the reduction is relatively greater than the corpuscular loss, with an even slower return to the normal. Ex-



Black—Red Corpuscles. Red—Hemoglobin. Blue—Colorless Corpuscles.

FIG. 3.—Chart showing Anemia of Typhoid Fever.—(*From Thayer's "Monograph."*)

treme anemia, with a blood count as low as 1,300,000 corpuscles in a cubic millimeter and hemoglobin as low as 20 per cent., has been met.

The number of *leukocytes* in a cubic millimeter, normal at the beginning, tends to *diminish* slightly throughout the disease, reaching a minimum toward the end of defervescence, increasing again with the beginning of convalescence, and reaching the normal after several weeks. More definitely the change consists in a diminution in the percentage of multinuclear or overripe elements, with a relative increase in the large mononuclear or ripe elements. The *absence of leukocytosis is regarded of real diagnostic value*, being in marked contrast with the distinct increase in the number of colorless corpuscles characteristic of most other infectious processes.

Very rarely the leukocytes may be increased with no signs except those of ordinary typhoid. In certain complications such as otitis, phlebitis, pneumonia, and perforation, there is a leukocytosis, one case at the Episcopal Hospital having 21,000 leukocytes within five hours of a local peritonitis. Typhus fever is unattended by blood changes. Pneumonia is usually accompanied by leukocytosis, while in a few cases of malignant pneumococcus infection there may be no leukocytosis. The leukocyte count in malarial fever is practically the same as in typhoid fever, but the presence of the malarial parasite in the former is distinctive. In pure miliary tuberculosis unassociated with local inflammatory processes there is also an absence of leukocytosis. It is important to remember that cold baths have the effect of producing a decided temporary increase in the proportion of leukocytes, probably rather in consequence of an accumulation of white cells in the vessels of the surface than as the result of a true leukocytosis.

Unusual Modes of Onset. Atypical Forms.—It has been mentioned that while slight chilliness is often an initial symptom, *severe rigor* at the same stage rarely occurs. It does, however, happen, as in 13 out of 79 of Osler's cases. More frequently, chills have been observed in the course of the disease from some one of the following causes:

1. At the onset of a relapse, or even during convalescence without apparent cause.

2. As a result of treatment, especially by antipyretics internally, guaiacol externally, or of a hypodermic injection of a sterilized culture of typhoid bacilli.

3. At the onset of complications, such as pneumonia, pleurisy or thrombosis.

4. From sepsis during convalescence in severe and protracted cases. Under these circumstances chills may be frequent, severe, and of grave import.

5. From concurrent malaria.

6. From constipation, according to Herringham.

In epileptics who acquire typhoid fever the latter disease is very apt to be ushered in by an unusual number of epileptic *convulsions*, which continue frequent until the fever becomes established, then diminish, and finally cease, often not recurring until some time after recovery, causing the victim and his friends to believe that the chronic malady has disappeared. It returns, however, sooner or later. The same is true of choreic attacks. Rarely, the disease is ushered in with convulsions in children. *Convulsions* are rare but acknowledged symptoms in the course of the disease as well as at the onset. Murchison recorded 6 cases in 2960 of typhoid, of which two died. In one diseased kidneys were discovered. Osler[1] reported eight cases of convulsions in from 1500 to 1600 of the disease. In two at the onset; in three during the course of the disease, supposed manifestations of toxemia, of which one died of perforation; two cases occured in severe cerebral complications as thrombosis, meningitis and encephalitis both fatal. One occurred of unknown cause during convalescence. The prognosis is not considered grave. *Aphasia* was noted by S. F. Blakely, of Ora, S. C., in the case of a girl of 15. The condition continued for four weeks.

[1] Osler, Wm., "Practitioner" (London), 1906, lxvxxi., p. 1.

In diabetes the sugar may disappear during the fever.

Among the more unusual modes of onset should be mentioned cases beginning with severe bronchitis; those with the initial symptoms of pneumonia, including chill; those with initial symptoms of nephritis or with intense nervous symptoms, suggesting cerebrospinal meningitis. Among the latter are intense headache and photophobia, combinations rapidly passing over into active delirium, with muscular twitching and retraction of the head, constituting the *nervous* or *meningeal* form. In accordance with recent views these varieties may be considered as representing forms in which the organs especially involved are the primary and chief seats of attack by the bacillus as contrasted with the more usual intestinal form. In certain long and severe cases septic infection occurs, manifested by fever, sweats, and local abscesses in various parts of the body, including the perirectal and perinephric regions.

Among *irregular* forms is the so-called *abortive* form. This doubtful form is said to be more sudden in its onset, beginning with shivering and fever of 103° F. (39.4° C.) or higher. The rose-colored spots appear at from the second to the fifth day. The fever falls at the end of the first week or beginning of the second, commonly by crisis with a sweat, after which follows convalescence. The *hemorrhagic* is a grave variety characterized especially by cutaneous and mucous hemorrhages, and is fortunately rare. Five cases of this variety have been reported by Samohrd[1] and one by T. H. Evans[2] of Philadelphia. One of Samohrd's cases was fatal. Possibly the hemorrhagic sites are foci of invasion by bacilli, which weaken the integrity of the vessel walls.

The *mild form* is sometimes so mild as scarcely to be recognized as typhoid fever and is often called gastric fever or simple febricula. There is, however, no more important lesson for the inexperienced practitioner to learn than that some cases beginning as mere febricula may pass over into forms of great severity, and may even terminate fatally. A vary rare form is the *tonsillar* typhoid, in which whitish elevations appear on the tonsils, subsequently becoming ulcers.

Complications and Sequelæ.—The Spanish-American war confirmed the possibility of the coexistence of typhoid fever and *malarial* fever, since a number of cases from among the soldiers have been reported in which not only all the necessary clinical features of typhoid fever were present, but also the Widal reaction, in which, too, the malarial organism was found in the blood. Such coexistence occurred in cases in the Hospital of the University of Pennsylvania and Saint Mary's Hospital, Philadelphia. It is, however, an infrequent event. On the other hand, a mongrel disease that is the product of the two causes, as was once supposed to be the case, and known as *typhomalarial fever*, does not exist. The term should be dropped, as it is confusing and gives rise to erroneous impressions.

Persons with tuberculosis, heart disease, diabetes, epilepsy and other forms of chronic nervous disease are as liable to typhoid fever as others, while scarlet fever, diphtheria, measles, chicken-pox, rheumatism, and especially erysipelas, may befall a typhoid case. Typhoid fever in diabetic cases is

[1] Samohrd, "Sbornik Klinicky," Tomo V, fasc. i, 1903, and abstracted in "Il Policlinico," Rome, No. 21, 1904.

[2] "Medical News," Sept. 3, 1904.

especially apt to be attended with low temperature. Typhoid fever itself may be followed by tuberculosis but in most instances the condition was tuberculosis from the beginning, such cases are in our records.

Perforation occurs most commonly in the third or fourth week of the disease. Though it may occur as early as the eighth day, and as late as the sixth week. The premonitory symptoms are sudden, severe, localized pain with local tenderness and muscular resistance, most commonly in the lower right quadrant of the abdomen, with rapidly rising leukocytosis. When the perforation is complete, sudden or large, there is generalized abdominal pain, loss of peristalsis, distention of the abdomen, disappearance of liver dulness and collapse. Occasionally the pain is so high up that the condition may be mistaken for pleurisy, or on the other hand a pleurisy may be mistaken for perforation.

Thrombosis of the femoral vein, more frequently the left, resulting in *phlegmasia alba dolens*, or milk-leg, is a complication that often greatly delays convalescence. It occurs, according to Murchison, in one per cent. of all cases. It sometimes invades both legs in succession, and may extend into the iliac veins and vena cava, thence even into the right auricle, causing death from syncope. Unless the latter event occurs, however tedious the recovery, it takes place ultimately almost without exception. Very rarely there may be suppuration. Bacilli have been found in the thrombus. More or less phlebitis is always present. The question as to the primary event, whether thrombosis or phlebitis, is seemingly settled by this finding of bacilli, in favor of the former. *Arterial* as well as *venous thrombosis* may occur, and the former may start with embolism; femoral arterial obstruction is most common, resulting in gangrene of the leg and foot. *Embolic abscess* may occur in the kidney and lung.

Arterial sclerosis sometimes succeeds as a consequence of the irritative effect of the toxins.

A gangrenous condition of the skin and underlying tissues, may occur over any point subjected to pressure, most commonly over the prominence of the sacrum, or over the heels. This complication for want of examination is often overlooked, until the continued fever, often of remittent type, forces a careful examination. Bedsores, formerly frequent complications in protracted cases, are much less frequent with modern nursing.

Noma, or gangrenous stomatitis, has appeared as a complication or sequel in children. W. W. Keen records nine cases, of which five proved fatal. Gangrene in other situations occurs more rarely, as in the vulva in females and in the perineum and about the anus in both sexes. This may be due to arterial thrombosis. Perineal fistulæ may follow in these cases.

It has been mentioned that *pneumonia* may usher in the disease, and a few words may be said here of the relation of the two conditions, pneumonia and typhoid fever. The term *typhoid pneumonia* is one in common use by many who have no definite notion of its meaning, and, like the term typhomalarial, has occasioned confusion. In the first place, the case may begin as a lobar pneumonia, the intestinal symptoms appearing at the end of the first week or later, at which time also the spots may appear, establishing the diagnosis, while the usual crisis of pneumonia fails to make its appearance. Again, a pneumonia may supervene in the second or third week of a

typhoid fever as a complication in which the true relation is less difficult to determine. Finally, there may be a true pneumonia, to which stupor, a dry tongue, and general adynamia may be added, without the distinctive lesions of typhoid fever. This is true typhoid pneumonia, which it may not always be easy to separate from the typhoid fever beginning with pneumonia. The typhoid bacillus and the pneumococcus may be present. Hypostatic congestion has been referred to. Many cases formerly thus named are really instances of catarrhal or lobular pneumonia belonging to the class of inhalation pneumonias. Such may terminate in abscess and gangrene. When *pleurisy* occurs, it has the same relations to the disease as pneumonia. It is, however, more rare, but may also be purulent. An *initial* nephritis has been mentioned on page 12.

Certain *suppurative processes* sometimes included as symptoms should be regarded rather as complications than symptoms. Of these those in the parotid gland and ear are the most serious. They are, however, less frequent in typhoid than in typhus fever. They are most common in the parotid gland, where, however, the inflammatory process does not always terminate in suppuration, occasionally resolving itself with or without local treatment. The duct of Steno is probably the route of infection in these cases by the pus organisms that find conditions favorable to their work. The middle ear may be invaded, producing *otitis media*. Here the Eustachian tube becomes the route of infection. Sometimes there are multiple abscesses due either to staphylococcus or to the typhoid bacillus convalescence often being delayed by these collections of pus.

The *bladder* may be a seat of suppuration, and *pyuria* is not infrequently present. George Blumer found it in ten out of 60 cases, or nearly 17 per cent., of a series admitted to the Johns Hopkins Hospital. It was present in a pronounced form in a series of 41 of our cases. It is probably caused by the typhoid bacillus. The inflammation may extend to the pelvis of the kidney or begin there. *Orchitis* and *epididymitis* are also occasional symptoms during convalescence. Thompson S. Westcott collected 32 cases for Keen's book.[1]

Cardiac complications, including pericarditis, endocarditis, and myocarditis, are sometimes present. The latter may be a cause of sudden death.

Neuritis is an occasional complication in both the local and multiple forms. Osler found it, however, in but four of 389 cases. The pain may be severe and associated with the usual tenderness of the nerve trunks. The *tender toes* first described by Handford, are not very rare. The tenderness is often so great that the bed-clothing must be kept raised by a cradle. Even cases of optic neuritis with atrophy of the optic nerve have been reported, but it is probable that these are sequelæ of meningitis mistaken for typhoid fever. *Tetany* sometimes succeeds typhoid fever.

Two sequelæ of typhoid fever, neither of frequent occurrence, are conspicuous by their symptoms. They are *insanity* and *tuberculosis of the lungs*. The former is often typical acute mania, requiring the utmost vigilance to prevent the patient from injuring himself and others, or from escaping from the house or jumping from a window. Although this form of insanity is often prolonged for many weeks, the prognosis is singularly favorable, and

[1] "Surgical Complications and Sequels of Typhoid Fever," 1898.

recovery, sooner or later, takes place. *Tuberculosis*, when it occurs, has its predisposing cause in the lower tone of cell life, favoring the successful implantation of the specific bacillus, and is followed by its usual consequences.

Post-typhoid *bone lesions* are surprisingly common. Sir James Paget, Murchison, W. W. Keen, Haywood, Harold C. Parsons, and others have collected many cases. They include osteitis, necrosis, and periostitis. The tibia is the favorite seat—91 times out of 216 of Keen's collection—next the ribs 40 times, the femur 22 times, the ulna 15, and the humerus 11. Ebermaier, in 1887, obtained from two cases of suppurative post-typhoid periostitis the bacillus of Eberth in pure culture, and since then quite a number of cases have been reported; whence pyogenic properties of this bacillus may be inferred. Other bacilli—viz., the staphylococcus, streptococcus, and pneumococcus—are, however, at times associated. Golgi also produced suppuration by injecting pure typhoid bacilli subcutaneously at a distance from the fractured ends of a long bone in a lower animal. The pus showed in culture only typhoid bacilli.

Perichondritis appears to be a frequent complication in Germany, as shown by the collections of Keen, Lüning, and Westcott—169, 13, and 14, respectively. Keen's and Lüning's lists include the same cases. The disease is certainly less common in England and America. Necrosis of the cartilages, as well as ulcers, are frequent results. *Arthritis* is an occasional complication. All of these surgical complications are easily explained since the discovery of the bacillus.

The *typhoid spine*, to which attention was called by Gibney, of New York, in 1889, is a sequel of undetermined nature. There is severe pain in the back, commonly aggravated by motion. The pain may be throughout the whole spinal region or limited to the cervical, dorsal, or lumbar portions. From the latter it may extend toward the hips. It may be a spondylitis, but is probably a pure neurosis. Allied to this condition is perhaps an obstinate periostitis of the sternum or the crest of the ilium or front of the spinal column after typhoid fever, alluded to by William Pepper in the "Text-book by American Teachers." These conditions are rare and sometimes, at least, may be coincidences.

Cholelithiasis is now a well-recognized sequel, Dufourt having first reported it in 19 patients who had their first attack after typhoid fever. Further interest attaches because there is every reason to believe that the bacilli in the gall-bladder may be the initial cause of the process which results in stone. Bernheim first called attention to this possibility in 1889, and is sustained by Dufourt, Milan, Hanot, Maurice H. Richardson, Mason, W. H. Welch, and W. W. Keen.

Relapses.—These occur readily. As long ago as 1871, Hamernjk, quoted by Murchison and Maclagan,[1] suggested that the relapse is really a reinfection of the large intestine from the small by the passage of sloughs over healthy lymphoid follicles. Hugh Stewart[2] reiterated this suggestion in 1894, but Murchison had early noted that the fresh lesions are sometimes higher up in the ileum than those of the first attack. Liebermeister believed that a part of the typhoid poison remained latent somewhere in the body,

[1] "Edinburgh Med. Jour.," vol. xiv., part ii., p. 865, 1871.
[2] "Practitioner," vol. liii., p. 185, 1894.

awaiting some exciting cause to bring it into activity. G. Fütterer[1] claims to have been the first to discover the typhoid bacillus in the gall-bladder in 1888;[2] also that he was the first to express the opinion that relapses are caused by typhoid bacilli entering the intestines with the bile. Duprè[3] and Chiari[4] were among the first to find typhoid bacilli almost constantly present in the gall-bladder of those ill with typhoid fever, and also suggested the possible responsibility of these bacilli for relapses. They may be discharged into the small intestine without harming it after immunity is secured. Prior to this, however, the patient may suffer a relapse. Thus may be explained the occurrence of relapses after indiscretions in diet, which stimulate the discharge of bile and bacilli into the bowel, thus increasing the chances of infection. Chiari's experience adds further confirmation, since in three cases of relapse the number of bacilli in the gall-bladder was very large. B. Curshmann, in his paper on typhoid fever in Nothnagel's "Encyclopedia of Practical Medicine," says of relapses: "Undoubtedly their development is to be attributed to the re-entrance into the circulation of living typhoid bacilli which, after the primary attack, were left behind in various organs; and associated with this, more or less complete development of the local and general typhoid lesions occurs."[5]

The signs necessary to the diagnosis of relapse are the presence of those symptoms, essential to the primary diagnosis—viz., the characteristic spots, a return of the tidal or step-like temperature, and, scarcely less so, the enlarged spleen, and all of these after complete defervescence. The attack is usually less severe, the duration *shorter*, and recovery the rule. Relapses are to be distinguished from *recrudescence*, which is a simple return of fever, often induced by numerous causes, including lapses in diet, too much excitement, and the like. Relapses may be multiple. Transverse markings on the finger-nails incident to multiple relapses are sometimes noted. The number of cases in which relapses occur varies greatly in the experience of different observers—from one to 18 per cent. Of 112 cases admitted to the Hospital of the University of Pennsylvania from the various military camps of the country, in the fall of 1898, there was a percentage of 10.7.

Relapses are more frequent in young persons than in older ones. A little girl of 14 in the Pennsylvania Hospital had six relapses with febrile periods of two or more weeks and a total duration of the illness of almost a year.

Diagnosis.—Typhoid fever is usually easily recognized, but sometimes the diagnosis may have to be delayed until the distinctive signs appears The peculiar range of temperature is the most distinctive symptom, and from it alone the diagnosis may be tentatively made. The rose-colored spots, occurring about the eighth day, are conclusive if present, but they are occasionally absent. Diarrhea is less constant, and nosebleed still

[1] "Medicine," November, 1898.
[2] "Münchener med. Wochenschrift," No. 19, 1888.
[3] "Les infections biliaires," "Thèse de Paris," 1891.
[4] "Prager medicinische Wochenschrift," 1893, No. 22. See also Brannan, "Twentieth Century Practice of Med.," vol. xvi., pp. 678 and 679.
[5] The term recrudescence is not always similarly used. Thus Curschmann, in the paper alluded to, regards relapse and recrudescence as due to the same cause and calls it relapse if it succeeds upon a perfectly afebrile period, and recrudescence if the rise in temperature occurs during the period of involution before the declining temperature has completely returned to the normal. I prefer to retain the distinction given in the text, which is also that adopted by Osler.]

less so, but more characteristic. Both, however, require to be weighed in association with other symptoms. No one symptom is pathognomonic.

The resemblance of typhoid fever to certain cases of *acute tuberculosis* has long been recognized. Certain cases of *malarial fever*, especially the autumnal type, also very closely resemble typhoid, but here, too, the temperature diagram is not identical, while the usually easy recognition of the malarial organism completes the solution. Where the two diseases are concurrent, as is sometimes the case, the difficulties are increased.

Mention has been made of the close resemblance of the so-called nervous variety of typhoid fever to meningitis, and it is sometimes so misinterpreted. In every doubtful case a spinal puncture should be made. This will decide the question. As the disease progresses, the distinctive signs develop and the correct diagnosis is gradually made. The popular term, "brain fever," now passing into disuse, doubtless included many of the cases of nervous typhoid.

More misleading, even though less frequent, are the cases beginning with decided pulmonary symptoms suggesting *pneumonia* rather than typhoid fever, and unless the physician is awake to the possibilities of such a beginning and watches further developments the case may be regarded as one of pneumonia with typhoid symptoms. Doubtless some cases that are still regarded as lobar pneumonia are typhoid fever. Such a mistake might have been made in the case reported by Osler in the third edition of his "Text-book," when only the symptoms and morbid anatomy of pneumonia were found, but in which pure cultures of the typhoid bacillus were isolated from the lungs, liver, kidneys, and spleen. No lesion of the intestine and no other organisms were present.

Certain cases of *concealed suppuration* resemble typhoid fever in the symptoms produced, and may for a time mislead. But again the temperature chart, after a few days' observation, will help to solve the question. It is in such cases that a study of the blood is of value—the presence of leukocytosis pointing to suppuration, and its absence, to typhoid. Brill's disease, which Anderson and Goldberg have proven identical with mild typhus fever, is characterized by mild fever, but the rash is different, and it may certainly be distinguished from typhoid by the absence of Widal reaction and the sterility of blood cultures.

Of specific aids to diagnosis the isolation of the bacillus is now done with ease in any good laboratory. Some recent studies by Warren Coleman and B. H. Buxton go to show that in 75 per cent. of 604 cases bacilli have been isolated from the blood at some stage of the disease.[1] *The serum diagnosis, or the Widal-Gruber* reaction, which depends upon the fact that the diluted serum of a patient suffering from typhoid fever will cause actively motile typhoid bacilli to lose their motility and to become aggregated into clumps, is the best aid at hand. The active principle underlying this reaction is the presence in the blood of a substance termed *agglutinin*. In many diseases this substance is present, and it is found to be specific in its reaction to the causal bacterium. However, in some normal sera a non-specific agglutinin is found, which will produce the agglutination of several varieties of bacteria.

[1] "Bacteriology of the Blood in Typhoid Fever." Proceedings of the New York Pathological Society, 1904, N. S. iv.

The test may be said to be pathognomonic, but, because of conditions to be spoken of later, not always applicable as an aid to the immediate diagnosis of a doubtful case. Kneass and Stengel[1] report that in 2383 cases of typhoid fever the reaction was present in 95.5 per cent. of the cases, and that in 1365 non-typhoid cases it was absent in 98.4 per cent. of the cases. Taking these statistics, the absence of the reaction in 4.5 per cent. of the typhoid cases may be due first, to faulty clinical diagnosis, for at the present time there is reason to believe that there are infections caused by bacilli of the typhoid-coli group, the sera of which will only agglutinate these modified types, which have been termed paracolon and paratyphoid infections. Second, it may be due to the fact that in these cases the test was not applied continuously during the supposed attack of typhoid fever, since from statistics collected by Hermann Biggs, of the Health Department of New York City, the serum of typhoid patients gave the reaction during the first week in about 70 per cent.; during the second week in about 80 per cent.; and during the third and fourth weeks in about 90 per cent. of the cases. Thus in cases clinically typhoid the test should be made every two or three days during the disease before it can be said that the reaction is absent. This late reaction, of course, is of little practical value, since the diagnosis will have been made much earlier by the more usual methods. The reaction has appeared for the first time as late as the 42nd day, and in a few isolated cases has remained absent throughout the course of the disease. The reaction has been found as long as eight years after recovery.[2]

The presence of the reaction in 1.6 per cent. of non-typhoid cases is due either to faulty technique, *i. e.*, the dilutions were not high enough since the agglutinin found in some normal sera will agglutinate the typhoid bacilli in insufficient dilution; or to the fact that the patient may have passed through a typhoid infection some months previous, because the reaction has been found in some cases to be present many months after the recovery from the disease. It may occur as early as the third day, but is usually observed about the seventh day. It gradually becomes more marked as the disease progresses, and is commonly present in the blood of convalescents, and for months after recovery, though in some cases it disappears before the end of the disease. It is also true that the severer the infection, the more marked the reaction, and *vice versa*. Pleural and pericardial effusions, the bile, the milk, and to some extent, the urine of typhoid fever cases, as well as the blood serum, possess this agglutinative property for typhoid bacilli. Widal reaction is present after vaccination by typhoid cultures.

Diagnosis of Perforation.—In view of recent increased success of operation for perforation, an early recognition of this accident becomes imperative, to which end a daily examination of the abdomen should be made. A case of typhoid at any stage of the disease presenting the signs of local pain, tenderness and rigidity in the abdomen, should at once be suspected of perforation. If these local signs are proven not to be caused by pneumonia or pleurisy and especially if they are accompanied by leukocytosis, not the result of some other complication they are signs of a preforative perito-

[1] Gould's "Year book," 1898.
[2] "Clinical and Scientific Contributions upon the Value of the Widal Reaction, based upon the Study of Two Hundred and Thirty Cases," Philadelphia Med. Jour., vol. iii., p. 778.

nitis, and a surgeon should be consulted. To wait for tympany, decrease of liver dullness, drop of temperature and rapid pulse is to wait for general peritonitis and the probable death of the patient. It may occur in the mildest cases, and after the temperature has been normal for ten days, and in such especially, the appearance of localized pain and tenderness may also be regarded as a warning. Hemorrhage from the bowel is occasionally followed by perforation. Every case should be carefully watched.

In a second class of severe cases where there is delirium or stupor, abdominal distention may be the only symptom. In a few instances there are no evident signs and the perforation may be first found at autopsy. This occurs commonly in cases of unusual gravity where the event is masked by the severity of the symptoms.

Prognosis.—The mortality of typhoid fever varies so much in different epidemics and under different circumstances that statistics are of doubtful value in measuring fatality. Extremes of mortality claimed are as low as one per cent., and even less by the Brand bath method as carried out on the continent of Europe, and as high as 55 in army practice during campaigns and among negroes. The average of all may be put down approximately at from ten to 30 per cent. before the Brand cold tub treatment was instituted. Prior to this, hospital treatment appeared less successful than that of private practice. Since its introduction, because of the greater ease with which that treatment can be applied in hospitals, this can hardly be said to be the case.

In private practice a decided majority get well, fully 80 per cent., with rest, liquid diet, and family nursing. With skilled nursing, judicious feeding, and symptomatic treatment, a larger proportion of recoveries takes place, say 90 per cent. In hospitals where the Brand method is correctly carried out there is an easy reduction of mortality to seven per cent. and less. In this country the results have not been quite so satisfactory as claimed on the continent of Europe. The mortality of William Osler's cases at the Johns Hopkins Hospital, Baltimore, has been 7.3 per cent. Tyson, at the Hospital of the University of Pennsylvania and at the Philadelphia Hospital has been 7.3; that of James C. Wilson and others at the German Hospital, up to January 1, 1896, 7.25 per cent.—astonishingly uniform results. Brand's own mortality has been but one per cent. Of Tyson's cases treated by the Brand method almost all who died perished through perforation or hemorrhage of the bowels, the remainder from exhaustion, or toxemia. Among the soldiers at the University Hospital in 1898–99 treated by the Brand method the mortality was 4.5 per cent. Of 1948 cases at the Pennsylvania Hospital in the years 1901 to 1903, inclusive, the mortality was 7.8 per cent. The Brand bath treatment is less rigidly carried out at this hospital than at the University Hospital or the German Hospital in Philadelphia. Among causes which have contributed to reduce percentage of deaths is the including of mild cases as determined by more accurate diagnosis.

Unfavorable symptoms are persistent high temperature, above 105° F. (40.5° C.), low muttering delirium, extreme tympany, hemorrhage from the bowels, and the signs of perforation. Walking typhoid is frequently fatal, exhaustion being apparently caused by the continued muscular effort during

fever. Complications such as hemorrhage and perforation are likely to occur.

Sudden death by syncope occasionally occurs, sometimes when least expected, during convalescence, or it may happen during the acme of the fever. In either event the immediate cause is not always discoverable, evident lesions being wanting in most cases. Pulmonary thrombosis and myocarditis have been found at autopsy in these obscure cases. Sudden death is much more frequent in men than women—114 to 26, according to Dewèvre's statistics—a surprising and almost incredible difference.

The *prognosis in children* under 15 is especially favorable. Recovery takes place in them with few exceptions, while the number of fatal cases in young people from 18 to 22 is remarkable. Then follows a period favorable to recovery, but after 40 the mortality again increases. The dangers at this older age appear to be from complications, especially pneumonia, as the symptoms peculiar to the disease are not increased in severity.

The *prognosis in pregnant women* is grave. In the first place, the pregnant woman usually aborts in the second week. According to L. Brieger, the mortality was 20 per cent. of cases treated by other than the bath method. The results of the bath treatment seem to be better. Recently Tyson had under his care two pregnant women at the end of the fifth and sixth months, respectively, now recovered, who were treated throughout by cold tub-baths without accident. Under any circumstances more women die of typhoid than men—this, too, though the disease is more frequent in men than in women. Fat persons bear the disease badly. Hemorrhage and perforation seem to be in no degree diminished by the Brand bath treatment. On the other hand, careful investigation shows that these accidents are not more frequent, as has been alleged.

Death in typhoid fever may be the result of any of the following causes: exhaustion incident to prolonged illness, hemorrhage, peritonitis, shock due to perforation, intoxication by the toxin of the disease, thrombosis or complications such as pneumonia or nephritis. As already intimated, sudden death sometimes occurs inexplicably.

Treatment.—*Rest and Diet.*—The primary conditions of a successful treatment of typhoid fever are rest in bed and a selected diet, of which milk forms an important part. No one questions the necessity of putting the typhoid fever patient absolutely at rest in bed and not permitting him to rise for any purpose until convalescence is thoroughly established. Coleman has shown that starvation leads to complications. The diet should be selected and contain a sufficient number of calories 2500 to 3000 for an adult to fully sustain the patient's strength, bearing in mind the fact that the vast majority of patients with typhoid fever have a local ulceration in the intestine that must not be unduly irritated. Each individual must be treated and given the diet best suited to his particular case. In the semi-conscious patients, or in those very delirious, a liquid diet made up largely of milk, is best suited. One quart of milk contains 700 calories. The calorie value can be greatly increased by the addition of cream and milk sugar, 1 ounce of cream being equivalent to 60 calories, and one ounce of milk sugar is equivalent to 36 calories. One and one-half quarts of milk, 8 ounces of cream, and 6 ounces of lactose will furnish 1700 calories. This has been

carefully worked out by Coleman, his patients all doing well upon the diet. Patients who are quite rational, and who are able to eat, can be given a carefully selected diet in addition to the above, the one selected by Shattuck in 1897 seems perfectly satisfactory. This diet consists of the following articles:

1. Milk, hot or cold with or without salt, diluted with lime water, soda water, Apollinaris, or Vichy. Peptonized milk; cream and water (*i. e.*, less albumen) milk with white of egg, buttermilk, kumiss, matzoon, milk whey, milk with tea, coffee, cocoa.

2. Soups: beef, veal, chicken, tomato, potato, oyster, mutton, pea, bean, squash, carefully strained and thickened with rice, powdered arrowroot, flour, milk or cream, egg, barley.

3. Horlick's food, Mellin's food, malted milk, somatose.

4. Beef juice.

5. Gruels: Strained cornmeal, crackers, flour, barley-water, toast water, albumen-water with lemon juice.

6. Ice cream.

7. Eggs, soft boiled or raw, egg-nogg.

8. Finely minced lean meat; scraped beef; the soft part of raw oysters; soft crackers with milk or broth; soft puddings, without raisins; soft toast, without crust; blanc mange, wine jelly, apple sauce, and macaroni.

Ice cream may be given in any quantity which does not cause nausea or diarrhea.

The Brand Bath Treatment.—In addition to rest and selected diet, the treatment that experience places easily at the head, in every case when it can be carried out, is the cold tub-bath treatment, commonly known as the Brand treatment. Our method is as follows:

Before the bath the patient is first encouraged to empty the bladder, and if sweating, he is wiped dry. He is then covered loosely with a sheet and gently lifted into the bath sufficiently filled with water at 70° F. (21° C.), provision being made to rest the head upon an air-cushion or platform. During the bath he is vigorously rubbed by the nurse, and encouraged also to rub himself. A compress wrung out of ice-water or an ice-cap is kept upon his head, or water at the same temperature is poured at intervals upon it, say, three times in the course of the bath, or the head is sponged with cold water from time to time. This is important in severe cases with decided nervous symptoms. At the end of 15 minutes he is lifted on the bed, which has been previously protected with a mackintosh and blanket. The wet sheet is replaced by a dry blanket, and the patient is rubbed dry. When this is accomplished, the under blanket and mackintosh are withdrawn and he is comfortably covered.

As soon as the patient ceases to shiver after his removal from the bath, which is usually in 20 minutes, the temperature is taken with a view to determine the effect of the bath. If delayed longer he may be in a restful sleep, and to wake him for the purpose of taking his temperature is needlessly disturbing. After this the temperature is not again taken until three hours after the bath. If then it exceeds 102° F. (39° C.), the bath is repeated. If the temperature is between 101° F. (38.2° C.) and 102° F. (39° C.), it is taken again in an hour; if between 100° F. (37.8° C.) and 101°

F. (38.3° C.), in two hours; if below 100° F. (37.8° C.), not until three hours, but whenever the temperature exceeds 102° F. (39° C.) the bath is given, provided three hours at least have elapsed since the previous bath. This makes more than eight baths in the 24 hours impossible.

The effect of the bath upon the temperature varies with the stage of the disease; the reduction during the first week being often less than one degree, while toward the end of the second week and in the third week a fall of two or more degrees is quite usual. Fig. 4 shows these effects very nicely. In

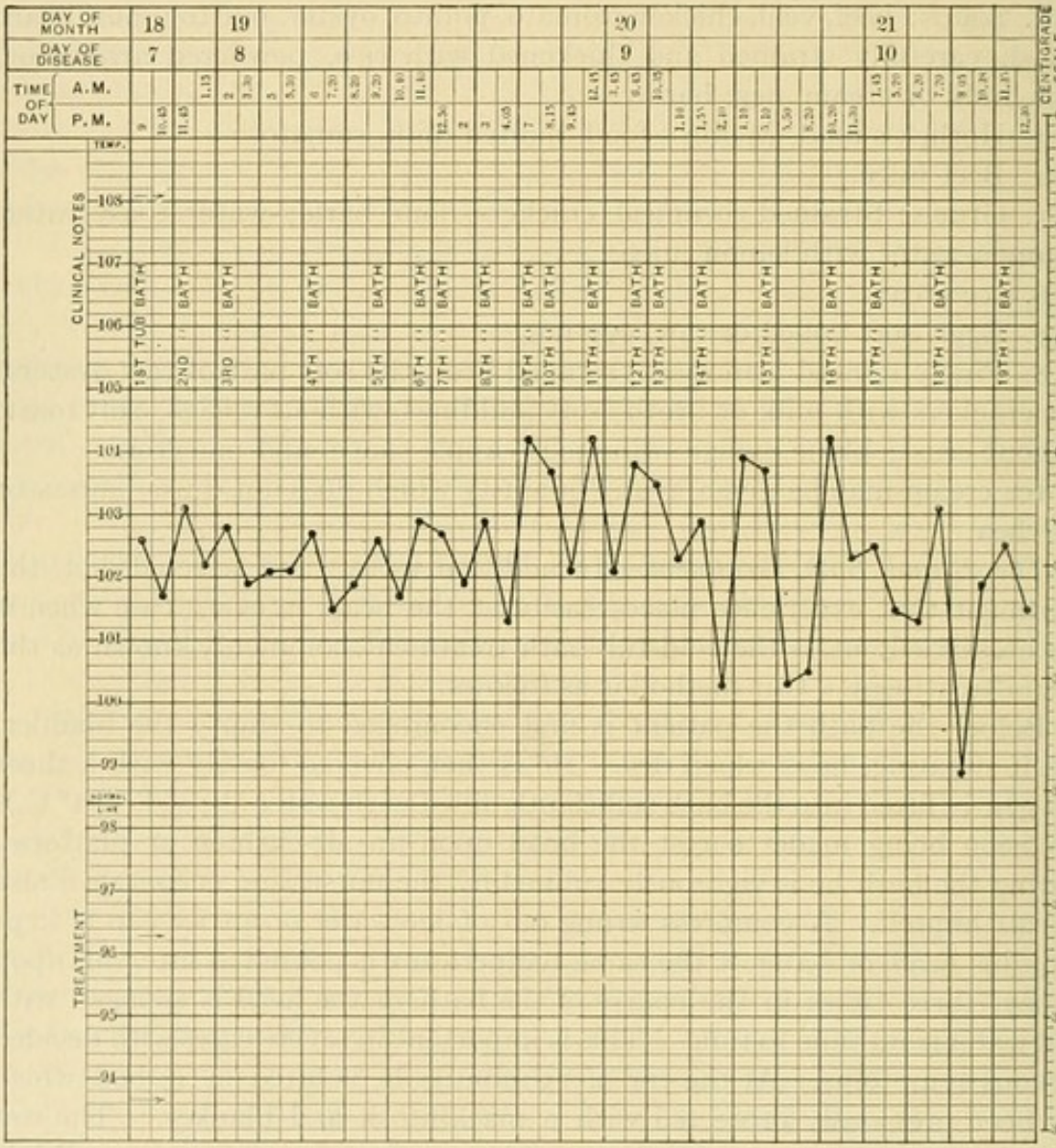


FIG 4.—Chart Contrasting the Drop in Temperature after the Bath Early and Later in the Disease.

addition to the lower temperature the immediate effect of the bath is to add strength to the heart and volume to the pulse. The shivering, which begins from five to ten minutes after the immersion, is not allowed to interfere with the continuance of the bath, and it very rarely happens—indeed, scarcely ever—that anything occurs to interrupt the bath. It would be wrong, however, to say that there are no conditions under which it should be discontinued and the patient at once returned to bed. Such conditions are an almost absolute pulselessness with a blue, cyanosed appearance

of the skin. Should this occur, hot-water bags should be applied to the feet and legs after the patient is put to bed.

The more remote effect of the bath may be said, in a word, to be amelioration of the symptoms in every particular. Delirium and stupor are lessened. The dry tongue is very much more infrequent, and diarrhea rarely demands other treatment. The majority of cases require no medicine but symptoms should be treated that demand it by appropriate treatment. Sometimes whisky and water during the bath are necessary.

None of the complications except hemorrhage from the bowels and signs of perforation is allowed to interfere with the carrying out of this treatment, nor is menstruation or even pregnancy. The baths are discontinued during hemorrhage, lest the necessary movements of the body should re-excite it. It is not claimed that the baths shorten the illness, they simply ameliorate it. While it is probably true of typhoid fever, as of pneumonia, that it may abort spontaneously, we cannot cause it to abort by any means we possess.

In private practice the difficulties of the Brand treatment are greatly increased—unfortunately, sometimes are insuperable. They consist chiefly in the difficulty in arranging the bath and the strain on the attendants. By means of a portable tub devised by A. H. Burr,[1] of Chicago, a very large part, if not the whole, of these difficulties is removed. A large rubber blanket spread upon the bed and lifted at each corner allows the giving of what is practially a tub bath. The blanket is large enough to extend far over the edges, the foot and head of the mattress, the corners are looped up and securely tied. The patient is stripped, then water of the desired temperature is dripped in a stream from a sponge held over the patient. The surplus water is caught by the blanket,. This bathing is continued 15 to 20 minutes. The water is removed by sponging or a hose. The patient is dried and covered.

Other Methods of Reducing Temperature.—As contrasted with the Brand bath, other methods of securing the good effects of hydrotherapy seem trifling; yet, as it may be impossible to carry out this treatment, such methods must be considered. Sponging is one of the most usual, and if rightly carried out may be quite efficient. It should be resorted to, as is the bath, when the temperature exceeds 102° F. (39° C.), and continued for 15 minutes, or until the temperature falls. An important condition of successful sponging is often overlooked. A thin film of water should be left on the surface sponged, as it is the evaporation of this, rather than the temperature of the water, which is effectual in cooling the body. Temperatures that cannot be thus controlled can often be kept down by a partial wet-pack, which is often very efficient: The patient's trunk is enveloped from the axilla to the thighs in a folded sheet, which is kept constantly wet, or as much so as is required to control the temperature, by the continual addition of cold water.

Antipyretics, including antipyrin, antifebrin (acetanilid), phenacetin,

[1] The Burr bath-tub is sold by E. H. Sargent & Co., 106 Wabash Avenue, Chicago, Ill. Another tub, as convenient and as easily managed, has been devised by S. Clifford Boston, West Grove, Pa., who dispenses with the framework, substituting strong iron supports, made by Jones, Leopold & Co., Southwest corner Ridge Avenue and Fairmount Avenue, Philadelphia.

A convenient tub which may be used on the bed, known as the "Coile Bath Tub" is made by the Roco Bath Co., Knoxville, Tenn. The price advertised is $100.

and others of the same class are mentioned to be condemned. They should never be used.

The Expectant-symptomatic Treatment.—Where the difficulties in the way of the Brand method are insuperable, we prefer to place the patient in bed on the diet described, combat the temperature by sponging or wet-packs, and for the rest adopt what may be termed the expectant-symptomatic method, meeting the symptoms as they arise in accordance with the following:

Alcohol is not a heart stimulant as formerly supposed. That a certain amount may be utilized as food is certain. That alcoholics afflicted with typhoid fever need some form of alcohol seems certain, therefore alcohol is used in good doses 30 to 60 c.c. every two or three hours in alcoholics in the early stages but is gradually reduced in amount. It is sometimes used in small amounts as a food when this seems necessary.

It is never used in large doses as a stimulant for it does not have this effect. However when the heart muscle begins to flag, the pulse becomes rapid, and the patient extremely weak, doses of 15 to 30 c.c. may be given every two or three hours with good effect. It may thus act as a fuel and indirectly stimulate the heart. A low, muttering delirium, feeble, dicrotic pulse, and dry tongue are among the indications which imperatively demand small amounts of alcohol; a high temperature does not contraindicate it, as an antipyretic effect also follows its use and delirium is sometimes calmed by it. Other diffusible stimulants which may be used in conjunction or alternation with alcohol are the aromatic spirit of ammonia and the carbonate of ammonium, while digitalis and strychnin may tide a feeble heart over a period of weakness. From 5 to 10 minims (0.333 to 0.666 gm.) of the tincture of the former, and 1/30 to 1/20 of a grain (0.00216 to 0.00324 gm.) of the latter may be given as demanded, while their hypodermic use may be availed of. We have found hypodermic injections of camphorated oil 1 grain (0.066 gm.) to 15 minims of the oil (1 gm.) very useful in tiding over extreme adynamia. The injections may be repeated once in four hours or oftener. Caffein, 2 grains (12); may be taken by the mouth. It is an excellent stimulant.

Transfusion or, what is more practicable and as efficient, hypodermoclysis of normal salt solution (0.8 per cent. sodium chlorid) may be availed of in the extreme adynamia which sometimes attends protracted typhoid fever. Vaccine *treatment* by killed bacilli is not of value. No serum has been found of value.

(*b*) *Treatment of Special Symptoms.*—Methods more directly adapted to control *delirium* are an ice-cap to the head, the bromids, spirit of chloroform, chloral, and Hoffmann's anodyne. With the cold-bath treatment they are rarely necessary.

Little difficulty is commonly experienced in controlling the *diarrhea* of typhoid fever. As stated, with the cold bath treatment very little special treatment is necessary. Often omission of all food is necessary. Simple preparations of opium, either alone or in combination with bismuth or nitrate of silver or acetate of lead, or salol, are usually sufficient. One-quarter grain of nitrate of silver and 1/4 grain of extract of opium is an excellent remedy for diarrhœa. The impression made by the teachings of the late

George B. Wood on the profession of the United States as to the effect of oil of turpentine has not yet been effaced. He held that the dry, leathery tongue so often presented in this disease is the indication for its use. Whether such view was correct or not, few who have used the oil of turpentine have failed to see the coated tongue clear up under its use. Turpentine is also useful as a stimulant. It should be administered in doses of 10 minims (0.66 gm.) in mucilage of acacia every six or eight hours.

Constipation, especially during convalescence, is not infrequent, and should not be too hastily interfered with. If it is necessary to interfere, it should be simple enema only. Aperients by the mouth in this stage are dangerous, they may be succeeded by perforation, peritonitis, and death. On the other hand, indifference to the condition of the bowels sometimes leads to fecal impaction, which can only be relieved by the finger. Such a state of affairs should be averted by watchful care.

Hemorrhage from the bowels should be treated by absolute quiet with cold to the abdomen. Food should be interdicted. The administration of food may be suspended for some hours without risk. In severe cases the foot of the bed should be raised, and a hypodermic injection of 1/8 to 1/4 of a grain (0.008 to 0.016 gm.) of morphin given at once and the extremities bandaged. In cases where prompt and decisive action is necessary, hypodermoclysis, or intravenous injection of normal salt solution, may be necessary, to which 10 minims of a 1–1000 adrenalin chloride solution may be added. This is rarely necessary. The lowered blood pressure is one of nature's ways of controlling the bleeding. Therefore do not give stimulation to every severe case of hemorrhage. In mild cases, astringents such as tannic acid or gallic acid and the acetate of lead, may be given by the mouth, the former in doses of 10 to 15 grains (0.666 to 1 gm.) hourly until some hours have elapsed without a hemorrhage. The acetate of lead should be given in 1 to 3 grain (0.066 to 0.194 gm.) doses every three hours, combined with extract of opium, 1/4 of a grain (0.016 gm.). Turpentine is of value in the treatment of hemorrhage from the bowels. In cases of extreme bleeding from the bowel apparently due to hemorrhagic diathesis, injection of normal blood serum may be used with good effect.

Tympanitic distention of the abdomen is often a distressing symptom. It is usual to treat it with turpentine in 10 minim (0.666 gm.) doses every four to six hours. The rectal tube should be cautiously used if the meteorism is great, and large quantities of gas are sometimes thus disengaged from the large intestine. The quantity of food should also be reduced to a minimum, as its fermentation and decomposition contribute to the gas.

Pain induced by meteorism or otherwise may be allayed by turpentine stupes over the abdomen, though sometimes it may be necessary to reinforce the stupes by small doses of opium, or a light, warm poultice may be substituted. Sudden, sharp pain, similar to that produced by tympanitic distention of the bowel, is also caused by *peritonitis*, of which tympany is likewise a symptom, and the two often occasion many anxious moments to the physician necessarily in doubt as to whether this serious complication may occasion them. Here the surgeon must at once be called. A local peritonitis is a call for a laparotomy.

Perforation is the most serious accident which can happen to the typhoid

fever patient, though it is claimed that recovery has taken place where peritonitis has been thus caused. Indeed, according to Murchison, 10 per cent. of all cases recover, five per cent. if general peritonitis supervene. Even this seems a large proportion, for in our experience no case of undoubted perforation has recovered without operation. On the other hand, recent results after operation have been so favorable as to make it imperative that every case should be operated upon. It is important to remember that early operations are those attended with largest success. In an exhaustive paper ("Jour. Am. Med. Assoc.," January 20, 1900) on the "Surgical Treatment of Perforation of the Bowel in Typhoid Fever," W. W. Keen collected 158 cases and summarizes as follows:

OUT OF 158 CASES OF OPERATION FOR PERFORATION.

When Done	Total	Died	Recovered	Percentage of Recoveries			
Within 4 hours	8	6	2	25	19.44	15	25.33
In 4 to 8 hours,	12	11	1	8.33			
In 8 to 12 hours,	16	12	4	25	30.76	29.09	
In 12 to 18 hours,	25	17	8	32			
In 18 to 24,	14	10	4	28.57			
After 24 hours,	44	38	6	13.63			
Not given,	39	27	12	30.74			
Total,	158	121	37	23.41			

Keen also formulates the rule that *if the operation is not done within about twenty-four hours after the perforation, there is probably no hope of a recovery.* Nevertheless an operation should always be done unless the patient is in extremis. A surgeon should therefore be immediately called, and if collapse is not too profound laparotomy should be done.

For *sleeplessness* the milder soporifics usually answer; 10 to 15 grains (0.666 to 1 gm.) of sulphonal generally furnish the required rest. Trional 15 to 30 grains (1 to 2 gm.), veronal in 5 grain doses, or chloral in 10 to 15 grains (0.666 to 1 gm.) may be used. If these remedies are insufficient, morphin must be used, 1/4 grain (0.016 gm.) being given by the mouth or half as much hypodermically, or more if necessary.

Bed sores can generally be averted by scrupulous attention to cleanliness, the thorough drying of the patient after washing, removing thus all traces of urine or other discharges, and by sponging the patient daily with alcohol or whisky. Above all, his position in bed should be frequently changed and all inequalities in the bed clothing should be smoothed out, while the bed should be kept clear of crumbs and other irritating particles. Should a sore appear it must be aseptically dressed, while the part should be protected from pressure by pads and air-cushions. In cases with extreme emaciation and weakness, a water bed is a necessity, and will cure or prevent bed sores.

For *hiccough* the measures effectual are counter-irritation by mustard, dry cupping, or blistering over the epigastrium; the various anodyne measures, including Hoffmann's anodyne, chloroform, and the hypo-

dermic injection of morphin. The anti-spasmodics, including sumbul, the oil of amber, and especially musk, have been useful. Cannabis indica is also recommended. In an obstinate case, after all measures had failed, including musk, the hypodermic injection of 1 grain (0.06 gm.) of camphor dissolved in oil, 15 minims (0.5 gm.) repeated hourly, relieved the case in six doses. A second case has been relieved in the same hospital by like treatment. In other cases we have found musk useful when all else failed, but it is a most costly remedy and its use is thus necessarily limited. The dose is 5 to 10 grains (0.3 to 0.6 gms.).

The *cystitis* sometimes present in typhoid fever is commonly easily relieved by washing out the bladder with boric acid solution, a dram (4 gm.) to a pint (0.5 liter) of sterilized water; or instead of this salol may be given in 5 grain (0.3 gm.) doses four or five times a day, as a urinary antiseptic. The best remedy is hexamethylenamine which is a derivative of formaldehyd. It must not be forgotten that large doses exceeding 1 to 1 1/2 grams a day may cause severe irritation of the kidneys and bladder. According to Mark W. Richardson daily doses of 30 grains (2 gm.) will remove typhoid bacilli permanently from the urine in a week.

The Management of Convalescence.—In no disease is watchfulness during convalescence more important. The effect of indiscretion in diet in producing relapse and recrudescence has been referred to. But there are other dangers during convalescence. It is to be remembered that the complete have disappeared except a slight elevation of temperature; a deep-seated healing of intestinal ulcers is often delayed after all other symptoms ulcer may thus remain with the thin peritoneum for its floor, rendered weaker by reason of imperfect nutrition. Such a membranous floor is known to have been torn by simply reaching over for a book and to be followed by a fatal peritonitis. These are reasons, too, for putting off the use of a table diet until the temperature has maintained the normal for a considerable time, certainly two weeks. Then the diet should be changed most gradually.

Emotional disturbance is a well-recognized cause of recrudescence, and should be carefully guarded against.

Constipation should be corrected by enemata only.

During convalescence the *hair* is very apt to fall out, but usually returns in a natural way. It may be desirable to cut it close, though scarcely necessary to shave the head, as some recommend.

Antityphoid Inoculation.—The experiments of A. E. Wright and his co-workers show that both the incidence and mortality of typhoid fever can be diminished by typhoid inoculation or vaccination. This is done by injecting subcutaneously dead cultures of the typhoid bacillus, sterilized at 53° C. These injections are made in convenient parts of the body with the usual antiseptic precautions; they are given the first 500,000,000 dead bacterii, the last two 1,000,000,000 each at intervals of three days. Both local and constitutional symptoms supervene after injection. Local symptoms may appear as early as 15 minutes to two or three hours, and are severe inversely as the constitutional symptoms are mild. They are manifested when they occur by a red blush and more or less serous exudation at the site, followed by some lymphangitis upward toward the axilla or

downward toward the groin, according as the inoculation is made above or below the middle line of the body.

Constitutional symptoms also supervene in from 15 minutes to two or three hours, and are increased by muscular exertion after, or fasting before the operation. These symptoms include some headache and malaise in mild cases, with rigors and symptoms of collapse in severe cases, lasting five or six hours. Neither the local nor general reaction is dangerous; 60,000 men have been vaccinated in India, 7000 in Africa and 85,000 in the United States, without mishap.

The general good effect is shown by Kunell and many other reporters. Spooner reports 1588 inoculations among 405 nurses and attendants in hospitals with no untoward results. The incidence of typhoid in vaccinated individuals is much less than in unvaccinated under the same conditions of exposure. After the vaccination Widal reaction is present in the blood just as though the patient had passed through an attack of typhoid fever. As to mortality, in the aggregate the proportion of deaths to cases among the inoculated was less than half that among the uninoculated. The duration of the protection conveyed by antityphoid inoculation persists at least through the second year, probably during the third year. Major Russell, of the U. S. Army reports the vaccination of over 85,000 troops with the incidence of only 3 cases of typhoid fever.

Hunt, American Journal of Medical Sciences, June, 1913, reports the result of vaccinations in an epidemic of typhoid fever which occurred at Troy, Pennsylvania. 1343 persons are known to have used the water supply. 229 cases of typhoid developed. 127 of these developed the fever before the vaccinations were begun, and 103 afterward. The number of persons vaccinated was 761. Of these 37 developed typhoid fever. The number of persons not vaccinated was 455. Of these 65 developed typhoid, that is 4.86 per cent. of the vaccinated, and 14.28 per cent. of the unvaccinated, developed the disease. In the 37 persons who developed typhoid fever, 28 developed it after the first vaccination, 7 after the second, and 2 after the third. The onset was prolonged in every case. The mortality among the vaccinated was 0.28 per cent., among the unvaccinated 8.85 per cent.

Prophylaxis.—Very important in the management of typhoid fever is the *disinfection of the excreta*, which are the contagium bearers, through the careless handling of which the disease is communicated to others. The same is perhaps true of the vomited matters and also of the urine.

Among the most suitable disinfectants, on account of its cheapness, harmlessness, and effectiveness, is *chlorinated lime* or bleaching powder, also called chlorid of lime, which contains from 25 to 40 per cent. of available chlorin. A solution made in the proportion of 4 to 100 of water, containing, therefore, at least one to 1.5 per cent. of chlorin, is sufficiently strong. Some of the solution is placed in the bed pan before it is used, and the remainder, in all say a pint, is added afterward. Thorough admixture should be made, and an hour allowed to elapse before the stool is thrown into the privy or water-closet, if disposed of thus. In the country the disinfected stool may be buried. Solution of chlorinated *soda*, or Labarraque's solution, is a more elegant but not more effective disinfectant. As it contains about two per

cent. of chlorin, it is nearly equivalent, when undiluted, to the above solution of chlorinated lime. Chlorinated lime rapidly loses its chlorin, and should be kept in tight vessels.

Carbolic acid, in the proportion of one part of the commercial acid to ten of water, is an efficient disinfectant for this purpose. The same method as that described for chlorinated lime must be employed, and an exposure of twenty minutes to half an hour maintained. Quite as good a disinfectant for intestinal evacuations is *milk of lime* or ordinary "whitewash," composed of lime in solution and in suspension. This should be thoroughly mixed with the evacuations until the mass is distinctly alkaline, and should remain in contact for one or two hours, since it is slower in its action than chlorinated lime or carbolic acid, and much longer exposures are required to destroy the bacillus. It is particularly adapted to the disinfection of privy wells and latrines, into which it may be thrown, freshly prepared in the proportion of 1 part by weight of recently burned calcium hydrate to 8 of water, or about 12 per cent. It is not harmful to water-closet pipes in such quantities as required to disinfect the stools of a single case of typhoid fever.

Acidulated *solution of corrosive sublimate* 1 to 500 is an admirable disinfectant for stools, but is not altogether harmless to plumbing, whence it is less satisfactory when excreta are thrown into city water-closets.

Sulphate of iron or *copperas* is a good deodorant, but not a true disinfectant. Above all, it must not be forgotten that simple *hot water* thoroughly mixed with the fecal discharges is an efficient disinfectant.

Most important in the prophylaxis of typhoid fever is *drainage*. It seems to be now definitely settled that the fever originates in every instance from the ingestion in some way of the typhoid bacillus, commonly in drinking-water or milk, or in food contaminated with it, more rarely by inhalation. Hence, it is of the greatest importance that the sources of water used in domestic economy should be protected against contamination by discharges containing the specific bacilli, which sometimes find their way into wells and other sources of water-supply.

Nurses should be enjoined to guard against their own infection by due attention to cleanliness after caring for the discharges of a patient and even after tubbing, while watchful care should be taken not to carry the hands to the mouth during the bath. Every nurse should wear rubber gloves when bathing or tubbing a patient. On the other hand, the infection is one of the easiest controlled, and the spread of typhoid fever can be effectually prevented if the precautions advised are followed. Moreover, it cannot be too strongly insisted upon that any infected water or milk may be rendered thoroughly harmless by boiling and filtration. Physicians should lose no opportunity to inculcate this truth as well as that limpidity of a water does not guarantee its innocuousness, while it may even be slightly turbid and yet harmless. The part that flies and other insects play in the spread of typhoid fever is now well understood. All food should be carefully screened in summer time to prevent the approach of flies. All dejecta must be carefully protected from flies, that these insects may not become contaminated by the infective material.

PARATYPHOID FEVER.

Definition.—A form of infectious fever presenting a clinical picture closely resembling that usual to typhoid fever, but usually the symptoms are less severe. It is due to a bacillus whose characteristics are intermediate between the typhoid and colon groups, and called, therefore, the paratyphoid or paracolon bacillus.

Morbid Anatomy.—While ulceration of Peyer's patches may be absent, recent studies have shown its occurrence. The spleen is always found enlarged. The ulcers, when present, are like those of typhoid while there is a characteristic absence of alteration of Peyer's glands and of the solitary follicles. Focal necrosis has been found in the liver. The anatomic lesions are said by Osler[1] to be, in a word, those of typhoid fever.

Symptoms.—The symptoms are those of typhoid. Differences observed are, in general, greater mildness and more favorable prognosis; greater frequency of diarrhea and more frequent termination of fever by crisis. Myositis and purulent arthritis, very rare in typhoid fever, are among the complications. The disease does not respond to the Widal test with typhoid bacilli, but the serum reacts upon fresh cultures of the paracolon or paratyphoid bacillus.

Treatment is in no way different from that of typhoid.

ROCKY MOUNTAIN SPOTTED FEVER.

Definition.—Rocky Mountain spotted fever is an acute infectious disease common in the valleys of the Rocky Mountains, in Idaho, Montana, and along the valleys as far as Mexico, and characterized by fever, headache, muscular pains and a petechial rash.

We owe our knowledge of this disease principally to Maxey and to Wilson and Channing; and to Ricketts and King who described the clinical course of the fever, and the latter its method of dissemination.

Etiology.—The actual cause of the disease is not known. King and Ricketts have proven beyond question that it is spread through the medium of a tick, the Dermacentor Occidentalis; the latter authors have transmitted the disease to animals by means of bites of these ticks; they also found that the ova and young of the ticks contain the infective material. "A certain percentage of the female ticks which have acquired the disease as a consequence of feeding on animals, the latter having been infected by other ticks, transmit the disease to their offspring through the egg. The new generation during the process of feeding, transfer the virus to certain of the susceptible small wild animals (ground squirrel, rock squirrel, chipmunks, ground hogs, and perhaps others) and this may take place either during the larval, nymphal or adult stage, hence at various times of the year. During the infection of the wild animal it is required that hitherto normal ticks either as larvæ, nymphs or adults, acquire the disease by feeding simultaneously with, or shortly after, the feeding of the infected ticks."

Apparently the type of the disease varies in severity in the various areas where it occurs. The disease occurs in the early spring months, just after

[1] Wills and Scott, "Journal of Infectious Diseases," Jan. 2, 1904.

the snow has melted, and is most common among males for the reason that these individuals, particularly herders, are lead by their business to be in the meadow lands and grasses in this region at this time of the year, and thus have an opportunity to be bitten by the tick.

Symptoms.—There is a short period of malaise, followed usually by a well-marked chill. These chills may be repeated throughout the course of the attack. At the beginning there is severe aching of the bones and muscles, and pains in the joints, and severe headache. Constipation is the rule. The skin is dry; the tongue is coated and sordes appear early, and the case has the appearance, except for the chill, of a typhoid in its beginning. The temperature rather rapidly develops and soon reaches 102 or 103 degrees on the third or fourth day. It may go much higher, reaching even 107 degrees. There is usually a slight evening increase and morning decrease. When recovery occurs, the temperature falls by lysis.

Skin.—The most characteristic part of the disease is seen upon the skin. From the second to the fifth day after the chill, a macular rash which rapidly takes on the characteristic petechial appearance appears around the ankles and upon the wrists, and then extends over the entire body. Occasionally the rash will cover the entire body in 12 hours. More usually, however, it takes a longer time. A desquamation occurs in about the second week of the disease, which is best seen on the soles of the feet and the palms of the hands. Occasionally there is jaundice. In severe cases the patients become delirious and pass into a typhoid state. However, there is no sign of meningitis. The pulse is rapid, out of proportion to the temperature. The blood is only slightly changed, the red cells being normal, the white cells being from twelve to thirteen thousand. Except for constipation, there is no unusual sign of disturbance of the digestion. The urine frequently shows the signes of a severe febrile condition, albunim and casts.

Diagnosis.—The diagnosis is usually easy, the condition occurring in the spring in the mountain valleys of the Rocky Mountains. The fever resembles in the beginning typhoid fever but with a petechial rash, the absence of Widal reaction, the rather rapid rise of temperature, and the geopraphical and seasonal distributions, it scarcely can be anything except Rocky Mountain spotted fever. It might be mistaken for typhus; for dengue; for cerebrospinal meningitis, but the characteristics of these latter diseases are so marked, and the geographical distribution of this disease is so certain, that there is scarcely any possibility of making a mistake.

Prognosis.—This seems to be different in different localities. Dr. Maxey's cases almost all recovered, while the cases described by Wilson and Channing show a high per cent. of mortality. There is no pathological condition which is peculiar to the disease.

Treatment.—The treatment is entirely symptomatic. However, the prophylaxis is an important part. Persons whose business calls them into these valleys in the spring time, should be warned of a possibility of infection, and should protect themselves against the bite of ticks in every possible manner, particularly by protecting the feet and hands. General principles should govern the treatment. The patient should be in a cool, well-ventilated room; the diet should be soft and properly regulated. Cold sponging

for the fever, with morphin or other opiate if the patient becomes extremely restless.

As a tonic afterward, the patient can be given nux vomica and gentian.

TYPHUS FEVER.

SYNONYMS.—*Typhus Exanthematicus*; *Petechial Fever*; *Pestilential or Putrid Fever*; *Ship Fever*; *Jail Fever*; *Camp Fever*; *Brill's Disease.*

Definition.—An acute fever frequently occurring where human beings are crowded together, as in jails and ships of olden times; especially characterized by sudden onset of high fever, by a petechial eruption, typhoid symptoms, and short duration as compared with typhoid fever; in favorable cases terminating suddenly at the end of the second week.

Etiology.—Though of acknowledged infectious nature, no organism has as yet been isolated that can be held responsible for typhus fever.[1] Anderson and Goldberg in "N. Y. Med. Journal," May, 1912, have proven by careful animal experiments that typhus fever in the mild form of Brill's Disease is common over the United States and Canada. Fostered by close crowding, filth, and famine, it each year becomes more infrequent as the conditions favoring it are eliminated, and there is reason to believe it will ultimately be stamped out. Thus, in 1897 there were only three cases in all the London fever hospitals. Ireland has been its home for centuries, but filthy and crowded sections and the almshouses of large cities have at different times furnished seats for its lodgment. Quite a serious epidemic prevailed in New York City in 1881–2, and a milder one in 1892–93. Nicolle has transmitted typhus fever to monkeys by injecting them with blood of typhus-fever patients, while Anderson and Goldberg have done the same with typhus fever of Mexico.

Nicolle also proved that typhus fever in France could be transmitted from monkey to monkey by the common body louse. Ricketts and Wilder confirmed these facts in regard to Mexican typhus (tabardillo) and also that this disease can be transmitted from man to monkey by means of the louse (Pedicullus Vestimente). These writers have also shown that the flea and the bedbug are not concerned in the spread of typhus. Anderson and Goldberg believe the disease can also be carried by the head louse.

Typhus fever is therefore not contagious, but great care should be taken to destroy all the clothing from an infected individual as well as all vermin on his body. Also he must be protected from vermin in order that the latter may not be infected. Nurses and others in constant attendance upon typhus patients are more liable to be attacked than those who, like the physician, merely visit them daily, although perhaps no disease in the past has included among its victims so many medical men.

Morbid Anatomy.—As to the morbid anatomy of this disease, there is really nothing distinctive. Rigor mortis is apt to be delayed. The petechial eruption remains after death, and gangrenous bed-sores may be found on the body. The most constant lesion is very *moderate enlargement of the*

[1] For a summary of the observations thus far made on the "Micro-organisms in Typhus Fever," see a paper with this title by J. B. Byron and Egbert Le Fevre, in vol. ii., "Researches of the Loomis Laboratory," New York, 1892, p. 130.

spleen, and in this enlargement the *liver* and *kidneys* may share, and their cells be the seat of cloudy swelling due to fever heat. Indeed, all the tissues, including the heart muscle, may be granular from this cause. The splenic enlargement is mainly due to vascular engorgement, but there may also be some hyperplasia of lymph-cells. The lymph-follicles of the intestine may be enlarged from the same cause, but there is no ulceration of these or of Peyer's patches. The blood is dark and liquid. Hypostatic congestion of the lungs is very frequently found; likewise bronchial catarrh. The permanence of the eruption after death is in strong contrast with that of typhoid fever, which disappears after death.

Symptoms.—The *period of incubation* is usually about 12 days. It may be less. There is seldom any prodrome, the *invasion* being *sudden*, an-

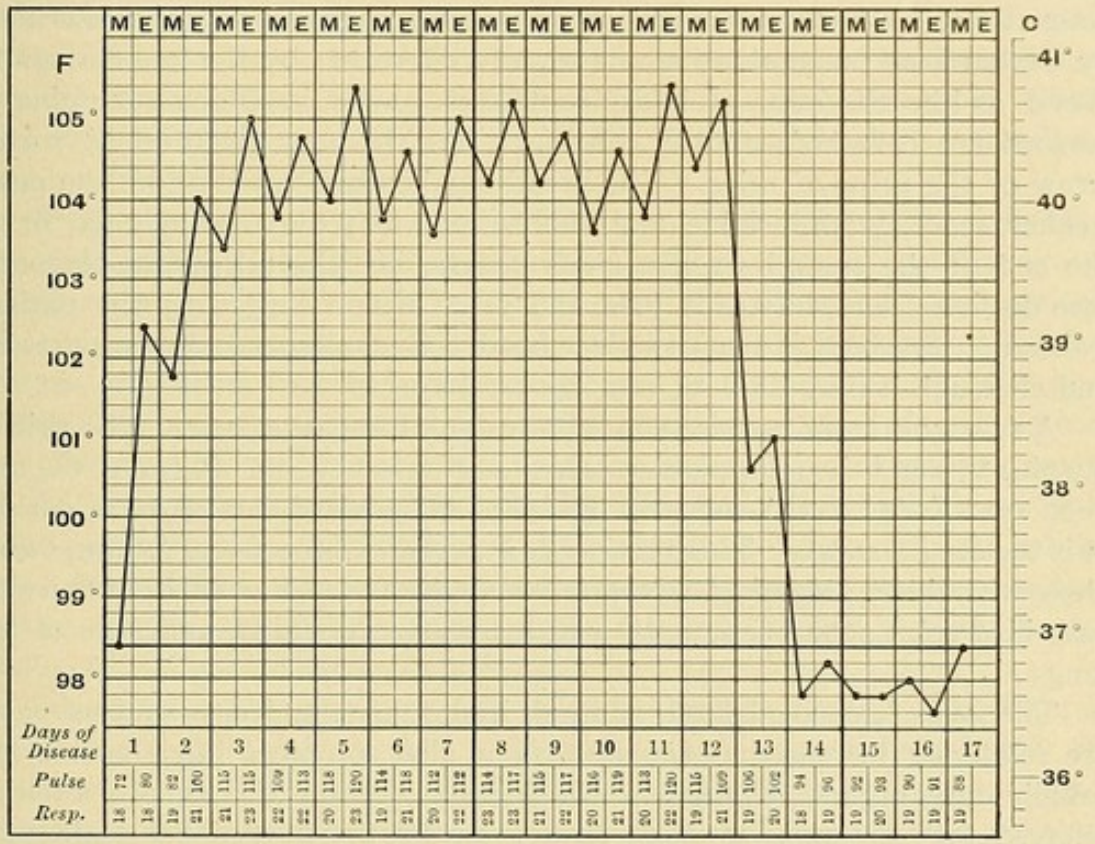


FIG. 6.—Temperature in Typhus Fever ("Pepper's American Text-book of Medicine").

nounced by a *chill* or *chills* followed by headache and great *muscular pain*, especially in the back, and by *high fever*, the temperature rapidly rising to 103°, 104°, 105°, and 106° F. (39.4°, 40°, 40.5°, 41.1° C.) without any of the tidal-wave rise characteristic of typhoid fever. The *pulse* is at first full and strong, but soon weakens and becomes frequent—120 and more. There is extreme *debility*. *Almost* characteristic are the *red, congested conjunctivæ*, the *dusky face, dull expression*, and *low, muttering delirium*, which contrasts strongly with the sometimes active delirium of typhoid. The tongue is early coated and becomes rapidly dry. The bowels are constipated.

On the third to the fifth day the *eruption* presents itself; it is of *two kinds*, the *petechial* and the *mottled*. The *petechial*, or more characteristic, is at first not unlike that of typhoid fever, but is darker in hue and disappears less readily on pressure; a little later it is barely influenced, and still later

does not respond at all to pressure. It has become hemorrhagic There may be spots exhibiting each one of these stages. This eruption is also more scattered than that of typhoid fever, appearing all over the body, while that of typhoid is limited to the chest and belly. In addition to the petechial eruption there is also a peculiar *dark mottling* of the skin, an alternation of purple blotches with others of a light hue, generally capable of being influenced by pressure, but these blotches, too, may become blood extravasations.

With the *beginning of the second week all of the symptoms become more severe;* the tongue becomes dry, fissured, and leathery; sordes collect on the teeth; stupor deepens, there are subsultus and nystagmus, *coma vigil*—the patient is unconscious, but the eyes are wide open—and picking at the bedclothes. At this time, too, the peculiar disagreeable odor said to be characteristic of typhus fever makes its appearance. It is variously described: by Gerhard, as pungent, ammoniacal, and offensive; by the late George B. Wood, as like the odor of badly ventilated rooms, in which a number of persons are collected; and by others, as like the odor given off by rotten straw or the urine of mice. The breathing becomes more rapid, the pulse weaker, scarcely appreciable, and the patient may die of adynamia; or at the end of the second week a *crisis* occurs, he falls asleep, the temperature declines as rapidly as it rose, and often after a long sleep the patient wakes up refreshed and with a clear head. Convalescence now progresses, and although it may be slow, relapses rarely occur.

A few symptoms require special allusion: First, the *fever*. The skin is burning hot and the temperature occasionally rises to 106° F. (41.1° C.) and even 108° F. (42.2° C.) and 109° F. (42.7° C.) toward a fatal termination. It is the *calor mordax*. There is always *hypostatic congestion of the lungs* and, along with this, a great deal of *bronchial catarrh* and *cough*. Such catarrh may pass into a broncho-pneumonia, which may terminate in gangrene of the lungs.

The *urine* is concentrated, as in all high fevers, and urea and uric acid are relatively increased. *Albuminuria* is also common, but there is not usually any organic change in the kidney beyond the cloudy swelling referred to. *Retention of urine* on account of the mental hebetude may occur, and should be guarded against by frequent examination and catheterization. *Bed-sores* are common, and there may even be gangrene of the extremities.

Instances of the ambulatory form of typhus fever are much more rare than of typhoid, but they are occasionally met.

Diagnosis.—How does typhus fever differ from *typhoid* fever? We have referred to the differences in the eruption in the two diseases. But the temperature of typhus fever is quite as characteristic as that of typhoid fever. In the latter disease we have the peculiar tidal-wave course described. In typhus fever, in the first place, the average maximum temperature is higher; for, while a temperature of 106° F. (42.1° C.) is not uncommon in typhus fever, 105° F. (40.5° C.) in typhoid is quite high. The temperature in typhus quickly reaches the maximum, usually from the third to the fifth day, continues with light remissions until the 12th or 14th, then there is a sudden decline. The ascent is steady and con-

tinuous, and only marked by slight morning remissions, while in typhoid fever the morning remissions are decided. The pulse, during the first three days, is usually about 100; after that it becomes more frequent and feeble, running up to 120 or higher, until the drop in temperature, when there is a corresponding fall in the rate of the pulse. It is seldom dicrotic, as in typhoid fever. Typhus fever more frequently begins with a *chill* than does typhoid; the important symptoms, including the eruption, appear earlier. In isolated cases, however, there may be difficulty in diagnosis.

Malignant measles, hemorrhagic smallpox, cerebrospinal fever, bubonic plague and Rocky Mountain spotted fever are diseases for which typhus fever may be mistaken. The eruption of malignant measles is not unlike that of typhus fever, and it appears first in the face. The extreme adynamia and the typhoid symptoms are very similar. There is bronchitis in both, but the coryza and acute nasal catarrh are not found in typhus, while concurrent with the case of malignant measles are others of a milder and more typical nature. The latter fact also aids the diagnosis in variola, where, too, in the malignant form the hemorraghic tendency is more marked and occurs early in the disease.

Meningitis has often been mistaken for typhus, and in the early stage the suddenness of onset, the eruption, and the nervous symptoms are all calculated to mislead. Here the spinal puncture will make the diagnosis. *Bubonic plague* has been confounded with typhus, it however resembling typhus only in its fatality. Bubonic plague is characterized by the same suddenness of onset, the chill, high fever, and prostration, as is typhus fever; but the eruption appears earlier, becomes carbuncular, while the course of the disease is much shorter. Rocky Mountain fever might easily be mistaken for typhus, the symptoms of the two conditions closely resembling each other. The non-occurrence of typhus, however, in the northern states and the rare occurrence of spotted fever in the region where typhus abounds, is a good diagnostic point.

Prognosis.—The mortality of epidemic typhus is high, but different epidemics vary in this respect. There is practically no mortality in the mild form of Brill's disease. During the epidemic at the Camden County Almshouse (1880–81) referred to, 103 of the officers and inmates were attacked. Of this number 23 died, giving a mortality of a little over 22 per cent. We might add that of the officers of the institution, seven, including an attending physician, the steward, the matron, the assistant matron, and two nurses, together with the builder of a new hospital building, were attacked, and all died. In some epidemics the mortality is even greater, reaching 50 per cent., but it commonly ranges from 12 to 20 per cent. The disease attacks either sex at any age. One of the modes of death is by acute fatty degeneration of the heart, and the peculiar dusky complexion sometimes seen may be due to the inability of a weak fatty heart to propel the blood through the capillaries. Sudden death is not unusual. It is more than likely that with the improved nursing and hygiene of the present day the mortality of typhus would be less.

Treatment.—Prophylaxis is highly important. The knowledge that the body louse carries the disease from patient to healthy individuals, makes personal cleanliness and the destruction by fire of all clothing of the

patient, imperative. Likewise the patient may be bathed in coal oil which will destroy the louse. Notwithstanding the fact that the louse adheres to the clothing and not to the body of the patient, it is wise to take these precautions. Whenever possible, typhus fever should be treated in the open air (in tents), as the safety of attendants as well as recovery of patients is favored thereby. No patient should be admitted to a ward or tent for treatment until divested of all his clothing, and given a scrub and bathed in coal oil. Hydrotherapy is as serviceable in typhus as in typhoid, but it is absolutely necessary that free stimulation should be associated with any treatment. We know that the greatest danger lies in the asthenia, which can be met only by stimulants. Aromatic ammonia, strychnin, caffein and camphor are here of the greatest value. Strychnin should be given in 0.002 (1/30 grain) doses every three hours, hypodermically if necessary; ammonia in teaspoonful doses; caffein in 0.060 to 0.120 (1 to 2 grains) doses every two or three hours hypodermically; alcohol should be given as in typhoid, remembering that it is indirectly a stimulant in that it may be used as food, 8 to 16 c.c., 2 to 4 drams, may be given as necessary. If the pulse becomes full and strong, the alcohol is doing good, otherwise it is useless. Quinin is also strongly indicated, as are digitalis and strychnin as heart strengtheners. When the temperature becomes high, if the cold bath be not used, sponging of the body in the way described under typhoid fever may be substituted. The same objection exists to phenacetin and antifebrin as in typhoid; that is, they dare not be relied upon as a means of reducing temperature. Other symptoms should be treated as they arise. Specific antiseptic treatment has proved to be without peculiar advantage.

After the crisis, which, as has been said, is strikingly well marked in this disease, it is simply necessary to treat symptoms as they arise. The accompanying bronchitis is treated, if it requires treatment, like any other bronchitis, but the ammonium salts are especially indicated on account of their stimulating qualities, while the aromatic spirit of ammonia is an especially convenient preparation for these purposes. Camphorated oil may be given hypodermically to tide over emergencies.

The patient should be nourished as in typhoid fever—by nutritious liquids, including milk, milk punches, egg-nogg, and nutritious broths, and all soft foods the patient can swallow.

RELAPSING FEVER.

SYNONYMS.—*Febris recurrens; Famine Fever; Seven-day Fever.*

Definition.—Relapsing fever is an acute infectious disease, characterized by two or more febrile relapses separated by periods of total remission and caused by the inoculation and multiplication of the spirochæta of Obermeier.

Etiology.—The specific cause of relapsing fever known in this country is the *spirochæta Obermeieri*. It was first discovered by Obermeier in the blood of victims, it is known by his name. It is a narrow spiral about 0.025 to 0.05 mm. (1/1000 to 1/500 inch) in length—that is, its length is three to

six times the width of a red blood-disc. *It is found floating among the blood-discs during the fever.* In the intervals the organism is not found, but small, glistening spherules, said to be its spores, take its place. Confirmation of the infectious nature of the disease is found in the fact that it has been communicated to monkeys by inoculation of blood. Transmission from one individual to another is undoubtedly favored by overcrowding, by filth, and by destitution. Yet the disease is not confined to the poorly fed. This was especially proved in the Philadelphia epidemic of 1869, when a considerable number of fairly well-to-do persons were affected, although they always resided in crowded districts. Neither age, sex, nationality, nor season is a factor in its causation. Bedbugs are unquestionably carriers of the disease, the spirochæta having been repeatedly demonstrated within the bodies of these pests infesting the bedding and beds upon which relapsing fever patients have slept.

There are relapsing fevers in different countries of much the same clinical character in which the organism is of a different species from that of the Obermeier organism. In the tick fever of Africa (which see) the disease is spread by ticks and repetition has a high mortality.

F. G. Novy[1] who has made the latest study of relapsing fever has concluded that a "plurality" of relapsing fevers is very probable. This conclusion was reached after a study of the blood from two cases of the disease, from different sources, one from New York, and one from Bombay, and is based on certain anatomical differences in the spirillum; that of Bombay being more like that of tick fever from which it, however, differs. The spirochæta of tick fever (the *spirochæta Dultoni*) is composed of cells 16 microns long while the s. Obermeieri is but eight microns long, and the number of turns in each cell is about the same. Moreover, the width of the spiral of tick fever is two or three times that of s. Obermeieri, being two to 2.7 microns as compared with one micron. Clinically, however, the diseases are very similar.

Morbid Anatomy.—There is no essential morbid anatomy, and such as is found corresponds with that of typhus. Most conspicuous is *enlargement of the spleen.*

Symptoms.—The *period of incubation* varies greatly, so that it is put down at from two to 14 days. According to Murchison, there may actually be no interval between exposure and the invasion. The latter is sudden by a *chill*, *fever*, *intense pain* in the back and limbs, with *dizziness*. This abrupt invasion is a distinctive feature, and in perhaps none of the contagious diseases is it, as a rule, so marked. Exceptionally only is there a short period of *malaise* with *loss of appetite*. On invasion the temperature rises rapidly and quickly reaches 104° F. (40° C.). The patient cannot remain on his feet, and promptly takes to his bed, feeling very sick, rather than profoundly weak. There may be *nausea* and *vomiting* and even *convulsions* in the young; the pulse rises rapidly, more rapidly than in typhus, reaching 140 on the second day, and later 150 and 160. The patient may be *delirious*, but the typhoid symptoms are not usually so profound as in typhus, and the tongue remains moist. *Jaundice* appears in a certain number of cases on

[1] "Relapsing Fever and Spirochetes," F. G. Novy and R. E. Knapp "Trans. Assoc. Amer. Physicians," vol. xxi., 1906.

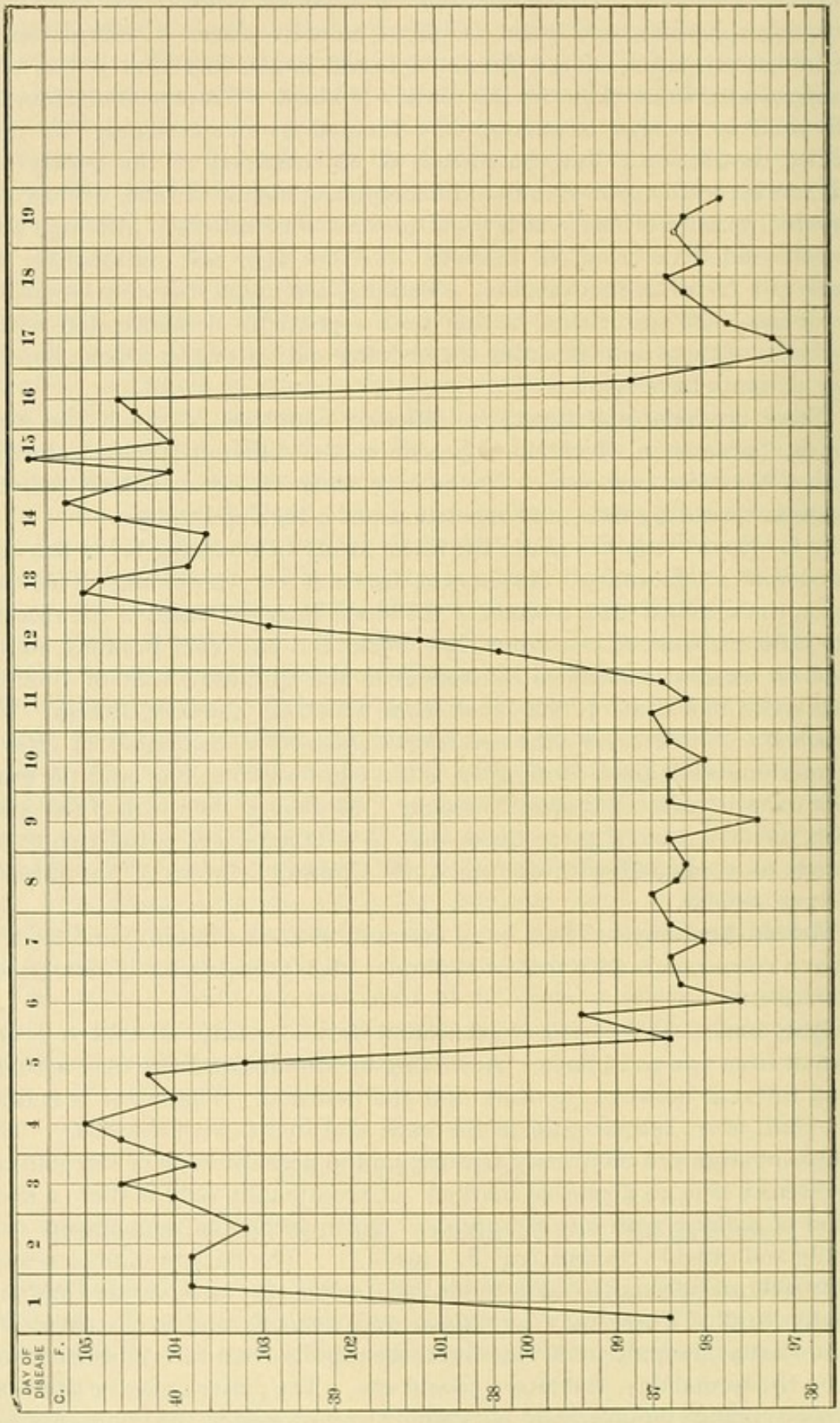


FIG. 6.—Temperature Chart of Relapsing Fever Showing Relapses.—(From Murchison on "Fevers.")

the third or fourth day, usually in one out of every 12 cases, occasionally as often as one in every four or five. The temperature during the paroxysm fluctuates slightly, being higher in the evening. *Sweating* and *sudamina* are often present, and occasionally *petechiæ*, but there is no characteristic eruption. Rarely, Murchison says in eight out of 600 cases, a roseolar rash appears, or there may be a *mottling* like that of typhus, which, however, always disappears on pressure, and disappears entirely in three or four days—differing in these respects from the similar eruption of typhus. *Herpes* may be present. There is occasionally *abdominal tenderness* in the epigastric or iliac region, and the *enlarged spleen* may be easily detected, but there are no active intestinal symptoms. The *liver* may be also *slightly enlarged*, extending lower than in health.

The *spirochæte* is to be found in the blood and should always be looked for. It may readily be detected with a power of 500 diameters without any special preparation of the blood, care being simply taken to secure a thin film.

Crisis.—If the invasion of relapsing fever be sudden, its termination is no less so. It is by crisis, beginning usually with sweating. After five or six days of unabated fever sweating sets in, which soon becomes profuse, the temperature falls rapidly to normal or even subnormal, the various discomforts fade away, and in the course of a few hours the patient is apparently well. Rarely, the crisis may be ushered in by a diarrhea, an epistaxis, or the appearance of menstruation.

The crisis does not always take place at the same stage of the disease. It may occur as early as the third day or not until the tenth, or even the fifteenth but most commonly on the seventh. While the crisis is ordinarily followed by some relaxation and faintness, there soon ensues a rapid recovery of natural and healthful feeling. Occasionally, however, the depression is greater and a sensation as of collapse occurs, especially in delicate or elderly persons.

Relapses.—Again, in a week from the crisis, generally on the fourteenth day from the primary chill, another occurs, or a series of them, with fever, and the paroxysm repeats itself, to be again succeeded by a crisis at a somewhat shorter interval. There may be a third or even a fourth and fifth paroxysm; more commonly they are limited to two or at most three. Each succeeding attack is shorter than the previous one. Occasionally there is no relapse, the disease terminating with the first crisis. Convalescence, usually rapid, is sometimes prolonged, and the *duration of the entire illness* may be put down at from 18 to 90 *days*, and the patient rarely returns to work within six weeks. One attack does not secure immunity from another.

Complications.—Among the complications may be mentioned bronchitis, pneumonia, nephritis and hematuria. The spleen may enlarge until it ruptures. It may attain a weight of four and one-half pounds (10 kilos), and may be the seat of infarcts. Albuminuria occurs as in other fevers characterized by high temperatures. Pregnant women usually abort in the relapse, and the child, if not still-born, survives but a few hours. Post-febrile paralysis may occur, and troublesome ophthalmia succeeds in some epidemics.

Diagnosis.—In its early stages relapsing fever is not unlike *typhus*. In

suddenness of onset, rapid rise of temperature, habitat, and subjects, the resemblance is close. The readiness with which a patient takes his bed is characteristic of each, but in relapsing fever the adynamia is not so great as in typhus, and it is rather because of a dizziness that he cannot keep about. Examination of the blood will show the spirochæte in relapsing fever. The crisis cuts short all doubt on this point of confusion with typhus. In the intense muscular pains, especially in the back, relapsing fever resembles *smallpox*, but the eruption in the latter disease sets doubt at rest.

Malaria fever may be suggested by the relapse, but the presence of an organism in the blood of each of these affections, widely different in appearance, permits the settlement of such confusion by the microscope. The prevalence of an epidemic is, of course, of great assistance in the diagnosis between relapsing fever and any of the diseases with which it may be confounded.

A diagnosis can certainly be made during the febrile paroxysm by finding the spirochæte in the peripheral blood. During the apyrexia the organisms are not found in the blood. According to Lowenthal a sero-diagnosis may be made during this period. The serum of a relapsing-fever case will immobilize the spirochæte of Obermeier and thus a diagnosis may be made.

Prognosis.—The prognosis of relapsing fever is not unfavorable. The higher mortality reported in some of the earlier epidemics in Great Britain and Ireland was doubtless due to an admixture of typhus. An average for several years in a number of cities in Great Britain and Ireland, according to Murchison, has been 4.3 per cent.; in the epidemic at Bombay in 1877–78, Vandyke Carter estimated the mortality at 18.02 per cent.; while in the Philadelphia epidemic the studies of William Pepper, 2d, and Edward Rhoads found it 14 per cent. It is sure that in private practice during this epidemic the mortality was not so great. There are some accidents, which have been already alluded to, that are responsible for a few deaths. Thus, the spleen has ruptured from extreme congestion. Pneumonia sometimes causes a fatal termination. It has been said that the crisis sometimes terminates in collapse with its characteristic clammy coldness, pulselessness, unconsciousness, and fatal end. A fatal nephritis occasionally complicates the disease, death being preceded by uræmic convulsions. Certain cases associated with jaundice, called by Griesinger "bilious typhoid," are often fatal. Some striking cases of this kind were noted by Pepper at the Philadelphia Hospital in the epidemic of 1869–70.

Treatment.—Prophylaxis here plays a great part in the treatment. All bedbugs should be destroyed in or about the rooms of the patient with this disease. The patient being freed of all bedbugs should at once be transported to a room known to be free from infection by bedbugs. The febrile paroxysm demands much the same treatment as in typhus—careful nursing, sponging or cool bathing, nutritious, easily assimilable food, and stimulation, although the latter is less important than in typhus. According to Meltzer[1] salvarsan can completely destroy the spirillum of relapsing fever in the animal body. Hence this drug or the neo-salvarsan should be tried in every case of relapsing fever. No drug has the power to prevent the recurrence of the relapse, although quinin is indicated, and, as in other adynamic

[1] "Transactions, Assoc. Amer. Phys.," vol. xxvi., 1911, p. 300.

fevers, is useful only as a tonic. It is reasonable to expect that acetylsalicylic acid, phenacetin, antifebrin, or antipyrin will relieve the muscular pains. Should they not suffice, morphin, hypodermically, can be relied upon.

The studies of Novy go to show that the serum treatment will probably be ultimately successful.

MALTA FEVER.

SYNONYMS.—*Mediterranean Fever; Neapolitan Fever; Rock Fever; Undulant Fever.*

Definition.—An anomalous fever, characterized by irregular remissions and relapses, copious sweats and rheumatoid pains, and caused by a bacillus known as *micrococcus melitensis*.

Distribution.—The various names of Malta fever indicate its distribution on the Mediterranean littoral, outside of which it has been thought infrequent; but in 1898 J. J. Kinyoun[1] suggested its presence on the Southern Atlantic coast of America and the islands of the Gulf of Mexico, a suggestion confirmed by the report of J. H. Musser and Joseph Sailer of a case originating in Cuba.[2] It is also reported from Africa and South America.

Etiology.—The *micrococcus melitensis*, the cause of this peculiar fever, has been studied by Bruce, whose results have been confirmed by Hughes. Its morphological and biological features have been accurately studied by H. E. Durham. It is found in large numbers in the spleen, but has not been isolated from the blood. The disease is spread through the milk of goats. It is not definitely known how the micrococcus leaves the body, but the urine of about 10 per cent. of patients contains the organism; the feces also in a certain number. No intermediary host save the goat is known, though Bruce claims that mosquitoes may be the conveying medium. Pure cultures have been obtained, the disease has been reproduced in monkeys, and the micrococcus isolated from the infected animal. A. E. Wright and F. Smith have shown that the blood of Malta fever patients reacts with pure cultures of the *micrococcus melitensis*. This would seem to settle its independent nature. The disease attacks mostly the young.

Morbid Anatomy.—Our knowledge of the morbid anatomy of Malta fever is not definite. Thus, Bruce says no characteristic lesion of Malta fever is found.

Symptoms.—There is usually a *period of incubation* of from six to ten days. The onset is gradual, with *headache*, *sleeplessness*, and *thirst*, *loss of appetite*, *without chilliness* or *high fever* at first. There is no diarrhea; spots are not found. These symptoms, more or less pronounced, last from three to four weeks, when the first remission sets in, simulating convalescence. It lasts a few days only, when the first relapse appears, this time with *rigors*, *high fever*, and often *diarrhea*, and the symptoms of the first attack intensified. This relapse lasts for from five to six weeks, to be followed by another remission of from ten days to two weeks. Then follows the second relapse, when recur the symptoms of the first relapse, to which

[1] "Gaceta de Caracas," July 15, 1898, and "Philadelphia Med. Jour." January 14, 1899, p. 63.
[2] "Philadelphia Med. Jour." December 31, 1898.

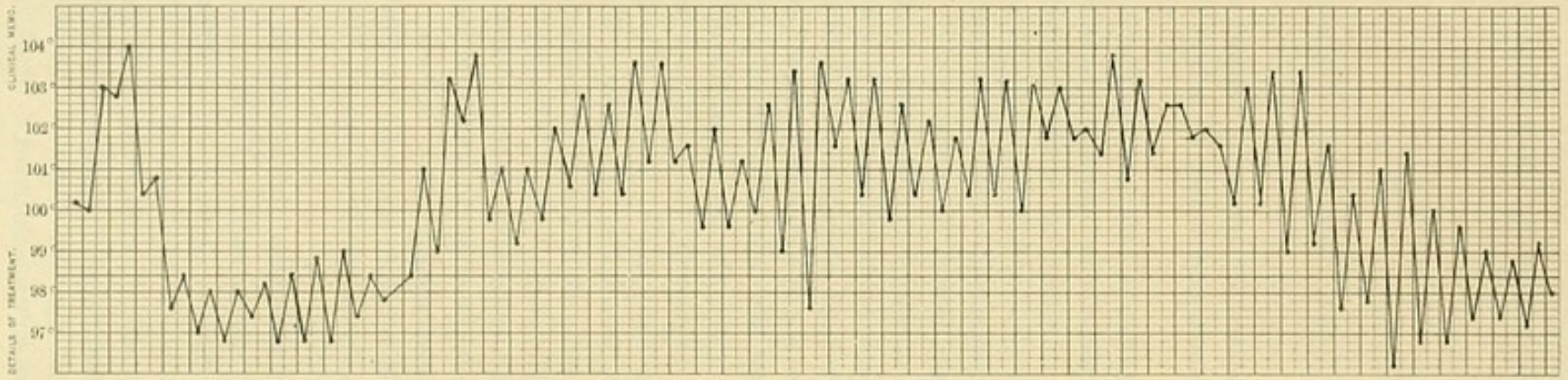


CHART OF MALTA FEVER SHOWING MORNING AND EVENING TEMPERATURES.

TWO DISTINCT RELAPSES ARE SHOWN.

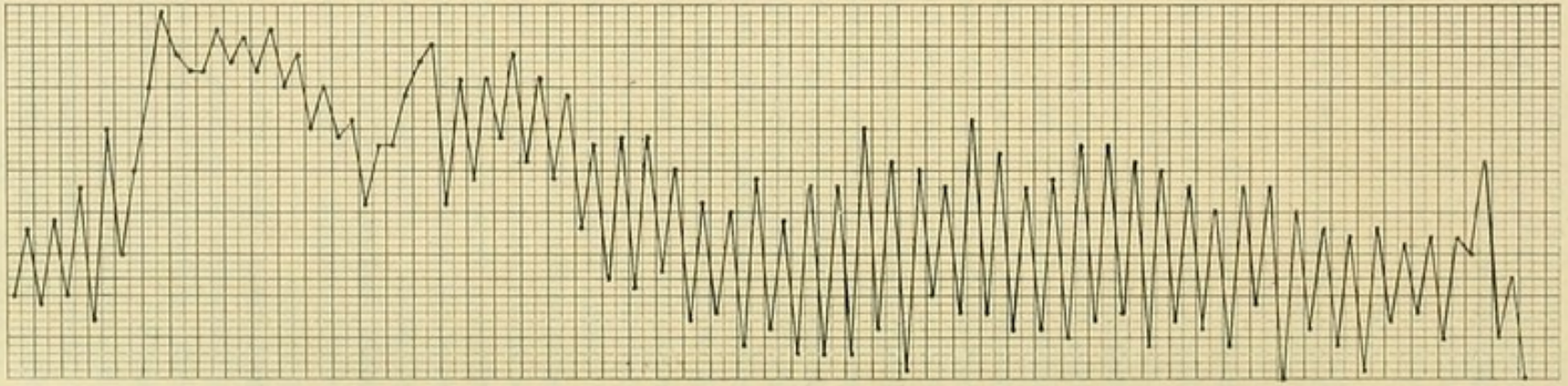


FIG. 7.—(After Musser and Sailer.)

are superadded great *debility, night-sweats, pain* in the larger joints, including hips, knees, and ankles, and in the testicles—one or both—lasting three or four weeks. Then follows a third remission, which may last for a month or six weeks. Then a third relapse of shorter duration, adding to the other symptoms a heavily *coated tongue*, a high temperature, 105° F. (40.5° C.) and above in the evening, but normal in the morning, the night-sweats, and especially the joint pains, being markedly severe. All the joints now seem to be involved, and motion is an agony. The fibrous tissues are also often involved in this relapse, especially the *tendo Achillis* and fibrous structures about the ankle; also the lumbar aponeuroses and sheaths of the nerves from the sacral plexus.

Diagnosis.—The rarity of the disease and the peculiarity of its symptoms may cause it to be overlooked for some time. It may at first be mistaken for typhoid fever, but there are no rose spots and no diarrhea, the course is much longer. The serum reaction is, however, characteristic, cultures of the specific bacillus responding to the serum of the blood of the disease as does the typhoid bacillus in typhoid fever.

Prognosis.—This is generally favorable, not more than two per cent. perishing.

Treatment.—Prophylaxis is the most important part of the treatment. The milk of goats in infected regions should not be used or when its use is imperative should be boiled. The actual treatment is symptomatic, being directed to the relief of the symptoms and the support of the patient against the exhaustive effect of the disease. A case seems to have been successfully treated with Malta fever antitoxin by Fitzgerald and Ewart.[1]

THE MALARIAL FEVERS.

Synonyms.—*Ague; Fever and Ague; Chills and Fever; Marsh Fever; Swamp Fever; Paludal Fever; Miasmatic Fever; Intermittent, Remittent, and Pernicious Remittent Fever; Bilious Fever; Estivo-autumnal Fever.*

Definition.—Malarial fever is an infectious disease, of intermittent or remittent type, due to an organism known as the *plasmodium* or *hæmocytazoa* of malaria.

A chronic cachectic condition due to the same cause is known as "chronic malaria" or "malarial cachexia." Chronic malaria has really a more definite morbid anatomy than the acute malarial fevers. The term "malaria"—meaning, in the Italian, bad air—was originally applied to the supposed specific cause of the fever, but it is also used to express the consequences of such cause.

Varieties of Malarial Fever.—The clinical varieties of malarial fevers are *intermittent* (tertian or quartan) or *remittent* (estivo-autumnal). The former is characterized by paroxysms of fever, between which there are total intermissions. In the remittent form there are remissions or abatements in the fever, but not intermissions. The remittent fevers exhibit much less regularity than the intermittent fevers, even in their remissions, and in consequence of their prevalence in the later summer and fall have among

[1] "The Lancet," April 15, 1899.

other irregular types been included under the head *estivo-autumnal.* The latter term embraces also all the malignant types, which are rarely seen in the spring months.

The paroxysms of fever may come on daily at the same hour, when they are called *quotidian;* they may occur every other day, when they are known at *tertian;* or they may occur every third day—that is, skip two days—when they are called *quartan.* More rarely occur quintan, sextan, septan, and octan fevers, with intervals of four, five, six, and seven days, respectively. It will be noted that in naming these periods the day of the paroxysm and that of the following paroxysm are both counted. The "*double tertian*" is a fever in which paroxysms occur each day but at different hours, the hours on alternate days corresponding with each other. In these cases the alternate paroxysms may also be of different intensities. The quotidian is really a double tertian, the paroxysms occurring at the same hour or nearly the same hour each day. In like manner there may be double quartans and even double quotidians.

Although the paroxysms in true intermittent fever commonly occur at the same hour, they may happen a little earlier each day, when they are called "anticipating" or they may happen a little later when they are called "retarding." The former is apt to occur when the disease is becoming more severe, the latter when it is abating. The paroxysm varies in length in the different varieties. In the quotidian form it lasts from ten to 12 hours, in the tertian six to eight hours, and in the quartan four to six hours.

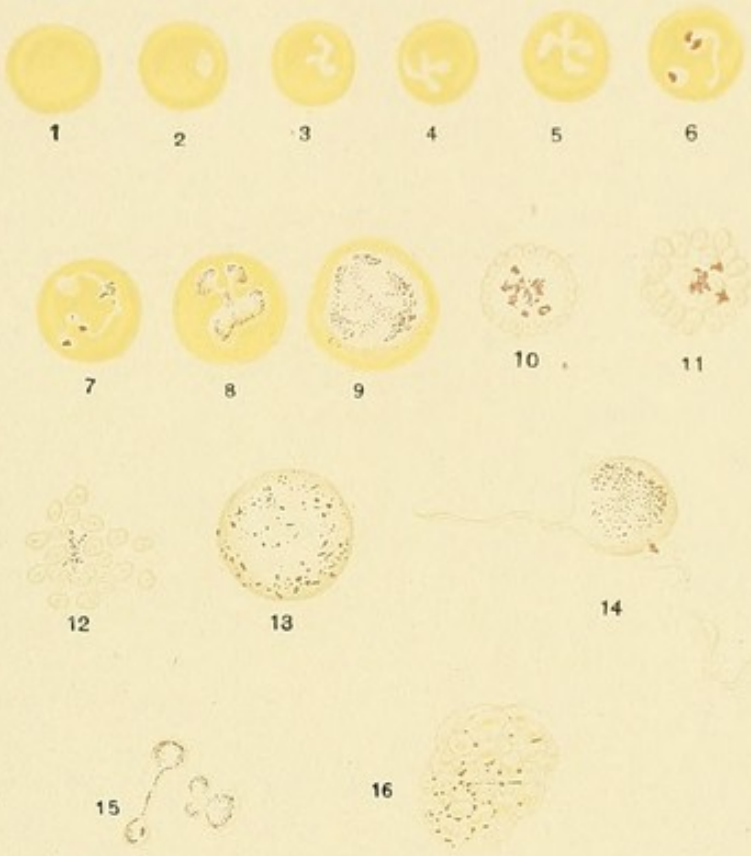
Malarial cachexia referred to in the definition, also known as chronic malaria, will be fully considered later.

Etiology.—The malarial fevers are caused by a protozoön known as the *plasmodium malariæ*, or *hæmacytozoa malariæ.*

Hæmacytozoa or parasites of the red corpuscles are not confined to man but are met in the blood of birds, fish, frogs and such mammals as monkeys, bats and cattle as well. In birds and frogs they are especially numerous and apparently harmless unless very abundant.

To Study the Malarial Organism in Man.—The malarial organism is best studied from fresh preparations as follows:

A drop of blood is taken from the finger or lobe of the ear during the chill, or an hour or two previously, while the temperature is gradually rising. It should be placed on a perfectly clean cover glass, which should be allowed to fall gently on a glass slide, without pressure, and carefully examined with an oil immersion lens. Careful searching will show certain red corpuscles (Plate I, Figs. 2, 3, 4, 5), containing minute hyaline bodies with an ameboid movement. Later the ameboid bodies are seen to contain pigmented granules, having a Brownian movement. (Plate I, Figs. 6, 7, 8, 9). These bodies stain with a Wright's stain. Later the plasmodium fills the entire body of the corpuscle, its ameboid movements cease, and the granules become more or less stationary. The pigment now tends to mass itself into clumps near the center, (Plate II, Fig. 9). Very soon this larger complete pigmented body begins to segment, making a Mulbery mass (Plate I, Fig. 11) of from twelve to twenty segments, with the pigment granules in the center. At the same time a body similar to the one within the corpuscle may be seen upon the slide outside of the corpuscle.



THE TERTIAN PARASITE.

1. *Normal erythrocyte.*
2, 3, 4, 5. *Intracellular hyaline forms.*
6, 7. *Young pigmented intracellular forms.* In 6 two distinct parasites inhabit the erythrocyte, the larger one being actively ameboid, as evidenced by the long tentacular process trailing from the main body of the organism. This ameboid tendency is still better illustrated in 7, by the ribbon-like design formed by the parasite. Note the delicacy of the pigment granules, and their tendency toward peripheral arrangement in 6, 7, and 8.
8. *Later developmental stage of 7.* In 7, 8, and 9 enlargement and pallor of the infected erythrocyte become conspicuous.
9. *Mature intracellular pigmented parasite.*
10, 11, 12. *Segmenting forms.* In 10 is shown the early stage of sporulation—the development of radial striations and peripheral indentations coincidentally with the swarming of the pigment toward the center of the parasite. The completion of this process is illustrated by 11 and 12.
13. *Large swollen extracellular form.* Note the coarse fused blocks of pigment. (Compare size with that of normal erythrocyte, 1.)
14. *Flagellate form.*
15. *Shrunken and fragmenting extracellular forms.*
16. *Vacuolation of an extracellular form.*

NOTE.—The original water-color drawings were made from fresh blood specimens, a Leitz $\frac{1}{12}$-inch oil-immersion objective and 4 ocular, with a Zeiss camera-lucida, being used.

(E. F. FABER, *fec.*)

(From *Da Costa's "Clinical Hematology."*)

These extracorpuscular forms frequently can be seen to develop flagellæ, which whip around on the slide, causing the corpuscles to be actively moved about. The same cycle of successive steps is kept up from paroxysm to paroxysm. The conclusion reached by all observers is that the large intracorpuscular body (Plate I, Fig. 9) is the mature parasite ready for sporulation, and the mulberry mass presents the sporules perfectly formed, which a few seconds later become free spherical spores. These attach themselves to the red disks, penetrate them, and grow at the expense of the hemoglobin, leaving the black granular residue as excrementitious substance, which is let loose in the blood at the time of sporulation. As a consequence of this, the presence of pigment in the blood and tissues is one of the most characteristic features of malaria.

The time required to attain the perfect growth, from the free sporule to the stage of sporulation, varies in the different varieties of malarial fever. During this period certain groups, perhaps numbering myriads of corpuscles, pass through the same stages, and the final sporulation of such a group of parasites is always followed by the malarial paroxysm. This is probably due to some toxic substance developed at the time of sporulation. Thus, with the typical tertian type, sporulation takes place every other day at the same hour, the quartan type every 72 hours. If, however, two groups ripen at different hours, we have the double forms, be it double tertian or double quartan. Or two groups of tertian parasites may mature on alternate days, causing a quotidian paroxysm, though at different hours. The typical quotidian is the result of maturation of two groups of tertian organisms at the same hour on alternate days making a daily paroxysm. A paroxysm may be expected at once in the tertian form, if radiating lines appear in the organism with concentration of pigment. The cycle of existence of the estivo-autumnal type has an undetermined duration, and probably varies from 24 to 48 hours. The irregular ripening of different groups would explain the irregularity of the estivo-autumnal forms of fever, which may begin as regular types. Three distinct forms of parasites are recognized, the tertian, the quartan, and the estivo-autumnal forms. The full-grown *parasite of tertian fever, plasmodium vivax*, is about as large as a normal red blood-corpuscle, beginning its cycle of development as a much smaller hyaline ameboid body, corresponding very closely with the spores of the rosette-body. It acquires rapidly at the expense of the surrounding blood-disc, fine, brown pigment granules which sharply outline its "ring" shape and subsequently fill it completely. The blood-disc itself becoming gradually decolorized, grows larger and more indistinct until it disappears. The granules exhibit active movement. In the sporule stage the segments number from 15 to 20, or even more. The parasite has a cycle of 48 hours, but the segmentation is often seen between the 20th and 24th hours.

The parasite of quartan fever, plasmodium malariæ, is very similar, but it is smaller; its ameboid movements are slower, and the pigment granules fewer, coarser, darker, more frequently arranged about the periphery, and less active in motion. The red corpuscle embracing it, instead of becoming larger and paler, shrinks about the parasite and assumes a deeper, greenish

hue. The sporulation segments are fewer, only from five to ten in number, and are arranged with great regularity about the central pigment (Plate II, Fig. 10). Its cycle is 72 hours.

The *parasite of the estivo-autumnal fever, plasmodium falciparum*, is still smaller, being, when fully developed, often less than half the size of a red blood-corpuscle, and the quantity of pigment is much smaller. Only the early stages of its development, represented by small hyaline bodies, often with one or two pigment granules, are found in the peripheral circulation, the later stage being seen in the blood of internal organs, such as the spleen and bone-marrow. The scarcity of the pigment granules is characteristic. The corpuscles containing the parasite are also often shrunken, crenated, and brassy in color. After a week or more, larger, crescentic, ovoid and round bodies with central clumps of pigment make their appearance, and are characteristic of this form of fever. This is also a gametocyte or sexual form. The cycle of this parasite is also about 48 hours.

The large *extracorpuscular* body (F_1, F_2,) which presents the same pigmentation and other features of the intracorpuscular body, is the latter escaped from the corpuscle. It is the gametocyte or sexual form. It is found in preparations watched with the microscope for some minutes after being taken.

The *crescent-shaped* body (Plate III, Figs. 15, 16, 17, 18, 19) is a striking object. The crescent develops in the interior of the red corpuscle from the small hyaline forms, which gradually increase in size, lose their ameboid movement, and assume a crescentic shape, while pigment granules collect in a group at the center. The corpuscle itself gradually becomes decolorized and ultimately destroyed, though for some time a delicate line can be seen running between the horns of the crescent, a shell, as it were, of the corpuscle in which the parasite is developed. The crescentic and ovoid forms are incapable of sporulation, they are the gametocyte, corresponding to the round extracorpuscular body of the tertian and quartan variety; they do not segment.

The *flagellate body* (G_1, G_2) (*Micro gametocyte*) may also come into view on the slide some 15 or 20 minutes after the blood is mounted, but is never seen on slides "fixed" immediately after the blood is drawn. It develops from the full-grown tertian and quartan parasites and from the round bodies with central pigment in estivo-autumnal infections. It is a very interesting object, the tentacular prolongations lashing about the central mass and agitating the surrounding corpuscles in a seemingly violent manner, throwing the latter and its own melanin particles into a state of extreme commotion. Sometimes portions of these tentacles break loose and float away in the blood plasma.

The Parasite in the Mosquito.—Though the mosquito theory of malaria is by no means new, it was not until 1894 that Patrick Manson gave it definiteness by suggesting the mosquito might be the intermediate host for the extracorporeal forms of the parasite, of which the flagellate form is the first stage; and that the flagella, breaking off from the residual body, may penetrate the cells of some organ of the insect. He first claimed that the crescentic form of estivo-autumnal malaria and the tertian and quartan spherical forms from which develop flagella are the "extracorporeal sporu-



THE ESTIVO-AUTUMNAL PARASITE.

1. *Normal erythrocyte.*
2, 3. *Young hyaline ring-forms.*
4, 5, 6. *Intracellular hyaline forms.* In 4 the parasite appears as an irregularly shaped disc with a thinned-out central area. In 5 and 6 its ameboid properties are obvious.
7. *Young pigmented intracellular form.* Note the extreme delicacy and small number of the pigment granules. (Compare with 6, Plate VI, and with 3, Plate VII.)
8, 9. *Later developmental stages of 7.*
10, 11, 12. *Segmenting forms.*
13, 14. *Crescentic forms at early stages of their development.*
15, 16, 17, 18, 19. *Crescentic forms.* In 15 and 19 a distinct "bib" of the erythrocyte is visible. Vacuolation of a crescent is shown in 18, and polar arrangement of the pigment in 17.
20. *Oval form.*
21, 22. *Spherical forms.*
23. *Flagellate form.*
24. *Vacuolation and deformity of a spherical form.*
25. *Vacuolated leucocyte apparently enclosing a dwarfed and shrunken crescent.*
26. *Remains of a shrunken spherical form.*

(E. F. FABER, *fec.*)

(From *Da Costa's "Clinical Hematology."*)

lating homologues of the intracorporeal organism; that the flagellum is the extracorporeal homologue of the intracorporeal spore." Both types of sporulating plasmodium possess the same function—the propagation of the parasite—one in the human body; the other, outside of it. Surgeon-Major Ronald Ross, whose studies were stimulated by Manson, found the flagellate form in the stomach of mosquitoes that had fed on subjects suffering with estivo-autumnal fever whose blood contained large numbers of crescents, thus confirming Manson's observations. Again, Daniels, working in Calcutta under Ross's direction, was able to confirm all the latters observations. Angelo Celli, while admitting that Ross partly saw the first stages of development of the estivo-autumnal parasites in the body of a dapple-winged mosquito, holds that Grassi, Bastianelli, and Bignami have given us all the details of its development.[1] To whomever be the credit, and it is probably a divided one, it is admitted that the hæmocytoa of malaria in man as in birds has two life cycles; the one asexual in the blood of malarial subjects, the other sexual in the body of special mosquitoes.

The above observations are the confirmed results of many different observers, whence it follows that inoculation is the only mode by which the infection is carried from the mosquito to man and from man back to the mosquito. Nay, more, the very genus and species of mosquito responsible have been isolated. They are the genus *anopheles*, species *claviger*, and species *pictus* (dapple-winged mosquito of Ross), both conveying the estivo-autumnal form, while the former is the conveyer of tertian infection. The *A. quadrimaculatus* of this country has been shown to be identical with the *A. claviger*, the insect chiefly responsible for the spread of the disease on the continent of Europe. In addition to the *A. guadrimaculatus*, only two species of anopheles have been positively recognized in this country, viz., *A. punctipennis* and *A. crucians*. The mosquito genus *culex* is not a host for the parasite of malaria.

It does not follow that every anopheles is infected, as it may infest a region where malaria does not prevail or the locality may be too cold to permit the development of the organism, warmth being an essential condition.

The harmless *culex* is comparatively easily distinguished from the malaria-breeding *anopheles*, both of which are shown in the annexed figures from the description of L. O. Howard, Entomologist of the United States Department of Agriculture.

The *anopheles*, (Fig. 1, Plate 1) has wings which are more or less spotted, while *culex* (Fig. 2) has clear wings. The palpi or projections on either side of the beak in *anopheles* are very long, nearly as long as the beak; in *culex* they are very short. Moreover, when *culex* is resting on a wall it is more or less hump-backed, that is, the head and beak are not in the same plane with the body and wings, the body and wings being parallel with the wall. With the *anopheles* (Fig. 3 on the left) the head and beak are practically in the same plane with the body and the body itself is usually placed at an angle with the wall and especially when resting on a horizontal wall such as the ceiling of the room its body is at a marked angle with the surface, as con-

[1] Celli, "Malaria," 2d ed. Translated by John Joseph Eyre, London, 1900.

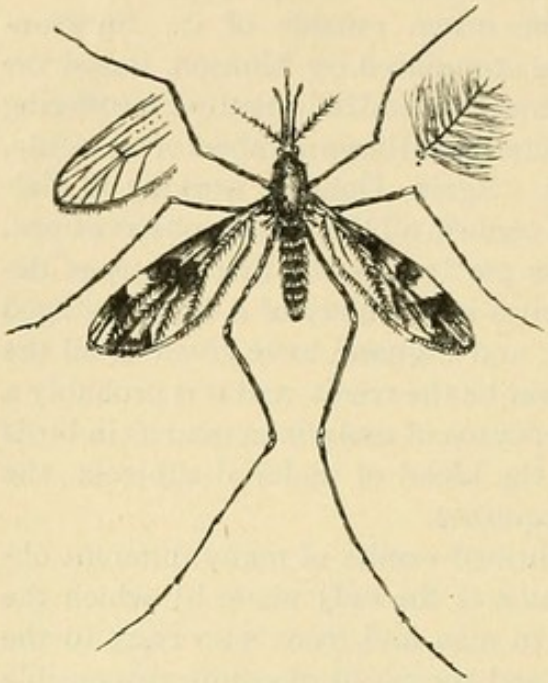


FIG. 8.—*Anopheles punctipennis*—female, with male antenna at left, and wing-top showing venation at left, enlarged.

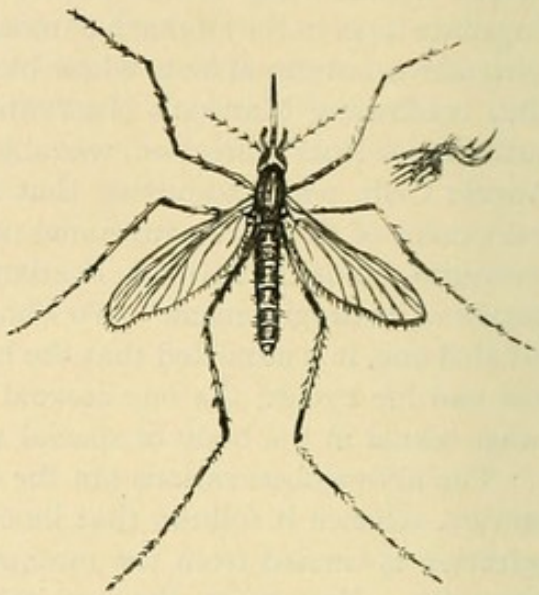


FIG. 9.—*Culex tæniorhynchus*—female, showing the short palpi which distinguish *culex* from *anopheles;* toothed front tarsal claw at right, enlarged.

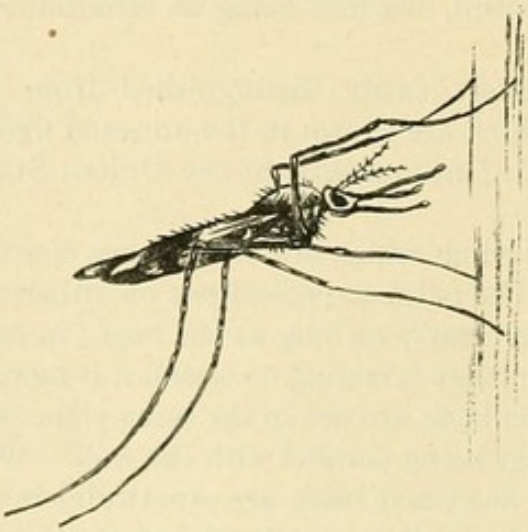


FIG. 10.—Resting position of *anopheles*, enlarged.

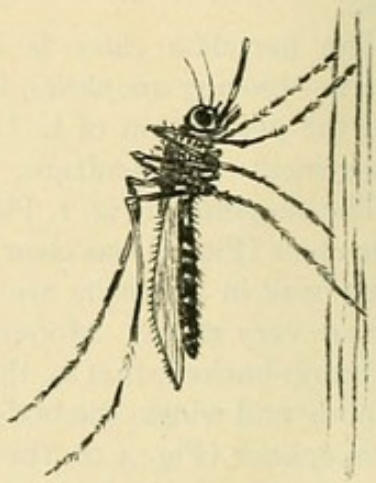


FIG. 11.—Resting position of *culex*, enlarged.

Made up of drawings from Dr. L. O. Howard's "Notes on the Mosquitoes of the United States." Government Printing Office, Washington, 1900.

trasted with *culex* (Fig. 4). This difference is well shown in the appended drawings from Howard's pamphlet.[1]

The studies of Ross, Grassi, Bastianelli, Bignami, Stephens, Christophers and Daniels have determined the following stages in the evolution of the sporozoön in the body of the mosquito. When the anopheles has "bitten" the malarial subject and drawn into its stomach the blood containing sex-ripe forms (gametocytes), the male elements or microgametocytes send out their flagella (microgametes) which penetrate and fecundate the female forms or macrogametes. These fertilized bodies or zygotes known also as oöcysts and sporoblasts, migrate as far as the muscular coat of the mosquito's stomach and begin there a cycle of development. After 40 hours appear small round refracting bodies which contain pigment granules like those contained in the malarial parasite. These grow until they have reached a diameter of 60 to 70 microns at the end of seven or eight days and are much larger than those seen in malarial blood. At this time they exhibit a delicate striation, due to innumerable small fusiform sporoblasts or sporozoids (polynuclear multiplication of the uninuclear parasite). With the complete formation of the sporoblasts the oöcyst ruptures and the *sporoblasts* escape in large numbers into the general cavity of the mosquito's body and by the lacunar circulation reach the salivary glands and ducts where they accumulate also in large numbers to be discharged into the blood of the bitten victim, where they develop into young asexual parasites. The sporozoid thus developed in the oöcyst corresponds to the spore resulting from the asexual segmentation of the full-grown parasite in the blood. Either one of these fastening on a red blood-corpuscle may develop the asexual or sexual cycle. As a rule, in the human body, in the first generations of parasites the asexual cycle is followed, the sexual form developing later. The sexual forms, sterile while in the human host, are the forms through which the life of the parasite is preserved, spreading infection through the mosquito bite.

Favoring Causes and Geographical Distribution.—These views are further confirmed by the conditions which favor malarial fever, and these notwithstanding exceptions, are hot climates and hot seasons plus decomposing vegetable matter, low river banks frequently covered and uncovered with water and exposed to the sun—in a word, conditions that favor the

[1] A vocabulary of terms has been created to cover the requirements of our new knowledge of this subject, some of which are here appended:

Gamete, a sexually capable element, including *macrogamete*, the female, cell and *microgamete*, the male element (a flagellum originating from a microgametocyte, the parent male cell).

Gametocyte, a cell capable of developing into or producing the gametes; including the macrogametocyte or cell capable of development into the female gamete, and the microgametocyte, the cell from which arise the male elements or microgametes.

Hyaline Body, a nonpigmented young form of the hematozoön.

Ring Form, a shape any young parasite may assume, not a "kind" of organism.

Pigment, the transformed hemoglobin, or "melanin," appearing as brown granules in the parasite (not chromatin granules).

Presegmenters, fully grown parasites, with pigment accumulated in masses, before segmentation occurs.

Schizogony, the asexual multiplication, seen in the human host.

Spozogony, the sexual multiplication, occurring in the mosquito.

Microzoit, or *segment*, one of the offspring of the asexual generation, in the human blood (one of the products of segmentation).

Sporozoit, one of the offspring of the sexual generation, produced in the mosquito, and inoculated by it into the human host where it becomes one of the hyaline forms of parasite, and subsequently grows into the mature form.

Schizont or *monont*, an adult parasite capable of or engaged in asexual reproduction or schizogony.

Gametoschizont, an adult parasite of the sexual generation.

Copula, the female cell or macrogamete fertilized by the male element or microgamete.

Vermiculus or *oökinet*, the motile stage of the copula in which it penetrates the alimentary wall of the mosquito.

Zygote, *oöcyst*, *sporoblast*, *sporocyst*, names given to the copula in the production sporozoits upon the alimentary wall in the body cavity of the mosquito.

breeding of mosquitos. Wherever these conditions occur, malaria is rife. Especially are they found in the southern borders of the north temperate zone, as in Southern United States, Southern Italy, and along the lower Danube; the northern border of the south temperate zone, in the tropics, as Central America, the West Indies, Central Africa and Southern Asia. A freshly upturned soil may furnish, under a sufficiently high temperature, a minimum of 60° F. (15.6° C.), as favorable a focus almost as a marshy river bank. All ages are susceptible, but children are especially liable to take the disease. More men have it than women, for evident reason. Currents of air are thought to have less influence than was formerly supposed, since the mosquitoes laden with the extracorporeal forms do not as a rule come out of their hiding places when the wind blows. Water ingested cannot be a cause of malarial infection, according to modern views, nor can it be inhaled.

Morbid Anatomy.—The morbid anatomy of malaria includes mainly changes in the blood, the liver, and the spleen—changes that vary with the duration and intensity of the disease, to which, however, they do not always correspond.

As to *blood changes* in *acute malaria:* In the true intermittent fevers there is a loss, sometimes considerable, of red corpuscles after each paroxysm, which is made up during the intermission. In the estivo-autumnal form the blood losses are greater and more permanent. *The absence of leukocytosis is characteristic.* In remittent and pernicious malaria—the latter a form characterized by the intensity of the poison and severity of the symptoms—the morbid changes may not be very striking if the patient die in the first attack, but more marked after a second. The blood is described as hydremic, the serum is sometimes tinged with hemoglobin, and the corpuscles, while containing the parasite, present all stages of destruction. *In chronic malaria* the *blood changes* are even more marked. There is a positive secondary anemia in which, as usual, the hemoglobin is decreased rather more than the corpuscles. The leukocytes are almost invariably diminished, the polynuclear leukocytes most, while the larger mononuclear forms are relatively increased. Pigment deposits are abundant, especially in the spleen, which is enlarged and hard.

The *spleen* is enlarged, but not nearly so much as in chronic recurring forms. It is, moreover, soft and its pulp is dark from accumulated pigment in the intervascular cords. In chronic malaria, of whatever form, the *enlarged spleen* is the most characteristic morbid product. It may weigh as much as ten pounds (4.5 kilos.) and measure ten inches (25 cm.) long and four (10 cm.) to six (15 cm.) in width; its capsule is thickened, its substance firm, and the trabeculæ prominent. Pigmented areas abound, due to the plugging with pigment of the intercommunicating lymphoid spaces of the pulp, and in some cases the melanosis is general. The pigment particles resulting from the disintegration of the hemoglobin in the vessels are retained in the spleen, as by a filter.

The *liver* is enlarged and dark-hued, sometimes described as bronze and sometimes slate-color. Even when not visibly altered to the naked eye, there may be no difficulty in recognizing the excess of pigment within and without the small vessels, some of which may be occluded. In fact,

by the aid of a microscope, almost all the tissues may be found abnormally pigmented, even the brain, some small vessels of which may also be occluded.

In chronic malaria the *liver* is also enlarged, to a less degree, however, than the spleen. It is indurated and presents various degrees of pigmentation, which may reach a slate-gray tint. The pigment is contained in the portal canals and beneath the capsule.

The *kidneys* may be similarly pigmented, the pigment lying about the smaller blood-vessels and the Malpighian bodies, and in the cells lining the tubules. In protracted cases of malarial cachexia other tissues may be pigmented. Thus, the small vessels of the brain may be surrounded by pigment and even occluded, so that hemorrhagic infarcts may occur. Even the mucous membrane of the stomach and the peritoneum may be pigmented in extremely chronic cases.

CLINICAL VARIETIES.

The chief varieties of malarial fever admit of easy separation by their symptoms.

Tertian or Quartan Fever (*Intermittent Fever*).

Definition.—This form of malarial fever is characterized by a total remission of fever between paroxysms.

Symptoms.—This, the well-known fever and ague, characterized by distinct paroxysms of chill, fever, and sweat, has a distinct *period of incubation*, which may be as short as 24 hours or even less, though usually it is from a week to 14 days. Sometimes it is very much longer, and even months are said to elapse after exposure before the first paroxysm sets in. The paroxysm is usually preceded by a prodrome of uneasiness and discomfort, sometimes languor and yawning, sometimes headache, sometimes nausea, which forewarns the patient of its coming. As often as not there is no such prodrome. The paroxysm consists of the chill or cold stage, the fever, and the sweat.

The *chill* commonly begins gradually. First there is a creep, then another a little more severe, then another, each growing in severity until the teeth chatter and the body shakes violently. There is, however, great difference in the severity of the chill. It may be a barely noticeable creep or such a chill as will cause the bed and even the windows of the room to shake. At the same time the patient's lips are blue, his face is pale and pinched, and he looks very cold. Yet he has fever. Even before the chill there is a slight rise in temperature, and during it the latter may reach 105° F. (40.5 C.) and 106° F. (41.1°C.) in the axilla or mouth. A surface thermometer may show a lower temperature of the skin, but the internal heat is in strong contrast with the apparent coldness. There may be nausea or vomiting and severe headache. The pulse is small, hard and frequent. The hands are pale, cold and the nails blue. The urine is increased, light-hued, of low specific gravity, though before the chill it may be concentrated and the specific gravity high. The duration of the chill varies from a *few minutes to an hour or more.*

To the chill succeeds the *fever*. The skin is intensely hot and dry and

the face flushed. There is intense thirst. The mouth is dry, the tongue coated, the breath foul. There is no mistaking this stage any more than the first. Yet the actual temperature is but little higher than during the chill, which is well shown in the appended chart, in which it will be seen that the temperature during the chill at two successive observations was 104.2° F. (40° C.) and 104.4° F. (40.18° C.); during the succeeding *fever* it reached at the first observation 104.8° F. (40.4° C.), and at the second, 105° F. (40.45° C.). The duration of this stage is from *half an hour to four or six hours.*

The *sweating stage* follows, with the appearance of drops of sweat on the face, whence it extends all over the body and is various in quantity.

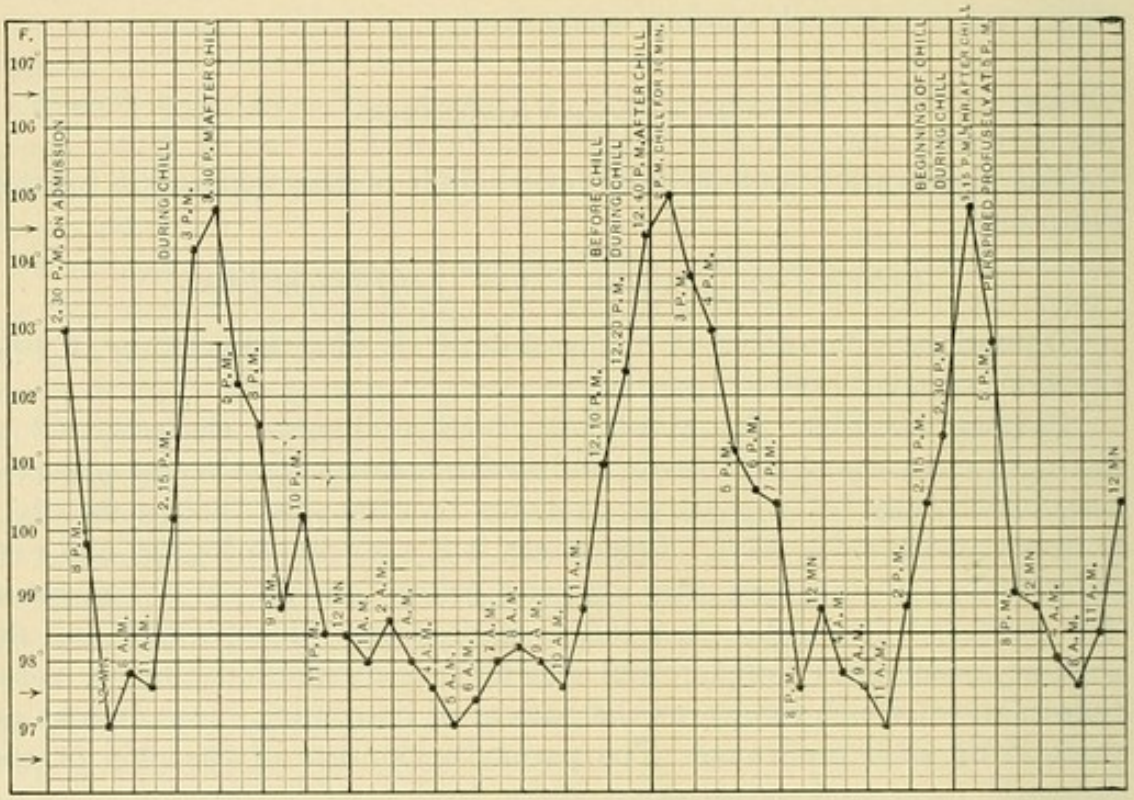


FIG. 12.—Temperature Chart in Intermittent Fever, showing Paroxysms and Intermissions. It will be noted that the temperature has been taken during the chill, and during the fever just after the chill, and that although, as is well known the fever is very high during the chill while the patient *feels* cold, it is still a little higher during the fever just after the chill.

With it comes relief to all the symptoms. Indeed, a sense of great comfort supervenes. It may be a mere suggestion of moisture, or it may be very profuse, drenching the patient's clothing and even wetting the bed. It is commonly proportionate to the severity of the chill. During the sweat the temperature falls rapidly, but if the paroxysm is severe several hours elapse before it attains the normal. It lasts for *half an hour to two hours*, after which the patient feels comfortable and well.

It is not easy to give a satisfactory *rationale* of the three stages. The first and second are undoubtedly the direct result of the same cause—a toxin generated by the plasmodium, since the actual fever which characterizes both is irritative. The superficial coldness and sense of cold of the first stage may be the result of vasomotor spasm contracting the blood-vessels of

the surface and due to an irritation of vasomotor centers by the toxin; the second stage to a derangement of the heat-regulating centers. The third stage is probably a reactive vasomotor paralysis, with the usual leakage from the skin incident to it.

The total duration of the paroxysm is from *eight to twelve hours*, and usually between the paroxysms the patient feels perfectly well. During the paroxysm the *spleen* becomes *enlarged* and the malarial patient has often *herpes labialis*. The size of the spleen subsides after the paroxysm, although

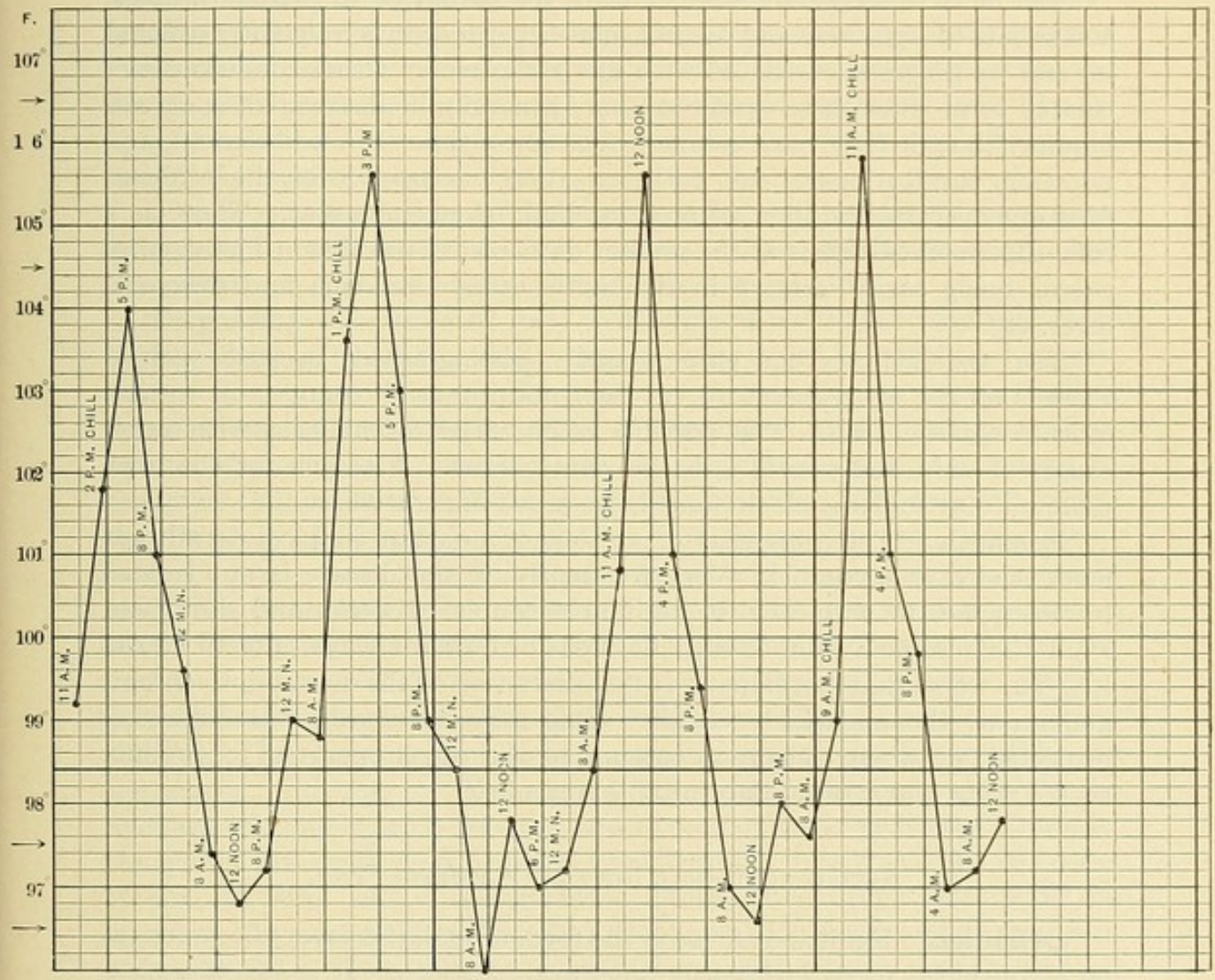


FIG. 13.—Temperature Chart in Intermittent Fever, Showing the Paroxysms and Intermissions.

with its repetition there is a disposition to permanent enlargement, resulting finally in the ague cake. In 48 hours or 72 hours the same attack occurs.

The types of the paroxysm so characteristic of intermittent fever have already been referred to. The order of frequency is quotidian or double tertian, tertian, and quartan; the first being by far the most frequent.

Diagnosis.—The diagnosis of intermittent fever is most easy, and a typical case should be easily recognized after the second paroxysm, if not after the first. The presence of the plasmodium in the blood makes a

positive diagnosis of malaria while its absence is not always a positive sign of the absence of malaria. If the case be less typical and the chill omitted or so slight as to escape recognition, a certain resemblance between such a paroxysm and the *hectic fever of tuberculosis*, with its subsequent sweat, must be admitted, and it not infrequently happens that such fevers are declared to be malarial by the attending physician.

The attacks of chills and fever accompanied by jaundice in ball-valve gall stone frequently require examination of the blood to rule out malaria, but the jaundice and abdominal pain should suggest gall stones.

Still more close is the resemblance of the paroxysm to the chills, fever, and sweats of *septicemia* and *pyemia*, while suppuration is frequently ushered in, and its progress associated with like symptoms. Other conditions calculated to produce these symptoms may generally be discovered on careful inquiry, though they may escape notice for a time. In addition to suppuration, surgical operations, catheterization, puerperal fever, the incidence of empyema, and the like, all produce chill, fever and sweat. Then, as a rule, the blood culture is at the present day an easy solution. The streptococcus being easily isolated in the septic cases, empyema and puerperal fever, etc.

The so-called nervous chill is easily distinguished, because with it there is no rise of temperature—at least nothing at all comparable to that of the malarial chill. The possible combination of malaria with other causes of chill and sweats is to be remembered. A search for the plasmodium may under such circumstances prove imperative.

Prognosis.—The prognosis of tertian or quartan simple intermittent fever is always favorable. Very frequently, if the disease is not treated by medicine, it will exhaust itself in a couple of weeks and disappear, while the administration of suitable doses of quinin always puts an end to it. The worst that can happen is the conversion of the disease into chronic malaria or the malarial cachexia. This may occur when treatment is neglected, or when constant exposure to the cause operates to produce such a state notwithstanding suitable treatment.

Estivo-autumnal Fever.—*Remittent Fever.*

Definition.—Remittent fever is the form of malarial fever characterized by a continued fever with paroxysmal exacerbations. It is also known as bilious fever. It has become rare at the present day in the North Atlantic States of America, and is confined mainly to the South Atlantic States, to Italy, the lower Danube sections in Europe, and to tropical countries. It occurs in the late summer and fall, and hence is included among the *estivo-autumnal* fevers.

Symptoms.—It generally begins with a *chill* after a *period of incubation* analogous to that of intermittent fever. It is more likely to be preceded by *prodromal symptoms* than is intermittent. There are malaise, intense headache, a coated tongue, and often obstinate nausea and vomiting. Vomiting of bilious matter is a conspicuous symptom. These gastric symptoms, formerly ascribed to gastritis, are probably caused by central nervous irritation due to the toxin. There may be *jaundice* resulting from

obstructing cholangitis; the liver may be tender on pressure. The chill may be less severe, and the other stages of the paroxysm less characteristic. The fever does not pass off, but continues with a full, bounding pulse and a temperature of 102° to 103° F. (38.9° to 39.5° C.). There are daily remissions, as in typhoid fever, but they do not follow the same rule of tidal rise. Yet the two diseases are very similar, and often thoroughly try the diagnostic skill, even of those who are accustomed to meet estivo-autumnal fever. The temperature rises quite as high as in typhoid fever, and the patient is usually very ill. The two diseases occur at the same time of the year—the autumn. It is not impossible for them to be concurrent.

There is little else that is peculiar in the symptomatology of the common forms of remittent fever besides the prodrome, the malarial organism, and the peculiar paroxysmal character. In prolonged remittent fever the typhoid state is sometimes assumed, manifested by dry tongue, hebetude, stupor, and feeble, frequent pulse.

The *urine* is high-colored, with high specific gravity, depositing a copious sediment of urates, and sometimes contains biliary coloring-matter. Not infrequently it contains blood-corpuscles or hemoglobin.

Diagnosis.—As intimated, it is with typhoid fever that remittent fever is most likely to be confounded. Occasionally in the South there has been confusion with yellow fever. To us who study *typhoid fever* in the North it seems surprising that there should be any confusion with this disease. It is ordinarily so easy, after watching the temperature chart for a few days, to recognize typhoid fever. In the South it is, however, different, and, before the discovery of Laveran's plasmodium, the therapeutic test—administration of quinin—was frequently needed to settle the question; for remittent fever, like intermittent fever, yields to quinin. In such cases a successful search for the hemocytozoa will settle the question promptly. This is the variety in which we have the small, actively motile hyaline forms of organism, while the larger crescentic, ovoid bodies are to be looked for as soon as the disease has existed over a week. An unsuccessful search may still leave the matter in doubt, but if the nasal hemorrhage, the typhoid spots, the diarrhea of typhoid, and the temperature are not sufficiently characteristic, the quinin test ought to put an end to all doubt. The Widal test has come to our aid, also, and if responded to affords conclusive evidence of the presence of typhoid fever. Study and observation impels us to say, with Osler, that all of the continued endemic fevers of the South may be resolved into typhoid or malarial fever, or the two diseases existing at the same time in the same individual.

The diagnostic distinction between remittent fever and *yellow fever* will be given when considering the latter disease.

Prognosis.—This is usually favorable when treatment can be promptly applied.

Pernicious Malarial Fever—The Congestive Chill.

Definition.—This variety of malarial fever is characterized by the extreme severity of its paroxysms.

Occurrence.—It still presents itself occasionally in the North, but is much rarer than it was 50 years ago. Up to a few years ago it was not un-

common to hear of the death from this cause of a prominent citizen at his country seat on the banks of the Delaware above Philadelphia. Later, the cases became confined to the servants and others out late at night or early in the morning, and, more recently still, even such cases as these are seldom reported, although the milder forms of malaria prevail. It is still prevalent in the Southern United States, in Italy, the lower Danube, the Niger delta, and other parts of tropical Africa. Pernicious malarial fever is a form of estivo-autumnal fever.

Two principal types present themselves—the *comatose* and the *algid*. Other adjective terms based on special features more or less characteristic, are the *hematuric*, the *bilious* and *ansthmatic*. As malarial hematuria is not confined to the pernicious variety, it will receive separate consideration. The bilious type is that of the ordinary severe form of remittent fever, while asthma characterizes the comatose and algid types.

1. The *comatose* type may or may not begin with a chill, but in its more serious forms the chill is a conspicuous feature, being severe. To it succeeds the comatose state, whence the term *congestive chill* often used in the South, where the popular notion prevails that if the first paroxysm does not kill, the second will. This is an exaggerated idea of its seriousness, although it is certainly a very grave affection and often terminates fatally. A low, muttering delirium may supervene, the eyes are bloodshot, the skin is hot and dry, the temperature rising to 105° or 106° F. (40.6° or 41.1° C.). The comatose condition is the result of occlusion of the small vessels of the brain with the organism, and lasts until a partial elimination of the poison has taken place, usually from 12 to 24 hours later. The patient may, however, perish without return to consciousness, or consciousness may return to be followed in a short time by fatal relapse.

2. The *algid* type is characterized by gastric symptoms, extreme nausea and vomiting, which are mostly followed by collapse, for there is intense prostration, with coldness of the surface and extremities. The symptoms are, indeed, comparable to those of the collapse of cholera. There are the same small, feeble pulse, frequent, shallow breathing, cramps, vomiting, purging, husky voice, and thirst with suppressed urine, and with these the same clearness of intellect until death steps in—the last scene in the drama, in which asthenia also plays a leading rôle.

In these cases there may or may not be a chill, while the patient feels cold and the surface temperature is never high, rarely exceeding 101° F. (38.3° C.), and falling as low as 96° F. (35.6° C.). The internal temperature is, however, high.

Diagnosis.—Pernicious malarial fever is to be distinguished in its comatose form from *typhoid fever*, and in its algid type from *yellow fever*. The presence of the plasmodium and pigment in the blood are the distinctive features to be carefully sought.

Irregular Forms of Malarial Fever.

It sometimes happens that the paroxysm in intermittent fever omits one or more of its stages. Especially is this the case with the chill in which event the disease has received the characteristic name of "dumb ague." Fre-

quently, however, what receives the name of "dumb ague" is something altogether different. The "malarial cachexia," for instance, is sometimes spoken of as "dumb ague." Like malarial cachexia "dumb ague" is found among the older residents of a malarial district.

Quite often it happens that the malarial paroxysm consists of nothing but a state of *drowsiness*, which recurs at regular intervals and is very characteristic. The temperature in these cases is elevated, but not very high, 100° F. (37.8° C.) or perhaps 101° F. (38.3° C.); there may be *slight delirium*.

The term *latent intermittent fever* is applied to a combination of symptoms affecting persons living in malarial districts—consisting in a weary, languid feeling, associated with want of appetite, headache, nausea, vomiting, constipation, and coated tongue. Sometimes the so-called "bilious attacks," which exhibit the above symptoms in an aggravated form, especially the headache and vomiting, are malarial in their origin, and may be broken up with quinin. Such attacks may be called *malarial migraine*.

Irregularity of fever or chills, or both, may be caused by infection with more than one group of the same kind of parasite occurring at different times, or there may be infections of different kinds of parasite maturing at their own specified time.

Much harm results from a diagnosis of malaria with vague symptoms quoted above. Such a diagnosis should never be made unless the malarial organism is found in the blood or the disease responds promptly to quinin.

Malarial Hematuria, or Hemaglobinuria, or Intermittent Hematuria—Blackwater Fever.

This form of hematuria or hemaglobinuria is the direct result of malarial poison. The first account of it in this country was published by George Troup Maxwell in the "Oglethorpe Medical and Surgical Journal," Savannah, Ga., July, 1860. It occurs in two varieties, a mild and severe.

The Mild Form.—While it is a very frequent symptom of the pernicious or malignant type of malarial fever, hematuria also occurs as a symptom, and, indeed, sometimes the sole symptom, of the milder varieties of malaria, such as occur in the Middle States of the United States. Rarely are they accompanied by a chill, and there may be no symptoms whatever except the bleeding. More frequently there is a cold feeling, the tips of the nose and of the fingers become cold, and the lips become blue, immediately after which the urine is found to be bloody. Microscopic examination of the urine will recognize in some instances blood-disks, in others no corpuscles can be found. It is a *hemoglobinuria*. The hemorrhage occurs daily or on alternate days, more rarely at longer intervals. Sometimes it is continuous, with exacerbations at regular intervals. In all cases of unexplained hematuria the blood should be examined for the malarial organism.

The Grave Form—Blackwater Fever.—When a symptom of pernicious malarial fever, the condition is more apt to be hemoglobinuria than hematuria; it is more aggravated and more continuous, although still intermittent. It may also be associated with hemorrhages from the nasal and oral mucous membranes, and even from the stomach, which add much to the gravity of the case.

To the grave form of malarial hemoglobinuria the name *blackwater fever* has been given.

The organism causing blackwater fever is usually the estivo-autumnal organism but the tertian and quartan may also cause it.

William Krauss, of Memphis, has furnished a description of the mode of onset and symptoms of malarial hemoglobinuria as it occurs in Tennessee, from which is condensed the following:

After a variable period of neglected intermittent or remittent fever, especially in a subject already cachectic, there occurs suddenly a violent chill, with the usual high temperature. The face is anxious, and cyanotic, and (in from 15 minutes to 2 hours) there ensues more or less vesical tenesmus, and a small quantity of urine of a port wine to inky black hue is voided. This may occur at very short intervals during the day (the onset is usually before sundown) and in favorable cases the quantity of urine increases while the color becomes lighter. There is great shock, the pulse is 100 to 150 and very small, the breathing is embarrassed and hurried, often sighing in character. The stomach becomes irritable, and projectile vomiting sets in, the vomited matter being of a spinach-green color. In the morning of the second day a very intense icterus is first recognized. The vomiting may continue in spite of all measures looking to its control, the parasites are still in the blood and the temperature is usually high, 103°–105° F., although if the toxemia is extreme there may be great depression and the temperature may be subnormal. A hardening pulse at this stage indicates grave renal involvement and the subsequent history is that of fatal uremia. A subsidence of the restlessness indicates re-established permeability and a favorable prognosis. The triple symptoms—hemoglobinuria, vomiting and icterus—are essential for diagnosis.

As to the hemolysis, there is reason to believe it takes place in the spleen and kidneys. The excessive waste hemoglobin discharged by the spleen into the portal vein furnishes the liver with more material for bilirubin, hydrobilirubin, etc., and the irritative hyperactivity of this organ results in an excess of both internal secretion and excretion, the latter being represented by an imperfect bile, engorging the bile capillaries and causing an overflow into the lymphatics. A hemo-hepatogenous jaundice is the result. The disease is not paroxysmal and lasts about four days in favorable cases. Some recent authors believe, Craig, for instance, that true hemoglobinuric fever is due to another organism than malaria, and hence is a different disease.

Chronic Malaria and Malarial Cachexia.

Definition.—This is a condition which often supervenes in cases imperfectly or ineffectually treated, or in persons living in malarial districts where there is constant exposure to the cause and consequent repeated attacks.

Symptoms.—The most striking symptom of this condition is *anemia* of a peculiar kind. The incident changes in the blood have been referred to on page 54. The skin exhibits a dirty-yellow or sallow appearance, often erroneously characterized as "bilious," as though it were a form of jaundice, which it is not, although there may be sometimes slight jaundice also.

Such persons have, in addition, deranged digestion. The tongue is pale, flabby, and coated and the breath sometimes foul. The bowels ar constipated. The hands and feet are cold, the circulation is generally bad, and the temperature is subnormal, though it may alternate with the feverish state. In consequence of the hydremic blood there is sometimes edema of the feet, and even general anasarca. The *spleen is enlarged*, often extending as low as the ilium.

Some very unusual symptoms are included in the symptomatology of this form of malaria—as, for example, *paraplegia* and *orchitis*. The former condition may be the result of deranged circulation in the spinal cord, but it is difficult to regard the latter as anything except a coincidence. A remarkable case of malaria with symptoms of disseminated sclerosis was reported by William G. Spiller in the "American Journal of the Medical Sciences" for December, 1900. The autopsy disclosed sclerosis of the right crossed pyramidial tract throughout the spinal cord, not intense, but unmistakable.

The *plasmodium* is found in this form of malaria also, and the *crescent* is said to be the form more or less characteristic of it. The recognition of the organism is of value in the diagnosis although the history of the case and the presence of enlarged spleen are also important aids to diagnosis, especially as the plasmodium may elude detection altogether. In leukemia there is also enlarged spleen, but the microscopic examination of the blood reveals at once in the latter disease the excess of colorless corpuscles.

Prophylaxis of Malarial Fever.—Much may be done to avert malarial infection. It is not possible for the organism to enter the system by the stomach or respiratory passages. This being established, prophylaxis must consist in measures to destroy the mosquito or escape its bite. To exterminate the adult mosquito is manifestly impossible. Yet it is not chimerical to look forward to the possibility of destroying the insect in the larval state as it exists in pools and ponds, uncovered rain barrels, old tins containing water, in the axils of leaves of tropical plants, indeed in any collection of stagnant water large or very small. Attention need only be called to the wonderful work of Gorgas and his associates in the Canal Zone which has made possible the practical completion of the Panama Canal. Formerly, continued work was impossible on the isthmus because of the blighting effects of malaria. Now the disease is rare in the Canal Zone due to the destruction of the mosquito and screening of houses. A most important measure is the protection of the malarial patient from the bite of the mosquito. Every malarial patient should live in a thoroughly screened house free from mosquitoes, until his blood is entirely free from organisms of malaria.

In addition to killing mosquitoes our prophylactic measures must consist in protecting against mosquito bites by netting, and in making the blood as uncomfortable a habitat for the plasmodium as possible by charging it with quinin—*quinin prophylaxis*. To this end a few grains of quinin, say five to ten (0.333 to 0.666 gm.), should be taken daily, especially by newcomers in malarial districts and by all residents at times. No less efficacious is the arsenical prophylaxis as conclusively shown by experiments on the Adriatic railways reported by Celli in his book on "Malaria." It seems pretty well

founded, too, that the cause of malaria is more active after nightfall. This is consistent with the mosquito theory. Hence, exposure at these times should be avoided.

Further prophylactic treatment consists in measures to prevent inundations, and stagnant waters in any form.

Treatment of the Different Forms of Malaria.—The treatment of *intermittent fever* is preëminently by *quinin*. The dose required varies, but in mild tertian variety 15 to 30 grains (1 to 2 gm.) are usually sufficient for an adult. Sometimes larger doses may be needed in inveterate cases, and always larger doses in the estivo-autumnal types of fever. It matters not much how the drug is administered, but there is a best way for each case. A good rule, is to give an hourly dose of 10 grains ($.\frac{3}{10}$ gram), beginning long enough before the expected paroxysm to get the quantity previously decided upon into the blood at least two hours before the chill is expected. If the dose first selected fails, the second should be made larger. It is to be remembered, however, that quinin, like other drugs, acts more efficiently after a free aperient, while constipation decidedly interferes with its prompt and efficient action. Some prefer a mercurial, as 8 to 10 grains (0.55 to 0.666 gm.) of blue mass, or 5 to 10 grains (0.33 to 0.66 gram) of calomel, but provided a free movement is secured, it does not matter much how it is accomplished. Having broken the paroxysm, it is well to continue the quinin for a few days in smaller doses, and to anticipate the seventh day subsequent to the last chill by another full dose of the drug, and to do so at intervals of seven days for some weeks. Under ordinary circumstances the freshly prepared pill of quinin made with aromatic sulphuric acid is to be preferred, or the quinin may be given in capsules. This is easily soluble and is not so unpleasant to take as the solution, which is, however, more readily absorbed. The sugar-coated and gelatin-coated pills are not so certainly efficient, as they sometimes, especially with deranged digestion, pass through the bowel undissolved.

Some physicians prefer to administer quinin during the decline of the fever. This was the practice of Sydenham in giving the bark. Among modern physicians disposed to follow this method are Bacelli, A. Plehn, Maclean, Manson and other East Indian physicians; and in this country George Dock.

The treatment of the paroxysm itself is by measures calculated to combat each stage. During the chill, to satisfy the patient artificial warmth should be supplied, and will give comfort; during the fever, if the temperature is above 102° F. (38.9°C.), the body may be sponged to reduce the heat, and during the "sweat" the patient should be carefully dried. If there be any reason why quinin should not be exhibited, the other alkaloids of cinchona, as cinchonidin, are equally effectual in doses about one-fourth larger. No substitute for cinchona or its alkaloids has ever been suggested which has stood the test of trial.

The treatment of *remittent fever* is essentially that of intermittent fever. It is in this form that the mercurial aperient is deemed especially valuable as a preliminary by those having wide experience in its treatment. The continued nature of the fever, and the tendency to a typhoid state which often develops, demands a liquid diet, with the careful addition of stimulants.

The *pernicious forms* of malarial fever are treated by quinin, as are the other varieties of the disease. Sixty grains or more may be necessary, and advantage must be taken of hypodermic injections. Soluble salts should be used, such as the bisuphate, hydrochlorate, and hydrobromate, of which 15 grains (1 gm.), dissolved in distilled water, are a dose. Double this dose may be given. The bisulphate of quinin may also be administered hypodermically in solution with tartaric acid, 30 grains (2 gm.) of the quinin to 5 grains (0.333 gm.) of the tartaric acid. The muriate of quinin and urea may also be given hypodermically in 10, 15 or 20-grain (0.666, 1, and 1.33 gm.) doses. It is especially commended by Solomon Solis-Cohen, who advises, as soon as the diagnosis is established, and without reference to the time of paroxysm, a single injection of from 10 to 15 grains (0.66 to 1 gm.) of the salt, dissolved in a syringeful (20 to 30 minims) of boiling water. Should a paroxysm recur at the following period, a second injection is given, and should further paroxysms occur, injections are given in corresponding number; otherwise but three injections are given the first seven days, and two injections during the second seven days. Bass suggests the use of nitroglycerine in comatose forms in order to dilate the smaller blood vessels and allow the large malarial bodies to pass through the capilaries and become exposed to the quinin which is in the circulating blood. It may also be given by the mouth in capsules in the same doses.

Even the intravenous injection of quinin has been recommended in intractable cases, and for this purpose the soluble bimuriate is most suitable. Fifteen grains (1 gm.) with 1 grain (0.066 gm.) of sodium chlorid are dissolved in 2 drams (8 c.c.) of distilled water and injected.

Methylene blue (methylthioninæ hydrochloridum, U. S. P.) was introduced by Ehrlich and Gutmann in 1891 in the treatment of the malarial fevers; and H. C. Wood, Jr., collected 425 cases up to 1906, in 362 of which final cures were affected. It is given in 3 grains (0.1 gm.) doses every three hours in pill or capsule. Its peculiar staining qualities constitute its chief objection.

In addition to the use of quinin, prompt measures must be taken to combat all symptoms which add to the dangers of the situation—stimulants for the asthenia; artificial heat for low temperature; morphin hypodermically, to relieve the pain and allay nausea; cool sponging or bathing to reduce the temperature, and saline cathartics to relieve congestion in the comatose form.

I. L. Van Zandt of Fort Worth, Texas, recommends atrophin, hypodermically in 1/60-grain doses repeated in 20 minutes if necessary, for the symptoms of collapse so characteristic of the algid type. He associates this drug with 1/30 to 1/20 doses of strychnin similarly administered.

The treatment of the *milder varieties of hematuria* is most satisfactory. The administration of quinin in almost any way, say 3 grains (0.19 gm.) every three hours for several days, will effectually break up the paroxysms, and its use in smaller doses for some time longer will prevent a return.

Not all practitioners, even those residing in districts where it is most rife, are agreed upon the treatment of the *graver forms* of *malarial hematuria* as it occurs in Southern latitudes. While some do not hesitate to use quinin,

in the way it is used in ordinary forms, many of the most experienced physicians in the Southern United States object to this drug, on the ground that it may even cause hemoglobinuria, a belief endorsed by such authorities as Tomaso Celli, who was the first to direct attention to it, Marchiafava, Bignami, Bastianelli, the brothers Plehn and Robert Koch abroad. I cannot better express the views of the Southern physicians referred to, than by quoting one of their number, Krauss,[1] already mentioned, who says:— "We are therefore forced to the conclusion that hematuria once begun, quinin has no place in its therapy." And again, "Only so long as the sporulating parasites are in the peripheral blood and there is no hematuria, is it fit to be used."

It seems that malarial hemoglobinuria is brought about by a hemolytic substance or anti-alexin set free in the blood-plasma. The hemolysis may be precipitated by an unusual sporulation of plasmodia on the one hand or anti-malarial treatment on the other. In the normal blood, quinin will not cause hemoglobinuria. Moreover, it is held that a long continued action of the malarial toxin is necessary and such is the experience of our Southern physicians, who found it especially prone to occur in neglected intermittent and remittent fevers. It is especially important to note that the quinin and malarial toxin are simply the agents which liberate the hemolytic substance and are comparable to the "addiments"[2] of Ehrlich and Morgenroth.

The effect of quinin on susceptible subjects is due entirely to the amount of hemolytic stored up, and this varies with different individuals. Some set free their storage after a single dose, and subsequent doses can do no harm. Others eliminate their storage after a few days' duration of the syndrome, and in some it persists. In these, all treatment must be suspended and they must be allowed to immunize themselves. As small a dose as 3/4 of a grain (48.6 mgr.) has produced hemoglobinuria. Hemoglobinuria, being therefore a consequence of malaria and not a cause, it does not *per se* call for quinin which, on the other hand, may precipitate it.

It is evident that the treatment of the graver forms of hematuria becomes a much more complex problem if these new views be accepted. Again quoting Krauss, "So long as sporulating parasites are present in the blood and there is no hematuria, push it (quinin) to the limit. If oral administration is not practicable give the neutral chlorid in very dilute solution with an antitoxin syringe." Otherwise the use of quinin must be cautious and tentative. If the urine redden or a chill supervene the drug must be omitted. On the other hand, before hematuria has occurred quinin may be given, because "a further neglect of an already neglected malaria is more provocative of a hemoglobinuria than any amount of quinin before the attack." If well borne the dose may be increased.[3] In a word blackwater fever (ma-

[1] "Memphis Medical Journal." April, 1902. Page 177.

[2] Addiment, Ehrlich and Morganroth's term (1899) for an active thermolabile substance (destroyed by a temperature of 56° C.) contained in normal serum and capable of liberating the immune body of Ehrlich and setting up bacteriolysis and hemolysis.

[3] Krauss has written in further detail the treatment as he would practise it in such a case and he has furnished me with the following:

1. If the onset follows the use of quinin I would suspend the use of it until the urine has cleared up, then begin it cautiously, and if there is no hemolytic reaction, proceed (intramuscularly, of course) until it is believed that 30 grains of it is in the blood at one time. If a reaction (rigor and dark urine) follows, wait a few days and begin still more cautiously.

2. If the patient has had no quinin, and the symptoms are not those of a malarial paroxysm, I would do the same.

3. If there are distinct paroxysms of fever without a quinin history, the physician may elect to take

larial hematuria) does not occur in individuals who have not had malaria. Quinin may be given if the malarial infection is severe—otherwise it often does more harm than good.

Treatment is otherwise symptomatic usually eliminative and restorative. Elimination is secured by water drinking and rectal injection of normal salt solution, or if not retained, by hypodermoclysis or even intravenous injection, one object being to prevent inspissation of the blood, favored by the sharp purging and vomiting. The skin must be kept active by cautious doses of pilocarpin, by hot packs and by the use of woolen clothing. Stimulating diuretics are strongly disadvised. Vomiting may be controlled by morphin injection and cold drinks, such as champagne, carbonated waters and Appollinaris water. Strychnin may be given hypodermically.

It is in *chronic malaria* especially that *arsenic* becomes a useful remedy. Fowler's solution being the best preparation. It should be given in ascending doses. Arsenic may be given in the form of "Neo-salvarsan"—Ehrlich's preparation, or the cacodylate of sodium, which may be given hypodermically in 2 grain (0.132 gm.) doses in obstinate cases. *Iron* is often advantageously associated with it, and for such a combination the solution of the chlorid of arsenic and the tincture of the chlorid of iron are especially suitable. Here, the constipating effect of iron, so justly complained of, would be obviated by giving doses little in excess of what can be absorbed. For it is this excess remaining in the alimentary canal that works the mischief. Three minims (0.333 gm.) of the tincture of the chlorid, combined with as many of the solution of the chlorid of arsenic, are a proper dose, but the arsenic should be increased until a slight puffiness of the face results. The carbonate of iron or the reduced iron sulphate may be given in doses not exceeding one grain (0.066 gm.). A modified Blaud's pill containing in addition to the carbonate of iron, arsenic in 1/30 grain (0.0026 gm.) doses is a very efficient and convenient remedy. The administration of iron should be kept up a long time.

Quinin should not be omitted in this form of malaria, but there is no advantage in giving it in large doses. It should rather be kept up a long time. *Strychnin* and *mineral acids* are also useful remedies for the gastric derangement, while constipation may be treated by an occasional mercurial purge, say a couple of grains (0.132 gm.) of blue mass, to which may be added as much compound extract of colocynth, and as much extract of hyoscamus, or 1/8 grain (0.008 gm.) of extract of belladonna.

YELLOW FEVER.

SYNONYMS.—*Febris flava; Bilious Remittent Fever* (Rush); *Kendall's Fever; Barbadoes Distemper; Elodes icterodes; Typhus icterodes; Typhus tropicus.*

Definition.—Yellow fever is an acute infectious disease, characterized by a febrile paroxysm succeeded by a brief remission and a relapse. It is

his chances with quinin, by giving four simultaneous injections in different places of 5 grains each. If, coincident with the time of absorption, a rigor occurs, its use is discontinued even though the case appear hopeless, else proceed to saturation. Time this dose so as not to meet a sporulation.

4. In comatose cases a blood examination may differentiate between active sporulation and other causes and treatment given according to rule 3.

5. During convalescence an attempt to get one good "saturation" should be made.

associated more or less constantly with jaundice, and tendency to hemorrhage especially into the stomach, whence the blood is vomited constituting "black vomit." Neither jaundice nor black vomit is essential to the disease. The infecting organism is not known, but is dessimated through the medium of the mosquito stegomayia fasciata.

It is in a sense an American disease, and except in Spanish ports it has been limited in Europe to ports to which it has been carried. It is, however, endemic on the west coast of Africa as well as in the West Indies until modern regulations succeeded in almost stamping it out.

Distribution.—John Guitéras makes three areas of infection: (1) The focal zone, in which, up to 1901, the disease was never absent, including Havana, Vera Cruz, Rio de Janeiro, and other Spanish-American ports. (2) Perifocal zone, or region of periodic epidemics, including the ports in the tropical Atlantic, in America, and Africa. (3) The zone of accidental epidemics, between the parallels of 45 degrees north and 35 degrees south latitude.

A very interesting fact in connection with yellow fever is its limitation to the sea and the seacoast, as it rarely invades interior cities or altitudes higher than 1000 feet (300 meters).

Etiology.—Reed and Carroll have shown that the infective agent is probably an ultra-microscopic organism, a member of the animal kingdom, in the blood of the infecting person.[1] These results are confirmed by the yellow fever commission of the Pasteur Institute. The Pasteur commission has further shown that the organism can be separated from the serum and the latter rendered innocuous by passing through the finest grade of Pasteur filter. As to the propagation of the disease, the same observers with Aristides Agramonte, all of the United States Army Medical Department,[2] have placed the *mosquito theory* of the *origin of yellow fever on so* substantial a basis that further discussion of other theories may as well be laid aside. This theory, which, it will be remembered, makes the mosquito the host of the unknown parasite of yellow fever, was first advanced by Carlos J. Finlay, of Havana, as far back as 1881,[3] but made little impression. The studies of Reed and his colleagues made in the island of Cuba in 1900, are most convincing. They included two divisions: First, the exposure of non-immunes to the bites of mosquitoes which had bitten yellow fever subjects; and second, exposure of another group of non-immunes to fomites by handling and sleeping in clothing saturated with the discharges of yellow fever patients. Their conclusions are as follows: 1. The mosquito—*Stegomyia fasciata*—serves as the intermediate host for the parasite of yellow fever. 2. Yellow fever is transmitted to the non-immune individual by the bite of a mosquito that has previously fed on the blood of those sick with this disease. 3. An interval of about 12 days or more after contamination appears to be necessary before the mosquito is capable of conveying the infection. 4. The bite of the mosquito at an earlier period after contamination does not appear to confer any immunity against a subsequent attack. 5. Yellow fever can also be experimentally produced

[1] "Etiology of Yellow Fever: A Supplemental Note." "Am. Medicine," February 22, 1902.
[2] "Etiology of Yellow Fever," "Philadelphia Medical Journal," October 27, 1900. "Etiology of Yellow Fever: An Additional Note," "Journal of American Medical Association," February 16, 1901.
[3] "Annales de la Real Academia," vol. xviii., 1881, pp. 147–169.

by the subcutaneous injection of blood taken from the general circulation during the first and second days of this disease. 6. An attack of yellow fever produced by the bite of a mosquito confers immunity against the subsequent injection of the blood of an individual suffering from the non-experimental form of this disease. 7. The period of incubation in 13 cases of experimental yellow fever has varied from 41 hours to five days and 17 hours. 8. Yellow fever is not conveyed by fomites, and hence disinfection of articles of clothing, bedding, or merchandise supposedly contaminated by contact with those sick with the disease is unnecessary. 9. A house may be said to be infected with yellow fever only when there are present within its walls contaminated mosquitoes capable of conveying the parasite of this disease. 10. The spread of yellow fever can be most effectually controlled by measures directed to the destruction of mosquitoes and the protection of the sick and well against the bites of the insects. 11. While the mode of propagation of yellow fever has now been definitely determined, the specific cause of this disease remains to be discovered.

Yellow fever attacks all races, both sexes, and all ages except the very young. Yet it is through the young that the disease is maintained in a native population, because protection is secured by a previous attack or long residence in a locality in which it is endemic, and it is the young who, as they grow up, furnish the pabulum for fresh cases. The negro and the creole, although not immune, are comparatively so. More males are attacked than females, because of their frequent exposure. Strangers are especially liable.

Morbid Anatomy.—Intense *yellow coloration* and *hemorrhagic extravasations* under the *skin* are present. The yellow coloration is due to a mixed hepatogenous and hematogenous jaundice. The serum of the blood is red-tinted, because of its containing dissolved hemoglobin. The *liver* is the organ which has always been regarded as exhibiting the most characteristic change. Yet this is not always so. It becomes ultimately fatty, when its color resembles the yellow of admixed coffee and milk—*a café au lait* appearance—as contrasted with the more bronzed appearance of the liver of remittent fever. Earlier in the disease the organ may be slightly enlarged from hyperemia. It may be a nutmeg liver. The liver-cells present various stages of fatty degeneration, with necrotic masses in and between the liver-cells, described by George M. Sternberg. The *gall-bladder* is generally empty. The kidney may exhibit cloudy swelling of even acute nephritis, and pale, fatty areas may be seen at the bases of the pyramids. Various bacteria are found in the liver and kidney.

The *stomach* after death contains more or less of the "black vomit," which is a mixture of transuded serum and altered blood pigment. The mucous membrane of the stomach is hyperemic and more or less swollen, and there are blood extravasations.

Surgeon Eugene Wasdin,[1] in a paper on the postmortem findings of yellow fever, says the morbid appearances postmortem cannot be regarded as sufficiently distinctive to admit a diagnosis from them alone.

Symptoms.—Yellow fever has a *period of incubation* of from 24 hours to five days, very rarely exceeding the latter. It is usually three or four

[1] "United States Marine Hospital Reports for the Fiscal Year 1898."

days. (See Reed and Carroll's conclusions.) After this follows the *stage of invasion* or *febrile stage*, with sudden onset and generally a chill, promptly followed by headache and severe pain in the back and limbs. The patient may be seized at any time, day or night. Surgeon R. D. Murray,[1] of the United States Marine Hospital Service, emphasizes the fact

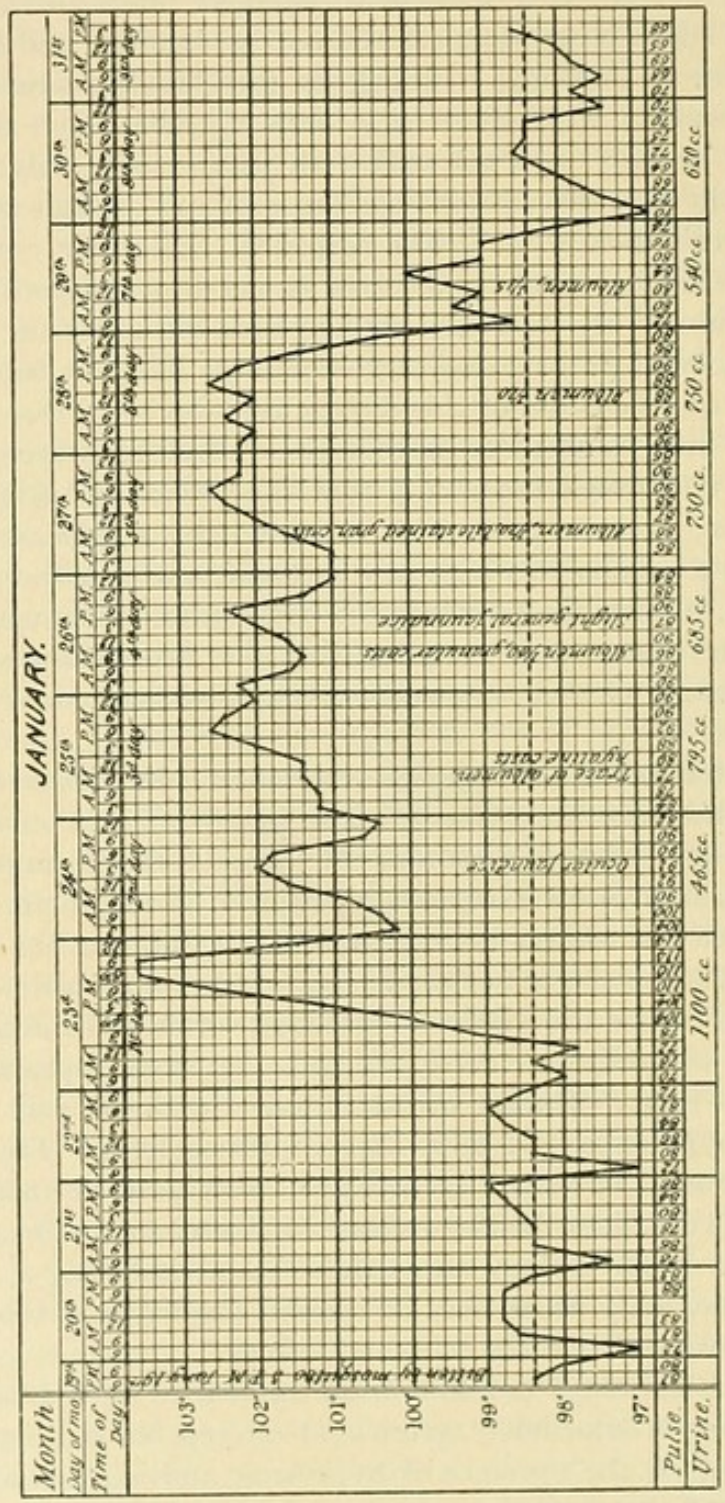


Fig. 14.—Chart of Yellow Fever—Produced by the Bite of Culex Fasciatus. After Reed, Carroll and Agramonte. Transactions Association of American Physicians, vol. xvi., 1901.

that yellow fever *usually begins at night* when the patient is relaxed, while malarial fever attacks him more frequently when at work. The fever rises rapidly to 102° F. (38.9° C.) and as high as 105° F. (40.5° C.). The pulse corresponds, until the second or third day when it begins to fall even while the fever keeps up. The skin feels hot and dry, but less pungently so than

[1] "Marine Hospital Reports," 1899, p. 303.

in typhus. Even on the first day the face is flushed, the eyes are injected, the lids perhaps slightly tumid, the tongue furred but moist the throat sore, the bowels constipated, the urine is scanty and often albuminous, though albuminuria does not generally appear until the evening of the third day. So, too, at this early stage there may be slight *jaundice*, and Guitéras says this "early manifestation of jaundice is undoubtedly the most characteristic feature of the facies of yellow fever." There may be nausea from the beginning, but it is not until the second or third day that it is aggravated and the characteristic "black vomit" makes its appearance. This resembles an infusion of coffee, and deposits a sediment comparable to coffee grounds, and which consists of broken-down red corpuscles and hematin. In the worst cases the vomited matter may be tar-like in appearance and consistence. On the other hand, "black vomit" is not always present, being generally confined to the severe cases. In some, the vomited matter is watery or bilious. *This stage lasts from a few hours to two or three days.*

Then follows the *second stage*, or *stage of calm*, in which there is a decline in the fever and of the other symptoms generally. This may be the beginning of convalescence in the mild cases. But in severe cases this stage is of short duration—*from a few hours to one or two days.*

Then the *third stage*, or *stage of febrile reaction*, sets in, lasting one, two, or three days. The temperature now rises again, *although the pulse may continue to fall;* the nausea and vomiting return—the latter becomes again hemorrhagic and may be accompanied by abdominal pain. Black and offensive stools occur. Jaundice, if not previously present, now makes its appearance, the tongue becomes dry and brown, and there may be bleeding of the gums—indeed, from all the mucous membranes. To albuminuria may be added *hematuria*. The strength rapidly fails, the pulse grows weaker, there is nervous trembling, suppression of urine, mental wandering, convulsions or stupor, and death.

Such, however, is not always the termination, even when there has been "black vomit." The symptoms may all gradually subside and the patient recover, although the jaundice may persist for a long time. In mild cases the calm stage, as stated, may be succeeded by convalescence.

Guitéras[1] regards as the three characteristic symptoms of yellow fever: First, the *facies*, including especially early jaundice. Second, *albuminuria*, which, he says, is rarely so early in other fevers, unless of an unusually severe type. "Even in the mild cases, that do not go to bed— cases of 'walking yellow fever'—on the second, third, or fourth day of the disease albuminuria will show itself," though it may be quite transient. Third, a peculiar *slowing of the pulse*, with a steady or even rising temperature. This symptom was first pointed out by Faget, of New Orleans. It is noted more particularly on the second or third day, when the fever is still keeping up, that the pulse begins to slow, dropping as much as 20 beats, while the temperature has risen 1 1/2° to 2°. On the evening of the third day there may be a temperature of 103° to 104° F. (39.4° to 40° C.), with a pulse running from 70 to 80. During defervescence the pulse may become still slower—down to 50, 48, 45, or even 30.

Diagnosis.—The three characteristic symptoms of Guitéras above

[1] "Diagnosis of Yellow Fever." "U. S. Marine Hospital Reports for the Fiscal Year 1898."

pointed out should be borne in mind, viz., facies, early albuminuria, and slow pulse. As to differential diagnosis, yellow fever is most likely to be confounded with severe fever of *malarial remittent* type. Indeed, the resemblance is sometimes very close, especially when the latter is accompanied by hematuria. But the remission occurs earlier in remittent fever and the chill is of much longer duration, while the presence of Laveran's plasmodium in the blood settles the question in favor of the latter. *Acute yellow atrophy* of the liver is a disease more insidious in its approach and less febrile. The urine in acute yellow atrophy is loaded with bile.

Relapsing fever resembles yellow fever only in the symptoms of the relapse, but this occurs much earlier in yellow fever. The presence of the spirochæte in the blood of relapsing fever makes the diagnosis. The similarity of the mild forms of yellow fever to *thermic* fever has been emphasized by Guitéras.

Dengue, or break-bone fever. The question is one which presents difficulties, for both jaundice and hemorrhage, including black vomit, have been in the past credited to dengue, while in the disputed cases black vomit, at least, was wanting. In favor of yellow fever were the authoritative names of Guitéras and H. A. West, of Galveston. The following table of contrasted symptoms was kindly prepared by H. A. West:

Yellow Fever.	Dengue.
1. One febrile paroxysm, characterized by a steady rise and lasting usually about three days. The temperature rises rapidly, the acme is often reached within a few hours from the onset.	1. Usually one febrile paroxysm, but sometimes two, a steady rise of temperature until the acme is reached; a short stadium, followed by a remission, then not infrequently a second rise. Duration four to eight days.
2. The pulse rate is characterized by abnormal slowness and want of correspondence with the temperature; while the latter is rising from three to four degrees the pulse continues to diminish in frequency.	2. The pulse usually increases in rapidity with rise of temperature, though an abnormally slow pulse may sometimes be observed.
3. There are cutting pains through the forehead, the eyes ache, the muscles of the back, loins, thighs, and calves are sore and often ache severely, even in mild cases. The pain is muscular rather than articular.	3. Headache is more or less intense, pains in the limbs and back are severe and apparently involving the bones and joints. The latter are not only painful and stiffened, but in many instances swollen.
4. There is no glandular involvement.	4. The lymphatic glands are enlarged with varying degrees of frequency in different epidemics.
5. The face is turgid, not infrequently a dusky red. The upper eyelid is often swollen. The appearance is that of typhus or of measles before the eruption, with the addition of slight or well-marked jaundice. The conjunctivæ are congested and shiny with a slight yellow tinge, the eyes sometimes intensely red and sensitive to light. The jaundice becomes more distinct after the first or second day, the skin showing the same combination of capillary stasis with an icteroid hue as the eyes. As the case progresses, jaundice may become intense.	5. The face is generally flushed, the eyelids swollen, the eyes injected and watery; there may be a slight jaundice, but this symptom is extremely suspicious of yellow fever.
6. The tongue is whitish in the center with red tip and edges, and is pointed; gums swollen and disposed to bleed. Epigastric tenderness and pain, nausea and vomiting are common; in the stage of depression black vomit is not infrequent; it is alarming and often of fatal import.	6. The tongue at first is covered with a white fur; it is swollen and the edges are red, and as the case progresses the coating increases in thickness and becomes a dirty yellow. In many cases there is nausea, but vomiting is rare.

7. Eruption absent, or extremely rare and insignificant.	7. An eruption occurs in quite a large number of cases; it may be a simple erythema or resemble that of scarlatina, measles, lichen, or urticaria.
8. Urine scanty, albumin usually found within seventy-two hours; there may be only a trace in the evening urine. In the second stage albumin may be abundant and accompanied by all the evidences of a severe nephritis, the presence of casts, hematuria, disposition to anuria and uremia. *In every severe case nephritic complications dominate the clinical picture.*	8. The urine, except in rare instances, is free from albumin; if present at all, it is evanescent. There is no evidence whatever that serious kidney complications belong to the pathology of dengue.
9. Tendency to hemorrhages common, from nose, gums, bowels, uterus, kidneys, and stomach, the last often fatal.	9. Hemorrhages from mucous membranes, nose, gums, intestines, uterus and kidneys not infrequent, but rarely of serious import.
10. Disease often fatal.	10. Prognosis proverbially favorable.
11. One attack protects from another.	11. One attack does not protect from another.
12. Not protective against dengue.	12. Not protective against yellow fever.

Prognosis.—Yellow fever is a grave disease, and in its severe forms one of the most fatal of the infectious diseases. The mortality ranges from 15 per cent. to 85 per cent. Among the dissipated, the worn-out, the poor, and in hospitals the mortality is higher; it is less in the colored race. "Black vomit" is not necessarily a fatal symptom. Many malignant cases terminate in a couple of days. Modern studies go to show the ravages of yellow fever will be greatly diminished in the near future, emphasized by the statement of Gorgas's work on the Isthmas of Panama. Here the yellow fever no longer exists.

Treatment.—Prophylaxis is more efficient than direct treatment, and modern etiology has overthrown rules formerly supposed well established and resolved it chiefly into (1) Guarding nonimmunes against infection by the mosquito. (2) Screening the house of the infected person against the insect in order to prevent the spread of the disease. (3) The destruction of as many mosquitoes as possible, by drainage, by covering breeding places with insecticides and larvicides, the best of which is petroleum. (4) Depopulation of infected places—that is, the removal of all susceptible persons whose presence is not necessary for the care of the sick. Early diagnosis is of the utmost importance in a prophylactic measure. The mosquito can become infected only by biting a patient during the first three days of illness, hence every suspicious case must be protected against mosquitoes at the very earliest moment. There is no specific treatment for yellow fever, and the symptoms are to be met as they arise. The practice quite general in the Southern United States to give an initial dose of castor oil is justified. Some prefer calomel 5 to 10 grains (0.33 to 0.66 gm.); others compound cathartic pills, 1, 2, or 3 at a dose followed, if necessary, by a saline such as cold citrate of magnesia, Epsom salt, or Glauber salt. The last is preferred by some who have had the longest experience. These measures are followed by efforts to cause perspiration, in which the hot foot-bath is included. We may seek to stop vomiting by ice internally and externally and hypodermic injections of morphin, by cold dry champagne and cold effervescing waters. Food should be withheld for from three to five days, and then be of the simplest kind, of which a mixture of equal parts

of milk and Vichy is the type. The hemorrhagic tendency may be combated by astringents, including iron. Washing out the rectum by warm water and soap enemas, carried high up in the bowel, is highly recommended by Marine Hospital Surgeon H. D. Geddings.[1] Two or three pints must be used. Normal salt solution may be thus used with a view to its being retained and absorbed.

The failing strength is to be supported by alcohol, strychnin, and digitalis; the high temperature reduced by sponging and cool baths. Nutrient enemas are to be relied on when vomiting is uncontrollable.

The following line of treatment laid down by Surgeon-General Sternberg appears to have been especially satisfactory in cases treated in United States, Cuba, and Brazil, with a mortality, according to Carroll, of only 7.3 per cent. In addition to sodium bicarbonate 7 1/2 grains (0.5 gm.), mercury bichlorid 1/60 grain (0.001 gm.) every hour, he advises a hot mustard foot-bath during the first 24 hours, cold sponging, cold applications to the head, protection from currents of air, sinapisms over the stomach and lumbar region, the promotion of perspiration, withholding of food during the first three days, and stimulants, in the form of iced champagne or good brandy, after the fourth day. If the stomach be irritable he advises milk and lime water, and if these do not agree, nutrient enemas. Later on he allows milk punch, ale, porter, etc.

Good judgment should be exercised in discriminating against the overuse of drugs.

DENGUE.

SYNONYMS.—*Break-bone Fever; Dandy Fever.*

Definition.—Dengue is an epidemic, infectious, disease, characterized by paroxysms of extreme pain in the joints and muscles, accompanied by fever and sometimes eruptions on the skin.

Etiology.—Dengue spreads extremely rapidly by the routes and means of travel. It attacks both sexes and all ages, regardless of season, although warm climates are its natural habitat, and it is rather more common in summer. It is not usual to have more than one attack. No morbid anatomical changes have been found associated with the disease.

Symptoms.—Dengue is usually sudden in its onset, after a *period of incubation* lasting from three to five days, at the end of which there may be some *sense of discomfort*, more frequently there is not, *headache*, and even *chilliness*. Suddenly, often at night, the patient is struck with *pain* in the muscles and joints, and especially the muscles of the back and loins. The pain is searching, as though extending into the bones themselves. The small as well as the large joints are affected, and the pain is aggravated on motion. The suffering is extreme, and it may be said that the patient is literally racked with torture.

Simultaneously there are *headache* and *fever*, the former serve and the latter quite high, rising rapidly to 102°, 103°, 105° F. (38.9°, 39.4°, 40.5° C.), and even 106° or 107° F. (41.1° or 41.6° C.), reaching its maximum

[1] "United States Marine Hospital Reports for Fiscal Year 1898."

from the second to the fourth day, then declining, reaching the normal about the fifth day. The face is flushed, the conjunctivæ are congested, commonly less so than in yellow fever; the pulse is frequent, 100 to 120, rising and falling with the fever. *Delirium is not a marked feature*, save in children. The *tongue* is *coated* and *red* at the *tip* and *edges;* there are *loss of appetite, slight nausea*, and extreme *thirst, scanty urine*, and *constipation;* at times, however, the urine is copious and clear. *Hemorrhage* from the nose and gums has been noted, and both Eugene Foster and D. C. Holliday have seen *black vomit* similar to that of yellow fever; and in one case copious hemorrhage from the bowels, which persisted three months and terminated in death, was observed.

The paroxysm lasts *three or four days*, at the end of which the temperature falls, the pain subsides, and a short period of comparative comfort, though one also of great prostration, succeeds that of great suffering. It is during this remission that an *erythematous rash* makes its appearance on the face, neck, and shoulders, and thence over the whole body in two or three days. At the same time the *lymphatic glands* at the back of the head and neck, in the axillæ and groins, swell, with some return of fever. The eruption is not constant or always uniform. It lasts from a few hours to a couple of days, when it subsides with the beginning of the second febrile movement, which is milder and shorter, after which true convalescence sets in. The eruption may also reappear, though rarely.

Diagnosis.—The resemblance of dengue to *yellow fever* has been referred to under the latter disease, where, too, the two conditions are contrasted. On account of the joint involvement, associated, as it often is, with redness, dengue has not inexcusably been mistaken for *acute rheumatism;* but the decided remission in three days, the altogether short duration of the disease, and its epidemic character, should soon resolve all doubt. The absence of any glandular swelling or eruption in rheumatism and the more close limitation of the pain to the joints aid in the discrimination.

After rheumatism, influenza is perhaps the next disease with which dengue may be confounded. It, too, is epidemic, and is attended often by extreme and sudden muscular pains, but the sudden intermission characteristic of dengue does not occur in influenza, nor does the eruption or glandular swelling.

Prognosis.—Notwithstanding the extreme suffering, recovery is the invariable rule.

Treatment.—Nothing can be done to cut short the disease. The most satisfactory method to control the pain is by the hypodermic injection of morphin and atropin. One-fourth grain (0.016 gm.) of the former and 1/150 grain (0.00044 gm.) of the latter may be given, supplemented by phenacetin, acetyl-salicylic acid and antipyrin, in doses of 10 grains (0.66 gm.) of the former and 5 grains (0.33 gm.) of the latter, every two hours, when the hypodermic injection may be repeated if relief has not been obtained. The coal-tar derivatives are also the best remedies for the fever, but they may be supplemented by sponging with cool water, or the cold bath in extreme cases. Prostration must be met by alcoholic preparations.

CHOLERA.

SYNONYMS.—*Cholera asiatica; Cholera algida; Cholera maligna; Cholera infectiosa; Epidemic Cholera.*

Definition.—Cholera is an acute infectious disease cause by a toxine evolved by a pathogenic organism known as the comma bacillus or spirillum of Koch, named after its discoverer. It is characterized especially by vomiting, purging, painful cramp, and collapse.

Etiology.—It is now generally acknowledged that cholera owes its existence to the comma bacillus or spirochæte, a semispiral rod-bacillus discovered by Koch in 1884. It is thicker, but not more than half as long as the tubercle bacillus. Sometimes, by the apposition of two bacilli, an S- or a corkscrew-shape is produced. Its multiplication is favored by heat, moisture, and filth. It is easy of destruction, even by weak acids and a temperature of 140° F. (60° C.). It can produce cholera only when it is taken in by the stomach, whence it quickly passes into the intestine, where the alkaline reaction of the secretions favors its multiplication in enormous numbers. Bacilli are rarely found in vomited matters, but are numerous in the fecal discharges, and are found in the intestines after death. They may invade the follicles and intestinal wall, but some time is required for this, and such invasion does not occur in cases speedily fatal. Nor has the comma bacillus been as yet isolated from the blood. The symptoms are caused by an endotoxin which is liberated after solution of the bacterial cells. Koch's conception is that cholera is an acute infectious process of the intestinal epithelium and the general condition is an acute intoxication. He believes that the primary intoxication comes from the organisms which have penetrated between and beneath the epithelial cells and here have undergone solution.[1]

Medium of Infection.—Drinking-water and contaminated food are the acknowledged media through which the bacillus is commonly introduced into the human organism, but it may be conveyed in clothing or food, on the hands, and may even enter the mouth while floating in the air, or it may be spread through the medium of flies. The postal service is regarded as a means of infection. It frequently follows in the train of moving masses of human beings, such as emigrants and pilgrims, but it prefers the sea-level and lower altitudes, especially less than 100 feet (305 meters) above the sea.

Anything that enfeebles digestion favors its permanent lodgment and multiplication. Hence, general ill-health, fatigue, the alcoholic habit, depression of spirits, fright, or anxiety, any one or all may be predisposing causes. All ages and sexes are liable to be infected, but young children seem most vulnerable.

Morbid Anatomy.—The appearance of a man dead of cholera may present no peculiarity. More commonly, there is a shrunken aspect of the whole frame, the skin of the exposed and nondependent parts is gray or ashen hued, while the dependent portions are livid. The eyes are deeply sunken, the temples hollow, the nose is pinched, and the skin clings

[1] "Ricketts and Dick, Infection Immunity and Serum Therapy."

closely to the bones beneath it. The appearances of such a body, in brief, are those of a wasted cadaver long immersed in the pickling vats of the dissecting room.

Very striking are the postmortem elevations of temperature and the phenomena of postmortem muscular contraction. The former has reached 109° F. (42.8° C.) and higher. The latter include movements of the lower jaw, rotation of the eyes, contraction of the arms and legs, sometimes startlingly life-like.

On section of the body the subcutaneous tissue is found dry, the blood in the vessels thick and dark. The condition of the stomach and bowels differs somewhat with death at different stages of the disease. If death takes place early the *stomach* is commonly, but not always, filled with a turbid liquid grayish-white in color, resembling rice water. In this the microscope may recognize columnar epithelial cells, isolated and in flakes; also the remnants of partially digested food, such as disintegrating muscular fasciculi and oil globules. The *mucous membrane* of stomach appears *congested*, and the course of the larger vessels can be readily traced in consequence of their being full of thick blood. A *papillated* appearance ascribed to enlargement of the solitary follicles is often present. The epithelium is detached in places; in others, intact.

The mucous membrane of the *small intestine* may also be much congested; the bowel is filled with rice-water fluid. On its surface lie numerous patches or flakes of detached epithelium, while the papillated appearance produced by the enlarged lymphadenoid follicles is everywhere present. The villi are largely denuded of epithelium, but in places they are intact.

If death takes place during imperfect reaction, the gastro-intestinal *mucous membrane* is still more *congested* and dark-red in color from hyperemia and *blood extravasation*. At such times, too, the solitary glands are conspicuous and cause also a *papillated* appearance even more striking than that in the stomach. Peyer's patches may also be swollen, and the same denudation of epithelium from the villi and elsewhere is present. The signs that suggest an inflammatory process are a slight cellular infiltration of the intestinal walls and the enlargement of the solitary follicles; also, at times, a diphtheritic exudate.

The *liver* is natural in size, but may be congested and darker hued than in health, while the cells exhibit cloudy swelling, and in places small areas of fatty change. The *spleen* is usually small, certainly not enlarged.

The condition of the *kidneys* varies with the stage at which the patient dies. If early in the disease the organ, superficially, is not much altered; it may be somewhat enlarged. The veins are slightly overfilled, but there is no marked capillary injection. There may be a few white or yellowish patches, where the epithelium is found compressed, cloudy, and fatty. The lumina of the tubes may, in places, be blocked with granular matter or well-formed casts, and there may be a few hemorrhagic foci, the changes starting from the pyramids.

If death takes place later, after reaction has set in, the kindey is enlarged. In the cortex are seen grayish-white and yellow patches, alternating with normal-hued portions. In these altered places the tubes are opaque with granular and fatty débris. Hemorrhagic infarcts may also

be found in the cortical substance. The Malpighian capsules, with their included glomerular capillaries are intact.

The *heart* is normal in size, but its walls flaccid. The right cavities are commonly filled with dark, liquid blood; the left cavities, empty.

In many instances the *lungs* also present an appearance more or less characteristic, being shrunken and small, lying back in the thorax, as though collapsed. Like the other tissues, they are empty of blood except in their dependent portions, which are the seat of hypostasis. They have been compared by Parkes to fetal lungs. Sutton found the two organs to weigh but 20 ounces (600 gm.,) as compared with 45 ounces (1350 gm.), when death occurred after reaction had been established—that is, after the blood had again occupied the pulmonary artery and its branches. Collapse may be interfered with by adhesions, in which event it is only partial.

Such appearances could, of course, occur in death from hemorrhage and, after all, the only distinctive condition is the presence of the rice-water fluid in the stomach and intestine, or in both, containing the "comma" bacillus and desquamated epithelium. The latter, to which the earlier descriptions attached great importance, is now generally regarded as *postmortem* in origin. The flakes thus produced are also what the older authors described as patches of lymph.

Symptoms.—After a *period of incubation* ranging from 36 to 56 hours, rarely five days, the symptoms of cholera commonly present themselves gradually enough to permit of arrangement into three distinct groups or stages:

1. The stage of preliminary diarrhea.
2. The stage of collapse.
3. The stage of reaction.

The stages are by no means always recognizable, and the severity of the symptoms varies greatly, such variations being reasonably ascribed to the varying quantities or virulence of the specific poison. Mildness in a given case is no guarantee against virulence in another caused by it.

1. *The stage of preliminary diarrhea* is characterized by moderate diarrhea, which is characteristically painless, but may be associated with colicky pains. The stools are yellow or yellowish throughout this stage, and are alkaline in reaction. Nausea and vomiting are not usual in it, and the patient may feel but slightly indisposed. There is generally a feeling of restless discomfort and depression, to which headache may contribute. The temperature remains normal. The first stage may last for a week or longer, or a few hours only, or it may be entirely absent.

2. *In the stage of collapse* the diarrhea has become profuse. The discharges have lost their yellowish color and resemble thin gruel or rice-water. The fluid gushes out with great profuseness and apparent force. There may be griping or tenesmus, but more characteristic are the very painful muscular cramps, which usually begin in the fingers and toes and extend thence to the calves of the legs and abdominal walls. Vomiting, bilious at first, is soon added to the diarrhea. The fluid vomited soon assumes the rice-water character, and gushes from the mouth as from the bowel, in enormous quantities.

Extreme weakness and exhaustion are by this time present. The skin is blanched and shrunken, the lusterless eyes are sunken and bounded below by great circles of blue. The nose is pinched, the lips are thin, the cheeks hollow, and the countenance pallid to bluish grayness. The extremities and entire body become clammy and cold, the superficial temperature falls 5° or 6°, while that of the rectum rises to 103° and 104° F. (39° and 40° C.). There is intense thirst, the mouth is dry, speech is husky, whispering, and labored. The pulse is feeble, frequent, or absent at the wrist, and the patient appears to be dying. Even the heart-beat and sounds are almost gone, but the breathing continues. Through all this, conciousness may be maintained to the end or coma may supervene. Death commonly occurs in this stage.

On account of the scantiness of blood certain secretions cease and there is neither urine nor saliva, while power to perspire and even the lacteal secretion in nursing women, remain.

A closer examination of the rice-water vomited matters and bowel discharges reveals flakes of epithelium, mucus, and granular débris, and, with sufficiently high powers and suitable preparation, the cholera bacillus together with numerous other bacteria. Occasionally a little blood is present. The fluid is albuminous and contains the salts of the blood, among which sodium chlorid is conspicuous. Sometimes, however, there may be no vomiting or purging, whence the term *cholera sicca*. In these cases, however, the stomach and bowels are commonly found containing the characteristic fluid after death.

This second stage is generally of shorter duration, commonly a few hours only, but it may be prolonged to 12 or 24. The disease is sometimes ushered in with the symptoms of this stage. It has been ascribed to the action of a toxin produced by the bacilli, which, when absorbed, produces the systemic effects of this stage, but it is likely that the flux is the principal factor in its production.

3. *The stage of reaction* is characterized by the return of warmth and color, the latter more slowly, and the re-establishment of secretions. Especially favorable is the return of the urinary secretion. Along with these changes the vomiting and purging occur at longer intervals. Such improvement is, however, often delusive. The diarrhea may return, the collapse repeat itself, and the patient die. Or there may supervene *cholera typhoid*, a state characterized by a frequent, feeble pulse, dry tongue, delirium, and sometimes an *erythematous* or *roseolar eruption* on the extremities. This may end in recovery. Or there may be superadded symptoms of *nephritis*, including uremia, coma, and death. Or there may be *inflammation*, diphtheritic or catarrhal, of the bowels.

Diagnosis.—In the matter of diagnosis it is well known that, so far as symptoms are concerned, cases of acute enteritis, have occurred with symptoms absolutely identical with those of true cholera, including the fatal termination.

There is one very important *etiological difference* between enteritis and true cholera, which is also of great diagnostic value, and that is that almost invariably cholera morbus is traceable to a severe and irritating exciting cause, such as a meal of indigestible fruits or vegetables, or

imperfectly cooked or decomposing fish or shell-fish, while cholera comes on without any such cause, or succeeds trifling derangements of digestion, which in other than cholera seasons pass away without harmful results. As a rule, too, the symptoms of enteritis are much more severe at first than those of true cholera, and the substances first vomited are undigested articles that have acted as exciting causes, succeeded by green, bilious matter. The discharge from the bowels is first also of a more bilious character, and above all, the mortality is much less serious; indeed, recovery is the rule. Yet these differences are not to be relied upon.

By bacteriological investigation only can a given case be identified with absolute certainty. The agglutinative reaction is a ready method. It is similar to the Widal test for typhoid fever, the blood-serum of the infected case agglutinates the comma bacillus. Some hours are, however, necessary to complete such a bacteriological diagnosis.

As to the microscopic examination of the dejecta, which is more feasible for the practitioner, it may be said if the examination reveals a preponderance of curved bacilli, comma-shaped, and sometimes joined end to end, so as to form figures somewhat resembling the letter S, and again appearing in long threads, we may feel justified in considering the case one for careful study by bacteriological methods. Although there are found in the alimentary tract other bacilli, the morphology of which is much like that of the cholera bacillus, they are not numerous.

How, then, shall we know a case of vomiting, serous diarrhea, severe colicky pain, followed by collapse, to be a case of cholera? Every suspected case should at once have an agglutination test made, and the stools should be examined for the comma bacillus. In this country, where such a thing as endemic cholera is unknown, it goes without saying that any isolated case, even if fatal, cannot be one of true cholera unless there be traceable some connection with an acknowledged focus of cholera elsewhere. Second, such communication must have taken place within the period of incubation required for the development of the case, say within six days. Of course, such communication need not be a personal one. It may be by clothing, merchandise, and possibly letters.

These conditions being fulfilled, the patient suffering with the symptoms of cholera must, for the time being, be regarded as a case of the true disease, and isolated until the bacteriological investigation can be made, but the rapid occurrence of similar cases increases the probability of its being true cholera, and finally establishes its certainty.

Symptoms similar to those of cholera arise from poisoning by corrosive sublimate, tartar emetic, arsenic, mushrooms, and ptomains from various sources, but their symptoms are rarely confounded with those of cholera.

Prognosis.—The prognosis, always grave, varies with the stage of the epidemic. It is well known that in the beginning a very large proportion of cases die, fully 80 per cent., but as the epidemic is prolonged the ratio of deaths to persons attacked grows less, the mortality falling to 30 per cent. or less. The habits and morals of the patient have an important influence. Intemperance and dissipation diminish greatly the powers of resistance, as do also fatigue, indigestion, fright, and fear.

Treatment.—The treatment of cholera is very appropriately divided

into prophylactic and curative; the former, when properly carried out, being more effectual than the latter.

Prophylaxis.—It consists mainly in the isolation of the patient and in certain precautions against the spread of infection by sterilizing the discharges. To this end:

1. The vomited matter and the discharges from the bowels are to be gathered in carbolic solution, 1 to 20, or chlorinated lime, 1 to 10, some of which should be in the vessels before it is used. After use, more should be added. The matter thus collected should be gently stirred and allowed to remain 20 minutes before being poured into the water-closet hopper. When the excreta can be thrown into a pit, or even, as may be done in the country, on the manure pile, milk of lime, or what is the same thing, ordinary whitewash, is a very efficient and cheap medium with which to disinfect them.

2. After vomiting, the mouth of the patient should be rinsed with a solution of hydronaphthol, 1 to 5000, care being taken that none is swallowed. After each evacuation from the bowels, the buttocks, thighs, and anus should be washed with soap and water.

3. All body and bed linen soiled with the discharges should be immediately moistened with carbolic solution, 1 to 60, and removed in a covered vessel from the apartment, placed in a wash-boiler, and boiled for half an hour in a one per cent. solution of washing soda.

4. Napkins, towels, and table linen should be placed in a similar vessel or canvas bag for removal and similarly boiled.

5. All dishes, knives, forks, spoons, etc., used by the patient should be boiled after each meal in a one per cent, solution of soda.

6. The remains of meals should be thrown into a vessel containing milk of lime or whitewash, and removed at the end of the day.

7. Door-knobs are liable to be soiled by the hands of one carrying out excreta, and should be carefully washed and sterilized, lest they, in turn, communicate the infectious material to another person.

8. In case of death, the body, without being washed, should be wrapped in sheets wet in a solution of bichlorid of mercury, 1 to 1000, and allowed to remain until removed for prompt burial.

Special Directions to Nurses:

1. They should, under no circumstances, take their meals in the same apartment with the patient, and before leaving the room the hands should be cleansed with soap and bichlorid solution, and such portion of the dress as is liable to be soiled should be changed. The hands should be again rinsed in bichlorid solution, 1 to 1000, after leaving the patient's room. A very convenient plan is to wear a slip or "overall" with a hood to cover the hair, which can be easily thrown aside before leaving the room. A canvas slipper or overshoe, readily removed, should also be worn in the sick-room.

2. The food of the nurse should be wholesome and plain, freshly cooked, and served hot. No uncooked vegetables should be eaten. Milk should be boiled and, if desired, cooled before using. Cold drinks should be taken moderately, if at all. Coffee and tea may be taken hot.

3. Teeth should be cleansed after each meal, as the mouth affords a peculiarly favorable nidus for decomposing matters and a favorable nidus for the multiplication of pathogenic fungi. A daily bath in warm water, with the use of soap, should be taken by each nurse.

4. Care should be observed to keep the body from being chilled by drafts or other cool exposures, and to this end woolen underclothing should be worn.

5. All nurses should wear rubber gloves and a gowns.

6. Courage and cheerfulness are amply justified, because it is really almost impossible to take cholera if the above precautions are carried out.

During an epidemic the public must be warned to boil all drinking water and all milk. To seek relief for the most trifling diarrhea.

It early appeared that a certain degree of immunity from cholera is secured by a first attack. This was also the conclusion of a collective investigation directed by the Academy of Medicine of Paris in 1884, and by Edward O. Shakespeare from information collected by him during his residence in Spain in 1885. From this standpoint Ferran and others sought to secure immunity by vaccination with protective virus. Ferran injected subcutaneously into each arm of the subject, 1 c.c. of a pure culture in bouillon of the *comma* bacillus, during the epidemic of 1885, in Spain. Notwithstanding the discouraging results of Ferran's method that of Haffkine, which proved successful, is essentially the same.

The vaccination of human beings is done in two stages. In the first .05 to .1 gram of a 24 hour agar tube of an attenuated culture, suspended in bouillion is injected under the skin. Three to eight days later the same amount of vrulent or fixed virus is inoculated. Only a slight local reaction is said to follow the injection of the attenuated culture, which modifies the reaction of the second. When thus injected the microbes die and disappear, setting free a substance which acts upon the organism and confers immunity on it. The same result follows the injection of their dead bodies only. Thus he was enabled to prepare vaccine, preserved in weak solutions of carbolic acid, which remains efficacious for six months, and may be used by persons without bacteriological training.

According to Haffkine[1] 70,000 inoculations against cholera were made in India on 42,179 persons, without a single accident which could be ascribed to the inoculations, and he regards the results as eminently satisfactory in cases where the vaccination is properly carried out.

Notwithstanding the claims of Haffkine it is held by others that the discomforts and more serious results which ensue are a drawback to its use.

The Treatment of the Attack.—Before any general plan is followed the intestinal tract must be freed by castor oil or calomel. The indications in the management of cholera, apart from isolation of the patient and the sterilization of the discharges, are, in the *first stage*, to combat the multiplication of bacilli and neutralize their toxic influence. In the *second stage*, to relieve the cramp and pain and check the flux.

I.—The former is to be attained by the judicious use of opiates and

[1] See a lecture by W. W. Haffkine on "Vaccination against Cholera" in "Baumgarten's Jahresbericht." vol. xi, 1895, p. 411.

acids on the one hand or opiates and antiseptics on the other. Any of the mineral acids, such as hydrochloric, nitromuriatic, and sulphuric acids in doses of 10 to 15 minims (0.66 to 1 c.c.) of the dilute acid with as much tincture of opium or a corresponding dose of paregoric or deodorized tincture of opium properly diluted, may be given every two hours. Or a lemonade of tartaric or citric or lactic acid, 2.5 drams to 1 quart of water (9.5 gm. to a liter), may be used in conjunction with the opiate. In addition, the rectum may be washed out by the warm solution of tannic acid in water or camomile tea.

Instead of the acid solutions, antiseptics may be given for the same purpose. Of these, salol is a favorite, and may be given in doses of 10 to 15 grains (0.66 to 1 gm.) every two or three hours, and it may be combined with subnitrate of bismuth in large doses, with wine of opium or deodorized tincture.

The greater or less usefulness of calomel in cholera, as attested by experience in so many epidemics, beginning in 1885, may be ascribed to its antiseptic qualities, although it is probably as efficient in controlling vomiting as any other drug. The plan pursued at the New Hamburg Hospital and at the Moabit Hospital in Berlin was to give an initial dose of 4 to 7 grains (0.3 to 0.5 gm.), after which 1/3 to 3/4 grain (0.02 to 0.05 gm.) was given every two hours through the first and second stages. A portion of the calomel becomes changed in the intestine to corrosive sublimate; and as corrosive-sublimate solutions have a fungus-destroying action, in a strength of 1 to 30,000, it is reasonable to suppose that the bacilli in the intestine are directly killed by the calomel.

II.—The indications in the *second stage* are to relieve the painful cramp, to continue to try to check the discharges, and to compensate for the loss of liquid by the vomiting and purging.

For the relief of cramps morphin hypodermically is to be preferred, because of the promprness of its effect and because absorption from the gastro-intestinal mucous membrane is much hindered, if not altogether prevented, in true cholera, while the vomiting is a further obstacle to the administration of medicine by the mouth. Full doses should be given, 1/6 to 1/4 grain (0.01 to 0.016 gm.), which may be repeated, if necessary. If circumstances compel the administration of anodynes by the mouth, chlorodyne is one of the best, and is well administered in brandy or whiskey. Such administration, too, fulfills any indication for opium to control the bowels. Some difference of opinion exists as to the propriety of checking the discharges in this stage, the chief reason assigned being that the bacilli, whose presence is directly or indirectly the cause of the flux, are thus retained. But such objection is offset by the fact that the flux itself is the greater source of danger and that, if it can be controlled, the bacilli in the bowels are comparatively harmless. Unfortunately, in the later stages, when the flux is established, nothing avails to control it, and the opiate may as well be limited to that hypodermically administered for the relief of pain. We quite agree with those who hold that, notwithstanding the opposition to it, opium will retain its place among the chief weapons against the disease.

The effect of the copious discharge is to produce the intense exhaus-

tion referred to under symptomatology, and it is imperative to counteract this, if possible, by stimulants freely administered. Champagne, brandy, and ammonia, combined with ice and carbonated waters, are suitable. If not retained by the stomach, whiskey, ether, and the aromatic spirit of ammonia may be given hypodermically in 30 minim (2 c.c.) doses frequently repeated. The hope of benefit from these remedies is justified, if reaction once sets in.

More serious still is the drainage of liquid from the tissues, and the most serious consequences ensue from the resulting stagnation in the blood. To restore the water is, therefore, of the greatest importance. Intraveneous injections of normal salt solution are of the greatest value. More easy in private practice are hypodermic injections of hot saline solutions or hypodermoclysis, also enemas or enteroclysis of similar fluids, slightly astringent. They were practised successfully by Cantani in Italy in 1892, and have been continued with various results in Europe, and in a more limited manner, with with satisfactory results, at Swinburne Island in New York Harbor. The benefit derived from the use of this measure under other circumstances—as, for example, succeeding large hemorrhages and uremia—together with the facility with which it can be carried out, commend it strongly. A heaping *teaspoonful of common salt to a pint of sterilized water furnishes with sufficient accuracy the proportion desired.*

Whenever the discharges have been so copious as to make it reasonable that the vessels are becoming drained, hypodermoclysis is indicated, and may be repeated every two, four, or six hours as required.

Enteroclysis is made with a one or two per cent. solution of *tannic acid* at a temperature of 113° F. (45° C.). For an adult 2 quarts (2 liters) may be administered; for an adolescent, 1 quart (1 liter). It is introduced slowly, by a fountain syringe, through a rectal tube with lateral outlets but closed at the end. The tube is inserted gently by a combined rotary and pushing motion to the depth of ten inches, when the fluid is allowed to enter very slowly, consuming not less than ten minutes. The patient should, of course, be encouraged to retain the fluid, and may be aided by pressure on the anus with a napkin. Enteroclysis is useful in any moderately severe case of cholera, and may be given night and morning, more frequently in severe cases.

It is in the algid stage that this treatment is especially useful, but other means must be taken to keep up the warmth of the body. To this end the patient is immersed in the hot bath at a temperature of 38° to 42° C. (100° to 107° F.). In favorable response the warmth of the body returns, the pulse is fuller and stronger, the respiration deeper. Hot-water bottles, hot-water bags, and hot bricks may be applied alongside the body.

III.—In the *third stage*, that of reaction, indicated by the return of warmth, pulse, and heart-beat, and especially the establishment of the urinary secretion, restorative measures are continued with the addition of judicious nutriment, preferably in the shape of peptonized foods, especially peptonized milk. Great care must be exercised lest diarrhea be induced by too liberal feeding. Convalescence is necessarily very slow in serious cases, and relapses are prone to occur.

DYSENTERY.

SYNONYM.—*Bloody flux.*

Definition.—The term dysentery, derived from the Greek words δυς difficult and Εντερον bowel is applied to inflammations of the large intestine, sometimes extending into the small bowel, which are infectuous in character. The condition can be best considered under two heads which represent varieties or different forms of the disease. These are: 1st, bacillary, and 2d, amebic dysentery.

BACILLARY DYSENTERY.

Definition.—An acute infectious disease due to the inplantation of some strain of the *bacillus dysenteriæ*. It is characterized by sudden onset, fever, abdominal pain, sharp and cramp-like, frequent stools at first large and loose, soon small containing blood and mucus accompanied by much tenesmus.

Etiology.—Beginning with the researches of Shiga in Japan, in 1898, which were followed by the investigations of Flexner and Barker carried out in Manila, in 1900, and afterward by Flexner in this country, and Kruse and others in Germany, the evidence has grown in favor of the B. dysenteriæ as being the specific cause of this variety of dysentery.

The *bacillus dysenteriæ* of which there are several varieties, is a well-characterized micro-organism belonging to the colon typhoid group of bacilli, which can be distinguished by its cultural and other characteristics. In morphology it differs only slightly from the typhoid bacillus, with which it has certain cultural properties in common. It is highly toxic, the poisonous character being due to an endotoxin. Vedder and Duval, late of the University of Pennsylvania have demonstrated the presence of this bacillus in sporadic cases in Philadelphia and in epidemics in other portions of Pennsylvania. Hunt[1] has recently reported several epidemics of dysentery from which he recovered bacilli of one or the other of three groups of dysenteric bacilli. The organism is pathogenic for a wide series of laboratory animals, and when injected into the intestine of cats, or fed to them after alkalinization of the gastric juice, it is capable of setting up an inflammation of the gut from which the bacillus may be recovered. Ingested by man it rapidly sets up a severe colitis. There are two instances on record of its actions on man: The first, reported by Flexner, in which a small quantity of a culture was accidently aspirated into the mouth by one of his assistants, the intestinal symptoms appearing with forty-eight hours; the second, reported by Strong, in which a Filipino prisoner voluntarily swallowed a portion of a culture of the bacillus, in which case the symptoms quickly developed and were of marked severity, the bacillus being recovered from the stools. The man finally recovered.

Morbid Anatomy.—The anatomical features of bacillary dysentery vary with the form and duration of the disease. The lesions vary from a mere hyperemia of the mucous membrane of both ileum and colon to destructive ulceration of the mucous membrane more particularly of the colon, there may be a grayish pseudo-membrane which is easily scaled off covering

[1] "Jour. Amer. Med. Assoc.," September 21, 1912.

the entire mucous membrane or there may be deep localized ulceration. Occasionally the whole mucous membrane is much thickened. As the condition progresses in severity the intervening mucosa is covered with pseudo-membrane. The entire mucosa is injected, swollen, and covered with blood-stained mucus, beneath which bleeding points may be discerned. Upon microscopical examination the pseudo-membrane is found to consist of a fibrinous and cellular exudation which lies upon the surface and penetrates into the substance, for a variable distance, of the mucosa. The glands of Liberkühn undergo necrosis and become invaded by pseudo-membrane. Large numbers of micro-organisms are present in the dead tissue, and the blood-vessels of the mucosa are extensively occluded by thrombi.

A demarcating inflammation takes place at the limits of the living and necrotic tissue, causing separation of the latter, which upon exfoliation leaves behind defects which constitute the acute dysenteric ulcers. The disease may come to an end at this stage or an earlier one, and the integrity of the mucosa be restored, or the necrosis may extend more deeply and involve the death depth of the mucosa and be associated with marked inflammatory changes in the submucous and muscular tunics. In these instances ulceration may extend through the mucosa and invade the submucosa, and even penetrate more deeply, and in the subsequent process of repair new tissue develops in the submucosa which leads to the permanent thickening of the intestinal wall.

It is the form of dysentery which tends to pass into the chronic disease, the ulceration is deep and persistent, and much new tissue develops in the submucosa, in the mucosa, and even in the muscular coat. Owing to the persistence of the ulceration and possibly to the interaction of secondary micro-organisms, including the pyogenic cocci, always present in the intestinal canal of man, the ulceration extends not only more deeply, but tends also to heal slowly and imperfectly, whence arise the symptoms characterizing chronic ulcerative dysentery. That the specific organism persists throughout long periods, where these pathological conditions are present, is shown by the acute exacerbations of the disease and by the association of the chronic ulcerative with fresh pseudo-membranous inflammation met, not infrequently, at autopsy. It is during the exacerbation that the specific bacillus is to be sought in the dejecta and the blood reaction looked for.

The new formation of connective tissue throughout the coats of the gut may be so extensive as to bring about, after its contracture, serious deformity and narrowing of the lumen. Inflammation sometimes extends to the peritoneal coat, whence adhesions to the neighboring parts take place. Only rarely does ulceration proceed so rapidly, or fail to be attended by connective tissue formation, as to perforate the peritoneal coat.

Symptoms.—The symptoms of bacillary dysentery vary greatly in intensity. Those cases due to the Shiga group of bacilli have the most severe symptoms. To this class belongs the bacillary type of tropical dysentery. The *fever* is high, the *pain* is great, the *tormina* and *tenesmus* are severe, the *stools* are bloody, and the *adynamia* is profound. *Delirium* is often present, and the tongue may be dry. The abdomen is tender and swollen, and typhoid fever may be simulated.

Those cases due to the other two groups of bacilli have much milder symptoms. There is sudden abdominal pain, fever, reaching 103° or sometimes over, but usually 101° to 102°, tenesmus, frequent stools every hour to every half hour. The stools containing blood and mucus. There is much prostration and emaciation, there is frequency vomiting. This latter group of cases belong to those formerly designated catarrhal dysentery.

Complications and Sequelæ.—The complications in this form of dysentery are more numerous. *Abscess of the liver* is rare, and is ascribed to thrombotic extension from the seat of inflammation along the vessels of the portal system into the liver, or to emboli carried from the primary focus to the liver. *Perforation* of the bowel is not a very rare complication, having been found by Woodward, in a study of the statistics of the late Civil War in America, 11 times in 108 autopsies. This accident is followed by a peritonitis, which is usually fatal, the local symptoms of which vary with its exact seat. If in the neighborhood of the cecum, perityphlitis ensues; if lower down in the rectum, a proctitis. A peritonitis may also arise by extension of the inflammation from the mucous lining of the bowel.

The same opportunities enabled Woodward to show the undoubted association of *malaria* with dysentery, though it is likely that the "chills" referred to in older reports were sometimes septic and due to the dysentery. The same is true of the *joint swelling* described by the older authors, among whom was Sydenham. They may be a part of pyemic processes. *Paralysis*, commonly paraplegia, as a sequel, is attested by Woodward and Weir Mitchell. Pleurisy, pericarditis, endocarditis, and Bright's disease are among sequelæ reported.

Diagnosis.—The condition must be distinguished from a simple ileocolitis due to improper food, or drink.

Bacteriological Diagnosis.—Diagnosis of this form of dysentery can be established in two ways: First, by recovery of the specific organism from the stools; second, by obtaining the agglutination reaction with the blood of the patient and the specific bacilli in a manner similar to that of the Widal test in typhoid fever.

In the acute disease the specific bacilli are abundant, and can be separated without great difficulty from the dejecta.

The agglutination test with the blood of persons ill of bacillary dysentery is easily obtained. For this purpose cultures, 24 hours old, upon agar-agar, are employed, from which suspensions are made in bouillon. In using the blood, it is preferable to employ the wet method by which the blood is obtained in capillary tubes, from which the serum can be collected. After proper dilution of the serum the tests are carried out in the usual manner. Positive reactions may be obtained in dilutions varying from 1/20 to 1/1000 in a period of from one-half to one hour, and as early as from the third to the fourth day of illness. This method is applicable to the study of all cases of dysentery, as well as the entero-colitides of children.

Prognosis.—The prognosis depends upon the locality and the type of infecting organism. In the tropics the mortality is high and a high mortality accompanies the infection of the Shiga type wherever it occurs. In temperate climates the mortality is very low especially where the infecting

organism is one of the less virulent types. Most cases perish, death being preceded by extreme adynamia and other symptoms of the typhoid state, including dry tongue, stupor, emaciation, and the cadaveric countenance. Consciousness is sometimes painfully persistent to the end.

Amebic Dysentery (*Amœbiasis*).

Synonyms.—*Amebic Enteritis*; *Tropical Dysentery.*

Definition.—An ulcerative inflammation of the large intestine due to *amœba coli*. This form has sometimes been termed tropical dysentery. One hundred and nineteen cases were treated in the Johns Hopkins Hospital, Baltimore, Md., from the date of its opening, May 15, 1889 to 1902, a period of nearly 13 years.[1]

Etiology.—The *amœba coli* or *dysenteriæ* is now the acknowledged cause of this form of dysentery.

The amebæ are found in the dejecta, in the intestinal ulcers, and in secondary liver abscesses complicating the disease. The organism varies from 15 to 20 microns in diameter and is actively motile when examined in the living state. It consists of two portions, an outer ectosarc and an inner endosarc. Its movements are brought about through the propulsion of the former, after which the granular inner substance flows into the pseudopodia. The ameba is phagocytic, taking up foreign substances from the intestine, etc., and especially englobing the red corpuscles. At present two varieties of amebæ are distinguished as occurring in the stools: the first nonpathogenic—amœba coli mitis—and the second pathogenic—amœba coli. The former has been found repeatedly in healthy stools, and it does not exhibit phagocytic properties for red corpuscles. In this country amebic dysentery has been found to occur as a sporadic disease, especially in the Southern States, but also in Pennsylvania, New York, and the New England States. In the latter three it is probably imported.

Morbid Anatomy.—The intestinal lesions are usually limited to the large intestine; rarely they are found in the ileum. The characteristic lesion is ulceration, involving the mucosa and submucosa. In early ulcers a small defect only is found in the mucosa; more rarely the muscular coat is invaded, and rarest of all the peritoneal coat. In the course of the ulceration the submucosa becomes infiltrated with a grayish gelatinous material, the exfoliation of which gives rise to the ulcer. In this material there are a few pus cells, but it consists chiefly of necrotic material. Amebæ may be discovered in the necrotic tissue, as well as in the adjacent portions of the mucosa and submucosa. In the immediate neighborhood of the ulcer proliferation of the connective tissue takes place which, in favorable cases, may completely restore the defect, and in chronic cases brings about permanent changes in the gut similar to those described in chronic bacillary dysentery. Pseudo-membrane is never present in uncomplicated cases, but instances of combined amebic and bacillary dysentery, in which pseudo-membrane has been present, have been described.

Symptoms.—The symptoms of amebic dysentery are similar to those

[1] See Thomas B. Futcher's paper "A Study of the Cases of Amebic Dysentery," occurring at the Johns Hopkins Hospital. "Jour. Amer. Medical Assoc.," Aug. 22, 1903.

of bacillary dysentery, but much more irregular and prolonged. The onset is usually less sudden, but may be equally so. The stools are less numerous, and are apt to be more liquid and more copious. They abound in the *amœbæ coli*. The straining at stool is less severe and persistent, while there may be several days of relief, to be followed by the usual train of symptoms. The fever may be severe or mild. Intestinal hemorrhage should be mentioned as an occasional symptom of amebic dysentery. This symptom rarely has resulted fatally being caused usually by extensive ulcerative gangrenous processes which prevail in this disease. The subject has been exhaustively studied by Richard P. Strong, Director of the Biological Laboratory at Manila, P. I. (Journal of American Medicine, January 27, 1906.)

Complications.—The most common and serious complication is *abscess of the liver*, which is now believed to be due to the wandering *amœba dysenterica*, which reaches the liver through the blood-vessels. The abscess may be single or multiple. In the former case it may be of large size, involving fully half of the bulk of the liver. The multiple abscesses are smaller in size and superficial. The abscess walls are peculiar, being ragged from the presence of necrotic projections. Only occasionally, in the older abscesses, are there firm, smooth, fibrous walls. Next to the *innermost necrotic zone* is a zone of *cellular infiltration* encroaching upon and destroying the liver-cells, and external to this again a zone of *intense hyperemia*. The contents of the abscess are not pure pus. In fact, the paucity of the pus-cells here is as significant as in the inflammatory infiltration of the mucosa, indicating a similarity in the etiology. The pyoid material consists rather of fatty and granular débris and the amebæ, which are also found in the walls of the abscess. These abscesses sometimes break into the lungs, carrying the amebæ with them, which, under these circumstances, may be found in the expectoration.

In addition to the abscesses described there are found also in the liver in amebic dysentery patches of *circumscribed necrosis*, scattered through the liver as the result of the action of the amebæ.

Diagnosis.—The diagnosis is rendered easy by the recognition of the *amœba coli* in the stools, which should be examined by the microscope in every case of dysentery as directed under microscopical diagnosis.

Microscopical Diagnosis.—Detection of the specific amebæ in the stools, or of secondary liver abscesses, confirms the diagnosis of the disease. Great care should be exercised to obtain fresh material for microscopical examnation, and bits of mucus, rather than fecal material, should be chosen for study. The mucus or pus is slightly pressed out, but not too firmly, under a cover-glass, and the slide slightly, but carefully, warmed up to body heat, before examination. Inasmuch as desquamated epithelial cells sometimes take on a round form and stimulate amebæ, it is desirable that a definite movement be detected before passing upon the nature of the suspected cells. Living amebæ, especially those enclosing red corpuscles, are taken to indicate the nature of the pathological condition of the intestine.

Prognosis.—The prognosis is more serious than that in the bacillary variety. The course of the disease is always prolonged, and a fatal issue is much more frequent. It would seem that the patient must outlive the

organism before he can recover, and even then recovery is delayed by the exhausted condition into which he has fallen. When the termination is most favorable, cases of amebic dysentery last from 6 to 12 weeks.

Treatment of Dysentery.—The first measure of treatment of mild or sporadic cases of *dysentery* duly recognized should always be a purgative. No aperient is better than castor oil. An ounce of oil (30 c.c.) is the proper dose for an adult. The saline treatment, especially when there is high fever and no marked adynamia, is also efficient, working a rapid cure in many cases. Two drams (8 gm.) of sulphate of magnesium, or 1/2 ounce (16 gm.) of Rochelle salts dissolved in water, should be given every hour until copious watery purgation results. The patient should always be required to use a bedpan.

After free purgation, an opiate may be given. Plain opium in doses of 1 grain (0.066 gm.) every three hours, or 1/2 grain (0.033 gm.) of the extract, is the favorite. Bismuth subnitrate in 10 to 30 grain (0.66 to 2 gm.) doses, one of the astringents, tannic acid in 2 to 5 grain (0.132 to 0.33 gm.) doses, or the acetate of lead, 1 to 2 grain (0.066 to 0.132 gm.); or maybe salol in 5 to 10 grains (0.3 to 0.6 gm.) may be given. Very comforting in quieting rectal irritation is an opium suppository containing 1 to 2 grains (0.066 gm. to 0.132. gm) of opium, or 1/2 to a grain (0.033 gm. to 0.066 gm.) of the extract.

High enemata of normal salt solution introduced very slowly will frequently give more relief than any other measure. The solution should be luke warm, from one pint to one quart may be used. If the funnel is held not more than 15 to 18 inches above the anus the whole amount may be introduced taking about one-quarter to one-half an hour.

It goes without saying that the food should be liquid and of the blandest kind: boiled milk, better still peptonized, light animal broths, and beef-juice, not beef-teas, are the type. Barley or rice may be added to such broths, and should be thoroughly cooked.

Severe Epidemic Forms.—The first consideration in the treatment of bacillary dysentery is a *bland and nonirritating, but nourishing, diet*, one that leaves as little residue as possible. The peptonized foods, such as peptonized milk, malted milk and beef-preparations, in addition to beef-juice and somatose, are the types. To these, stimulants should be freely added. *Opiates* are needed to relieve the pain, and their hypodermic use is sometimes especially efficient for this purpose. When the necrotic membrane is removed, an extensive ulcerated surface remains to be healed. Such healing is favored by the restrained peristalsis that opium produces. The same purpose may be served by the use of ipecacuanha. Directions for its administration are given below.

On the other hand, it is uncertain whether soluble remedies intended for the direct healing of the ulcers ever reach these surfaces in an active state, when administered by the mouth. Nitrate of silver, when administered, does sometimes, however, reach the lower bowel. Bismuth, being largely insoluble when administered in large doses, 30 to 60 grains (2 to 4 gm.) every two or three hours, undoubtedly reaches the bowel, and may produce some healing effect.

Amebic Dysentery.—The same indication as to *diet* exists in the amebic

as in the other forms of dysentery. It is in this form, as well as in the bacillary type of severe grade of which only isolated cases are met in temperate climates, that the *ipecacuanha* treatment of the East Indian physicians has been so successful. It is claimed to act as a muscular sedative and secretory stimulant; by its effect the former allays the exaggerated peristaltic activity so characteristic of the disease, by the latter it augments the secretion of mucus as well as stimulates the activity of the liver-cells in bile formation—a function which in dysentery is in abeyance. Great stress is laid on the mode of administration. It is best given in pills coated with salol or keratin, so that it reaches the intestines undissolved. The stomach should be empty when the pill is given, at bed time. One dose is given each night, the first 60 to 90 grains (4 to 6 grams), which is reduced about 5 grains each night until the dose is down to 10 grains (6/10 of a gram). Milk should form the staple food. Later, farinaceous foods and soups may be carefully given, but no solids should be permitted for a long time.

Warm injections of quinin, 1 to 5000, 1 to 2500, and 1 to 1000, have been employed at the Johns Hopkins Hospital with good results, the amebæ being rapidly destroyed by them. Perhaps ipecacuanha acts similarly. For the relief of pain opiates must also be administered, preferably by the rectum in suppository or small starch-water enemas; or morphin may be given hypodermically if the stomach be sensitive. Appendicostomy has been lately practriced in chronic forms. The appendix is stitched to the belly wall, is kept open and the gut flushed through this opening.

Serum Therapy.—The immunizing protective effect of vaccines against the dysentery bacillus and the protective and curative effect of the antidysenteric sera demand allusion. Curative serum was first employed by Shiga in 1898 in the treatment of 65 cases in the hospitals in Tokio and by Rosenthal and Kruse. Shiga also practised vaccination to some extent in Japan, making a prophylactic vaccine out of dead dysentery bacilli. The availability of sera received fresh support from experimental studies by Simon Flexner and Frederick P. Gay.[1]

Dysentery vaccines were made of dead cultures as described in Gay's paper. Guinea-pigs which received one or more subcutaneous injections of subminimal lethal doses showed a marked protection against multiple intraperitoneal lethal doses of the living organism. It is interesting to note that while protection afforded by a given vaccine against its own strain of bacillus dysenteriæ was absolute, within limits, it was found that under similar conditions such protection may not be secured against other strains, suggesting the advisability of combining several strains of bacilli after their cultivation in the preparation of vaccines.

Antidysenteric curative serum was obtained from the horse after immunization. It was found to possess agglutinative properties for *bacillus dysentriæ*. This serum also had *protective* as well as curative properties against multiple fatal intraperitoneal doses in guinea-pigs. Gay concludes that this protective power may be regarded as proven beyond peradventure.

While a very considerable reduction of the mortality of dysentery has

[1] "Vaccination and Serum Therapy against the Bacillus of Dysentery. An Experimental Study." By Frederick P. Gay, "University of Pennsylvania Medical Bulletin," November, 1902.

appeared to result in Japan and Russia, from 32 to 12, 9 and even four per cent., the results thus far obtained in this country have not been sufficient to justify any conclusions as to the efficiency of sera although they show it to be harmless even in the case of little children.

Chronic Dysentery.

Any one of the forms of dysentery described may become chronic, but bacillary dysentery is the more usual form.

Morbid Anatomy.—All the lesions described as occurring in the different varieties of dysentery may be present. The most common is ulceration, which is variously extensive and exhibits also efforts at healing. On the other hand, cases of chronic dysentery are met with in which there are no ulcers whatever. The coats of the bowel are thickened, especially the submucosa and the muscularis, while patches of black and slate-gray discoloration are scattered through it, the resuit of blood extravasation and disintegration. Puckering, pseudopolyposis, and cystic degeneration may be present as described under Morbid Anatomy of bacillary dysentery.

Treatment.—The patient should be put to bed on a *diet* easy of assimilation and furnishing a minimum of waste. Its quantity should be just what is needed and no more. From what has been said it may be inferred that we have little confidence in methods of treatment, the object of which is to get remedies to the diseased bowel by way of the mouth. *Bismuth* in large doses, *iodoform*, and even *nitrate of silver* may, however, be tried for the purpose. One-half to 1 dram (2 to 4 gm.) of bismuth should be given at a dose, so that from 12 to 15 drams (48 to 60 gm.) are administered in the course of a day. Iodoform may be given as above directed.

The *topical treatment* of chronic dysentery by way of the rectum is that on which most reliance is placed at the present day. Its object is to get remedies to the diseased part. To this end they are dissolved and their solutions are introduced into the lower bowel. *Nitrate of silver* is the favorite remedy, but alum, sulphate of zinc, sulphate of copper, and acetate of lead are also used in the same doses. Twenty to 30 grains (1.3 to 2 gm.) are dissolved in a pint (1/2 liter) of water, and from 3 to 6 pints (1.5 to 3 liters) are injected at one time through a long tube gently introduced well up into the bowel, but at the onset weaker solutions and smaller quantities are injected. The patient should be placed on his back with the hips elevated by a pillow, so that there may be the coöperation of gravity. The treatment is sometimes painful. More may be expected from the irrigation of the colon by like solutions, through an appendiceal fistula.

THE PLAGUE.[1]

Synonyms.—*The Bubonic Plague; Oriental Plague; Black Death; Black Plague; Pestis Hominis.*

Definition.—The plague is a febrile infectious disease, characterized by a tendency to buboes or carbuncles, in addition to the usual phenomena of the typhoid state.

[1] For an admirable series of papers on the Plague see "British Med. Jour.," October 27, 1900; also, "Bubonic Plague," by Simon Flexner, "University of Pennsylvania Medical Bulletin," November, 1902.

Etiology.—The epidemic of 1894 gave the opportunity of isolating the specific germ *bacillus pestis* of plague which was discovered by Kitasato and later by Yersin. It is a short rod with rounded ends, and resembles the bacillus of chicken cholera. It is found in the blood, glands, and other viscera, and in no other disease excepting the plague. It is comparatively easily isolated from the blood and it becomes an important aid to diagnosis in those cases where the rapidity of the disease does not permit the development of other distinctive symptoms. Obtained in pure cultures, it can produce in inoculated animals the same effects as in human beings. Filth is a potent predisposing cause, as the description of Aoyoma, who was a member of Kitasato's expedition and himself fell a victim, vividly portrayed. The rat and the ground squirrel are the means of transmission from house to house, while man in his travels is the agent of transmission through long distances. The rat is the subject of plague. The fleas which infect its body become infected, they in turn bite an uninfected individual thus transmitting the disease.

Plague is a disease of hot countries and of hot seasons, but it may break out in midwinter. The disease is kept alive by plague in rats and sporadic cases in man. It attacks all ages and classes, but the poor, who live in crowded quarters and amid unfavorable hygienic surroundings, are its favorite victims. Small animals, such as monkeys, squirrels, rats, and mice die in great numbers during epidemics, and seem, indeed, to be the first victims. In this respect it is similar to anthrax and tetanus. Persons who live in upper stories are less frequently attacked than those who live on the ground floor. The boating population of China, which lives mostly on the water, is comparatively exempt. Body linen, bed clothing, carpets, rags, and baggage are frequent media of communication.

On the other hand, virulent as is the plague, its contagium appears to be more controllable than that of such diseases as smallpox and scarlet fever, as evidenced by the fact that with ordinary cleanly precautions few physicians, nurses, or others attendant on the sick acquire the disease, and even those employed to guard and disinfect houses commonly escape. In the epidemic in Canton, during which upward of 30,000 Chinese died, not one of 300 American and English residents was affected. It is of the greatest importance to know that a considerable interval may exist between the importation of an infection and the outbreak of an epidemic.

Morbid Anatomy.—There is no morbid anatomy to the plague beyond the buboes and internal suppurating processes, which seem to be essential symptoms, the cutaneous and other hemorrhages, and the various tissue alterations that attend high fevers generally. The liver and kidneys are congested and the spleen is enlarged to two or three times its normal size.

Varieties of the Disease.—Four principal forms are easily separated: (1) *Pestis minor*, abortive or larval form, which commonly appears before the outbreak of an epidemic. It is also the form which is endemic. It is characterized by moderate swelling of the lymphatics, little fever or other constitutional disturbance, and usually terminates favorably at the end of about two weeks. (2) *The bubonic form* is the more common severe epidemic form— the *malignant adenitis* of James Cantlie. Until recently all plague was called "bubonic," but it is now known that only about 70 per

cent. of cases are accompanied by glandular enlargement. (3) The *septicemic form*, also known as *toxic*, *fulminant*, or *siderans*, a severe form, in which death may occur in twenty-four hours with associated hemorrhages, but in which glandular enlargement is slight; the time between the onset and the fatal termination being too short to allow its development. Prostration is extreme. (4) The *pneumonic form*, in which no buboes appear on the surface, but the force of the disease is spent on the lungs, the sputum swarming with bacilli. The processes in the latter organs are septicemic.

Symptoms.—*Of the bubonic or ordinary form.*—A *period of incubation* of from two to seven days usually precedes the appearance of the *intense weakness* which is one of the earliest characteristic symptoms of the plague. A second period or period of *prodrome* may follow the incubation, though it is not common. It is short, from a few hours to a couple of days, and includes headache, prostration, marked nausea, vomiting, vertigo, and rarely lumbar pain. A chill is not usual, but there may be *chilliness*, after which the usual *fever* of the infectious diseases sets in with great severity and with its accompaniments, among which severe headache, backache, delirium, and the typhoid state are conspicuous. The *temperature* rises rapidly to 102° and 104° F. (39° and 40° C.) and even higher. The *pulse* ranges from 90 to 120, of fair volume, often dicrotic. Before the fever sets in great weakness is manifest. The patient reels like a drunkard, with weakness and vertigo. He breathes hurriedly and is anxious, restless, and depressed. The features are drawn and haggard. *Petechiæ*, *vibices*—the plague-spots of the Bible—*albuminuria*, *hematuria*, and even *hematemesis* may be included. Slight enlargement of the spleen is present.

Pre-eminently characteristic is the *bubo* or suppurating gland. It appears on the second or third day, if the patient live to it. It occurs in order of frequency in the glands of the groin, the armpit, the neck, or in the popliteal region. It commonly reaches the size of a walnut or egg, when it ruptures, if not opened with the lance. It may, however, subside without discharging. Suppuration is a desirable termination. It is painful and tender, as buboes commonly are. Coincident with the appearance of the bubo the fever subsides, a *profuse sweat* breaks out, and the pulse falls to 90 or 100. In addition to the bubo, *carbuncles* may also be present in the lower extremities, the buttocks, or in the neck. In some epidemics hemorrhages are common, and even the buboes may contain blood.

In the *pneumonic* form there are the usual symptoms of pneumonia, chill, high fever, severe pain in the side, dyspnea, cough, rusty sputum, and physical signs of consolidation,[1] and marked prostration. Bacilli may be found in the sputum; and this form is infective through the sputum.

In the *septicemic form* the patient is stricken by a virulent poison and the prostration is extreme. The glands are enlarged, but there are no buboes. The enlargement is slight and may only be detected at necropsy, but it is general. Hemorrhages from the nose, bowel, and kidney are most frequent in this form. Apyrexia is not uncommon, fever reaction being impossible because of the extreme depressing influence of the disease. The delirium is of the typhoid type.

Diagnosis.—In its fever, its intense prostration, its petechiæ and vibices

[1] For reports, see "Sajous' Annual," vol. v., 1900, article "Plague."

of the early stages the plague resembles typhus. No other fever is characterized by such intense prostration. The bubo and the carbuncle seem to be the distinctive signs, although they are said to be sometimes absent in the milder cases of a declining epidemic, as well as in the intense *pestis siderans*. The bacillus may be isolated from the blood in suitable cultures and should be thus sought in doubtful cases. The diazo reaction of the urine is usually absent.

Prognosis.—The plague is said to be the most fatal of all diseases, 70 to 90 per cent. perishing, districts and towns being half depopulated, while whole families have been annihilated. Death occurs from the second to the fourth day, and if recovery take place it is delayed by the slowly healing buboes and carbuncles. These may, however, heal rapidly.

Treatment.—Prophylaxis is the most important part of the treatment, all rats must be exterminated—no rat, no plague. Cleanly habits which insure absence of the infected flea from the body are necessary. Free stimulation, nutritious food, as in the most adynamic forms of typhus and typhoid fevers together with cool baths to combat the fever, are the measures indicated. Antiseptic treatment of the buboes and abscesses should be practised, and may shorten the duration of these plagues of the skin as compared with the older treatment. Morphin should be given to produce sleep and relieve pain. Kitasato's general directions, so often quoted, can hardly be improved. They are as follows:

"The disease prevails under faulty hygienic conditions; it is, therefore, urged that general hygienic conditions be carried out. Proper receptacles for sewage should be provided, a pure water supply afforded, and streams cleansed; all persons sick of the disease isolated; the furniture of the sick-room washed with a two per cent. carbolic solution in milk of lime; old clothes and bedding are to be steamed at 212° F. (100° C.) for at least one hour, or exposed for a few hours to sunlight. If feasible, all infected articles should be burned. The evacuations of the sick are to be mixed with milk of lime, and those who die of the disease are to be buried at a depth of three meters (about 12 feet) or, preferably, cremated. After recovery the patient is to be kept in isolation at least one month. All contact with the sick is to be avoided, and great care exercised with reference to food and drink." Instead of carbolic acid and milk of lime for the disinfection of buildings, Haffkine suggests sulphuric acid in the proportion of 1 to 200 of water.

Serum Therapy.—Preventive inoculation was introduced by Yersin, Calmette, and Borrell conjointly in 1895. Dead cultures of plague bacilli were injected subcutaneously into rabbits and guinea-pigs and found to convey a certain degree of immunity against plague. Haffkine extended this method of preventive inoculation to man. The dead bacilli, suspended in bouillon, were injected subcutaneously, first in lower animals, notably monkeys, with the result of protecting them against subsequent inoculation with virulent plague bacilli; then upon human beings in India and China. Haffkine's vaccine, or prophylactic, is a solution of toxic substances produced during the growth of plague bacilli, and should not be spoken of as a serum as no animals are used in its preparation.

The results of these inoculations are more definitely stated in the fol-

lowing conclusions reached by the Indian Commission: "(1) Inoculation sensibly diminishes the incidence of plague attacks on the inoculated population, but the protection which it affords against attacks is not absolute; (2) Inoculation diminishes the death-rate among the inoculated population. This is due not only to the fact that the rate of attack is diminished, but also to the fact that the fatality of the attacks is diminished; (3) Inoculation does not appear to confer any great degree of protection within the first few days after it has been performed; (4) Inoculation confers a protection which certainly lasts for some considerable number of weeks. It is possible that the protection lasts for a number of months. The maximum duration of protection can only be determined by further observation; (5) The varying strengths of the vaccine employed have apparently had a great effect upon the results which have been obtained from inoculation. There seems to be a definite quantity of vaccinating material which gives the maximum amount of protection; and provided that this quantum can be injected in one dose, and provided also that the protection turns out to be a lasting one, reinoculation might with advantage be dispensed with."[1] Colonel Bauner, Director of the Plague Laboratory and successor of Haffkine, devised a number of methods by which the vaccine could be produced in a purer state, uncontaminated by other bacteria which had been productive of serious complications.

As contrasted with preventive treatment Yersin's antipest serum[2] and Lustig's serum are intended for *curative* purposes, after the manner of diphtheritic antitoxin. Yersin's is not only curative but antitoxic and may be employed at any stage. It is prepared by injecting the horse, first, with dead and then with living plague cultures. The serum, to insure sterility, is heated to 140° F. (60° C.) before being sent out. Lustig's serum is prepared from the horse after injection with a substance derived from bacilli by treatment with alkali and precipitation by hydrochloric acid.

Small animals have been rescued from infection by plague germs by Yersin's serum, but in human beings the results have been less conclusive, Arnold[3] claiming that it reduced the mortality of cases 70 to 90 per cent., while Cremow[4] denies any therapeutic value. Calmette and Salimbini[5] claim to have shown from their observations and experiments during the plague epidemic in Oporto, Portugal, that much larger doses of the antipest serum may be used, and are sometimes demanded, than have heretofore been deemed sufficient. The use of a preliminary immunizing dose of antipest serum, followed by an injection of a dose of Haffkine's prophylactic, removed the element of danger and conferred an immunity of probably longer duration than would be produced by the exhibition of the serum alone.

Notwithstanding the seeming inconclusiveness of these observations, the Indian Commission reports that "though the method of serum therapy as applied to plague has not been crowned with a therapeutic success in any way comparable to that obtained in the treatment of diphtheria, never-

[1] See Simon Flexner's paper on "Bubonic Plague, its Nature, Mode of Spread, and Clinical Manifestations." "University of Pennsylvania Med. Bulletin," November, 1902.
[2] "Sajous' Annual," vol. v., p. 491.
[3] "Med. News," January 1, 1898.
[4] "London Lancet," May 6, 1899.
[5] Calmette and Salimbini," Philadelphia Medical Journal," Feb. 10, 1900.

theless, the method of serum therapy is in plague, as in other infectious diseases, the only one which holds forth the prospect of success."

Walter Wyman, Supervising Surgeon-General United States Marine Hospital service, has directed that between 160 and 200 c.c. of antipest serum should be given during the first 48 hours of the disease. In severe cases, 20 to 40 c.c. of this amount should be injected into a vein. In immunizing with Yersin's serum inject 5 to 10 c.c. every 15 days. In case the Haffkine prophylactic cannot be administered on account of exposure to the disease, the mixed plan of immunization may be used. This consists of giving 5 to 10 c.c. of antipest serum, and, three days later, 1 c.c. of the Haffkine prophylactic.[1]

MEASLES.

SYNONYMS.—*Rubeola; Morbilli.*

Definition.—Measles is an acute, highly contagious disease, characterized especially by a mottled eruption and naso-bronchial catarrh.

Etiology.—Measles is in all probability due to a micro-organism, which, however, has not as yet been isolated. Anderson has shown experimentally that the disease may be transmitted to monkeys by injecting the blood, the expectoration, and nasal and conjunctival mucus of cases of measles, and that it cannot be transmitted by injecting the desquamating skin. Whatever the infecting agent may be, it is very unerring, since the disease is more unfailingly communicated to those unprotected by previous attacks than is scarlet fever. The contagium has been transmitted by the inoculation of morbillous blood and nasal mucus, and it is most active when the breath is its medium. It is communicable by a third party and by fomites; though more active and unfailing than the contagium of scarlet fever, it is less so than that of smallpox. It is not, however, so tenacious as these. Measles is a disease of childhood, but adults often get it, and that very severely. No age is exempt. Repeated attacks are possible, but as other eruptive affections resemble it and diagnosis is often careless, some of the repeated attacks may be thus explained. It is milder and rarer in sucklings under six months. Further, the studies of Carr, Mayo and Edw. Graham go to show that the new-born are very slightly susceptible. Six months would appear to be the age at which susceptibility begins, although Bartsch reports a case of intrauterine infection. Finally the age during which the disease is more commonly contracted is from one to five years.

Morbid Anatomy.—There is no essential morbid anatomy of measles beyond the nasal and bronchial catarrh, and the signs of these generally disappear with death. When death occurs it is usually the result of complications, and the morbid anatomy of such is present. The most frequent complication is bronchopneumonia. There may be lobar pneumonia, and among the morbid phenomena are to be included sometimes those of collapse of the lung. In rare instances of hemorrhagic or "black" measles there is the usual discoloration of hemorrhagic extravasation. Rarely also the morbid states of intestinal catarrh are found.

[1] "Philadelphia Medical Journal," February 10, 1900.

Symptoms.—The *period of incubation* of measles varies, but is commonly between seven and 14 days. Rarely it is a day or two longer. A prodrome, if present, in measles is of short duration. It may be manifested by sneezing, fretfulness, chilliness, and feverishness; or, if the child is old enough to express itself, by headache. Then comes, on the first day, the initial or prodromal fever, a peculiarity of which is a remission on the third day. This is shown by the appended cut from Eichhorst. But very early, and even almost suddenly, *coryza*, with red and watery eyes, and photophobia present themselves, closely followed by troublesome *cough* and corresponding feverishness reaching 103° and 104° F. (39.4° and 40° C.). Much less frequently than in scarlet fever is there *vomiting*, and the tongue is apt to be furred. The cough is sometimes croupy. Convulsions very rarely usher in the disease. In the very beginning Koplik's spots are present.

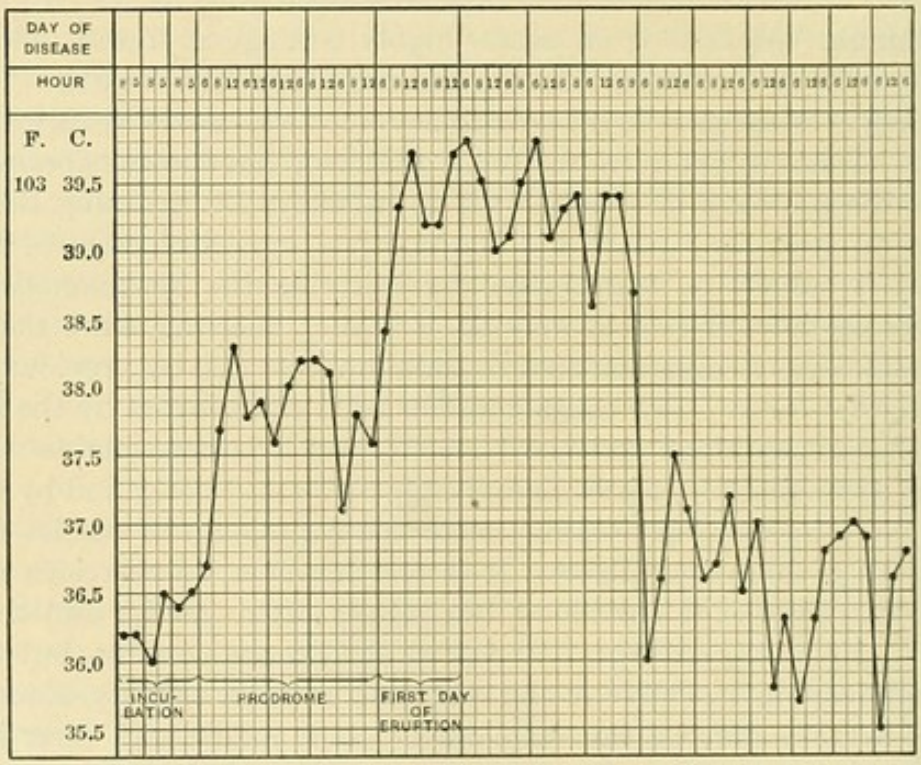


FIG. 15.—Temperature Chart of Measles.—(*Eichhorst.*)

On the fourth day from the onset the *eruption* makes its appearance. With the eruption the fever usually increases for 24 to 48 hours. It appears first in the *face* in the form of papules and blotches, which coalesce more or less imperfectly, leaving sometimes islands of white skin between them. Sometimes after coalescence the eruption quite resembles that of scarlet fever. Under any circumstances the boundary between the eruption and the sound skin is uneven and crescentic. The eruption is somewhat raised above the surface, and the whole effect is to make the face appear swollen. This elevation of surface at times becomes distinctly papular and even shot-like, resembling closely the papular stage of smallpox. In fact, this appearance has quite often lead to a diagnosis of smallpox, which 12 hours later had to be withdrawn. From the face the eruption spreads to the neck, thorax, abdomen, and extremities. It is bright red, as a rule

Fig. 1.



Fig. 2.

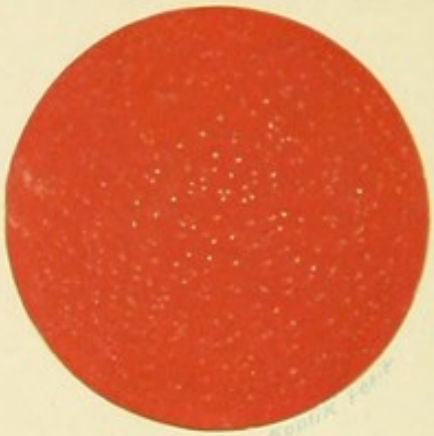


Fig. 3.

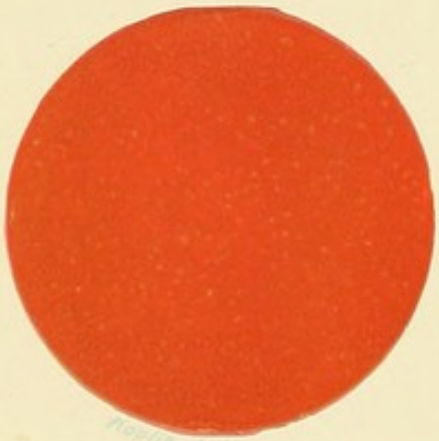
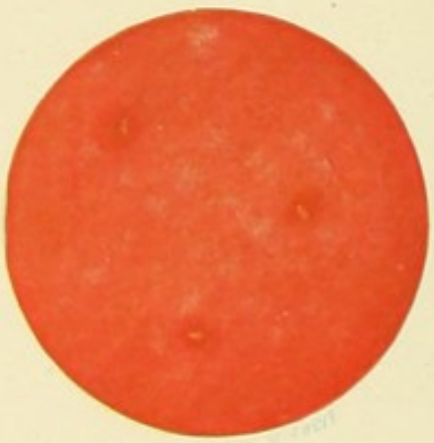


Fig. 4.



THE PATHOGNOMONIC SIGN OF MEASLES (KOPLIK'S SPOTS).

FIG. 1.—The discrete measles spots on the buccal or labial mucous membrane, showing the isolated rose-red spot, with the minute bluish-white centre, on the normally colored mucous membrane.

FIG. 2.—Shows the partially diffuse eruption on the mucous membrane of the cheeks and lips; patches of pale pink interspersed among rose-red patches, the latter showing numerous pale bluish-white spots.

FIG. 3.—The appearance of the buccal or labial mucous membrane when the measles spots completely coalesce and give a diffuse redness, with the myriads of bluish-white specks. The exanthema on the skin is at this time generally fully developed.

FIG. 4.—Aphthous stomatitis apt to be mistaken for measles spots. Mucous membrane normal in hue. Minute *yellow points* are surrounded by a red area. Always discrete.

—(*From "Medical News."*)

disappears on pressure. Sometimes, however, even in mild cases, there are petechiæ, and in malignant cases the extravasations are extensive. At the same time, the mouth and fauces are bright red in color and the rash is present in these positions. Not infrequently there is diarrhea, as though the eruption extended throughout the entire mucous tract as well as over the skin. At the maximum of the eruption there may be slight swelling of the cervical lymphatic glands. At the end of two or three days after its appearance the rash fades gradually, first from the situations in which it appeared earliest, and a fine, branny desquamation occurs, easily overlooked. The fading takes place in the order of invasion. The typical rash may be accompanied by sudamina.

In 1896 Henry Koplik[1] called attention to a sign that has been found of real value in the diagnosis of measles. It is the appearance, on the first day of invasion, on the buccal and labial mucous membrane, of a scattered eruption of minute *bluish-white specks* each surrounded by a bright red areola. They have been found 45 times in 52 cases and 31 times in 32 cases. They may appear four days before the characteristic rash, and rarely before the fever. The spots somewhat resemble those of thrush, from which they are distinguished by their roundish shape and their color, as contrasted with the more *yellowish* center of those of thrush and by the fact that scrapings do not show the oidium albicans. While thoroughly discrete in the beginning, later in the disease the spots may coalesce, and the characters of a discrete eruption or spotting disappear, producing an intense general redness, "which is simply dusted over with myriads of these bluish-white specks." They cannot be wiped off, but the whitish portion can be removed by forceps without causing pain or bleeding. They consist of thick layers of epithelium in a state of partial fatty degeneration. They require a good light for their demonstration. (See plate opposite.)

The other symptoms described continue until the eruption begins to fade— that is, on the fifth or sixth day, when they abate. The *cough*, often hangs on quite stubbornly, especially in tuberculous children, and sometimes even persists as the catarrhal symptom of a tuberculosis, the development of which seems peculiarly favored by the disease. Hence, the cough of measles should never be slighted, and early exposure to cold and dampness should be guarded against.

It has already been intimated that a malignant form of measles sometimes occurs, called also "black" measles, which is very serious—often, indeed, fatal. It is generally epidemic, occurs in institutions and camps, and its presence is characterized by subcutaneous extravasations of blood and hemorrhages from the mucous membranes. Hoarseness is especially found in black measles, as contrasted with black smallpox.

Complications and Sequelæ.—These furnish most that is serious in the disease, and of them the most frequent and dangerous is bronchopneumonia, the bronchitis creeping into the smaller air-tubes. The occurrence of this form of pneumonia seems to be favored by bad hygiene. Collapse of the lung is also prone to occur, caused by an accidental valve-like plug of secretion. Bronchopneumonia is recognized by the persistent and aggravated cough, the continued high temperature,

[1] "Archives of Pædiatrics," December, 1896, and "Medical Record," April 9, 1898.

and physical signs of a circumscribed pneumonia. More rarely lobar pneumonia supervenes and is recognized even more easily. In view of these possible complications, frequent physical examinations of the chest should be made.

Among the complications may be mentioned laryngitis, catarrh of the middle ear leading to suppuration and perforation of the drum, and chronic or intractable ophthalmic trouble. Ulcerative and even gangrenous stomatitis or *cancrum oris* are met under unfavorable hygienic conditions; also ulcerative vulvitis.

Nephritis, although not often a complication of measles, does, however occur. Tuberculosis has long been recognized as a sequel of measles, yet it is not a very frequent one. Any of the varieties of pulmonary tuberculosis may be present. Even nervous lesions are reported, such as hemiplegia, paraplegia, neuritis, and myelitis.[1]

Diagnosis.—Measles is easy of diagnosis; but the physician must not be too precipitate. Allusion has already been made to the possibility of mistaking it for *smallpox*, on account of the similarity of the eruption in the early stage, an error which a few hours' delay would have averted. Koplik's spots should be helpful here, as they appear at least twenty-four hours before the skin eruption. From *scarlet fever* there is sometimes difficulty, as there is occasionally slight sore throat and the eruption may be diffuse, while the difficulty is increased if there be glandular swelling in measles; but the catarrhal symptoms of measles are essential to it. The time of appearance of the rash is much later in men. In measles there is no leucocytosis. In scarlet fever there is leucocytosis. The mildest cases are probably those that give most trouble. The distinction between measles and rubella is sometimes more difficult, but this will be considered when treating of rubella.

Typhus fever and measles have been confounded, and it must be admitted that in the asthenic variety of measles the eruption may resemble that of typhus fever. It will be remembered that the eruption of typhus is described as "rubeoloid." Confusion is further favored by the fact that the eruption occurs at about the same time in each disease.

Prognosis.—The vast majority of cases of measles get well, yet measles is not the slight disease it is often considered. In epidemics of the malignant form, in hospitals, camps, and foundling asylums, that death occurs as a direct result of the disease. In these the mortality is sometimes very high. Epidemics among the aborigines in North and South America, in the Mauritius and Feejee Islands, and in the Confederate Army in the War of the Rebellion in America were of signal fatality. Other deaths are due to complications, especially pneumonia. Out of 24 fatal cases collected by Pott, 21 died of bronchopneumonia and pneumonia, and three of croup.

Treatment.—After surrounding the patient by a uniformly warm temperature, best secured in bed, in a well ventilated room, the treatment of measles is mainly that of the fever and the cough. The former is sufficiently treated by the simple diaphoretics and febrifuges, such as citrate of potash and sweet spirit of niter, or tincture of aconite. The latter is efficient and

[1] See a paper by Imogene Bassette entitled "The Paralyses in Children which occur during and after Infectious Diseases," "Jour. Nerv. and Ment. Dis.," vol. xix., 1902.

tasteless. The coal-tar derivatives, acetanilid, antipyrin, thermol, and phenacetin may be used.

The cough calls for positive anodyne measures, of which, for children, paregoric is the best because the safest. Laudanum or deodorized tincture of opium may be used in smaller doses, but not morphin. It is best to prescribe the opiate separately in order that the dose may be decreased or increased at will. It is comparatively rare that cool sponging is needed to reduce the temperature, but cold water drinking should be allowed *ad libitum*. A case of measles under our care received the cold tub-bath treatment under the impression that it was typhoid fever. The rash came out brilliantly at the proper time and the case did splendidly. Complications should be treated as they arise. Stimulants and tonics are necessary in the adynamic form. When the cough is prolonged, cod-liver oil is a valuable remedy. Watchfulness during convalescence is more important than is supposed by many, and carelessness and indifference are sometimes responsible for unfortunate results.

It occasionally happens that the eruption is "suppressed," or its appearance may be delayed. Under these circumstances the hot pack is very effectual. The child is wrapped in flannel wrung out in hot water and then enveloped in a mackintosh. Copious perspiration soon sets in, the eruption appears, and general reaction begins.

RUBELLA.

SYNONYMS.—*Rötheln; Rubeola;*[1] *German Measles; Rubeola notha; Epidemic Roseola; False Measles; Hybrid Measles; Hybrid Scarlet Fever.*

Definition.—Rubella is a mild, acute, contagious disease, characterized by a punctiform rash that fuses into patches less plainly crescentic than those of measles. There is often slight sore throat, more rarely mild catarrhal symptoms, and trifling fever. Many so-called second attacks of measles and scarlet fever are attacks of rubella.

Etiology.—The relation of the disease seems rather closer to measles than scarlet fever, and may be said to bear to the former the same relation as varicella to variola. Though contagious, it is much less so than measles or scarlet fever. It affects children chiefly, very rarely adults, sucklings less frequently than school children, because the latter are more exposed to contagion. Isolated cases occur, but it is apt to prove in large cities epidemic. Such epidemics are sometimes widespread. No special bacillus has as yet been isolated.

Symptoms.—After a *period of incubation* ranging from two to three weeks, the disease sets in, as a rule, with no distinctive prodromal symptoms prior to the eruption. There may be chilliness, moderate muscular pain, mild catarrh, and slight fever, with temperature barely reaching 100° F. (37.8° C.), for a day or two previous to the eruption. Rarely these prodromal symptoms may be prolonged to two, three or even four days.

More frequently, an indistinct macular *eruption* of a pale rose color is the first symptom noted. The papules are not elevated, and vary in size

[1] It is unfortunate that the Germans have selected for their technical term for this affection the word *Rubeola*, which is the word used in English for *measles*.

from a pinhead to a split pea, the smaller being more numerous, much smaller than the papules of measles. They may, however, fuse and form large, irregular patches, with little or no disposition to form small crescent-shaped groups like those of measles. The rash may appear as late as the second day, rarely on the third, after the indistinct symptoms of invasion mentioned.

Two types of the spread of the eruption are possible. In the one it appears almost simultaneously all over the body, reaching its maximum by the second day, after which it rapidly fades. In the second mode of invasion the rash appears first on the face, and extends rapidly thence all over the body, reaching the hands and feet last, and beginning to fade on the face and trunk before attaining its maximum on the extremities, or even before it appears there at all. Thus it has a wave-like course, reaching its maximum in twenty-four hours, when it begins to decline rapidly. It is, therefore, of shorter duration than the eruption either of measles or of scarlet fever. It may terminate in a branny desquamation, less evident even than that of measles.

The most constant symptom after the eruption is the *sore throat.* It varies in severity, but is for the most part mild, never becoming ulcerative. It is really, perhaps, the eruption in the throat. Somewhat less constant than the sore throat, though varying somewhat in different epidemics, is *swelling of the lymphatic glands* of the neck, especially the superficial cervical, postcervical, and postauricular glands. This swelling is present during the eruptive stage and may occur even earlier. Its possible, though rarer, occurrence in measles and scarlet fever also is to be remembered.

The remaining symptoms of rubella are not marked nor distinctive. There is little or no constitutional disturbance, and, as already mentioned, rarely any *fever* above 100° F. (37.8° C.), although 102° F. (38.9° C.) and even 103° F. (39.4° C.) have been noted. There may be slight catarrh, watering of the eyes, and running at the nose, all much less marked than in measles. There are no complications, as a rule, though albuminuria, nephritis, pneumonia, colitis, and icterus have been reported, but it would seem as though measles or scarlatina must have been mistaken for rubella in these cases.

Diagnosis.—Such are the symptoms of a typical case. Unfortunately, there are many deviations, some approximating *measles* and some *scarlet fever*, differing from either mainly in mildness. The absence of decided catarrhal symptoms, the earlier appearance of the eruption, its more diffuse character, and the swelling of the lymphatic glands are its chief differences from measles. The careful studies of J. P. C. Griffith[1] show the latter of less significance than has been usually supposed. The course of the eruption differs also, that of measles lasting longer. The absence of Koplik's sign must hereafter be helpful in distinguishing it from measles. The same mildness and absence from fever, with the more distinct mottling, distinguish it from scarlet fever. In rubella the symptoms of invasion are all very much milder than in either measles or scarlet fever, even mild cases of the latter. Most cases of supposed second attacks of measles are cases of rubella.

[1] "Differential Diagnosis of Rubeola and Rubella, with Special Reference to Enlargement of the Glands of the Neck," "University Med. Magazine," June, 1892.

Prognosis.—The prognosis of rubella is invariably favorable.

Treatment.—Very little if any is required, except rest in bed. A simple febrifuge with potassium citrate may be useful.

SCARLET FEVER.

SYNONYM.—*Scarlatina.*

Definition.—Scarlet fever is an acute contagious disease, especially characterized by sore throat and a diffuse scarlet eruption, terminating in more or less membranous desquamation.

Etiology.—The organism that causes scarlet fever has not been isolated. Streptococci have been found in the blood by many observers. Less frequently staphylococcus aureus and the influenza bacillus but the consensus of opinion is that these are cases of secondary infection and that the special bacillus of scarlet fever has not as yet been isolated. Such secondary infection has been moreover held responsible for many serious complications attended with suppuration.

Whatever the agency, it is the most tenacious of all the contagia, retaining its power to infect for at least a year after the occurrence of a case. It is especially difficult to dislodge from organized substances, such as bedding, clothing or straw, letters and books, and the disease has been communicated to newcomers even after an infected apartment has been thoroughly cleaned and fumigated with sulphur. Physicians have doubtless conveyed it, and the beard and hair are contagium-bearers more frequently than is supposed. Hence, physicians in attendance should cover the head with a cap which will protect both the scalp and beard, and nurses, before passing from one case to another, should disinfect the hair as well as the rest of the body.

While the contagium itself has never been isolated, there is every reason to believe that the bearers are the exudate from the eyes, nose and throat, from the skin at the time of the appearance of the exudate, as well as the exfoliated epithelium. The virulence of these exudates often lasts long after desquamation ends. Discharging ears, discharging sinuses are frequently infective, the ease with which the scaly particles are disseminated through the air and the tenacity with which they adhere to textures readily explain the communicability of scarlet fever and the difficulty in destroying its cause. On the other hand, until the eruption makes its appearance the disease is not likely to be spread. Hence, children removed from association with the disease promptly after its discovery, and kept apart, generally escape it.

The route of infection is mostly the respiratory tract, although the alimentary canal may also convey it. In confirmation of this is the fact that in a number of instances milk has been the medium of infection. Trask ("Hygienic Laboratory Bulletin," No. 56) cites 51 epidemics of scarlet fever spread through milk. The infecting cause was variously a lack of care at the place of milk supply, unsterilized bottles, or a nurse.

The disease occurs more frequently in children, because a single attack, as a rule, protects against a second. Infants, however, even under exposure, are less liable to the disease, and it would seem, too, that adults who have

escaped exposure during childhood are less liable. The primary attack is not always protective; second and third attacks are reported. But here, again, careless diagnosis and defective memory are responsible for a certain number. The disease is most common between the ages of four and seven.

Holt says that about 50 per cent. of all children exposed to the disease are attacked. Koplik gives the same figures and Carr places the number as high as 56 per cent.

Morbid Anatomy.—There is no morbid anatomy peculiar to scarlet fever. The eruption fades after death, unless there happen to be hemorrhagic extravasation. There may be lesions the result of ulcerative destruction in the neighborhood of the throat. The intensity of the fever sometimes produces granular fatty change in muscles, which is pronounced in the case of the heart; also cloudy swelling in the cells of the kidney and liver. Glandular swellings present at death maintain themselves afterward. The morbid anatomy of the complications and sequelæ is appropriately considered under the diseases constituting them.

Symptoms.—The *period of incubation* varies greatly. It is sometimes as short as 24 hours, and again as long as 12 days; more frequently, perhaps, from two to four days. At the end of this time there is usually a very short prodrome, sometimes none at all. *Vomiting*, occurring either as an initial symptom or a couple of hours later, is often present; more rarely a *convulsion*, still more rarely a *chill*. *Sore throat* is early complained of, and *high fever* is conspicuous. The fever is early, the face is flushed, and the temperature rapidly rises to 103° F. (39.4° C.), 105° F. (40.5° C.), and even 108° F. (42.2° C.), and the pulse to 110, 120, or more.

The *eruption* appears, as a rule, on the second day, and it generally happens that, if it is not present at the physician's first visit, it is sure to be found at his second. Its striking character is its uniform redness. It is like a diffuse, broadly spread blush, appearing first upon the neck and chest, and extending thence rapidly over the whole body, so that at the end of the third day it has completed its invasion. The appearance of a child covered with a frank scarlet fever eruption is very characteristic. The cheeks and forehead are flushed while around the mouth the lips are white forming a marked "anemic ring" in the midst of the flushed face. It has been well compared with that of a boiled lobster in its bright redness. It is further characterized by the readiness with which it disappears on pressure and the promptness with which it returns after the pressure is removed. It is, however, no sooner complete than it begins to fade, and does so with great rapidity in the order of invasion. The eruption is not, however, always thus typical, and presents every degree between that described and that which is barely recognizable. It is also at times more "patchy," but never presents the crescentic or otherwise irregular edges or mottled appearance of the eruption of measles. In the lower and more malignant forms the redness is of a darker or dusky hue, and in the worst of these, petechiæ are present. Vesicles are even found with turbid contents, producing *scarlatina miliaris*. The eruption is sometimes entirely absent from the face. The thorax and inner surface of the thighs are more favorable sites. The eruption, when severe, is constantly accompanied by an itching or burning more or less intense, and there is a feeling of slight roughness at times.

The *tongue* is red at the edges and tip, furred at the center, but through the fur the papillæ stand out in distinct points, producing an appearance that is regarded as more or less characteristic. This has been called by some the *strawberry tongue*. But further examination into the subject leads us to adopt the view that the strawberry tongue is the red and raw-looking tongue with enlarged papillæ, as originally held by the late Dr. Flint,[1] who wrote as follows: "In the progress of the disease the coating exfoliates, leaving the surface of the tongue reddened; and the papillæ being enlarged, the appearance is strikingly like that of a ripe strawberry." The term *raspberry tongue* is also applied to this condition. The rest of the mouth, including the roof and the palate and tonsils is bright red, as though the eruption extended to it, as it doubtless does.

With the abatement of the eruption comes *desquamation*, and it is generally proportionate to the intensity and extent of the former. It sets in about the *tenth* day, and continues in bad cases for two or three weeks and even longer. When the eruption is slight, the little scales are scarcely noticeable, and the closest examination is necessary to discover them, while, where there is a vivid and extensive eruption, the amount of desquamation is enormous. Glove-like casts of the fingers, including the nails, are sometimes exfoliated, and the bed contains each day numerous flakes of epiderm that have come off, while many days are required for complete separation of the dead skin. Great care should be taken in gathering it up, for it is still believed that, in the desquamation resides the contagium. On the other hand, when slight it should be carefully sought for, as it has great diagnostic value. At the same time it should not be regarded as something peculiar and confined to scarlet fever, for every dermatitis is followed by desquamation, as especially exemplified in the exfoliation that follows an attack of erysipelas on the face or irritation by iodin or mustard.

The *urine* from uncomplicated scarlet fever is like that of fever cases generally—scanty, high-colored, and precipitating uric acid and urates on cooling. The chlorids are diminished during active fever.

The *blood* in scarlet fever exhibits a sudden hyperleukocytosis, 18,000 to 40,000 white cells per cubic millimeter, falling gradually to the normal in from three to six weeks. There is also a moderate secondary anemia.

The *duration* of simple uncomplicated scarlet fever ranges from three to fourteen days, according to the degree of severity. Its decline is, however, usually gradual as compared with the suddenness of onset.

Such is the general picture of scarlet fever in its simple, uncomplicated form, so characteristic that early in its history it received the name *scarlatina simplex;* owing to further combinations of symptoms, there have been added three other varieties: the anginose form, or *scarlatina anginosa;* the malignant form, or *scarlatina maligna*, and the *hemorrhagic* form.

In the *anginose* variety the throat symptoms are conspicuous and severe. In no well-developed case is there an absence of throat redness. On the other hand, there may be intense soreness with swelling of the fauces and tonsils, giving rise to extreme dysphagia. The neck may be so swollen as to fill up the depression beneath the jaw. There may be a false membrane

[1] For paper containing the views of various authors on this subject see "The Strawberry Tongue in Scarlet Fever," by M. H. Fussell, "University Med. Magazine," Philadelphia, May, 1897.

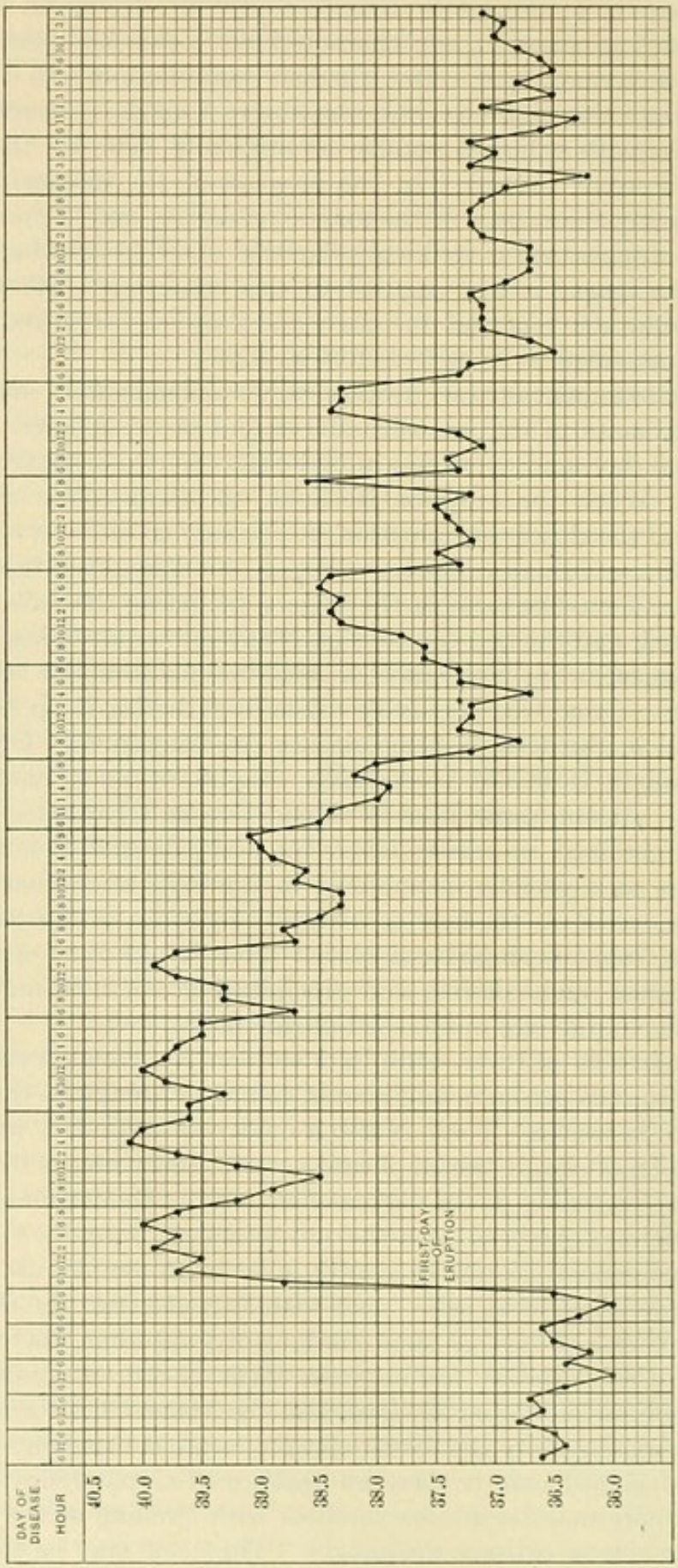


FIG. 16.—Temperature Chart of Scarlet Fever.—(*Eichhorst.*)

involving the fauces, the posterior pharynx, the nasal cavities, the trachea, and the bronchi. The throat may present all the features of a severe diphtheria. Abscess and destructive ulceration may result, which may proceed even to perforation of the carotid artery, and rapid death ensue therefrom. The inflammation almost certainly ascends the Eustachian tubes, producing severe ear symptoms. The false membrane is usually the result of the intensity of the inflammatory process, due to the specific cause of the disease or to a streptococcic infection and not to that of diphtheria, but there may be true diphtheritic membrane containing the Loeffler bacillus. This can be decided only by the microscope. Especially is this true of the cases in hospitals for infectious diseases. The *streptococcus pyogenes* is perhaps the most frequent cause of the throat inflammation. It has been found also in the skin, the blood, and the glandular organs in fatal cases. Scarlet fever has, indeed, been called a streptococcus infection. Follicular tonsillitis may also be one of the forms of sore throat.

In the *malignant* variety there is an overwhelming intensity of the cause which may result in almost immediate prostration and death of the patient, giving no time for the development of the usual symptoms, or these may be so feebly manifested that they present no distinctness. When the disease is not immediately fatal, there is intense adynamia, the heart and pulse sharing it. The breath is rapid; the capillary circulation is feeble; the skin dusky; the eruption is imperfectly developed; the temperature is very high, reaching 105° to 108° F. (40.5° to 42.2° C.); there is delirium, which may pass over into coma, and convulsions may occur. The pulse ranges from 120 to 150.

In the hemorrhagic form there are more or less extensive hemorrhagic extravasation, epistaxis, and hematuria. It attacks, for the most part, the feeble and badly nourished, and, like the previous variety, is almost invariably fatal.

Epidemics of scarlet fever vary greatly in severity. In some all the cases appear to be mild, in others all are of extreme severity. Families of children may be exterminated. Again, a mild case may give rise to one of the most intense forms.

Complications.—*Acute nephritis* is the most frequent complication of scarlet fever. It makes its appearance usually after desquamation is more or less complete—in the second, third, or fourth week. A slight albuminuria, which is common at the height of the fever, is not to be confounded with that of nephritis, and probably does not predispose to it, although the cells lining the tubules are at this stage in a state of cloudy swelling. The rationale of its production is not precisely understood. It is probably the direct result of the toxic power of the infecting agent upon the kidney substance. The fact that the complication is usually more severe the earlier it appears and the more severe the case would go to show that the specific toxin is the cause. It is true, too, that with the skin functionally dead the complemental work thrown upon the kidney increases its susceptibility to the ordinary causes of nephritis, of which cold is one. It is to be remembered also that other diseases in which the skin is seriously affected predispose to nephritis. This is pre-eminently true of burns and scalds.

However it may be brought about, the result is generally a typical example of parenchymatous or tubal nephritis, although instances of acute

interstitial inflammation are also found. Every grade of severity is met, but early recognition increases our power to control this severity. The majority of cases thus recognized get well, recovery takes place after sup pression of urine has lasted for a week. The clinical picture is that of actue nephritis otherwise caused, and its consideration may be deferred until that disease is studied. This complication was formerly often over-looked, but in modern times cases are more closely watched for it. The possibility of Bright's disease without albuminuria must be borne in mind.

Adenitis producing a moderate degree of glandular enlargement occurs in almost all cases of scarlet fever, but in severe cases it becomes a painful and grave complication. A majority of cases subside, but some go on to extensive and destructive suppuration, of which we have known ulceration through the carotid artery a consequence.

Arthritis ensues in a certain number of cases, and closely resembles that of acute rheumatism. The term Scarlatinal rheumatism is not justified any more than is the term gonorrheal rheumatism. Each is the result of the specific cause of the disease, and not of the cause of rheumatism. It occurs usually at defervescence, and recovery is almost invariable. Suppuration in the joint has, however, occurred.

Otitis is one of the most serious and permanently harmful of the complications. It is commonly considered the result of an extension of inflammation from the throat through the Eustachian tube to the middle ear, and is associated with the streptococcus. It has occured after recovery was supposed to have taken place. On the other hand, it sometimes happens quite early in the disease. Suppuration and perforation of the membrane of the tympanum are common, and more rare is destructive suppuration of the mastoid cells. As a consequence of one or both of these, it almost always leaves impaired hearing or total deafness. The facial nerve may become involved in the disease of the labyrinth, producing facial palsy, while thrombosis of the lateral sinuses may be another result of the same condition. Meningitis and death may be later consequences.

Meningitis may arise independently of otitis; in fact, scarlet fever is the most frequent cause of meningitis, after cerebrospinal fever, tuberculosis, and syphilis.

Various other *nervous affections* develop as rare complications. Among these may be mentioned chorea, convulsions followed by hemiplegia, and ascending spinal paralyses with wasting limbs.

Of *thoracic complications* endocarditis and pericarditis not infrequently develop during the course and during convalescence from scarlet fever. Endocarditis is not always discovered, and a few unexplained chronic valvular defects may have originated in this way and thus be accounted for. Pericarditis is less likely to be overlooked. Pleurisy may also occur, and more rarely pneumonia.

Diagnosis.—The diagnosis of scarlet fever is easy if the symptoms are well developed, but it is the mild cases that escape detection. In the absence of the eruption in a distinctive form, it is sometimes impossible to aver the presence of the disease. Two new diagnostic signs of scarlet fever have been recently suggested, first by Umber (see Medizinische Klinic, February 25, 1912). This is a urinary action brought about by the addition of two

drops of a preparation made by tritrating in a mortar 30 grams of concentrated hydrochloric acid, and 2 grams of paradimethylamidobenzaldehyde and diluted with 70 c.c. of water. This will bring about a red coloration, sometimes in the cold urine, sometimes in urine boiling.

Another sign discovered in 1911 by Dohle (Centrablett f. bact., November 23, 1911) there were certain inclusion bodies found in the polymorphonuclear-leucocytes of scarlet fever. These inclusions by further examination have been proved to be due to streptococcic infection, and may be of value in differentiating the various skin eruptions. If there be a doubt as to the eruption, close watching will sometimes discover signs of desquamation in the shape of scales beneath the underclothing or in the stockings. In the absence of this the question must occasionally remain forever unsettled. At others the unfortunate development of a nephritis sets the matter at rest. If there has been exposure to the contagion, it is best to regard every case of sore throat as a possible case of scarlet fever, and treat it accordingly. While the throat affection of *diphtheria* closely resembles at times that of scarlet fever, where this symptom is at all conspicuous in scarlet fever the eruption is not generally wanting, or is, at least, present to such extent to permit recognition of the disease. The fact that the one or the other of the two diseases is prevailing may settle the question. It must be admitted, too, that the two affections may succeed each other, and even, perhaps, coexist both events being, however, exceedingly rare. The diagnosis of diphtheria is rendered certain by obtaining a successful culture of the Klebs-Loeffler bacillus. The facilities furnished at the present day by the municipal laboratories to this end make it easy to obtain this test.

The coryza and cough in *measles* characterize the stage of invasion, while the eruption occurs later than in scarlet fever. When it does come it is very different, being at first, at least, in patches bounded by irregular and crescentic outlines, more uneven and elevated, and is conspicuous in the face, where the scarlet fever eruption is faintest. The time of the appearance of the rash is important. This is early in scarlet fever. The absence of sore throat is distinctive of measles, though its occasional presence in mild degree must be admitted in the latter disease. Leucocytosis is present in scarlet fever and absent in measles.

Rotheln, or rubella, has an eruption more like that of scarlet fever than is the typical measles eruption, but it is not usually followed by desquamation. There are no uncomfortable throat symptoms, and the constitutional disturbance is much less. It is also of much shorter duration. It is possible, too, that these affections may succeed each other, as is true of real measles and scarlatina.

Acute exfoliating *dermatitis* resembles scarlet fever during the eruption, but the exfoliation in the former is not like that of scarlet fever. As in erysipelas, it has more the appearance of scales and crusts before it is thrown off, and there is more apt to be a moist surface left behind, followed by a second exfoliation. There are no throat symptoms, and the tongue characteristic of scarlet fever is wanting. The eruption caused by belladonna, both on the skin and throat, resembles that of scarlet fever, but it is of short duration and without constitutional symptoms.

Prognosis.—The prognosis of scarlet fever varies greatly in different

epidemics. There are epidemics of great severity, in which the mortality is large, and certain fulminating cases are beyond treatment. Yet most physicians of large experience in surveying their work will recall that the percentage of deaths in their scarlet fever cases has not been large, and that it has been greatest among the very young. The percentage of deaths is put down at from five to ten per cent. in mild epidemics, and 20 to 30 per cent. in severe ones. The mortality is greater in hospitals than in private practice. In the fulminating cases death takes place before a chance for treatment is offered; but in the next grade of cases, characterized by high temperature and severe throat symptoms, a survival of five or six days generally means recovery, unless the supervening complications carry off the patient. Among these, nephritis and adenitis passing over to abscess are conspicuous, but even of those so afflicted a majority recover.

Treatment.—After isolation and protection in bed against changes of temperature, the treatment of scarlet fever is, in the main, a symptomatic one, associated with a vigilant nursing that will guard against complications. The patient should be isolated, if possible at the top of the house, and all communication with those of the family who have not had the disease interdicted. The temperature of the room should be uniform, while effective ventilation should be secured. The diet should be liquid as long as the fever persists, and the best of all liquids is milk, though light broths are allowed as is also an abundance of water.

If the *fever* is high, say above 103° F. (39.4° C.), cool sponging may be resorted to, but it is to be remembered that high temperature in this disease is usually of short duration and not likely, therefore, to produce the mischief it may cause in long-continued febrile diseases like typhoid fever. Very high temperature, such as 105° F. (40.5° C.), with meningeal symptoms, may require the tub-bath or cold pack, but the temperature of the tub-bath should not be so low as that used in typhoid fever. It is safer to put a patient in a bath at 90° F. (32.2° C.) and gradually reduce the temperature. The warm bath allays the itching of the skin, but this is as well accomplished by inunction with cold cream or sweet oil, and this unguent is important for another purpose as soon as desquamation takes place, to keep the scales from flying about and spreading the contagium. An ice-cap may be applied to the head if the temperature be high, and especially if there are head symptoms. While cool applications are allowable during fever, they are positively contra-indicated in its absence, as they may act in the development of complications of nephritis and otitis.

Fever is best controlled by these measures, but it is desirable to give medicines which tend to the same purpose, especially if they dispose to diuresis as well. Hence, the officinal solution of citrate of potassium or of the acetate of ammonium combined with the spirit of nitric ether, or a couple of drops of aconite with a little flavoring syrup, is useful. Constipation should be guarded against.

The *throat* symptoms require to be treated according to the degree of their severity. Iron and potassium chlorate may be added to the above mixture. If more active local measures are needed, the throat may be sprayed frequently with a saturated solution of boric acid or a normal salt solution. Irritating applications should never be applied. The first is

the best. Great care must be taken not to exhaust the child by spraying. Much harm may be done by holding a struggling child and applying a spray. Cold water applications, and ice to the exterior of the throat, are very comforting to the patient. Very efficient and soothing is a bandage for the throat with pockets opposite to the tonsils, into which pieces of ice are placed and the whole covered with a dry towel; or little india-rubber ice-bags may be similarly used. In adynamic cases stimulants and restorative treatment in general are indicated. Due regard should be had to the tendency of the disease in severe forms to produce degeneration of muscle and the liability of the heart to share in this.

The proper treatment of the throat tends to save the *ear*, but should the middle ear become involved, the membrane should be watched daily, and if the tension be extreme, perforation practised, even more than once, if needed. Too little attention has been paid to this complication, and if circumstances permit, an aural surgeon should be called in.

The *prophylaxis* against *nephritis* should be most careful. Whatever may be the immediate cause of the renal involvement, it is certain that cold often becomes its exciting cause. Hence, the patient should be scrupulously guarded against drafts, and, tedious as it may sometimes seem to mother and child, "six weeks in the room" is a precaution which will avert many a case of nephritis.

The treatment of complicating nephritis is the treatment of that affection under other circumstances, and the reader is referred to the appropriate section on it.

Serum Treatment.—An important addition to the treatment of scarlet fever has been made by Paul Moser[1] who suggested the use of antistreptococcic serum, not with a view to combating the disease itself, but the complications which are the result, not of the scarlatinous, but of the streptococcic infection. G. A. Charlton,[2] of Montreal, and W. R. Hubbert, of Detroit, have repeated Moser's treatment with gratifying results. Charlton says that he employed it in 15 cases, the majority of which would, in his judgment, under ordinary treatment, have terminated fatally, or, at least, have suffered from lingering and troublesome complications. There were 13 prompt recoveries and two deaths, one case having been in a dying condition, and the other complicated by pneumonia when they came under treatment. The frequency of mixed infection is shown by Moser's statement that in 99 cases of scarlet fever streptococci were obtained from blood 63 times. These observations have been amply confirmed by other bacteriologists.[3] The injections should be made early in the disease. Shick[4] says the use during seven years is satisfactory. Two hundred cubic centimeters must be used for young children, 100 c.c. for infants. After the injection of the serum a rapid subsidence of the pyrexia supervenes, also a corresponding decrease in the pulse rate, with improvement in its tension and rhythm. This seemingly harmless treatment demands a prompt trial for the relief of the dangers of this serious disease.

[1] "Ueber die Behandlung des Scharlachs mit einen Scharlachsstreptococcen serum," "Wiener klinische Wochenschrift," October 9, 1902.
[2] "Montreal Medical Journal," October, 1902.
[3] For a more detailed account of these observations see "Die Bakteriologie des Blutes bei Infektionskrankheiten," von Dr. Med. Canon Jena, 1905.
[4] Therap. Monasthefte, April, 1912.

Prophylaxis against the spread of the disease should be rigid and is accomplished by the same measures as those against the spread of diphtheria. (See p. 122.)

DIPHTHERIA.

SYNONYMS.—*Membranous Croup; Angina maligna; Angina membranacea; Cynanche contagiosa; Diphtheria faucium.*

Definition.—Diphtheria is an acute, contagious, inflammatory disease, caused by inoculation with the Klebs-Loeffler bacillus, and especially characterized by the formation of false membrane and by secondary constitutional infection. It may attack any mucous membrane, and even the skin, but, as usually employed, the term means diphtheritic inflammation of the oral, faucial, nasal, laryngeal, tracheal, or bronchial mucous membrane.

Etiology.—The specific organism which by common consent at the present day is the cause of diphtheria is the so-called Klebs-Loeffler bacillus, a bacillus, non-motile, with rounded club-shaped ends, 2.5 to 3 microns[1] in length, and from 0.5 to 0.8 micron in thickness. It stains readily by Loeffler's methylene alkaline blue in cover-glass preparations and in sections. Its cultures in blood-serum are small, round, grayish-white colonies that are characteristic. These, with the clubbed ends of the bacillus and clear spaces in its interior, giving it an appearance as if broken, suffice for its recognition. It grows on all the usual culture-media, but ceases to grow at a temperature below 68° F. (20° C.). If inoculation cultures are practised on the lower animals, the nature of the virus is declared by the exudation, the bacilli, the swelling of adjacent lymphatic glands, and the invariably fatal results of such inoculation. The bacillus produces in its growth a potent toxic substance, or tox-albumin, the absorption of which from the seat of local infection causes the general symptoms of the disease, which are therefore due to this toxin and not to an invasion of the blood by the organism producing it. The toxin is an albuminous substance, but its composition in unknown. When injected into animals, it produces paralysis, nephritis, and albuminuria. Roux and Yersin were the first to show, in 1888, the pathogenic property of cultures that had been filtered through porcelain.

The successful implantation of the bacillus of diphtheria is, dependent on various circumstances. Certain temporary states of the individual doubtless favor it, while others retard it. While general weakness or feeble resisting power may be one of these conditions it is likely also that purely local states, such as uncleanness of the mouth, teeth, and fauces, as well as chronic inflammatory conditions, may act as predisposing causes. Enlarged tonsils and nasopharyngeal catarrh predispose. It has been shown that there are different degrees of virulence in the contagious organism itself. Diphtheria bacilli are rarely found in the blood especially in that of the heart, in very severe infections.

The bacillus of diphtheria is associated with other pathogenic bacteria, such as *streptococcus pyogenes* and *staphylococcus albus* and *aureus*, *micrococcus lanceolatus*, and *bacillus coli communis*, which are probably responsi-

[1] A micron is a 1/1000 millimeter, or 1/25400 inch.

ble for suppurative processes often associated, as well as for certain deep-seated inflammatory conditions and certain forms of pseudo-diphtheria, which often complicate the disease and are sometimes mistaken for it. The streptococcus is probably the most active. In fact secondary streptococcus infection is often more dangerous than the diphtheria infection.

It was formerly believed that defective drainage, and to a less extent also the upturning of soil, were conditions favoring the production of diphtheria, but such views are not sustained by modern studies. The contagion is communicated, as a rule, through the air and not by fluids ingested, although epidemics have been traced to milk, in which the bacillus multiplies. In the vast majority of instances the source of the contagion is the throat or nose of another individual affected, whence the infective material is propelled by acts of coughing or expectoration. Hence it happens that the physician and nurse are not infrequently infected. Perhaps in this disease, more than any other, excepting typhus, are doctors and nurses the victims of contagion. Much may, however, be done to secure protection by caution during such ministrations, as by keeping the mouth closed and carefully cleansing the hands after contact. The practice of wearing a gauze mask over the mouth when examining patients is a further protection against inoculation of the examiner. The contagion is less tenacious than that of scarlet fever, but is highly so, having been found to live on blood-serum for 155 days; dried on silk threads, 172 days; and in gelatin, for 18 months. It has been found on a child's toy that had been kept in a dark place for five months and in the hair of nurses. It resides also in the healthy throats of immune persons, in simple catarrhal angina without membrane, and in what appears to be simple lacunar tonsillitis; whence it is plain that the cause is difficult to find in sporadic cases.

It is believed by some that diphtheria affects the lower animals, especially the cat, and may be transmitted from them to children. It is said, also, that such an affection attacks calves and heifers, and is from them communicable to man.

The disease is much more common in children than in adults, though no age is exempt. It is rare in very young children, and more girls are attacked than boys. Abraham Jacobi, whose experience has been very large, has seen only three cases in the newly born. Several cases in children about six months old have come under our notice. Epidemics vary in severity, and winter is the season in which the disease is most prevalent. While crowding in cities favors it, it is often widespread and virulent in the country.

Morbid Anatomy.—The morbid anatomy of diphtheria consists, on the one hand, in the presence of the false membrane and of the more ordinary phenomena of inflammation, most of which latter disappear after death; in the deep-seated ulcerative processes that sometimes result; and in the results of the complications and sequelæ to be considered later. The paralyses do not furnish palpable morbid products.

Under morbid anatomy the constitution of the false membrane is suitably considered. At its first appearance it is yellowish-white, but later may assume a grayish hue. Whether superimposed on a mucous membrane or set into it as in a frame, depends much upon the character of the epithelium

with which the surface is normally covered. To squamous epithelium the membrane is more deeply and thoroughly attached; to columnar epithelium, such as lines the larynx or bronchi, it is more loosely adherent; but in both situations it tends to become looser with the lapse of time.

The membrane itself is to-day considered a product of what is known as *coagulation-necrosis*, our knowledge of which is based on the studies of Wagner, Weigert, and especially of Oertel. The mechanism of its production is as follows: The diphtheritic poison, probably admixed with fibrin from the blood, infiltrates the wandered-out leukocytes and the epithelial cells of the part, especially the more superficial, causing first their death and then a hyaline transformation, and simultaneously coagulation. The resultant is a plate of *necrotic* tissue and *coagulated fibrin*. Hence the word "coagulation-necrosis." The membrane presents, also, a laminated structure, probably due to the involvement of successive layers of tissue and wandering cells. If forcibly separated, especially when recent, it is apt to leave a bleeding surface, on which new membrane is generally promptly deposited. The process proceeds from without inward, and, though usually superficial, may extend more deeply, invading lymphatic glands and adjacent tissue, producing foci of necrosis, which may be extensive. Blood-vessels may also be invaded, especially capillaries. Bacilli are everywhere present in the necrotic tissue, but they do not directly produce the mischief. It is caused by the toxin they generate. The same results may be produced experimentally.

Symptoms.—The *period of incubation* varies from two days to 12, seldom exceeding one week.

According to what may be the primary or principal seat of invasion we may speak of the *pharyngeal*, *laryngeal*, and *nasal* forms of diphtheria.

In the pharyngeal variety, *fever* and *sore throat* appear simultaneously, sometimes preceded by a chill or chilliness. Both increase rapidly. There may be aching or a sense of weariness. More rarely a *convulsion* ushers in the attack. At times at the beginning, at others on the second or third day, an *erythematous eruption* more or less extensive appears on the skin and may lead to the diagnosis of scarlet fever. Usually, as soon as attention is called to the throat, white patches are found on one or both tonsils, which spread with varying rapidity. *It is this spread from the original focus by which the disease is especially characterized as something distinct from follicular tonsillitis.* Commonly, the extension is anterior, over the anterior half-arches to the uvula, and to the palate or up into the nasal passages, or both. With the invasion of the uvula and palate, commonly reached about the fourth day, the diagnosis becomes certain. Bacteriological examination must settle the diagnosis before this, however. More serious is the extension backward into the larynx, producing croup.

The *temperature* rises to 103° or 104° F. (39.4° or 40° C.), but is not characterized by extreme or persistent elevation. The *pulse*, which ranges from 120 to 140, is never very full and strong, but tends early to smallness and weakness. *Delirium* is rarely present. *Deglutition* becomes more and more painful, and is increased by *external glandular swelling*, involving the lymphatic and salivary glands, although this swelling is not invariably present. As the *nasal passages* become involved, breathing becomes more

and more obstructed, until, finally, it is possible through the mouth only. The *Eustachian tube*, *middle ear*, and even the *antra* may be invaded. So, also, there may be *diphtheritic* conjunctivitis, and even keratitis, and, though rarely indeed, dermatitis. Should there be, however, excoriations or wounds, these may be invaded by the diphtheritic pseudo-membrane. Such false membrane may, however, be due to the streptococcus, which requires a bacteriological examination for its recognition.

As intimated under the head of morbid anatomy, the *ulcerative process* may extend much more deeply, producing destruction of tissue and even gangrene, resulting, as in scarlet fever, in a fatal erosion of blood-vessels. Usually, the membrane gradually disappears from the fauces as convalescence is established, or is coughed up if deeper in the respiratory passages. At times, on the other hand, it remains on the tonsils for some days after all constitutional disturbance has disappeared.

If the inflammation and membrane formation extend downward, *laryngeal cough* and the signs of laryngeal obstruction become superadded —in a word, the symptoms of *laryngeal diphtheria* supervene. Of if the process begins in the larynx—*primary laryngeal diphtheria*—we have croup at the outset, which differs from spasmodic croup in being less sudden in its onset. The seriousness of the disease is greatly aggravated by the possibility of complete obstruction and suffocation unless averted by operative interference. Not the larynx alone, but the trachea and bronchi may be invaded by false membrane. While the onset is slower than that of pharyngeal diphtheria, the course is more rapid. To the phenomena of congestion and membrane formation with resulting obstructions are added those of spasm, which bring on at intervals the alarming paroxysms that add to the terrors of this horrible affection.

Nasal Diphtheria.—When the nares are invaded by the membrane in the course of pharyngeal diphtheria the child frequently becomes seri-seriously ill because of the greater absorption of the toxin from this area. *Membranous rhinitis* while it gives rise to few symptoms has been proven by Albott and by Park to be a true diphtheria of low virulence. The child is not sick, the nares are plugged by a dense membrane but this condition can give rise to a virulent case of diphtheria in another individual. The membrane contains diphtheria bacilli of varying virulence.

In three to five days after the onset, if the case is one of ordinary severity, the phenomena of *constitutional infection* make their appearance in the shape of extreme *adynamia*, *feebleness of pulse* and *heart-beat*, while a sense of *intense weariness* is complained of. From this time a new period of danger begins, the danger of death from vaso-motor paralysis. At times in diphtheria, as in scarlet fever, the signs of constitutional poisoning appear at the outset, and the patient is struck down as by a blow, but this is less common than in scarlet fever. In such cases the temperature may not rise, and may even be subnormal. Constitutional poisoning is not so prone to take place in primary laryngeal croup as in secondary croup. This lesser tendency to constitutional poisoning together with the more gradual onset, the spasm, the slighter contagion, the shorter duration, and more serious mortality, constitute the chief clinical features of the laryngeal variety.

Complications and Sequelæ.—The most frequent complication of

diphtheria is *nephritis*, which pursues a course somewhat similar to the nephritis of scarlet fever, but is less frequently accompanied by dropsy, and generally terminates more favorably. On the other hand, albuminuria is present in almost every severe case. There may be the other signs of nephritis—viz., blood-casts, epithelial casts, scanty and even suppressed urine. *Capillary bronchitis* and *bronchopneumonia* are serious complications, especially if the result of insufflation of the virulent membrane. *Endocarditis* and *arthritis* sometimes occur.

The most important sequel of diphtheria is *paralysis*. This is now generally regarded as the result of a *toxic neuritis*. It may come on as early as the seventh or eighth day, or as late as the second and third week, when convalescence is apparently established. It is quite as likely to follow mild cases as severe ones. It may even follow wound-diphtheria. It most frequently affects the *palate*, producing nasal speech and permitting the passage of fluids into the posterior nares and through the nose. There is simultaneous *anesthesia of the pharyngeal mucous membrane*, destroying reflex excitability. Next in frequency of involvement are the muscles of *deglutition;* more rarely, the *eye muscles*, especially those of accommodation, which is thereby rendered defective. There may be also ptosis and strabismus, or paralyses of the distribution of the facial nerve. Still more rarely the nerves of the lower extremities are involved, producing paralysis, partial recovery from which leaves lameness that may last through life. Generally, however, recovery takes place in the order of involvement, usually in two or three weeks. Sometimes there is a general multiple neuritis giving rise to *ataxic symptoms*, with *loss* of the tendon *reflexes*, but no involvement of sensation.

The most serious of the local palsies is that of the *heart*, due probably to fatty degeneration of the heart muscle fibers, though a degeneration of the nerves may take place in some cases. In this there may be bradycardia and tachycardia but the most frequent result is the sudden cessation of the heart's action, and this tragic termination may take place during convalescence. Indeed, the event is more frequent during convalescence, and is often as late as the sixth or seventh week. At other times the phenomena of heart failure are more slow in their development. The pulse may become weak and rapid, or more rarely become slow, while the extremities become cold, the temperature falls, and there supervene in a few hours all the signs of collapse. A most striking instance of bradycardia in diphtheria was met by Baumgarten, wherein, toward the close, the pulse fell to 25, though very regular. It must be remembered that the sudden failure of circulation at the height of an attack of diphtheria as in other infectious diseases is not really due to cardiac failure, but to a failure in the peripheral tone, a massing of the blood in the splanchnic area.

Diagnosis.—The only two conditions with which diphtheria is liable to be confounded are, first, the different forms of sore throat, including follicular tonsillitis, and, the sore throat of scarlet fever. The difficulty in deciding between the former condition and dipthheria at the outset is sometimes extremely great, and time or the bacteriological investigation may alone settle it, therefore, routine examination of exudative sore throats should always be made. The primary fever, constitutional disturbance, and dysphagia are often equally as great in follicular tonsillitis due to strep-

tococcus or some other infecsion. As a rule, however, the follicular exudate remains limited in extent—it does not spread, and in the second or third 24 hours is apt to drop out, leaving a clean-cut ulcer that heals rapidly, while the constitutional symptoms disappear with equal rapidity. In the form of follicular tonsillitis attended by multiple white spots on the tonsils the local resemblance to diphtheria is even greater, but the white spots remain isolated while those of diphtheria spread.

Sometimes, however, the mass of desquamated epithelium, fibrin, and fungous filaments, which make up the contents of the follicles in follicular angina extend outside of the follicles and over the surface of the tonsils. Then it becomes more difficult to decide. It does not, however, pass the boundary of the tonsils. The follicular fungi are said to stain bluish-red with an iodopotassic iodin solution. Further certainty is secured by making cultures from the membrane, a small portion being removed by the sterilized platinum loop or cotton swab, and planted in gelatinized blood-serum. In diphtheria in the course of 24 hours characteristic colonies of the Klebs-Loeffler bacillus will develop, and the microscope will confirm the diagnosis. A smear of the exudate may be made and microscopic examination give an immediate diagnosis. This can and should be done by every physician. If he has not learned the use of the microscope, he should at once do so, or have proper clinical facilities to have it done for him.

From *scarlet fever*, diphtheria is usually easily distinguished by the absence of eruption, although this aid is wanting in those few cases of scarlet fever in which there is no eruption, and in those of diphtheria where there is an erythematous redness. Under these circumstances the distinction becomes more difficult if the throat symptoms be similar, as they sometimes are. The prevalence of an epidemic of one or the other disease aids in the decision. Later on, the desquamation that takes place in scarlet fever, but not in diphtheria, also settles the question.

Diagnosis is sometimes delayed or the disease entirely overlooked by concealment of the membrane in localities not easily open to examination, as in the nasal chambers. Hence, in all obscure cases these should be examined. Indeed, it is not impossible that diphtheria may exist without membrane, as evidenced by prompt recovery after the use of antitoxin in certain obscure throat cases with continued adynamia and fever.

The larger cities in the United States now offer, through their health bureaus, to make bacteriological examinations for physicians in all cases of possible diphtheria. Outfits are left at stations. They consist of a box containing a tube of blood-serum and another containing a sterilized swab.

Prognosis.—The introduction of the serum treatment for diphtheria, which may be dated April, 1893, when the first 30 cases treated by Behring's normal serum were reported,[1] marks an era prior and subsequent to which the prognosis of diphtheria presents very different aspects. Even prior to 1893, while the prognosis was so unfavorable as to justify a wholesome dread of the disease the world over, many moderately severe and most mild cases got well. Allowing for the great variation in the percentage of fatal cases in different epidemics, and especially at different ages, the very careful and

[1] The prior trials of immune serum in the treatment of human diphtheria, made in v. Bergmann's clinic in Berlin in 1891, and by Henoch and Huebner in Berlin in 1892, were tentative and made with weak serum and in insufficient doses.

reliable studies of Professor William H. Welch,[1] of Johns Hopkins Hospital, make it safe to put such mortality at a minimum of 40 per cent. Where the larynx was involved, it amounted to almost 100 per cent. Of the remaining nonlaryngeal cases probably one-third died. Since the introduction of the antitoxin treatment the studies of the same observer (Welch) show a reduction in mortality of between 50 and 60 per cent. As near as it may be possible to put in, the mortality since the introduction of antitoxin has been from 8 to 25 per cent. This improvement affects all classes of cases, including those operated on as well, and is attested from many sources. For example, in the report of collective investigation by the American Pediatric Society we have the following: "Formerly, 27 per cent. approximately represented the recoveries, while now 27 per cent. represents the rate of mortality" also "Formerly, only ten per cent. of laryngeal cases did not require operation, while now with antitoxin treatment 17 per cent. do require this procedure." Finally, the most remarkable results are shown in the "Bulletin of the Department of Health," city of Chicago, for February, 1899, which reports that out of 4071 cases of *bacterially verified* diphtheria, 3705 recovered and 276 died, giving a mortality rate of but 6.77 per cent. In New York City for 1899 there were 8240 cases reported with a mortality of 1087, or 13 per cent. The sooner the antitoxin is given in the disease, the less the mortality. Holt quotes the following statistics:

During 13 months ending October, 1896, 1972 patients were treated with antitoxin at the Boston City Hospital, and of this number post-diphtheritic paralysis occurred in 5.8 per cent., which percentage is smaller than that of cases not treated with antitoxin. A fair ratio of the causes of death in 25 fatal cases prior to the use of antitoxin was given in a paper by William P. Munn[2] as follows: from septic intoxication eight, laryngeal stenosis seven, cardiac paralysis six, hemorrhage from the bowels one, nephritis one, unknown two; total 25. Thus the chief causes of death are adynamia, laryngeal obstruction, heart paralysis, or suffocation from paralysis of deglutition; more rarely nephritis and bronchopneumonia. Hemorrhage from an eroded blood-vessel is a possible cause of death. Morse analyzed 366 deaths occurring in 1972 consecutive cases treated since 1895 in the Boston City Hospital, and found the mortality only 18.5 per cent. Seventy of these cases died on the day of admission, and 38 on the following day; in other words, 100 were moribund on admission. The following are the causes of death: sepsis, 107; bronchopneumonia, 91; cardiac complications, 52; exhaustion, 13; tuberculosis, one; empyema, one; typhoid fever, one; moribund when admitted, 100; total, 366.

Under the use of antitoxin the average duration of an ordinary case may be put down at about five days and of a very bad case ten days. It is important to remember, however, that actively growing bacilli can be cultivated from the throat of cases treated early with antitoxin, two weeks after the membrane has disappeared.

Treatment.—In the management of every case of diphtheria there are two principal indications: first, to combat the toxin and thereby neutralize

[1] "The Treatment of Diphtheria by Antitoxin." Reprint of paper read before the Association of American Physicians, May 31, 1895, and published in the "Transactions" for that year.
[2] "Diphtheria: A clinical Study," "Medical News," Philadelphia, March 25, 1893.

constitutional infection; second, to co-operate with this object by suitable supporting treatment.

I. To combat the toxin and to prevent constitutional infection. This is accomplished (*a*) by serum therapy, that is, by antitoxin; (*b*) by local antiseptic measures.

(*a*) *Antitoxin.*—The treatment of diphtheria by antitoxin is based on the fact that animals may be made immune to diphtheria by the injection of diphtheria toxin, and that the serum from such animals is antitoxic to the toxin of diphtheria. This was shown by Behring in 1891, after some preliminary experiments had been made by Fränkel in the same year. In 1892 Behring and Wernicke employed this method successfully in immunizing sheep, and also ascertained the second important fact mentioned that blood-serum from an immune animal could be used with success in arresting diphtheritic infection in susceptible animals. To this was added the further important fact that a *smaller amount of serum is required to produce immunity than is necessary* for the cure of *an animal already infected. If the injection be made immediately after infection, from one and a half to twice as much is required; eight hours after, three times as much, and 24 to 36 hours after infection the dose required is eight times the immunizing dose.*

One of the objections to the serum treatment at first was the necessarily large bulk of the injection. This has, however, been reduced by increasing the strength of the serum, so that the dose now injected gives no more discomfort than a hypodermic injection of morphin. Reliable preparations are now made in this country by various well-known firms, and in some cities by the official authorities under direction of the city board of health. The manufacture should be placed under governmental control.

Technique of the Administration of Antitoxin.—Antitoxin should be administered *at once* if there is a reasonable probability of the presence of diphtheria, without waiting for the bacteriological diagnosis. Antitoxin does no harm where the disease is not diphtheria, and delay in a true case may be fatal.

Very much larger doses of antitoxin are given now than formerly. Thus the beginning dose was 1000 *units* for ordinary pharyngeal diphtheria. Now 3000 units are a frequent initial dose, and even more is given in bad cases, 7000 to 10,000. In laryngeal diphtheria at least 5000 units should be given at the first dose. The "concentrated" form is preferred on account of its small bulk.

A large hypodermic syringe is used for the administration. The syringe must be made sterile by boiling for five minutes just before being used. Always test the syringe with water before filling with serum. *After* the administration the syringe should be washed out with clean cold water. At the present day the serum is dispensed in proper dose in a glass syringe with a needle point attached, by which the serum is injected, thus avoiding the manipulation necessary of transfer from bottle or tube to syringe. The injection is given in the back just below the scapula or in the flank or buttock, the skin being cleaned with soap and water followed by alcohol. If the smaller bulk be used, it can be injected quickly. If the larger be used, inject slowly in order to avoid injury to the underlying

tissues by too rapid stretching. Immediately after the injection there is an occasional rise of temperature, which need give no concern, or an eruption may appear which is equally harmless.

In favorable cases, after 24 hours have passed, the temperature will not have risen; the pulse will be slower; the membrane will *not* have spread; the mucous membrane at the edge of the exudation will be bright red in color. There will be a feeling of diminished discomfort and revival of spirits. These are favorable signs, and a second dose need *not* be administered. A second dose is administered after 12 hours if the temperature has risen, if the membrane is spreading, and if the general condition of the patient is not so good as at the previous injection. As might be expected, improvement is more rapid in mild cases.

W. K. Sutherlin reports a case of diphtheria in which 498,000 units of antitoxin were used[1] in a protracted case with relapses between Aug. 22 and Oct. 17. On the 7th, 8th and 9th of October, she took 32,000 units daily.

For Immunization.—For producing immunity to those subject to infection from diphtheria, immunizing doses should be administered. These range from 500 to 1000 units, according to the age of the person to be protected. Infants and very young children are easily protected by the smaller dose. Adults, especially those in attendance upon the sick, should receive the larger dose. Persons who have been exposed and probably are already infected should receive 500 units. The throat irritation so common in those who are attending diphtheria is said to have yielded promptly to a dose of 500 units. If suspicious symptoms have appeared, not less than 1000 units should be given.

Immunization cannot be too strongly insisted upon. The protection afforded by one dose will last for at least three or four weeks, at most not more than eight or ten weeks; within which time, with proper means of disinfection, the source of infection should be eliminated.

Behring and others declare that the diphtheria antitoxin has no injurious effect upon animals in the largest dose in which it has been employed, and that, aside from its antitoxic powers, its properties are entirely negative, as far as human beings are concerned. This is essentially true; but a few cases have died suddenly as the result of the injection of the serum. Therefore it is a wise precaution to inject only a drop or two at first, wait ten or fifteen minutes and inject the rest if no reaction has taken place. If there is any sign of shock no more should be injected. The few fatal cases are probably the result of anaphylactic shock.

Serum sickness is now very well recognized. It is an anaphylactic phenomenon. It is the result of the introduction into the economy of a foreign proteid. Its symptoms vary in severity from a local irritation to severe constitutional symptoms. In from six to twelve hours after the injection the temperature rises and severe local pain at the site of injection accompanied by redness and edema appear. The pain is frequently so severe that the patient has curled up on his side refusing to be moved. In from 24 to 36 hours the soreness disappears without treatment. Occasionally there are severe joint pains or the patient's skin may be covered with an urticarial rash. These symptoms may appear as late as six days

[1] "Medical Record." Shreveport, Louisiana, Jan., 1905.

after the injection. Cases are on record where the mucous membrane of the respiratory passages have been covered with urticarial rash.

Local treatment should be confined to keeping the throat clean and clear of mucus. To this end a spray of boric-acid solution or normal salt solution should be used. The spray should be used through the nose as well as through the mouth. No irritating solution should be used. As stated above the child should not be *forced* to have the spraying done. The wash may be gently applied by washing out the nares by means of a douche.

II. The second object includes measures which also have for their purpose, first, checking the spread of the membrane, its loosening and solution, and, second, maintaining the strength of the patient against the depressing action of the absorbed toxin. (*a*) The former is accomplished by the preparations of mercury. Of these, the bichlorid of mercury in doses of 1/48 grain (0.0027 gm) to 1/12 grain (0.005 gm.) for an adult, in conjunction with tincture of the chlorid of iron, may be given every two hours, taken freely diluted. The former dose makes 1/4 grain (0.0162 gm.) of the bichlorid in 24 hours, but as much as one-half (0.032 gm.) may be given in that period. These doses are given to adults, and they need not be much reduced for children. There need be little fear of poisonous effects from the bichlorid, as bowel irritation, pain, and loose movements give warning before any more serious consequences supervene. When these symptoms appear, the bichlorid should be discontinued or the dose decidedly diminished.

(*b*) Iron is also useful in supporting the strength of the patient. Strychnin in 1/60 grain .001 dose every 3 hours and caffein 2 grains every 3 hours for an adult are the most useful stimulants. The strychnin can be used hypodermically. Stimulating, nourishing, and easily assimilated food is necessary. Milk is to be preferred to all else, fortified with whiskey or brandy, 1 dram to 2 drams (4 to 8 c.c.), every two hours, being required in all cases of severity, and proportional doses for children. The milk may, of course, be alternated with nutritious animal broths. In extreme cases of difficult deglutition nutrient enemas may be useful, but nourishment by the stomach-tube, if possible, is more efficient. For enemas, peptonized milk is the most suitable. To this brandy or whiskey may be added, if needed. Rectal alimentation has sometimes to be discontinued because the enema is made too large and is too frequently administered. Once in six hours is often enough, and 4 ounces at a time are as much as the rectum will commonly bear, although this quantity may be gradually increased. Smaller quantities should be used for children.

Treatment Demanded by Special Indication.—Where *laryngeal obstruction* is imminent, intubation or tracheotomy should be performed. Lives have been saved by both of these operations. Intubation may precede tracheotomy, as its use does not preclude the more serious operation at a later date, if the obstruction increases.

In the *nasal* variety of *diphtheria* special means must be employed to disinfect and cleanse the nasal passages. The solutions recommended to spray the throat may be used for such cleansing. Gentle injections into the nostril may be more efficient than the spray, precaution being taken to keep the mouth open, by which the entrance of fluid into the Eustachian

tube is guarded against. The injections should be continued until the fluid has free exit either by the other nostril or through the mouth. Jacobi recommends that when about to bring the injection to a close, the nasal cavities should be pressed together for an instant with the fingers, as in this way the fluid is forced backward into the pharynx and swallowed or ejected through the mouth, thus washing both at the same time.

The Treatment of Complications and Sequelæ.—Complications are treated as the same conditions under other circumstances, and the paralysis so frequently succeeding upon diphtheria, alone requires special allusion. The prognosis is, on the whole, good, and time, under favorable circumstances, mainly effects the cure, and during this the most important measures are those that save the patient from accident. Thus if there is paralysis of the muscles of deglutition, liquid food only should be used, and it may be necessary to nourish for a time by the rectum or by means of the stomach-tube. So, too, undue exertion should be avoided. Electricity and tonics, especially strychnin, are indicated. The former is applied to wasting muscles, and may be advantageously associated with massage. Strychnin should be given in full doses, ascending gradually to 1/20 grain (0.003 gm.) three and four times a day, with appropriate reduction for children. Iron and quinin should also be given.

The electrical treatment for paralysis of the pharyngeal muscles is applied in the following manner: An electrode is placed at the back of the neck and a very small electrode is touched to the velum palati, and a rapidly interrupted faradic current of moderate strength applied. Galvanism may be similarly used. A specially constructed electrode is also applied to the throat.

Prophylaxis Against Diphtheria.—Most important are the precautions necessary to prevent a spread of the disease. To this end the patient should be isolated, all carpets and unnecessary furniture and hangings should be removed from the room, and all utensils used in treatment should be kept apart and separate for the patient's own use. Spoons should be kept in carbolic acid solution, or, better, thrown into water kept boiling. Tongue depressors should be made of wood and burned immediately after using. All bed linen and clothing removed from the patient should be boiled, being immersed in water before removal from the room. Mattresses, pillows, and woolen garments too good to be destroyed should be exposed to superheated steam in establishments provided for the purpose in the cities; or they may be disinfected at the same time with the apartment occupied by the patient. They should be opened and suspended in this apartment, of which all the doors and windows must be closed tightly and the room fumigated with formaldehyd gas, of two to four per cent. volume strength, for not less than 12 hours. Suitable lamps are provided for this purpose. If formaldehyd is not available, sulphur may be used. The sulphur, in the amount of two pounds to every ten square feet (2 kilos to every 2.5 meters) should be placed in iron pans and these supported by bricks in washtubs containing a little water. The sulphur is then ignited by glowing coals or by burning alcohol. The room should be kept closed for twenty-four hours. After this fumigation the articles of clothing should be hung out in the open air for several hours, and the doors and woodwork washed

well with a solution of corrosive sublimate, 1 to 1000, while the walls should be wiped down with a similar solution.

Finally, physicians and nurses in attendance on the patient should wear gowns which will be left behind on leaving the room. The gown can be kept in a tight receptacle containing formaldehyde between visits. The hands should be washed before leaving the room, first in soap and water, and finally rinsed in corrosive sublimate solution, 1 to 1000.

As the bacillus has been found to multiply in milk, it is safer to use sterilized milk during an epidemic.

The convalescent patient should also be kept isolatedu ntil two consecutive negative cultures are obtained from the throat. On leaving the sick room they must be given first a hot water and soap bath, then washing the body of the patient with a solution of bichlorid of mercury, 1 to 2000, or two per cent. solution of carbolic acid, or, what is more agreeable, 25 to 50 per cent. alcohol. This should be done two or three days in succession. The hair should be cut or similarly washed with these solutions.

FOLLICULAR TONSILLITIS.

SYNONYMS.—*Angina follicularis; Lacunar Tonsillitis.*

Definition.—An infectious disease due usually to a streptococcus or staphylococcus and characterized by swelling of the tonsils with a whitish-yellow exudate in the crypts, or extending over the entire tonsil.

Symptoms.—The patient is suddenly seized with headache, aching limbs, sore throat and high fever. The tonsils are red and swollen. In the crypts of the tonsil is a yellowish exudate which can be easily removed by a swab. Sometimes the exudate covers the entire tonsil. Usually the exudate is easily removed but in certain cases there is a membranous formation which leaves a bleeding surface. The cervical glands are sometimes swollen.

Diagnosis.—It is extremely important to differentiate this condition from diphtheria and scarlet fever. From diphtheria the diagnosis is only certainly made by means of examination of a smear or of a culture. The absence of Kleb's Loeffler bacillus means no diphtheria. From scarlet fever the condition may be separated by the fact that the throat of scarlet fever is more uniformly red, the redness covering the whole soft palate and pharynx and also by the fact that a skin rash appears in scarlet fever within the first 24 hours. The exudate in scarlet fever is not confined to the lacunæ of the tonsils and is apt to be streaked over the part. Unquestionably many cases of endocarditis have their origin in acute tonsillitis.

The disease occurs in children and young adults, and is one of the affections sometimes mistaken for diphtheria. It is, however, something very different. It is a much less serious disease, of shorter duration, and patients never die of it. It is, however, probably infectious in origin, caused by a germ other than the diphtheritic, perhaps the streptococcus or staphylococcus. There is often very decided fever.

Treatment.—The treatment of this form of tonsillitis is definite and easily carried out. In the first place, cold should be applied to the neck by cloths wrung out in cold water or by ice, which is conveniently applied in

little muslun bags made to fit under the angle of the jaw and held in place by a bandage. Bicarbonate of soda may be used as a wash or the throat should be sprayed or gargled with a normal salt solution. The patient should be given a mixture of iron and bichloride of mercury. Acetphenetidin (phenacetin) and phenol salicylates (salol) will relieve the *joint-pains*.

VINCENT'S ANGINA.

Definition and Causes.—A form of sore throat described by Professor Vincent, of Paris, as due to certain spirilla and fusiform bacilli. It occurs in children eight to ten years old, or at about the time of the second dentition, and in adults 18 to 20 years, or about the time when the wisdom-teeth are appearing. It is met in all races and climates and, though more common in alcoholics and tobacco users, is found also in those who are not cleanly in the care of their mouths. Bad hygiene favors it and it is inoculable. The bacillus is bobbin- or cigar-shaped and associated almost always with the long wiry motile spirilla. The bacilli may reach 10 to 12 microns in length and a micron in width. They do not stain by Gram's method, but may be colored by ordinary stains, especially Wright's stain, thionin or Ziehl's solution. The spirilla do not stain as well.

Symptoms.—Clinically, the disease occurs in two forms: One, quite rare (two per cent.) resembles closely diphtheria, and the exudate contains bacilli with spirilla, though they may be associated with other organisms, as staphylococci and streptococci. The false membrane may be one to two centimeters in thickness, and be made up of an almost pure culture of the organisms described. There is a slight fever, the submaxillary glands are enlarged and there is pharyngeal pain. The illness lasts from four to six days.

In the second form (98 per cent. of cases) there is a "membranous ulceration" containing both organisms described. The onset is characterized by malaise, lassitude, pain in the limbs, headache and fever to 102.2° F. (39° C.). Most frequently one tonsil only is involved. The breath is fetid and there may be salivation. There may be an eruption like that of scarlet fever.

The pure form is characterized by the absence of the Klebs-Löffler bacillus of diphtheria, but the two conditions may coexist. It is also distinct from syphilitic stomatitis, though it may be engrafted on it.

Diagnosis.—The disease closely resembles certain cases of diphtheria, therefore a culture should always be made from the throat.

Treatment.—It is said to be easily cured by painting with iodin twice a day, but is not amenable to antitoxin.

If the diphtheria bacilli as well as the spirilla are present in the throat, a full dose of antitoxin must be given. This will cure the diphtheria and will not interfere with the Vincent's angina.

SMALLPOX.

SYNONYM.—*Variola*.

Definition.—Smallpox is an acute contagious disease especially characterized by an eruption which passes through the successive stages of papule, vesicle, pustule, desiccation, and desquamation.

Etiology.—The contagium of smallpox, probably the most unfailing of all the contagia in its effect upon the unprotected victim, is not certainly known an intracellular parasitic protozoön, was first clearly described by Guarnieri in 1892, and named by him the *cytoryctes variolæ*. The studies of Guarnieri were confirmed by Wasielewski in 1901, and more recently in 1903–03 by the exhaustive work of Councilman, Calkins, Tyzzer and their colleagues, but these observations are not confirmed by all observers.

The degree of mildness or severity of a case does not influence that of another caused by it, the severest cases being at times followed by the mildest, and *vice versa*. The contagium is very tenacious, and may be dormant for months in clothing or furniture hangings. No age nor sex nor race is exempt, but the number of cases in successive decades diminishes because of the immunity furnished by a previous attack. The *fœtus in utero* may acquire the disease from the mother, and the child may be borne with the eruption on it. Certain individuals are invulnerable even though unprotected by vaccination, while the mortality in aboriginal races is very great. Many alleged immunes respond to a proper vaccination.

Some difference of opinion exists as to the period at which smallpox is contagious. Welsh and Schamberg, in their book on Contagious Diseases, make the following statement, which may be considered as embodying the most recent views:

"Smallpox is undoubtedly infectious in all stages characterized by symptoms. It is alleged by some that the disease is even infectious during the period of incubation, but we think there is very little reason to believe that such is the case."

"The disease is least infectious during the initial stage, and most highly so during the suppurative and early period of the desiccative stages."

Morbid Anatomy.—The essential morbid anatomy of smallpox is that of the eruption as represented by its various stages and modifications, including hemorrhagic infiltration. To the anatomy of the eruption is added that of the complications that may occur.

The histology of the pustule shows that it starts from a single point in the *rete mucosum*, close to the true skin, whence it extends in all directions to a varying extent. The center or older area is a focus of coagulation necrosis, and about it the reticular spaces are filled with serum, leukocytes, and fibrin filaments. In the older area, too, the most highly developed of the cytoplasmic inclusions are found and in the peripheral area the smallest and presumably youngest forms. As long as the process does not extend deeper, healing takes place without a scar. In the more severe cases the papillæ of the true skin are invaded to various depths and destroyed by the infiltration, producing a loss of tissue constituting the pit.

Among other morbid phenomena may be mentioned a *hardness and firmness of the spleen*. *Cloudy swelling* of the *secreting cells of the liver and kidney* occur, as in other fevers with high temperature. True nephritis is rarely present.

Symptoms.—After a *period of incubation* of from seven to 12 days, and sometimes longer, the victim is seized with *violent muscular pain*, especially in the back. Often a *chill* or *chills* usher in the disease, and in children a *convulsion* may be the initial symptom. *Intense headache* is also

present. *Fever* sets in rapidly and the temperature reaches 103° to 104° F. (39.4° to 40° C.) the first day. The *pulse* is rapid, hard, and strong at this stage. *Delirium* may be present and is sometimes very violent. *Vomiting* and *diarrhea* are sometimes initial symptoms and may continue later *in the disease.*

About the second day the *initial rashes* make their appearance. These have been especially studied by Theodore Simon,[1] Knecht,[2] Scheby-Busch,[3] and William Osler,[4] although they are mentioned by some of the older authors, including Sydenham, Wood, Watson, Niemeyer, Trousseau, Marson, Munro, and others.

They include a *diffuse scarlatinous rash* and a *macular or measly form*, dark red in color and occupying a variable extent of surface. Either may be associated with petechial ecchymoses. Sometimes they are general, but as a rule they are limited to the abdomen, the inner surface of the thighs,

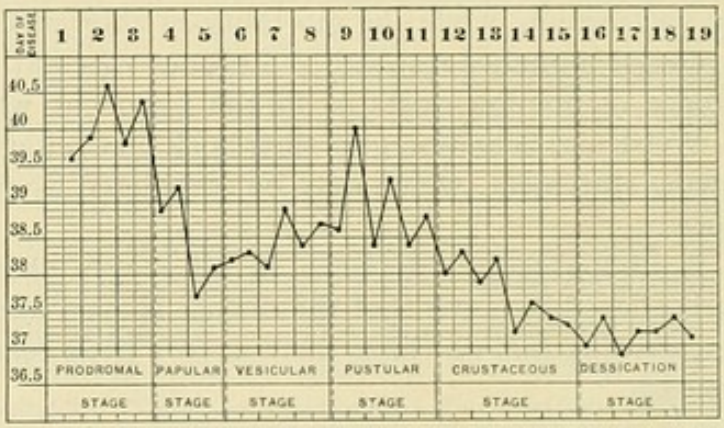


FIG. 17.—Temperature Chart of Smallpox.—(*Eichhorst*).

or the lacteral region of the thorax and axilla. Among Osler's cases was one of a true urticarial prodrome. While it is to be remembered that the coexistence of smallpox and measles and of smallpox and scarlet fever is possible, it is more than likely that the eruptions on which the diagnosis was based were really the initial rashes of uncomplicated smallpox.

On the *third day* of the disease, the *distinctive eruption* makes its appearance in the shape of *small red spots*, first on the forehead and wrists, whence it extends rapidly over the face and extremities, becoming quite general in the first 24 hours. At this time the temperature rapidly falls to again rise at the stage of suppuration. At this stage the eruption is not unlike measles, but in another 24 hours it is decidedly different. The papules have acquired shot-like hardness. With the appearance of the eruption the fever falls and the patient feels comfortable. On the *fifth* or *sixth day* a clear or slightly turbid *serum* makes its appearance. Coincident with this a depression is seen in the middle of each vesicle. It is *umbilicated*, and this umbilication is the most characteristic feature of the eruption. Frequently, a hair follicle passes up through the center of it. Umbilication is not usually present in the papular stage, its presence is not pathognomonic. By the *eighth day* the turbidity has increased until it is

[1] "Das Prodromal-Exanthema der Pocken." "Arch. f. Dermatol. und Syph.," Prag., Heft iii., 1870, 346; 1871, Heft ii., 242; Heft iii., 309; 1872, Heft iv., 541.
[2] "Arch. f. Dermatol. und Syph.," Heft iii., 1872, 372.
[3] "Arch. f. Dermatol. und Syph.," Heft iv., 1872, 506.
[4] "The Initial Rashes of Smallpox," "Canada Med. and Surg. Jour.," 1875.

bright yellow and the umbilicus has disappeared. The *pustule is complete.* The maturation takes place in the same order as the eruption appeared. With the appearance of suppuration the fever again returns, known as the secondary fever, and with it elevation of temperature and other signs of fever. There is a good deal of pain in the inflammed parts because of the tension. On the 10*th* or 11*th* day the *pustules* become *dry*, and by the 14*th* are converted into *crusts*, which drop off, leaving in mild cases a simple discoloration, in severe cases a more or less deep ulcer, or, if cicatrization be complete, a simple pit. The eruption may be found on the tongue and buccal mucous membrane and even in the pharynx, larynx, and esophagus, and pustules have been found in the stomach and rectum. In the trachea and bronchi there may be ulcers; also on the cornea. Sore throat, nausea, hoarseness, vomiting, and diarrhea may be consequences. With the drying of the eruption the fever disappears.

This description is typical of the course of the eruption in the simple discrete variety. It may be variously modified. The attack may be so virulent that the patient dies before the eruption makes its appearance, or it may be arrested at any stage. Sometimes blood forms the contents of the pustule, and there may be subcutaneous infiltration of the blood in addition. Along with this there may be hemorrhage from the mucous surfaces of the nose, stomach, or bowels, or there may be hematuria.

The pustules may be so close to each other that they join, when the case is *confluent;* or they may be separate and distinct, producing the *discrete* form. The variety with bloody infiltration is called hemorrhagic. The diagnosis as to whether the confluent or discrete form is present is generally made by an examination of the face, for it is an interesting fact that nowhere are the pock-marks more abundant than upon the face.

Sydenham early called attention to the fact that in the confluent variety the eruption appears earlier (on the third day), and its early appearance, according to him, is an indication that the case will be one of that variety. All the symptoms are much more severe. There is not the abatement of fever described as occurring on the appearance of the eruption. The face, hands, and feet present an almost continuous pus-vesicle, which often bursts in places, and the pus partly drying, there results a picture which is revolting. Such pronounced morbid changes must produce wide systemic exhaustion, as is manifested on the tenth or 11th day by growing weakness of the patient, an adynamia that frequently terminates in death. When recovery takes place, the secondary fever is the more prolonged the more widespread the suppuration.

The *hemorrhagic* variety of smallpox is still more severe. Two forms of it are described: One, the *purpura variolosa*, or hemorrhagic variola, in which the hemorrhagic symptoms appear early in the shape of a hemorrhagic rash while hemorrhage takes place from the mucous surfaces, generally on the evening of the second or third day. The patient dies in from two to six days, sometimes before the eruption makes its appearance. In the second form, *variola hemorrhagica pustulosa*, the case progresses at first like any other, and it is not until the vesicular or pustular stage that blood makes its appearance in the pocks.

VARIOLOID.—A third variety of smallpox is *varioloid*, which is variola

modified by vaccination or a previous attack of smallpox. In general, varioloid is smallpox bereft of all its serious features, each symptom being milder. The initial fever is less, the eruption is less general and may abort in its development, the secondary fever is less marked, and convalescence sets in earlier. Yet it has happened that both classes of individuals referred to, those having had smallpox and those having been vaccinated, have had very severe attacks, from which, indeed, the patients have perished. Generally, the longer the interval between the attack and vaccination, the more severe the former is. Similar is the mildness which characterizes smallpox produced by the direct inoculation of an individual from the pus of another, though the attack thus caused is more severe than that which follows vaccination.

Other names given to less important varieties are *variolæ sine variolis*, or variolous fever without eruption; the "crystalline pock," in which the eruption continues vesicular; and the "stone pock," "horn pock," and "wart pock," in which the vesicles dry up into horny, tuberculated or warty elevations.

Complications.—Among complications of smallpox may be mentioned laryngitis with fatal edema of the glottis, bronchopneumonia, parotitis, diarrhea, albuminuria, but rarely nephritis. Prolonged delirium, and even insanity, have supervened, while neuritis may occur during convalescence; so may arthritis. On the skin may be boils and painful acne. A troublesome and painful conjunctivitis used to be the result of indifferent care of the eyes, but it is now less common because of greater care in this respect. Corneal ulceration does, however, occur in two per cent. of cases and complete destruction of the eye has occurred in 24 hours in confluent cases, and in India is the most frequent cause of blindness. The specific pock does not, however, invade the cornea. Myocarditis and pericarditis sometimes occur, and most rarely endocarditis.

Diagnosis.—With the appearance of the perfect papule all doubt in the diagnosis of smallpox generally ceases. Ignorance of the initial rashes, measly and scarlatinal, has often led to errors of diagnosis. On the other hand, the resemblance of the eruption of *measles* to smallpox has also given rise to errors the result of which has been no less serious, because in consequence cases of measles have more than once been sent to smallpox hospitals with disastrous results. Never in measles is there such severe pain in the back as in smallpox, while the early cough and coryza are found only in measles. The lesson taught is to defer a positive diagnosis, because less serious mischief can result from an error thus occasioned than as the result of an opposite course. The possibility of *relapsing fever* being taken for smallpox has been alluded to in considering the former disease. *Cerebro-spinal fever* may also be simulated by the hemorrhagic form of smallpox. *Pustular syphilids* and accidental *croton-oil eruption* have been mistaken for smallpox, as has also chicken-pox. Secondary umbilication in the croton-oil pustule from collapse of the pustule may simulate the umbilication of the smallpox pustule, but it occurs late. Mild cases have been mistaken for acne.

Prognosis.—Unmodified smallpox is a serious disease, and the death-rate is always relatively large. It varies, however, at different ages, in different

races, and in different epidemics. The young die almost always. Thus, in the Montreal epidemic of 1885, 86 per cent. of the deaths were children under ten years. The African, American Indian and Native Mexican have perished by thousands. The range of the mortality in different epidemics is put down at 25 per cent. to 35 per cent. The recent epidemic in the United States was an especially mild one, the mortality being but 3.3 per cent. The hemorrhagic cases are always serious; those of *purpura variolosa* all die, and although some cases of *variola pustulosa hemorrhagica* get well, the majority are usually fatal on the seventh, eighth, or ninth day. The pregnant woman usually aborts, it is said in 50 per cent. of cases, and commonly perishes, but not always. The complications of pneumonia and laryngitis are serious.

From the statistics of Gregory, based upon London hospital practice, most die on the eighth day; but in private practice, according to the experience of George B. Wood, the greatest number of deaths occur between the 12th and 18th days.

Treatment.—Here as in all other conditions, prophylaxis is the most important part of the treatment. Vaccination should be universal. An unvaccinated community should not be allowed to exist. It is not possible to cut short a case of smallpox. The patient should be isolated and taken to a smallpox hospital, if possible. If at home, an uppermost room should be selected, all hangings and carpet removed, and communication with the rest of the house cut off by closed doors fortified by a sheet dampened with a solution of carbolic acid, 1 to 60. Separate dishes and utensils should be provided, and nurses should hold no communication with other members of the family. All clothing removed from the patient should be put in scalding water, and sweepings should be burned. The nurse should wear an overall, to be removed on leaving the room, and her head should be covered with a close fitting cap.

The treatment must consist in combating the symptoms. Morphin, or in less severe cases one of the salicylates, must be given to control the pain in the back. Nourishing liquid food and stimulants are required in adynamic cases. The fever is treated by sudorifics including potassium citrate, sweet spirits of niter, and by aconite, or by cool sponging or even by cold baths, as in typhoid fever, if the temperature be high. Cool drinks should be permitted *ad libitum*. The complications must receive the treatment appropriate to them. Tracheotomy may be demanded by edema of the larynx.

To Prevent Pitting.—It has always been the object of the physician to find some means of preventing the disfiguring scars which so invariably remain after very severe cases. No one method is always successful. It has long been thought that the absence of light favored healing without leaving pits. For the painful ophthalmia that so often attends smallpox, darkness is certainly a comfort, but that it diminishes the pitting is doubtful. It is a comforting fact that even the deepest and ugliest pits gradually lose their distinctness as time passes, and that much of the marking disappears in the course of a few years. The surface should, however, be anointed with vaselin, cold cream, or similar substance, as they allay the burning and itching, keep the scabs moist, and prevent the contagium from

spreading through the air. The odor, which is often intolerable, is perhaps best covered by adding carbolic acid to the vaselin or other unguent employed, say 10 grains (0.666 gm.) to the ounce (30 gm.); or a watery solution of carbolic acid may be made of the same strength and applied on cloths. Bichlorid of mercury, 1 to 2000, may be used in the same way. These preparations applied cold on lint are soothing and comforting. Schamberg, the assistant physician to the smallpox hospital in Philadelphia, says that, as the result of his experience in the epidemic of 1901–1902 in Philadelphia, painting with iodin seems to be more efficient in averting pitting than any other treatment. J. F. Romero claims to have used with most satisfactory results picric acid as a local measure to prevent pitting. He advises a lotion made with 2 grams (30 grains) picric acid, 15 grams (1/2 ounce) alcohol, and 185 grams (6 1/2 ounces) water. An ointment may be made instead. He suggests that the picric acid may destroy the pyogenic germs that may find their way into the pustules.

The eyes, nose, mouth, and throat should be kept clean, all crusts being carefully removed. This may be accomplished for the eyes by cold compresses frequently changed, while the nose, mouth, and throat should be cleansed with borated gargles and lotions. As soon as convalescence is established the patient should bathe daily, using carbolic soap, the bathing being kept up until the skin is perfectly smooth, because only then does the patient cease to be a source of infection.

Special Modes of Treatment.—As in the case of the other infectious diseases, smallpox offers encouragement to similar specific modes of treatment. The infecting organism of smallpox, whatever it may be, does not seem to develop a toxic substance so virulent as that of diphtheria. The extensive inflammation and suppuration of the skin is probably the chief debilitating agency. The internal administration of antiseptics has been recommended, but seems to have furnished no results that particularly commend it. The substances tried are the usual ones—namely, sodium salicylate, salol, mercuric chlorid, carbolic acid, creosote, the sulphites, and sulphocarbolates.

Upon the same principle as the serum treatment for diphtheria, serum from vaccinated subjects both human and lower animal, and from smallpox patients in the advanced stage of the disease, has been used by Kinyoun, Lundmann, and Béclère. Analogy would lead us to expect similar results to those obtained by antitoxin in diphtheria, but such has not been the case as yet.

Special modes of external treatment, as by baths impregnated with antiseptics, have also been used and brilliant results claimed. Galewouski[1] used solutions of potassium permanganate of such strengths as to make the baths a rose-red color. He claims reduction of temperature, disappearance of pustules, and speedy recovery. Talamon[2] recommends external application of mercuric chlorid spray to the skin, using a solution made up of

℞ Corrosive sublimate,
Tartaric acid, of each, 1 gram (15 grains).
90% alcohol, 5 c.c. (1.25 fluidrachms).
Ether, enough to make 50 c.c. (1.33 fluidounces).
Spray three or four times daily for a minute, being careful to protect the eyes.

[1] "Med. Press and Circular," 1890.
[2] "Jour. of Cutaneous and Venereal Diseases," February, 1891; "Gazette medica Lombarda," 1890.

The treatment is commenced on the first day of the eruption, being preceded by thorough washing of the face with soap, which is rinsed off with boric-acid solution, and the skin then dried with absorbent cotton. After the spray has been used the face should be covered with a layer of 50 per cent. of glycerolate of mercuric chlorid to keep the skin antiseptic. After the fourth day the number of sprayings is gradually diminished, and after the seventh day they are discontinued, though the glycerolate dressings are kept up. Talamon also recommended in the confluent and grave forms of the disease mercuric-chlorid baths lasting from three-quarters of an hour to an hour, with internal treatment including the usual supporting measures. These treatments commend themselves to reason and common sense and as being at least disinfectant and cleansing.

VACCINE DISEASE.

SYNONYMS.—*Vaccinia; Vaccina; Cowpox; Kinepox.*

Definition.—Vaccinia is an infectious disease produced by inoculation of man with lymph from the vesicle of kinepox. It is characterized by local and general symptoms. Persons successfully vaccinated are, in the vast majority of cases, immune from smallpox. The local product of such vaccination is the vaccine vesicle, the contents of which, when again inoculated, are capable of producing the same disease with immunity in another person not previously vaccinated. It is pre-eminently characteristic of vaccine disease that it can be communicated only when introduced directly into the blood.

Efficiency of Vaccination.—There can be no doubt that, if vaccination were thoroughly carried out, smallpox could be stamped out. This is, however, not done, and in point of fact, a few cases occur annually everywhere, while at intervals an epidemic of greater or less severity occurs. A false sense of security leads to indifference about vaccination and revaccination, and thus gradually accumulate a number of susceptible persons who are liable to the disease. In Germany the nearest approach to exemption exists. In 1904, 189 cases of smallpox occurred in the German Empire of which 25 per cent. were fatal while 28 per cent. were of foreign origin. The results of organized effort are better appreciated by comparing the death-rate from the disease per 100,000 in that country compared with other countries. In the German Empire, in 1904, it was 0.04; in Switzerland, 0.12; in Holland, 0.22; in 70 districts and cities of Austria-Hungary, 0.176; in 77 Belgian cities and in 8 of the suburbs of Brussels, 14.11; in 71 of the largest cities of France, 6.39, and in 76 of the largest cities of England, 1.26.

Nature of Vaccinia.—There can be no doubt that vaccinia is smallpox modified by transmission through the cow.

Etiology.—The earlier efforts of Quist, Harold Ernst and Martin, of Boston, and of Klein and Copeman in England, to establish a bacterial origin for vaccine were not successful, nor were those of Pfeiffer and Ruffer to find the cause a psorosperm any more so. On the other hand, there is every reason to believe that the inoculating element is the same as that of smallpox—a protozoön though of diminished virulence. In all vaccine lesions

studied by Guarnieri, Wasielewski, Councilman and their colleagues (and especial attention is called to the work of Calkins and Tyzzer), whether in man, in monkeys of various species, or in the calf or rabbit, bodies essentially similar to those included in the epithelial cells in smallpox in man, and undergoing the same changes and development, were found. There was no difference in size, relative numbers, and course of development, whether the seat of vaccination was the skin, mucous membrane or cornea, the only difference being the physiological one of diminished virulence, producing vaccinia instead of variola, in accordance with which the name *cytoryctes vacciniæ* instead of *cytoryctes variolæ* is given it. As Councilman further says, "The result of the two processes are in all respects the same, and immunity from both smallpox and vaccinia is conferred. Neither the calf nor any other of the domestic animals is susceptible to smallpox. The disease which they acquire by inoculation with smallpox virus is vaccinia."

Lymph in Use.—At the present time it is almost the universal practice to use animal lymph or the lymph directly from the cow, although humanized lymph, that from another person having vaccine disease, can also be successfully used. The chief reason for using animal lymph is that all danger of communicating other affections, especially syphilis, is thus avoided, although there is reason also to believe that protection is more certainly secured by animal lymph. For securing the cow-lymph numerous farms exist in this country and in Europe, where, under the most perfect sanitary precautions, inoculation is practised on the udder of heifers, whence the lymph is gathered and distributed. In Belgium the heifers are slaughtered after the lymph is taken, and if they are found diseased, the lymph is not used. In this country the more usual method is to allow the lymph to dry on ivory points or quills, or to collect it in capillary tubes.

Operation.—The operation of vaccination is variously performed. After thorough cleansing the skin of the arm with alcohol, the contents of the capillary tube are expressed on the cleansed spot of skin. With a needle sterilized by heat or the sterile needle in a package, a small area not exceeding one-eighth of an inch in diameter is gently scratched *through* the drop of virus. Blood should not be drawn if that is possible. This area is then thoroughly rubbed by a sterile piece of wood or the same sterile needle used in making the inoculation. It is best to make the inoculation upon the arm in the region of the insertion of the deltoid muscle. If the left arm is always used it will facilitate general inspection later. Vaccination upon the leg should not be practised. There is much more danger of infection of the site. After the spot is dried (from five minutes to a half an hour) the area is covered by a small pad of sterile gauze held lightly in place by small adhesive strips. This gauze should be left in place for seven or eight days, then it should be removed for inspection of the area. If the vaccination is successful another pad should be applied. If unsuccessful a revaccination should at once be done. Vaccination thus performed with perfect lymph is harmless. Carelessly done, it may be followed by dire results and bring opprobrium upon this great boon to humanity. Prolonged friction after the scratching is desirable to secure success.

The Phenomena of Vaccination.—Immediately succeeding the opera-

tion a slight inflammatory redness appears, which usually subsides rapidly, and sometimes has entirely passed away before the first phenomenon of the vaccine disease appears. Thus, there is a true period of incubation, after which, usually on the *third* day, but often two or three days later, a slight red elevation or papule makes its appearance. By the *fifth or sixth* day this has already become an umbilicate vesicle filled with a transparent viscid fluid, surrounded by a delicate red areola. The vesicle presents a shining silvery appearance; by the *eighth* day it becomes a lustrous silver-gray, and by the tenth day the vesicle and areola have both reached their maximum. Each individual vesicle is about one-eighth of an inch in diameter. The pock is by this time 1/3 inch in diameter (about 1 cm.), one to two lines in height, umbilicated at its center, and presenting frequently a minute brown spot or scab in the same situation. This larger lesion is the result of the fusion of two or more of the primary lesions. The areola is quite angry looking, often two inches (5 cm.) or more in diameter, and shows under a magnifying glass numerous minute vesicles on its surface, and the skin may be indurated. At this stage, too, it itches and burns to a degree which causes in adults an almost irresistible desire to scratch, while in the child it gives rise to fretfulness, peevishness and to slight fever. Even in the adult there is slight rise of temperature. On the *11th or 12th* day the disease begins to decline. The areola narrows and becomes less bright, the lymph more turbid and begins to dry. By the end of *two weeks* the vesicle has been converted into a dry, brown scab, which generally drops off on the 21st to 25th day. A scar remains, which is very distinct at first, but gradually assumes even a whiter hue than the surrounding integument.

The course described is the typical one in a healthy vaccinated child. In other cases the amount of local irritation is much greater, with a corresponding degree of constitutional disturbance. There is often adenitis in adjacent glands. Sometimes, in ill-conditioned children, deep, unhealthy ulcers supervene that are very slow to heal, while erysipelas and gangrenous ulcerations have even occurred and been followed by death. Even tetanus has succeeded upon vaccination and it has been claimed that the bacillus of tetanus has been inoculated with the germ of vaccine resulting in the simultaneous development of tetanus. Tetanus resulting from simultaneous inoculations should appear five to nine days after its introduction, whereas, in the cases commonly reported, three to four weeks have elapsed before tetanus developed. This seems to have been the case with the epidemic in Camden, N. J., in the fall of 1901. All of these untoward results are the outcome either of bad vaccine, careless vaccination or carelessness in treating the case after vaccination.

Since the incubation period of vaccination is shorter than that of smallpox, the prompt vaccination of a person exposed to smallpox may protect him, or at least modify the disease.

Vaccination Rashes.—In certain cases, especially when vaccination is done with the liquid lymph from the cow, a general eruption of vesicles takes place, constituting *vaccinia bullosa;* associated with miliary vesicles it is called *vaccinia miliaria.* At times a roseolar eruption is associated—*roseola vaccinalis*—not unlike the roseolar eruption of syphilis. The vesicles may be filled with blood—*vaccinia hæmorrhagica.*

Revaccination.—Should a considerable time elapse after vaccination, a revaccination will generally be more or less successful. Usually, the entire set of phenomena is less characteristic, although it sometimes happens that the same typical course is repeated. Such successful vaccination is regarded as evidence that immunity from smallpox is no longer present, and the person, if exposed to smallpox before vaccination, would have taken it. Such an attack is almost invariably less severe, and presents the modified symptomatology known as that of varioloid. The period of exemption after vaccination varies greatly. It is often life long. More frequently, it lasts from ten to 12 years, and every person should be revaccinated at 7 to 8 years, and thereafter every seven or eight years and whenever an epidemic of smallpox is raging.

At times, even in first vaccinations, an abortive result obtains, the vesicle drying and dropping off much too early. Should this occur, the operation should be repeated.

Possibility of Transmitting Disease by Humanized Lymph.—The possibility of transmitting disease by vaccinating with humanized lymph has been a potent influence in stimulating the employment of animal lymph. Syphilis seems the only disease that can be thus transmitted, although it has been claimed also for tuberculosis. It is, nevertheless, important that every precaution should be taken against such accidents. If humanized lymph be used, as it sometimes must be, only that from children of healthy parents, free from syphilis or tuberculosis, should be selected, and under all circumstances *lymph admixed with blood should be rejected.* Lymph should be taken from fully matured and perfect vesicles on the eighth day. It is scarcely justifiable to use humanized lymph.

It is exceedingly important that the physician should have at hand the data of discriminating between the ulcer of vaccinosyphilis and of uncomplicated vaccination; and between secondary vaccinosyphilis, the vaccination rashes, and hereditary syphilis occurring about the time of vaccination. Such data are found in the following table compiled by C. S. Shelly from Fournier, in Fowler's "Dictionary of Medicine":

Vaccinosyphilis or Vaccino-Chancre.	Vaccination Ulcers.
Chancre never developed before the fifteenth day after vaccination; usually not until after three to five weeks; it is still in its earlier stage twenty days after vaccination.	Ulceration is present twelve to fifteen days after vaccination and is fully developed the twelfth day after vaccination.
Chancre developed on the site of usually one or two only of the vaccination punctures.	Ulceration affects all the punctures, as a rule.
Inflammation is slight.	Inflammation and ulceration severe.
Loss of substance superficial only.	Ulcer deeply excavated.
Suppuration scanty or absent, scabs, or crusts.	Much suppuration.
Border of chancre smooth, slightly elevated, gradually merging into floor.	Margin of ulcer irregular, as in "soft chancre."
Surface of floor smooth.	Floor of ulcer uneven, suppurating.
Induration "parchment-like," and specific, not merely inflammatory.	Induration inflammatory only.
Inflammatory areola very slight.	Areola inflammatory and erysipelatous.
Gland swelling constant, indolent [syphilitic] bubo.	Gland swelling often absent; if present, merely inflammatory.
Complications rare.	Complications—sloughing, erysipelas, etc. —often present.

Secondary Syphilitic Eruption due to Vaccinosyphilis.	Vaccination Rashes.
	[Including roseola vaccinalis, miliaria vaccinalis, vaccinia bullosa, vaccinia hæmorrhagica; also accidental eruptions—rubeola, scarlatina, lichen, urticaria, etc.]
Appears, at the earliest, nine or ten weeks after vaccination.	A true vaccinal rash appears between the ninth and fifteenth day after vaccination.
Requires, in every case, the pre-existence of a specific ulcer [chancre] at the site of vaccination.	Absence of inoculation chancre.
Exhibits the character of a true specific eruption.	Eruption does not exhibit specific characters.
Fever often slight.	Fever always present.
Lasts for a long time. Usually accompanied by specific appearances on mucous membranes.	Evanescent.
Vaccinosyphilis.	**Hereditary Syphilis, Showing Itself about the Time of Vaccination.**
Begins with local infection chancre and indolent bubo.	No chancre; begins with general phenomena.
Typical development in four stages—viz., incubation, chancre, second incubation, generalization [secondary eruption], etc.	Has no typical development in connection with vaccination.
Never appears earlier than the ninth or tenth week after vaccination.	Time of development quite independent of vaccination. Is attended by the characteristic syphilitic bodily aspects. Other manifestations of hereditary syphilis may be present. The history may indicate syphilis.

Some idea of the efficiency of vaccination may be obtained from the fact that through it smallpox has been blotted from the Germany army. Further, it was early shown by Marson that of those who have acquired smallpox after vaccination, the disease is vastly less severe than in those who have primary smallpox. This is confirmed also by the statistics of W. M. Welch, Physician-in-charge of the Municipal Hospital of Philadelphia. From a study of 5000 cases, he showed that where there were good cicatrices, only 8 per cent. died; with fair cicatrices, 14 per cent.; with poor cicatrices, 27 per cent.; unvaccinated cases, 58 per cent. The history of vaccination in the province of Santiago, Cuba, where smallpox was banished by its use should be read.

Treatment.—No treatment for the vaccine vesicle is usually required beyond protection by means of sterile gauze from friction and contamination by the clothing. Shields should not be used, but the wound covered with a piece of surgeon's gauze fastened by adhesive plaster.

CHICKEN-POX.

Synonym.—*Varicella.*

Definition.—Varicella is an acute contagious disease of children, characterized by an eruption of vesicles with pearly contents and attended with little or no constitutional disturbances.

Etiology.—The disease is eminently contagious, but no specific causal organism has been isolated. It is almost purely a disease of childhood, though attacks in adults are known, it occurs most frequently between the second and sixth year. It is a distinct and separate disease from smallpox, an attack bringing no exemption from that disease.

Symptoms.—The *period of incubation* is from 10 to 15 days. So slight is the constitutional disturbance that very commonly the appearance of *the eruption* is the first notification of the child's illness. At times there are *slight prodromal peevishness, restlessness*, and *feverishness;* at others there is a *slight chill* followed by *fever*. Some muscular pain may be present.

A prodromal scarlatinal rash may rarely present itself, but for the most part the *suddenness* of the *eruption* is *distinctive*. It presents itself in the shape of isolated pimples scattered over the body within the first 24 hours after constitutional disturbance. They are more prone to occur on the parts covered by clothing, as the trunk, but they may appear first on the face. In another 24 hours they are vesicles filled with perfectly clear fluid, as a rule, without umbilication or areola; the liquid rapidly becomes turbid and by the end of the third day the vesicles begin to dry up, and in another day are converted into dark-brownish crusts, which drop off, usually leaving no scar. Sometimes, however, a distinct pit is left, especially if the pock be scratched by the child, as it sometimes is, because of the irritation it excites. Occasionally, too, the pustule is distinctly umbilicated and may also have a pink areola. The pustules appear in crops, so that on the fourth day they can be seen in all stages, but at the end of a week again all have disappeared. Rarely are there more than half a dozen on the face, though they may be quite numerous and the victim well dotted over. They occur also on the scalp and in the throat.

Complications are rare with varicella, and in most cases would have been overlooked but for the eruption. It is, however, true that *hemorrhagic pocks* sometimes occur accompanied by hemorrhage from the mucous membranes; that *nephritis* and even gangrene—*varicella gangrænosa*—have occurred, and *infantile paralysis* has developed during an attack of the disease. This paralysis not belonging, however, to the *disease* anterior poliomyelitis.

Diagnosis.—The diagnosis should not detain one long. The trifling constitutional disturbances, the rapid, almost sudden, development of the pustules during the first day—all distinguish the disease from smallpox.

Prognosis.—This is invariably favorable, except in rare cases of *varicella gangrænosa.*

Treatment.—Usually none is needed save the application of a simple lotion or ointment to allay the itching. The principal need of the physician is to make the diagnosis.

The following table, somewhat modified, from T. M. Rotch, may be helpful in diagnosis.

	Scarlet fever	Measles	Rubella	Variola	Varicella
Incubation	Two to four days	Seven to fourteen days.	Fourteen to twenty-one days.	Seven to twelve days.	Ten to fifteen days.
Prodrome	Two days.	Three days.	A few hours.	Three days.	A few hours
Efflorescence	Erythema.	Papules.	Papules.	Macules, papules, vesicles, pustules.	Vesicles.
Desquamation	Lamellar.	Furfuraceous.	.....	Large crusts.	Small crusts.
Complications and sequelæ	Kidney, ear, and Heart.	Eye and lung.	.....	Larynx, lungs, eyes.	.....

WHOOPING-COUGH.

SYNONYMS.—*Pertussis; Whooping-cough.*

Definition.—Whooping-cough is an infectious disease, characterized by spells of coughing accompanied by a long drawn inspiration producing the "whoop," whence the disease is named.

Etiology.—It is interesting to note that Linnæus ascribed whooping-cough to the larvæ of insects in the nose. The infecting organism is probably the Bordet Bacillus—as described by Bordet and Genyou.[1]

Whooping-cough attacks children of all ages not rendered immune by previous attacks, though it is most usual between the first and second dentitions; nor is it a very rare affection in adults, in whom it may become serious. It is said to be more frequent in girls. Epidemics are more common in the spring and winter, and often precede or follow those of scarlet fever and measles. The disease is generally communicated from one child to another, and few escape who are exposed. Sporadic cases also occasionally occur. The delicate and those suffering from bronchial and nasal catarrh are more vulnerable. Some persons are immune.

Morbid Anatomy.—There is no morbid anatomy peculiar to whooping-cough beyond the catarrhal inflammation. According to Myer-Huni and v. Heroff, this is most marked in the mucous membrane of the nose, larynx, and trachea down to the bifurcation, but especially on the posterior wall of the pharynx, and in the interarytenoid region—the so-called "cough region." The morbid states found after death are those of the complications—viz., bronchitis, bronchopneumonia, and collapse of the lung. Vesicular and interstital emphysema are sometimes present, the former from over-distention of the air-vessels, and the latter from their rupture.

Symptoms.—Whooping-cough has a *period of incubation* of from seven to ten days. There is no prodrome separable from the *preliminary stage*, beginning with cough which is in no way peculiar, being that of an ordinary cold with slight fever and without expectoration. There may be *coryza* and *injection of the conjunctiva*. This cough may go on for a *couple of weeks* and, if there be nothing in the history to suggest the nature of the disease, may occasion no suspicion. Toward the end of this period, however, the observing mother will have noted that the cough is gradually growing worse and becoming paroxysmal, that it occurs "in spells," and frequently at night. Then suddenly a "whoop" is noted and the nature of the disease is suspected.

The *paroxysmal stage* has replaced the catarrhal, and soon the diagnosis is plain. The paroxysms become more frequent and more severe. Each one begins in a succession of short expiratory coughs, which grow in intensity. All efforts lie in the direction of expiration, and all expiratory muscles are brought into play to this end. *The chest is compressed laterally, and bulges in the sternal region.* As the result of such efforts, the face is flushed, turgid and sometimes cyanotic, the eyes are injected and bulging, the tears start, and the nose discharges. Finally, the paroxysm terminates or is interrupted by a loud, whooping inspiration—that is, it may end for the

[1] "Soc. Roy. des Sc. Med. et Naturelles de Brex," vol. lxiv., 1906, page 305; 1907, page 107.

time or be immediately succeeded by another similarly concluded paroxysm. Severe paroxysms commonly terminate in an act of vomiting, which brings up considerable mucus, often accumulated before the paroxysm begins and seeming to be its exciting cause. The whoop may precede or begin the paroxysm. The number of paroxysms in the 24 hours varies greatly. They may be as often as every half-hour or only four or five times in the day. Emotion or exertion will precipitate a paroxysm, as will the inhalation of irritant matters. The little patient resists the paroxysms as long as possible, and when the inevitable comes it will run to the basin or bowl, knowing full well what is to happen. The demure method pursued by little children under these circumstances is often at once touching and amusing. Rupture of a conjunctival or nasal blood-vessel sometimes occurs and occasionally an involuntary urination. An ulcer may form at the frenum of the tongue, said to be done to pressure of that part of the organ against the incisor teeth. The termination of the paroxysm is followed by temporary relief.

The paroxysmal stage, if uncomplicated, is unattended by fever, and physical examination of the chest is barren of results as compared with the severity of the cough. The percussion note is clear, clearer during inspiration. Auscultation may discover a few moist râles soon after a paroxysm; but during it, nothing. Even during the whoop the vesicular murmur may be absent, because of the slowness with which the air enters the chest.

The *length* of the *paroxysmal stage* is usually from *four* to *six weeks*, although in mild cases it may be shorter. Indeed, there are mild cases of whooping-cough in which the paroxysms are scarcely noticeable and would not be noted except for an occasional "whoop." Toward the end of this period the paroxysms become less severe and less frequent, and soon the stage of decline or convalescence is established. In the course of it the paroxysms become still milder and less frequent, and finally subside altogether. They are, however, liable to be renewed for a time if the patient takes cold, and even digestive disturbances are said to have a similar effect. The other phenomena of the stage of convalescence are return of appetite, weight, and strength. The period of convalescence occupies another four weeks, so that the entire length of an ordinary attack of whooping-cough is from ten to 12 weeks, and even longer.

Complications and Sequelæ.—The complications that attend whooping-cough are bronchitis, bronchopneumonia, collapse of the lung, pleurisy, and interstitial emphysema. The bronchopneumonia is apt to be of the insufflation kind. Collapse of the lung may succeed it. Interstitial emphysema and even pneumothorax may result from rupture of the air-vesicles, and it is apt to become general and serious. In a case of this kind under the care of my friend, Horace Williams, which terminated fatally, an abscess formed at each point at which the emphysema approached the surface. Cerebral palsy and death from subdural hemorrhage are said to have occurred in whooping-cough. Among sequelæ may be mentioned, as a rare event, tubercular consumption; also permanent changes in the shape of the chest including the so-called pigeon breast, sometimes the result of a prolonged attack of whooping-cough.

Diagnosis.—The diagnosis cannot be delayed after the appearance of the

whoop, and it is scarcely possible without it. Spasmodic cough may occur from other causes, but it is not whooping-cough unless there be the whoop. A differential blood count shows a preponderance of lymphocytes, as many as 50 to 60 per cent. This is of diagnostic value.

Prognosis.—Notwithstanding the enormous number of children who have whooping-cough and get well of it, many of them without any treatment whatever, it is not so harmless a disease as many suppose. Its mortality is greater than that of diphtheria or scarlet fever. At the same time we cannot believe that the position assigned to whooping-cough by Thomas M. Dolan,[1] of being third among the fatal diseases of children in England, is true of this country. The chief danger is from the complication of bronchopneumonia. The younger the child, the greater the danger. As already stated, cases in which interstitial emphysema occurs from rupture of the air-vesicles may terminate fatally. The disease is more serious in the negro race—more than twice as fatal as in whites.

Treatment.—Prophylaxis is important. The Bordet bacillus is found in the nasal and bronchial discharges, therefore the pernicious habit of allowing children to vomit the mucus on the street and in the cars should be stopped. When the mucus is discharged it should be collected in a suitable receptacle and at once destroyed. Children affected should not go to school. The treatment of whooping-cough is one of the opprobria of medicine. Notwithstanding the claims of many to the contrary, it remains a fact that we possess no means of cutting it short. We may, however, palliate the disease by diminishing both the frequency and the severity of its paroxysms. Fresh air is important. The patient should sleep in a room with windows wide open, and be out in the air as much as practicable, during the day. Violent exertion should be prohibited. The best remedies are the opiates, chloral, and antispasmodics. The former two, as a rule, should be reserved for night, though in severe cases chloral in doses sufficient to secure somnolence is recommended by Willoughby.[2] Of the latter, the most efficient are belladonna, the bromids, and asafetida. Belladonna should be given in full doses. It is difficult to name them, and they must for the most part be arrived at by trial. We may begin with 1 minim (0.066 gm.) of the tincture every two hours to a child of six months, or 1/12 grain (0.0055 gm.) of the extract, and increase the dose until the characteristic redness of the skin is produced.

The bromids, preferably of sodium, should be given as often, in doses of 2 or 5 grains (0.1 to 0.3 gm.) for every year of age. Asafetida is useful. Antipyrin has acquired some reputation and has been especially recommended by F. J. Taylor,[3] and Von Genser. The former says in many cases its action is little short of marvelous. He recommends beginning with a small dose, increased until a child of two years is taking 2 or 3 grains every three hours. The bromids of potassium, sodium, and ammonium may be combined with it. The same writer recommends alum to check excessive secretion in the later stages, 3 grains, every three or four hours, to a child two years old. Von Genser recommends 2 grains a day for each

[1] "Whooping-cough," London, 1882.
[2] "Am. Jour. Obstetrics," June, 1898.
[3] "Annals of Gynecology and Pediatrics," July, 1899. See also very full paper, giving the experience of many physicians in "Gazette Hebdom. de Med. et Chirurg.," October 22, 1896, by Le Goff. Abstracts in "New York Med. Jour.," November 14, 1896.

year of age and reports recovery in 24 days. Good results are claimed for quinin in doses of 5 grains a day for a child five years old. An abdominal binder very tightly applied sometimes controls the severity of the paroxysms.

The intervals between the paroxysms at night may be prolonged by the judicious use of paregoric, deodorized tincture of opium or codein combined with antispasmodics, including belladonna and the bromids.

The inhalation of germicidal solution suggested by the probable germ origin of the disease has not as yet produced any results. The use of agents which require a room to be closed so the air may be impregnated with the drug is not only useless but it is harmful. The various patent preparations on the market should be shunned.

Parents should be enjoined to protect their children from undue exposure, because it is this that causes complications, and it is the complications that are dangerous. Such complications, and other symptoms which arise in the course of the disease, should be treated by appropriate remedies.

The possibilities of serum therapeutics extend to the treatment of whooping-cough, and Walsh, in the paper alluded to, refers to results obtained by him which encourage further trial.

MUMPS.

SYNONYM.—*Epidemic Parotitis.*

Definition.—Mumps is an acute infectious disease characterized by inflammation of the partoid gland, sometimes of the submaxillary.

Etiology.—Although a *bacillus parotidis* has been described, it is generally conceded that the real contagium of mumps has not been isolated. Children and adolescents are its favorite subjects, the very young as well as adults being equally exempt. More boys are attacked than girls. The disease is more common in the spring and fall. It is more commonly epidemic, but may be sporadic. It may be associated with measles and whooping-cough. One attack protects against a second.

Morbid Anatomy.—The swollen and hardened salivary gland is the sole morbid product. The swelling is mainly due to infiltration.

Symptoms.—From seven to 14 days intervene between exposure and the invasion, which is ushered in by *moderate fever*, rarely exceeding 101° F. (38.33° C.), although 103° and 104° F. (39.44° and 40° C.) have been noted. The first symptom is usually *pain below* and *in front of the ear*, but pain in swallowing may be first experienced. Simultaneously, there may be *swelling* about the ear, which extends rapidly in front of the ear and below it until the entire neck in this vicinity is involved.

The maximum swelling is reached in about 48 hours, after which the involvement of the other side begins and extends with equal rapidity. The most prominent point is in front of the ear. The swelling does not, however, subside as fast as it comes on, but presists from seven to ten days.

At the height of the disease the pain and difficulty in swallowing are extreme, the former extending often to the interior of the ear, producing earache, and the hearing may be affected. The parts are so tense and

swollen as to make opening of the mouth almost impossible, mastication equally difficult. Suppuration is an exceedingly rare event. In cases of great severity delirium is sometimes present for a short time.

Occasionally the other salivary glands are affected.

Complications.—The most frequent complication is *orchitis*, and occurring, as it commonly does, after inflammation of the salivary glands has subsided, it has been regarded as a metastasis; but this is probably not the case, since both conditions may be the result of the same cause, as originally held by Niemeyer. The swelling may affect one or both testicles, the duration being longer in the bilateral form. The organs are heavy and painful, but not so much so as in gonorrheal epididymitis. The inflammation lasts for three or four days and then subsides gradually. Usually, the gland itself is involved, but occasionally there occurs acute epididymitis with acute hydrocele and edema of the scrotum. Atrophy is said to have supervened.

Inflammation of the mammary glands and of the vulva sometimes occurs in girls, and more rarely of the ovaries.

Otitis media with resulting deafness, meningitis, and facial palsy are occasional complications.

Diagnosis.—The diagnosis usually presents no difficulties, and any doubt is commonly cleared by the acuteness of the attack. Certain enlargements of the *cervical lymphatic glands* resemble contagious parotitis, and in tuberculous children the swelling in mumps is sometimes prolonged, but the physiognomy in this disease is different and distinctive. There is more swelling in front of the ear in parotitis, and in the first stage a triangular shape is produced with the apex downward, while the lobe of the ear is raised in a characteristic manner.

Prognosis.—The prognosis is favorable, no fatal cases of uncomplicated mumps being recorded.

Treatment.—No means of shortening the duration of the disease exists. The patient should be kept uniformly warm, and to this end the bed is desirable. It is usual to anoint the gland with some simple ointment, as cold cream, and it may be that the feeling of drawing and tension is thus relieved. No commensurate advantage results from leeching. It is thought by some that the so-called metastasis is occasioned by exposure to cold, and if this be true, there is even better reason for keeping the patient warm. Warm applications are generally better borne than cold. Cotton—wool or flannel, warmed and greased, gives a sense of comfort. Fever should be treated by appropriate remedies and other symptoms met as they arise.

Secondary Parotitis.—This term is applied to parotitis occurring as a complication in acute infectious diseases, typhoid fever, typhus fever, and pneumonia being the most frequent. It may be a complication of pyemia, phthisis, and carcinoma. Except in pyemia, when it is metastatic, it is probably caused by the germ of the disease present, or the bacteria of decomposing matters in the mouth, which reach the gland through the duct of Steno.

It is a much more serious affection than mumps, with which it has nothing in common, and often terminates in suppuration. Facial paral-

ysis may result from destruction of the facial nerve, or there may be deafness from invasion of the middle ear.

The *treatment* of secondary parotitis is that of phlegmonous inflammation elsewhere.

INFLUENZA.

SYNONYMS.—*Catarrhal Fever; Grip; La Grippe.*

Definition.—Influenza is an acute infectious disease characterized by fever, by catarrhal irritation of any or all of the mucous tracts, especially the respiratory, by muscular pain, and by great prostration. It is commonly epidemic.

Etiology.—In 1892 Pfeiffer discovered in the pus-cells of tracheal mucus an organism which he regarded as that of influenza. It is 0.8 to 1 micron long and 0.1 to 0.2 micron broad—*i. e.*, about the same width as the bacillus of mouse septicemia and half as long. It forms colonies on glycerin agar 24 hours after inoculation, visible under the microscope as clear, water-like drops. These drops do not coalesce, but remain separate. The bacilli are best stained in dilute Ziehl-Neelsen solution of carbol fuchsin or hot Loeffler methylene blue solution. Later studies tend to sustain Loeffler's claim. The bacilli are very numerous in the nasal and bronchial mucus, whence they are conveyed to others, constituting a true contagium. P. Canon[1] claims to have found them in the blood, in large numbers and in clumps, although he admits that his observations have not as yet been confirmed. The contagious nature of influenza is further sustained by the fact that it travels only as fast as people travel, even contrary to the direction of prevailing winds. The complications and sequelæ of the disease—pneumonia, pleurisy, endocarditis—may be the result of a toxin, or the bacillus may be transmitted in the blood to the seat of secondary infection. One attack does not, however, protect against a second, and I know persons who have had an attack each winter for several winters.

VARIETIES.—There is much carelessness at the present day in the application of the word "grippe." Commonly, when a person is said to have "grippe" it means that he has a bad cold in the head, with more or less bronchial catarrh. This seemingly is what Leichtenstern calls endemic *influenza nostras*, pseudo-influenza, or catarrhal fever, a special disease of unknown etiology, which bears the same relation to the true influenza as *cholera nostras* to Asiatic cholera. In addition, Leichtenstern makes two other divisions—(1) *epidemic influenza vera*, caused by Pfeiffer's bacillus; (2) *endemic influenza vera*, which often develops for several years in succession after a pandemic, also due to Pfeiffer's bacillus.

Morbid Anatomy.—The anatomical changes are those of the complications. Whatever alterations are the direct result of the disease itself for the most part promptly disappear after death.

Symptoms.—Influenza has a *period of incubation* of from two to three days or longer. It attacks all ages, infancy less commonly, more frequently persons from 20 tc 50 years old. The mode of onset is by no means the same. The attack may be ushered in by a *chill* or continued chilliness.

[1] Canon. "Die. Bacteriologio des Blutes bei Infektionskrankheiten. Jena, 1905. p. 105.

Most frequently, perhaps, there are *coryza* and *sneezing*, with or without watering of the eyes. To this succeeds *cough*, to which is commonly added, very soon, copious expectoration. The cough may be paroxysmal and be attended with prostration at the end of the spell. It is often persistent, while the bronchitis may pass into *bronchopneumonia* or a *croupous pneumonia* may supervene. Less frequently, there may be *faucitis*, simple, however, and not accompanied by ulceration or white patches, but causing intense pain in deglutition. These symptoms are more or less associated with *muscular* pain elsewhere, although not invariably. At other times the attack begins with severe *pain in the back* or *back of the head*, the *chest walls*, the *extremities*, or *throughout* the *muscular system*. Such pain is sometimes severe and sudden. Severe *headache* may be a symptom, associated with other symptoms such as pain in the back and neck with delirium which suggest meningitis.

Another mode of onset is by an extreme and *sudden prostration*. This prostration is apt to be prolonged even in mild cases far beyond what seems reasonable. *Mental depression* is a frequent symptom, and suicide and even manslughhter have been said to be its terminal acts.

There is always more or less *fever*. Commonly, it is slight at first, but sometimes very high, ushering in the febrile variety of the disease. We have known it to be 106.2° F. (41.2° C.) at the first observation of a patient. More frequently, it does not exceed 103° F. (39.4° C.), and it is often but slightly above normal. During convalescence the temperature may become subnormal, and in the patient alluded to there was a fall from 106° F. (41.1° C.) to 96° F. (35.6° C.) in a very short space of time. Further, the temperature chart may exhibit fantastic changes, as seen in that of the case of a medical student who made a good recovery after 28 days' illness (see Fig. 16). *Delirium* is sometimes associated with the fever, and may come on suddenly and actively. The *pulse* is usually corresponding frequent, but some cases of uncommonly slow pulse have fallen under observation.

While pulmonary catarrh is perhaps the most frequent catarrhal manifestation, it is by no means always present, even when there are pulmonary symptoms. An obstinate case of bronchial spasm was seen by one of us without any secretion whatever. In the epidemic, especially of 1893–94, in Philadelphia and vicinity, *gastric catarrh* was frequent, producing distressing nausea with vomiting, and adding greatly to the physical weakness. Severe *vomiting* may even usher in the attack, especially in children. More rarely there is *diarrhea*. Other cases begin with nervous symptoms of which headache and delirium are conspicuous, suggesting meningitis. Herpes is sometimes present.

According as one or another set of symptoms predominates, the disease is said to belong to the respiratory, nervous, muscular or rheumatic, gastro-intestinal, or febrile form of influenza.

Complications.—The most serious complication is *pneumonia*. It is often invited by exposure during convalescence or in the attempt of a patient to fight out the disease without giving up. In these events it is usually ushered in by a chill and extends rapidly through the whole of one lung or both lungs. When a part of the primary attack, the pneumonia is more apt to be catarrhal and circumscribed, creeping from the bronchi

into the air-vesicles, and is less serious, although it may also be fatal, especially in old persons. At other times the inflammation is confined to the minute bronchioles, and we have the physical signs of a capillary bronchitis. It may be associated with pleurisy. Of *cardiac* and *vascular complications* endocarditis, pericarditis, irregularity of the heart unassociated with evident endocarditis or pericarditis, may arise. Sudden heart failure is to be remembered as a possible cause of death.

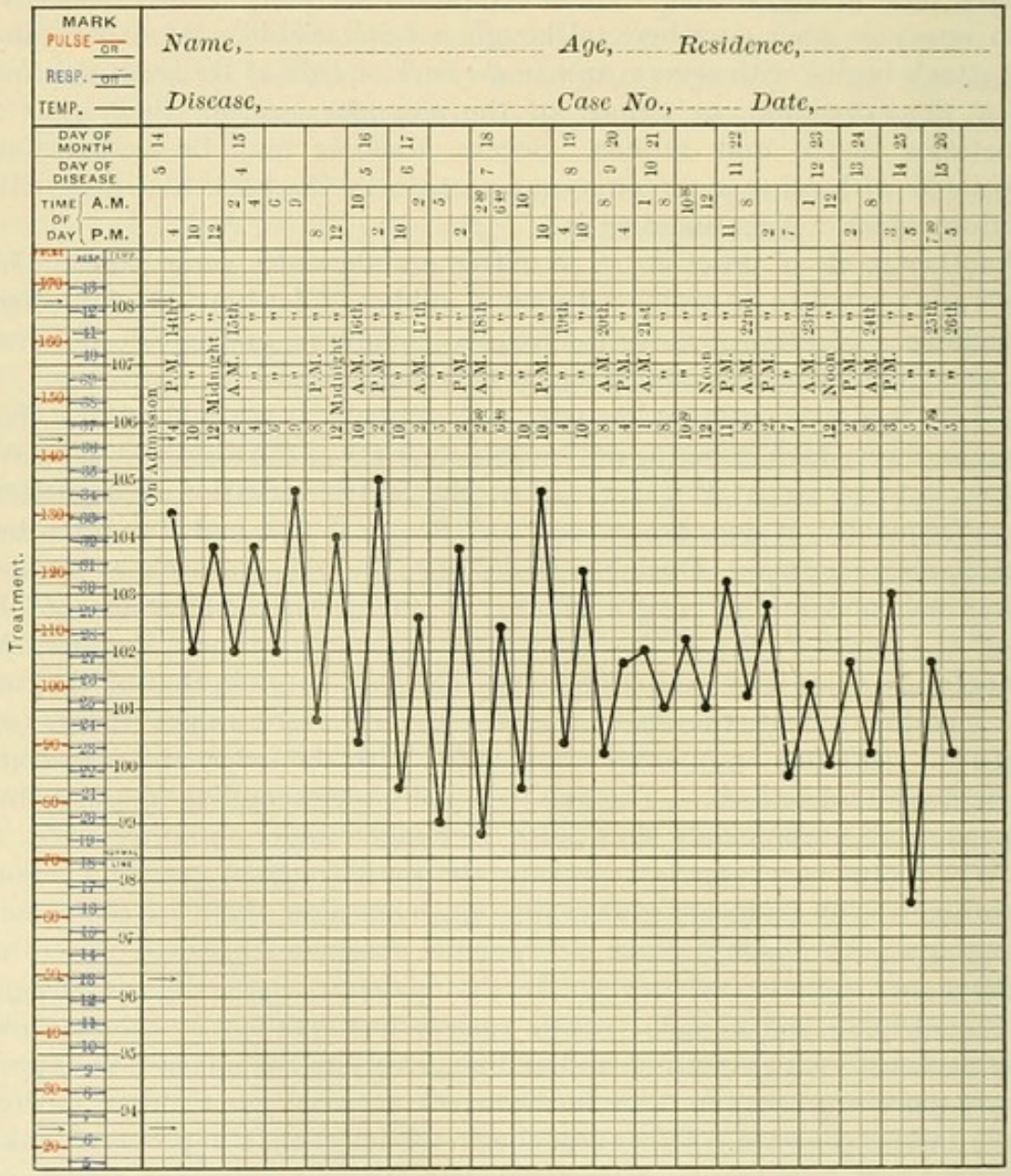


FIG. 16.—Chart of a Case of Influenza—Medical Student.

Of *nervous* lesions meningitis and encephalitis have been noted, even abscess of the brain; also neuritis and optic neuritis; in fact, almost every form of nervous disease, though some of the conditions must be referred to errors of diagnosis, cerebrospinal fever being probably responsible for some. *Herpes*, when present, is probably a result of neuritis. Mention should not be omitted of *venous thrombosis*—phlegmasia alba dolens—as a complication of influenza, Leyden and Guttmann having collected 28 cases.[1]

A most important fact to be remembered in this connection is the tend-

[1] "Deutsche med. Wochenschrift," No. 6, 1897.

ency of influenza to develop *latent disease into active disease*, and to make slight grades of organic affections more serious. This is particularly seen in connection with heart disease and kidney disease. A small albuminuria with no other symptoms may become, after an attack of influenza, an incurable and rapidly fatal Bright's disease.[1] A mild cardiac affection, scarcely noticeable by its symptoms, may become a grave illness with degeneration of muscular substance and dilatation of the cavities.

Diagnosis.—The diagnosis is ordinarily easy, although doubtless during an epidemic many cases are called influenza that are cases of simple bronchitis, faucial angina, or nasal catarrh. The diagnostic features in addition to the catarrhal factor are the *suddenness* of attack, fever of *short duration*, extreme disproportionate *prostration*. Muscular pains are characteristic, but not always present. *Cerebrospinal fever* and *influenza* are sometimes confounded. The distinction will be considered when treating of the former. Some cases in their incipiency resemble typhoid fever, but the suddenness of onset, absence of the typical temperature of typhoid, of epistaxis, of diarrhea, together with the shorter duration of the illness, turned the scale in favor of influenza.

Prognosis.—The prognosis is generally favorable, especially if the patient goes to bed at once, or at least houses himself. Such a one is almost sure to be well in three, four or five days. It is possible, however, for one attacked to fight through the disease without losing a day's time. But especially unfortunate is he if he fails in this attempt because of exposure or inability to hold out longer against the dibilitating effect of the disease. In the former he is apt to have pneumonia, in the latter he has to contend with extreme prostration. The prostration of the epidemic variety is something peculiar. The weakness is extreme, and the slightest effort, physical or mental, promptly convinces the patient of this. The duration of the weakness may be greatly prolonged, months being sometimes necessary to overcome it.

Treatment.—The treatment in the majority of cases is very simple. Rest in bed, without medicine answers for a large number. Beyond this the treatment is mainly symptomatic, phenacetin, acetanilid, or antipyrin being generally sufficient to subdue the pains when present. Quinin is necessary in many cases to keep up the strength. In ordinary cases requiring treatment 5 grains (0.324 gm.) of phenacetin with 5 grains of salol if of value given every four hours. When the pains are very severe, the phenacetin may be given more frequently and even in larger doses. When headache is present caffein should be added in doses of 1 1/2 to 3 grains (0.1 to 0.3 gm.). Larger doses of quinin may be needed. A favorite prescription is a capsule of 2 1/2 grains (0.16 gm.) each of Dover's powder, salol, and phenacetin, every two or three hours. Still another is phenacetin and salicin, of each 2 1/2 grains (0.16 gm.), and powdered camphor, 1/2 grain (0.035 gm.).

The cough may be treated with turpentine stupes and sinapisms to the chest; and when there are positive laryngeal symptoms, "Dobell's solution," sprayed into the larynx, is very soothing. It may also be sprayed

[1] See a paper by G. Baumgarten on "Renal Affections Following Influenza," in "Transactions of the Association of American Physicians," vol. x., 1895.

into the nasal passages, or cocain may be applied locally. Internally, the officinal solution of citrate of potassium in 1/2 fluidounce (15 c.c.) doses, every two or three hours, is helpful. When the cough is disturbing, small doses of morphin or heroin may be necessary; and if secretion has set in, ammonium chlorid in 5 to 10 grain (0.324 to 0.648 gm.) doses, with 15 minims (1 gm.) of syrup of squills and 2 drams (7.4 c.c.) of compound licorice mixture are sufficient to answer the purpose. If more stimulating effect is required on the secretion, the aromatic spirit of ammonium in 1/2 dram doses (2 gm.), or carbonate of ammonium in doses of 5 to 10 grains (0.324 to 0.648 gm.) may be substituted. On account of the fact that hexamethylenamin is excreted by the mucous membranes this drug may be used in doses of 3 to 5 grains (0.2 to 0.4) repeated every two or three hours. Opium may be given in large doses, or morphin in corresponding doses, to relieve pain, if required.

For the prostration, supporting measures are necessary, and stimulants may be called for. Whisky and milk are efficient. The entire absence of appetite and the complaint that all things taste alike are to be ignored, and the patient must be encouraged to take food, which should be made as attractive as possible. Strychnin is an admirable heart tonic, and may be given, 1/30 grain (0.00216 gm.), every six hours, increased, if necessary.

Treatment for the pneumonia, often so grave a complication, is at times extremely difficult. In a few cases "pneumonia fulminans" strikes the patient down so suddenly and violently as to make all treatment unavailing. Referring the reader for details to the section on pneumonia, it may be said that, as a rule, in the pneumonia of influenza, stimulating and restorative measures of a very positive character, rather than depressing agents, are indicated. The free use of alcohol and ammonia is especially necessary. Dry-cupping is never out of place. It may be repeated and should be followed during convalescence by a jacket of wool, to maintain warmth and a uniform temperature, but this is of doubtful propriety during the height of fever when we need measures to dissipate heat rather than retain it.

One need not wait for the physical signs of pneumonia to present themselves before beginning the treatment. Given a chill after exposure, with no other cause to explain it, a pneumonia is almost inevitable. Oftentimes a pneumonic focus in the center of a lung does not furnish any physical signs, while to wait until it approaches the surface causes a fatal delay in the treatment.

Other complications of influenza are treated as when they are simple diseases. Overmedication should be avoided.

CEREBROSPINAL FEVER.

SYNONYMS.—*Epidemic Cerebrospinal Meningitis; Spotted Fever; Petechial Fever.*

Definition.—An infectious disease of sporadic and epidemic occurrence, microbic in origin and especially characterized by inflammation of the membranes of the brain and spinal cord.

Etiology.—The direct cause of cerebrospinal fever is a lancet-shaped

diplococcus resembling the pneumococcus, discovered by Weicheslbaum in 1887, and called by him *meningococcus* or *diplococcus intracellularis meningitidis*. It lies largely within the polynuclear leukocyte found in the spinal fluid in the disease. Weichselbaum's observations were confirmed by Heubner[1] in 1891, by Jaeger in 1895, and by Councilman,[2] Mallory, and Wright in 1898, and Osler[3] in 1899. While the disease may be regarded as contagious, it is not highly so, being somewhat like tuberculosis in this respect. That the infectious agent is always derived from an infected person is at least doubtful, the disease not being, as a rule, traceable to another having it, but appearing to arise rather in certain houses or localities where the necessary conditions prevail. Neisser has shown that the bacillus is transmissible by feeble atmospheric currents.

The following are the characteristic features of this bacillus: It occupies a position within the polynuclear leukocyte, whence the adjective term intracellularis. It takes the usual stains, and is decolorized by the Gram method. It forms on Loeffler's blood-serum "round, whitish, shining, viscid-looking colonies, with smooth, sharply defined outlines, which attain a diameter of 1 to 1 1/2 microns in 24 hours" (Councilman). It is found in the cerebrospinal exudate, and has been isolated from blood, pus, the joints, from pneumonic areas in the lungs, and from nasal mucus.

Predisposing causes are—cold, moisture, exposure, defective sanitation. Crowded buildings, barracks, and tenements have been favorite localities, especially in Europe. Depressing influences and the fatigue of long marches favor it. During the Civil War in America both armies suffered from the disease, but the mortality was not large. The disease is more prevalent at times in the country than in the city. It is more common in the young, attacking even infants of less than a year old. Sex and race seem to have no influence on the etiology. The organism probably reaches the blood stream through the nasal mucous membrane.

Morbid Anatomy.—The external appearance of the body after death is not peculiar. Most characteristic are the remnants of the eruption, petechial or herpetic, but they are not constant. The *brain* and *spinal cord* are naturally the seats to which we look for morbid changes, and we find every degree of inflammatory condition, from slight hyperemia, such as may be found in any form of infectious disease, to intense congestion with injection of the pia-arachnoid, and finally a stage in which pus and fibrinous deposits, more particularly in connection with the pia mater, are abundantly present. Higher degrees of hyperemia involve even the calvarium as well as the dura mater. The arachnoid spaces may contain serum and pus, but it is under the pia mater that we look for the inflammatory products—serous, fibrinous, or purulent, especially at the *bottom of the sulci*, in the *longitudinal* and *Sylvian fissures* and at the base over the *pons*, the *chiasm*, and *cerebellum*. To a less degree the convexity of the brain is also involved, and even the brain substance may share in the hyperemia, while actual softening has been noted. Adhesions between the pia and the cortex are common, removal of the pia carrying the substance

[1] "Jahrbuch für Kinderheilkunde," 1891, and "Deutsche med. Wochenschrift," 1897.
[2] "Epidemic Cerebrospinal Meningitis," "Report of the State Board of Health of Massachusetts, Boston, 1898."
[3] Cavendish Lecture, "On the Etiology and Diagnosis of Cerebrospinal Fever," "West London Med. Jour.," 1899.

of the cortex with it. More rarely there is an effusion into the ventricles and the choroid plexus is congested. The walls of the ventricles may be softened, and in cases of long standing there is even hydrocephalus.

The *cranial* nerves, especially the auditory and optic, may be the seat of a neuritis, or bathed in pus infiltrating the lymph-sheaths. The muscular and trophic phenomena resulting from such involvement may be permanent.

The *spinal membranes* are similarly hyperemic, even to the extent of extravasation of blood at times. The same inflammatory products are found upon them as on the meninges of the brain. They are more frequently seen on the posterior aspect of the cord, but may be general. Ounces of pus have been removed from the spinal canal. Even the central spinal canal has been found dilated and filled with pus. There may be likewise inflammation of the substance of the cord. The roots of the spinal nerves may be compressed by exudate, producing localized paralysis, or may be themselves the seat of a neuritis, whence the characteristic clonic muscular contractions often present, while the irritation of the sensory roots gives rise to more or less intense pain. Certain malignant cases are of so short duration that there is no time for morbid changes to occur. In such the results of necropsy are negative.

Minutely examined, the exudate consists of polynuclear leckocytes inclosed in a fibrinous mass in which also diploccocci are found. The brain and cord may also be infiltrated with pus-cells. In the more chronic cases there is thickening of the meninges, with scattered yellow patches representing exudate.

As to *other organs*, there is no characteristic involvement. The *spleen* may be normal in size or, if the illness has lasted some time, it may be slightly enlarged. There may be congestion of the *liver*, *kidney*, *stomach*, and *intestines*, and even extravasation of blood. The same is true of the *lungs*, in which there may be bronchitis and pneumonia, the latter not very rarely. *Endocarditis* and *pleurisy* are sometimes found.

Symptoms.—Cerebrospinal fever does not present an unvarying picture in its symptomatology, and to attempt to portray every unusual symptom would occupy undue space. Several varieties are described, viz., (1) the ordinary form, (2) the malignant form, (3) the mild form, (4) the abortive form, (5) the intermittent form, (6) the chronic form. Only the most characteristic symptoms will be given, first of the ordinary form and then of the most important modifications of it.

I. *The Ordinary Form.* No definite time of incubation is known. A *prodromal period* of short duration with headache and pain in the back or headache and vertigo may precede, but sudden onset is characteristic, often associated with a decided *chill*. *Projectile vomiting* is also a frequent early symptom. *Headache* and *pain in the back of the neck* and back promptly appear. Though usually severe, this pain is sometimes so slight as to cause the real condition to be overlooked. It is sometimes so sudden and severe as to be compared to the sting of a bee. The muscles are rigid, and pain is increased on motion.

There is *fever*, but the temperature does not usually exceed 102° F. (38.9° C.). There is nothing characteristic in the fever, and the graphic chart shows no regular evening rise and morning fall. On the other hand,

it is extremely irregular. *Hyperesthesia* of the skin is a characteristic symptom. It is sometimes extreme, and as the disease increases in severity *rigidity* of the muscles of the neck and back becomes more marked. This muscular contraction may cause backward curvature of the head and even *opisthotonos*. *Clonic spasm* may also occur, though less frequent than tonic contraction. It is more common in children, in whom it may amount to convulsion and take the place of the chill. *Spasm of the muscles of the face* may occur, and of the eye-muscles, causing *strabismus*. Strabismus in any febrile case of doubtful nature should always lead to suspicion of meningitis. On the other hand, there may be *paralysis* of the face and eye-muscles, producing inequality of pupils, nystagmus, diplopia, and ptosis. More, rarely there are paralysis and wasting of trunk muscles, including those of respiration. The auditory nerves may be involved, affecting the *hearing*. and intolerance of sound is a characteristic symptom, as is also photophobia due to hyperemia of the retina. On the other hand, *anesthesia* of the *cornea* is found in some cases.

Delirium is very frequent, occurs early in the disease, and may pass into stupor or coma. It may be maniacal considerable effort being necessary to control the patient.

It has been stated of the *temperature* in this disease that it is rarely high. In some of the earliest descriptions of the disease—and there have been most interesting ones written almost a century ago—the writers speak of the skin as being cool. This was before the days of the clinical thermometer and the accurate measurement of temperature growing out of it. High temperatures do occur, though rarely, 105° and 106° F. (40.5° and 41.1° C.) being noted, and others even higher just before death. There is, however, no constant type. The temperature chart of the intermittent form resembles somewhat that of remittent fever, while sometimes the chart resembles that of the fastigium of typhoid fever in its spike-like delineation.

The *pulse* goes hand in hand with the temperature—that is, it is not very frequent at first, at least in adults. As the disease advances it grows more feeble and more frequent as the result of increasing debility of the patient. So, too, the *breathing rate* is not apt to be markedly influenced unless there be a lung complication.

The *urine*, as in other infectious fevers, may be scanty and albuminous; but it may also be increased because of the involvement of the nervous system. For a like reason there is sometimes glycosuria occasionally, associated, in severe cases, with Cheyne-Stokes breathing.

Another characteristic symptom is the *eruption*, although it is not present in more than one-half the cases. It is of at least two kinds—*herpetic* and *petechial*. *Herpes labialis*, although not always present, is nevertheless more frequent than in pneumonia. The herpes may be noted elsewhere than on the face—viz., on the trunk and extremities, extending exceptionally even to the ends of the fingers. The contents of the vesicles may be purulent; they may coalesce, break and dry, forming crusts. The petechial eruption is more general. It is an extravasation, and, like the similar eruption in typhus, does not disappear on pressure. The number of spots varies greatly; there may be only a few, or they may be very numer-

ous, fully justifying one of the names of the disease—spotted fever. It will not do, however, to exclude the disease by reason of the absence of these skin symptoms. The petechial eruption seems less common in the sporadic than in the epidemic form.

Other eruptions, as erythema, urticaria, sudamina, rose-colored spots like those of typhoid fever, pemphigus and ecthyma, have been noted. Gangrene of the skin has occured as the result of pressure. Some trophic influence may, however, be responsible for it.

Arthritis is not infrequent, varying in different epidemics, reaching nearly 20 per cent. of the severe cases in the epidemic described by S. Flexner and L. S. Barker.[1] The arthritis is deforming and is analogous to the arthropathies more or less common in spinal cord diseases.

Sometimes the disease sets in with *diarrhea*, though more commonly there is *constipation*. The *tongue* is less apt to be dry than in typhus, probably because the patient is less disposed to breathe through his mouth. Jaundice has been met with, and may be due to infectious inflammation of the bile-ducts.

Leukocytosis is a constant symptom, the increase being chiefly of the multinuclear variety of white cells. Vacuolation of blood-cells has also been noted.

Kernig's Sign.—Kernig, of St. Petersburg, called attention to a symptom which is at times a valuable aid to diagnosis in meningitis where the spinal membranes are involved. It is tested for in the following way: The patient is propped up in bed in a sitting posture, with the thighs flexed upon the abdomen and the legs partially flexed upon the thigh—a position commonly assumed by patients with prolonged spinal meningitis. An attempt is then made to extend the leg, when it will be found to be resisted by contraction of the flexor muscles, preventing its full straightening. When the patient cannot sit up in bed, the thigh may be flexed upon the abdomen and then an attempt made to extend the leg, which again fails if meningitis be present. Friis found the sign in 53 out of 63 cases, Netter in 45 out of 50, and J. B. Herrick in 17 out of 19.[2]

Recent studies by J. E. Miller and Robert N. Willson go to show that not only in this sign wanting in a certain proportion of cases of meningitis, but that it may also be present in a few normal individuals and others ill of other diseases. Of the nonmeningeal cases examined by Miller (190) the sign was found in 23.6 per cent. and by Willson in 26.8 of 120 cases. The sign is apparently no measure of the degree of intensity of the disease. Netter explains it as follows: "In consequence of the inflammation of the meninges the roots of the nerves become irritable, and the flexion of the thighs upon the pelvis when the patient is in the sitting posture elongates and consequently stretches the lumbar and sacral roots, and thus increases their irritability. The attempt to extend the knee is insufficient to provoke a reflex contraction of the flexors while the patient lies on his back with the thighs extended upon the pelvis, but it does so when he assumes a sitting posture."

The *Babinski* or extension toe reflex may be sought, though it is incon-

[1] "Am. Jour. of the Med. Sc.i," 1894, vol. cvii.
[2] "Am. Jour. of the Med. Sci.," July, 1899.

stant and occurs in hemiplegia and other results of lesions of the motor tract. *Brudzenski's* sign is a flexure movement in the ankle, knee and hip joints when attempt is made to flex the head on the chest. Passive flexion of the leg may cause the fellow limb to draw up a *Macewen's sign* is a hollow note developed on percussing the inferior frontal bone and is said to indicate fluid in the ventricle.

II. *Malignant Form.*—The malignant form of cerebrospinal fever is characterized by the suddenness of its onset and severity of its cardinal symptoms—the chill, headache, coma, collapse—followed by early fatal termination. There is little or no fever; indeed, the temperature may be subnormal. The pulse is feeble and slow, falling to 50 or 60 a minute, increasing, however, in frequency, as the disease progresses. The breathing is labored. The urine is scanty and albuminous. But for the prevalence of the epidemic such fulminating cases could not be distinguished from like attacks of other infectious diseases. Such cases may, however, occur even sporadically. They last but a few hours. They are more frequent in the beginning of an epidemic. The malignant form of smallpox is similar, and the presence of an epidemic of one or other disease must settle the question.

III. The mild *form* presents a corresponding mildness of symptoms, and only the presence of an epidemic leads to its recognition.

IV. The *abortive form* terminates abruptly after a sharp development of characteristic symptoms.

V. The *intermittent form* is characterized by remissions and exacerbations in the fever every day or second day, without, however, the regularity of intermittent fever, for which it is sometimes mistaken. The fever resembles somewhat that of pyemia. This form is very trying, the remissions and intermissions giving rise to delusive hopes which are as often shattered.

VI. Finally, the term *chronic form* is applied to cases prolonged beyond the usual duration, in which the headache, gastric irritability, and vague neuritic pains reduce the patient to such an extremity of exhaustion and emaciation that he welcomes death as a relief to his suffering; or partial recovery may take place with crippled motion, defective senses, and severe pains, which are a source of constant discomfort. On the other hand, some remarkable recoveries, even in these advanced stages, are reported, so that one should not be discouraged from continuing therapeutic effort.

Complications and Sequelæ.—Of the complications of cerebrospinal fever, *croupous pneumonia* has already been mentioned as not infrequent as well as that it is sometimes difficult to say which disease is primary. The initial chill and herpes are characteristic of both affections, and close attention to other conditions must be given, such as the presence or absence of an epidemic, the order of appearance of the symptoms, the nervous and muscular preceding in cerebrospinal fever, and coming on later in pneumonia. *Other complications* are those which not infrequently accompany infectious diseases, including *pleurisy*, *endocarditis*, *pericarditis*, *polyarthritis* with possible suppuration, and others.

Of the sequelæ the most important are *blindness due to optic neuritis* and more rarely *keratitis*, *deafness* from disease of the labyrinth, *paralysis*

more or less extensive, invading especially groups of muscles, including those of the face. There may be aphasia and defective articulation. There may be also persistent headache, shooting muscular pains, and mental weakness. Next to scarlet fever cerebrospinal meningitis is the most frequent cause of deafness. Even *chronic hydrocephalus* and *abscess of the brain* are included among sequelæ. Von Ziemssen says the former is indicated by "paroxysms of severe headache, pain in the neck and extremities, without vomiting, loss of consciousness, convulsions and involuntary discharges of fęces and urine." He also says that of the deaf and dumb in the institutions of Bamberg and Nuremberg, in 1874, a majority of the pupils had become deaf from cerebrospinal meningitis.

Nasal catarrh may be an early symptom, and Strümpell suggests it may precede and be the starting point of the invasion. The discharge often contains the meningococcus, as in ten out of 15 cases in the Boston epidemic alluded to.

Diagnosis.—A certain diagnosis may be arrived at by the use of spinal puncture described below. This should be done in every suspicious case. The fluid obtained which is usually cloudy should be centrifugated also and the cells stained after Gram's method. If the diplococci are found within the cells by the use of a simple stain and do not stain by Gram's method the case may be considered as one of epidemic cerebrospinal meningitis and treated as described below. The diagnosis in epidemic cases is usually easy, although it is more than probable that under such circumstances some cases are classified as cerebrospinal fever when they are really something else. During epidemics *typhus fever* is the disease with which it is most frequently confounded, especially as epidemics of typhus and cerebrospinal fever sometimes prevail simultaneously. The difficulty is greatest at the beginning of the attack, for as time passes, the diseases diverge in symptoms. Typhus fever is not characterized by the severe pain in the head and back of the neck, nor by opisthotonos, both of which may, however, be absent in cerebrospinal fever or be so slight as not to attract attention. In typhus fever the spots are more constant and numerous than in cerebrospinal fever. Herpes does not occur in typhus. The typhoid state may be equally pronounced in both, but in general it may be said to be more marked in typhus. The two diseases differ in their duration, typhus having a pretty definite duration of about two weeks, whereas cerebrospinal fever is either shorter or longer.

The joint complications not infrequently associated cause a resemblance to articular rheumatism, which may lead to confusion at first.

The isolated cases give most trouble. *Typhoid fever*, especially the meningeal from of typhoid, in which there is extreme headache and active delirium, sometimes simulates cerebrospinal fever in the beginning. The onset of typhoid is also slow; as a rule, there is no vomiting nor severe muscular pain. In typhoid fever there is *no* leukocytosis. Widal reaction is present and the spinal fluid is clear as a rule and does not contain diplococci within the cells of the fluid.

Pneumonia is another source of confusion, especially as the two diseases are sometimes associated, and it is almost impossible to say which is primary. Here too spinal puncture will make the diagnosis If there is a cloudy

spinal fluid the diplococci will stain by Gram's method, and will be found largely outside the cells. The meningeal complications in pneumonia are more apt to invade the convexity, whence there arise muscular contraction and tremor, but not retraction of the head.

Tuberculous meningitis presents some resemblance to cerebrospinal fever. While usually less sudden in its development, it is not always so. Delirium and stiffness of the neck, retraction, and even opisthotonos occur. It is, however, scarcely ever primary, and there are no skin symptoms. The termination of tubercular meningitis is invariably fatal. The presence of a focus of tuberculosis is a great aid to diagnosis. Here also the character of the cerebrospinal fluid is diagnostic. It is usually perfectly clear, if turbid the lymphocytes prevail, and tubercle bacilli may be demonstrated by carefully centrifuging the fluid and examination of the pellicle which forms on standing.

Influenza, too, in one of its many forms occasionally simulates cerebrospinal fever, at times very closely. Extreme muscular pain is characteristic of both, and when influenza is associated with actual cerebrospinal meningitis, with delirium and stupor, as it sometimes is, one may be excused for being in doubt. Although both are diseases of short duration, influenza spends its fury earlier, and is thus a shorter disease unless prolonged by one of its complications.

Quincke's lumbar puncture is necessary to establish a diagnosis. The operation is done with the patient lying on the side, with knees drawn up, and the upper shoulder turned forward. A needle furnished with a stillette 10 to 20 cm. in length and of large caliber and a short beveled point. In lieu of this needle one in use in an ordinary aspiration set may be used, and is introduced midway between the third and fourth or the fourth and fifth lumbar vertebræ, below the spinous process, a little to one side of the median line, the thumb of the left hand of the operator being placed between the spinous processes as a guide. The needle should enter one centimeter from the median line on a level with the thumb, and be directed slightly upward and inward, or the patient may be held upright and the puncture made. Under these circumstances the needle need not be pointed upward. At the depth of two centimeters in infants and four to six in adults it should enter the canal. The fluid may be collected in a sterilized test-tube, care being taken not to allow it to run down the side of the tube. Fifteen to 30 cubic centimeters should be withdrawn, for chemical, bacteriological, and microscopical examination. A cloudy fluid is almost always present in epidemic meningitis; rarely, it may be clear, or the fluid from an upper puncture may be clear, and from a lower turbid. Blood may be present in both. Marfan's site is in the median line and is preferred by J. P. Crozer Griffith. Chipault's site is between the fifth lumbar vertebra and the sacrum.

Prognosis.—Cerebrospinal fever is a grave disease, but the mortality varies greatly in different epidemics, ranging from 20 to 75 per cent. according to Hirsch, while v. Ziemssen places it for mild epidemics at 30 per cent., and for severe ones at 70 per cent. The death-rate is higher for children, those under two years almost invariably perishing, while few under five survive. The old likewise succumb easily.

Of few diseases is the course more variable and uncertain. It is favorably affected by the early use of Flexner's serum. From a duration of two to three days only it may be prolonged to weeks and even months, and its consequences may be permanent. Usually, however, improvement may be looked for if the patient survives five days, more than half the deaths occurring within this period. A remission of symptoms may take place on the third day, to be followed after a very short time by a relapse. This often misleads and gives the illusive hope of permanent improvement. Convalescence is characteristically slow, the symptoms yielding gradually. If the termination be fatal, the cardinal symptoms likewise gradually subside, but are replaced by growing debility and exhaustion.

Relapses are prone to occur, prolonging the case indefinitely, while a chronic or protracted form, to which reference has been made is probably due to the presence of one of the persistent or progressive lesions above referred to.

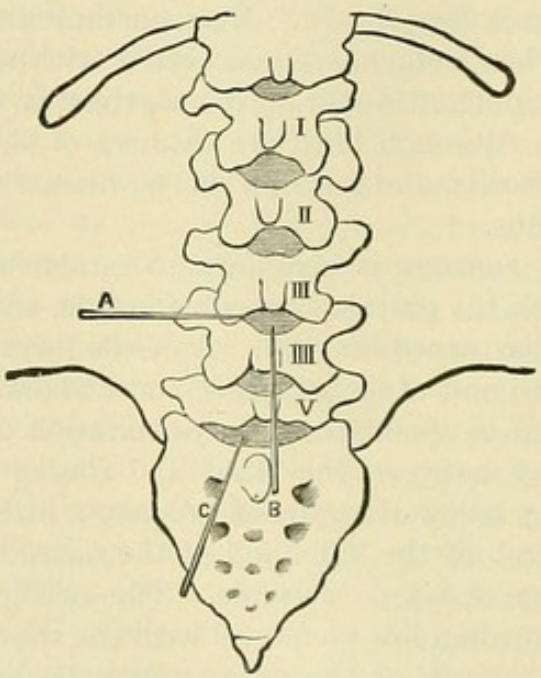


FIG 19.—Method of Puncture for Spinal Drainage. A. Quincke's site. B Marfan's site. C. Chipault's site.—(*Chipault.*)

Sporadic Cerebrospinal Meningitis.—This form of cerebrospinal meningitis may not be surely differentiated from the epidemic form without examination of the cerebrospinal fluid. It requires a separate, though brief, consideration. It has been already said that such cases occur at intervals, and more especially at odd times succeeding the prevalence of an epidemic in a city. Osler, in his Cavendish lecture, 1899, has taken some pains to analyze the cases of cerebrospinal meningitis treated at the Johns Hopkins Hospital, Baltimore, with a view to ascertaining what proportion was strictly sporadic and noncomplicating. He finds that after eliminating pneumococcic meningitis complicating pneumonia and pneumococcic meningitis due to local infection and streptococcic cases of the same class (surgical cases), there remained a few primary cases due to the pneumococcus, a few of miscellaneous meningitis—*i. e.*, caused by unidentified bacilli—and a few due to the diplococcus intracellularis. The following table will show the organisms causing the various forms of cerebrospinal meningitis.

TABLE OF CHIEF FORMS OF ACUTE LEPTOMENINGITIS.

Primary.	1. Cerebrospinal fever	(a) Sporadic (b) Epidemic	Diplococcus intracellularis.
	2. Pneumococcic M.	Meninges alone involved in a gen. pneumococcic infection.	Pneumococcus.
Secondary.	1. Tubercular M.	Bacillus tuberculosis.	
	2. Pneumococcic M.	(a) Secondary to pneumonia, endocarditis, etc. (b) Secondary to disease or injury of cranium or its fossæ.	Pneumococcus.
	3. Pyogenic.	(a) Following local disease of cranium (or a local infection elsewhere). (b) Terminal infection in various chronic maladies.	
	4. Miscellaneous acute infections.	In typhoid fever, influenza, diphtheria, gonorrhea, anthrax, actinomycocis, and other acute diseases.	Typhoid bacillus Influenza " Diphtheria " Gonococcus.

Treatment. SERUM THERAPY.—Every case of cerebrospinal meningitis due to micrococcus intracellularis, indeed every case of cerebrospinal meningitis where the infecting organism in unknown should be given at once a dose of Flexner's serum. This may now be obtained from reliable manufacturers under the name of Antimeningitic serum. The use of this serum has reduced the mortality to about 20 per cent. It must be given into the spinal canal. A needle is introduced, the fluid withdrawn and the serum introduced through the same needle. The amount of serum should be injected should about equal the serum withdrawn. Quiet and the absence of disturbing causes, such as excess of light, too much company, are absolutely essential. The food should be simple and liquid, with an abundance of water. The symptom demanding the promptest relief is pain, and for this there is no substitute for *opiates*, and of these the best preparation is *morphin*, and the best mode of administration is by hypodermic injection. Doses sufficient to accomplish their purpose should be given, say 1/4 grain (0.016 gm.) to 1/2 grain (0.032 gm.), night and morning, for an adult. The tolerance for the drug is great. It may be combined with 1/150 grain (0.00054 gm.) to 1/100 grain (0.00064 gm.) of atropin. The same preparation may be given by the mouth if the hypodermic administration is not convenient, but the *deodorized tincture of opium* may be better borne, and where the more frequent administration of opiates is necessary, as hourly or bihourly, this preparation is to be preferred because of the possible harmful effects of the too frequent use of the hypodermic syringe. The action of the drug is, of course, to be carefully watched. Phenacetin, antipyrin, salicylic acid, and this class of drugs are no substitute for opium in this painful malady. *Hexamethylenamin* (urotropin) should be given at once in doses of 5 grains (0.3) every 3 hours when the disease is suspected. Hot baths may be employed for the same purpose.

When there are spasms or convulsions there is no remedy equal to *chloral*. If it cannot be administered by the mouth, a dram (4 gm.) dissolved in 2 ounces (60 c.c.) of water may be given to an adult, without hesitation, per rectum. In extreme cases chloroform or ether may be inhaled for the same purpose. The bromids may be used as adjuvants in mild cases, but of themselves are altogether inefficient.

Cold may be applied to the head for the headache and other meningeal symptoms, and is best used in the shape of an *ice-cap* or ice-bladder or Leiter's coil. Cold may also be applied to the back of the neck and spine, and according to James Barr over the splanchnic region. These measures must be discontinued when the temperature falls to normal. *Counterirritation* to the back of the neck and spine has long been employed, by blisters, but is not recommended. General bleeding is not recommended. Free movements of the bowels must be maintained by castor oil or calomel, and the bladder watched.

Measures of a very decided character to reduce the temperature are not, as a rule, needed. Simple sponging suffices for the most part. Should this be insufficient, however, tub bathing may be used as in typhoid fever.

The *nourishment* should be of the best, including animal broths and milk, and where, as is frequently the case in the early stages, they cannot be tolerated by the stomach, they may be given peptonized per rectum, not more than 4 ounces (120 c.c.) at one time. I have thus nourished for several days until the stomach became retentive a case despaired of, which ultimately recovered. Forced alimentation by the stomach-tube is recommended by Heubner. Alcohol is contraindicated in the early stages unless there be unusual adynamia. Later, when exhaustion begins to show itself, it may be used and pushed as under similar conditions in other diseases.

The lumbar puncture is strongly recommended by Williams, Brower, W. Cuthbertson,[1] and others as a curative measure; Osler admits possible benefit therefrom. Temporary relief undoubtedly ensues. Laminectomy and local therapeutics, including drainage, have not furnished encouraging results at the Johns Hopkins Hospital.[2]

The resulting paralyses should be treated by massage and electricity, and as already suggested we should not be discouraged from persisting, as remarkable cures have been accomplished.

Satisfactory results have followed the subaqueous treatment recommended by Goldscheider[3] to which attention was called by William G. Spiller. It consists in active movements by the patient while submerged in a bath at a comfortable temperature. The movements are not passive, but active and voluntary.

ERYSIPELAS.

SYNONYMS.—*The Rose; St. Anthony's Fire.*

Definition.—An acute, contagious dermatitis associated with the usual signs of inflammation—swelling, heat, pain, redness, the formation of blebs and a peculiar disposition to spread.

Etiology.—The *streptococcus erysipelatis* of Fehleisen is a minute, cleft fungus, a micrococcus in the narrow sense, three to four microns in diameter, arranged in pairs (diplococci) or chains (streptococci) of from six to 12 cells. The erysipelas coccus resembles closely the *streptococcus*

[1] "Chicago Med. Recorder," June, 1899.
[2] Osler, "Cavendish Lecture," June, 1899.
[3] "Ueber Bewegungstherapie bei Erkrankungen des Nervensystems," Goldscheider, "Deutsche medicinische Woschenschr.," January 27, 1898.

pyogenes of Rosenbach—in fact, cannot be distinguished from it microscopically, while even the cultures of the two organisms resemble each other very closely. The *streptococcus pyogenes* is said by Hoffa to grow more slowly and less uniformly than that of erysipelas, and presents also a brownish discoloration in the middle of its colony. They behave very similarly when inoculated in animals. Simon asserts that the micrococcus of erysipelas is identical with that of pyemia, and this belief is now quite general. Klebs suggests that more than one organism may be concerned in the causation of erysipelas.

The organism probably operates as a local irritant producing the dermatitis. From this as a focus constitutional infection is set up, as in diphtheria, probably through the influence of a toxin generated by the micrococcus. The bacterium is found in the lymph-vessels and lymph-spaces of the periphery of the inflamed area, and not in the center, by which fact the peripheral spread of the disease is explained. It is readily found by cultures made from the blood of the veins and heart and even the urine.

The organism is transferred from one person to another by direct contact, or by the intermediation of a third person, or through the atmosphere. It cannot be said, however, that the disease is highly contagious in the absence of surgical injury, for in Tyson's experience as a hospital interne at the Pennsylvania Hospital and later as a visiting physician in the Philadelphia Hospital, though it was the custom to keep the erysipelas cases in the ordinary medical wards, he cannot recall a single instance where the disease was communicated to another patient in the ward. It was very different, however, in the surgical wards, where the disease would spread rapidly from one patient to another, showing the importance of the open surface as a condition of the spread. The lying-in woman is very readily inoculated, so that no physician should attend a case of labor while attending one of erysipelas. Certain kinds of wounds, as lacerated wounds and scalp wounds, are especially prone to erysipelas. Clean-cut wounds in other locations suffer less frequently. Leech-bites, vaccination punctures, the wounds of the cupping scarificator and of the subcutaneous syringe, are also favorable starting points. Chronic inflammatory processes and skin diseases may also have erysipelas engrafted upon them.

Erysipelas is prone to occur in the epidemic form, more especially in the spring[1] of the year in old and unclean hospitals, but such epidemics have become much rarer in the last 20 years. This is doubtless one of the results of antisepsis, now so generally practised. The feeble, the intemperate, and those having Bright's disease or other affections weakening natural resistance are more prone to the disease. An interesting case of Bright's disease in the Philadelphia Hospital had frequent attacks of facial erysipelas, always accompanied by hematuria. *Relapses* and *recurrences*

[1] The influence of the seasons is very well set forth by James M. Anders in a paper on "Seasonal Influences in Erysipelas, with Statistics," wherein he has 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 months 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.

of erysipelas are prone to occur, and a person once attacked by erysipelas, far from being protected, is rather predisposed to a second attack. A family predisposition of erysipelas may exist.

Morbid Anatomy.—Like all acute inflammatory states of the skin, erysipelas fades away after death and leaves little, if anything, to be seen unless it has proceeded to the formation of blebs or abscesses. Swelling and corresponding deformity of the part, especially of the face, when extensive, may remain, but even this subsides with the lapse of time after death and may totally disappear.

Minute examination finds the cocci in the lymph-vessels and spaces at the periphery of the inflamed area, as already stated, and even in the uninflamed tissue beyond the margin.

Various complications attend erysipelas and add their morbid anatomy to that which is more essentially that of the disease. The most important of these are *pyemic abscesses* of internal viscera and *hemorrhagic infarcts* of the lung, spleen, and kidneys. The kidneys are especially apt to be congested, and the lesions of acute or subacute nephritis are sometimes found, and more rarely suppurative orchitis.

Symptoms.—The form of erysipelas which more particularly concerns the physician is the so-called *idiopathic erysipelas*, which arises independently of any apparent traumatic lesion, but since all erysipelas implies some lesion, however minute, the term is a misnomer. The fact remains, however, that the physician is most frequently called upon to treat the form of erysipelas in which there is no discoverable local lesion.

There is a *period of incubation* of from one to eight days, after which this variety of erysipelas begins at times with a *chill* or succession of chills associated with a *loss of appetite* and feeling of general discomfort. At other times the chill is wanting. In either event there soon appears a *small, red, burning spot* a few lines in diameter, usually on the face, oftenest on the bridge of the nose or on the chin. It spreads rapidly, and as soon as sufficient size is attained there is a very characteristic elevation of the patch above the surrounding tissue, which can be recognized by carrying the finger across it. This is of diagnostic value. The future extension of the process is upward over the forehead and laterally toward the ears until the whole face, and more rarely also the neck, is invaded. The eyes become closed by *swelling*, the features are distorted, and the sum of changes produces an appearance not soon to be forgotten. In other parts of the body, as the arms and legs, the same process may go on, but there is not the unsightly distortion found as in the case of the face and head. In some cases the process proceeds to suppuration, and deep-seated *abscesses* form. These must result from mixed infection with other pyogenic organisms, unless indeed the organism be the same as that of suppuration. *Blebs* form, particularly on the lobes of the ears and on the eyelids, while little vesicles are always visible through a lens. From these a serum may exude and dry on the skin. As the dermatitis extends to new areas, the earlier spots dry up and desquamate. The disease seldom lasts more than four days in one spot, although it may revisit the same spot during one attack.

There may be erysipelas of the mucous membranes, which may extend

to the skin, or the reverse may take place—extension from the skin to the mucous membrane.

Fever probably always precedes, though not noted in the beginning, and it rapidly becomes higher, reaching as high as 105° F. (40.5° C.). There is a corresponding *frequency* of pulse, associated with *headache* and sometimes *delirium*. The fever continues as long as the disease continues to spread. Often a sudden drop, a crisis, occurs on the fifth to the seventh day, followed by another rise if the disease takes a fresh start.

In more serious cases fever and delirium may be followed by *drowsiness*, *stupor* and a *coated, dry tongue*—all the symptoms, in fact, of a typhoid state. The *urine* is scanty and a febrile *albuminuria* may be present—in fact, to a degree, may be said to be constant[1]—and nephritis sometimes results, while a pre-existing nephritis may have an acute exacerbation engrafted upon it. Mention has already been made, under the head of Etiology, of *hematuria* occurring in these cases.

Gangrene may be associated with the deep-seated varieties, constituting gangrenous erysipelas.

Complications and Sequelæ.—The possible complications are numerous, but in practice are really not often encountered. The most frequent is *meningitis*, the result of extension by continuity through the openings of the cribriform plate of the ethmoid bone or by contiguity from the scalp through emissary veins of the skull, but I have never seen such a case. William Osler, however, traced the extension from the face along the fifth nerve to the meninges, causing an acute meningitis and thrombosis of the lateral sinus.

Edema of the glottis is the result of extension of the disease to the mucous membrane of the glottis. It is promptly fatal, unless relieved.

Malignant ulcerative *endocarditis* is also with comparative frequency secondary to erysipelas, three cases out of 23 being sequelæ of this disease. Of *cardiac complications*, pericarditis, endocarditis, and myocarditis; of *pulmonary*, bronchitis, pneumonia, and pleurisy may be mentioned as possible; also jaundice, dysentery, and hemorrhages from the nose and bowels. *Purpura* is an occasional complication. Nephritis of hemorrhagic variety has already been mentioned, and even glycosuria has been noted, possibly an accidental association. *Septic* and *pyemic complications* do, however, occur and are among the causes of death. *Suppuration in the testicle has been referred to.*

Among the sequelæ may be mentioned a *loss of hair*. *Cicatricial new formations* replace the parts destroyed by gangrene and may produce deformity by their contraction. On the other hand, hyperplastic new formations resembling elephantiasis Arabum may result. *Hyperesthesia* and *neuralgia* of the involved areas, *anesthesia* with which atrophy of the skin may be associated, symmetrical gangrene of the fingers, and painful affections of the joints have all occurred as sequelæ.

Erysipelas may be associated with other infectious diseases, such as typhoid and typhus fevers, diphtheria, scarlet fever, and the like.

Diagnosis.—The diagnosis of erysipelas is usually not difficult, although

[1] See paper by J. M. DaCosta on "The Internal Complications of Acute Erysipelas," "Am. Jour. of the Med. Sci.," October, 1877.

many conditions are called erysipelas by the ignorant which are not of this nature. The acuteness of the disease, the rapidity of its spread, the constitutional disturbance and fever distinguish it from other conditions that superficially resemble it.

Prognosis.—The prognosis, in the vast majority of instances, is favorable. Only in the aged, the intemperate, and those of broken health from other causes does it prove fatal, as a rule. Complications, especially meningitis and septic states, are causes of death. On the other hand, erysipelas is said to exert a favorable influence on certain acute diseases, such as acute rheumatism, choroiditis, and even morbid growths. It has even been suggested to inoculate erysipelas for the cure of such affections.

Treatment.—The patient should, of course, be isolated. It is more than likely that a decided majority of cases of idiopathic erysipelas would get well without any treatment whatever. In other words, the disease is *self-limiting*. As the disease is exhausting, internal *treatment* should be *restorative* and *supporting*. Quinin, iron, nutritious food, and stimulants are indicated, while the patient should be kept at rest. The *tincture of the chlorid of iron* is used throughout North America because of some supposed specific influence over the disease, and doses as large as a dram every three or four hours have been given. We have always given iron, but never in such doses, and we are doubtful whether it exerts any specific effect of the kind claimed. The natural duration of the disease is short, and the effect claimed from the iron is no prompter than that which nature brings. Ten minims (0.666 gm.) every two or three hours are sufficient, and it is exceedingly doubtful whether larger quantities than this are absorbed. Where debility is marked, alcohol in some of its forms should be administered.

J. M. DaCosta first suggested the use of *pilocarpin* in the treatment of erysipelas, more particularly in the early stages. J. L. Salinger,[1] A. A. Eshner, and S. D. Barr also report favorably on the same treatment, which should, however, be employed cautiously. It is recommended that 1/6 grain (0.01 gm.) be administered hypodermically every three hours until free sweating ensues. After this the interval is increased to four or six hours.

Serum Treatment.—Antistreptococcic serum may be tried. It is far better to use an autogenous vaccine, however, the stains of streptococci are so numerous that a stock serum made by their use may be easily useless in any given case.

An infinite variety of *local measures* has been suggested to arrest the spread of the disease, all of which are useless to this end, although some of them are useful in allaying the burning. For this purpose nothing is better than the old-fashioned mixture of lead-water and laudanum in the proportion of four parts of the liquor plumbi subacetatis dilutus, U. S. P., to two of laudanum. Or a mixture may be made of acetate of lead 1 ℈ (1.3 gm.), powdered opium 90 grains (6 gm.), and water 6 f℥ (180 c.c.). Lead-water alone is an efficient local application for this purpose; so is cold water. Dusting the surface with finely levigated oxid of zinc or subnitrate of bismuth also has a soothing effect. Of late, *ichthyol* has become a popular local dressing. It should be added to glycerin or collodion in the

[1] "Therapeutic Gazette," March 15, 1894.

proportion of 2 drams (8 c.c.) to the ounce (30 c.c.) of glycerin or collodion. A saturated solution of magnesium sulphate, epsom salts, is grateful and efficient.

SEPTICEMIA AND PYEMIA.

SYNONYM.—*Bacteriemia.*

Definition.—Pyemia and septicemia are general febrile conditions caused by the entrance into the blood of pathogenic micro-organisms. They are distinguished from sapremia, which is the condition of local development of micro-organisms associated with the entrance of their *toxic products* into the circulation but not of the organisms themselves. Septicemia and pyemia are sometimes included under the single designation of bacteriemia. They are in man caused usually by the entrance of pyogenic organisms—*streptococcus pyogenes* and *staphylococcus pyogenes aureus* or *albus*—into the blood. In septicemia the development of the organisms is not associated with a special localization of the micro-organisms in the internal organs with the production of abscesses, whereas in pyemia the presence of secondary pus foci in different organs of the body constitutes the distinguishing feature of the condition.

Etiology.—While the *pus* organisms have been heretofore held responsible for the majority of intoxications of the blood by their pathogenic products or toxins, from the medical standpoint the term *septicemia* may be applied to the toxic condition produced by any of the pathogenic bacteria which invade the blood and tissues with or without a visible site of infection. The proportion of these last has of late enormously decreased, because of the antisepsis practised by surgeons, while the medical septicemias have not much diminished.

Illustrative cases of the more usual form of septicemia are puerperal fever following retained placenta, infection by scarlet fever or erysipelas or during difficult labor involving laceration, and the poisoning by a dissecting wound. Among medical septicemias may be mentioned those arising from typhoid fever, pneumonia, diphtheria, and gonorrhea. These are all primarily local infections. The symptoms set in in from three to four hours to three or four days, more frequently within 24 hours.

The same essential cause lies at the bottom of pyemia as of septicemia, but associated with the former as important etiological factors are *thrombosis* and *embolism*. To this association Virchow first drew attention, and it is to thrombosis or embolism that the pyemic abscesses are due. Fragments of a venous thrombus due to phlebitis at the seat of putrid inflammation are broken off and carried in the circulation until a lodgment is affected. These fragments swarm with bacteria, causing intense inflammation which goes on to abscess formation, producing the metastatic or embolic abscess. Emboli may be multiple and there will be as many abscesses as lodged emboli. A frequent source of multiple abscesses is the disease, malignant or ulcerative endocarditis, itself a specific inflammation caused by some pathogenic organism floating in the blood and lodging on the heart valves, where it excites a septic valvulitis. The vegetations produced by this may be broken off and become emboli. These are carried through the

arterial system to points of lodgment and constitute the arterial pyemia of Wilks. Osteomyelitis is also a cause of pyemia. The term *idiopathic pyemia* is applied to that form in which multiple abscesses coexist with the other symptoms of pyemia, but no infective focus is discoverable. It will be remembered that the noninfectious embolus produces simple hemorrhagic infarct.

The *seats of election* for abscess in pyemia in their order of frequency are as follows: The lungs, liver, spleen, kidneys, brain, and joints, the subcutaneous connective tissue, and subperitoneal connective tissue, including pelvic connective tissue. The marrow of long bones and the parts about the cavity of the middle ear are also seats.

Abscesses occur in the lungs when the septic emboli originate in osteomyelitis or in inflammatory affections of the periphery; in the liver, when they arise from septic foci in the portal area, especially in the intestines; the pelvic connective tissue, when they start in the uterus and its appendages; in the spleen, kidneys, and brain, if the emboli arise in the left heart or are so small that they can pass from the right heart through the lungs to the left heart. Emboli may also, in rare instances, pass from the right heart to the left heart through a pabulous foramen ovule. Suppuration is not limited to the agency of streptococci and staphylococci. The *gonococcus*, the *bacillus coli communis*, the typhoid bacillus, the *bacillus lanceolatus*, and others are equally capable of producing suppuration.

Symptoms.—A rapidly rising *fever* is the first symptom of pyemia and septicemia, often so closely followed by a chill that its pre-existence is not suspected. The severity of the chill corresponds with the intensity of the infection and the degree of inflammation resulting from it. The temperature during the chill reaches 103° to 104° and 105° F. (39.4° to 40° and 40.5° C.) and is followed by a *sweat* and fall of temperature, after which the latter again arises to a point even higher than that first attained. Then follows another sweat and fall and thereafter a succession of intermissions, variable but quite characteristic. The rise is generally toward evening, and thus there is a certain resemblance to typhoid fever, while the rigors and sweats suggest malaria. The evening rise is by no means constant, and irregular fluctuations in the temperature are characteristic. There are other symptoms of fever—viz., *thirst*, *loss of appetite*, and *nausea*. The *strength* of the patient rapidly wanes, he soon sinks into a condition of exhaustion and semiconsciousness, from which, however, he may be aroused to take medicine and nourishment.

The various local involvements cause *localized symptoms*. Emboli in the lungs cause cough and hurried breathing, but there may be no distinctive physical signs; in the liver, they may cause tenderness and enlargement with jaundice; if in the kidney, there may be no sign or there may be albuminuria and hematuria; if in the intestines, diarrhea; if in the skin, superficial abscesses; if in the joints, swelling, tenderness, and fluctuation; if in the brain, paralyses occur depending upon the seat of the abscess. Delirium and unconsciousness are common. There may also be secondary abscesses of the parotid gland and pancreas, the former, producing hard painful swelling and the latter deep-seated pain in the epigastric and umbilical regions.

The *abscesses* contain the pyogenic bacteria, which are responsible for them.

Diagnosis.—The diagnosis is not usually difficult, though sometimes the disease is overlooked and the symptoms ascribed to some other cause. Reference has always been made to its resemblance to *typhoid fever* and *malarial fever*, but the physician should not be long in doubt. A careful study of the case will show marked differences in history, while the *status præsens* exhibit only a superficial resemblance. There are no rigors followed by sweats in typhoid fever, as a rule, and the temperature chart in pyemia is much more irregular. Especially confusing are those cases of septicemia in which the blood responds to the Widal test of which two are in the wards at this writing. The suddenness of the pyemia is characteristic, though it is by no means invariable. In *remittent* fever the chill, fever, and sweat are more regular, the prostration is not so extreme, and, above all, it is promptly cured with quinin. The plasmodium, if found, definitely settles the question as to the malarial fever, and the Widal test that of typhoid fever. There should be no confounding of pyemia with simple intermittent fever. The complete absence of symptoms between paroxysms is in no way comparable to the evident desperate illness despite the temporary absence of fever in pyemia.

Among the causes of pyemia that have been overlooked is osteomyelitis. Gunshot injuries of bones and compound fractures, if followed by suspicious symptoms should lead to investigation. Malignant or ulcerative endocarditis is often overlooked, and not without reason, as it is so often overshadowed by other symptoms. A cardiac murmur, with irregular temperature and sweating and unusual prostration, should excite suspicion.

Gonorrhea and prostatic abscess are occasionally causes, as are also tuberculosis of the kidney and calculous pyelitis, the last two, perhaps, more frequently than the first two.

Prognosis.—The prognosis is very grave. Even when recovery takes place in comparatively mild cases, it is with shattered health. More fortunate are the rarer instances of recovery after puerperal pyemia, which, when they do occur, are more apt to be complete. When calculous pyelitis and even tuberculous pyelitis are causes, operation often furnishes prompt relief more or less complete.

Not all fatal cases are promptly so. There is a form of chronic pyemia lasting for months, in which the symptoms are less distinctive and in the history of an infected wound may be the only cue to its real nature. One such case came under Tyson's observation, that of a young physician who received a dissecting wound from which the symptoms started and which terminated fatally with meningitis after many months' illness.

Treatment of Septicemia and Pyemia.—First *remove*, if possible, the *primary surgical focus* and *relieve secondary foci* as they appear. After that the symptoms are to be combated and the *strength supported* to the utmost. To the latter end the most nutritious and easily assimilable food, alcohol moderately, and strychnin are the sheet anchors. To these may be added sponging to lower the temperature. To check sweating, atropin, oil of erigeron in doses of 10 to 30 minims (0.65 to 2 gm.) in a capsule or on sugar; ergot 15 to 30 minims (1 to 2 gm.); the dilute mineral acids, 15 to 30

minims (1 to 2 c.c.). Antipyretics should not be used to reduce temperature, it is better to accomplish the same thing by hydrotherapy.

Among the more favorable cases, in which operative treatment is followed by prompt and sometimes more than temporary relief, are cases of septicemia originating in vesical and prostatic disease and calculous and tuberculous pyelitis. In tuberculosis of the kidney, as tuberculosis elsewhere, especially illustrated in the peritoneum, exposure to the air seems to have a destructive influence upon the bacillus. If the source of the infection cannot be reached by surgical measures, antistreptococcic serum should be tried without hesitation though frequently there is no relief by its use. Twenty to 30 cubic centimeters should be injected every six to eight hours daily until decided improvement in symptoms takes place, after which the interval between injections should be increased. Smaller doses may be injected in milder degrees of the poisoning.

Prophylaxis is much more efficient than treatment, and with modern aseptic surgery and aseptic obstetrics septicemia and pyemia are becoming much more infrequent.

HYDROPHOBIA.

SYNONYMS.—*Rabies; Lyssa.*

Definition.—Hydrophobia is an acute infectious disease of animals, communicable to man, and characterized by intense tonic spasm beginning in the larynx.

Etiology.—All warm-blooded animals are subject to the disease. The dog is the most frequent victim, and it is from that it is almost invariably communicated to man. The wolf, cat, and skunk are also frequent subjects, and may communicate the disease to human beings by their bites, that of the wolf being especially virulent. In such cases, whatever the contagium may be, its bearer is conceded to be the saliva of the animal. The contagium is a fixed and not a volatile one. The researches of Pasteur go to show that it is also contained in the central nervous system, especially the spinal cord, medulla and brain. Klebs suggested that the disease is caused by a bacterium found in the salivary glands of those affected with hydrophobia. Gibier, Fol and Babès claim to have found micrococci in the brain-substance, but these claims have not been confirmed by others, though their experiments have been repeated. There can scarcely be a doubt that an organism is the medium of infection.

The *period of incubation* is extremely variable, ranging from one week to two months or longer. Even two years are said to have elapsed before symptoms set in. The average may be put down at from six weeks to two months; Stevenson's records in Public Health reports for 1912 confirm the above statements. In 65 cases in which the incubation time is discussed 57 developed the disease in less than four months, and three over 12 months —but by no means all persons bitten take the disease, a most important point to be remembered in estimating the efficacy of supposed curative measures. Not more than 15 per cent. of those bitten by dogs, according to Horsley, become affected. Various causes contribute to this. Thus, the saliva may be wiped off in the transit of the tooth through the clothing, and

such removal or virus may reduce the danger of the second bite of the same animal, even though it be on the unprotected skin. Again, the young are more susceptible. Statistics by Watson, in America, and by Bollinger, in Germany, show more cases to have resulted from bites in the upper extermities, while, according to Horsley, wounds about the face and head are more apt to cause the disease than those on the hands, which are second in order, and after these comes bites on other parts of the body. A much larger proportion of those bitten by wolves perish, from 40 to 80 per cent., according to different authorities. Wounds about the face and hands are much more serious than in parts covered by clothing.

To a very important practical question, How long after a bite may the dreaded suspense of an expected outbreak last? Accurate answer seems now possible. Cases treated promptly by inoculation after the Pasteur method rarely develop the disease. Cases not so treated are still uncertain.

Morbid Anatomy.—The morbid anatomy of rabies, so far as recognized, is limited to the upper spinal cord, medulla, pons, and cortex of the brain, and is revealed only by the microscope. The blood-vessels are dilated and congested, the perivascular sheaths are invaded with leukocytes, and there are even small hemorrhages. There is hyperemia of the pharynx, larynx, trachea, bronchi, and even of the mucous membrane of the stomach, which may be covered with blood-stained mucus. Often there are no discoverable changes.

During the year 1900, important discoveries in the minute morbid anatomy of rabies were announced by Van Gehuchten and Nélis. The changes were found in the peripheral ganglia of the cerebrospinal and sympathetic systems, and are especially marked in the plexiform ganglion of the pneumogastric nerve and Gasserian ganglion. In the normal state these ganglia are composed of a framework of tissue in the meshes of which lie the nerve cells, each one inclosed in a capsule made up of a single layer of endothelial cells. The rabic virus stimulates these cells to proliferation leading to the ultimate destruction of the normal ganglion cell and replacing it by a collection of round cells. The ganglion cells are sometimes only slightly altered, at others destroyed, the extent of the process varying in different animals, being most pronounced in the dog and less so in man and rabbit.

These changes are claimed to be especially valuable in diagnosis, since the examination can be completed within six hours after the death of the animal. It is important, however, that the animal should be allowed to die and not be killed prematurely. The ganglion selected for examination is by preference that of the pneumogastric nerve. The laboratory of the State Live Stock Sanitary Board of Pennsylvania was the first in this country to take up this method, under the direction of Mazyck P. Ravenel, bacteriologist to the board. Fifty-two cases were examined between May, 1900, and July, 1901, without a single failure. In 1903 Negri described certain bodies within the plasma of the nerve cells which he claims are protozoa. Whether the latter fact is correct or not, it is now agreed by observers that when they are present with the changes described by Van Gehuchten and Nélis biological diagnosis becomes entirely unnecessary.

Symptoms.—Rabies is usually divided, corresponding to the promi-

nence of symptoms, into two varieties—*furious* or *convulsive* and "*dumb*" or *paralytic* rabies. Professor W. H. Welch, of Johns Hopkins University, suggests a third form of *mixed* rabies, representing a combination of convulsive and dumb rabies. The variety common to human beings is the furious or convulsive, though paralytic rabies also occurs in man, especially after bites on the lower extremities, and would seem to be increasing as compared with the convulsive form. So, too, in dogs furious rabies is the more usual, while in rabbits the paralytic form is more common.

It is true, also, that a sharp distinction cannot always be made between the two forms, while a stage of excitation and a stage of paralysis may be made out in the same case, and it amounts largely to this: that in the furious form, the stage of paralysis may be short or wanting, while in the paralytic form the stage of excitement may be short and may be manifested only by acceleration of breathing, elevation of temperature, and symptoms referable to irritation of the vagus nerve. The most reliable observations go to show that there is no difference in the quality of the virus producing the two forms, but that the differences are due rather to peculiarities in the individual, the seat of inoculation, or perhaps the quantity of the virus.

The *first* or *premonitory stage* succeeds upon the period of incubation and lasts about 24 hours. The *cicatrix* of the bite, which has been for some time healed, may become painful or the seat of radiating pain, or become livid, or even break out again. The patient is *morbidly depressed* or irritable, is *feverish, loses appetite*, and is *sleepless;* there is *hoarseness* or huskiness of voice. A feeling of intense anxiety and a moodiness are very characteristic, his probable fate being the sole subject of contemplation. There is an increased excitability, as a result of which the banging of a door or a flash of light causes the patient to start. Fever is not marked.

The *second* or *spasmodic* stage is the true hydrophobic stage, setting in usually after the first 24 hours. It is also called the furious stage. The sum of its symptomatology depends upon an *exalted irritability* of the *muscles of the larynx*, as the result of which they contract upon the slightest irritation in their vicinity, the act of swallowing being the most frequent exciting cause. Attempt at swallowing is followed by the most *powerful contraction* associated with *dyspnea*, even when the glottis is open or tracheotomy has been performed; whence the *fear of water*, the contact of which with the throat is followed by such frightful spasm of the muscles of the larynx and elevators of the hyoid bone. Even the saliva, which is secreted in increased quantity, cannot be swallowed without exciting paroxysms. Hence it is discharged from the mouth, sometimes forcibly, giving rise to the popular idea that the patient is frothing at the mouth. A breath of air or the slamming of a door may produce a paroxysm.

The paroxysm may be associated with *maniacal excitement* in which the patient is sometimes uncontrollable, rolling his eyes, striking about with his arms, and making snapping noises with the mouth, which are compared to the biting of dogs. These noises are altogether due to uncontrollable spasmodic shutting of the mouth. On the other hand, between the paroxysms, when the mind is clear and the reason sound, there is often found a touching concern on the part of the patient lest he does some harm to those whom he loves. There is more decided *feverishness* in this stage,

the temperature rising as high as 103° F. (39.4° C.), while the *pulse* is frequent and sometimes irregular. Albuminuria and glycosuria have both been found in this stage. The second stage lasts from *one* to *three* days, sometimes a little longer.

In the *third* or *paralytic* stage the patient has become exhausted. There are no more paroxysms and he is quiet. His heart gradually fails, and he dies by syncope, although he may die in a convulsion or in asphyxia. This stage usually lasts from *six* to 18 *hours*. Happily, the disease is one of short duration, ranging from two to six days, notwithstanding its long period of incubation.

Diagnosis.—Hydrophobia most resembles *tetanus*. Yet the diseases are very different. Hydrophobia has a long period of incubation, while tetanus has a short one from three to ten days. Tetanus begins with trismus and is associated with opisthotonos. Neither of these symptoms is present in hydrophobia. Tetanus has no laryngeal symptoms, no spasms in swallowing. The mental depression so characteristic of hydrophobia is wanting in tetanus.

More difficult is it to distinguish hydrophobia from the imaginary condition known as *pseudophobia* or *lyssophobia*, numerous cases of which have been reported, and the occurrence of which doubtless furnished the foundation for the relief by some that there is no such disease as hydrophobia, and that all cases are lyssophobia. The resemblance is often very close, especially the depression and mania, and it is even said that strong men have been so overcome by this fear that they die as a consequence. The condition, however, generally passes away. Especially is this the case when it transpires that the biting dog was not rabid. Hence, the usual practice of immediately killing the dog supposed to be rabid is not a wise one, since it makes it impossible to settle the question conclusively as to its madness. It is better to confine the animal until the possibility of recovery is settled. If the dog be killed careful microscopic examination of the ganglion of the brain and medulla should be made by an expert and inoculations from the medulla should be made under the dura mater of rabbits and results awaited. If true rabies, the paralytic form of the disease will be developed in from 15 to 20 days. A much more rapid method of diagnosis is that recently announced by Van Gehuchten and Nélis, for which see Morbid Anatomy.

Prognosis.—The diagnosis once established of hydrophobia, the prognosis is, unfortunately, totally unfavorable. The possibility of spontaneous recovery cannot be denied, but it is certainly exceptional. The preventive treatment is eminently successful. The claims of Pasteur will be considered under treatment. Bollinger's statistics go to show that out of 134 cases in which the bite was cauterized, 92, or 69 per cent., were attacked, while 42, or 31 per cent., died of the disease; of 66 not cauterized 83 per cent. died of the disease.

Prophylaxis.—Statistics show that hydrophobia can practically be banished as a human disease by the muzzling of dogs in countries where the dog is the chief animal which disseminates the disease. Care in regard to the bites received from unknown dogs will also help to lower the incidence of the disease.

Treatment.—The preventive treatment consists first in prompt measures to eliminate the poison. *Suction* is the promptest measure available and should be practised, if possible, by the victim himself, as it is not without danger to a second person. An abrasion in the mouth or a carious tooth may be the medium of inoculating such person with the dreaded virus. If suction be practised, the mouth should be promptly rinsed. It is doubtful if the cupping-glass is as efficient, even if at hand.

Next in availability is *cauterization*, which should be practised by a *glowing hot iron* or other instrument of the kind, a galvanocautery or *Paquelin's cautery*, failing this pure carbolic acid or pure nitric acid should be applied to every part of the wound—or the wound being thoroughly opened should be washed with 1–1000 solution of bichloride of mercury. When the symptoms once set in, palliation alone is possible. A case is reported of true hydrophobia which was treated by Tonin in 1912 by the use of salvarsan. This observer is a physician in charge of the Pasteur Hospital at Cairo. As no case has heretofore been reported which gives any good evidence of cure after the symptoms have developed, this method should be tried.

Pasteur's Treatment by Attenuated Virus.—This is of the nature of preventive treatment and should be used in every suspected case. It is of no value once the symptoms of the disease are present. Pasteur discovered that the virus of hydrophobia is located in the nervous system, especially in the brain, medulla, and spinal cord. He then ascertained that inoculations by virus from this source in rabbits produced a virus of such increased virulence that after 25 successive inoculations there resulted a virus that acted after a period of incubation of eight days; and after 25 additional inoculations in seven days. The virus from the medulla of rabbits, with this short period of incubation, is called "fixed" virus as contrasted with the "street" virus. Now, although the spinal cords of such animals contain the virus in a state of great intensity, Pasteur ascertained that its intensity could be greatly reduced by preserving the cords in dry air, and that it disappeared altogether in two weeks.

Careful examination of the results of this treatment by the most exact and conscientious observers, such as Victor Horsley, of London, and William H. Welch, of Johns Hopkins University, as well as the records of the numerous Pasteur institutes throughout the world, goes to show that the treatment is a powerful agent in saving life. As soon as a person is bitten by a rabid dog or one supposed to be rabid, the person should at once be subjected to the Pasteur treatment. This can be done either in one of the Pasteur institutions, which now cover practically the entire globe, or by purchasing the virus from one of the various Health Boards. In the eastern part of the United States and probably in other portions of the country, the New York Board of Health will under proper regulations send the virus to responsible individuals with proper directions for its use. Various manufacturing firms now dispense the virus in forms that can be used at home. The use of the Thermos bottle makes it practicable to ship the virus to long distances and have it perfectly efficacious.

If the dog is not certainly known to be rabid, it should be confined until it shows some symptoms of disease or is known to be healthy. If there is

an opportunity for examination, the dog should be killed and the head packed in ice and sent to a laboratory for examination.

The *paroxysms* should be controlled by inhalations of chloroform, and averted as far as possible by full doses of opium, preferably, as a rule, morphin hypodermically. Chloral may at first suffice. As light and noise excite paroxysms, the patient should be kept quiet and secluded, and even in a dark room with two attendants. Water and nourishment may be given by enema.

Beyond these measures the treatment of the disease is the treatment of the symptoms.

TETANUS.

SYNONYM.—*Lock-jaw.*

Definition.—Tetanus is an infectious disease characterized by paroxysms of tonic spasm, repeating themselves with increasing severity. It is a disease of human beings and lower animals.

Etiology.—The specific cause of tetanus is a bacillus, which was isolated by Nicolaier in 1884 and obtained in pure culture by Kitasato in 1889. It is a slender rod with rounded ends, develops at ordinary temperatures, and is found in the soil, in the alimentary canal of animals, in manure, in pus and putrefying fluids of wounds; sometimes forming threads, sometimes irregular masses. It is slightly motile, anaerobic, refusing utterly to grow in the presence of oxygen; develops spores within itself, though when studied early in pus is often sporeless. During sporulation one end becomes rounded, giving the bacillus a drum-stick like appearance.

It is one of the most invulnerable of bacilli, its spores resisting a temperature of 176° F. (80° C.), while the bacilli retain their vitality in the dried condition for months. According to G. M. Sternberg, they resist a five per cent. carbolic solution for ten hours, but will not grow after 15 hours' immersion. If five per cent. hydrochloric acid be added, they are destroyed in two hours. They are destroyed in three hours by a 1 to 1000 bichlorid solution, but when five per cent. hydrochloric acid is added the *spores* are destroyed in 30 minutes. Exposure to passing steam for from five to eight minutes kills the spores. The toxin, on the other hand, is rapidly destroyed by heat and light, being unable to resist a temperature above 140° to 149° F. (60° to 65° C.). In the dark in a refrigerator it can be kept indefinitely. Cultures of the tetanus bacillus in all media give off a peculiar characteristic odor—a burnt-onion smell with a suggestion of putrefaction.

The bacilli do not, however, pass into the blood, as a rule, but at the site of the wound manufacture with great rapidity a ptomain or toxin, which is absorbed by the ends of the motor nerve trunks, carried along them to the motor center where it excites the disease. This was first shown in 1890 by Kitasato, who found that the bacteria-free filtrates of bouillon cultures of the tetanus bacillus produce the same symptoms as inoculation with cultures containing the bacillus, including ultimate death. Indeed, Brieger, in 1886, isolated from impure cultures three ptomains, which he called *tetanin*, *tetanotoxin*, and *spasmatoxin*. The first of these causes the char-

acteristic symptoms of tetanus; the second, tremors, convulsions, and subsequently paralysis; and the third, intense tonic and clonic spasms. More recently, Kitasato and Weyl obtained Brieger's tetanin and tetanotoxin from pure cultures; while Brieger himself, with Fränkel and Kitasato, has succeeded in isolating from tetanus cultures a far more deadly ptomain, *toxalbumin*, which was purified by Brieger and Cohn, who have shown that it is not a pure albuminous body. Brieger has also isolated such poisons from the organs of those dead of tetanus, and Nissen has demonstrated toxin in the blood of those ill of tetanus.

Further, it has been shown by Behring and Kitasato that there exists in the blood of animals immune to tetanus a substance with opposite properties, therefore called antitoxin, and by the gradual introduction of the toxin into animals these observers have been able to produce in their blood a potent antitoxic substance. Such serum is prepared by Behring and by Roux abroad, and by manufacturing chemists in America. The methods for its production is similar to that for diphtheria antitoxin, but slower. Tizzoni and Cantani have successfully prepared it in a solid form, in which, it is claimed, it can be kept indefinitely and shipped as wanted, and applied to treatment of cases of traumatic tetanus with success.

Predisposing Causes.—The excitation of tetanus is favored by certain conditions. *Wounds*, particularly contused and punctured wounds, especially of the hands and feet, are favorite foci, whence the term *traumatic* for such cases of tetanus. A similar focus is the badly cared-for umbilical cord whence *tetanus neonatorum*, affecting especially the colored race. In certain parts of the West Indies it is said that more than half the deaths among negro children are due to this cause. It is probably because the contused wound affords a more favorable nidus for the growth of the bacilli rather than that there is any peculiar laceration of nerves, as formerly thought. It is more common, too, in *hot countries* and in places and seasons where there are decided alternations of heat and cold. It affects both sexes and all ages, but it is more frequent in men for obvious reasons. Children are especially susceptible.[1]

Tetanus occasionally prevails in the epidemic form.

Morbid Anatomy.—There is no essential morbid anatomy of tetanus. There may be congestion, extravasations, and perivascular exudates due to impediment of the movement of the blood during spasm, granular changes in cells from modified nutrition—all results rather than causes of symptoms.

Symptoms.—A *period of incubation* of from ten to 15 days is required for the operation of the specific cause of tetanus. Occasionally only does a *chill* precede the other symptoms. There appears first usually a *stiffness in the neck* and *jaws* and the patient opens his mouth with difficulty, but not with pain. Then the stiffness extends to the back and abdominal muscles and to the legs, which may be fixed in extension, more usually during a paroxysm. The result is that the abdominal muscles feel like a board and the whole trunk is inflexible. If an attempt be made to flex the thighs on the abdomen the whole body comes up in single piece; if the body is turned over, it is like turning over a wooden man. There is, in a word, *orthotonos*. Again, as in a striking case of Tyson's, the symptoms

[1] Article "Tetanus," Keating's "Cyclopedia of Diseases of Children," vol. iv., p. 913, 1890.

may begin in the abdomen and by their intermittent character simulate cramp.[1] These symptoms are present in various degrees, less marked in the mild cases, more so in the severe ones. In severe cases the jaws become locked, in milder ones they may partly yield to forcible extension. The eyebrows may be raised and the angle of the mouth drawn up, producing the *risus sardonicus*, or tetanic grin.

In the so-called *head tetanus* described by E. Rose, there may be *paralysis* of the facial muscles and difficulty of swallowing, with violent *spasm* of the *pharynx* and *esophagus*. It is associated more particularly with injuries to the fifth nerve.

These symptoms are more or less constant in various degrees. All are further increased during the paroxysm, which is excited by various sensory impressions, sometimes exceedingly trifling, as a breath of air or the contact of a dress, a footfall, or the slamming of a door. The muscles of the trunk contract more strongly, and if the patient be on his back, the body may be so bowed that only the back of the head and heels touch the bed—opisthotonos; or the side of the face and leg, producing pleurosthotonos; or the abdominal muscles may bend the body forward—emprosthotonos. Spasmodic closure of the jaws sometimes causes the tongue to be bitten. The paroxysm may then relax, and during its relaxation the patient will be able to walk about. In severe cases the spasm may involve also the muscles surrounding cavities, as the thorax, compressing as in a vise their contents, causing extreme pain. Indeed, *pain* is almost everywhere an accompaniment of these spasmodic contractions, and the perspiration stands out in great drops on the face and covers the body. An attempt to speak is transformed into a fit of crying. The frequency of the spasms varies greatly; they may occur every couple of hours or minutes or almost incessantly.

The temperature is generally, but slightly, if at all, elevated, rising to 101° F. (38.3° C.) and more rarely to 102° F. (38.9° C.). At times, however, it rises higher, to 105° to 106° F. (40.5° to 41.1° C.), and it is said also in fatal cases to reach 108° to 110° F. (42.2° to 43.3° C.). The pulse is generally frequent, 130 to 150, *respirations* 30 to 45. There is often *constipation*, which is a more serious symptom in severe cases, because the efforts to relieve it are apt to bring on a spasm. Among the rare events have been the rupture of muscles and spasmodic closure of the glottis, producing fatal asphyxia. Generally, death is produced by exhaustion, the mind remaining unclouded throughout.

Diagnosis.—Tetanus is liable to be confounded with strychnin poisoning, cerebrospinal meningitis, and hydrophobia. *Strychnin poisoning* differs from tetanus in the absence of rigidity between the paroxysms and of trismus, and in the more marked involvement of the extremities, as well as in the history of the case. In *hydrophobia* there is no trismus, and while convulsive dysphagia occurs sometimes in tetanus, it is very rare. (See also hydrophobia.) As in strychnin poisoning, too, the individual paroxysms are more distinct.

Cerebrospinal meningitis produces a rigidity similar to that of tetanus, but the cerebral symptoms give it its stamp, and fever is a much earlier

[1] "Philadelphia Med. Times," vol. i., 1871, p. 418.

symptom than in tetanus. Leucocytosis is present in cerebro spinal meningitis and a spinal puncture will fix the diagnosis. The stiffness of the jaws in *parotitis* and severe *tonsillitis* is similar to that of tetanus, but there the resemblance ends.

The interesting and rare condition known as *tetany*, or intermittent tetanus, characterized by the paroxysmal tonic contraction in *groups of muscles*, more frequently in the extremities, is hardly likely to be confounded with tetanus.

Prognosis.—The prognosis of traumatic tetanus once established is exceedingly unfavorable, not less than 80 per cent. perishing.

In children the prognosis is more favorable than in adults, and some very severe cases get well. Most cases die within the first six days, and cases living to the sixth day are very much more apt to get well. The aphorism of Hippocrates, that "such persons as are seized with tetanus die within four days, or if they pass these they recover," is frequently substantiated. On the other hand, a late onset makes a case more hopeful. Localization of the spasm to the muscles of the face, neck, and jaw is favorable to recovery, and the so-called Rose's head tetanus most commonly gets well. The cases in which there is very little elevation of temperature are more apt to do well. Convalescence is likely to be protracted even in mild cases.

Treatment.—Prompt local treatment is important. The wound should be excised and cauterized by the hot iron or nitrate of silver, and antiseptic dressings should be applied. The patient ought then to be secluded and surrounded by the utmost quiet. After such seclusion Bacelli recommends the subcutaneous injection of ten drops of a ten per cent. solution of carbolic acid every two or three hours. The injection is made deep into the muscles. He claims that carbolic acid gives better results than the antitoxin by antagonizing the toxin and quieting the nervous system.

Recent studies, however, give the palm to the antitoxin treatment. To be of any value it must anticipate the symptoms of the disease, since if tetanus is fully established serum therapy, however administered, avails little. As a prophylactic it is to be confidently relied upon. To be of the greatest service it must be administered before the motor nerves have absorbed any toxin. It should, therefore, be administered as soon as possible after the infliction of the injury and to every person who has sustained an injury in which dirt, manure or foreign substance of any kind, such as powder or fragments of fire-arms, could have entered the wound. Ten c.c. of a reliable serum should be injected early into the muscles, repeated on the third and fifth days, and on the 15th to 20th day if suppuration continue. Of Tizzoni's solid antitoxin 2.25 grams should be the first dose, and 0.6 gram afterward at about the same interval. It is also recommended to use the dried serum locally in the wound, dusting it over before the dressing is applied. Thus used, the antitoxin acts locally, but when injected it travels along the blood stream in contrast to the route of toxin. The use of antitoxin in no way precludes the employment of spinal antispasmodic remedies, such as chloral, bromids, morphin, eserin, etc.

The further treatment of tetanus must be the treatment of the symptoms. *Morphin* is indispensable to control the pain and defer the paroxysms or diminish their severity, and anesthesia by ether or chloroform may be

required during the paroxysm. The milder sedatives, like chloral, may suffice in mild cases, but they are insufficient in severe ones. *Chloral* may be used as an adjuvant in not less than 15 grain (1 gm.) doses for adults when the quantity of morphin otherwise required would be excessive. Even larger doses of chloral than those named may be given in connection with the antitoxin treatment.

Subdural injection through a trephined opening is recommended by A. E. Barker,[1] who injected 7.5 c.c. of antitoxin at one time, and 20 c.c. subcutaneously daily for the following four days. In addition massive doses of chloral were given. A week later the rigidity commenced to diminish, and in the course of three weeks the patient had completely recovered. The efficiency of chloral is also increased when combined with double the dose of bromid of potassium. To a less degree phenacetin, antipyrin, and antifebrin may be useful. Salicylic acid in large doses has been thought to be of value. Later studies have shown that neither the subdural nor intracerebral method of injection of antitoxin has any peculiar advantages.

The intraspinal injection of magnesium sulphate was suggested by Meltzer, the thought arising from the fact that a long, deep, lasting anesthesia with complete relaxation of the voluntary muscles and abolition of some of the less important reflex activities terminating in recovery, succeeds upon such injection in the lower animals. The same effect was produced on the human being, 25 per cent. solutions, 1 c.c. to every 20 pounds of body weight. There were some untoward effects, the most serious being slowing of respiration, relieved by washing out the spinal canal with normal salt solution. Following this treatment some excellent results have been obtained in the treatment of tetanus. Up to this writing four cases of tetanus thus treated have been reported, of which two recovered, one at the Pennsylvania Hospital by Canby Robinson. Robinson pleads for the use of the remedy. He injected 1 c.c. of the 25 per cent. solution for each 23 pounds at first injection, 1 c.c. for 20 pounds at second injection, and finally 1 c.c. for 17.5 pounds. The injections were given daily or every two or three days whenever the symptoms returned. Later three more cases were treated at the Pennsylvania Hospital by the magnesium solution with fatal results, although the control of the paroxysms was striking.

Reasoning from its physiological action on the nerve-centers, *calabar bean* ought to be a useful remedy, and it is commonly used in doses of 1/4 to 1/2 grain (0.0165 to 0.033 gm.) three to five times a day. *Curare* should also be useful for its sedative effect on the terminal nerves, but experience has not confirmed expectation as yet; 1/25 grain (0.0026 gm.) may be given hypodermically and cautiously increased. The strength of curare varies greatly. *Warm baths* are serviceable in relaxing spasm and often very comforting to the patient.

The most nourishing food in liquid form is necessary, and usually, also, stimulants are freely administered in tetanus, with a view to sustaining the patient against the exhaustion that sooner or later causes death unless the disease is arrested.

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[1] "Philadelphia Med. Jour.," December 8, 1900.

ANTHRAX.

SYNONYMS.—*Malignant Pustule; Contagious Carbuncle; Splenic Fever; Splenic Apoplexy; Gangrene of the Spleen; Carbuncle Fever; Blood-striking Choking; Quinsy and Bloody Murrain; Wool-sorters' Disease; Rag-sorters' Disease.* In France it is known as "*Charbon*," and in Germany as "*Miltzbrand*."

Definition.—An acute infectious disease of animals, especially affecting cattle and sheep, but transmissible also to man; caused by the implantation and multiplication of the bacillus of anthrax.

Etiology.—The bacillus of anthrax, the largest of the pathogenic bacilli, is a minute cylinder 5 to 20 microns in length and one to 1.25 microns in breadth. It is found in enormous numbers in the blood and tissues of the animal infected with anthrax, where it multiplies rapidly by division and whence it may be obtained by cultures. In artificial cultures it grows in long threads, in the interior of which appear minute ovoid spores, which are loosed by disintegration of the bacilli, which have but a transient existence, while the spores are very tenacious of life. Their vitality may remain in abeyance for long periods of time, and revive with the return of favorable condition of heat and moisture. Introduced into the blood of animals they develop into bacilli. The medium of their transfer to others, including human beings, is the blood, secretions, flesh, and hair from those infected. An extremely common source of the infection in America is infected hides and hair. Malignant pustules not uncommonly appear on the exposed portions of the bodies of workmen. Here, as in glanders and hydrophobia, an abraded surface is necessary for successful inoculation, although the possibility of absorption through intact mucous membrane and skin is asserted. Those most frequently infected are herdsmen, stable-hands, butchers, and wool-sorters.

It is thought that anthrax bacilli may exist elsewhere than in animals, as in marshes and on the banks of streams, whence they may be carried by freshets into pastures and so infect the grazing animals. Commonly, however, the affection spreads from other animals having the disease. Pasteur has found the bacilli in the herbage over the buried bodies of animals dead of the disease. It is primarily a disease of herbivora, from which it is transmitted to carnivora and man.

Hoffa has isolated a toxin, which he calls *anthracin*.

Morbid Anatomy.—The body after death is cyanotic. The blood is dark and viscid, coagulating slowly; the spleen is enlarged and soft. On the skin are carbuncular and gangrenous patches, the subcutaneous tissue is infiltrated with bloody serum, the blood is uncoagulated, and all the tissues and organs are more or less infiltrated with blood. The gastrointestinal mucous membrane is edematous and ecchymotic, there are enlarged follicles and gangrenous patches infiltrated with bacilli, constituting the so-called *carbuncle of mucous membrane*. Even the nervous tissues are the seat of analogous lesions.

Symptoms.—Anthrax has a *period of incubation* of about seven days, after which there are several ways in which the disease shows itself, of which the chief are *external anthrax* and *internal anthrax*.

EXTERNAL ANTHRAX manifests itself as *malignant pustule* and *malignant anthrax edema*.

1. *Malignant pustule* starts most frequently on exposed surfaces of the skin—the arms, hands, or face—at the seat of inoculation. It begins as an *itching* and a *burning, smarting pain*, resembling often that from the bite of an insect. The spot becomes red and develops rapidly into a *papule*, in the center of which a *vesicle* soon appears, which is filled with clear, or at times bloody serum. The vesicle bursts, the papule enlarges and becomes indurated, surrounded by a number of small vesicles. The induration extends, while the center becomes dark and discolored. Within 36 hours a brown eschar makes its appearance and rapidly undergoes disintegration. The vicinity becomes edematous, the lymphatics inflamed, swollen, and painful.

To these local symptoms are added those of *general infection*, with its thirst, high temperature, and frequent pulse. The tongue becomes dry, the liver and spleen enlarged, the breathing rapid, and death supervenes in from three to five days, in fatal cases.

Frequently, recovery takes place, but it is only in mild cases, in which all the symptoms, local and general, are less severe, that the vesicles dry up into a crust or scab, and the induration dies away.

2. *Malignant anthrax edema* begins in the eyelids and passes thence to the head, hands, and arms. The skin reddens and becomes edematous, vesicles may arise, but there are no papules, although the edema may proceed to extensive gangrene. The local symptoms in this form follow rather than precede the constitutional disturbance, as is the case with the papular form, and the termination is even more invariably fatal than in the latter.

Anthrax presents an interesting contrast to hydrophobia in the absence of the anxious mental condition so characteristic of the latter.

INTERNAL ANTHRAX manifests itself as *mycosis intestinalis* or intestinal anthrax, and *pulmonary anthrax*. The latter is also called *wool-sorters'* disease.

1. *Intestinal anthrax*, or *mycosis intestinalis*, is often ushered in by chill followed by nausea, vomiting, bloody diarrhea, abdominal pain and tenderness. With these symptoms are found after death endmatous and ecchymotic gastrointestinal mucous membrane, enlarged follicles and gangrenous patches. In addition to these symptoms pustules may form on the skin. It arises from the ingestion of meat infected with anthrax.

2. *Wool-sorters' disease* is a form of internal anthrax acquired by inhaling the bacilli into the lungs by those engaged in sorting wool, especially that imported from Russia and South America. It begins with chill, fever, high temperature, pain, dyspnea, bronchitis and cough, together with the physical signs of lung involvement. There are rarely premonitory symptoms and often no external lesion. It is rapidly fatal, the patient often dying in 24 hours in collapse. Other cases are more protracted, and there may be vomiting, diarrhea, delirium, and unconsciousness, while the brain may be the chief seat of involvement, the capillaries being filled with bacilli. *Rag-pickers'* disease is a special etiological variety, invading the lungs and pleura, with general infection.

Diagnosis.—The diagnosis of external anthrax is usually easy from the symptoms, in connection with the history of exposure to the cause. The

fluid of the pustule may be examined for the bacilli, which are large and easily recognized. Cultures may be made and a mouse or guinea-pig inoculated.

Internal anthrax is more difficult to recognize and may escape it altogether unless a knowledge of the occupation of the patient suggests it.

Prognosis.—The prognosis is unfavorable; yet not all cases perish. The anthrax carbuncles common in American workmen frequently recover. They vary from a mere irritated patch of skin without general symptoms to severe large gangrenous carbuncles with general toxemia. The intestinal form and wool-sorters' disease are especially fatal, though it is said also that those who survive the latter one week recover.

Treatment.—Prophylaxis is exceedingly important. Animals dead of the disease should be cremated—burying is not safe a plan; their hides should not be used; infected pastures should be shut off; disinfectants should be freely used in the wake of the disease. Hides, wool, and rags should be disinfected by superheated steam. In the case of wool and rags this is quite possible, but the necessary temperature is so high that hides are damaged by it, hence the latter should be destroyed.

The treatment of the local lesion varies with various practitioners and cases encountered. Wolf and Weinvowoski report thirteen cases with no mortality. Conservative treatment was applied in ten cases. *Absolute* rest of the part with boric acid poultice. The excision practised in one case was followed by severe fever and general symptoms. Other cases demand operation. *Deep crucial* incisions should be made, and to these the actual cautery, caustic potash or strong carbolic acid should be applied and the wound dressed with a strong solution of carbolic acid, 1 to 20 or the entire diseased part may be excised under ether and the excavation treated with carbolic acid.

With the local treatment should be associated *stimulating and restorative measures*, including alcohol, highly nutritious food, quinin, and strychnin. Sclavo's serum has been used. Lazaretti treated 23 cases with but one death.

Bacillus pyocaneus toxin has been used also with apparently brilliant results.

GLANDERS AND FARCY.

Synonyms.—*Farcy; Malleus humidus; Wurm* (German).

Definition.—Glanders is an infectious disease more especially of the horse, communicable to man and certain domesticated animals but not to cows; characterized by nodular growths in the nares, when it is known as glanders, and under the skin, when it is called farcy. Among animals to which it is communicable are the lion, sheep, rabbit, guinea-pig, cat, dog, and mouse.

Etiology.—Glanders and farcy are the direct results of a bacillus—the *bacillus mallei*—described by Leoffler and Schütz in 1882. It is a short nonmotile bacillus not unlike that of tubercle and leprosy, but shorter than either. It is commonly seen among the cells of the growth, but has also been found in the blood. The disease is communicated by the discharge

from the infected animals to an abraded skin surface or intact mucous membrane. The human victims are usually hostlers or others working among horses.

Morbid Anatomy.—The infection presents itself in the shape of nodules ranging in size from that of a lentil to that of a fist, or in many infiltrate more diffusely. It is composed of round cells which invade the skin, mucous membrane, and muscles. Internal organs—as the lungs, liver, spleen, kidneys and even the stomach, the nervous system, bone, and cartilage—may be invaded. The ulcers on the skin are often serpiginous, whence the name *Wurm* among Germans. A few of the cells develop into epithelioid cells, but all soon break down, leaving ulcers on mucous membrane and skin, and abscesses under the latter.

Symptoms.—Glanders and farcy have a *period of incubation* of from three to five days, rarely a week. There is an *acute* and *chronic* form. The acute terminates within three weeks, while the chronic may last for months and even years.

In *acute glanders* of the nasal mucous membrane there is, first, *redness* and *swelling* at the point of inoculation with *burning* and *dryness* of the adjacent mucous membrane. *Intense pain* in the forehead from involvement of the frontal sinuses may also be present. This is promptly followed by nodule-formation and the rapid breaking down of the nodules and discharge of fetid hemorrhagic or muco-pus. The destructive process extends to the nasal septum, the mouth and pharynx, and even the larynx, lung, and other organs. The submaxillary glands swell and suppurate. From these lesions result the usual symptoms of *painful deglutition*, *cough*, and *hoarseness*, with *fetid expectoration*.

Chronic glanders is less easy of recognition. The symptoms are more like those of incurable coryza and sometimes of chronic laryngitis. It may be necessary to make cultures and inoculate an animal, preferably the guinea-pig, which perishes in 30 days and presents already testicles swollen and suppurating.

In *acute farcy*, after the *period of incubation*, a *feverish state* develops. At the point of infection on the skin there appears a nodular *swelling*, or an ulcer which tends to spread and discharge a fetid hemorrhagic pus. The adjacent tissue becomes red and edematous and the lymph-vessels and lymphatic glands are inflamed. Papules that become pustules may also develop in the neighborhood. Such an eruption has been mistaken for that of smallpox, but is soon replaced by open ulcers. The so-called *farcy buds* are nodular, subcutaneous enlargements along the course of the lymphatics, and may suppurate. The nose is not involved.

In *chronic farcy* the localized tumors form under the skin, especially of the extremities, and break down, but the process is more slow, and there is no special involvement of the lymphatic glands.

Further symptoms in both forms are: *chilliness*, *fever* with high temperature, *intense prostration* and depression, muscular and joint *pain* and soreness, *abscess* formation, and finally *typhoid symptoms* and death.

The *spleen* and *liver* may be enlarged, *albuminuria* may be present, and it is said even *leucin* and *tyrosin* are found in the urine.

Diagnosis.—The diagnosis in the acute form is easy. It has, however,

been confounded with pyemia and smallpox. Chronic glanders is to be distinguished from syphilis and tuberculosis. The history of exposure is helpful. In doubtful cases cultures should be made. Especially characteristic is that on the cooked potato, which by the thrd day furnishes an amber-hued film, that on the sixth to eighth day is red and turbid, surrounded with a pale-green area. Inoculation with "mallein," a product of the bacillus of glanders, comparable to the tuberculin of tuberculosis, should be made. It causes a rise of temperature in affected cases as do tuberculosis cases with tuberculin. A reaction of 3.50° F. (2° C.) in horses is regarded as positive proof of the presence of the disease; a rise of 1.85° F. (1.50° C.) is strong presumptive proof, and 1.25° F. (1° C.) suspicious. The absence of the Waserman reaction will distinguish an uncomplicated case from syphilis.

Prognosis.—The prognosis in the acute variety is invariably fatal; in the chronic form 50 per cent. recover.

Treatment.—In the cutaneous form excision and cauterization should be practised as early as possible, followed by antiseptic dressings. In the nasal variety sprays of carbolic acid, bichlorid of mercury and peroxid of hydrogen should be introduced into the nose and throat. "Mallein" has also been used internally as a remedy, but its value is not as yet determined.

ACTINOMYCOSIS.

SYNONYMS.—*Big Jaw; Swelled Head; Bone Tumor.*

Definition.—An infectious inflammatory disease of cattle, communicable also to man, and depending for its existence on a peculiar fungus named by Hartz, a Munich botanist, *actinomyces* or ray-fungus.

Etiology.—The fungus belongs to the species Streptothrix Actinomyces, and is known as the ray-fungus. As found in the pus from man and cattle affected with the disease, it appears as a small, yellowish granule from one to two millimeters (1/25 to 1/12 inch) in diameter, detectable by the naked eye. By the microscope the granule is resolvable into conical threads, radiating from a center to which they are attached by their small ends, the other club-like ends being outward. This gives the external surface a mulberry appearance. The center is composed of a granular substance, containing numerous bodies resembling micrococci. The disease has been reproduced by inoculation of the fungus from a diseased animal, as well as by the inoculation of cultures. It is thought to arise primarily in animals in the course of their feeding on vegetable matter. This is the more reasonable, because the ray-fungus has been isolated from vegetables. A similar origin is ascribed to it in man.

The effect of the parasite is to produce granulomatous and fibromatous new formations, which ultimately become the seat of suppuration. The former, like tubercle, is composed of small round cells, epithelioid cells, and giant cells. The fibrous matter consists of proliferated connective tissue about the granulation growth, expanding and enlarging the bones until it resembles an osteosarcoma, for which it was for a time mistaken.

The tendency to suppuration is more marked in man than in cattle, where the process too is more localized. In man the disease runs its course

with the formation of multiple abscesses and chronic pyemia. Such course is supposed to be due to an admixture of pyogenic organisms with the true ray-fungus. Associated with the suppurative process in man is a tendency to fatty degeneration of the cells of the granulation tissue.

Morbid Anatomy.—In addition to the lesions presently to be described about the jaw and head, there are found in the lungs, when the latter are invaded, the miliary nodules alluded to, made up of groups of fungi, surrounded by granulation tissue. Bronchopneumonic areas and abscesses large enough to be recognized by their physical signs during life may also be present. Erosion of the vertebræ, ribs, and sternum may also occur.

Symptoms.—The route of infection is generally the mouth, while the special seats seized upon are carious teeth, whence the *jaw* is invaded and becomes swollen. The swelling may extend thence to the face and temporal region, and even to the neck, producing *discharging sinuses* like those associated with dead bone. Alongside of these are cicatricial marks of healing. More rarely the tongue, fauces, and even the intestines (large and small), and the liver are invaded. The latter organ may also become involved metastatically. The fungus has been found in the stools first by Ransom, and pericecal abscess has been found due to it.

The *lungs* are also favorite seats of invasion by actinomycosis, and it was in these organs in man that Israel recognized the fungus which proved to be the ray-fungus also. The *symptoms* produced are *those of tuberculosis* —fever, cough, and more or less fetid expectoration, in which the fungus is occasionally found. In the lungs the posterior and lateral parts are affected rather than the apices. They may be invaded simultaneously with the jaws. The course of lung actinomycosis is chronic, and resembles that of pulmonary consumption, the average duration in man being ten months. The physical signs are those of consolidation, often of a pleural effusion.

Actinomycosis may occur in connection in the *skin* alone, and even in the *brain*, abscesses may occur containing the mycelium. Bollinger has reported a case of the primary disease in the brain of man, while Gamgee and Delpine and O. B. Keller have found it in the brain secondary to pleural invasion. The metastatic abscesses are the direct result of the transfer of a portion of the fungus.

Diagnosis.—*Sarcoma* of the jaw presents a macroscopic picture very like that of actinomycosis, but its course is more rapid and there is less suppuration, yet these signs are of themselves insufficient, and there recognition of the fungus is necessary to a diagnosis.

More frequent, perhaps, than any other error is that which mistakes the disease for *pyemia*, of which, indeed, as it occurs in man, it is a chronic variety. There are the same sort of metastases in the lungs and elsewhere; in man with pus formation, in animals with or without slight suppuration.

Treatment.—The treatment is surgical, consisting in thorough extirpation, the opening of the abscesses and removal of the dead bone, followed by thorough drainage. Iodid of potassium in doses of 40 to 60 grains (3.66 to 4 gm.) was recommended by Thomassen in 1885, and cures are reported from its use. DaCosta also reports success with this drug.[1] In Boston Med. and Surg. Jour., July 18, 1912, Kinnicutt and Mixter report eight

[1] "Proceedings of the Association of American Physicians," 1900.

cases treated by either autogenous or stock vaccines, six of these cases recovered. Certainly the procedure should be tried.

FOOT-AND-MOUTH DISEASE.

SYNONYM.—*Aphthæ epizooticæ.*

Definition.—An acute infectious disease of lower animals, communicable to man. It affects especially cattle, sheep, swine, more rarely the goat and horse, and still more rarely of fowls, dogs, and cats. The disease in cattle spreads rapidly and entails often serious loss. It is characterized by fever and the presence of vesicles and ulcers in the mucous membrane of the mouth in the furrows and clefts about the feet, and on the teats of animals. It is communicable to man especially during epidemics.

Etiology.—The microbe responsible for foot-and-mouth disease has not been settled upon, though a streptococcus has been isolated from the fluid of the vesicle by Klein, and a micrococcus from milk by Cnyrim and Libberitz; the specific power of neither has as yet been determined. The contagion bearer is especially the contents of the vesicle alluded to, but milk, blood, urine and feces are also media. It is communicated to man through the ingestion of unboiled milk, butter, and cheese, or through contact with the fluid of the vesicles on the teats by milkers. It is said to be communicable even by the saliva from the affected animal.

A certain relation is believed to exist between the aphthous sore mouth of children and the foot-and-mouth disease, chiefly because it has been observed that aphthæ are apt to prevail in children at the same time with the foot-and-mouth disease in cattle.

Morbid Anatomy.—As recovery invariably takes place, no lesions other than those to be noted under symptoms have as yet been observed.

Symptoms.—The disease has a *period of incubation* of from three to five days. At this time there is a *febrile movement* with *malaise* and *loss of appetite*. On the mucous membrane of the lips and tongue, and sometimes on the hard plate and pharynx, come *vesicles* containing a yellowish serum. There is a sensation of *heat* and *burning* throughout the mouth, and the *swelling* may be so great as to make speech difficult and swallowing painful. There is *copious salivation*. Almost simultaneously appear vesicles between the fingers and toes and around the nails. Vesicles have also been noted on the nipples of women. Indeed, they have been found scattered all over the body, so that the case resembles smallpox. The hands, especially may be extensively involved. Gastrointestinal symptoms are sometimes present.

Diagnosis.—This must depend largely upon the history of exposure of the patient to the infective disease in animals. There is no specific way of making a diagnosis from catarrhal or severe aphthous stomatitis.

Prognosis.—The prognosis is favorable in man, recovery being the rule. Very young children may perish. The suckling young of animals perish in large numbers, because of the infected milk on which they subsist.

Treatment.—The disease can be easily avoided by simple prophylactic measures by those in contact with animals, of which the use of boiled milk

is the most important. Cleanliness of man and beast conduce to the same end.

Curative measures of a simple kind generally suffice. Mouth-washes of a saturated solution of chlorate of potassium should be frequently used. Powdered borax and alum may be directly applied. The separate ulcers or vesicles should be touched with the solid silver nitrate. The skin lesions should be washed in corrosive sublimate solution and dressed in sublimate cotton or salicylated cotton. The fever should be combated with suitable antifebrile measures.

MILK-SICKNESS.

SYNONYMS.—*Trembles; Puking Fever; Slows.*

Definition.—An infectious disease prevailing in the western and southwestern parts of the United States, characterized especially by trembling, vomiting, constipation, and a peculiar fetor of the breath.

Etiology.—A like disease called "trembles" prevails among the cattle of the infested districts, and it is supposed to be communicated to man through the milk and its products—viz., cheese and butter, and also flesh when used as food. It is more common in summer and autumn and in dry seasons. Nothing more definite is known as to its cause. Recently E. L. Mosely[1] has called attention to Eupatorium ageroteides or white snakeroot as a cause of milk-sickness, communicated to animals while grazing; this he attempts to prove by experiments on animals, apparently refuted by Albert C. Crawford, of the U. S. Agricultural Dept.

Morbid Anatomy.—Our knowledge of the morbid anatomy of milk-fever is chiefly by inference from that obtained by necropsies on cattle, those on man being few and imperfect. The lesions noted by Graff under these circumstances are as follows: Cerebral sinuses, meningeal vessels of the brain and cord distended with blood; pia mater opaque and overlaid with purulent exudate; brain soft; stomach and intestines contracted and mucous membrane injected; lungs, liver, kidneys, and spleen engorged with blood, the liver and spleen soft, the latter enlarged in some cases to twice the normal size, the blood fluid.

Symptoms.—There is usually a *prodrome* of two or three days, manifested by simple *uneasiness* and *discomfort*, after which the disease is usually ushered in suddenly by severe epigastric *pain*, *constipation*, *nausea*, and *vomiting*. Hence the term "puking" sickness. There is also moderate *fever* and disproportionate *thirst*. The *pulse* at first is full; later, small and rapid. There is marked *tremor* or muscular twitching on attempt at motion. The constipation is characteristic. The tongue is swollen and the breath is peculiarly foul. This is said to be diagnostic. A typhoid state may supervene, preceded by restlessness, irritability, coma, and even convulsions.

Prognosis.—The duration of the disease is from two to ten days or longer. The short cases are the fatal ones. When recovery takes place, convalescence may be protracted three to four weeks.

[1] Mosely, The Cause of Trembles in Cattle, Sheep and Horses and of Milk-sickness in People. Ohio Naturalist, vol. vi., p. 463 and 477. 1906.
Crawford, The Supposed Relationship of White Snake-root to Milk-sickness or Trembles. Government Printing Office, 1908.

Treatment.—The treatment is symptomatic, and consists chiefly in combating by alcohol, aromatic spirits of ammonia, and food the tendency to weakness. Happily, the disease appears to be dying out as land is improved.

Prophylaxis may be secured by fencing off cattle affected and carefully guarding against the use of infected food and milk.

SYPHILIS.

SYNONYMS.—*Lues venerea; The Pox.*

Definition.—Syphilis is a specific constitutional disease acquired by contact with the lesions of the disease or their excretions, and by heredity. Under the former condition it is known as *acquired syphilis;* under the latter, as *hereditary syphilis*. It is apparently confined to the human race and to monkeys.

Etiology.—The infecting organism is the Treponema pallidum of Schaudinn first described in 1905. This is a delicate spiral, 4 to 10 microms long, averaging 7 microns (about that of the red corpuscle of man) and in width may be of unmeasurable thinness to 1/2 micron.[1] It is found with striking constancy in the primary and secondary local lesions of acquired syphilis, whether the lesions are on the surface or interior of the body. It was found in the pharyngeal secretions of a congenitally syphilitic child and in the conjunctival secretions, but rarely in the general blood stream, explaining the difficulty in producing syphilis by inoculating the blood of syphilitics. The evidence in favor of the important rôle assigned to this organism is derived from its presence in syphilitic lesions and from experiments on the anthropoid apes which are susceptible to syphilis which is communicable from one ape to another.

Among the most important papers upon this subject those by Flexner, and Uhle and Mackinney[2] appear to confirm fully previous observations which go to show that the Treponema pallidum is the cause of syphilis.

Syphilis is one of the most highly contagious diseases. In the first place, the blood of the syphilitic is inoculable though with difficulty and capable of producing the disease. Further, the secretions of all primary and secondary lesions of the skin and mucous membranes are similarly potent. The products of the third or gummatous stage are not so regarded, although opinions are not unanimous on this point. A raw or abraded surface is a necessary condition of inoculation. The physiological secretions, such as the tears, milk, nasal and bronchial mucus, do not communicate the disease when inoculated, although they may become virulent by contamination with the poisonous secretions. Exceptions to this law are the spermatozoid of man and the ovule of woman.

The acquired disease has three stages—a *primary*, *secondary*, and *tertiary*. The *primary* is characterized by a primary sore associated with glandular

[1] See papers by Schaudinn and Hoffmann, "Deutsche med. Wochenschrift," 1905. Numerous other papers have been written since the original of Schaudinn and Hoffmann, by Neisser, Metschnikoff and Roux, Rekzet, Neoggerath and Straehlen, Simon Flexner and others. For a good bibliography see Flexner's paper, "Medical News," 1905.

[2] The Demonstration of Spirocheta Pallida in Lesions of Acquired Syphilis. "Journal of the American Medical Association." February 16, 1907.

enlargement in the neighborhood of the seat of inoculation. The *secondary* stage furnishes general lymph node enlargement, lesions of the skin and mucous membranes, etc., among which sore throat is especially conspicuous. The *tertiary* is characterized by the affections of deep-seated structures, the osseous and nervous systems, the liver, spleen, kidney, and testicle; also the subcutaneous and submucous tissues.

The initial sore makes its appearance within six weeks after exposure, usually in two or three weeks. The phenomena of the second stage usually show themselves about six weeks after the appearance of the initial lesion. Rarely this period may be somewhat longer. The third stage is more difficult to define by temporal limits. It is by years rather than months, and is characterized, as stated, by the involvement of the deeper-seated organs.

In the vast majority of cases, acquired syphilis comes from sexual intercourse, but it may be the result of any contact such as kissing. Drinking-cups, utensils, and other articles used by the infected in common with others sometimes convey the infection. Physicians are not infrequently infected in midwifery practice, the initial lesion making its appearance around the nail or in the web between the first and second fingers. Wet-nurses acquire the disease from syphilitic nurslings, the chancre occurring in a fissure or abrasion of the nipple.

Hereditary syphilis may be transmitted through the father or mother. In the former instance it is called *sperm inheritance;* in the latter, *germ inheritance.* Syphilis may be communicated by the father while the subject of the active disease or after all signs of it have disappeared. On the other hand, a syphilitic father may beget healthy children.

Syphilis and Marriage.—The question has sometimes to be decided by a physician as to whether a syphilitic, apparently recovered, may marry with safety to offspring. It will be seen from the above that an absolute answer dare not yet be given; but this much may be said, that the longer the interval since the primary attack the less likely is the offspring to be tainted, and it is generally acknowledged that systematic and continuous treatment may eliminate the disease altogether. Since the discovery of the complement reaction and its application to syphilis by Wassermann the question may be more definitely answered. An individual whose blood gives a Wassermann reaction should not marry. If the Wassermann is negative a second test should be made. If both tests are negative the patient may safely enter into marriage provided an interval of not less than three years be insisted upon between the disappearance of the last symptom and the patient's marriage. It is to be remembered also that each successive child of syphilitic parents shows less signs of the disease, until finally healthy offspring results.

A syphilitic mother may, of course, bear syphilitic children from germ infection, producing thus true hereditary syphilis; but a child may also be infected at the moment of its birth, when the syphilis is congenital but not inherited. On the other hand, a woman may bear a syphilitic child, and, though herself without signs of the disease, will not, according to Colles' law, be infected by her child should she suckle it while it has syphilitic ulcers of the lips and tongue. Such women, however, show the Wassermann

reaction. Yet a healthy nurse who suckles this same child or merely handles and dresses it may be infected. Such a mother is supposed to have received protective inoculation without evident signs of the disease; and we may have here an example of protection through a natural immunity of a nonsyphilitic mother.

According to Profeta's law in contrast with Colles', a child born of a syphilitic mother may suckle the mother without being infected even if there be lesions on the nipple, immunity having been acquired, but such children show the Wassermann reaction.

A woman may be infected after conception, when the child may be born nonsyphilitic or syphilitic by placental transmission.

Of course, when both father and mother are infected, the chances of the offspring being infected are doubled.

Morbid Anatomy. I. *Of Acquired Syphilis.—At least five sets of lesions* may be traced to acquired syphilis. The *first* is the initial lesion, the chancre or primary sore at the point of inoculation making its appearance two or three weeks after exposure. This constitutes primary syphilis. Beginning as an abraded spot, a vesicle or papule develops, forms an ulcer with a hard, gristly base and edge, constituting the hard of indurated chancre. It is found to consist in a dense infiltration of small cells, some of which develop into large formative (epithelioid) cells and others even into giant cells, but no further differentiation takes place; for the most part the infiltration breaks down and is absorbed, a few of the cells going to form the cicatrix. In the lesion is found the tryponema pallidum. The chancre is found usually in males on some part of the penis, especially on the prepuce, and in females on the labia or vaginal part of the cervix. It may be so small as to escape notice, especially when within the urethra. The sore lasts from three or four weeks to as many months. Its peculiar induration is easily recognized by taking it up and pinching it between the fingers, though it is often not characteristic on the flat mucous membranes of the genitalia of women.

Along with the chancre there is a *second* lesion, an adenitis of the adjacent lymph glands, forming a bubo, or there may be a hyperplasia of connective tissue, terminating in persistent induration of the gland. It usually appears soon after the induration. Buboes may be long stationary and are then said to be indolent. They may be multiple. They belong to the symptoms of primary syphilis.

The *third* lesion is the mucous patch, soft papule or *condyloma latum*, which is one of the events of the secondary stage of syphilis. It has its seat on mucous membrane of the pharynx, tonsil, or on soft, moist skin, as in the perimeun, groins, between the toes, at the junction between the skin and mucous membrane at the angle of the mouth, and about the anus. It consists of an inflammatory infiltration of the epidermis and corium with small cells. A more highly differentiated infiltration of the papillæ of the mucous membrane is the acuminate condyloma, or *venereal* wart, especially common about the vulva and anus.

The *fourth* lesion is the cutaneous affection, or *syphilid*, of which there is a roseolar or macular, a papular, a pustular, a squamous, and a tubercular variety. All are characterized by a copper-colored hue, especially permanent after the other features have subsided, and a tendency to symmetrical

distribution. The *macular* or roseolar syphilid affects more particularly the abdomen, the chest, and the front of the arms, while the face is exempt. This syphilid persists a week or two. The *papular* eruption is in groups on the face and trunk. The *pustular* eruption often closely resembles that of small-pox. The *squamous* syphilid resembles other squamæ, but it is especially distinguished by its coppery hue. It involves perferably the backs of the arms and the front of the thighs—the extensor surfaces—and is, moreover, rare. The skin syphilids are symmetrical in the early stages, but in the latest stages become irregular and unilateral in their distribution.

The *fifth* or remaining set of lesions constitutes the tertiary manifestation, and involves the deeper tissues, such as the subcutaneous tissues, the osseous and the nervous systems, the liver, lung and kidney. They include especially the *tubercular* and *fibroid induration*. The first occur in single nodules or may coalesce to form a solid tubercular patch, or serpiginous patches or segments of circles. They are confined to certain regions, as a rule, face, back, and more rarely extremities, and are usually unilateral. The most widespread is the *fibroid induration*, consisting in a development of fibroid tissue like that of chronic inflammation. The new tissue thus formed arises around the blood-vessels, and consists, at first, of a small-celled infiltration, which later is converted into fibroid tissue. It is found also in the periosteum, the sheaths of the nerve trunks, the capsules, and interstitial tissue of organs and muscles. It occupies, for the most part, small areas surrounded by normal, unaffected structures. When in the capsules of organs it sends prolongations into their interior, which partition off the organ and by their subsequent contraction give rise to irregular thickening and cicatricial puckering.

A differentiation of this fibroid change, a most characteristic lesion of syphilis, is the *gumma*, a yellowish white fibrous nodule, closely continuous by its outer layer with the connective tissue of the organ in which it is imbedded. It varies in size from that of a pin point to three to five centimeters (1 to 2 inches) in diameter. Histologically, it is with tolerable ease separated into three parts—a central or oldest part in a state of atrophic cheesy degeneration, an intermediate layer of imperfect fibrous tissue, and an external layer of vascular granulation-tissue rich in cells. It is frequently associated with the fibroid change above described. In the degenerative changes to which the gumma is subject it may produce extreme destruction of the organ in which it is imbedded.

The seats of the *gumma* are the subcutaneous and submucous tissues, muscles, fasciæ, bone, where it forms the *syphilitic node*, the connective tissue of organs, especially the liver, brain, testicle, and kidney, less commonly the lungs. When in submucous tissues, it may give rise to deep-seated ulceration and suppurative processes, leading to destruction, not only of soft tissues, but also of bone. Especially frequent and repulsive in its result is the destruction of the nasal bones with perforation of the palate.

Another variety of deep-seated syphilids, *syphilitic rupia*, consists primarily of large pustules, which dry and crust over with laminated scabs, while beneath is a deep ulcer. This may subsequently heal, leaving a scar.

Large pustular lesions and tubercular syphiloderms occur especially in the neighborhood of the sacrum.

Another tertiary lesion of syphilis, although probably not peculiar to it, is *syphilitic arteritis*, which consists in a cellular thickening of the vessel-walls, beginning in the intima and intruding thence on the lumen of the vessels. The outer coat is abnormally vascular and infiltrated with small cells that also invade the muscular coat. These are the phenomena of obliterative endarteritis, which have thus far been studied only in the vessels of the brain by Greenfield and Huebner.

Symptoms.—The symptoms of acquired syphilis are so largely the morbid states described under the head of morbid anatomy that most of them need only be enumerated in connection with the date of their appearance. The chancre or primary sore and the bubo, which together constitute primary syphilis, have been sufficiently described.

The secondary symptoms manifest themselves usually from the sixth to the 12th week, beginning with general lymphadenitis followed by *sore throat* commonly associated with *fever*, which rarely exceeds 101° F. (38.3° C.). It may be remittent and even strikingly intermittent, and in rare instances rises much higher than 101° F. (38.3° C.), reaching 104° F. (40° C.), and even 105° F. (40.5° C.). The sore throat alluded to is associated with hyperemia of the fauces, often with intractable, gray-based ulcers, and less frequently with mucous patches. The inflammation may extend from the throat into the Eustachian tube and middle ear, producing impaired hearing. The larynx is especially liable to become the seat of ulceration, which may heal and produce marked deformity.

Then there are the syphilids named. Along with these, a very common symptom is the *falling out of the hair*, and especially from the eyebrows, giving rise to a striking change in the facial expression. An inflammatory condition at the root of the nails, *syphilitic onychia*, causes them to become brittle and distorted. Other secondary symptoms not mentioned are *iritis*, and more rarely *choroiditis* and *retinitis*. The former presents itself in from three to six months after the primary chancre, and is one of the most painful and trying of symptoms, requiring prompt and energetic treatment. Involvement of the *ear* ossicles is rare but possible, producing deafness.

Joint affections are sometimes associated with tertiary syphilis. These may, of course, result from the invasion of the joint ends of the bones by the gummatous syphilitic disease, to which they are subject, but there may also be direct involvement of the *serous* tissues themselves by inflammatory and gummatous processes that give rise to pain and interfere with motion. The *bone affections* of syphilis are characterized by nocturnal pains.

The involvement of internal *glandular* organs occurs later, ten or more years after the primary lesion, though precocious tertiary lesions of this kind have been reported much earlier. *Amyloid disease* is a very common tertiary affection, involving liver and spleen and producing some of the most striking enlargements of the former. But cirrhosis and cicatricial markings are also common. Syphilitic lesions of the liver are of such a degree and importance as to demand separate consideration under the diseases of that organ.

An *atrophy of the follicular glands at the base of the tongue*—smooth atrophy of the base of the tongue—was early (at least as early as 1863) pointed out by Virchow as a symptom of late syphilis. Lewin and Heller[1] made a special study of it. They ascribed it to an interstitial inflammation and probably irreparable. Sixty-two per cent. of cases investigated were over 40 years old, more frequently found in men.

A *sarcocele* involving the whole testicle is among the tertiary affections often mistaken for tuberculosis, from which it may be distinguished by the fact that the latter is accompanied by tuberculosis elsewhere, and involves the proper structure of the testicle instead of the whole organ. *Sclerosis of the spinal cord* is frequently associated with syphilitic history, and it is often ascribed to it. A special condition is an involvement of the nervous system of such importance as to require a separate section. *Gumma of the brain* occur, producing pressure symptoms; a similar association is true of *arteriosclerosis* as well as the *arteritis obliterans* alluded to.

Sooner or later the syphilitic becomes *anemic* and an examination of the blood recognizes a reduction in the number of red corpuscles, in the hemoglobin and an increase in the white cells. Repeated abortion is a common symptom of syphilis in a married woman. Frequently four, five or even more abortions occur, while each successive one usually takes place longer after conception until finally a living child may be born.

II. *Of Hereditary Syphilis.*—Except the primary chancre all the symptoms described as occurring in acquired syphilis may be present in the congenital form. It may be said, in a word, that visceral alterations are more prominent, especially those involving abdominal organs. It is necessary, therefore, to mention here only those that may be regarded as additional.

Among the most important of these is premature birth. Such aborted products are shriveled, the skin exfoliates, and there is often reason to believe they have been some time dead. Syphilitic children born at term have evidently been arrested in development, are shriveled and wizen-faced, and may suffer from cutaneous syphilids.

The so-called *pemphigus neonatorum*, with blebs occurring about the wrists, hands, ankles and feet, is characteristic. There is also apt to be enlarged liver and spleen. Or a child may be born apparently healthy and take on these symptoms after three or four weeks. This is, however, unusual. *Rhinitis*, or nasal catarrh with *snuffles*, is one of the earliest symptoms, often followed by cutaneous lesions, particularly about the nates. *Fissures* about the lips and ulcerations on the muco-cutaneous surface may be present, and the discharges from these are inoculable.

Disease of the *epiphyseal cartilages* of long bones and of the cartilages of the ribs is a very common symptom of hereditary syphilis. The zone of the cartilage adjacent to the bone exhibits proliferated cartilage cells and prolongations over the end into the diaphysis instead of being sharply separated. There is *tendency to hemorrhage*. A *syphilitic cry*, high pitched

[1] Lewin & Heller, "Die glatte Atrophie der Zungenwurzel und chr Verhaltniss zur Syphilis." "Virchow's Archive," 138 p., 1894. The latest paper which also reviews the literature is by Nathaniel Bowditch Potter entitled "The Value of Virchow's Smooth Atrophy of the Base of the Tongue in the Diagnoses of Syphilis," published in the "Boston Medical and Surgical Journal," March 8, 1906.

and harsh, is described. To these may be added any of the symptoms already mentioned under acquired syphilis.

A later symptom is "notched teeth," first described by Jonathan Hutchinson as characteristic and distinctive of hereditary syphilis. The teeth affected are the permanent incisors of the upper and lower jaws. The appearances are not uniform, and are better appreciated by examining the accompanying drawings than from descriptions. Other late symptoms are *keratitis*, *iritis*, *impaired hearing* from ear affections, *periostitis*, and *splenic* and *hepatic* enlargement.

If it survive the earlier lesions or escape them, the syphilitic child remains undeveloped and stunted in its growth, and in consequence of arrest of development a singular reversal of the appearance of premature age,



FIG. 20.—The lower incisors of a girl, aged fifteen, the subject of inherited syphilis. The teeth are very short, rounded and peglike, with wide interspaces. This set shows the most typical condition ever exhibited by the lower set—(*after Hutchinson*).

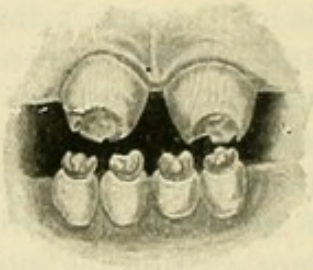


FIG. 21.—The two upper and four lower incisors (permanent) of a girl, the subject of inherited syphilis, all recently cut. The upper teeth are narrow from side to side, at their edges, and show a thin middle lobe, bounded above by a crescentic line. The lower teeth are rounded and show foliated extremities. All the teeth are small and spaces occur between the adjacent ones. In the upper ones the crescentic thin mid-lobe, and in the lower ones the foliated extremities will, before long, break away—(*after Hutchinson*).

described as characteristic of the syphilitic child at birth, takes place. The new-born syphilitic child looks prematurely old. A popular novelist has aptly described the appearance of the syphilitic child in the terse phrase, "a little old man with a cold in his head." The syphilitic who outlives his childhood remains, however, younger looking than he actually is, insomuch that a young man of 20 may appear as though he were but 12, a condition to which Fournier applies the name *infantilism*. In such the forehead is prominent, the frontal bosses are marked, the bridge of the nose is depressed, its tip turned up. The head may be asymmetrical.

Diagnosis.—The recognition of general syphilis is not usually difficult. The symptoms described are of themselves distinctive, but the complement deviation test of Wzssermann or Nagouchi's modification should always be made use of when the diagnosis is the least doubtful. According to Ricketts and Dick, it is present in from 64 to 100 per cent. of cases in the various stages of syphilis. A positive reaction may be misleading in from 0.3 to 3.6 per cent. of positive findings. This test is time consuming and requires much experience for accuracy. It therefore should be given into the hands of a trained laboratory worker.

Prognosis.—The prognosis of acquired syphilis depends wholly on the treatment. With early treatment properly conducted it is favorable; without treatment or with defective treatment the most serious consequences

result, while the physical inconvenience and suffering scarcely exceed the mental misery which the knowledge of the presence of so loathsome a disease entails. In congenital syphilis treatment is less satisfactory for the severer manifestations, and it is perhaps fortunate that so many perish in infancy or early childhood. Even those most fortunate remain delicate and vulnerable to disease through life, and too often fall victims to causes which but slightly affect the healthy man and woman.

Treatment. *Prophylaxis.*—Against sexual syphilis the only prophylactic measure to be relied upon is sexual purity. The duty of the physician is plain in respect to this, and the medical man who advises illicit sexual intercourse for any reason degrades his calling. Medical men should be exceedingly cautious in their necessary professional contact with all suspected of having syphilis and protect themselves against accidental infection. It is to be remembered that the secretions of all primary and secondary lesions, as well as the blood of syphilitics, may transmit the disease.

Treatment of the Primary Sore.—With the appearance of a suspicious sore the exudate from the lesion should at once be examined for the tryponema pallidum, and a Wassermann test should be made. If the organisms are found or if the Wassermann reaction is positive, the seat of the primary sore should at once be excised, if such excision is not mutilating, and the constitutional treatment recommended below at once be carried out.

The Constitutional Treatment.—This should begin at once. All syphilologists are agreed that Ehrlich's neo-salvarsan should be used in practically all cases of syphilis of whatever stage. The drug is best given intravenously with every asceptic precaution, and should only be given by one accustomed to such surgical procedures. If given very early it controls the disease better than any other drug. If given on the appearance of secondary or tertiary lesions, these lesions usually disappear magically. It, however, does not effect a cure in one dose. The neo-salvarsan should be followed by some form of mercurial medication described below. After about three months a Wassermann's reaction should be taken, if this is positive another dose of neo-salvarsan should be given to be again followed by mercurial medication, after another interval of three months a second Wassermann should be done. A third Wassermann reaction should be done at the end of a year after infection. The occurrence of three negative Wassermann reactions indicates a cure. *Mercury* is the remedy *par excellence* of the *second stage*. The best method of administration for mercury is undoubtedly by *inunction*. The following is the plan to be pursued: A warm bath is taken, if possible, each day, and immediately thereafter 1 dram (4 gm.) of mercurial ointment is spread between the hands and rubbed, one day on the inside of one thigh, the next on the inside of the other; again, under the arm, on the chest, and so on until each part of the body covered by softer skin is treated, after which the same course can be repeated. The friction is to be kept up until the skin is thoroughly dry, half an hour being usually necessary. The part rubbed should be washed off the following day. Parts covered with hair are to be avoided, because mercurial eczema, characterized by pustules starting from the hair follicles, is more apt to be produced in these localities. During this time the patient should not smoke, and the teeth should be

frequently and carefully cleansed and the mouth washed with solution of chlorate of potash with a view of averting mercurial sore mouth. Sooner or later, however, sore mouth may manifest itself by a fetid odor, swollen gums, and a sensation as though the teeth were loose, when the treatment should be suspended for a week or ten days, or until the signs of ptyalism have disappeared. The inunctions may then be resumed, this should be continued until the Wassermann reactions are negative.

The inunctions may be substituted by the use of hydrargyri iodidum flavum (protiodide of mercury) 1/4 grain (0.016 gm.) three times a day, or the biniodid, 1/16 grain (0.004 gm.) three times a day. The former is usually preferred because less irritating. This last addition to the treatment should be kept up until a cure is indicated by a negative Wassermann reaction. By such means as these tertiary symptoms can be averted if the patient is but willing to continue the treatment. The great difficulty is to secure this. He tires of the monotony and the trouble involved in a faithful adherence to the directions, and symptoms sooner or later return. Should secondary symptoms recur a course of inunctions may be repeated.

Most recently mercury has been administered by direct *injection* into the muscles. One-third grain (0.0216 gm.) of bichlorid dissolved in 20 minims (1.333 c.c.) of water is injected once a week, or from 1 to 2 grains (0.066 to 0.132 gm.) of calomel in 20 minims (1.333 c.c.) of glycerin and water. The salicylate of mercury may also be used. The injection is made deep into the muscles, and not in the subcutaneous tissue, through silver canulæ. The points selected are the sides of the thorax and back, where abscesses are said to be less likely to occur. Great care should be taken in sterilizing instruments. The nicest attention to these points is, however, still followed at times by abscesses. Many experts use this method almost exclusively.

In the treatment of the *third stage the iodids* are especially useful. It is here that large doses of iodid of potassium are indicated and often produce such magical results. The most convenient mode of administration is the solution, of which 1 drop contains one-half a grain (0 .033 gm.) Starting with 10 drops, a drop may be added each day to the dose until the symptoms yield, that is, until the tumors disappear. Pressure symptoms and head and bone pains are relieved. The iodid is well administered in milk. The indications for its discontinuance or reduction in the dose are the erythematous rash, coryza, and salivation and constriction about the throat due to swelling of the salivary glands.

Diseases Due to Animal Parasites

Out of the vast number of animals which in one or other period of the life cycle are parasitic upon some higher form of life, the group which infests man either as the definitive host or as host of some inermediate stage is comparatively small; and that which includes only the more important forms capable of actually working harm to the human host is much smaller. Representatives of the animal parasites of sufficient importance to be here considered are distributed among the *protozoa*, the *worms* and *arthropods*.

I Class: RHIZOPODA;

Order: Amœbina;

Genus: *Amœba.*

Amœba Coli, Lösch.

(*Amœba dysenteriæ; entamœba coli, Schaud.; entamœba histolytica, Schaud.; entamœba hominis, Casagrandi.*)

The amœba coli is commonly observed in the stools and in the wall of the colon of human beings subjects of so-called *amebic* dysentery. Its demonstration in the dejecta is to be made by placing a bit of fresh warm material upon a slide, and covering with slip without further precaution than to obtain a thin and even layer. The blood-stained mucus in the dysenteric stool is especially suitable for the purpose. The parasite may be recognized by obtaining the cells in active motion. They are very susceptible to the influence of cold and then quickly become quiescent; it is therefore best to insist that the stool should be quite fresh and warm from the body or with its warmth artificially maintained, and that the observation be made in a warm room or with the aid of a warm stage. In such case the movements are usually quite active and readily draw attention to the parasites; but even if slow and uncertain they may be surely determined by making outline drawings of a cell at frequent intervals for a few minutes and comparing these. It is difficult to reinduce movement when the animals have become chilled. In such preparations of dejecta, especially after they have become cold, encysted forms (spherical and with an apparent cell-wall) are often to be observed; this condition being apparently assumed as more resistant to the influences of heat, cold, drying, etc., and believed to be that most favorable for prolongation of life and the usual condition of amœbæ in transmission from one to a second host. In the walls of the colon the amœbæ are found in the necrotic matter of dysenteric ulcerations and in the surrounding tissues of the mucosa and submucosa, often being seen in great numbers in the lymph spaces of the latter layer, even at some distance from the base of the ulcer. (For the general description of the pathological changes, symptoms, and treatment of amebic dysentery reference should be made to the special section dealing with this disease (p. 88).



Fig. 22.—Amœbæ coli in fecal matter; several of the parasites show included red blood cells. In the fecal matter in addition to the granular (largely bacterial) matter, one may note red corpuscles, muscle and elastic tissue, a vegetable spiral duct and numerous crystalline bodies.

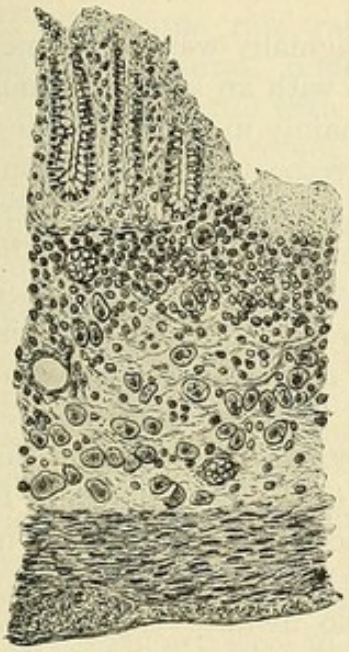


Fig. 23.—Section of wall of colon at border of dysenteric ulcer; showing loss of substance of mucosa, thickening of submucosa from inflammatory changes and in the latter large numbers of amœbæ coli.

Errant examples of the parasite are most frequently met in the pus of secondary hepatic abscesses in dysenteric subjects and in the bordering hepatic tissue, and in the lung and expectoration where such abscesses have perforated the diaphragm and penetrated the lung. They have been found rarely in other positions. The transmission of amœba coli is by no means clear. It is thought that the parasites when passed from the intestine of the original host assume an encysted condition and are carried to the alimentary canal of the second individual in impure water, or perhaps by foods tainted by such water or by contact with insects carrying the amœbæ from the dejecta in which they originally existed. Arrived in the large intestine the parasite multiplies by division; Grassi believes, too, that he has seen endogenous formation of small amœbæ within the encysted form.

A remedial measure of considerable value which should be mentioned in addition to the procedures recommended in the section upon amebic dysentery (p. 88) is the use of infusions or the fluid extract of the *chaparra amargosa*, a simarubacea growing in Mexico and Texas, given preferably by the mouth but also employed in enemata. The infusions of the entire plant seem to be more valuable than the fluid extracts of the pharmaceutical houses; and, given in wineglassful measure three or four times daily.

II. Class: FLAGELLATA; Subclass; *FLAGELLIDIA;*
Order: Polymastigida; Monostomea;
Genus: *Trichomonas.*
Trichomonas vaginalis (Donné),
Trichomonas intestinalis (Leuckart, etc.).

Originally it was believed that trichomonas vaginalis was to be met solely as a parasite of the vaginal canal, in women with an acid, spumous type of vaginal secretion, occurring mainly in young females, but possible at any time of life and irrespective of conditions of pregnancy or of actual menstruation at the time of examination. A number of instances of its occurrence in the urinary bladder and urethra of the male have, however, been recorded; and at present it is generally accepted that the organism described under the name *trichomonas intestinalis* is identical. It is believed, too, that similar organisms met in the mouth, stomach, and in pulmonary cavities and described as separate species are really of the same nature. It may be accepted, however, that it most frequently occurs in the vaginal secretions. It is apparently unproductive of any important results. There need be no definite vaginal discharge or any recognizable degree of vaginal catarrh; doubtless in the existence of such condition the parasite may have some minor influence in maintaining the irritative state, but is apparently unable or unlikely to initiate it. So, too, while symptoms of cystitis coexisted with the presence of the parasite in the bladder and urine in certain cases, it is not to be held as the essentially influential agency in their production. In the intestine it is present along with other protozoa, either with diarrheal symptoms or entirely without any symptoms.

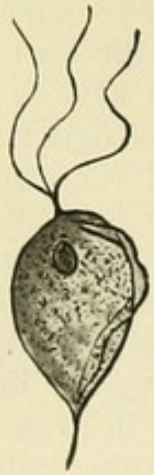


Fig. 24.—Trichomonas vaginalis.

As to its origin in the human host practically nothing is known. For

the intestinal occurrence it is natural that unfiltered water should have been suspected and several instances suggestive of this mode of acquirement have been published; perhaps females may by the use of similarly unclean water in bathing the genitals transmit it to the vaginal mucous membrane; and it has been suggested that it may be air-borne to explain its occasional occurrence in the lung. Experiments seeking to transmit the organism by the mouth to lower mammals have failed. Its persistence is variable. Often in young females it would appear that the menstrual discharge mechanically rids the canal of the parasites, yet it may in instances not infrequently be found persisting over months and years. It is not difficult of destruction by the use of alkaline vaginal douches or douches of very hot water. In the intestinal occurrence it often disappears without treatment of any kind; and generally the use of calomel and intestinal antiseptics is followed by the early disappearance of the organism.

Family: RHIZOMASTIGIDA;

Genus: *Trypanosoma.*

Trypanosoma gambiense (Dutton), (*Sleeping Sickness*).

Elongate flagellates, two to four times the length of a human red blood-corpuscle: with an undulatory membrane extending the length of the more or less curved and slightly spirally twisted, delicately fusiform body, and prolonged into a long, single flagellum at one end; at base of undulatory membrane at nonflagellate end is a small refractile body regarded as a centrosome or by others as a micronucleus; an oval nucleus near middle of body; reproduction by cell division (believed to take place after sexual fertilization and ookinet formation in the intestine and body of the human tse-tse fly, the definitive host); parasitic in human blood and cerebrospinal fluid (human beings regarded as the intermediate hosts).

Trypanosomes of different species have been discovered in the blood of frogs, birds, rats, rabbits, guinea-pigs, of horses suffering from diseases known as surra, douraine, mal de caderas, of cattle with tse-tse fly disease; and within recent years the form above outlined has been encountered in the human blood by Nepveu, Dutton, and Mason. Quite recently Castellani has demonstrated with much uniformity the presence of the same species in the cerebrospinal fluid of individuals, almost invariably negroes, presenting the symptoms of the African *sleeping disease;* and has apparently led to the solution of this mysterious and fatal affection, the etiology of which has hitherto been entirely a field for surmise. This malady is pratically unknown save in Africa; and is but infrequently met in Europeans in the locations where it is endemic. It runs a rather long course, the period of incubation being of months or even a year or two in duration, the period of the active manifestations extending over three, four or five months in addition before the fatal termination. When fully developed the disease is essentially a meningoencephalo-myelitis and is characterized by progressive lassitude and mental dullness, deepening into somnolence and coma, by tremors and uncertainty of gait and eventually inability to progress, edema of moderate degree, especially about the face, irregular temperature, rapid

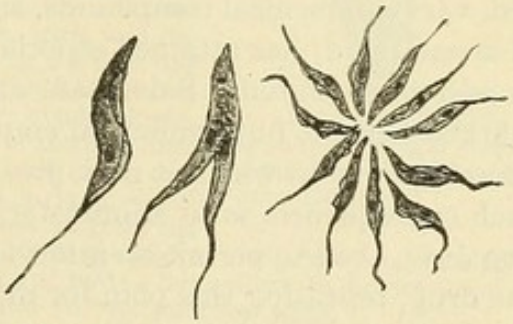


FIG. 25.—Trypanosomes; showing ordinary structural appearance on left; in middle a trypanosome undergoing division; on the right an agglutinated group.

pulse, emaciation, glandular enlargements, a papulovesicular eruption becoming superficial ulcers, and eventually death. L. Lorand ascribes the malady to a degeneration of the thyroid consequent on the action of the toxins generated by the trypanosome.

The parasites are readily found in the cerebrospinal fluid and at times also in the blood. They may also be obtained in the juices removed by a hypodermic syringe from the enlarged lymph nodes. They are to be sought for in ordinary fresh moist films or in dried preparations just as one examines the blood for malarial hematozoa; and in dried and fixed films are stained in the same manner as are the malarial organisms. In the moist film the trypanosomes may be seen winding their way among the corpuscles, imparting a slight motion to the cells. There can be little doubt from the symptomatic picture of the disease and from the experimentally discovered fact that the pathogenic power of an allied species (*T. evansi*, of the rat, cultivated by Novy on agar with defibrinated rabbit blood added—the first successful artificial cultivation of a protozoon) may be lost by prolonged artificial culture, that the effects of the parasite are largely the result of some toxin in some way elaborated by it. It has been shown both by clinical studies and by experiments upon monkeys that this species is transmitted by the human tse-tse fly (*glossina palpalis*), thus closely following the transmission of *t. brucei* in cattle which is conveyed by *glossina morsitans*, the tse-tse fly of cattle. It is of interest to know that Schaudinn has recently suggested that spirochætæ, regarded as of bacterial nature, and known best in connection with relapsing fever, are in reality trypanosomes.

Treatment.—In the treatment of sleeping sickness a number of trypanocides are available, as members of the benzidin group such as trypan red, various arsenical compounds, among which atoxyl (an aniline compound of arsenic acid) has attained especial prominence, and various basic anilines, as well as mercurial. Salvarsan, 0.4 or 0.5 gm., has been widely used with marked benefit, but cannot be employed with impunity because of serious complications to which it may give rise. The method best suited to escape such consequences is to administer the chemical in the dose mentioned for two days, then to permit an interval of ten or 15 days for the elimination of the drug, repeating this plan for months. In the intervals mercurials may be given or some of the anilines. The combination of trypanocides in this fashion is further sustained by the fact that Ehrlich has shown in experimentations that trypanosomes may attain immunity against the various chemicals used for their destruction and that such resistance may be carried forward for many generations; but that where a strain of trypanosomes is encountered which is resistant to a given chemical it may be readily destroyed by some other type of trypanocide. After administration of atoxyl the parasites are soon diminished or even lost from the fluids used for examination, but improvement of the general condition of the patients is not likely to be recognized until after some weeks of persistence of treatment.

Prophylaxis must of course be of great importance. It contemplates the destruction of herbage about the damp places where the human tse-tse flies abound, the careful screening of all dwellings, the proper protection of those exposed to their bites by suitable clothing, the removal of all infected individuals and animals from districts where the disease does not

ordinarily prevail to situations where the disease is endemic, and the destruction of such animals (crocodiles) from which the flies seem ordinarily to obtain the blood which seems necessary for their life and reproductive ability.

II—VERMES OR WORMS.

Worms are bilaterally symmetrical, more or less elongate animals, without articulated members, with the body usually showing a number of apparent rings or segments (metameres), and are either flat or cylindrical in transverse section. Excluding a number of classes as of no interest in the present connection, there remains the classes of Flat worms and Round worms as including various genera and species in which occur human parasites.

Family: FASCIOLIDÆ;
Genus: *Fasciola.*

Fasciola hepatica (Lambé),
(*Distomum hepaticum; d. caviæ; fasciola humana; cladocœlium hepaticum;* the common liver fluke).

A comparatively large fluke, measuring 20.–50. mm. long and 8.–13. mm. wide, of leaf shape, with anterior extremity prolonged into a small cone; greatest width of body about anterior third of length; light brown color; cuticle provided with alternating transverse rows of spines, extending on ventral surface to the posterior level of testes, but not as far posteriorly on dorsal surface; oral sucker at anterior end of cephalic cone, inclining to ventral surface (1. mm. in diameter); ventral sucker near anterior end behind cephalic cone (1.6 mm. in diameter); well-developed pharynx and short esophagus; intestinal branches extending nearly to posterior extremity of worm, approaching the median line posteriorly, with few median and numerous lateral branches; excretory pore at posterior extremity, with well-developed system of excretory tubes; genital pore in median line anterior to ventral sucker; two large, highly branched testes, mostly posterior to the transverse vitelline duct; ovary single, branched, lying in front of testes and to one side of median line; uterus coiled into a rosette, showing as a brown spot (from ova contained) just back of ventral sucker on ventral surface; vitelline glands numerous, ranging along each lateral border from the level of the ventral sucker to the posterior extremity of the worm; vitelline ducts running transversely at about the end of the anterior third of body; ova yellowish-brown, oval, operculated, measuring 130.–145. microm. long and 70.–90. microm. wide.



FIG. 26.—*Fasciola hepatica:* natural size; cleared in oil. (*Gould after Leuckart.*)

This parasite is a common one in sheep; and is also found in cattle, deer, goats, hogs, horses, asses, camels, rabbits, guinea-pigs, and other mammals. It is occasionally (23 cases recorded according to Braun) met in man. It has a wide geographical distribution over the world, and is not infrequently found in this country. Its usual habitat is the gall ducts; but it has been encountered ("errant flukes") in the gall-bladder, intestines, in the portal and other venous channels, and rarely in subcutaneous cysts. The life-history has been unusually well followed out and may be outlined as follows. The ova after oviposition are carried by route of the gall-passages and intestines to the exterior where, if fortune favors, they are deposited in water. Here the embryo develops and escapes through the operculum, swimming about as the free miracidium until it can gain entrance to the tissues of a snail (some form of *Limnæa*), in which the sporocyst is formed and the rediæ (half a dozen or more) develop; these presently emerge from the parent cyst and wander further into the tissues of the snail,

each then again encysting and dividing into a number of cercariæ. By this time probably the snail has died from the effects of the parasites; and its tissues disintegrating, the tadpole-like cercariæ escape in the water and for a time swim free. These later become attached to a blade of grass or other similar object, lose their tails, and become covered with a hard covering, a product of their cuticular glands, and thus remain protected for a variable time. Later the definitive host, probably a sheep, devours the grass and with it the cercaria, which on arrival in the alimentary tract

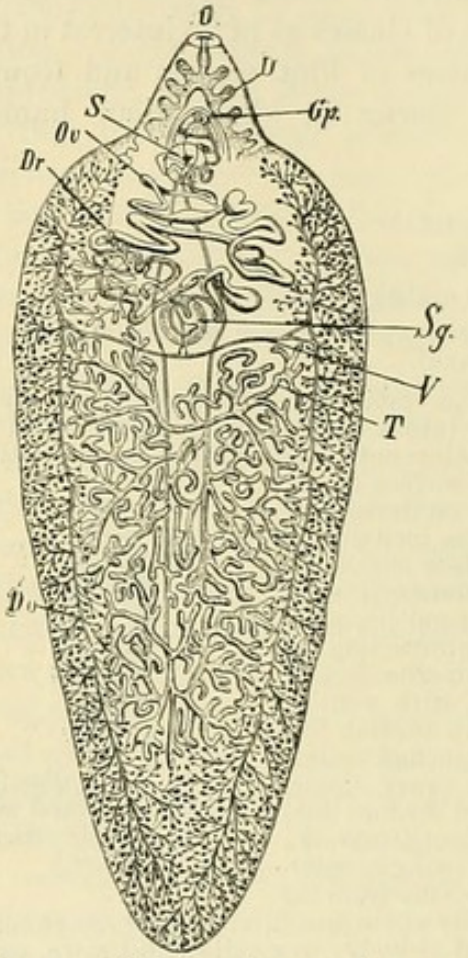


FIG. 27.—Showing the sexual organs of fasciola hepatica; 5×1. *O*, oral sucker; *D*, intestinal ceca; *Do*, vitelline glands; *Dr*, ovary; *Ov*, uterine canal; *T*, testicles; *Sg.*, "shell gland," *V*, transverse vitelline duct; *Gp*, genital pore; *S*, ventral sucker. (*Braun.*)

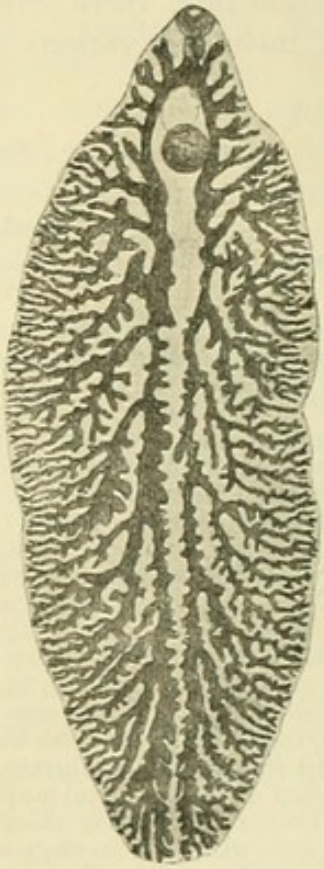


FIG. 28.—Showing the alimentary system of *fasciola hepatica*, other parts suppressed; 5×1. (From a fluke as yet undeveloped in its sexual organs.) (*Braun.*)

has its covering removed through the action of the digestive juices and in some manner (whether by the common duct into the hepatic duct and its radicals, or by boring through the intestinal wall into the portal vein and thus carried to the liver, is unknown) finds its way into the bile ducts, where it grows into the adult worm. Doubtless in the occasional cases of human acquirement it is similarly conveyed upon some water vegetables, as cress, to the stomach. In the lower animals there are often found large numbers (from dozens to hundreds) of the flukes in the bile ducts of a single host; but in man, while this is possible, ordinarily one finds but very few.

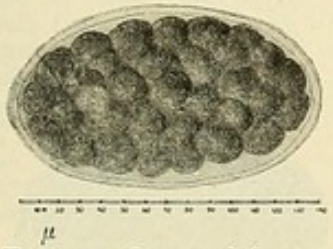
The effects as seen in the lower animals—quite similar in general char-

acter in case of man—are of little severity and may well be overlooked in case the parasites are but few; but when the parasitism is of serious grade a condition often proving fatal, and known in sheep as "liver-rot," is induced. The worms are well anchored in the small tubes by their suckers and posteriorly inclined cuticular spines, and the ducts are often at site of the parasites distended into small cysts. The flukes suck blood from the walls of the ducts, and cause considerable biliary and peribiliary irritation with fibrous thickening of the walls and a cirrhotic extension into the surrounding hepatic tissue, which undergoes atrophic and degenerative changes in some degree. Hemorrhages into the ducts often occur, constituting one of the influences producing the more or less severe anemia characterizing the affection. Gallstone formation is quite common, mixed concretions of biliary matter, mucus, and blood remnants sometimes forming complete casts of greater or less length of the infested ducts. Thus biliary obstruction with the development of icterus, and portal interference with the production of ascites, are common. At first, the liver is enlarged, but eventually becomes atrophic from the cirrhosis and tissue degeneration. Digestive disturbances with anemia, loss of flesh and enfeeblement appear, and eventually death is apt to take place from exhaustion. These symptoms, usually first appearing in a month or two after the parasites gain access to the liver, are evidence of an active but definite inflammatory and degenerative hepatic affection; and for the absolute diagnosis of the nature of the case recourse must be had to the microscopic examination of the dejecta for the ova of the parasites. The majority of instances encountered in man have been met at autopsy, without previous suspicion of their existence.



FIG. 29.—Miracidium of *fasciola hepatica*. (*Gould after Leuckart.*)

It is probable that the cases of flukes in the eye and described under the names *Distomum oculi humani* (Ammon, 1833), *Monostomum lentis* (v. Nordmann, 1832) and *d. ophthalmobium* (Diesing, 1850), were but errant and immature examples of the worm under discussion.



μ

FIG. 30.—Ovum of *fasciola hepatica*.

Treatment.—But little of value can be said in connection with treatment of the condition, the most important measures being prophylactic and looking to the restriction of the flocks and herds from pastures on badly drained land where the common grass snails are known to be present. In active treatment it must be clear, because of the position of the flukes, that difficulty exists to reach the parasites with medicaments capable of destroying them. Numerous remedies have been suggested for the affection in sheep and the same substances may be tried in human cases. Salol is said to have lethal effects upon the trematodes in the liver, but must be given in comparatively large doses and persistently; the ethereal extract of male fern is sometimes employed, as well as naphthalin, various salts of iron, sodium chlorid, and a number of other substances. In addition

measures may be indicated to combat the digestive and nutritive symptoms, but these are in no wise peculiar.

The Asiatic lung fluke inhabits the bronchial tubes of man, cat, dog,

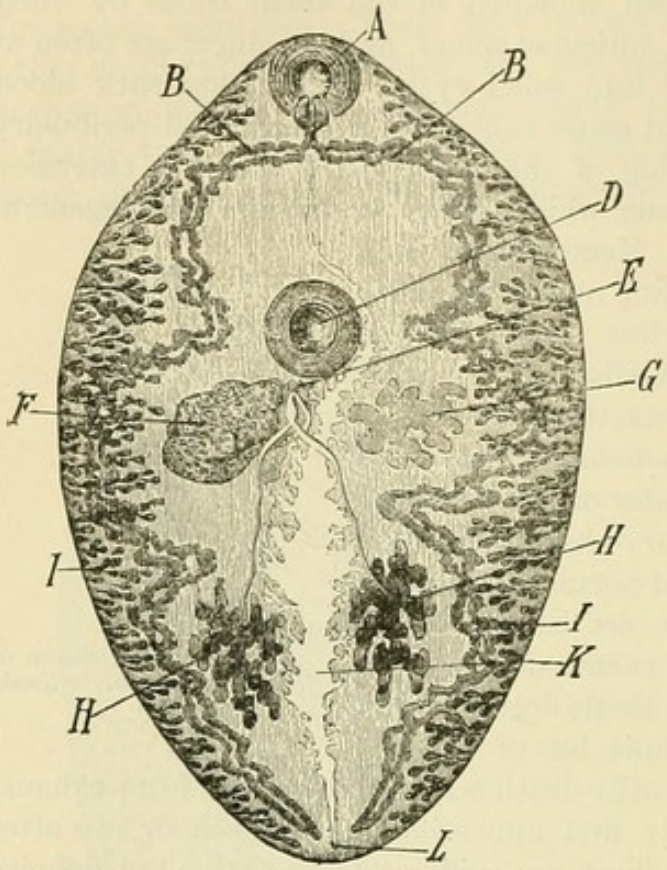


FIG. 31.—*Paragonimus westermanni* (ventral view). 10×1. *A*, oral sucker; *B*, ceca; *D*, acetabulum; *E*, genital pore; *F*, uterus; *G*, ovary; *H*, testicles; *I*, vitelline glands; *K*, excretory canal; *L*, excretory pore. (*Braun, after Leuckart.*)

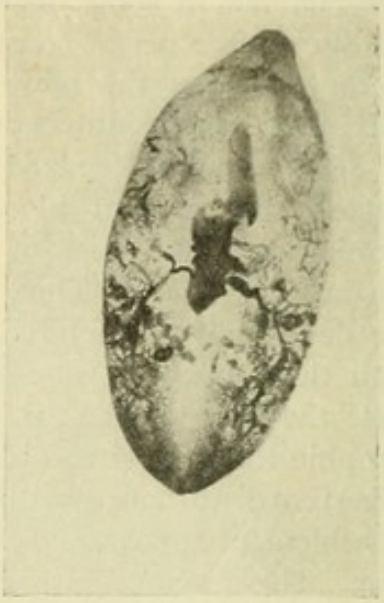


FIG. 32.—*Paragonimus westermanni;* photograph from a sexually immature specimen.

hog, and mouse; it has also been recorded from the bronchial tubes of a tiger in the zoological gardens of Amsterdam; the worm has also been found in the brain and a few other situations in the body. Usually there

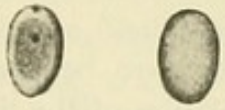


FIG 33.—*Paragonimus westermanni:* natural size; to left showing ventral surface; to right showing dorsal surface. (*Braun, after Katsurada.*)

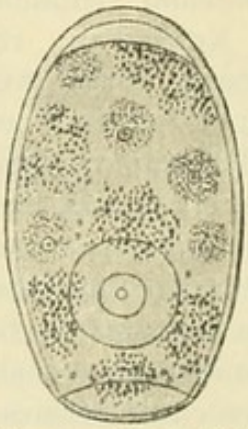


FIG. 34.—Ovum of *paragonimus westermanni*, from sputum; 1000×1. (*Braun, after Katsurada.*)

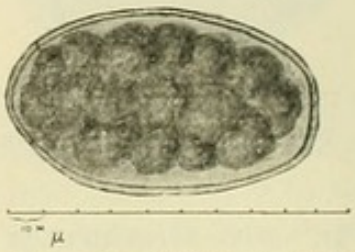


FIG. 35.—Ovum of *paragonimus westermanni*, from sputum.

are small bronchiectatic cysts in the lungs, in which in the midst of a reddish-brown mass of blood and mucus the worms are found, generally two in each such cyst; the pulmonary tissue about these cysts, fibroid or

otherwise changed, and the bronchial mucous membrane in the vicinity of the cyst more or less inflamed. From tissue destruction in the cyst-wall bronchial hemorrhages from time to time are apt to take place, constituting the most pronounced evidence of the affection and leading to some degree of anemia. As a rule, the patient suffers but little inconvenience beyond a slight cough, and the hemoptysis rarely requires special attention. In the sputum the ova are readily found, their recognition establishing the diagnosis. These are known to develop in from six to eight weeks in water into a ciliated miracidium; but all further knowledge as to the intermediate stages and their hosts is lacking. The parasite has been encountered most frequently in Japan, Formosa, Korea, and China; in the hog, dog and cat, it is known to exist in this country, and one imported case in man has been reported from Portland, Oregon.

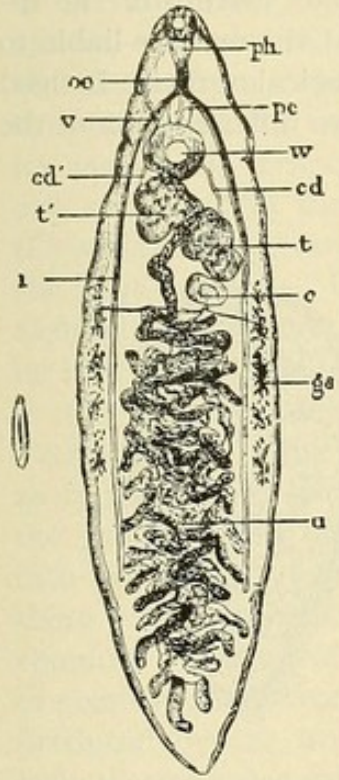


FIG. 36.—*Dicrocœlium lanceatum*: on left natural size; on right enlarged 10 times (ventral view); *ph*, pharynx; *œ*, œsophagus, *i*, cecum; *t* and *t'*, testicles; *cd* and *cd'*, vasa differentia; *pc*, cirrus pouch; *o*, ovary; *ga*, vitelline glands; *u*, uterus; *v*, vagina; *w*, acetabulum. (*Railliet.*)

The condition is not incurable; inhalations of balsamic material, of chloroform short of anesthesia, and similar measures holding out some promise of success in destroying the parasites and allowing them to be dislodged by coughing efforts, providing they are not too deeply encysted in the bronchial pockets. Nothing is known of the modes of acquirement of the worm and hence no known prophylactic measures exist at present.

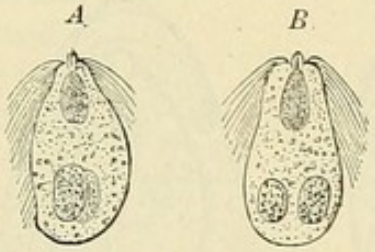


FIG. 37.—Miracidium of *dicrocœlium lanceatum*: *A*, lateral and *B*, flat view. (*Braun, after Leuckart.*)



FIG. 38.—Ovum of *dicrocœtium lanceatum*. ×600.

Family: SCHISTOSOMIDÆ;
Genus: *Schistosomum.*

1. Schistosomum hæmatobium (Bilharz).

(*Distomum hæmatobium; d. capense; Bilharzia hæmatobia; gynecophorus hæmatobius; thecosoma hæmatobium;* African blood fluke.)

Sexes separate. *Male:* whitish, 12.–14. mm. long (often less), with margins back of ventral sucker folded on ventral surface so as to form a long groove (gynecophorous groove) and when thus folded the thickness is about half a millimeter; oral and ventral suckers close together in the attenuated anterior end of the worm on ventral surface, prominent; esophagus short, dividing into two simple intestinal tubes which back of the testes reunite to form a single cecum; numerous gland cells about esophagus; excretory pore opening at posterior extremity slightly dorsally; genital opening in median line back of ventral sucker; no cirrus pouch; testes five or six in number, vesicular; cuticle of dorsal surface covered with small spinulated tubercles, of ventral surface bearing spines except along the median line. *Female:* whitish to reddish-brown; filiform; length 15.–20. mm.; suckers as in male, prominent; cuticle with spines; digestive tube as in male (single cecum from reunion of intestinal tubes posterior to ovary); ovary oblong, lobate, lying in posterior fork of intestine; vitelline follicles extend posteriorly to extremity of body

from just back of ovary; oviduct and longitudinal vitelline duct run forward to unite at "shell-gland" with the uterus in a dilated oötype; uterus straight, tubular, extending forward to the genital pore, which opens at posterior border of ventral sucker. The two sexes lie constantly together, ventral surfaces in contact, the female in the gynecophorous groove of the male. Ova oval in shape, thin-walled, without operculum, with a spine at one end or along the side of one end, 135.–180. microm. long, 55.–60. microm. broad.

The blood fluke is found in the veins, commonly the portal veins and its branches, and the plexuses about the bladder and rectum, in man and a few species of monkeys. It is met mainly in Africa (Egypt, Abyssinia, Sudan, Mozambique, Natal, Tunis, Algeria, etc.); but has been reported in imported cases from this country and elsewhere. It is said also to occur in Cuba and Porto Rico. It is more common in the young than in adults.

The presence of the adult worm is not the serious feature of the infection, the fault depending rather upon the fact that the ova are liable to obstruct the vessels and give rise to important pathological results. Lodged for example in the small veins of the vesical plexus, an inflammation of the

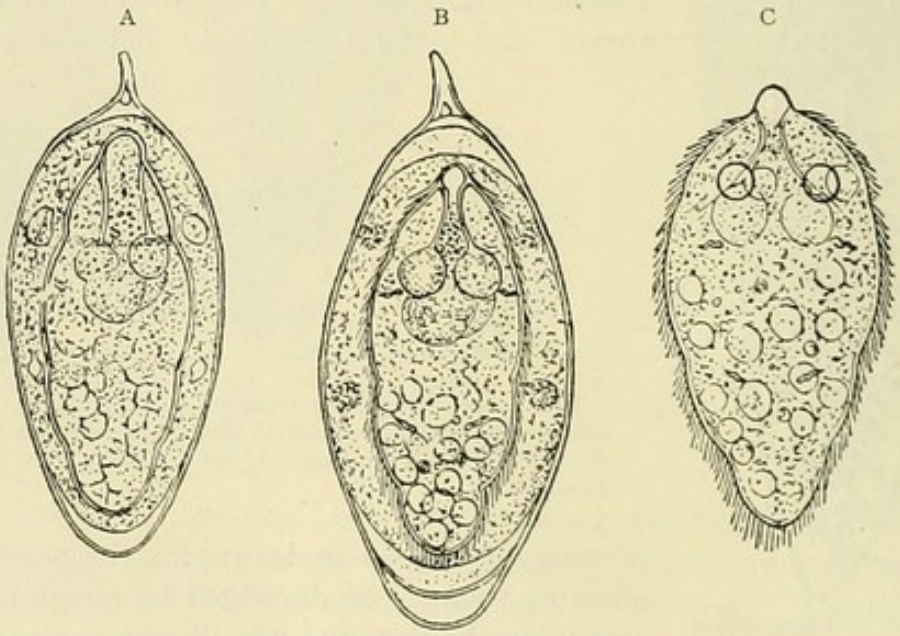


FIG. 39.—Ova and miracidium of *schistosomum hæmatobium*, ×300; A, ovum as seen in urine; B, the same after addition of water; C, miracidium. (*Railliet.*)

bladder and rupture of the occluded vessel with appearance of blood in the urine are apt to result; or if in the wall of the lower bowel, a proctitis or colitis with dysenteric hemorrhages. The vesical symptoms are not infrequently marked, pain and tenderness in the hypogastrium, a burning pain in the urethra, especially on micturition, difficulty of micturition, sometimes evidence of prostatic swelling, with the urine containing blood, pus, and mucus as well as the ova of the parasite. Sometimes the inflammatory disturbances may extend along the ureter to the pelvis of the kidney and the latter organ itself, inducing the symptoms of a more or less grave nephritis. Occasionally from convection of the ova into the liver or lungs symptoms referable to these organs may also be met. Fatal results are to be apprehended after a variable period of such symptoms, with secondary anemia, debility, and exhaustion.

No knowledge is had of the intermediate stages beyond the miracidium, which is not infrequently seen in the ova in the urine or feces. It is suspected

that the infection is carried through unfiltered drinking-water, and as a measure of prophylaxis this should be strictly cared for in infested districts. No plan of successful medication is known; but all means of ordinary character for conservation of nutrition and repair of blood loss should of course be undertaken as well as the usual methods of overcoming the actual hemorrhage, as the use of ergot, etc.

This fluke has been met in cats and in man in Japan and China. It is believed to be responsible for an endemic affection known in Japan as Katayama, characterized by enlarged liver and spleen, disturbances of appetite, diarrhea (the dejecta often containing blood and mucus), and in severe cases anemia, fever, ascites, and edemas, and occasionally death from exhaustion. The worms inhabit the vena porta and its mesenteric branches, and the ova are found in the liver and in the walls of the intestine, especially the large intestine. Unlike *s. hematobium*, these ova are not apt to be met in the urine but are found in the intestinal dejecta. In the liver and wall of the bowel, as well as in mesenteric lymph-glands, in the pancreas and other situations into which the ova are carried, there ensue either from the mechanical irritation of these objects or possibly partly from toxic factors produced by the worms and similarly disseminated (or from both of these influences) chronic hyperplastic and indurative changes which may be of serious importance. Anemic symptoms are best seen in heavy infestments, and are thought to be partly due to the blood destruction and partly to toxic influences. Yamagiwa, according to Katsurada, believes that a case of epilepsy which he originally attributed to lodgment of ova of *paragonimus westermanni* in the brain of the patient was due in reality to ova of the parasite here mentioned.

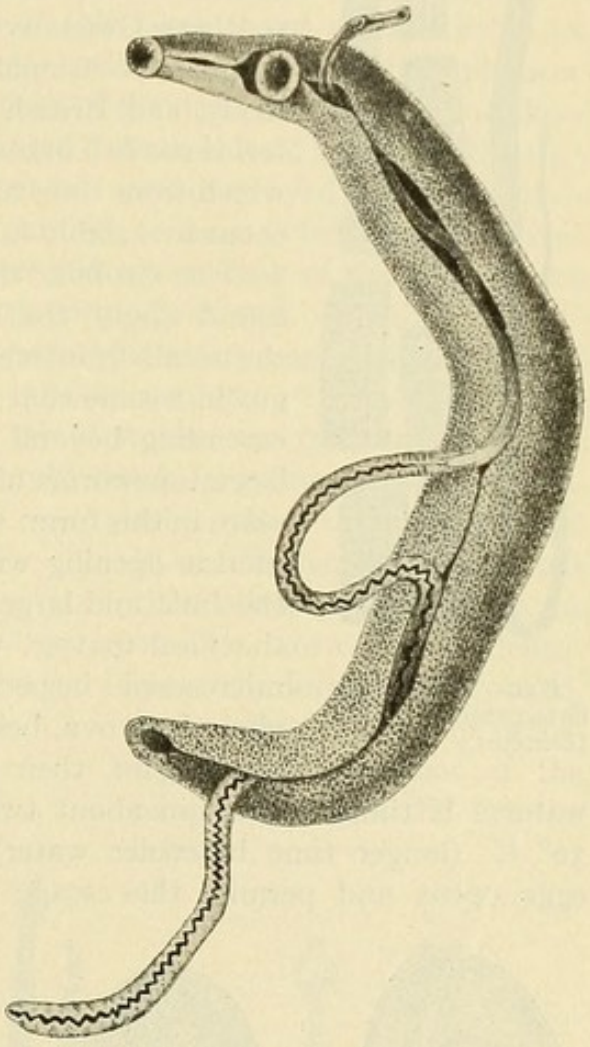


Fig 40.—*Schistosomum hæmatobium:* male with female in gynecophorous groove. (*Braun, after Loos.*)

1. **Dibothriocephalus latus** (Leuckart).

(*Tænia lata; bothriocephalus latus; dibothrium latum; bothriocephalus latissimus;* fish tapeworm; etc.)

Description.—Strobile two to ten meters or more in length (reported 20 in one or two cases); strobile yellowish; marked in ripe segments by brownish central rosette (uterus with ova) when specimen is soaked in water; head elongated almond-shape (2.–5. mm. long, and 0.71 mm. transversely), with two lateral grooves or bothridia as suckers; neck variable according to degree of contraction; 3000–4000 segments; the

anterior links poorly defined, in their growth increasing slowly in length but markedly in breadth; ripe links back of middle of strobile measure 2.–4. mm. long and 10.–12.–20. mm. wide, with opaque brownish rosette in middle line; terminal links shrunken and narrower than above in proportion to the length after discharge of ova; vagina and cirrus open in small prominence in midventral line near anterior border of link and just back of this a third opening (uterine); testes numerous, best seen toward the margins; uterus of a number of plicated tubes in form of rosette; ova brownish, ellipsoidal (68.–71. microm. long, 44.–45. microm. transversely), operculated.

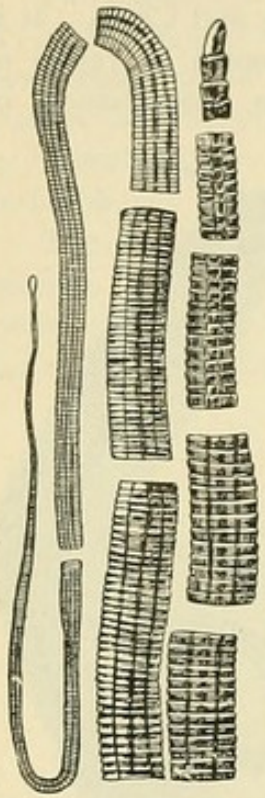


FIG. 41.—*Dibothriocephalus latus.* (*Leuckart.*)

This parasite in its adult stage is most commonly met in the human intestine, but has been encountered also in dogs and cats. It is most common in parts of central Europe (Switzerland, northern part of Italy, southern Germany and this vicinity), along the Baltic borders, occasionally in Denmark and in the Netherlands, and British Islands. In Asia it has been met a few times in Turkestan and in Japan. The few examples which from time to time are reported from this country occur invariably in foreigners.

The strobile, the longest met in man, is generally found singly, the head attached in the upper part of the small intestine and the length trailing through the gut in a somewhat plicated fashion and not infrequently extending beyond the ileo-cecal valve. Unlike other large tapeworms of the human intestine ovulation takes place in this form, the eggs being discharged through the uterine opening without the necessity of destruction of the link, and large numbers are usually encountered in the fecal matter, where they are readily recognized on microscopic inspection. In the life-history of the parasite, these ova, being carried out with the fecal matter, require for their further development immersion in water. If thus favored, in about two weeks at a temperature of about 30° C. (longer time in cooler water) the operculum at the end of the eggs opens and permits the escape of a minute hexacanthous embryo,

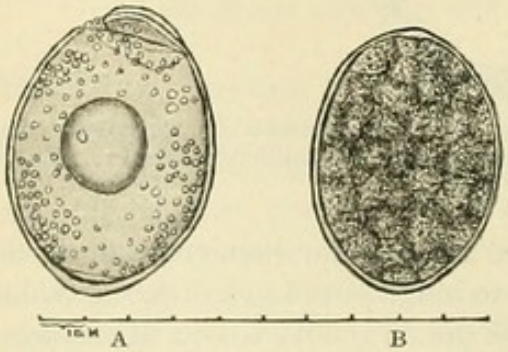


FIG. 42.—Ova of *dibothriocephalus latus: A*, after treatment with sulphuric acid so as to render lid apparent; *B*, natural appearance in fecal matter.



FIG. 43.—Free swimming embryo of *dibothriocephalus latus.* ×500. (*Leuckart.*)

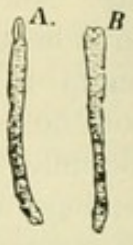


FIG. 44.—Plerocercoid of *dibothriocephalus latus: A*, with head projected; *B*, head retracted. (*Braun.*)

which is surrounded by a ciliated membrane and which lives a free existence in the water for a time. It is not known how this embryo passes to the intermediate host, one or other of several fresh-water

fishes, but it is probably swallowed; in event of failure to attain this secondary host, it dies after some days. Within the fish it develops into a small worm-like larva (8. mm. long, 3. mm. thick), known as a *plerocercoid*, which, without a surrounding (adventitious) capsule, is found in the ovary, wall of the intestine, liver and other viscera, and in the muscular system. It possesses a slight vermicular movement. Essentially it is the anterior end of the future adult tapeworm. The head is generally invaginated, but may be caused to protrude if the specimen is placed for a time in warm water, when it will be found to present the characteristics above outlined for the adult head; unlike the common bladder-worms of many tapeworms the posterior part of this larva is not cystic. From experiments it has been found that under ordinary conditions these plerocercoids will retain their vitality in the flesh of the dead fish for about 18 days, that they are quickly killed in a saturated sodium chlorid solution, that they are comparatively resistant to cold but are soon destroyed by a heat above 50° C.–53° C. Transmission of the parasite to man is believed to be confined to the swallowing of the plerocercoid in the flesh of the infested fish imperfectly salted or cooked; although formerly it was believed if the ciliated embryo were taken into the human alimentary canal with unfiltered water, it was able to develop directly into the strobile. The fish most liable to contain the larval worms are fresh-water fish, as pike, turbot, perch, tench, grayling, etc. In countries where these fish abound it not infrequently happens that certain parts, as the roe or liver, are eaten as delicacies very imperfectly cooked, such habits distinctly favoring the acquirement of the parasite, should the fish be infested.

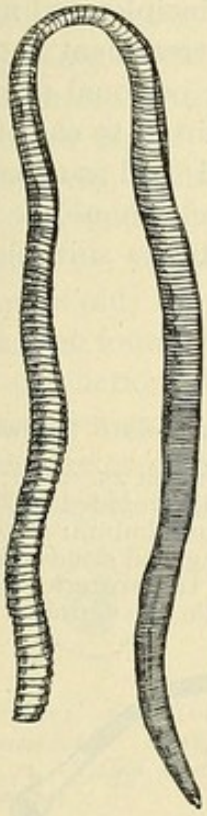


FIG. 45.—*Dibothriocephalus cordatus:* adult. (*Leuckart.*)



FIG. 46.—Young specimens of *diothriocephalus cordatus;* natural size. (*Leuckart.*)

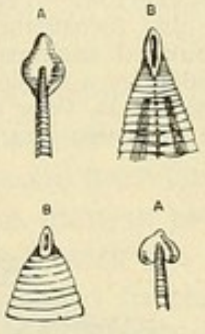


FIG. 47.—Head and anterior segments of *dibothriocephalus cordatus: A*, seen from margin of strobile; *B*, seen from surface of strobile. (*Leuckart.*)

The *symptoms* of parasitism by *dibothriocephalus latus* are for the most part quite similar to those occasioned by other tapeworms, *alimentary* and *nutritive* and *reflex nervous*. Usually the alimentary or local symptoms are the most pronounced, and proportionately more marked than in the case of

infestment by the beef and the pork tapeworms. These may be very trivial and be entirely neglected, however, although there is commonly some degree of abdominal discomfort, fullness, and a sense of weight, with now and again some little pain. Often the pains, irregularly intermittent in occurrence, are severe. Diarrhea alternating with constipation is common, and occasionally nausea and vomiting are noted. The appetite is apt to be capricious. In time some loss of weight and strength and a more or less striking and notable anemia may be expected which resembles in every particular so far as blood-findings are concerned the progressive pernicious anemia, the latter being supposed to be due to the absorption of some toxic principle elaborated by the parasite or developing under conditions of its presence in the alimentary tract. The nervous phenomena are generally of little moment, but may exceptionally be pronounced. There may be nasal and anal pruritis, ocular disturbances, sometimes a functional and transitory strabismus, choreiform twitching, epileptiform seizures, headaches, mental hebetude, tinnitis, etc.

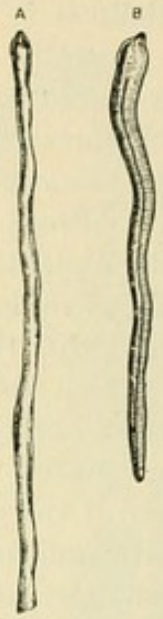


FIG. 48.—*Bothriocephalus mansoni: A*, after Leuckart; *B*, after Cobbold.

Treatment rests upon the general principles and measures detailed in the section devoted to treatment of tapeworm disease in general (p. 211). For personal prophylaxis the individual should not be permitted to eat of the flesh of fish from infected districts except it be well cooked; and as a general measure to prevent the infestment of the fish, human excreta should be kept from the drainage into lakes and rivers inhabited by fishes suitable for the plerocercoid stage.

Genus: *Hymenolepis.*

1. Hymenolepis nana (von Sicbold).

(*Tænia nana; t. ægyptica; diplacanthus nana; hymenolepis muria;* dwarf tapeworm.)

DESCRIPTION.—Average length of strobile 10.–15. mm. (may' reach 25. mm.); head subglobular, measuring 0.25–0.30 mm. in transverse diameter; head provided with four large rounded suckers and a large rostellum retractile into an infundibulum; rostellum surrounded by a single crown of hooklets (24–30); neck rather long and slender; about 150 proglottids, very small, broader than long; the largest, near the posterior end of strobile, measure 0.14–0.30 mm. long by 0.4–0.9 mm. broad, while the terminal links

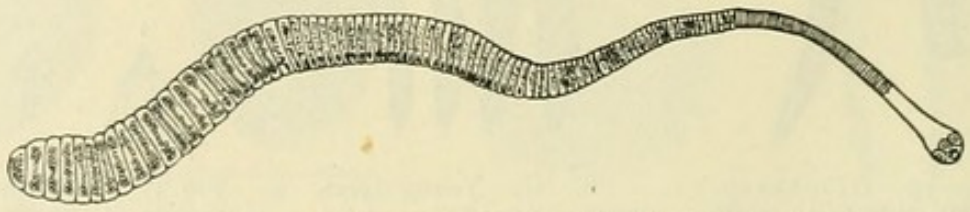


FIG. 49.—*Hymenolepsis nana:* ×10. (*Gould, after Leuckart.*)

narrow slightly and lengthen, so as to give a rounded posterior extremity to the strobile. Genital pores all on the same (left) margin, near anterior end of margin of links; three spherical testes, in posterior part of segment near dorsal wall; vas deferens small, straight, slightly distended before reaching cirrus pouch; cirrus pouch club-shaped, near anterior end of segment. Vagina distended into prominent receptaculum seminis, in anterior part of segment; ovary bilobed, extending transversely just anterior to middle of segment; back of it the yolk-gland and between the two the "shell-gland"; uterus distended with ova occupies nearly the whole segment, obscuring the other parts (ova often free in

parenchyma of segment). Ova round or oval, double-walled; outer diameter averaging 40. microm., but variable (36. : 32. microm. to 56. : 42. microm.); inner wall showing measurements ranging from 20. : 18. to 32. : 24. microm.; at each pole of inner membrane a small protuberance from which spring a number of clear, refractile thréads which are distributed in a waving fashion through the substance intermediate to the outer and inner walls; within the egg a hexacanthous embryo slightly separated from the inner membrane.

This parasite, now regarded by most authorities as identical with *hymenolepis murina* of rats and mice, is an intestinal parasite of these animals and of man. It has a wide geographical distribution, being perhaps best known in Italy and neighboring parts of southern Europe. Recently a number of cases have been encountered in the eastern and southern states of this country, and there is some reason in the supposition that it will come to be regarded as one of the most common tapeworms of this portion of the world. It is most frequently met in children, especially in those of the poor and those living in poorly cared-for homes. The parasite inhabits the ileum, usually from the middle toward the ileo-cecal valve, and is commonly met in large numbers in the individual infested (sometimes a thousand or more.) The usual life-history is not finally established. For a long time certain cysticercoids met in the common meal-worm were suspected as representing the intermediate stage, but the evidence now held would refer these to another tapeworm; although it is not excluded that some such intermediate host may perhaps at times serve as a connecting link. However, it has been shown by Grassi that if the ova be fed to rats (best from one to three months old) the adult worm will develop in the intestine of the rat; and it has been found in such cases that the embryos having had the shell removed by the action of the upper digestive juices penetrate into the villi of the mucous membrane, and there become encysted as minute cysticercoids, which later drop into the intestinal lumen and develop the adult worms. This would suggest the possibility of direct multiplication within the intestine of the definitive host, but this is a mistake. The ova are not affected by the juices of the lower intestinal canal and are passed from the original host without change and for their further progression must gain access to a second suitable host (rat, mouse, man) or perhaps to the original host of reinfecting himself with ova from his own intestine. The means of convection to the second host probably include unfiltered water tainted with human or murine dejecta, food to which rats or mice have had access, or in case of man the fingers soiled with fecal matter after scratching about the anus.

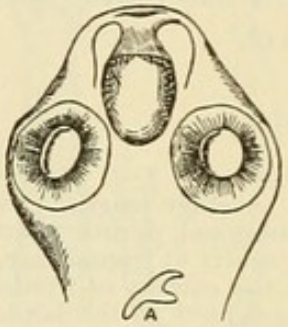


FIG. 50.—Head of *hymenolepis nana:* with rostellum retracted, ×75; *A*, an isolated hooklet, ×300. (*Gould, after Leuckart.*)



FIG. 51.—Ovum of *hymenolepis nana*, ×300. (*Gould, after Leuckart.*)

The symptoms caused by the parasite are of the same general nature as those induced by the common larger tapeworms; but in children, and when in large numbers, may be of marked severity and even terminate fatally. When present in but small numbers and in resistant individuals, the symptoms may be trivial and overlooked. In their severer manifestations, in addition to the intestinal discomfort and pain (sometimes severe) and

irregular diarrhea, there may be marked nutritive disturbances with more or less severe anemia; and the nervous phenomena, as epileptiform convulsions, may be a marked feature of the case, continuing for years until the parasites are gotten rid of or perhaps until death. The presence of the parasite being suspected, the diagnosis may readily be confirmed by the discovery of the ova by microscopic examination of the fecal matter, large numbers being usually found where the parasites are present in any marked degree of infection.

The treatment is much as in other types of tapeworm disease; but male fern has proved most satisfactory among the common anthelmintics (p. 211).

Subfamily: TÆNIINÆ; Genus: TÆNIA.

1. Tænia saginata (Goeze).

(*Tænia mediocanellata; t. inermis;* beef tapeworm.)

Average length, 3.–8. meters (in relaxed condition often reaching 10. meters), head tetragonal, pyriform, without hooklets or rostellum (in place of latter a central depression in center of frontal face, often slightly pigmented), with four cup-shaped suckers placed at the corners of frontal face, these provided with rather thick lips and often slightly pigmented at the borders; head measuring two millimeters in transverse diameter at frontal face; neck rather long and slender; first segments very short and broader than long; the segments increasing in their development so as to become much longer than broad (almost cord-like at posterior extremity) and when fully ripe (near posterior extremity) of long quadrate shape, 18.–20. mm. long and 5.–7. mm. broad; genital pores in adjacent segments irregularly alternating upon opposite margins and situated a little back of the middle of the margin of each; gravid uterus showing a median longitudinal trunk with lateral single or dichotomously branching and slender diverticula (25–35 on each side); embryophore ovoid, nearly spherical, slightly brownish, 30.–40. microm. long and 20.–30. microm. transversely, with thick radially striated shell and containing granular hexacanthous embryo.

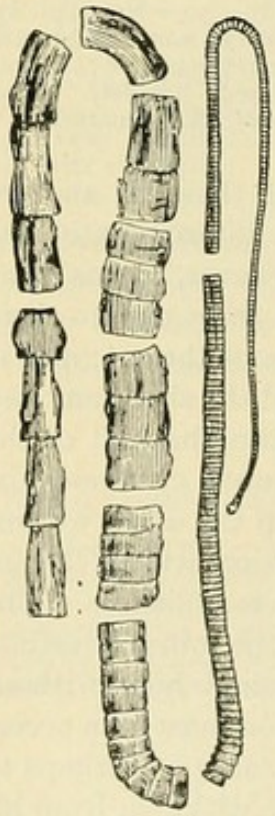


FIG. 52.—*Tænia saginata.* (*Gould, after Leuckart.*)

Habitat and Transmission.—This tapeworm, in its adult stage almost solely found in the small intestine of man, is widely disseminated over the world, especially in districts where beef is largely consumed, particularly when by habit of populace the flesh is not well preserved or thoroughly cooked. At present it is by far the most frequent tapeworm of man in this country, and is also the most common cestode of man in western Europe. It is a mistake to trust in the popular idea prevailing in the United States that of the large tapeworms met in man, most are derived from pork. The late Professor Leidy, to whom large numbers of worms were constantly being sent for opinion, informed the author of this work that all specimens sent him for examination during a period of 15 years were *tæniæ saginatæ;* and while in central Germany *tænia solium* has in previous periods been comparatively common, Heller has found in Holstein the beef tapeworm four times as prevalent as the pork worm.

The parasite is obtained from eating improperly cooked fresh beef containing the bladder-worm of the cestode. This, the *cysticerus bovis*, is a small spherical or ovoid vesicle, colorless and glistening, usually about half a centimeter in diameter (occasionally reaching or exceeding one

centimeter), and surrounded by a thin connective-tissue capsule formed at the expense of the host. After separating the bladder from this envelope one may note a small, opaque, whitish point on one side of the cyst. This is the invaginated head of the future tapeworm, which can be expelled from the cyst by careful pressure of the latter between the thumb and finger or may be caused to protrude by immersion in slightly warmed water; thus exposed, the head will be found to possess the characters of the future head of the adult tapeworm, with its neck faintly striated transversely (future segments) and with its posterior end distended into the cyst into which the head was invaginated. This bladder-worm because of its small size and small numbers (usually) is often missed in inspections of slaughtered beef; it may be found in any part of the striated muscular system and sometimes in the solid viscera, but Hertwig has pointed out its special frequence in the muscles of the masseteric region, in the internal and external pterygoid muscles. It develops from the ingested ovum apparently in a period of about three or four weeks; as a rule, is not of long life in this stage, as experimentally produced cysticercosis of but a few months' duration has repeatedly shown numbers of dead and calcified specimens in the muscles of the experiment animals. There is no doubt of the statement that transmission of the parasite to man follows only when the meat eaten is imperfectly cooked, as it has been shown that brief periods of exposure (several minutes) to temperatures of 45°–48° C. will kill the larval parasite, and it is safe to say that well-roasted meat (70° C.) cannot transmit the worm in vital condition. Moreover, while temporary drying of the flesh is by no means lethal to the bladder-worm (which while thus shrunken will again assume its vesicular appearance on soaking in water). Perroncito has found all cysticerci dead at the end of two weeks in a well dried and salted piece of veal. After eating infected beef the subject requires from *seven to ten weeks* before manifesting evidence of the presence of a fully developed *tænia saginata* by the passage from the anus of ripe segments of the worm.

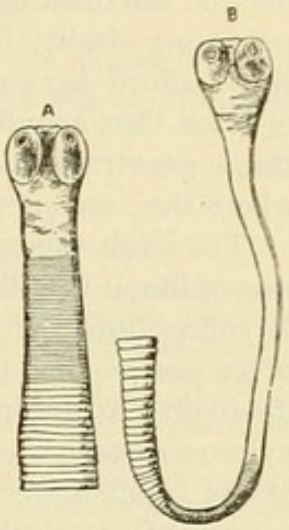


FIG. 53.—Head and neck of *tænia saginata:* A, retracted. B, extended. (*Gould, after Leuckart.*)

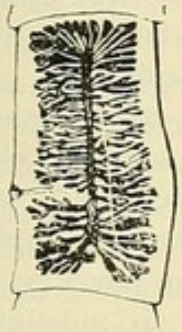


FIG. 54.—Proglottid of *tænia saginata.* ×2. (*Braun.*)

These separated segments, usually single, may be found in the dejecta or may be passed in the interval between stools; they were formerly regarded as special intestinal parasites and spoken of as "cucumber-worms," water-worms," etc.; they are yellowish-white in color, range up to about two centimeters in length, and are either distinctly quadrate and flat or are shrunken into nearly a cylindrical shape; they are usually quite active, having a mode of motion not unlike that of "measuring worms," and in this country among the ignorant there is a superstition that they will, if undisturbed, always crawl toward the nearest water (whence the name "water-worms"). Quite commonly they do gain access to water, but

only in a passive convection with ordinary drainage, and when immersed may retain their vitality and activity for a number of days. Ferdinand Herff, of San Antonio, speaks of having found a number of actively moving proglottids of this parasite in the water of a well into which surface drainage carried the contents of a cesspool. If deposited on a dry surface they soon die and are disintegrated and the ova are scattered over the herbage, these not losing vitality for some time because of the protection which their thick shells afford against drying and the elements. Cattle eating the blades of grass thus acquire the embryos within their alimentary tubes, and later these penetrate the intestinal walls and gain access to suitable parts, where they are encysted as larvæ or bladder-worms.

The adult tapeworm in the human intestine has an undetermined duration of life; it is well known to persist for years, and Railliet quotes Wawurch in connection with a case known to have passed segments over a period of 35 years. The parasite is found invariably in the small intestine when discovered at autopsy; usually the head is fixed a short distance below the pylorus among the villi, the strobile ranging loosely through the extent of the small bowel and only rarely found extending beyond the ileo-cecal valve. (Recently Fussell, Phila. Path. Soc. Repts., found several segments in the lumen of an appendix veriformis which was removed from a case with symptoms of appendicitis.) Although not provided with hooklets as is the pork tapeworm, it is apparently more firmly fixed than the latter to the mucous membrane.[1] Usually but a single example of the beef tapeworm exists in one host, but cases are reported furnishing as many as five or six.

But little difficulty can be experienced in attempting to distinguish between this worm and the *tænia solium*, with which alone it is likely to be confused at first. As a rule, the strobile is considerably larger and longer; the segments are larger and more active in their movements, and are more likely to be discharged from the anus in the intervals between the stools; the uterus in each link is more highly branched than in case of the tapeworm of the hog; the head is not provided with hooklets; the ova are somewhat larger than those of the latter and a little less spherical. In examining a link for study of the arrangement of the uterus, it is usual to compress it between two glass slides in a small amount of a 1 per cent. solution of caustic potash,or in a 20 per cent. solution of acetic acid.

Symptoms.—The symptoms occasioned by the parasite are partly local and partly of a reflex nervous type, and are both often so trivial as to pass unnoted. The former are generally of the nature of an indefinite abdominal discomfort, of weight and fullness, of occasional indistinct pain, usually most marked at meal-times, capricious appetite, irregular periods of diarrhea alternating with constipation, occasional nausea and vomiting, slow loss of flesh and strength, and eventually a moderate degree of anemia. The nervous phenomena are also very variable and indefinite, such as nasal pruritus, slight vertigo, choreiform twitching and occasionally epileptiform convulsions, visual disturbances and restless sleep. When the parasite is

[1] At least this is the opinion of Railliet, page 239 of his "Treatise on Medical Zoology," although it does not seem reasonable that the worm armed with suckers only should be more difficult to dislodge than the worm armed with suckers and hooklets. I cannot speak from my own experience, for I have never had a case of pork tapeworm.

suspected, the stools should be carefully watched for discharged proglottids and the patient instructed as to the appearance of these should they pass in the intervals from the anus upon the clothing. By microscopic examination occasionally the ova may be discovered, but probably the safer and almost as quick a recognition will be obtained by noting the links themselves as they appear.

The treatment of parasitism by this worm presents no special features differing from the treatment of tapeworms generally and will be discussed (p. 211) in the latter connection.

2. Tænia Solium (Leuckart).

(*Tænia cucurbitina; t. dentata; cystotænia solium;* pork tapeworm.)

DESCRIPTION.—Average length of strobile, 2.–3. meters, occasionally reaching twice this measurement; head more spherical than that of the *tænia mediocanellata*, but with a somewhat tetragonal shape given by the four rather prominent cup-like suckers with thick lips; head provided with a short thick rostellum bearing a double crown of hooklets (22–32 in number, usually 28); transverse diameter of head 0.6–1. mm.; neck rather thin and approaching one centimeter in length; about 800–900 proglottids; fairly developed links found unusually close to head; fully grown and ripe segments measure 10.–12. mm. long and 5.–6. mm. broad; regular alternation of genital pore on opposite margins of adjacent links, back of middle of margin; uterus consisting of a median longitudinal trunk with from seven to ten coarsely dendritic branches on each side; embryophore nearly spherical, light brownish, with thick radially marked shell and containing a granular hexacanthous embryo, measures 31.–36. microm.

Habitat and Transmission.—This parasite in its adult stage is practically limited to the small intestine of man in its occurrence. It is extremely rare in this country, popular impressions to the contrary notwithstanding; and is most frequently met with in Germany, France, Italy, and the British Islands. Because of the more careful meat inspection prevailing at present it is, however, decidedly less frequently met with than formerly. That it does occur occasionally among us is strongly indicated by the fact that the bladder-worm in pork is occasionally found in America; although the above statement as to its rarity is the experience of practically all American helminthologists. While the bladder-worm is occasionally encountered in other animals than the hog (and rarely in man), human beings obtain the intestinal parasites practically solely from eating improperly cooked pork containing the cysticerci. The encysted larva, known as *cysticercus cellulosæ*, is generally when found in pork encountered in large numbers, commonly in the muscles and especially in those of the shoulder, neck, tongue, diaphragm, and loins, but often in any part of the muscular system and sometimes in the solid viscera and even in the central nervous system. It is somewhat larger than that of the beef tapeworm, is a pale, shining cyst of ovoid shape, measuring from one-half to two centimeters in length, and from three to ten millimeters in transverse diameter; and as above described for cysticerci in general is surrounded by an additional thin connective-tissue wall derived from the surrounding tissues through inflammatory

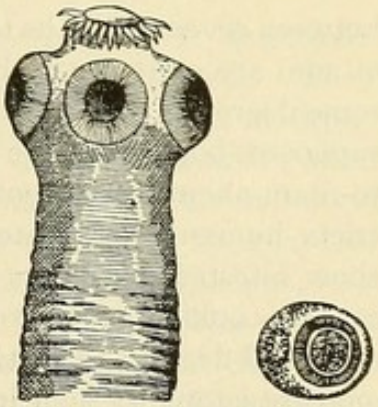


FIG. 55.—Head and neck, and ovum ×300, of *tænia solium*. Embryophore surroundedby vitellus. (*Gould, after Leuckart.*)

reaction. When picked out of such a situation there may be noted on one side of the cyst a small, slightly elevated spot of opaque white color, with a minute opening at the tip of the prominence. The white mass is the invaginated head, the tiny opening the outer end of the canal of invagination; and by careful pressure or by immersion in warm water the head may be caused to protrude from its bed through the opening referred to, showing the characters belonging to the scolex of the adult worm. The actual relationship between the cysticercus and the developed *tænia solium* has frequently been demonstrated by feeding experiments. In pork there are usually large numbers of these cysticerci, the muscles of favored parts being literally riddled with the bladder-worms; many of which are usually found in a shrunken and calcified condition (dead). The duration of life in this larval stage encysted in pork is unknown, but probably varies

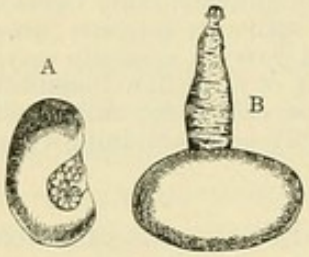


FIG. 56.—*Cysticercus cellulosæ:* A, with invaginated head, B, with evaginated head. (*Leuckart.*)

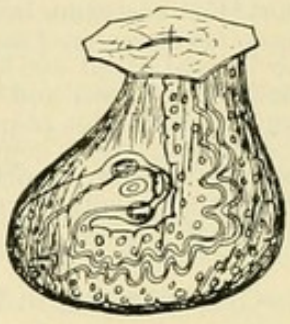


FIG. 57.—*Cysticerus cellulosæ*, ×12. (*Coplin and Bevan, after Leuckart.*)



FIG. 58.—*Cysticerus cellulosæ* after digestion of the bladder, ×10. (*Leuckart.*)

between several months to several years. Young hogs (less than six months of age) are more apt to be infested than older ones, which seem to possess some degree of immunity against the larval worms; and as would readily be supposed, hogs which are not carefully stalled and fed, but which are allowed to roam about, often rooting about manure-heaps (in which in country districts human fecal material is apt to be deposited), are most likely to show infested flesh. As in case of the *cysticercus bovis* above mentioned, careful cooking and prolonged and thorough salting and drying of the meat will destroy the vitality of the bladder-worms; but they seem slightly more resistant to such influences than the cysticerci of beef. About two and one-half months or over are required for the development of the adult worm after the infested pork has been devoured; after which the ripe links may be found in the stools of the patients. They are readily known from those of the beef tapeworm by their smaller size, their less active movement, and by the coarser and less highly branched appearance of the uterus as seen in compressed links cleared with acetic acid, caustic potash solution, or glycerin. The ova are less frequently encountered in the stools than those of the beef tapeworm, being more apt to be retained in the ripe segment until after its discharge and disintegration.

The habitat of the strobile in the human intestine, as seen in cases discovered at autopsy, is similar to that noted for the *tænia saginata;* usually but a single specimen is found (whene the name "solium," "the solitary worm"), but occasionally several are encountered in the single host.

The duration of its existence in the intestine is unknown, but instances of infested persons who passed links for years are common.

Treatment of Tapeworm Diseases.—There is, perhaps, no morbid condition which has brought more opprobrium upon the regular profession and more "grist to the mill" for advertisers and those who use secret remedies than tapeworm, and to our humiliation it must be said that these persons do seem to have more success in getting rid of tapeworm promptly than we do. There are, I think, two reasons why this is so. In the first place, it is certain that they do not use different remedies from those commonly in use by the profession, but they give larger doses. In the second place, they see a larger number of cases and develop a sort of specialty which, like all specialties, produces greater skill in treatment. In order that a tapeworm may be successfully removed it is necessary that it shall be of a certain size; so that, if a large part of the worm has been brought away by medicine, it is useless to give anything more until the remaining part increases sufficiently in size.

It is sometimes useful to know the exact course pursued in a given successful case. Thus, in such a case the patient was fasted for 29 hours. Twelve hours after fasting began he was given one ounce of castor oil. Twenty-four hours after fasting began he was given 1 1/2 drams (5.55 c.c.) of oleoresin of male fern. In 5 hours more he was given another ounce of oil. The worm came away entire in a mass.

There are half a dozen remedies for tapeworm, and they are all good. The two best are probably the ethereal extract of male fern and kousso flowers. Some prefer the first of these, while others prefer the second. The ethereal extract of male fern is prefered by us.

Patients require some preparation before any remedy is employed. In all cases they should be kept absolutely quiet during treatment. They should eat nothing from breakfast time of one day until the next morning, during which time the bowels should be moved by a saline cathartic; when 1 dram (4 c.c.) or 2 drams (8 c.c.) of the extract of male fern either in several capsules or in an electuary is to be taken. If at the end of six hours no movement of the bowels has taken place, a promptly acting aperient, as a dose of oil, compound jalap powder, or elatrium, is taken. The worm is usually discharged entire. Of course, one is never certain that this is the case unless the head is found. At the same time, it does not follow because the head cannot be found that it has not been passed, for it is very small, and may be lost in the discharges. In the *tænia solium* the head is about the size of a small pin's head; in the mediocanellata it is a little larger, and in the bothriocephalus it is still larger. If the head has not been removed, it is certain that in from 10 to 16 weeks the worm will grow out again and begin to discharge links.

The third remedy, in order of efficiency, is the bark of the root of the pomegranate. This has been given in the shape of a decoction, from 2 to 4 ounces (60 to 120 c.c.) to the pint (0.5 liter). Boil the bark half an hour, strain, and drink. The fluid extract is more convenient in the dose of from 45 minims to 2 fluidrams (3 to 8 c.c.). Two hours later a purgative should be given. An alkaloid is obtained from pomegranate, named pelletierine, in honor of the chemist, Pelletier. This is given in a

single dose from 8 to 25 grains (0.5 to 1.6 gm.). When first introduced it was vaunted as a "sure cure," but the experience of practitioners has not been uniform, and success has been by no means invariable. I have been successful with it.

Kamala, the hair of the *rottlera tinctoria*, is said to be very efficient in tapeworm and may be used. It is given in doses of from 1 to 2 drams (4 to 8 gm.) suspended in syrup, repeated in from eight to ten hours if it does not purge. The fluid extract is also given in doses of 1/2 a dram to 1 dram (2 to 4 c.c.). It is purgative, sometimes drastically so. It may also cause nausea and vomiting.

Another efficient remedy is the oil of turpentine. It is, however, apt to produce symptoms so unpleasant that it should be last used. The dose is from 1 ounce to 2 ounces (30 to 60 c.c.), mixed with twice that amount of castor oil—a horrid dose; but if others fail, it may be tried.

Still another is pumpkin-seed. There are two ways in which it may be given. Three or 4 ounces (30 to 120 gm.) of the seeds may be crushed in a mortar with water, then strained, and the emulsion taken fasting, after a day's dieting. A few hours later a brisk purge should be taken. Second, the seeds may be made into an electuary which is almost as pleasant as sugar candy, and often is about as effectual. These different remedies in the order of their efficiency are about as follows: male fern, kusso, pomegranate, kamala, turpentine, and lastly, pumpkin-seed.

Combinations are sometimes very efficient. The following is recommended by Strümpell:

℞	Granati corticis radicis,	℥ iv–v (120 to 150 gm.)
	Aquæ,	Oij (1000 c.c.)
	Macerate for 24 hours and boil until it is reduced to five fluid-ounces (150 c.c.).	
	Add:	
	Oleoresinæ filicis,	gr. lxxv (5 gm.).

The whole amount is to be taken in three or four doses as close together as possible.

Thymol, in doses of 10 grains (0.66 gm.) three times a day in a wafer, has been recommended. Another method is to give five grains (0.33 gm.) every hour with or without preparation. Papain, juice of *carica papaya*, is given in doses of from 1 to 10 grains (0.066 to 0.66 gm.). If thymol is given the purgative should *not be oil* but some saline.

Prophylaxis is of the greatest importance. Great attention should be paid to the cooking of meats, especially of large joints, in order that they may be thoroughly "done." Rare meats should not be eaten.

Family: ANGIOSTOMIDÆ;
Genus: *Strongyloides.*

Strongyloides intestinalis (Grassi).

(*Anguillula intestinalis* et *stercoralis; leptodera intestinalis* et *stercoralis; pseudorhabditis stercoralis; rhabdonema strongyloides; rhabdonema intestinale.*)

Living as two different (heterogonous) generations; the first diöic and free; the second parasitic as parthenogenetic females. *The parasitic form lives in the upper intestinal tract of man;* 2.5 mm. long; cylindrical, with pointed tail end; smooth cuticle; simple mouth with four (3?) lips; long, slender cylindrical esophagus, reaching one-quarter of the length of the worm; anus close to tail; vulva at posterior third; containing yellowish-green oval ova (50.–58. : 30.–34. microm.); larvæ develop in intestine (at first 200.–240.

microm., in length, but increase to two or three times this length) and are passed in the fecal material. The larvæ differ essentially from the parent in having an esophagus with two bulbs (rhabditiform). In the discharged feces at suitable temperature (about 30° C.) these develop with one moulting of the cuticle to a free-living generation with separate sexes (at lower temperature are apt to remain at least in part asexual). In this free sexual generation the worms are smooth, cylindrical, and tapering, with pointed tail end; mouth as in parasitic form; esophagus rhabditiform (two bulbs) with its anterior portion long and with the posterior pyriform and containing a Y-shaped chitinous armature; anus at base of tail; male with tail curved and two spicules, body length 0.7 mm.; female 1. mm. long, with straight pointed tail, vulva a little back of middle; ova few, yellowish, ellipsoid, thin-shelled, 70. : 45. microm., sometimes hatching in uterus. The larvæ of this generation look much as their free parents, are at first 0.22 mm. in length, but grow to 0.55 mm., then moult and assume a filariform or strongyloid character like that of the parasitic grandparent. In unknown manner these gain access to the intestine of a new host or shortly die.

The above outline follows that known for the tropical *strongyloides;* it is said that in the European examples the intermediate generation with separate sexes fails, that the rhabditiform larvæ of the parasitic worms pass with moulting to the condition of the strongyloid larvæ and these, if introduced into the intestine of the next host, directly develop into the parasitic females as above.

The worm is commonly spoken of as *strongyloides intestinalis* when one refers to the parasitic parthenogenetic females and their larval offspring; as *strongyloides stercoralis* in the free form having separate sexes. The parasite was first met in the stools of persons suffering from Cochin-China diarrhea, and was supposed to be the cause of this affection; and at one time, too, was suspected of being in causal relation to Asiatic cholera. Both these views are erroneous, the worm probably having no direct pathological significance; although perhaps, when present in large numbers, it is capable of aiding in keeping up the intestinal irritation of a diarrheal affection caused by some other original influence. The parthenogenetic females are found in the upper part of the small intestine, burrowed in the crypts or a little way in the mucous membrane. Here they deposit their ova. These retained in the crypts or between the folds rapidly incu-

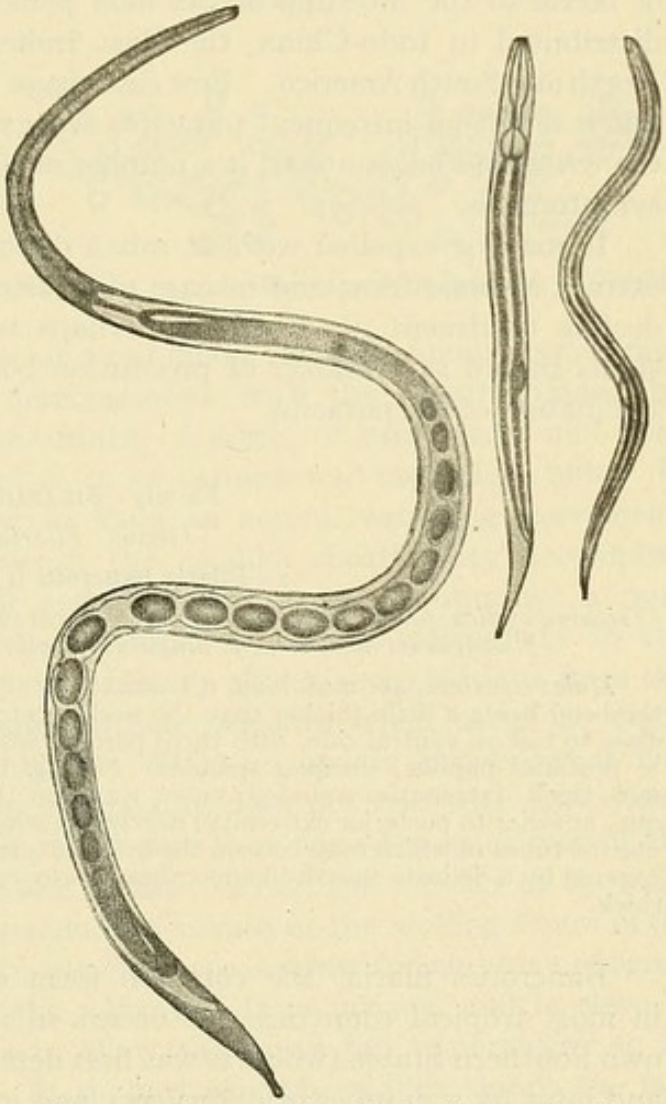


FIG. 59.—*Strongyloides intestinalis:* on the left a gravid female from human intestine (natural size, 2.5 mm.). In the middle a rhabditiform larva from fresh fecal matter, ×120; to the right a filariform larva from culture, ×120. (*Braun.*)

bate and give origin to the larvæ found in the stools. These are usually found in great numbers, scarcely a bit of the fecal matter but contains at least two or three of the young worms in ordinary cases. They are readily observed with low powers of magnification in thin layers of the fecal matter, actively wriggling. In autopsies there are occasionally found a few sexually developed examples of the intermediate generation (*s. stercoralis*) in the intestine of the cadaver; but it is probable that these develop after the death of the host from the intestinal larvæ in the same manner that they usually follow after ordinary discharge to the exterior.

Nothing is known as to the mode of transmission to the second host, but presumably unfiltered water or unclean vegetables (uncooked) which may have been grown in soil watered with fecal infusions (as is rather common in China, Japan, and elsewhere) may bear the second generation of larvæ to the intestine of the next person. The worm is found widely distributed in Indo-China, the East Indies, Africa, Europe, and in both North and South America. Few cases have been reported from this country, but it is not an infrequent parasite, at least in our Southern States, where the writer has encountered it a number of times, invariably without notable symptoms.

It may be expelled without much difficulty by the use of the ethereal extract of male fern; and in case of infested persons who for some reason decline treatment, or in whom perhaps treatment temporarily fails, the stools should as a matter of precaution be disinfected to prevent the dissemination of the parasite.

Family: FILARIIDÆ;

Genus: *Filaria*.

1—Filaria bancrofti (Cobbold).

(*Trichina cystica; filaria sanguinis hominis; filaria sanguinis hominis ægyptica; f. wuchereri; f. sanguinis hominum; f. sanguinis hominis nocturna; f. nocturna.*)

Male: colorless; 40. mm. long, 0.1 mm. thick; filiform; anterior end slightly clubbed, the head being a little thicker than the neck; posterior end curved, but not spiral; anus close to tail on ventral side, with three pairs of small preanal papillæ and same number of postanal papillæ; unequal spicules. *Female:* brownish; 76.–80. mm. long, 0. 2–0.3 mm. thick; extremities rounded; vulva 1.27 mm. back of anterior extremity, anus 0.28 mm. anterior to posterior extremity; nearly the whole of the body is occupied by the two uterine tubes in which may be seen the ova and already developed larval filariæ. *Larvæ;* covered by a delicate sheath-like membrane, 130.–300. microm. in length, 7.–11. microm. thick.

Bancroft's filaria, the common form of human blood filaria, is met in most tropical countries. It occurs in the West Indian islands, in our own Southern States (where it was first demonstrated by Dr. John Guitéras, and later by a number of observers), and in South America. It is encountered frequently in India, China, and Japan, in the East Indian islands and the South Sea islands, in Australia, commonly in Africa, and has even been met in southern Europe (Spain). The worm was first known only as the larva found in the circulating blood, no recognition being made of the species *f. diurna*, *f. perstans* and others later described as separate species by Manson. From the fact that these larvæ were met in the blood of infested individuals, the name *filaria sanguinis hominis* was first attached; but later, when the adults were recognized, it was found that the proper

habitat of the worm in man is in one of the lymph passages, usually one of the large lymph vessels of the trunk, as in the groins, the pelvis, or thoracic duct; although as errant forms they are also to be found in subcutaneous lymph vessels, and even in the heart and blood-vessels. In such situation are usually found two worms, male and female, intimately coiled together and probably living thus for long periods and producing their larvæ. The female generally gives birth to the larvæ (occasionally ova), in large numbers, each inclosed in a so-called "sheath" (the shell-membrane). These are carried along the lymph vessels by the current and eventually are poured into the blood-vessels, each larva being probably capable of several months of life in the blood. The establishment of a positive diagnosis of filariasis is made by the discovery of these larvæ in the blood of the subject examined. They are readily detected with ordinary laboratory powers of the microscope in thin moist films of the fresh blood made just as in examination for malarial parasites, each larva being about as thick as the diameter of a red blood cell, and about forty times as long. They are nicely demonstrated with the sheath stained by drying a film of blood over the fumes of acetic or osmic acid and then staining with hematoxylin and eosin or carmine and methylene blue. In the fresh blood they are seen to have an active, wriggling movement, but because of their inclosure in the sac-like sheath they accomplish but little progression by their activity. They may be obtained at any hour from the interior blood; but show a peculiar periodicity in the peripheral blood, that drawn as in ordinary clinical examinations from the subcutaneous tissues only showing their presence when taken at night (best between dusk and midnight). While no certain knowledge is had explanatory of this periodicity it probably does not rest with periodicity in the life of the worm, but is rather due to a variation in caliber of the peripheral capillaries at night and in day. From the tonicity of the walls of the capillaries and their surrounding tissues in the waking hours of the subject, probably the capillary lumen is too narrow for the easy progression of these larvæ; while in the relaxation from fatigue, and in sleep, it may become sufficiently wide to allow the worm fair opportunity to be borne along with the blood. In support of such an idea stands the fact that if the habits of the infested individual be changed so that he sleep in day and be awake and active at night, after a short period of indifference the larvæ are to be found in the peripheral blood only in the day. While in the blood the larvæ retain their sheaths and are only passively carried in the blood stream; were it otherwise they could probably actively progress at any hour through the finest blood channels, and could in all likelihood be found in the peripheral blood at all hours, and perhaps might penetrate the vessel walls and tissues of the host. It has repeatedly been

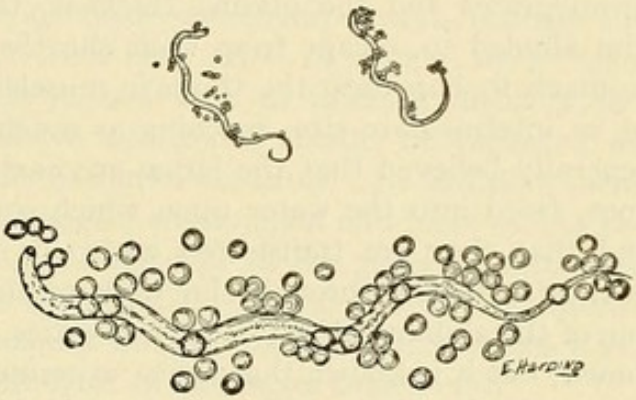


FIG. 60.—Larval *filaria bancrofti* in blood. (*Coplin.*)

noted that in blood preparations after the cells disintegrate and give off their hemoglobin, the plasma thus becoming thicker (although not coagulated), the larvæ are able by their active movements to break through the sheath (which is somewhat fixed by the denser plasma) and then are actively progressive, each showing on close examination a small boring apparatus at the head end.

The further life-history of these larvæ is not certainly established; but it is known that at least some are removed from the infested subject by mosquitoes of the genus *culex* in withdrawal of blood by these insects. It has been observed that in the stomach of the mosquito, as the blood disintegrates and the plasma thickens, the larvæ, just as in the blood-film alluded to, escape from their sheaths and penetrate the walls of the stomach to bore into the thoracic muscles of the gnat. Here they grow to an intermediate size, reaching as much as 1.5 mm. in length. It was generally believed that the larvæ are next, with the death of the infested gnat, freed into the water upon which commonly the mosquito has died, and that they are transferred with this to the alimentary canal of the next host, boring through the walls of the digestive tube and attaining one of the abdominal or pelvic lymphatics. This idea is by no means abandoned; but it is known that larvæ experimentally kept in water die in the course of a few days, and attempts to infect monkeys with water containing the larvæ have failed. On the other hand, it is well established that the larvæ after growth in the muscles of the gnat, may travel and get into the proboscis; and it is thought quite possible that if the culex at such times should bite a fresh human subject these larvæ may readily gain access to the wound and thus enter the new host. Strong analogy exists in the established similar mode of transmission of *f. immitis* of the dog by mosquitoes shown by Noé; and the present attitude favors this view.

Pathology and Symptomatology.—The symptoms of filariasis, aside from the presence of the larvæ in the blood, vary much in individual cases. It is well known that the infested persons, with numerous larval filariæ in their blood, may for years show no symptoms and be apparently in excellent health. Doubtless in such cases the parent worms are located in some portion of the lymphatic circulation which they do not occlude (as the receptaculum chyli) or in such part where free anastomosis prevents serious fault in the lymph flow. If, however, the parasites should occasion obstruction and cause stoppage of the lymph circulation, then in a limited or extensive portion of the body, according to the lymphatic area affected, there follows dilatation of the lymph vessels. This may result in rupture of the distended vessels and lymph edema; or a lymph fistula may develop to the external surface of the body or into one of the body cavities or hollow viscera. Not infrequently such fistula opens into a ureter or the urinary bladder, the urine becoming milky from the lymph admixture (chyluria); and at times in the development of the fistula some of the small blood-vessels may also be broken into and blood is added (hematochyluria). The distention of the lymphatics may involve the lymph glands; thus those of the inguinal region sometimes form tumor-like masses of a peculiar boggy quality ("varicose glands"), giving on palpation the sensation of interior small solid areas, occasionally growing to half the size of a fist, and re-

quiring to be differentiated from hernia. Along with such conditions the tissues about the dilated lymph vessels and passages become hyperplastic, especially in the skin, where the sometimes enormous thickening of the corium known as elephantiasis (the possibility of a combined or secondary bacterial infection in the production of which should be held in mind) is occasioned. This last is usually met in the skin of the lower members and about the genitals, but is occasionally seen elsewhere. These changes are essentially permanent, and for this reason not every case showing elephantiasis, lymph edema, varicose glands, lymph-fistula, or chyluria need necessarily show the presence of the filarial larvæ in the blood, all of the parent and larval parasites having perhaps died—moreover, lymph obstruction with any of the above secondary results may arise, of course, from other than parasite cause; yet it is safe to say that in infested districts the majority of such conditions as above mentioned should be regarded as due to existing or previous presence of filarial parasites. In addition, there are likely to develop some blood changes, eosinophilia and more or less reduction in the number of red cells, enlargement of the general lymphatics and of the spleen, some indefinite febrile disturbances, more or less alteration of nutrition, together with possible inflammatory changes of the peritoneum, bladder, pelvis of the kidney or of the latter organ itself.

The mechanism of the obstructive phenomena and their results are thus outlined by Manson:

"A parent filaria is lodged in the left thoracic duct. In some way not yet understood it injures the walls of the vessel, causing ulceration or inflammatory thickening. In time this lesion leads to stenosis of the *duct*. *Pari passu* with the development of the stenosis the thoracic duct on the distal side of the stricture dilates owing to the rising lymph. After a time the stricture becomes so narrow that the lymph and chyle no longer find their way past it to the left subclavian vein. They seek, however, to reach the blood by another route; a retrograde movement down the thoracic duct sets in, and so, by way of the pelvic lymphatics in the walls of the abdomen and the anastomosis between these and the lymphatics of the upper part of the body, the chyle from the intestines and the lymph from the lower extremities find their way into the circulation by the right thoracic duct. Possibly there are other routes, as by the lymphatics of the esophagus, diaphragm, and back. It is certain, however, that a frequent course pursued is that described, which is much the same as that pursued by the blood in the case of observed portal circulation. To accommodate this diverted chyle and lymph, the lymphatics by which they pass become enlarged and in many places varicose. The tendency to varicosity is very evident in such places as the scrotum, mucous membrane of the bladder, or wherever the lymphatics are abundant and feebly supported. In many instances these varices, when superficial, can be seen or felt and their nature readily recognized. If the inguino-femoral glands are involved, the varicose groin glands, so characteristic of filaria infection, are produced. Sometimes the varix is apparent on the surface of the abdomen even, as in a case related by Sir William Roberts and in another by Havelhing. That these varices are really part of an anastomosis conveying chyle from the abdominal viscera to the blood is proved by the nature of their contents, which are usually milky-white or slightly red-tinted chyle—not clear and limpid lymph, such as comes from the legs. As the lacteals are the only source of chyle, these chylous contents of the varicose lymphatics must have come from that source, and the route followed must have been the retrograde one described. Now, if the lymphatics of the bladder happen to be involved in the compensatory anastomosis, and if they give way, as the lymphatics of the scrotum so frequently do in similar circumstances, the result is a leakage of chyle in the bladder, and chyluria. It is evident from this that the embryo filariæ, although they are generally present in the blood and the urine in chyluria, have nothing whatever to do with its production. This is further proved by the fact that in some few cases of genuine and persistent tropical chyluria no embryo filaria can be found either in blood or urine. Proper treatment of chyluria is in principle the same as the treatment of acquired varix in any accessible region. This should consist of rest, elevation, lowering of the tension in the lymphatic vessels by the use of saline purgatives limited and appropriate food, and abstinence from fluids as much as possible. Certain drugs have been vaunted as specifics for chyluria. Temporary recovery from time to time is the rule, and the drug which was being used at the time the urine cleared spontaneously from the healing of the rupture in the varix of the bladder is often credited

with the cure. I cannot understand how a drug introduced by the mouth can possibly cause the closure of a gaping varix in the bladder."

Besides *f. bancrofti* reference may be made at this point to the following species of filariæ, the larvæ of which are to be met in the blood and liable to be confused with those of the ordinary form of human blood filariæ:

2—*Filaria diurna* (*f. sanguinis hominis*, var. *major*): a larval filaria found by Manson in the blood of negroes in West Africa, and differentiated by this observer from the larvæ of *f. bancrofti* by its presence in the peripheral blood in the day rather than at night, and by the fact that its intestine is not as granular as that of *f. bancrofti*. It is of the same size and general appearance as the latter, however. Manson has suggested this as the larval form of *f. loa*. In the few cases in which it was seen it presented no special symptomatology. It seems quite possible that this species is identical with *f. bancrofti*, the time of appearance in the peripheral blood being perhaps dependent rather upon conditions of the host than of the parasite, the less granular condition of the intestine being scarcely enough to justify the idea of specific difference.

3—*Filaria perstans* (*f. sanguinis hominis*, var *minor*): This form was met as the larva by Manson in the blood of negroes along the west coast of Africa and by him erected into a separate species. It is distinguished from the ordinary filariæ of human blood by the fact that it is present in the peripheral blood at all times, without diurnal or nocturnal periodicity; is smaller; as seen in the blood has no sheath and is actively progressive; has its posterior end truncated and abruptly rounded; and the boring spicule at the head end is more prominent than that of *f. bancrofti*. The adult parasites, subsequently recognized, inhabit the mesenteric and retroperitoneal tissues. The parasite is apparently of little or no pathogenic importance, as the hosts present no symptoms or important lesions thus far referable to the worms or their embryos. The male reaches a length of 45. mm. and is 0.06 mm. in thickness; the female is about twice as long and as thick as the male. The head end is rounded. The tail of the male is curled, and is marked by by four pairs of preanal and one pair of postanal papillæ; spicules unequal; a pair of small cuticular appendages at the tip of the tail. Vagina 0.6 mm. from the head end; tail with cuticular appendages at tip as in male; one anal papilla.

The intermediate hosts are as yet unknown.

6—*Filaria magalhæsi:* At autopsy in the heart of a child in Rio Janeiro Magalhæs found two sexually mature filarial worms, male and female, to which the above name has been applied. *Male:* 83. mm. long and from 0.28 to 0.4 mm. thick, with thick finely transversely striated cuticle, rounded head end without papillæ, posterior end with double curl and four large pairs of preanal and of postanal papillæ, with one (probably two unequal) spicule, anus 0.11 mm. anterior to tail, mouth round and unarmed. *Female:* 155. mm. long, 0.6–0.8 mm. thick, with cuticle a little more coarsely striated than in male; head-end as in male; tail slender, ending buntly; vulva 2.5 mm. from anterior end; anus 0.13 mm. in front of tail; two ovaries, with the ova contained measuring 38. microm. long and 14. microm. wide, the larvæ 300.–350. microm. in length.

Treatment of Filariasis.—There are no established remedial measures in case of these forms of filarial infection. Thymol has been lauded as almost a specific and has at times seemed to have given excellent results, but in many other cases it has been apparently of no value. So, too, benzoic acid and benzoate of soda have been recommended for the destruction of the parasites, but are of not more promise than the thymol. At best treatment has most to deal with the effects of the parasites in the way of the elephantiasis, lymph edema, fistulæ, chyluria, etc., and of course, the above drugs can have no value in such relation. Such conditions must be met individually and symptomatically. Elephantiasis is at times to be dealt with surgically, as in case of elephantiasis of such restricted regions as the scrotum, or of the female genitalia. It may be dealt with in cases of general involvement of the skin of the limbs by elevation of the member, application of pressure by bandage from foot upward, and perhaps some value may be realized from the internal administration of iodid of potash. Inasmuch as there is reason to believe that the dermal thickening of elephantiasis is, at least in the early stages of the inflammation, in part due to associated infection of the skin by

various common bacteria of the surface of the body, there is reason in employing internal antiseptics (as methylene blue or ichthyol) or applications of ichthyol or other antiseptics to the surface beneath the bandages advised. Treatment of chyluria or hematochyluria demands rest in the recumbent position, the lowering of lymphatic tension by saline purgatives, appropriate food, and limitation of fluids; such measures being appropriate also in connection with the pressure treatment of elephantiasis and in that of all of the mechanically induced lesions.

As measures of prophylaxis the careful screening of infested individuals from mosquitoes, particularly at night, all efforts to destroy mosquitoes and prevent their breeding and entering human habitations, and the careful filtration and boiling of water used by inhabitants of infested districts (in recognition of the possibility of correctness of the old theory of water transference of the larvæ) should all be practised—such measures much more than attempted remedy of existing cases holding out promise of valuable results.

7—**Filaria medinensis** (Linneus).

(*Vena medinensis; dracunculus Persarum; gordius medinensis; filaria dracunculus; f. æthiopica; dracunculus medinensis*, Guinea-worm.)

Only the female certainly known; whitish or yellowish; 50.–80. cm. or more in length, 0.5–1.7 mm. thick; cylindrical; anterior end rounded; oral orifice terminal; small, with two lips back of which are two lateral and four submedian papillæ, posterior extremity curved into a hook and terminating in a blunt point; intestine missing (probably atrophied by pressure of gravid uterus) up to esophagus; vulva and vaginal tube not recognized; nearly the whole body occupied by the double uterus full of larvæ and ova in various stages of development. Males probably represented by the small worms found by Charles in an autopsy on a native in Lahore, in whom in the subperitoneal tissue he encountered two female Guinea-worms each having a small worm about four centimeters in length attached about 14 centimeters back of the head end; it is from this believed that males are much smaller than females and that after coition they perish and are lost. *Larvæ* in uterus measure 0.5–0.75 mm. in length, slight tapering toward the head extremity and gradually tapering from the middle to a long fine straight posterior extremity, finely striated transversely, somewhat flattened laterally; escaping to the exterior of host through the mouth of the parent by rupture of uterine sac when the mother worm comes to the surface of the body of the infested individual.

The Guinea-worm, known of old as the "fiery serpent" of the children of Israel in their wandering through the wilderness and mentioned in the ancient writings of other races, occurs in India, Persia, Turkestan, in Egypt, and in fact all through tropical Africa, especially on the west coast. It has been introduced with negroes from Africa into this country, but has not taken permanent hold in the western hemisphere save in a few isolated localities in South America. It has been found occasionally in cattle, horses, the dog, jackal, leopard, and wild cat, but it is most common in man. It occurs without reference to race or age, is more common among males, but probably only because of some difference in exposure from habits; and generally is most frequently seen during the wet season and in the succeeding hot months of the year. Its usual habitat in the host is in the subcutaneous tissue of the lower extremities, down near the ankles, but it has been found in the trunk, in the face, and about the eyes and in the tongue, and elsewhere. It does not remain in one locality, but wanders through the tissue, usually causing some minor itching as it travels; and after maturation, which is reached after some months or a year or more after entrance of the larval worm, it coils itself in some locality, as about the ankles, and there

occasions a red and painful tumefaction. Probably, as above suggested, in the original infestment both a male and a female larva enter the host; after coition it is thought the male dies and is absorbed and the female continues to grow, developing an enormous number of larvæ in the uterus, pressure from the distended uterine sac causing the atrophy of the other structures internally and being largely responsible for the size of the mature worm. After several days the swelling, which may be as big as a pigeon's egg, becomes vesicular at the top and breaks down into an ulcer. When moisture is applied over this ulcerated tip (either water purposely dripped upon it or incidentally applied when the host is wading) the worm seems to be stimulated to discharge the larvæ, and the head end is slightly protruded through a small opening in the midst of the ulcer. The uterine sac seems to be forced through the oral orifice, breaks, and from it escapes a milky fluid abounding in actively moving larvæ. Some days may elapse before the entire discharge of the latter, the worm coincidentally shriveling and eventually spontaneously passing out from the tissue.

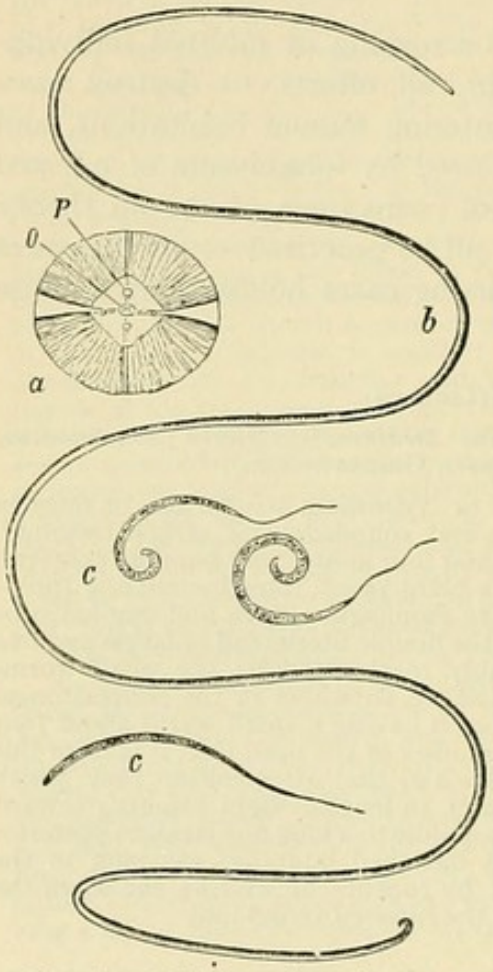


FIG. 61.—*Filaria medinensis*: *a*, anterior extremity; *O*, mouth; *P*, papillæ; *b*, female, reduced to less than half normal adult size; *c*, larvæ, enlarged. (*Braun, after Claus.*)

Nothing is really certainly known as to the life-history of the larvæ. It has been definitely shown that they may be directly passed with the water into which they have escaped to the next host and reach full development in such individual; but whether this is the rule is not established. Manson and others believe that, having escaped from the host into water, they enter the bodies of certain minute water arthropods (*cyclops*), therein moulting several times, becoming cylindrical, losing the delicate tail, and developing a small tripartite tail appendage. Whether the larvæ enter the next human host ordinarily with the water into the intestine (either free or inclosed in the arthropods), or whether they penetrate the skin of some individual who may be wading in water containing them, is not known. It is known that in clean water they are not of long life, dying in five or six days; in muddy water they live for several months as free-swimming larvæ; and when parasitic in cyclops are still more enduring.

There is rarely more than one parasite of the species in one host, although there have been placed on record as many as 50. The symptoms occasioned are practically entirely localized to the place of escape of the worm and under conditions of cleanliness after the removal of the parasite entirely disappear. Occasionally, in the height of the local inflammation, there may be a slight febrile general state induced, but this is not of importance; however,

should the worm be broken in an endeavor to drag it out, suppuration and more or less septic absorption with general symptoms may ensue.

Treatment.—Attempts to destroy the worm before its maturation and pointing are practically useless with means at present known; and the treatment is limited to the safe withdrawal of the worm and antiseptic dressing of the sore occasioned. The natives are in the habit of allowing water to drip over the ulcerated surface to cause the protrusion of the head of the worm; this is then fastened to a small stick, which is bound loosely to the surface by a well moistened bandage. From time to time several times daily, a turn is taken upon the stick, thus winding the protruding part upon it until finally the entire length is safely and without breakage withdrawn. A more excellent method has been suggested by a French surgeon, Emily, who injects with a fine hypodermic needle a little corrosive sublimate solution into the head end of the worm, which kills the parasite and allows it to be more readily withdrawn. Or if this cannot be done he would inject a small amount of the bichlorid solution into the swollen tissue about the worm, which is also likely to destroy the worm, and then cuts down upon it and carefully extracts it. Faulkner has said that he has been able to expel the worm entire within an hour by applying one pole of the constant current over the tumefied area.

It is impossible to be specific as to prophylaxis; but at least with our present knowledge it is advisable to refrain from the use of water for drinking which has not been boiled or well filtered, and lest perhaps the larvæ enter the host through the skin it would be well to refrain from wading in muddy water in countries where the parasite is known to exist.

8—**Filaria loa** (Guyot).

(*Filaria oculi; dracunculus oculi; d. loa; f. subconjunctivalis; f. lachrymalis.*)

Male: length, 20.–30. mm.; width, 0 3–0.4 mm. whitish or yellowish; cuticle not striated but, except at extremities, beset with numerous irregularly placed protuberances; anterior end as a truncated cone; at base of cone, dorsally and ventrally, a small papilla; posterior end slightly curved ventrally, pointed; mouth unarmed; anus 82. microm. in front of tail; three pairs of prominent preanal papillæ, two pairs of smaller postanal papillæ; two unequal sexual spicules. *Female:* 30 –40. mm. or more long, 0.5 mm. thick; surface and anterior end as in male; posterior end straight and tapering, round at tip; vulva at end of first fourth of body; double uterine tube nearly filling the body, the ends of tubes as ovaries; the uterus filled with ova and larvæ (253.–262. microm. in length) with rounded head ends and long pointed tails.

This worm is, as far as now known, confined to Western Africa; it has been known in America, having been conveyed in negro slaves to this country, but has never become permanent here. The adults live and wander through the subcutaneous tissues, especially about the face, nose, and eyes, and have been especially found in the subconjunctival tissue. Crawling in the skin, the worms cause considerable itching and burning pain; and in the eyelids and conjunctivæ induce more or less troublesome inflammation and swelling. The adults are known to persist for months or even several years in these situations in the host; but, as a rule, there are only a few present in one individual. The mode of escape of the larvæ from the host and their subsequent history are unknown. The treatment is limited to the removal of the parasite by surgical methods, as the clipping of an opening in the conjunctiva and withdrawal of the worm cautiously by means of suitable forceps.

Family: TRICHOTRACHELIDÆ;
Genus: *Trichiuris.*

Trichiuris trichiura (Leuckart).

(*Ascaris trichiura; trichocephalus trichiurus; trichocephalus hominis; trichocephalus dispar;* whip-worm.)

Male: 35.–45. mm. long; whitish; anterior three-fifths slender and thread-like; posterior two-fifths thicker, cylindrical, terminally rounded and curled; anus terminal; single spicule in a tubular sheath containing small spinules. *Female:* 35.–50. mm. long; shape as in male for front and body; posterior extremity straight, bluntly pointed terminally; vulva at beginning of thick posterior portion of body; ova brown, oval, thick-walled with a colorless shining button-like protuberance at each pole (50.–54. microm. long; 23. microm. broad).

This parasite, commonly known as the "whip-worm" because of the shape (the anterior filiform end suggesting the lash, the posterior thicker part the handle of the whip), is a very common and widely distributed parasite of man, finding its habitat in the large intestine, where the worms are found adhering to the wall by the anterior ends, which are buried a short distance in the tissues of the mucous membrane. It is one of the most common intestinal parasites in this country, although but little attention is given it and few records are to be had. The ova are discharged with the fecal matter from the intestine, and in water or moist earth the embryo develops within the shell, but does not escape to free larval life. The thick shell affords considerable protection and the embryo may live for months before destruction, thus incased. Probably with water or food the developed ova are introduced into the intestine of the next host, where in about a month or less the fully matured adult worms will be found. The parasite is of little pathological importance; there are not often more than a dozen present in one host, and apparently they do but little damage and practically never give rise to appreciable symptoms of their presence. Doubtless some little irritation and a very slight loss of blood from the lesions occasioned in the mucous membrane of the cecum and colon may result, but these are not of sufficient gravity to be noted. It is a somewhat difficult worm to dislodge with the ordinary parasiticides, probably yielding more readily to male fern than to other drugs of this class.



FIG. 62.—*Trichiuris trichiura*, natural size: *A*, male; *B*, female.



FIG. 63.—Ovum of *trichiuris trichiura.*

Genus: *Trichinella.*

Trichinella Spiralis (Owen).

(*Trichina spiralis.*)

Male: Length, 1.4–1.5 mm.; thickness 0.04 mm.; cylindrical; anterior end tapering, posterior end gradually and slightly thickening and terminating in bifid extremity with two lateral somewhat conical tail appendages; cloacal aperture between these, which form a sort of bursa; back of cloacal aperture two pairs of papillæ. *Female:* 3.–4. mm. long; anterior end as in male; posterior end nearly of same thickness to tail, which is rounded; anus terminal; vulva at anterior fifth of body; viviparous. *Larvæ:* when born, 90.–100. microm. in length, obtuse anteriorly, posteriorly prolonged to a pointed tail; when encysted as "muscle trichinæ" the larvæ measure about 1. mm. long and 0.04 mm. in thickness, tapering anteriorly, more thick and obtuse posteriorly, with complete organization as in the adult and showing the characters of the different sexes.

This important parasite in its adult, sexual stage infests for a brief period the intestinal tract of man and a number of animals (mainly mammals), gives origin to a large number of larval worms after which the adults

die; the larvæ make their way into the muscles of the same host and pass an indefinite encysted stage in this situation until transferred to the next host by the ingestion of the infested flesh by the latter. The species was first established by Owen in 1835, from encysted larvæ in the muscles of human anatomical subjects; in 1846 Leidy announced the discovery of the

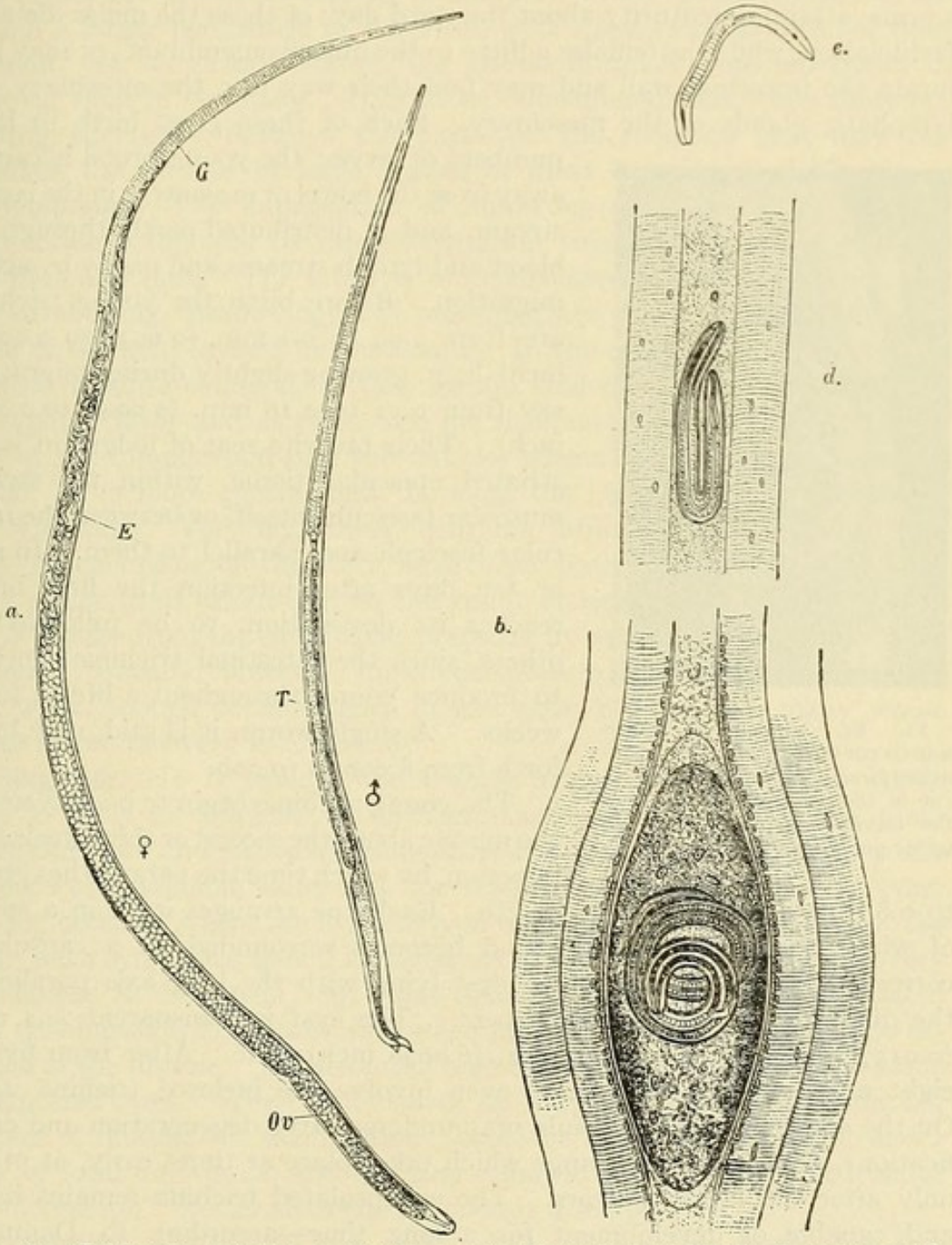


FIG. 64.—*Trichinella spiralis: a*, gravid female "intestinal trichina"; *E*, embryos; *G*, vulva; *Ov*, ovary; *b*, adult male "intestinal trichina"; *T*, testicles; *c*, young larva; *d*, larva in musculature; *e*, encapsulated larva in muscle. (*Braun, after Claus.*)

encysted larvæ in pork; but it was not until 1860, mainly through Zenker, that the full relation with the intestinal form and the development of the worm were understood.

Besides in man the worm is commonly found in the hog (domestic and wild), in rats, and in mice; it has also been met in rabbits, guinea-pigs, cow,

sheep, horse, dog, cat, fox, marten, badger, bear, raccoon, mole, skunk, hedgehog, hippopotamus, hamster, and in birds as hen, pigeon, and duck. It is most common in man, hog, rat, guinea-pig, and rabbit.

Man commonly acquires trichiniasis by eating infected ham insufficiently cooked. The capsules are digested and the trichinæ set free; they pass into the small intestine and there develop into the sexually matuer worms, attaining maturity about the third day; of these the males die after fertilization, while the females adhere to the mucous membrane, or may perforate the intestinal wall and may find their way into the mesentery and lymphatic glands of the mesentery. Each of these gives birth to large numbers of larvæ; the young brood is carried away from the bowel or mesentery in the lymph stream, and is distributed partly through the blood and lymph streams and partly by active migration. Before birth the young trichinæ are from 0.09 to 0.1 mm. (0.0035 to 0.00393 inch) long, growing slightly during migration, say from 0.12 to 0.16 mm. (0.0047 to 0.0063 inch). Their favorite seat of lodgment is the striated muscular tissue, within the striped muscular fasciculus itself, or between the muscular fasciculi and parallel to them. In nine or ten days after infection the first brood reaches its destination, to be followed by others, since the intestinal trichinæ continue to produce young throughout a life of seven weeks. A single worm, it is said, may bring forth from 8,000 to 10,000.

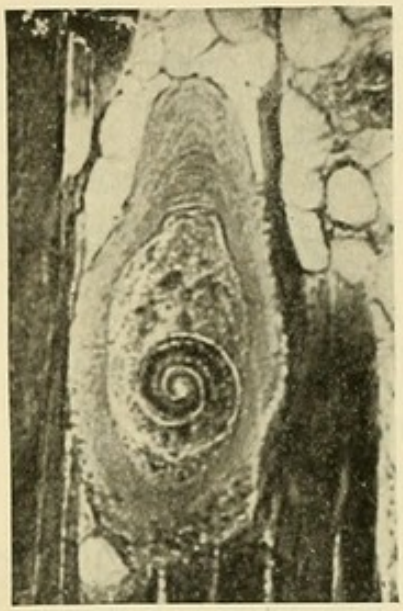


FIG. 65.—Section of human muscle containing encysted *trichinella spiralis;* parasite and its cyst cut in section, and but a part of the larval worm shown; about upper pole a local fat deposit.

The young trichinæ begin to be encysted in the muscle about the second or third week after infection, by which time the parasite has grown to 0.8 mm. (0.0314 inch) in length. Each one arranges itself in a spiral, of which the outline is oval, and becomes surrounded by a capsule of corresponding shape, the worm cyst lying with the long axis parallel to the direction of the muscular fibers. The cyst is transparent, 0.4 mm. (0.0157 inch) long, and 0.25 mm. (0.0098 inch) wide. After from five to eight months calcification may even involve the inclosed trichina itself. On the other hand, the capsule may undergo fatty degeneration and calcifications, a pathological change which takes place at times early, at others only after the lapse of years. The encapsulated trichina remains living and capable of development for a long time—according to Damman, in hogs eleven years, while in man they have remained living 25, 27, 30, and 40 years after infection. It has been shown by Zenker that the encysting is not a necessary condition to the mature development of young trichinæ.

Human infection having been conclusively shown to be due to the eating of raw pork infested with trichinæ, it is not at once evident how swine become infected. It is well known that the rats which infest slaughter houses are infected in large numbers, but it is plain also that they may acquire

trichinæ by eating pork. The two probably contribute mutually to the perpetuation of the disease.

As to the distribution of the trichiniasis: most epidemics have been in Germany. Even in America, where there have been two or three epidemics, it has been in German immigrant communities. Apparently it is rather the imperfect cooking of the pork which is responsible, for although a larger percentage of American pork appears to be infected than German, yet, as already stated, the disease is much more infrequent in America than in Germany. It is to be remembered that while thorough cooking effectually destroys the parasites, the requisite heat may fail to reach the interior of large masses of meat containing viable larvæ.

Symptoms.—The immigration of numerous active parasites in muscular tissue is followed by intense irritation, manifested at first by fever and muscular pain. The latter is especially severe during motion. The acts of chewing, swallowing, and breathing are particularly difficult, because of the pain excited by these acts. In the early stage of the disease diarrhea is quite common, so that certain epidemics have been mistaken for typhoid fever and as often also for rheumatism. In the very beginning of the immigration into the muscles edema has sometimes been observed. The more general and thorough the invasion, the more intense the symptoms. Very high fever, delirium, infiltration of the lungs, and fatty degeneration of the liver have been observed. Death may take place either from exhaustion as the result of extreme irritation, or later in the disease from the same cause preceded by anemia and gradual loss of strength. Usually, however, improvement sets in about the fourth or fifth week, though convalescence in bad cases is slow, and many weeks elapse before recovery is complete.

Diagnosis.—It is usually the unexpectedness of the disease which leads to delay in diagnosis. The resemblance of the symptoms to those of *typhoid fever* and *muscular rheumatism* has been referred to, yet in the presence of a possible cause—as, for example, a German picnic or other feasting occasion where the favorite ham or sausage has formed part of the feast—such symptoms should immediately excite suspicion. The discovery by Thomas R. Brown in 1897[1] that eosinophilia is constantly associated with trichiniasis is important and, when present, is confirmatory of the existence of the disease. A differential blood count should therefore be made in suspected cases. When doubt exists, the harpoon, designed for obtaining samples of muscles for examination, should be unhesitatingly used, under ether or local anesthesia, and the part removed carefully examined under the microscope. Better than the harpoon is a scalpel.

Treatment.—Salting of the pork, while causing the death of a few of the encysted larval trichinæ, is insufficient to destroy any large proportion unless prolonged much more than is usually practised; smoking is also lethal to the larvæ, but insufficient to guarantee the death of all; cold storage is of little or no value; but an exposure to heat of 70° C., as should be assured in thorough cooking, is known to be uniformly fatal to any remaining parasites and should render the infested flesh innocuous. However, it must be remembered that heat does not well penetrate to

[1] "Johns Hopkins Hospital Bulletin," April, 1897.

the interior of large masses of meat; and nothing but certainty of the thoroughness of cooking can be relied upon; and imperfectly cooked pork is more apt to be eaten than well cooked when hams are boiled entire.

Here, as so often elsewhere, an "ounce of prevention is worth a pound of cure." Such prevention consists in thorough official inspection of all pork brought to market, because cooking may fail of its purpose for the reasons already mentioned. For a similar reason swine should be grain-fed, rather than allowed to feed on offal. It is doubtful whether any direct measures can be used for arresting the disease after the muscles have once been invaded. It is a simple conflict for the mastery between the strength of the patient and the life of the trichinæ. In the majority of cases the former triumphs, though death is not infrequent from the causes named. If the disease is recognized early, the alimentary canal should be treated with vermicides and purgatives, with a view to getting rid of all the sexually mature worms which may happen to remain there, since it will be remembered that successive broods develop from the same mother-worm while in the intestinal tract. Glycerin, given in a table-spoonful (30 c.c.) dose hourly, is said to destroy the trichinæ. Benzine, in 1 to 2-dram (4 to 8 gm.) doses in capsules, and picric acid in dose of from 5 to 8 grains (0. to 0.5 gm.), are also recommended, but are regarded as less reliable. To relieve the pains, hypodermic injections of morphin, 1/4 grain (0.0165 gm.), or warm baths may be used. Restoratives and stimulants should be given to keep up strength.

Family STRONGYLIDÆ;
Genus: *Eustrongylus.*

Eustrongylus gigas (Rudolphi).

(*Ascaris canis* et *martis; a. visceralis* et *renalis; strongylus gigas; s. renalis; eustr. visceralis.*

Male: red in color; 14.–40. cm. in length, 4.–6. thick; slightly tapering anteriorly; mouth terminal, with a hexagonal orifice surrounded by six lips bearing papillæ; cuticle thin and transparent, finely striated transversely; about 150 papillæ along the longitudinal lines laterally (best marked near middle of body length); caudal extremity with an oval plate-like expansion serving as a bursa (transverse diameter the longer), its margin bearing small papillæ and slightly indented dorsally and ventrally; single sexual spicule. *Female:* general appearance and head end as in male; 20.–100. cm. in length and 5.–12. mm. thick; caudal extremity obtuse, straight, with anus subterminal; vulva 50.–70. mm. posterier to mouth; single ovarian and uterine tube plicated from near anterior end along the intestine nearly to anus, then returning to vulva near anterior end. Ova brown ellipsoid, with thick shell marked by external cribriform depressions, 64.–48. microm. long and 40.–44. microm. broad.

This worm, more common in the dog (and also found in other animals as seal, otter, wolf, horse, cow, marten, and skunk), has been recorded a number of times as a parasite of man, although in most cases with some reservation as to the correctness of diagnosis. It is the largest of the nematodes and has its habitat in the pelvis of the kindey, where one or several of the parasites may exist. It has been known, too, to be free in the abdominal cavity, and is said to have in rare instances been found in the liver and in the pleural cavity (in lower animals). Little is known of its life-history. At the time of oviposition the interior of the ova is segmented. Passed with the urine into water or moist earth outside the host, a larval worm develops in the course of five or six months in winter and probably much more rapidly in summer; this remains for a long time living within the shell, apparently several years. If removed

experimentally from the ovum it soon dies in pure water, but may be kept alive for a longer time in albuminous fluids. As yet no successful transference of the larval worm has been accomplished experimentally, and in consequence it is thought that in nature it passes to some intermediate host, possibly some fish.

In its usual habitat the worm causes considerable dilatation of the renal pelvis, and sometimes the whole kidney becomes reduced to a thin hydronephrotic sac, in which in the midst of a red and bloody urinous fluid the parasites are found. In man the worm has not been recognized antemortem, having only accidentally been observed in autopsies; and

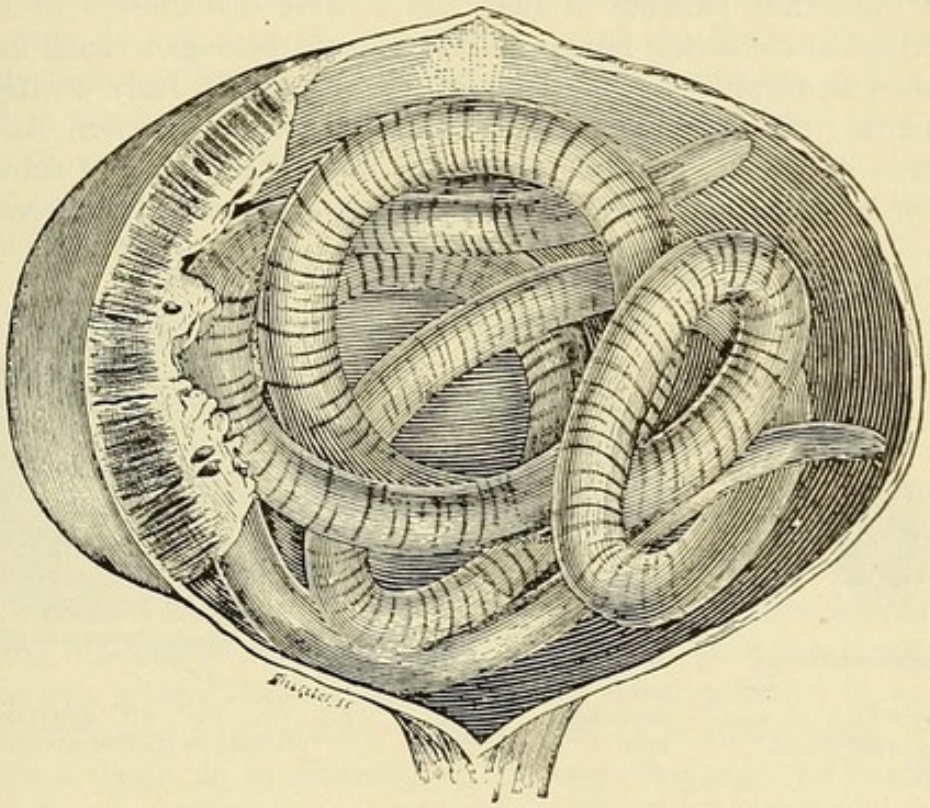


FIG. 66.—*Eustrongylus gigas:* female, natural size, in kidney of dog. (*Railliet.*)

this is also usual in case of infestment of the lower animals. However, in the dog the urine often becomes notably bloody, the animal sometimes whines as if in pain, the gait becomes tremulous, the bark altered, the animal becoming depressed and showing nervous symptoms which have been confused with those of rabies. The diagnosis in man must, of course, rest upon the discovery of the characteristic ova in the urine. It might be located in one or the other kidney by catherization of the ureters; and the only treatment, should a safe conclusion as to the presence of the worm in one or other renal pelvis be arrived at, would rest with the enucleation of the worm or of the entire kidney by surgical procedure. Thus far but one kidney has been found infested in a single host, although, of course, care should be exercised to exclude the possibility of a bilateral infestment.

Genus: *Uncinaria.*

Uncinaria duodenalis (Dubini).

(*Anchylostoma duodenale; strongylus quadridentatus; dochmius anchylostomum; sclerostoma duodenale; strongylus duodenalis; dochmius duodenalis;* European or old-world hook-worm.)

Male: whitish or blotched with brownish, spots when intestine contains blood; 8.–10. mm. long; cuticle finely striated transversely; tapering to a blunt point ante-

riorly and with head curved upon dorsum so as to give a slightly hooked anterior end; on each side of median line on ventral side of oral border two hook-like chitinous teeth and on dorsal border on each side of median line one less curved chitinous tooth; with a dorsal conical tooth extending along back of oral cavity from base of cavity; in oral cavity about esophageal opening a delicate armature consisting of two dorsal and two ventral lancet-like pieces; posteriorly the body ends in an abruptly pointed tail in a copulatory bursal expansion of the cuticle, this having one dorsal and two lateral lobes; in folds of bursa one dorsal subdivided muscular ray each division ending tridigitately, and on each side symmetrically an undivided dorso-lateral, a divided lateral, undivided latero-ventral, subdivided ventral and undivided small subventral muscular rays; cloacal aperture superterminal; two equal spicules. *Female:* General appearance and anteriorly like male; 12.–18. mm. long; posteriorly tapering to a finely pointed tail; anus subterminal; vulva about posterior third of body length; two uterine and ovarian tubes. Ova: colorless, elliptical thin-shelled, 50.–60. : 30. microm.

This important parasite of man has a wide distribution in tropical and subtropical countries, but probably properly belongs to such localities in the older hemisphere as southern Europe (especially Italy, Switzerland, and Austria), the Mediterranean borders of Africa, southern Asia, and the eastern archipelago. It is found also in the tropical and subtropical regions of America, but here has been much confused with the American

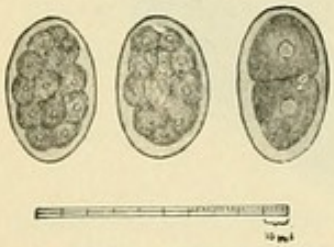


FIG. 67.—Ova of *uncinaria duodenalis.*



FIG. 68.—Anterior end, showing mouth parts of *uncinaria duodenalis* (dorsal view).

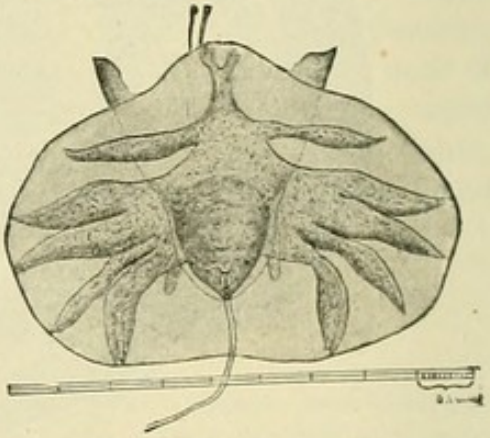


FIG. 69.—Tail, with expanded bursa, of male *uncinaria duodenalis.*

species of hook-worm recently recognized as a separate species. As imported cases, perhaps occasionally giving rise to small endemic foci, it has been met in the cooler parts of the United States.

Its habitat is in the duodenum, jejunum, and upper part of the ileum of man, where it is found in numbers varying from a few to considerably more than a thousand. With its strong armature it attaches itself to the intestinal mucous membrane producing a small excavation, and thus fixed, sucks the nutrient juices, lymph and blood, from the mucosa. Through the agency of certain glands situated in the anterior end the worm produces a substance inhibiting blood coagulation; and thus from the tiny lesions produced by the worm, which are frequently forsaken for fresh situations, considerable bloody oozing takes place. From this factor, as well probably from nutritive faults following upon the intestinal disturbances induced and perhaps also from some undiscovered toxic influences, there results a loss of bodily weight and strength and an anemia which in its severer forms ranks among the pernicious anemias. Fatal cases are not infrequent. When from the first but few parasites are present there may be practically no symptoms appreciated (unless the parasites are but a complication of other serious disturbances as malaria, the anemic results

of which they are likely to accentuate, or unless the host be quite young or a weakling). In severer grades of infestment, discomfort and actual pain in the abdomen, nausea, altered appetite (often kakophagism), and alternating diarrhea and constipation are apt to be noted. In course of time flesh and strength are lost, the patient becomes dull and slothful; the young do not develop with the usual vigor; and an anemia of varying grade comes to be appreciated (miner's anemia, tunnel anemia, etc.). This latter may be profound in loss and change of the red cells, is apt to show in its typical appearance some increase in the eosinophilic leukocytes, and a comparatively low hemoglobin proportion. With the severer anemia, and probably largely secondary to this, arise wide-spread degenerations, mainly fatty in type, involving almost any of the body structures, but especially noteworthy in the important parenchymatous structures as the wall of the heart, the liver, and kidneys. These in turn give further manifestations of disease; the urine becomes albuminous, the circulation becomes feeble, and a cachectic type of dropsy is apt to develop; and eventually, if no relief be afforded, the patient may die, as in any severe anemia, from exhaustion, intercurrent affection, or, perhaps, accidental terminal hemorrhage. The course of the case is apt to be a prolonged one, the parasites often persisting in the host for years.

The affection, known as *uncinariasis* or *anchylostomiasis*, is readily recognized from the general picture and the discovery of the ova of the worms in the stool, these being very numerous in the dejecta from subjects of even moderate infestment. They are readily determined by means of the ordinary laboratory powers of the microscope in thin layers of fecal matter, diluted if needed with a drop of water. There is eosinophilia.

Of the life-history of the parasite it is known that the larvæ escape from the ova within from 24 to 48 hours at a temperature of 25° C. or thereabouts in the fecal matter, in moist soil or in dirty water. Hence, if there is a doubt as to diagnosis of the eggs they may be hatched out and the characteristic larva sought more easily after centrifugation (Dock and Bass). The incubation goes on best in fair access to air, and therefore the most favorable situation is in moist sandy soil. When first emerged the larva measures about 0.2 mm. in length, is obtuse anteriorly, and posteriorly tapers to a finely pointed tail and shows a rhabditiform type of esophagus. In 48–72 hours a moulting occurs, the larva having grown in size, but preserving its structural features unchanged; a second moulting follows about the fifth day, the larva remaining, however, in the old cuticle (so-called "encystment") and assuming the adult type of esophagus. In this encysted stage it is still motile and now lives well in water or moist soil for several months, eventually dying or gaining access to a fresh host. From actual personal experience and from experimentation on man and dogs, Loos has established the fact that at this stage the parasite may pass into the human host by penetration of the skin. Should the moisture containing these larvæ come in contact with the skin, as about the feet of persons walking barefooted on the wet and infested sand, or wading in infested water, the larval worms attack the exposed surface, rapidly penetrating the skin and leaving their mantles (old cuticle) behind. In so doing, if there be many of the larvæ entering, considerable irritation

and consequent mild inflammation may be induced. It is supposed that this feature is the origin of certain inflammatory skin affections common in tropical regions and known as "ground itch," "water itch" and by other local terms; probably only a portion of cases of such affections depends upon this cause, as there are doubtless many other possible irritants which may act in a similar manner, and, moreover, it is probable that much of the inflammatory mischief is caused and prolonged in these cases by bacteria of one or other sort conveyed by the larval worms to the subcutaneous tissues. From the position of entrance into the skin the larvæ make their way, probably largely by passive convection by the blood and lymph, to the lungs. Here they penetrate to the air-passages, where it is thought they undergo another ecdysis, or moulting. They are still minute; are supposed to be carried by the bronchial mucus upward to the mouth and then to be swallowed, thus gaining their proper habitat and growing into adult size and sexual ability in the upper part of the intestine.

While this mode of infestment may be regarded as established, the older belief that the encysted larvæ are transmitted to the host by direct ingestion in dirty water or on unclean vegetables, or in dirt (in kakophagism), etc., cannot be as yet excluded, and must be kept in mind in considerations as to prophylaxis.

Genus: *Necator.*

Necator Americanus (Stiles).

(*Uncinaria americana; anchylostoma americanum.*)

Male: differs from *uncinaria duodenalis* in being of smaller size (6.–9. mm. long and more slender than *u. duodenalis*), in the smaller size and more conical shape of the head, in having no hooklets on the oral rim, but instead on each side a large ventral and smaller dorsal chitinous lip extending from the rim toward the median line; in a greater prominence and projection into the oral cavity of the dorsal conical tooth; in the smaller size of the copulatory bursa, its dorsal lobe being subdivided and the ventral margin being extended so as to form an indefinite ventral lobe and showing the dorsal muscular ray of the bursa divided, each division ending in a bipartite tip. *Female:* differs from *u. duodenalis* in being shorter and more slender (8.–15. mm. long), with similar difference of the anterior end as above outlined for male; vulva just in front of the middle of body length instead of at posterior third, as in *u. duodenalis.* Ova somewhat larger than those of *u. duodenalis* (68.–70. : 38.–40. microm.), but otherwise similar.

Necator americanus, originally established as a species of uncinaria by Stiles, but subsequently determined as generically distinct and given the name now used, is found especially in tropical and subtropical America, and in the West Indian islands; and prior to 1902, when the species was established, was probably often confused with *uncinaria duodenalis.* The latter is also met in imported instances and has been encountered not infrequently either alone or in association with the American form; but is scarcely to be regarded as properly an American parasite, especially since it is almost certain that many of the records of its occurrence in America are based by mistake upon *necator americanus.* The two worms are analogous in their influences and the term *uncinariasis* is usually employed to indicate the state of infestment by *necator americanus* or *uncinaria duodenalis* more or less indifferently. The worm in question is very common on our own Southern States, where it is apparently responsible for a group of anemic conditions in the inefficient, undernourished, pallid, and complaining classes of population known by various contemptuous terms in different localities, as "poor white trash," "crackers," "sand-lickers," "dirt-eaters," etc. The condi-

tions produced by the American hook-worm are comparable to those caused by the old-world form, but are probably less intense for a given degree of infection in the individual host. The affection in this country has long been popularly known as dirt-eaters' disease, sand-lappers' disease, mountain anemia, etc.

Treatment of Uncinariasis.—For the expulsion of the parasites, thymol is perhaps the most efficient remedy. It is given in large doses of the undissolved drug with precautions as below indicated, reliance being had upon its slow and partial solution in the intestine in close contact with the parasites, thus directly influencing the latter, but not being sufficiently dissolved to afford ease of serious absorption and intoxication of the host. The patient is prepared the day prior to the administration by a mild cathartic and by taking but a light evening meal or none. The following morning thymol is given in capsule or cachet, in three doses an hour apart of 0.6–2. g. (9.–30. grains) each, making from 1.8 to 6. grams in all. During the period

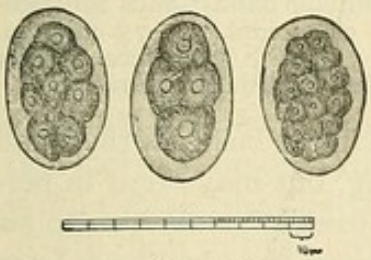


FIG. 70.—Ova of *necator americanus*.



FIG. 71.—Anterior end, showing mouth parts, of *necator americanus* (dorsal view).

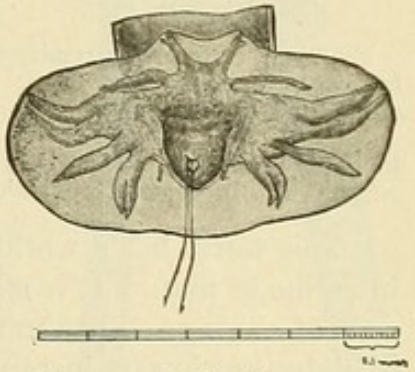


FIG. 72.—Tail, with expanded bursa, of male *necator americanus*.

in which the drug is in the alimentary canal oils, alcoholics, and other solvents of thymol are withheld to prevent massive solution and absorption of the substance. Within an hour after the last dose, if free purgation has not meanwhile taken place, a purgative (an ordinary saline) is administered; and the stools are to be closely examined for the discharged worms. In the course of a week or ten days, if examination of the dejecta continue to show the presence of ova, the above procedure may be repeated. Sometimes mental wandering, dizziness, and faintness appear as toxic symptoms from absorption of thymol, but usually rest in bed, a little weak coffee, and a small amount of hot bouillon after purgation has begun allay these symptoms. Male fern is strongly recommended by a number of European writers for the old-world hook-worm; but the writer's experience with the American form would indicate the greater efficiency of thymol. Filmaron, the non-toxic active principle of filix mas, has been recommended by Nagel.

As measures of prophylaxis there should be recommended the use of only boiled or well-filtered water for drinking purposes, thorough cleanliness of all vegetable food which has been grown in suspicious soils and which is eaten uncooked, together with refraining from going barefooted and wading in dirty water or mud in infested districts. The drainage of soils contaminated by the dejecta of infected persons, together with its exposure to

the sun by plowing, should also be considered; and the stools of infested persons should be disinfected before disposal. Persons should not go barefoot in infected places.

Family: ASCARIDÆ;
Genus: *Ascaris.*

1—Ascaris lumbricoides (Leuckart).
(Max-worm; common round worm of children.)

Male: whitish to reddish-yellow; 15.—17. cm. long, 3.—3.5 mm. thick; elongate, fusiform; cuticla finely ringed; oral orifice terminal, with three lips (one dorsal and the other two meeting in median ventral line), each with fine denticulations on margins; at base of superior lip two papillæ, one only at base of other two lips; posterior end terminating conically, curved ventrally, with two slightly curved, short, equal spicules projecting from subventral cloaca; 70.—75. papillæ on ventral face of posterior end, of which seven pairs are postanal. *Female:* 20.—25. cm. long, 5.—5.5 mm. thick; anterior end and general appearance as in male; posterior end tapering, ending in conical, pointed, straight tail; vulva at level of first third of body length (in a slightly depressed annular band); anus subterminal. Ova ellipsoidal, 50.–75. microm. long and 40.—58. microm. broad; thick-shelled; stained yellowish from fecal matter when found in dejecta, but colorless in uterus; covered with a mammilated envelope.

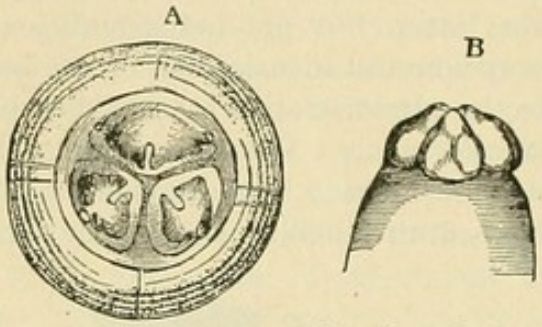


FIG. 73.—Anterior extremity of *ascaris lumbricoides: A*, seen from front; *B*, seen from dorsal surface. (*Railliet.*)

This worm has a world-wide distribution, its habitat being in the small intestine of man. It is more common in the young, but may occur in persons of any age. The number in a single host is usually small, two to six or eight, but in rare instances there have been reported some hundreds from one individual; and Cruveilhier found in the small intestine of a young idiot girl great masses of the worms, the number of which he estimated at about one thousand.

The presence of but a few of the parasites may pass unnoticed, but even where the parasites are but few there may result in children severe nervous disturbances, either reflex from intestinal irritation or possibly from absorption of some toxic material elaborated by the worms or generated in the intestine in their presence, as epileptiform attacks, cerebral congestion and headache, vertigo, chorea, ocular disturbances, or manifold hysterical manifestations. Capricious appetite, nausea, indefinite abdominal pains, symptoms of maldigestion, restless sleep are often complained of; occasionally swelling and congestion of the lachrymal papillæ, undue lachrymation, itching about the eyes, itching and swelling of the fingers are encountered. The worms possess active motility and not infrequently wander from their proper habitat, either up or down the canal and perhaps into some of the collateral passages. Thus, the writer some years since met an instance in which an adult ascaris was found in the cavity of a periappendiceal abscess, the worm having penetrated the appendix and escaped through a perforation in its distal end into the abscess cavity, having probably had mcuh to do with the appendicitis and perforation of the wall. Not infrequently they wander to the rectum and spontaneously pass from the anus. They have been found in the biliary duct, producing obstructive jaundice; in the pancreatic duct; in the stomach, whence they are commonly expelled by vomiting excited by their presence and movements. A specimen was formerly

in the collection of the University of Pennsylvania, in which the worm, having been thus carried from the stomach to the pharynx, had been retracted into the larynx in the deep inspiration following the retching, obstructing the lumen and causing the death of the child. Bunches of these worms have been known to cause intestinal obstruction, and occasionally at such positions of obstruction perforation of the wall has taken place and the parasites have been found in the abdominal cavity.

The ordinary life-history is about as follows: The ovum, after discharge in the fecal matter, slowly develops in water or moist earth, the larval worm being retained within the shell and preserved for months from destruction by the resistant shell; it is transferred directly to the alimentary canal of the next host with unclean water or food, there quickly freed from its wall by the action of the digestive juices, and developing to adult stage in the course of about five weeks.

The recognition of the presence of these parasites, while perhaps suggested by the presence of the more common symptoms above indicated, is only established by the discovery in the stools of the host of the ova or by the recognized passage of one or more worms.

Treatment.—The remedy which has been most satisfactory in my hands is santonin in combination with calomel. Powders containing santonin and calomel, of each 1 or 2 grains (0.066 to 0.132 gm.), may be prescribed rubbed up with sugar of milk. One is given night and morning until the bowels are freely moved. The santonin may color the urine and produce yellow vision, or xanthopsia, but I have never seen harmful results in a large experience, though poisoning, manifested by convulsion is said to have been produced. For very young children the dose may be reduced to 1/4 to 1/2 grain (0.0165 to 0.033 gm.). The worm tablets extensively advertised usually contain santonin as their basis. There is an official troche, U. S. P., containing 1/2 grain (0.033 gm.) of santonin. Santonica, or Levant worm-seed, whence santonin is derived, is no longer used. What is known as wormseed oil, the oil of chenopodium, another excellent remedy for round worm, is derived from the *chenopodium anthelminticum*, or American worm-seed. The dose is 10 minims (0.65 c.c.) to a child of five years, on a lump of sugar or in emulsion—before breakfast,

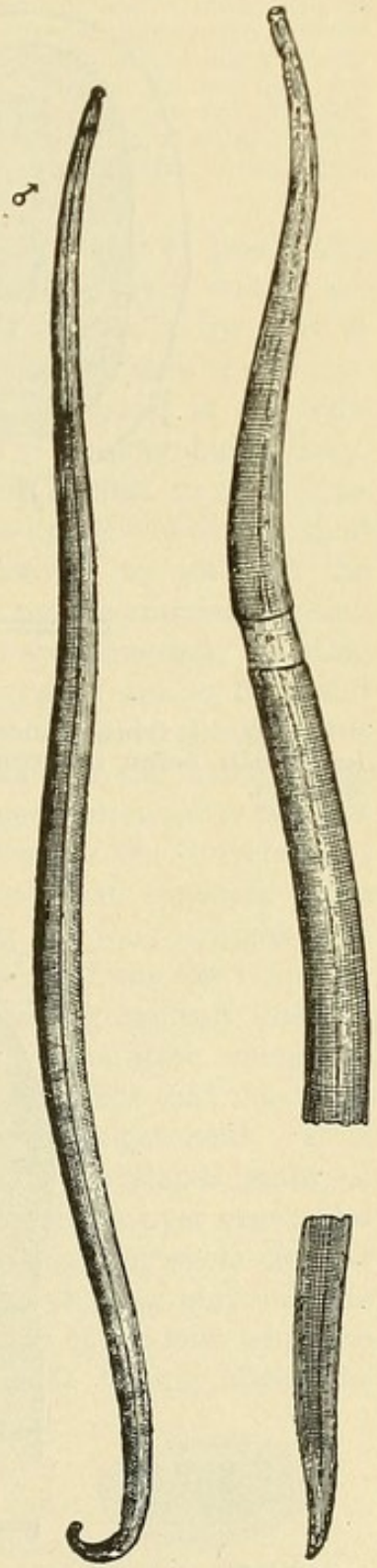


FIG. 74.—*Ascaris lumbricoides:* to left, male in lateral aspect; to right, female, ventral aspect, natural size. (*Railliet.*)

dinner, and supper for two days—followed by a purge, of which none is more suitable for children than calomel, itself a vermicide.

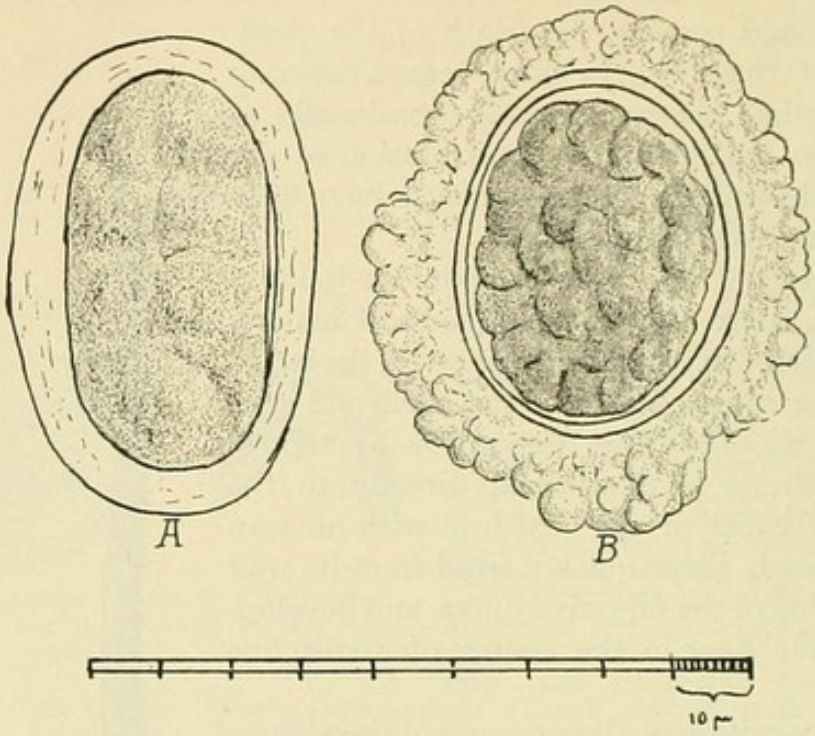


FIG. 75.—*A*, Ovum of *ascaris texana* drawn from specimen in uterus; *B*, ovum of *ascaris lumbricoides* drawn for comparison from examples taken from uterus of formaldehyd specimen.

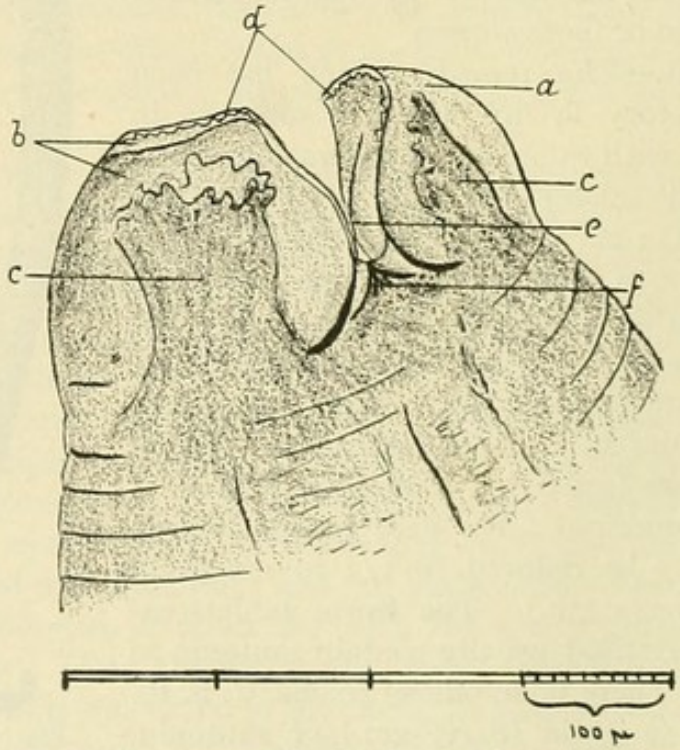


FIG. 76.—Lips of *ascaris texana* (camera lucida drawing from compressed specimen): *a*, superior lip; *b*, inferior lips, the left overlying the right; *c*, pulpa; *d*, denticulate anterior margin of superior and right inferior lips; *e*, keel of superior lip on inner surface; *f*, interlabium.

The prophylactic measures are principally the careful filtration or boiling of all water used for drinking purposes, and thorough cleanliness of all uncooked food.

Genus: *Oxyuris.*

Oxyuris vermicularis (Leuckart).

(*Ascaris vermicularis; fusaria vermicularis;* pin-worm; thread-worm; seat-worm.)

Male: whitish; 3.–5. mm. long, 0.3–0.4 mm. thick; cuticle transversely striated and at head end showing a vesicular swelling along the dorsal and ventral median lines; lateral lines distinct; mouth terminal, with three retractile lips; esophagus with distinct bulb; posterior end conical, curved ventrally, with six pairs of papillæ and slight cuticular expansion on each side; one spicule hooked at free end. *Female:* 10. mm. long, 0.6 mm. thick; anterior end and general appearance as in male; posterior end straight, extended to a long mucronate tail; anus 2. mm. in front of tail; vulva at anterior third of body length. Ova oval, flattened on one side, 50.: 16.–20. microm.; thin-shelled; colorless with embryo developed at oviposition.

This worm is an extremely common parasite of man, of practically world-wide occurrence, having its proper habitat in the lower end of the ileum and the cecum. It is especially frequent in children, but is also found in individuals of any age. The worms usually are in large numbers in the individual host; and are possessed of considerable activity, wandering from their natural habitat so that occasionally they are found in the upper end of the small intestine and have been known to get into the stomach and be vomited, but more commonly passing downward to the rectum, and spontaneously crawling from the anus. They are not as apt to be found in errant positions as *ascaris lumbricoides,* but in this respect much that has been said of the latter is true also of the present type. A specimen recently brought into the pathological laboratory of the University of Pennsylvania shows in a catarrhal appendix large numbers of the parasites. It was long thought that the entire evolution of the worm from the ovum to adult stage takes place in the original host, and Vix has actually seen the larval worms after emergence from the egg in the rectal mucus; but that this takes place, save exceptionally, is no longer held. It is believed that the ova with the developed embryos within are scattered after defecation over vegetables and fruit, being strongly resistant for some time at least to the effects of drying; or they may become adherent to the nails and fingers of the host when the latter, because of the intolerable itching about the anus, scratches himself. It is thought possible, too, that they may be transferred from the fecal mass to food-stuffs by flies. Ingested with foods they are directly swallowed. Upon the hands of one host they may readily be transferred to the hands of a second human being and thus endanger the latter. From the fingers they may be transferred to the mouth, or perhaps, may be carried into the nose, when the habit of nosepicking exists; and in the nasal mucus the larvæ may emerge from

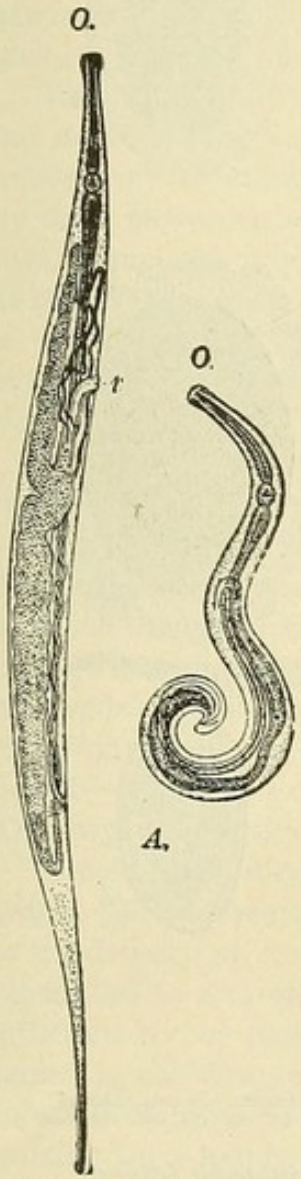
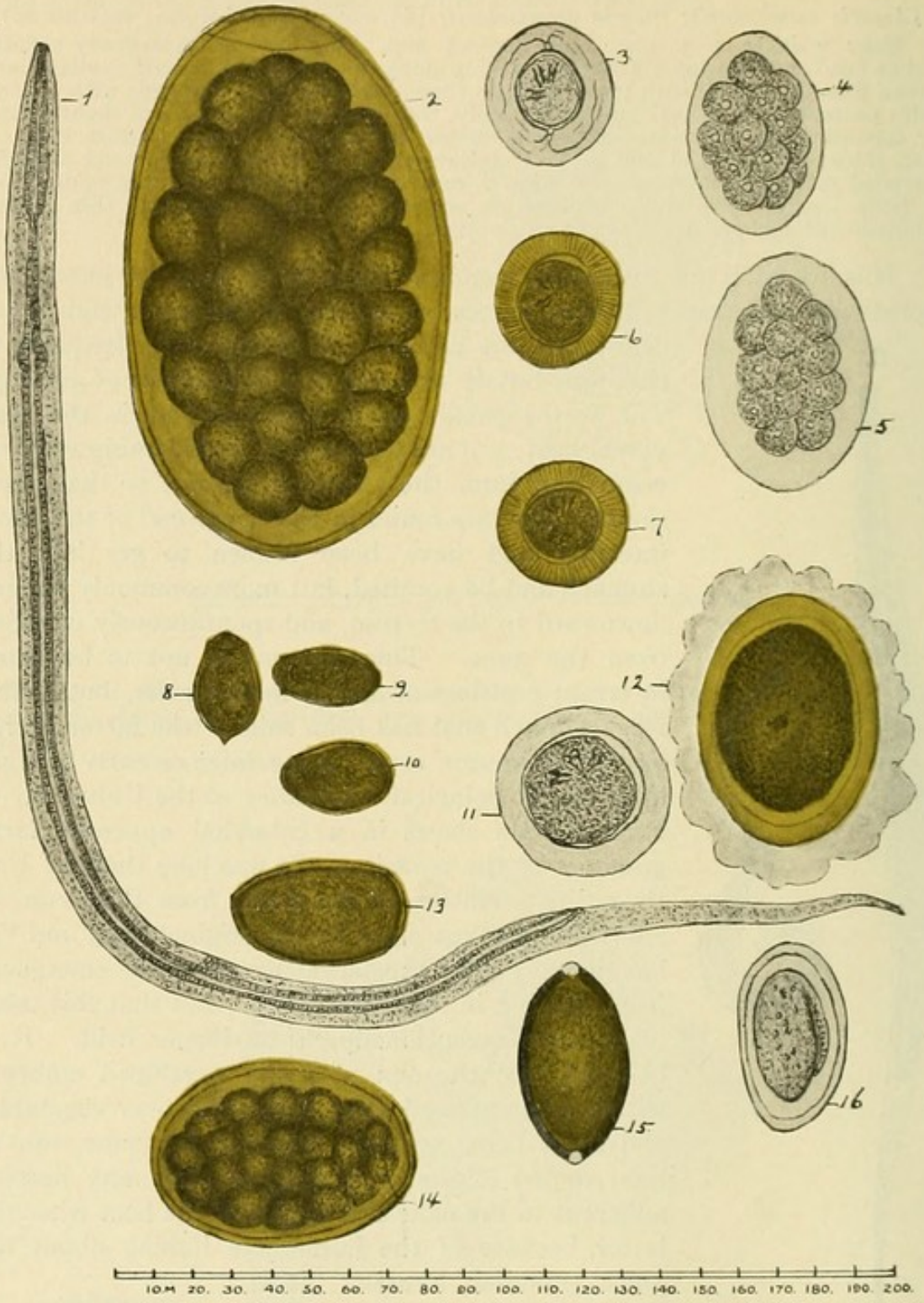


FIG. 79.—*Oxyuris vermicularis:* to the left, female; to right, male (considerably enlarged). *A*, anus; *O*, mouth; *v*, vulva. (*Braun, after Claus.*)



FIG. 80.—Ovum of *oxyuris vermicularis.*



1. Larval *strongyloides intestinalis.*
2. Ovum of *fasciola hepatica.*
3. Ovum of *hymenolepis nana.*
4. Ovum of *uncinaria duodenalis.*
5. Ovum of *necator americanus.*
6. Ovum of *tænia mediocanellata.*
7. Ovum of *tænia solium.*
8. Ovum of *opisthorchis sinensis.*
9. Ovum of *episthorchis felineus.*
10. Ovum of *cotylogonimus heterophyes.*
11. Ovum of *dipylidium caninum.*
12. Ovum of *ascaris lumbricoides.*
13. Ovum of *dicrocœlium lanceatum.*
14. Ovum of *dibothriocephalus latus.*
15. Ovum of *trichiuris trichiura.*
16. Ovum of *oxyuris vermicularis.*

FIG. 81.—Parasitic bodies, ova and larva met in human feces; color approximate only.

the shell and later be swallowed. It is not likely thay they are transferred by water, soon dying in the latter fluid. If the eggs have been swallowed, the larvæ probably emerge from the shells in the stomach and upper intestine. Here they undergo several moults before maturation, copulate, and the females become gravid. The males after copulation apparently soon die and are carried off in the intestinal contents, explaining the comparative rarity of the latter among samples obtained. The females in their wanderings lodge for the most part about the ileo-cecal region, where most of the ova are deposited; but are apt to continue moving slowly along the gut to the rectum, continuing to deposit their eggs. Their duration in the intestine is apparently at least some months; and the common persistence of parasitism in spite of treatment argues for the ease and frequence of self-infection by the host.

When in small numbers but little disturbance is ordinarily occasioned, but nervous symptoms, much as outlined in connection with *ascaris lumbricoides*, may be induced. The greatest common inconvenience is occasioned by their movements and the irritation of mucous membrane of the rectum, inducing a proctitis and troublesome pruritus. Sometimes, especially at night, the worms spontaneously escape from the anus and may be found in the bedclothes; or they have been known to crawl into the genital canal of females, where they may set up a vaginal catarrh and by the itching occasioned lead children to take up the habit of masturbation.

Treatment.—Some perseverance is commonly necessary to get rid of the thread-worm. I usually prescribe the same powder of santonin and calomel as for the round worm—*i. e.*, from 1 to 2 grains (0.066 to 0.132 gm.) of each—but at the same time order nightly injections into the rectum of vermicides, of which there are many—the infusion of quassia, of aloes, lime-water, vinegar, corrosive sublimate (1 to 5000), salt and water. The injection should be retained for some time, and to this end the buttocks should be raised, or the child may be placed on its hands and knees. Only as much should be introduced—from 2 to 4 ounces (60 to 120 c.c.)—as can be conveniently retained. Too large a quantity is promptly rejected.

Stools from infested individuals require to be disinfected before disposition; the anal region, especially in children, should be well washed after every defecation; and under no circumstances should the infested person be permitted to scratch about the anus lest the ova of the pin-worms become adherent to the nails, and through careless and uncleanly habit be transferred to the nose or mouth. This can be partially prevented by clothing infested children in stout muslin underdrawers, thus preventing them from putting their hands to the anus. Cleanliness and prevention of self-infestment, if persevered in will eventually be followed by the natural death and disappearance of the parasites; it is safe to say in all cases of very protracted presence of these pin-worms that in some way these essentials have not been fully maintained.

A—*ARACHNOIDEA.*

Among a number of arachnoids of more or less importance as parasites of man, either transitorily or permanently, the following species may be selected for brief mention.

Order: Acarina (Mites, Ticks).

1. *Sarcoptes* or *acarus scabiei*—the itch insect. This is the most frequently met of the arachnide parasites. Its oval, nearly circular little body, provided with horns and bristles, is barely visible to the naked eye under

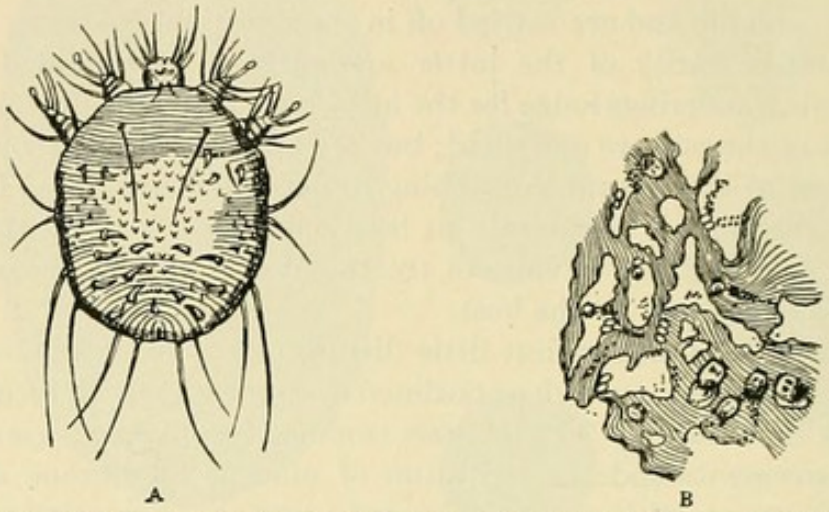


Fig. 80.—*Acarus scabiei A:*, female, dorsal view; *B*, portion of human epiderm, showing burrows with contained ova and young acarians. (*Gould, after Leuckart.*)

favorable circumstances, the male being from 0.2 to 0.3 mm. (0.0078 to 0.00118 inch) by 0.145 to 0.19 mm. (0.0057 to 0.0074 inch); the female, from 0.33 to 0.45 mm. (0.0129 to 0.0177 inch) by 0.25 to 0.35 mm. (0.0098 to 0.0137 inch).

The female lies at the end of a burrow in the epidermis, in situations where the skin is most delicate, as between the fingers, at the elbows, and under the knees, in the groin, and on the penis, very seldom in the face, but in any delicate part. In this burrow, some millimeters to a centimeter long, the female deposits her eggs. The male is seldom seen, dying after copulation, and the female after depositing her eggs. The eggs hatch in from four to eight days, and in about 14 days the larvæ are sufficiently matured to make their own burrows. The disease is communicated by personal contact or by clothing.

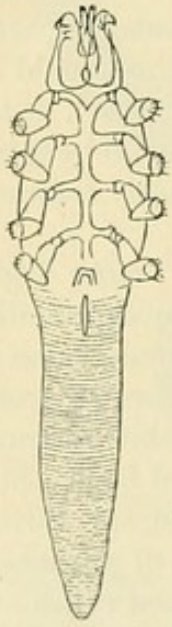


Fig. 81.—*Demodex folliculorum:* from dog, enlarged. (*Braun, after Mégnin.*)

Symptoms.—These are first an intense itching which incites to scratching, which, in turn, causes excoriations, papules, vesicles, and pustules.

Diagnosis.—The diagnostic feature is the shining little vesicle readily recognized by a moderate magnifier in the webs of the fingers, though it is often obscured and obliterated by the eruption and marks caused by scratching.

Treatment.—This is very simple. Sulphur ointment is a prompt specific. The body should be first bathed thoroughly with soft soap, and then as thoroughly anointed with the ointment, which should be allowed to remain until the next day, when there should be another bath, followed by another vigorous application of the ointment. Three or four days of this treatment should

suffice. An ointment of naphthol, one dram to the ounce (4 gm. to 30 gm.) is recommended.

2. *Demodex folliculorum*, a minute parasite from 0.3 to 0.4 mm. (0.0118 to 0.0157 inch) long, which resides in the sebaceous follicles, with the grease of which it can sometimes be squeezed out. It is oftenest met on the face and nose. It is said to be present in about 50 per cent. of persons, but this is probably exaggerated. It usually gives rise to no symptoms, but is said sometimes to be the cause of obstruction of the follicles and produces thus the little worm-like accumulations of fat which may be squeezed out of the follicles, and which cause inflammation and acne.

Treatment.—Acne is well treated by a lotion of corrosive sublimate, 2 to 1000, and it may be by its effect on the demodex that it is useful.

3. *Leptus autumnalis*, or harvest bug, is a minute red parasite, from 0.3 to 0.5 mm. (0.0118 to 0.0196 inch) long, which has three pairs of legs, with rows of bristles upon its back and belly. It prevails in summer on grasses and plants, attaches itself to the skin of man and animals by its hooklets, and gives rise to irritation.

Treatment.—It is successfully destroyed by sulphur ointment and corrosive sublimate, 2 to 1000.

Order: Hemiptera.

Family: *Pediculidæ* (Lice).

1. The *pediculus capitis*, or head-louse. The male is from 1 to 1.5 mm. (0.0393 to 0.059 inch) long, the female from 1.8 to 2 mm. (0.0708 to 0.0757 inch) long. The color varies somewhat with the races. In the white it is gray with a dark border, in the negro and Chinamen darker. Its eggs are 0.6 mm. (0.0236 inch) long, of which the female lays about 50, which mature in about a week, and in 18 days are ready to reproduce. The eggs are attached to the hairs, and are easily visible, being known as nits.

The head-louse is found the world over, upon the hairy heads of men and sometimes in other parts of the body where there are hairs. Even when they are quite numerous they may produce no symptoms. Generally, however, they cause itching and scratching, especially when the louse bores deep into the skin and produces pustular dermatitis, with resulting crusts and scabs in which the hair becomes matted and tangled, forming the *plica polonica*, so called from its frequency in Poland.

2. The *pediculus vestimenti*, or body-louse, is considerably larger, being from two to five mm. (0.1574 to 0.1968 inch) long and whitish-gray in color, the back part of the body being wider than the thorax. Its eggs are from 0.7 to 0.9 mm. (0.0275 to 0.0354 inch) long, and about 70 are laid by the female. It lives on the clothing, in which it deposits its eggs, about the neck, back, and abdomen. The puncture incident to sucking is often covered by a hemorrhagic point. It, too, causes itching and scratching, with irritation and inflammation of the skin, and in old cases a roughness and pigmentation causing dark spots and a condition known as *morbus errorum* or vagabond's disease, which has been mistaken for Addison's disease.

3. The *pediculus pubis*, *phthirius inguinalis*, or crab-louse, is smaller

than the head-louse, grayish-yellow or grayish-white, the male being from 0.8 to 1 mm. (0.0314 to 0.0393 inch) long, the female 1.12 mm. (0.0441 inch) long. The eggs are pear-shaped, from 0.8 to 0.9 mm. (0.0314 to 0.0354 inch) long, and from 0.4 to 0.5 mm. (0.0157 to 0.0196 inch) wide. They infest the parts of the body covered by shorter hairs, such as the pubis,

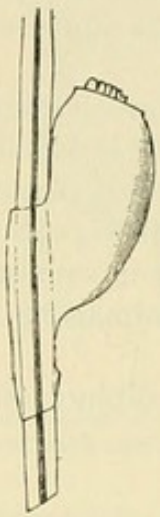


FIG. 82.—Ovum of head-louse glued to hair: ×70. (*Braun.*)

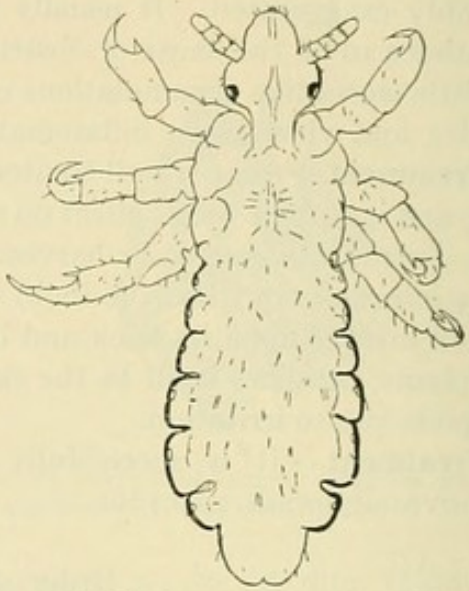


FIG. 83.—*Pediculus capitis:* ×15. (*Braun.*)

axilla, and eyebrows. The *pediculus pubis* does not wander so much as the *pediculus capitis* or *vestimenti*, but adheres more closely to the skin and therefore removal is often with difficulty.

These lice give rise to annoying itching about the pubes.

Treatment of Pediculosis.—For the head lice: The hair should be cut

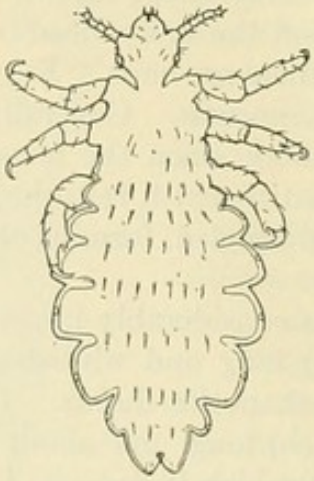


FIG. 84.—*Pediculus vestimentorum:* ×10, circa. (*Braun.*)

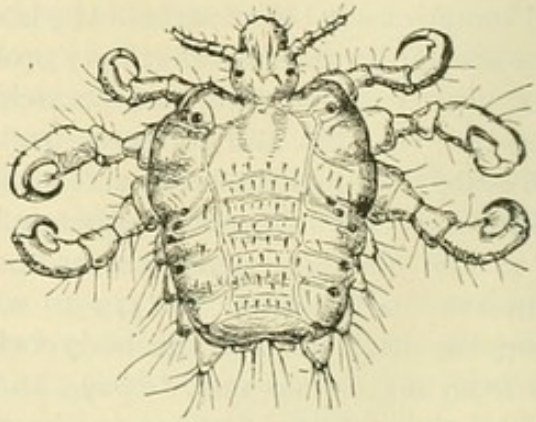


FIG. 85.—*Phthirius inguinalis.* (*Braun*).

short and burned, the head thoroughly washed with soap and water, and then anointed with mercurial ointment or washed with tincture of cocculus indicus, or with coal-oil or turpentine, or carbolic acid, 1 to 50. Cocculus indicus is to be preferred because of its freedom from odor. The washing should be repeated for several days in succession.

The treatment for the crab-louse is the same, but, as mentioned, it adheres firmly to the skin, and it is generally necessary to pick off the individual louse, and to make an application of mercurial ointment.

To get rid of the body-louse the clothing, if not too valuable, should be burned, but may be boiled, or, when this is not admissible, treated by superheated steam.

The itching promptly disappears with its cause, but, if necessary, it may be allayed by a warm bath to which 4 or 5 ounces (120 to 150 gm.) of sodium bicarbonate are added.

Repeated bathing with soft soap should be done until it is absolutely certain that the parasite and its ova are removed.

Destruction of the body louse is now doubly important because of its known relation to typhus fever.

4. The *cimex lectularius*, or common bedbug. This familiar insect is reddish-brown, oval in shape, from four to five mm. (0.0574 to 0.1967 inch) long, and three mm. (0.1181 inch) wide. The female lays three or four times a year about 50 eggs, 1.12 mm. (0.0441 inch) long, which require about 11 months for their perfect development to the sexually ripe condition. They live in the crevices of beds, floors, and rafters, in furniture, behind wash-boards and wall-paper, in the habitations of man. During the day they lie concealed; at night they wander in search of the blood of the human being, which they draw by means of a long proboscis. The peculiar odor of the insect is due to a secretion of a special organ with which the bug is provided.

Human beings are variously susceptible to the bite of the bedbug, some being quite indifferent to it, others being, as it were, special favorites of the little creature.

Treatment.—The irritation is confined to the moment of the bite. The aim to be sought is the extermination of the insect. This is often difficult when a thorough lodgment is secured, and it is often necessary that all wall-paper should be removed as well as loose woodwork. Bedsteads should be thoroughly scalded and then treated with the following: Two tablespoonfuls of metallic mercury should be thoroughly beaten up with the white of one egg until a froth is attained. Apply freely with a small paint-brush, filling in carefully all cracks and crevices. The pest is less apt to invade iron bedsteads, but even these must not be neglected, for they, too, in careless hands, may become infested. Solution of corrosive sublimate, 2 to 1000, may also be applied in the same manner.

Order: Diptera.

Suborder: Aphaniptera (Fleas).

1. The *pulex irritans*, or common flea. Of these little creatures, the male is from 2 to 2.5 mm. (0.0787 to 0.0984 inch) long, the female as much as four mm. (0.1574 inch), red or dark-brown in color. It is also highly capricious in its tastes, disturbing some persons not at all, others seriously. It is not a parasite of man, and invades him usually because of its great abundance in certain places and countries. Though of world-wide distribution, it is more troublesome in hot countries where cleanliness of house-

hold, city, and person is a matter of indifference. The eggs are not laid on human beings, but in the cracks of boards, sweepings, and wooden spit-boxes.

Treatment.—The essential oils applied to the infested parts cause the retreat of fleas when applied.

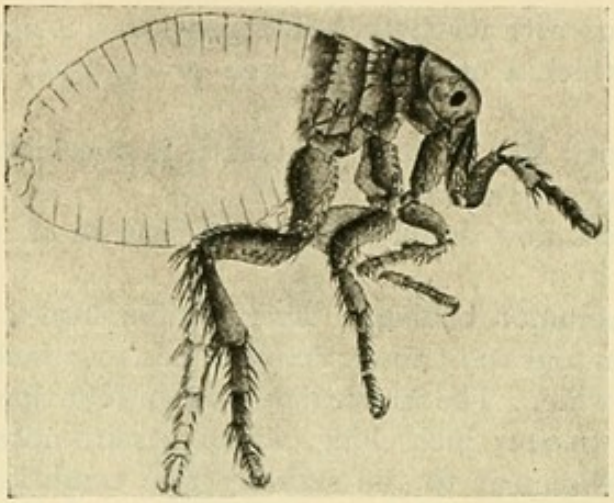


FIG. 86.—*Pulex irritans:* ×14. (*Braun.*)



FIG. 87.—Larva of *pulex irritans.* (*Gould.*)

2. The *pulex penetrans*, or sand-flea or jigger. The female buries herself in the skin of human beings as well as of dogs, swine, and other mammals, producing painful irritation, circumscribed swelling, and even suppuration. It especially attacks the feet. It prevails in tropical countries, especially in Central and South America. The eggs are land-hatched.

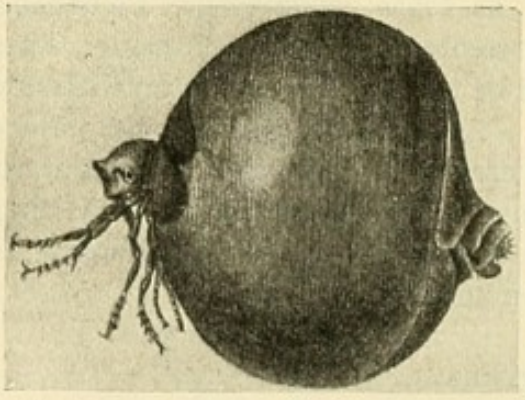


FIG. 88.—*Sarcopsylla (pulex) penetrans:* gravid female, enlarged. (*Braun, after Moniez.*)

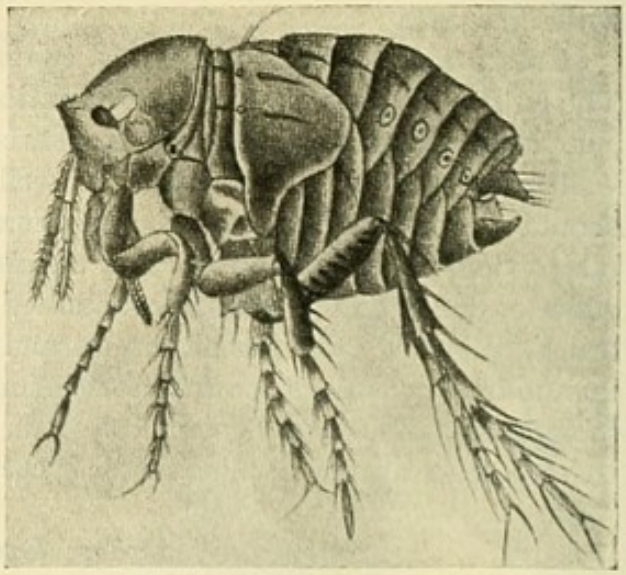


FIG. 89.—*Sarcopsylla (pulex) penetrans:* young female, enlarged. (*Braun, after Moniez.*)

Treatment.—The flea may be picked out with a needle, after which the essential oils are rubbed in on the parts to keep it away.

Suborder: BRACHYCERA (Flies).

Myiasis.—The diptera also contribute to parasites through their larvæ, which are deposited sometimes in open sores which have been neglected and

sometimes in the nasal passages and cavities—the ear, pharynx, vagina, etc. The condition is called myiosis, from the Greek μυία, a fly.

The most common of these is *myiasis vulnerum*, in which an ulcer becomes filled with maggots, which are the larvæ of the blue-bottle or common flesh-fly, *sarcophaga carnaria*.

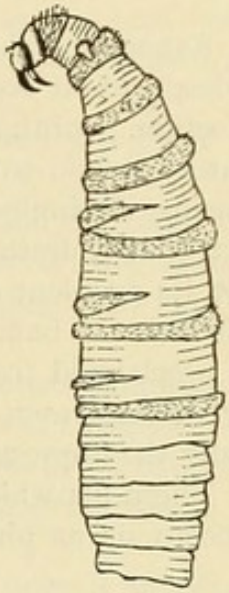


FIG. 90.—Larva of *lucilia macellaria:* ×4. (*Braun, after Conel.*)

Myiasis narium, aurium, conjunctivæ, vaginæ, etc., are due to the *lucilia macellaria*, whose larva is deposited in these situations usually when they are diseased, and may produce serious mischief, perforating mucous membrane and even cartilage. The larvæ of the *lucilia nobilis* have also been found in the auditory passages, producing ringing of the ears as a symptom. The larvæ of *sarcophaga magnifica* had been found in ulcers and other situations, throughout Europe, and especially in Russia.

Cutaneous myiasis is commonly due to the larva of the *hypoderma bovis* or bot fly, the female of which lays her eggs on the skin of cattle and sheep, in which the larva bores its way and forms the gad boil, about as large as a pigeon's egg. Rarely in tropical countries this happens in the skin of man. Cutaneous myiasis is sometimes caused by the larva of the *musca vomitoria*, one of the domestic flies. More frequently it causes internal myiasis, having been swallowed and again discharged by vomiting.

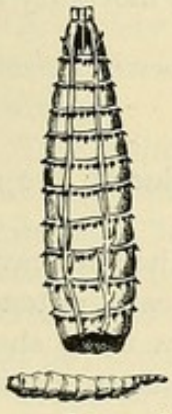


FIG. 91.—Larva of *musca vomitoria* (*calliphora vomitoria*): below, of natural size; above, enlarged. (*Leuckart.*)

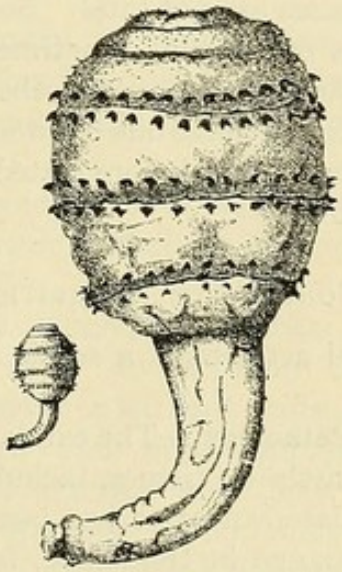


FIG. 92.—Larva of *dermatobia cyaniventris* ("Macaque worm: to left, natural size; to right, enlarged. (*Braun, after Blanchard.*)



FIG. 93.—Larva of *anthomyia canicularis*, enlarged. Rarely found in the stool. (*Gould.*)

In the tropics the macaque or moyaquil worm, the larva of a dermatobia, is not uncommon. More rarely dipterous larvæ are found in the feces, including those of the common house-fly and the *trichomyza fusca*, which has also been vomited.

GONOCOCCUS INFECTION.

Experience shows that the gonorrheal infection is no less harmful and widespread in its effects than syphilis. These effects include the primary infection, ophthalmia and gonorrheal arthritis, the majority of inflammatory pelvic troubles in women that make life a martyrdom and childbearing an impossibility. The explanation of this appears to lie in the fact that a urethral discharge continues to be infectious long after it has lost its purulent character, and the only test of recovery from gonorrheal infection is a bacteriological one. This is a startling statement, but should be proclaimed from the housetops if it will have any influence in preventing infected men from infecting innocent women whom they have married under the impression that they are free from disease.

These ills which have been referred to are largely surgical and do not concern us as physicians, for medical treatment is generally unavailing.

Gonococcus Septicemia.

This is a true generalized infection with the gonococcus, and has all the characteristics of streptococcic septicemia. Thayer and Bloomer, however, cultivated the gonococcus from the blood in Osler's wards, and the cases have been thoroughly studied by Cole since that time. The mortality, however, does not seem to be so high as the mortality from streptococcic septicemia. Sometimes there is an actual endocarditis complicating the conditions, and all of the cases are not fatal. Sometimes there is local suppuration with the features of pyemia; sometimes there is a long-continued fever without any local manifestations. At other times there is a purpural septicemia due to gonococci. Twelve out of twenty-nine cases, reported by Cole, died. It would thus seem as before stated, that the mortality is not so great as in streptococemia.

Gonococcus Arthritis.

Definition.—Gonorrheal arthritis is a septic arthritis due to the gonococcus.

Morbid Anatomy and Pathology.—The exudation into the joint cavity is rarely purulent. The periarthritic tissues, including the sheaths of tendons, are invaded by the exudate, and pus has been found in these sheaths. There may be not only change in the shape, but impairment also in the motility of the joints. They may become stiff and swollen.

Usually arthritis appears from six to ten days after the discharge is seen. It may appear, however, much later—as much as four or five months or even a year after the discharge sets in, or during a chronic gleet. A lately married woman may be infected by a husband who has gleet, indeed, as has been mentioned, after all visible objective signs of gonorrhea have disappeared from him, though a bacterial examination may discover the gonococcus. There seems to be no relation between the severity of the symptoms and that of the original disease. The discharge, if present, gen-

erally continues with the onset of the joint symptoms, although it often abates, and may even cease altogether for a time. It may even recur with the disappearance of the joint symptoms.

Gerhardt found that out of 928 cases of arthritis 7.43 per cent. were gonorrheal, while Gricolle found that out of 4423 cases of gonorrhea 16 per cent. developed arthritis.

Symptoms.—A study of these admits a classification as made by R. P. Howard, of Montreal, into seven subdivisions:

1. The purely *arthralgic* form, *i. e.*, cases characterized by pain, but not much other evidence of local inflammation. Fever is also absent, although the condition is apt to be polyarthritic, wandering from joint to joint.

2. *Gohorrneal Polyarthritis*, resembling very closely acute inflammatory rheumatism. In this division fever is added to the local symptoms of rheumatism. The fever, however, is less severe than would be expected from the severity of other symptoms. The maximum temperature may be 102° F. (39° C.), more frequently it is less than this.

3. *Acute gonorrheal monarthritis*, in which one joint only is involved, with severe pain and swelling and moderate fever. It is the knee-joint that is most commonly attacked in this monarthritic variety. Next in order follow the ankle, shoulder, elbow, and wrist; any one of these is liable to be the seat of the trouble. Suppuration is rare.

4. *Chronic gonorrheal arthritis*, without or with effusion (chronic hydroarthrosis). Suppuration, though rare, does take place and pus is found in the joint cavity. In these cases, too, there is generally slight elevation of temperature.

5. The *periarthritic* variety, including cases in which the periarthritic tissues are involved, including the capsule, ligaments, tendons, and adjacent fibrous structures. The periosteum is included among these, but the joint cavity itself is not affected.

6. A variety which invades *fibrous tissue not connected with joints*, as the plantar fascia, the sclerotic coat of the eye and iris, the pericardium and endocardium.

7. The *septicemic form*, where, in addition to the arthritis, there is general sepsis and endocarditis. In this event there are the usual signs of blood invasion, high fever with or without chills, and sweats.

Complications.—Isolated and even multiple cases of endocarditis associated with gonorrheal arthritis have been reported by German and French physicians during the past. The studies of Gluzinski (1888) and R. L. MacDonnell[1] (1891) have settled the question in favor of a causal relation, the latter having found endocarditis present in four out of 27 cases of gonorrheal arthritis, while Gluzinski collected 31 cases. They may reasonably be attributed to the action of the microorganisms on the valves. Malignant endocarditis may be thus caused.

Pericarditis and pleurisy similarly caused may complicate the disease, as may also iritis and sclerotitis.

Diagnosis.—This depends chiefly on the history of infection.

[1] "Gonorrheal Rheumatism," "Am. Jour. of the Med. Sci.," January, 1891.

Prognosis.—The disease is difficult to cure at times and prolonged treatment is necessary.

Treatment.—This is not always satisfactory. The primary urethritis must first be attended to. The salicylates frequently relieve the pain but have no effect on the morbid process. Iodid of potassium is perhaps the drug most commonly found useful, and its effect is increased when combined with the bichlorid of mercury. It must, however, be associated with rest and local treatment. Every gonorrheal joint should be *fixed* in a comfortable position by a properly applied splint. Often active surgical interference is necessary. A surgeon should be consulted in regard to the special form of local treatment. General treatment by tonics and good food may also be necessary. In selected cases gonococcic vaccine or serum may be tried, though its use has not been followed by the success anticipated.

RHEUMATIC FEVER.

SYNONYMS.—*Acute Rheumatism; Acute Articular Rheumatism; Inflammatory Rheumatism.*

Definition.—An acute febrile, infectious, but noncontagious fever, characterized by arthritis, usually multiple.

Etiology.—While no distinctive bacterium has as yet been isolated, Hermann Sahli found in inflamed joints in which there was no suppuration a bacterium closely resembling the *staphylococcus citreus*, and Leyden a diplococcus differing from that of pneumonia. F. J. Poynton and F. A. Paine with the diplococcus isolated from rheumatic fever have obtained in rabbits results which go to show that the organism with which they experimented is able to produce lesions of rheumatic fever, namely, mitral valvulitis, pericarditis, and polyarthritis. The diplococcus experimented with was obtained from the joints, from the throat in a case of rheumatic angina, from the bladder, and after death from the morbid product of rheumatic pericarditis and endocarditis. Again, by injecting a young rabbit with the organisms from the blood and cerebrospinal fluid of the infected rabbit they also produced polyarthritis and endocarditis in the second animal. Some of the animals recovered and others perished. In addition to the symptoms mentioned, there were wasting and involuntary clonic movements like those of chorea and the animal was also very nervous. With the chorea there was valvulitis.[1] In another instance the *micrococcus lanceolatus* was found. In view of the fact that several organisms have been found associated with rheumatic polyarthritis it may be true, as Flexner and Barker[2] suggested, that acute articular rheumatism has no etiological unity, but may be brought about by the entrance into the blood of one of several different pyogenic organisms under circumstances incompatible with the development of the phenomena of a general septicemia, but which may give rise to an inflammation of one of the several serous membranes, including the synovial, as well as the meninges, pleura, pericardium, or endocardium.

Among sources of infection always to be thought of are suppurative

[1] Communication to the Pathological Society of London, Tuesday, October 16, 1900; published in the "British Med. Jour.," October 20, 1901.
[2] "Am. Jour. Med. Sci.," 1894.

affections of the mouth cavity, such as tonsillar abscess, follicular tonsillitis, carious teeth and the stumps of roots of teeth. The studies of Goodale,[1] Geo. B. Wood,[2] Gurcih,[3] Isac Adler,[4] among others, go to show that the tonsils are the route of many general infections, of which rheumatic fever is the most common. In all cases of articular rheumatism the mouth should, therefore, be examined, and if lesions of the tonsils or adjacent parts are found, they should be treated and cured.

A *predisposing cause* seems, however, to be necessary in the majority of cases, and exposure to cold is the most common, although epidemics of acute rheumatism occur quite independently of such exposure. While sudden changes in temperature, also, often afford the needed conditions, the continued action of moderate degrees of cold, especially when accompanied by moisture, is almost as frequently responsible. If to these be added a lowered vitality due to insufficient food, fatigue, overwork, or all these combined, we include the majority of predisposing causes. The winter and spring, being the seasons in which the conditions of temperature and moisture operate most strongly, are those in which the disease is most prevalent. For a like reason it is more common in the temperate zones, the extreme North as well as the extreme South being for the most part exempt. It is a disease especially of young adults, being rare before fifteen and after fifty; while the exposing occupations, including those of driver, servant, and laborer, favor its development.

Morbid Anatomy.—There is little to be added to what will be described in treating of symptoms, and to what is furnished by the complications, whose morbid anatomy will also be considered in connection with the diseases that constitute them. The synovial membrane is hyperemic and swollen, and in some cases the fluid in the joints is increased, is turbid, and contains flakes of lymph, rarely pus. The fibrin of the blood is usually increased.

Symptoms.—While rheumatic fever is seldom ushered in by a *chill*, there is more frequently a short *prodrome* of a day or two, during which the patient feels uncomfortable or has an unpleasant aching feeling in his joints. More often, however, the *painful* arthritis, which is the first symptom to attract attention, develops rapidly, coming on in a single day or night, or seemingly in a much shorter time, making locomotion at once difficult or impossible.

The joint affection has some peculiarities. In the first place, the involvement is almost always multiple, and generally includes the larger joints, such as the knee, ankle, elbow, wrist, shoulder, and hip, although none are exempt, and the phalangeal and metacarpophalangeal articulations also suffer. The toe-joints escape most frequently. It rarely happens that a single joint is involved, but its occasional occurrence must be admitted. More rarely, if ever, does it happen that all are affected, although even the vertebral articulations are sometimes invaded. The inflammation is further characterized by a tendency to involve various joints in succession.

[1] Goodale, "Archiv for Laryngologie," vii, 1.
[2] Wood, The functions of the Tonsils, "University of Pennsylvania Medical Bulletin," 1904.
[3] Gurich, "Der Gelenkrheumatismus, sein tonsilarer Ursprung and seine tonsilare Heilung," Breslau, 1905, aus "Verhandlungen des Congresses für innere Medicin, 1905.
[4] Adler remarks on some General Infections through the Tonsil, "New York Medical Journal," March, 1906.

Now it will be the elbow, then the wrist; again, the knee, and then the ankle or shoulder or hip, either on the same side or the other; but while there will be a reduction in the degree of inflammation, and correspondingly of pain in the relieved joints, the relief will not be total. On another day, again, the pain will have returned to the joint which had been temporarily relieved.

While the joint-affection always includes a synovitis, the process is by no means confined to the synovial membrane. The whole joint is red, swollen and tender. The adjacent tissues, including the capsular and lateral ligaments, and the tendons, with their sheaths, coursing over the joint, and even muscles, are all the seat of involvement, contributing to the swelling and to the pain by the exudation pervading them. Comparing two hands, one of which is involved and the other not, the normal depressions between the metacarpal bones in the former may be obliterated by swelling, while they remain distinct in the latter. It is for such reasons that we prefer the name *acute rheumatism* to that of acute *articular* rheumatism, which would limit the process to the joints. Rheumatic fever is probably the best term.

Finally, mention should not be omitted of the nonarticular rheumatic fever to which Kohler[1] has called attention, in which there are no joint-symptoms.

The *pain* is almost always extremely severe, making all motion an agony, while jarring of the bed, or even the weight of bed clothing, may cause the patient to cry out. To diminish the tension, which aggravates the pain, the patient is disposed to lie with all the limbs semiflexed.

From the beginning there is *fever*, but being seldom high at this stage, it is not commonly the first symptom to attract attention. Later, it usually increases proportionately to the extent of joint involvement, but only in the meningeal forms is it extremely high. Nor does it pursue a course at all distinctive. In one case, for example, the temperature remained at 102° F. (38.8° C.) and a fraction, night and morning and throughout the day for a number of days. More rarely it rises to 104° F. (39.9° C.). Occasionally, however, there is intense hyperpyrexia, when the temperature rises rapidly from 104° to 110° F. (39.9° C. to 44.3° C.), and even higher. With this are associated cerebral symptoms of an alarming and dangerous kind, intense headache, and delirium—symptoms otherwise rather unusual in acute rheumatism. To these are often added unconsciousness, pulselessness, and cyanosis, rapidly followed by death, unless the temperature is promptly reduced. The sudden onset of these symptoms adds to their alarming character. This combination of severe symptoms is known as the meningeal form of rheumatism.

The *pulse* in rheumatic fever is rapid, often disproportionately so to the fever, probably because of the nervous demoralization caused by the acute suffering.

Next to the fever and joint-inflammation, the most distinctive symptom of acute rheumatism is the *sweating*, which is copious and usually acid in reaction, sometimes even to such an extent as to impart an acid odor to the air of the room. *Sudamina* are a frequent consequence of such profuse sweating.

[1] "Zeitschrift f. klin. Med.," Bd. xix, 1891.

Discolorations of the skin, varying in intensity and character, make their appearance in certain cases. There may be a simple diffuse erythema, or it may be papular or tuberculated or marginate. There may be true *urticaria*, or there may be extravasations of blood, *purpuric* patches of such extent and depth as to result in sloughing of the tissues, hemorrhages from the mucous membranes, and hematuria. In one case under our observation there ensued permanent blindness from extravasation into the retina. These cases of *peliosis rheumatica* are not acknowledged by all to be truly rheumatic, the joint-affection being declared to be of a different nature, analogous to that of scorbutus and hemophilia.

The *urine* is scanty, of high specific gravity, very acid in reaction, and deposits a copious sediment of pink-hued mixed urates.

Subcutaneous nodules, attached to tendons and fascia, which have long been observed as occasional events in connection with acute rheumatism, and have been especially studied by Barlow and Warner. They vary in size from a shot to that of a pea, and may be numerous or but few. They occur on the fingers, hands and wrists, elbows, knees, scapulæ spines of the vertebræ, and more particularly after the acuteness has passed away. They may last a few days or for months, and are more common in children than in adults.

Disposition to recurrence must be mentioned as a characteristic feature of acute rheumatism. Quite rarely does a person who has had one attack escape another, and it is these successive attacks which, augmenting previous cardiac lesions, finally cripple the heart until its work is greatly hampered. The intervals between successive attacks are various—from a year to four or five years—and they are the more frequent and more liable to occur the younger the subject.

Complications. *Pulmonary Affections.*—Both pneumonia and pleurisy may occur, usually as the result of an extension of the pericarditis, or from the more severe cardiac involvement—carditis.

Cardiac disease, including endocarditis and pericarditis, carditis and myorcarditis, the former being by far the most frequent, and confined almost exclusively to the left leart. Again, the mitral leaflets are much more frequently attacked than the aortic. While the cardiac involvement bears some relation to the severity of the disease, the mildest cases may become complicated as well as the severest. Hence, during an attack of rheumatism of whatever severity, the heart should be daily examined, a further reason is that the approach of the cardiac complication is often exceedingly insidious. Cardiac oppression and palpitation may occur, however, without actual structural change, and even a functional murmur may be present in acute rheumatism, and this, too, not only at the base, but also at the apex of the heart, an unusual site for such a murmur.

The proportion of cases in which cardiac complications occur, though difficult to estimate, is not less than 25 to 33 per cent. for endocarditis, with 10 per cent. more for pericarditis, making in all 35 to 43 per cent., while some estimate even a larger proportion.[1] Young subjects are more vulnerable than adults, and Fagge mentions an interesting difference in the sexes

[1] De Lancey Rochester in a paper published in the "Jour. of the Am. Med. Assn.," December 15, 1900, says 60 per cent. for endocarditis and 10 per cent. for pericarditis.

after adult life, which is, that *pericarditis* is more frequent in men above 25 than in women of the same age, probably because at this age men work much harder than women.

The variety of endocarditis is usually the verrucose, or warty—ulceration, laceration or perforation of the valve flaps being very rare. The malignant form of endocarditis does, however, occur. While the endocardial murmurs in the endocarditis of acute rheumatism are commonly soft, the pericardial murmurs are often loud, rough, and rasping, and the vibration resulting from the friction may even be communicated to the hand laid upon the precordium. Both conditions may result in complete recovery, but the former more commonly is the beginning of a chronic valvular defect and the latter may result in adhesive pericarditis.

Carditis may occur where all the structures of the heart are involved.

Myocarditis is not uncommon. In the grave cases of carditis it is one of the must serious features.

The *sequelæ* directly traceable to acute rheumatism are also few. Chorea, acute nephritis, are among those so regarded. The nephritis is, perhaps, better considered a complication resulting from the same cause, just as is the endocarditis.

Diagnosis.—The diagnosis of acute rheumatism is seldom difficult, the multiple painful involvement of the joints, the fever, and sweating seldom mean anything else; but pyemia and scarlatinal and gonorrheal arthritis must be remembered as possible events. It is the monarticular variety which demands most discrimination in its determination. Traumatic synovitis, tuberculosis, gonorrheal infection, and the so-called nervous arthropathies are to be eliminated.

It is not always easy at first visit to distinguish *gout* from acute rheumatism, but the most serious possible error in diagnosis is to mistake a pyemic arthritis for a rheumatic arthritis. This is not an uncommon mistake where there is no evident surgical lesion to suggest it. Osteomyelitis is said to be the most common cause of such pyemias; but other bone diseases, puerperal sepsis, and gonorrhea are also causes.

Acute arthritis deformans frequently begins exactly as an acute rheumatic fever. The longer course, its failure to react to salicylates and the finally deformed joints make the diagnosis.

Prognosis.—The course of acute rheumatism is characterized by many fluctuations independent of treatment, and its duration is various. Sooner or later recovery generally takes place, although it may be with a crippled heart and a susceptibility to return. More rarely the attack passes over into a subacute condition which makes the patient a sufferer for a long time.

Subacute Rheumatism.—This term is applied to forms in which all the symptoms are less marked and more prolonged. The fever is not so high, ranging from 99° to 101° F. (37.2° to 38.3° C.). The inflammation of joints is not so intense and the joints involved are less numerous. It exhibits the same "flying" tendency. It may also be associated with cardiac complications, especially in children.

Treatment.—Absolute rest in bed, and fixation of the affected joints are of prime importance. Many cases are prolonged and many complica-

tions occur from neglect of those important measures. The drug which is most useful is salicylic acid or one of its preparations.

Salicylic acid and salicylate of sodium are equally efficient, but the former has been largely superseded by the latter, because less irritating and easier of administration. Still better borne is strontium salicylate. Whichever is used, there is one necessary condition of its efficiency, and that is its constitutional impression. The aim in the administration is, of course, to relieve the patient, but this effect is seldom obtained or, if obtained, is of fleeting character, until the peculiar ringing in the ears is secured. To do this in the adult 1 1/2 to 2 drams (5.8 to 7.7 gm.) of salicylic acid and from 2 to 3 drams (7.7 to 11.6 gm.) of the sodium salicylate in the first 24 hours are required. If the salicylic acid is given, it should be in capsules or compressed pills containing 7 1/2 to 10 grains (0.49 to 0.65 gm.) every two hours, followed by a little water or milk. This drug is now rarely used.

The salicylate of sodium may be given in doses of 10 to 15 grains (0.65 to 1 gm.) well diluted every three hours or oftener, if the pain be severe, until relief comes, after which it should be kept up until the toxic effect is produced, when the dose should be diminished, but the drug continued; or the interval may also be prolonged. The doses laid down may be pushed more rapidly if the suffering is extreme, but it is seldom necessary. Under this treatment the pain fades away, the swelling diminishes, and the anxious expression of the patient is changed to one of comfort in from 24 to 48 hours. Those who object to the salicylate treatment do so on the ground that the relief is not permanent, and it must be admitted that relapses do occur; however, this is often because the remedy is discontinued too soon. As stated, the drug, while it should be cut down with the appearance of relief and toxic effect, must be continued for some time after relief is obtained.

We should not, however, rely wholly upon the treatment by salicylates. Warmth is commonly a useful adjuvant, and to this end the joints and limbs should be kept surrounded by warm flannels or carded wool or cotton. The patient should, further, sleep between blankets and in a flannel gown so made that it may be easily removed, with split sleeves and split skirt, because of the extreme sensitiveness of the sufferer. The bed, if possible, should be narrow because of greater convenience in handling. The joints should be surrounded by cotton or flannel soaked in a saturated solution of magnesium sulphate or lead water and laudanum, both these applications frequently giving much comfort to the patient.

Sometimes the salicylates are not tolerated by the stomach, even in the smallest doses likely to be useful. They may then be given by injection as follows: The rectum is washed out with warm water, and after a short rest, 20 to 40 grains (1.3 to 2.6 gm.) or more of sodium salicylate in solution are injected well up into the bowel. This may be done once in six hours with the happiest result, as I can attest from personal experience. If larger doses are thus given, 90 to 120 grains (6 to 8 gm.) being recommended by some, it is well to guard them with a little tincture of opium.

But the salicylate treatment is not always successful, even when the drug is well borne. Then the oil of wintergreen, which contains 90 per cent. of salicylate of methyl, may be tried, in doses of 10 to 15 minims (0.6 to 1 c.c.) every two hours, in capsules or in emulsion. Or it may be alternated with

the salicylate, if it be a question of tolerance of the latter, the gaultheria being usually better borne for a time by the stomach.

Oil of gaultheria is also used locally, at times with excellent results. It may be used as an embrocation in the proportion of one part of oil of gaultheria to two parts of olive oil. More usually it is applied to the affected joint on lint, which is thoroughly moistened with the oil, wrapped about the joint, and surrounded by gutta-percha, oiled silk, or other impermeable covering to prevent evaporation. This is further prevented by bandaging the whole limb. That the salicylate of methyl is thus absorbed is seen from the fact that salicyluric acid appears in the urine a few days later, while the usual evidence of the physiological action of salicylates—viz., headache or fullness of the head with ringing in the ears—takes place. In view of the gastric disturbances which the salicylates cause in some persons, this mode of administration should not be overlooked. Salophen, acetyl salicylic acid (aspirin) and phenol salicylis (salol), may be substituted for the salicylates when the latter are not well borne.

The *alkaline treatment* of acute rheumatism, most relied upon before the salicylic treatment came into vogue, is a treatment which is by no means worthless. This, originally instituted by Sir A. Garrod, received an additional impulse from H. W. Fuller, who insisted upon the administration of such doses as secured and maintained an alkaline reaction of the urine. This is accomplished by sufficient doses of almost any of the alkaline salts, as potassium citrate, potassium acetate, sodium carbonate, or *liquor potassæ*. Twenty grains (1.33 gm.) every two hours of the first three are generally sufficient, or 20 minims (1.3 c.c.) of the last. The dose may then be reduced, but enough should be given to maintain the alkalinity of the urine.

Failing for any cause in the treatment with salicylic acid, the alkaline treatment, or what is called the "mixed" treatment, may be employed. By this is meant the combined alternate use of the salicylates and alkalies. This may be tried, for example, where sufficient doses of the salicylates are not well borne by the stomach, when they may be supplemented by alkalies.

For relief of pain, opium or its derivatives is sometimes necessary, but less frequently than formerly. Here, again, the hypodermic injection of morphin, 1/4 grain (0.016 gm.) is most comforting, but sometimes Dover's powder in 10 grain (0.6 gm.) doses acts most kindly. Phenacetin, acetphenadin, aspirin, acidum, acetyl salicylates, or acetanilid, may be used for milder degrees.

The treatment of the *hyperpyrexia* of acute rheumatism must be prompt and energetic, as the danger to life is imminent, the extraordinarily high temperatures thus encountered being inevitably fatal in a few hours. There is but one treatment. It is the application of cold. The bath is to be preferred, although in its absence affusions of ice-cold water and rubbing the head and body with ice may be substituted. As soon as the temperature begins to mount rapidly above 105° F. (40.3° C.) it should be used, and if delirium or unconsciousness is associated with such temperature, its need is even more imperative. When time permits, the application of cold may be more gradual. Thus the patient may be put in the bath at 70° F. (21° C.) and the temperature further reduced, if necessary, by the addition of ice or colder water. As stated, there seems now to be no doubt about the necessity

of this treatment. Numerous cases of recovery have been reported, some even where the temperature had reached 107°, 108°, and even 109° F. (41.6°, 42.2°, 42.7° C.). With the reduction of temperature, the cerebral symptoms gradually disappear.

As the disease becomes more subacute or chronic, the necessity for more active local and tonic treatment becomes urgent. It would seem that at such a stage the pathogenic cause has exhausted itself, and the disease has become more a local one, maintained by the dyscrasic state of the blood, itself brought about by the prolonged suffering. Hence treatment with iron, arsenic, and nourishing food becomes necessary. Indeed, the patient with acute rheumatism should be well fed throughout. Massage is especially valuable, and often surprisingly soothing ultimately, even although at first somewhat painful, while by it the mobility of the joints may be gradually restored. There results sometimes in the muscles in the neighborhood of the joint, and especially in the case of the shoulder, a paretic state, which is also benefited by massage, especially when associated with electricity.

When a case runs over weeks, the focus of infection, ear, sinus, tonsil, or suppurating area should be carefully sought for. Excision of the tonsils has been followed by the cure of subacute and chronic cases, confirming the fact that these organs may be a route of infection.

Diet in Rheumatic Fever.—The diet of the patient with rheumatic fever should be simple and easily assimilable, but nourishing. While there is fever the food should be liquid, but the rule of conduct should be: feed well—do not starve.

PNEUMONIA.

CROUPOUS PNEUMONIA.

SYNONYMS.—*Pneumonitis; Lobar Pneumonia; Fibrinous Pneumonia; Genuine Pneumonia.*

Definition.—An acute infectious disease characterized by inflammation of the lungs and high fever, usually terminating by crisis in from five to nine days. A bacterium especially prone to occur in pairs or chains, known as the *pneumococcus, diplococcus pneumoniæ and micrococcus lanceolatus*, is found in 75 per cent. of all cases of lobar pneumonia and is commonly regarded as its cause.

Varieties.—The term *lobar pneumonia* is used for this form because it generally involves at least a single lobe or the greater portion of one. The term *pneumonia of the apex* is used where one or both apices of the lung are involved. A rare form of croupous pneumonia is *double pneumonia* in which both lungs are involved, though not necessarily the whole of each lung. A *massive pneumonia* is an inflammation not only of the air-vesicles, but of the bronchi and interstitial tissue of a lobe or even of the whole lung. A *creeping* or migratory pneumonia affects successively different lobes of the lung. *Epidemic pneumonia* invades large numbers or communities. The term *larval pneumonia* is applied to a form of the disease in which but a partial development of symptoms occurs, such as a moderate chill, slight

fever, and imperfect local signs. It is found more particularly in connection with epidemics or with pneumonias in crowded places, as ships, camps, and garrisons.

Etiology.—The *diplococcus* of Fränkel, to which the name Weichselbaum has also been added, is the true pneumococcus. It occurs in pairs, sometimes in rows or beads. It is also pointed at one end. When stained by the carbol fuchsin solution the occus is intensely red, while the capsule assumes a light reddish tint. It can also be stained by Gram's method. It thrives on agar and in bouillon, but not on gelatin. It is probably the same organism as that found by Sternberg in rabbits inoculated with his own saliva in 1880, but not announced until April, 1881. Pasteur had also recognized the same organism in the saliva and published several notes on the same subject, January to March, 1881. The coccus occurs, according to Netter, in 20 per cent. of *all* persons. Fränkel, Talamon, and especially Weichselbaum showed the relations of this organism to pneumonia. The latter found it in 92 per cent. of cases of croupous pneumonia. William H. Welch found it in every one of ten cases of croupous pneumonia studied at the Johns Hopkins Hospital at Baltimore. It has been found in the blood, in the spleen and kidney, in endocardial vegetations, in the spinal fluid as well as in the saliva of healthy persons and in the dust on the floors of houses. Its route of entrance is probably the respiratory passages, since it has been found in the nose, larynx and Eustachian tube, and is said to persist for months and even years in the saliva of healthy persons who have had pneumonia. On the other hand, it is a very perishable organism, maintaining its virulence outside of the body for four or five days only.

That the pneumococcus of Fränkel is not the only organism capable of producing pneumonia is, however, evident from the experiments of Fränkel himself, of Weichselbaum, and of Pansini and Neumann. It may be accompanied by pus organisms and others which may be responsible for complications and modifications of the ordinary pneumonic process. Streptococcus-pneumonia has come to be recognized as a variety of pneumonia having a more or less distinct clinical picture that will be again referred to.

Pneumococci have been found in cultures from the blood by many observers, but by no means uniformly in pneumonia. The presence of pneumococci in the blood is said to emphasize gravity of prognosis, constituting a pneumococcic septicemia.

Nature of Pneumonia.—Thus *caused*, pneumonia may be regarded as a general disease with a local expression in the lungs, analogous to the inflammation of Peyer's patches in typhoid fever. As in the case of typhoid fever, there were facts which pointed to the infectious nature of pneumonia long before the discovery of any organism that could be regarded as its specific cause. The occurrence of pneumonia in epidemic form was recognized by Laennec and Grisolle, and since their day innumerable epidemics have been described—house epidemics including those in which a number of individuals, from three to ten or more, have been attacked under the same roof, and general epidemics, invading institutions, ships, and garrisons, in which large numbers of persons are congregated. Out of a ship's crew of 815, 410 were attacked in rapid succession, and out of 720 attacked, 298 perished.

The lowered vitality consequent on exposure must be regarded as a predisposing cause, preparing the system for the operation of the ever-present organism as the exciting cause. Pneumonia is much commoner in the winter months. Other predisposing causes are: a *previous attack*, fatigue of mind or body, and debilitating conditions of all kinds, such as previous or present illness, especially a chronic complaint, such as Bright's disease. Traumatism is certainly a cause. Pneumonia may follow a traumatism without actual injury to the lung having been done.

Morbid Anatomy.—The lung in croupous pneumonia exhibits three distinct stages:

1. Congestion or engorgement.
3. Red hepatization.
3. Gray hepatization.

Pneumonia seeks, by preference, the lower lobes of the lungs, and the right lung more than the left. Pneumonia of the apex, however, not infrequently occurs, more often in children than in adults.

The Stage of Congestion.—In this stage the lung is engorged with blood, yet permeable to air. The capillaries surrounding the air-vesicles are turgid and intrude upon the lumina of the air-vesicles. There is a small amount of transudate, in which may be found a few exfoliated alveolar cells and red blood-disks. The part of the lung invaded is redder than normal and heavier, but not nearly so heavy as in the next stage. On section, blood transudes from the cut vessels and bathes the surface.

The Stage of Red Hepatization.—In this the lung is dark red in color, hard, and very much heavier than in health—as much as three and four times the normal weight. A piece dropped in water rapidly falls to the bottom. The lung pits on pressure, and in consequence the marks of the ribs are often seen on it after removal. On section the aptness of the name red hepatization is at once apparent. The surface is darker in color than in the first stage, and it has the appearance of a section of liver. On passing the finger over it, innumerable little hard spots like grains of sand are felt. These are air-vesicles filled with the croupous exudate. Corresponding to this, a granular appearance is recognized by the eye, the distended air-vesicles appearing as glistening points. By scraping, little plugs of fibrin and cellular detritus mixed with serum can be removed. The lung, though thus hard, is nevertheless friable, and may be broken up by the fingers.

Histologically, the air-vesicles are found to contain a delicate reticulum, the meshes of which are filled with red blood-disks, and with alveolar cells in different stages of degeneration, including numerous granular fatty cells or compound granular cells. The vesicular walls are found infiltrated with lymphoid cells, which extend even into the interlobular tissue beyond them. Plugs of fibrin may sometimes be traced into the smaller bronchi from the air-vesicles.

The *pneumococcus* may be demonstrated in cover-glass preparations made from the exudate. It may be associated with the streptococcus and staphylococcus.

The Stage of Gray Hepatization.—This is also well named, the cut lung exhibiting a grayish-white coloration. It is still dense and heavy, but much moister and softer, and more friable. The granulations are less distinct,

and on microscopic examination the alveoli are found filled with white blood-corpuscles, while the red corpuscles and fibrin filaments have disappeared. Sometimes all three stages are seen alongside of one another.

A stage beyond gray hepatization is sometimes spoken of as a *stage of purulent infiltration*. In this stage the lung has assumed a more yellowish appearance, it is much softer, almost liquid in consistence, and more like pus. On minute examination the air-vesicles are filled with pus-cells, the points of greatest softness constituting small abscesses as large as a pin's head and larger. The stage of gray hepatization is the stage of beginning resolution, while that of yellow hepatization represents the same stage in which the proportion of leukocytes undergoing fatty degeneration is larger.

If recovery takes place the contents of the air-vesicles liquefy, the product being mostly absorbed.

The *pleura adjacent* to the inflamed lung is almost always inflamed, the most distinctive sign of this being a plastic deposit. There may also be thickening and some serous effusion.

After death from pneumonia, the *heart* is found in a pathological condition typical of the disease. The left cavities are generally found empty or nearly so, while the right are distended with firm coagula, which often extend into the branches of the pulmonary artery. The *spleen* is often enlarged. The cells lining the *renal tubes* are often found in a state of cloudy swelling; rarely there is nephritis.

Symptoms.—Perhaps no other disease except malarial fever is so invariably ushered in by a *chill* as is croupous pneumonia, and often a chill of great severity. It may come on at night, waking the patient out of a deep sleep. It may or may not be preceded by a day or two of *prodromal discomfort*, with *headache*, which may be very severe. Almost immediately there succeeds a high fever, in which the temperature rises rapidly to from 103° to 105° F. (39.4° to 40.5° C.). A significant *flush on each cheek* is characteristic, occasionally more marked on the affected side. The pulse is full and strong, resisting pressure, rate 100 to 120. There is *thirst*, and *the urine is scanty and high colored*, sometimes albuminous. Equally promptly ensues a *pain in the side*, which may be dull, but is often also sharp and severe, caused in the latter instance by involvement of the pleura. The *respirations* rise rapidly in frequency, and there is *cough*, at first dry and hard. It is often restrained on account of the pain it occasions. Soon there is a small amount of *mucous expectoration* from the coincident bronchitis, but usually in 24 to 48 hours after the chill the sputum exhibits distinctive characteristics. It is tenacious, light red in hue—"rusty"—and is ejected from the mouth with difficulty. At other times it is much thinner and darker, and has received the name "prune-juice" expectoration. The amount of blood and the degree of coloration vary greatly. The *respirations* are exceedingly rapid—30, 40, 50, 60, and even more in the minute. The appearance of a patient at this stage is very striking. The face is flushed, the eye is brilliant, the breathing is rapid, the alæ nasi move with each breath, while a frequent short cough, held back until irresistible, increases at times the already anxious expression of the patient. There is practically always a leukocytosis the white cells averaging from 16,000 to 25,000 per millimeter.

This state of affairs continues unchanged for from five to nine days, when, if recovery takes place, a sudden drop in the temperature occurs, accompanied often by free perspiration, while a state of comparative comfort succeds to one of great distress, to be further followed oftentimes by a long and refreshing sleep. This is known as the *crisis*. It may be preceded by a fall of temperature a day or two earlier, which is again followed by a rise, whence such fall is called the pseudo-crisis. The accompanying temperature chart (Fig. 94) illustrates the actual crisis. The fall during crisis is sometimes as much as 7° F. (12.6° C.) in 24 hours, and the minimum is quite often slightly subnormal, whence it rises rapidly to the normal. Sometimes the fall takes from two to four days when the case ends by lysis.

From this point onward convalescence is rapid, and in four or five days more the patient is seemingly well, the temperature and pulse-rate normal, the breathing natural. A muscular weakness and vulnerability, however, remain, which demand care for a long time.

Physical Signs.—The physical signs of a typical pneumonia are very distinctive.

The *first*, or *stage of congestion*, in which the air-vesicles are still open, is of short duration, terminating within the first 24 hours, and may therefore be overlooked. *Inspection* shows the face flushed, increased frequency of respiration, with restricted movement upon the affected side and increased excursion of motion on the sound side. The patient lies by preference on the affected side because of the greater comfort it gives him. This posture not only diminishes the pain by hindering the motion of the affected side, but also lessens the dyspnea by permitting unrestrained expansion of the side that is doing the work.

Palpation at first may even find vocal fremitus diminished on account of the relaxation of the air-vesicles, but it becomes decidedly increased as the latter fill up. The skin is hot and the pulse is frequent, full and strong, as a rule. *Percussion* obtains but slight, if any, impairment of resonance. In fact, tympany, or the vesiculo-tympany of Flint—Skoda's resonance—may be present in this stage as a result of the relaxation of the partially filled air-vesicles, giving resonance by immediate relaxation. In the latter part of the first stage there is, however, impairment of resonance.

Auscultation in the very earliest stage may find the vesicular murmur feeble and less well heard over the affected area, but very soon is heard the distinctive physical sign of the first stage of pneumonia, the *crepitant râle* at the end of inspiration. If there be coincident pleurisy—the closely simulating friction sound may be added. Under such circumstances it may be difficult to distinguish between these two physical signs. Over the normal part of the lung there is exaggerated vesicular breathing.

But all these physical signs, even if carefully sought for, may be wanting if the pneumonia be central and deep-seated, as is not infrequently the case. They appear as the surface is approached, or they may not be recognized at all if the disease remains central.

The *second* stage, or *stage of red hepatization* or solidification, lasting four or five days, furnishes unmistakable signs. All the signs revealed to *inspection* in the first stage are intensified in the second, and the breathing

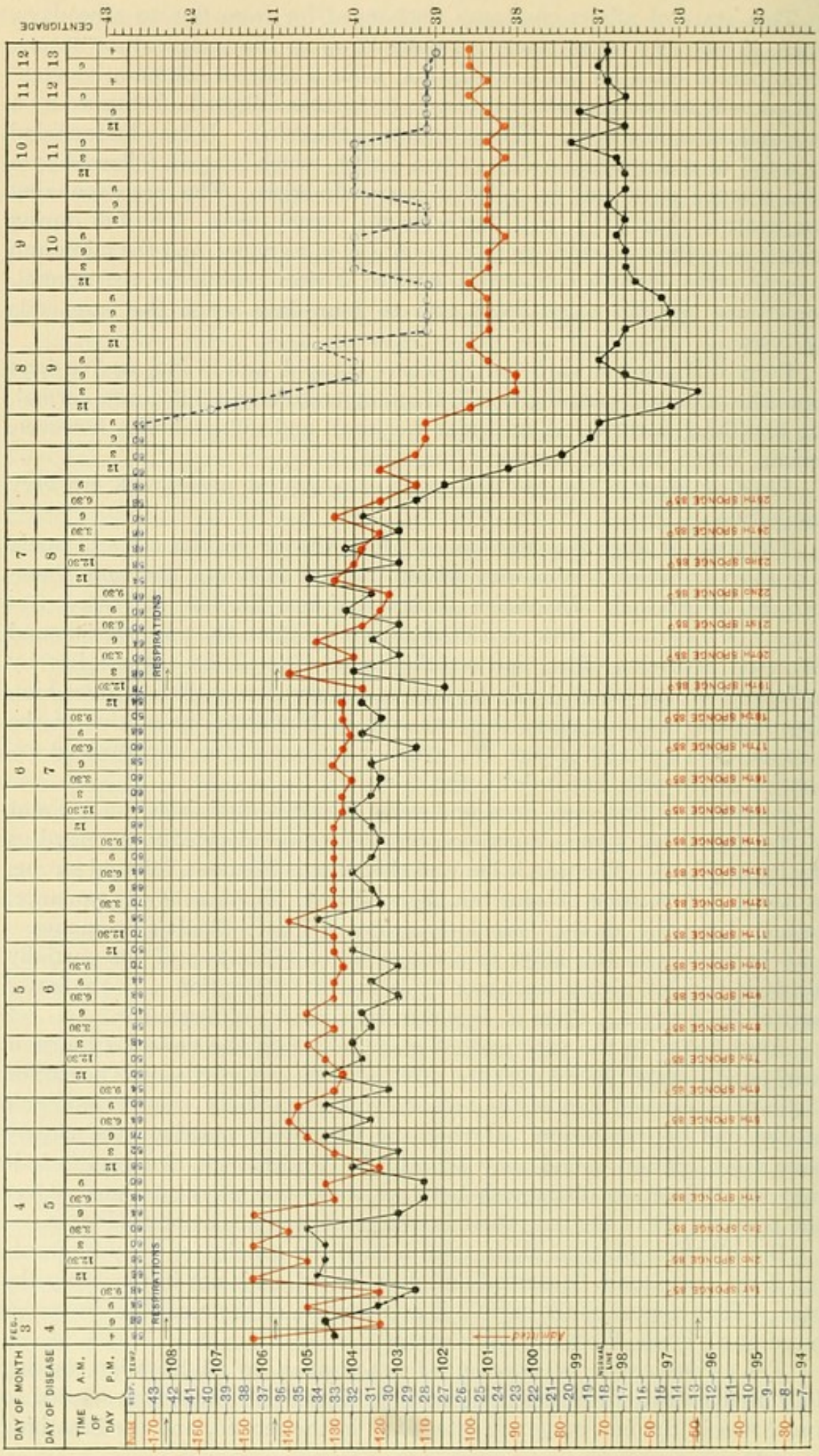


FIG. 94.—Showing Crisis in Pneumonia.

is markedly abdominal. To *palpation*, vocal fremitus is now increased. Occasionally the fremitus is decreased on account of a layer of liquid or a plugged bronchus. The skin is hot and dry, and the pulse continues frequent. Mensuration almost always and even inspection may recognize an enlargement of the involved side, the former to the extent of 0.5 to 2.5 cm.

Percussion gives dullness over the solidified area, with high pitch and short duration, except in those very rare instances where the extreme consolidation throws the column of air in the trachea and bronci into vibration, producing tympany. Over the adjacent normal areas, also, resonance is exaggerated, not so much, perhaps, in consequence of supplemental function as from relaxation of the adjacent air-vesicles—Skoda's resonance by mediate relaxation. Even cracked-pot sound may be produced by percussion over the solidified lung as a result of the sudden expulsion of air from a large bronchus leading to the solidified area.

Auscultation discerns high-pitched bronchial breathing over the solidified lung. Indeed, these are the circumstances that give the typical bronchial or tubal breathing. The air-vesicles are obliterated, and the resulting excellent conducting medium brings the tracheo-bronchial blowing to the ear. In very rare instances, when the larger bronchi are filled with exudate, there may be no bronchial breathing. The ausculted voice gives us typical bronchophony and occasionally even pectoriloquy, as well as whispering bronchophony and pectoriloquy. The heart-sounds are also heard with great distinctness over the consolidated lung, owing to the improved conduction, while the sounds of a concurrent bronchitis are similarly intensified. A lingering crepitant râle may also be heard.

The *third* stage, or *stage of gray hepatization* or resolution, occupies six to ten days. It repeats largely, to inspection, palpation, and auscultation, the phenomena of the first. Resonance continues impaired for a variable time, the lung sometimes clearing up in a couple of days, at others in a longer time. The normal manner of breathing gradually returns, the temperature of the skin is notably less, the crepitant râle returns, technically known as the "crepitans redux," and is finally replaced by the normal vesicular breathing sound, by which time the dullness has disappeared.

Croupous pneumonia may rarely terminate in *abscess* or *gangrene;* in either event the signs of the second stage continue and the temperature does not fall—in a word, the crisis does not occur. No changes in the physical signs take place as a rule, and it is rather by the general symptoms, viz., the failure to recover, the continued high temperature, the expectoration of pus, and, in the case of gangrene, the intensely disagreeable odor, that we are informed of the issue. The termination in abscess probably represents on a large scale what takes place in every instance in minute areas in the third stage of all pneumonias which terminate favorably. The occasional termination in tubercular phthisis exhibits a similar arrest of the resolving process in the second stage, and the phenomena of catarrhal or fibroid phthisis supervene.

The obscuring effect of a thickened pleura upon all these signs is to be remembered, and too much stress cannot be laid upon the fact that we may have a central deep-seated pneumonia that may give no physicial signs; also

that in old persons the physical signs of a pneumonia are very apt to be delayed from one to three days.

Careful differential percussion and palpation may recognize a moderate enlargement of the *spleen*.

The *heart* should be carefully watched in pneumonia. The sounds, at first loud and clear, becomes less so as the disease progresses and the lungs become engorged. The pulmonic second sound is particularly sharp as long as the heart is strong, and its failure is an unfavorable sign, as it means that the right ventricle is failing in power and may be yielding to distention.

Modifications in Symptoms and Special Symptoms.—The foregoing is the course of a typical case of pneumonia, perhaps of three-fourths of all cases, and the symptoms mentioned suffice for a diagnosis. All of them are, however, subject to modifications.

Thus, the *chill* may be absent or imperfectly developed, in which case all the symptoms arise more gradually. The *temperature*, especially in old persons and drunkards, may not be nearly so high; in children it may be higher. The same is true of the *respirations*, which may be increased to 100 to the minute in children. *Pain* is especially absent in old persons, *cough* and *expectoration* also, so that a careful physical examination of the lungs should be made in all ailments in the old and in drunkards also, as it not infrequently happens that pneumonia is overlooked in them. The *pulse* is often feeble and rapid instead of full and strong. Nay, more, even the *physical signs may be absent in the old*, and they are especially apt to be delayed in their development. It is unsafe to say of an old person at the first visit, after a negative physical examination, that he has not pneumonia, for the physical signs may not make their appearance until the second or third day and even later. It would seem, too, that central pneumonia is more common in the old than in the young, while even an afebrile pneumonia is a possibility in the old. Even in younger persons the appearance of physical signs is sometimes delayed three or four days.

The *expectoration* varies a good deal when present, especially as to the quantity of blood. Sometimes it is bright red and quite liquid, almost like a hemorrhage. More frequently it is viscid and glutinous, simply stained with blood. The term "prune-juice expectoration" has long been associated with pneumonia, and sometimes, when it is thin and dark-hued, the comparison is an apt one. Under the microscope the sputum is found to contain blood-discs, leukocytes, and alveolar epithelium in various stages of degeneration, including numerous compound granule-cells, also ciliated epithelium. *Fibrinous bronchial coagula*, sometimes large enough to be seen by the naked eye, are also met with in the expectoration, and, after suitable staining, diplococci. Should gangrene supervene, the expectoration becomes very fetid.

The *urine* is especially characterized by a reduced amount of chlorids, which are often absent until the crisis is passed, when they reappear. It is supposed that during this period they are transferred to the exudate in the lungs. A trace of albumin is often present and it presents the other features of febrile urine.

There is sometimes *marked jaundice*. It may even be the first symptom.

The cases attended by it are rather more serious. Sometimes the jaundice is of catarrhal type. Frequently it is due to a hemolytic action of the toxin liberated.

The *blood* exhibits usually a *leukocytosis*, the number of corpuscles being increased from 6000 per cubic millimeter to 19,000, or more. As many as 68,000 have been found. A moderate leukocytosis is regarded as a favorable symptom. The increase is almost always in the polymorphonuclear cells. The proportion of fibrin is also increased from four to ten parts in 1000. This increase of fibrin shows itself also on the microscopic slide in the shape of filaments of fibrin. According to Hayem, the blood-plaques are increased.

Herpes is very common on the lip—present, it is said, in from 12 to 40 per cent. of all cases. It may occur elsewhere, as on the nose and genitals.

Phlegmasia alba dolens, or milk-leg, is a rare sequel. J. M. DaCosta[1] collected nine cases, of which three were his own. The complication occurs late and has been more frequent in the left leg, and in women. W. H. Welch, W. R. Steiner, Sears and Larrabee and D. J. Milton Miller have increased the cases reported to 48 up to 1903.[2] They are due to bacteria or their toxins not always from the concurrent pneumonia, but from streptococcus and other cocci.

When typhoid fever coexists with croupous pneumonia the tongue is coated, and becomes dry and leathery. Constipation is usual, but occasionally there is diarrhea, especially in epidemics. Except in typhoid cases *delirium* is not common, but may be very active in the young. In old persons it may be low and muttering. In drunkards, in whom the disease is common and very grave, especially after a debauch, the delirium may be taken for *mania a potu*, or the two may coexist. Such a patient may rise from his bed and wander out into the city or to another hospital that he prefers, having just intelligence and strength enough to accomplish this purpose and will die after its attainment,

Streptococcus-pneumonia has been mentioned, with the statement that it presents some clinical features different from those of the ordinary croupous pneumonia, at least at times recognizable. We must say, however, that we have never been able to recognize such pneumonia by these symptoms, since many of them are the same as those heretofore regarded as peculiar to bronchopneumonia as ordinarily caused. In the first place, it is held that the serious form of pneumonia, which often complicates influenza, is thought to be a streptococcus pneumonia. Such pneumonias, like bronchopneumonia, commonly begin obscurely, are atypical, while the local signs are slow to develop. The rusty expectoration is delayed; in like manner the crisis which may be substituted by lysis; or death supervenes instead of crisis. The physical signs also rather resemble those of bronchopneumonia, while it is said[3] that the disease is more frequently found in the upper lobe, not at its apex, but in its lower part between the inferior angle of the scapula and the axilla. It may also be irregularly migratory. The sputum may be mucopurulent at the outset, and is always

[1] "Philadelphia Med. Jour.," vol. ii., 1898, p. 510.

[2] See Miller's paper in "Philadelphia Medical Journal," May 16, 1903, where references to other authors will be found.

[3] G. Baumgarten, "Variations in the Clinical Course of Croupous Pneumonia," "International Clinics," vol. ii. Sixth Series, 1896.

less conspicuously red or rusty. Like bronchopneumonia, it is also insidious in its onset, the fever is irregular, and there is often chilliness or actual rigor with sweats; in a word, septic symptoms are prominent.

Termination.—1. When the pneumonia *terminates favorably*, promptly after the crisis is passed, it is said to terminate by *resolution*, by which is meant that the inflammatory product liquefies, is absorbed or expectorated, and the lung resumes its natural state and normal physical features. The time at which these events are thoroughly established varies greatly, and if there happen to have been associated pleurisy, with resulting thickened membrane, impairment of resonance may last a long while. On the other hand, it may terminate spontaneously even earlier than the periods named for the crisis. In such event the pneumonia is said to abort. This promptly favorable termination does not always take place. Resolution may be unduly delayed and yet ultimately take place. Such cases naturally occasion anxiety, for resolution may not take place at all.

2. When the disease *terminates unfavorably*, it is usually by one of five causes, viz.:

(*a*) Death from cardiac failure.

(*b*) Abscess.

(*c*) Gangrene of the lung.

(*d*) Interstitial or fibroid pneumonia.

(*e*) Tubercular phthisis.

(*f*) Some complication.

(*a*) Cardiac failure may be due to overdistention of the right heart or to toxic influence on the cardiac muscle.

(*b*) *Abscess of the lung* is a termination of pneumonia in about four per cent. of fatal cases. Flint, Sr., found it in four out of 133 cases recorded. When this occurs, the interstitial tissue of the lungs becomes infiltrated with pus cells, small foci of leukocytes aggregate to form larger, until a large abscess results, which may occupy a whole lobe or even a whole lung. In such cases the fever continues high, there is expectoration of pus containing elastic tissue of the lung, and the physical signs of a cavity may rarely be present. It is not impossible, however, for such a process to be arrested by a reactive inflammation, by which a tough protective layer of embryonic tissue is formed about the abscess.

(*c*) *Gangrene of the lung* occurs in about three per cent. of fatal cases. It is especially prone to occur where the pulmonary vessels become so engorged that the circulation is arrested, and where, as a consequence, the hemorrhagic element is conspicuous. Bronchiectatic cavities in an inflamed lobe that are swarming with putrefactive bacteria are an important predisposing cause. It is recognized by the sickening fetor, which pervades a whole ward, and which, once met, is never forgotten. The expectoration is thin and similarly fetid, and contains large quantities of elastic tissue from the lung. The lung is converted into a gray-green, fetid pulp, in which cavities with ragged walls arise, from disintegration and expectoration of lung tissue. Gangrenous portions may be surrounded by a zone of true inflammation, contrasting by its red color with the gray of the gangrene. Such sloughs have been successfully excavated by surgical treatment.

(*d*) In *fibroid induration or cirrhosis*, which is occasionally met with,

there is also invasion of interstitial lung tissue, but instead of being infiltrated by such an excess of leukocytes as to produce pus, only as many wander out as can undergo organization and conversion into permanent tissue. Sometimes this results from the lung failing to expand after resolution and absorption of the exudate, the walls of the unexpanded alveoli collapsing and uniting. In other cases there is partial absorption of the exudate, repeated infiltration takes place into the alveolar septa, and organization takes place in both. The fibrinous plugs may also be transformed into connective tissue. Three successive stages may be present. In the first the cirrhotic patches are gray, grayish-red, or grayish-yellow, and a small amount of turbid exudate can be here and there squeezed out of them. In the second stage, where the formation of the fibrous tissue in the alveoli or their walls has set in, the lung is dense, firm, airless, and fleshy, whence the term *carnification*. In the third stage the fibroid transformation is complete; the tissue is tough and slate-gray in color. Such induration is generally in bands and patches that merge gradually into the normal vesicular structure.

(*e*) Tuberculosis of the lungs is another termination of pneumonia. It results from infection by the tubercle bacillus. Pneumonia of the apex terminates thus most frequently.

Complications.—The most frequent complication is *pleurisy*. It is probably always present to a certain extent, except in the central forms. It manifests itself in the first stage more by the characteristic severe cutting pain than by physical signs, as the friction sound characteristic of that stage is commonly obscured by the physical signs of the pneumonia. Should the stage of effusion be reached, the physical signs of the pneumonia are obscured. Such a pleurisy is especially apt to be followed by an *empyema* with its septic fever. This empyema is very frequently interlobar and confusing in its physical signs, and in its symptoms.

Diagnosis of Empyema.—When a frank case of pneumonia has a crisis there may continue for two or three days very slight rises of temperature which may be disregarded. But when the temperature rises decidedly, when it becomes intermittent in character, especially with an increasing leukocytosis, in all probability there is a focus of pus somewhere in the chest cavity. This must be carefully searched for by daily physical examination, by the use of the *x*-ray and by the insertion of a good sized exploring needle. These signs may be due to tuberculosis but are much more frequently due to an interlobar empyema. If the pus is free in the pleural cavity the condition can be easily recognized by the ordinary signs of pleural effusion. No case should escape constant examination. In severe cases a pleurisy may surround the entire lung and bind it to the chest-wall. A pneumonia on one side and a pleurisy on the other is a possibility. That very interesting pathological state known as pleurogenic pneumonia is sometimes seen in the human being as a form of tubercular pleurisy. In it the lung becomes partitioned off by an interstitial framework starting from the pleura. It has its typical anatomical product in the pleuropneumonia of cattle. The extension takes place chiefly by way of the lymphatics.

Endocarditis is a comparatively frequent complication. William Osler especially called attention to this fact in his Gulstonian lectures for 1885.

He ascertained that of 209 cases of malignant endocarditis 54, or over 25 per cent., occurred as complications of pneumonia. It is more prone to attack persons with old valvular disease, and to involve the left heart. There is good reason to believe that the specific lancet-shaped bacillus is responsible for this form of valvulitis as a complication of pneumonia. The endocarditis constantly escapes detection, since physical signs are sometimes absent, at others deceptive, but it may be suspected:

1. When the fever is protracted and irregular.

2. When signs of a septic condition arise, such as irregular temperature with chills and sweats.

3. When embolic pneumonia develops.

4. When a loud, rough murmur, especially a diastolic aortic murmur, develops in the course of the disease.

Pericarditis may also be a complication, to be diagnosed first by pericardial friction sounds, soon to be replaced by signs of pericardial effusion. The exudate may be purulent or serous.

Meningitis is another complication to which Osler has called especial attention, finding it in eight per cent. of fatal cases. It usually comes on at the height of the fever, and may be confounded with delirium. Meningitis can frequently be diagnosticated by spinal puncture the pneumococcus being found in the spinal fluid. It is often associated with endocarditis, and it *may be accompanied by cerebral embolism, producing hemiplegia.* *Neuritis* is a possible complication.

Parotitis occasionally occurs, commonly in association with endocarditis. In children middle-ear disease is not an infrequent complication.

Any of the above cases may produce a postcritical fever.

Acute dilatation of the stomach is a more frequent complication than has been supposed.[1] It is usually mistaken for obstruction of the bowel. It is characterized by pain, constipation, abdominal distention, vomiting of large quantities of ill smelling material and collapse.

Diagnosis.—The diagnosis of a case of typical pneumonia is easy. The chill, the rapidly developed fever, the physical signs and early leukocytosis are, as a rule, easily recognized. It is to be remembered, however, that the physical signs may be delayed or not appear at all in the central varieties.

Pleurisy is the disease from which pneumonia has most frequently to be distinguished. The resemblance between the friction sound and the crepitant râle in the first stage is often very close, while there is impaired resonance to percussion in both. Most valuable in diagnosis is vocal tactile fremitus, which is invariably increased in pneumonia and as invariably diminished in pleurisy of any variety. In the not very rare instances of pleurisy with effusion, second stage of pleurisy, attended by bronchial breathing and bronchophony, *Bacelli's sign*, tactile fremitus being diminished, whereas it is increased in pneumonia. Commonly, too, in this stage of pleurisy with effusion we have a change in the line of dullness as the patient changes position, though this is not invariable. Dislocation of the heart and other organs is the rule in pleural effusion. The exploring needle, if needed, may also help settle the question.

Frequent examination of the lungs should be made in alcoholism, in

[1] "Trans. Assoc. Amer. Phys." 1911.

chronic valvular disease of the heart, in diabetes, and in Bright's disease, since all these affections are prone to become complicated with insidious forms of pneumonia.

Typhoid fever and pneumonia are sometimes confounded. The former is apt to become associated with hypostatic congestion of the lungs, and pneumonia with a typhoid state. The hypostasis, however, occurs late in typhoid fever; the dullness in pneumonia sets in early. In typhoid fever there is the Widal reaction, and usually a leukopenia, in pneumonia there is a polymorphonuclear leukocytosis. Acute tuberculosis or an acute exacerbation of tuberculosis may begin with a chill, while the resemblance to ordinary pneumonia is otherwise very close, especially in physical signs. Microscopic examination of the sputum should recognize the bacilli of tuberculosis. An examination of the sputum should be frequently repeated when a pneumonia is prolonged beyond two weeks without a crisis. In pneumonic phthisis the appearance of bacilli is generally late. It must be remembered that pneumonia is not uncommon in tuberculosis.

Appendicitis and lobar pneumonia have been sometimes confounded owing to the production of certain reflex symptoms including pain and rigidity in the appendicial region reflected through the lower intercostal nerves. Careful examination should therefore be made for the presence of right lobar pneumonia in such cases.

Prognosis.—Pneumonia is a treacherous and uncertain disease at any age. Young, robust men of 25, taken mildly ill with every reasonable expectation of recovery, sometimes die suddenly and unexpectedly. On the other hand, while in the old and intemperate the disease is especially dangerous, old men and women over 70 often recover completely. The intemperate are less fortunate, yet even among them some surprising recoveries are observed. The mortality ranges from 20 to 40 per cent., or about one in four or five die. It is the most fatal of the acute infections of adults in temperate climates. Children recover often, even when desperately ill. The disease seems to be more fatal in cities than in the country, and is certainly so during epidemics, or in ships or other crowded places.

Terminations in the cases of croupous pneumonia treated at the Pennsylvania Hospital in five years prior to 1907:

1902	254 cases	53 deaths	20.8% mortality
1903	328 cases	46 deaths	14.0% mortality
1904	249 cases	48 deaths	19.2% mortality
1905	282 cases	64 deaths	22.6% mortality
1906	238 cases	25 deaths	10.5% mortality

The seriousness of an attack varies more or less with the extent of lung involved, pneumonia of a whole lung being more dangerous than that of a part, double pneumonia more than that affecting one lung, while massive pneumonias are always fatal. Meningitis is frequently fatal, but its presence must not be inferred from every violent delirium. Endocarditis is almost as fatal. Death is usually by heart failure, the right ventricle becoming stretched by the accumulated blood, and the valves and columnæ carneæ embarrassed by fibrinous coagula, which may extend from auricle to ven-

tricle and even into the branches of the pulmonary artery. Heart failure may be caused by toxins inherent to the disease.

A high leukocytosis indicating a corresponding phagocytic power is regarded as favorable to recovery. At any rate in cases which have taken a favorable turn a leukocytosis takes place, while in fatal infections leukocytosis and presumably phagocytosis is absent.

Some aid to prognosis is derived from a study of the blood pressure. When the arterial pressure estimated in millimeters of mercury does not fall below the pulse rate expressed in beats per minute the indications are favorable; while if the blood pressure falls below the pulse rate, it is unfavorable (G. A. Gibson). The same general truth is applicable to all acute fevers.

Treatment.—The tripod upon which the treatment of all cases of pneumonia must be based, are *rest*, *fresh air* and *proper food*. Beyond these three essentials the treatment is always symptomatic. Some cases need no drugs, others need drugs and other measures promptly and properly applied.

Rest must be absolute even in the mildest cases. The patient must use a bed pan rising neither to urinate nor to defecate. Food must be taken with the patient in the reclining position. This absolute rest must continue several days after convalescence is established.

Fresh air means all the words implies; the patient is best on a protected porch with the front entirely open. If this cannot be obtained then all the windows in the room must be raised to their fullest extent. If the weather is cold the patient must be protected by abundant warm clothing, a cap and woolen socks if necessary, and hot water bottles also.

Food may be of any character that can be taken and digested by the patient. Milk reenforced by cream and sugar as directed for typhoid fever must be the basis. Care of the mouth is important. The bowels should be moved daily.

These essentials must be carried out by a capable trained nurse. If this is impracticable the patient should be in the wards of a good hospital.

Bleeding from an arm vein in the beginning of certain cases, with high blood pressure, a full bounding pulse, high temperature and a flushed face sometimes does good. The same results may be accomplished by wet cups, provided a sufficient amount of blood be taken, and cupping has the appearance of being less formidable, although it is actually more painful and disturbing to the patient. After the removal of the cups a poultice or warm cotton jacket is comforting. If doubt is entertained as to the propriety of either of these two methods of bleeding, the affected lung may be covered with dry-cups, and after the removal of these the hot poultice or hot jacket applied. Even by this method the relief to the pain and dyspnea is often very great, but is it more likely to be temporary. Dry-cupping may, however, be repeated daily, if it affords relief. While there are cases in which the adynamia is so great as to make blood-letting in any form of doubtful propriety, there can be no possible objection to the dry-cups.

Bleeding may be cautiously done in the second period, when the right heart is over-distended, as indicated by rapid breathing with cyanosis and laboring pulse. At this stage the removal of 10 to 16 ounces of blood is often of signal service.

These measures may also relieve the *cough*, but usually something addi-

tional is required. Until expectoration sets in, *opium* is pre-eminently the remedy, and no preparation is so good as *morphin* in doses of from 1/16 to 1/12 grain (0.004 to 0.005 gm.) for adults every two to four hours in 1/2 ounce (15 c.c.) of the solution of citrate of potassium flavored with lemon or other syrup. *Dover's powder* sometimes acts admirably. It is best given in pill form Commonly in 2 1/2 or 5 grain doses (0.16 to 0.32 gm.). Hypodermics of morphin are essential in the delirium and restlessness which so frequently accompany the disease. The patient should not be strapped to restrain him, he should be given rational doses of morphin; one-fourth of a grain to one-sixth usually being sufficient. Expectorants are rarely needed at the outset, but *ammonium chlorid* in doses of 5 to 10 grains (0.32 to 0.65 gm.) in brown mixture, also combined with morphin if necessary, may meet the indications. If a still more stimulating expectorant is required, the *carbonate of ammonium* may be used in doses of 5 to 10 grains (0.32 to 0.65 gm.) frequently repeated. It is an important fact, often overlooked in prescribing diffusible stimulants, that to get a desired effect they should be frequently repeated, and it is better to give small doses often than large doses at longer intervals.

Alcohol is not always required. The indications for its use are a former alcoholic habit when it seems absolutely necessary, and a running feeble pulse, but here it must be given as directed in typhoid fever not in large amounts, but sufficient for food, not more than a tablespoonful every two hours. The index of sufficiency or the reverse is the state of the pulse and heart. Whisky or brandy, as selected, should be given in milk, which is the most suitable nourishment. From 4 to 8 ounces (120 to 140 c.c.) of milk every two or three hours, containing the proper dose of alcohol may be given.

Strychnin is an invaluable heart tonic in pneumonia, and may be given in doses of 1/30 grain (0.002 gm.) or more, every four to six hours.

Digitalis is a remedy much used in pneumonia, and it is a useful drug, but it is not always judiciously used. It is best given and indicated when the heart dilates as shown by falling blood pressure, cyanosis, rapid heart. It should then be given in full doses, preferably hypodermically in the form of a fat free tincture in doses of 15 to 30 minims every 3 hours. Occasionally in sudden adynamia very large doses, say 1 dram (3.7 c.c.), hypodermically, may turn the tide toward recovery. Aromatic spirit of *ammonia* is an important adjuvant in straits like these, and may be substituted with advantage for the carbonate. Camphor in the form of camphorated oil is useful.

Inhalation of oxygen is of undoubted advantage in relieving the dyspnea and thus comforting the patient. It is not curative.

High temperature may be reduced by sponging, though the temperature itself in pneumonia cannot be regarded as dangerous *per se*. It is better accomplished by the local application of ice to be presently described.

The use of veratrum viride is warmly recommended by some instead of bleeding in the earliest stages of the disease. It diminishes the force of the heart, furnishes a diverticulum for the excess of blood, and, as our colleague, Horatio C. Wood, says, "The patient is bled into his own circulation." We have never felt comfortable in relying upon it.

The treatment of pneumonia by *ice-cold applications* is in vogue. It

is of some value in relieving the local pain, and perhaps helps to lower the temperature.

Hypodermoclysis of normal hot saline solution was used in desperate cases of pneumonia in the Philadelphia Hospital by Frederick P. Henry as early as the spring of 1889. The injection is made in the usual way, under the skin, at any stage in bad cases of pneumonia—from 1/2 pint to a pint (236 to 473 c.c.) of a .6 of one per cent. solution being injected daily or oftener—about 50 grams to the pint (3.3 gms. to the liter). If there is distention of the right heart as evidenced by cyanosis, flagging pulse, and sharp accentuation of the second pulmonic sound, the injection of a large quantity of salt solution will be disastrous—causing more dilatation with all its symptoms. However, if the dilatation be relieved by bleeding the hypodermoclysis or, what is better, intravenous injection of a pint of warm salt solution is of value.

The eliminative treatment has of late assumed importance because of the rôle assigned to the toxins in causing certain serious symptoms. Hence the free ingestion of liquids and measures to promote perspiration and purgation are advised.

Serum Treatment.—Pneumonia was one of the first diseases the treatment of which by serum engaged attention. Unfortunately up to the present the antipneumococcic serum seems to be of as little value as most other internal remedies.

BRONCHOPNEUMONIA.

SYNONYMS.—*Catarrhal Pneumonia; Capillary Bronchitis; Suffocative Catarrh; Lobular Pneumonia; Aspiration Pneumonia; Deglutition Pneumonia.*

Definition.—Bronchopneumonia is an inflammation of lobules or *patchy areas* of lung tissue caused by microbic or other irritants that find their way to it through a *bronchus*.

Etiology.—The effects of recent studies go to show that the bronchopneumonias of children are the result of the same causes as the lobar pneumonias of adults, producing however in the latter lobar consolidation and in the former lobular or patchy consolidation.[1] Usually bronchopneumonia succeeds a bronchitis of the terminal bronchus leading to the part. Some would consider bronchopneumonia and capillary bronchitis one and the same thing. Parts of a lobule, a whole lobule, or scattered groups of lobules are thus affected, and may unite to form larger areas. Thus, while a bronchopneumonia is primarily lobular, we may have even a lobar bronchopneumonia if all the lobules of a lobe are simultaneously affected. *Aspiration* pneumonia is a bronchopneumonia caused by the irritation of inhaled or indrawn particles, including bacteria, among which must be included also streptococci and staphylococci, as well as pneumococci and tubercle bacilli. Tubercular bronchopneumonia is one variety of this. Syphilitic bronchopneumonia is a rare, but possible, affection.

The recognition of bronchopneumonia as a separate disease is usually credited to Barthez and Rilliet.

[1] See Samuel West, "Clinical Lecture on Bronchopneumonia," to show that pneumococoal pneumonia in a child takes the lobular and not the lobar form. Reprinted for the author from the "British Med. Jour., May 28, 1898.

Simple bronchopneumonia is pre-eminently a disease of the very young and the old. In the young it occurs as an idiopathic affection, though it is also a frequent complication of the infectious fevers, measles, whooping-cough, scarlet fever, diphtheria, and small-pox. In adults, especially the old, it occurs during influenza, erysipelas, typhoid fever, and all debilitating affections, including Bright's disease and organic disease of the heart. The inhalation variety especially occurs in comatose states, however induced. William Pepper laid especial stress on vesicular emphysema as a predisposing cause. In both young and old it may succeed a simple bronchitis from cold, but it is as a complication of the infectious diseases named that it becomes during the first five years of life a very common, serious, and fatal disease, causing, it is said, more deaths among children than any other disease except infantile diarrhea. Diarrhea itself and rickets are also to be included as predisposing causes. All influences depressing to life, such as overwork, fatigue, the air of badly ventilated and crowded houses, insufficient food, and defects of hygiene, act similarly. Collapse of the lung is at once a cause and a consequence of bronchopneumonia.

Another cause of bronchopneumonia more common in adults and the aged is the inhalation of fine irritant particles or the aspiration of particles of food. In comatose states from any cause the sensibility of the larynx is benumbed, and minute particles of food are permitted to pass beyond the *rima glottidis* to enter the larynx, and thence the smaller bronchial tubes, where they excite inflammation. Hence the term aspiration or deglutition pneumonia above mentioned. Glossopharyngeal palsy is often associated with deglutition pneumonia which may follow tracheotomy and cancer of the larynx and esophagus. The inflammation thus excited is sometimes so intense as to cause suppuration and even gangrene. Stone-cutting, steel-grinding, and coal-mining become causes by the irritating particles inhaled in these occupations.

Morbid Anatomy.—The morbid anatomy of simple bronchopneumonia is quite definite, yet somewhat complex and difficult of description. The lungs may be superficially unaltered or they may be large and heavy. The exterior, especially at the base, may be mottled because of an alternation of dark-blue or bluish-black depressed areas with projecting portions more natural in hue. The depressed areas represent collapsed lung, and can, for the most part, be reinflated. In places they are continuous, forming large patches. Where there is much of this diffuse pneumonia corresponding patches of fibrin may be seen on the pulmonary pleura.

On section the surface of the lung is dark red in color and from it project reddish-gray spots representing areas of bronchopneumonia. These may be separated by tracts of uninflamed and collapsed tissue, or may unite to form more extensive inflamed areas. A section made transverse to the lobule will be found penetrated by a central bronchiole filled with muco-pus, while if the section is parallel with the length of the bronchiole, the central alveolar passage with its alveoli may be readily recognized, being rendered distinct by the same muco-purulent contents. Around the bronchus, from 1/8 to 1/5 inch (3 to 5 mm.) or more, is an area of grayish-red consolidation elevated above the surface, usually slightly granular to the touch, but still lacking the hard, shot-like feel of croupous pneumonia.

On pressure, a mixture of pus and desquamated cells may be squeezed out, which, at a later stage, becomes almost pure pus, appearing as white points in the nondepressed tissue. Surrounding the imperfectly hepatized areas and at a lower level is a smooth, dark, airless tissue, representing collapsed lung, which may be the seat of beginning inflammation. At a later stage, if the patient survive, especially in adults, the inflammatory areas may assume a darker hue, even that of gray hepatization. Still later, in the persistent forms, the areas may contain the white foci above described resembling miliary tubercles, from which they may be always distinguished by the fact that the white droplets can be squeezed out, while the tubercle remains firm. These areas may be converted into cirrhotic patches. During the progress of a bronchopneumonia the air-cells in the adjacent lobules are found dilated, and the edges of the lung and upper portions have also become emphysematous. The bronchioles themselves are also dilated in places. The uninflamed areas are generally congested.

The contents of the bronchioles and air-vesicles are pus-cells and swollen exfoliated epithelium. The walls of the bronchiole and of the air-vesicles are thickened and infiltrated with leukocytes. Rarely do they contain blood or the fibrin-network characteristic of lobar pneumonia. Occasionally, minute extravasations of blood may be found.

The phenomena in the aspiration form of bronchopneumonia are more intense in every respect than in the other forms, the infiltration of the air-vesicles with leukocytes leading sometimes to suppuration or even to gangrene.

Symptoms.—The initial symptoms vary with the precursory disease. In a child—and here the disease has its greatest practical interest—there may have been measles, whooping-cough or diphtheria, in which convalescence may or may not have set in. To incipient or aggravated *cough* decided *fever* is added, a temperature of 102° F. (38.9° C.) and higher being attained; the cough becomes more severe and painful, the *breathing becomes rapid*, and an easily visible, distressing *dyspnea* supervenes. The embarrassed breathing grows worse, the fever is higher, the *lips* and *face become cyanosed*, the short, incessant cough is ineffectual in the raising of expectoration, and the little sufferer is a picture of pitiable distress. For such a state of affairs the term *suffocative catarrh* given by the older authorities is well chosen. Happily, as the disease advances and the blood becomes charged with carbon dioxid, sensibility wanes, the suffering abates, and the cough grows less; but the frequent breathing, often 60 to 80, the lividity of the face, and the frequent pulse show that the fury of the disease is not spent, but will probably terminate only in death, which is directly due to exhaustion of the muscle of the right ventricle. At times, however, and even when least expected, a favorable turn takes place and a surprisingly rapid convalescence sets in.

In adults, as in children, the symptoms vary with the mode of origin. In the idiopathic form, which is recurrent in some old persons, there are fever, a burning spot in the cheek, and shortness of breath, but a cough less troublesome than would be expected. The physical signs rather than the symptoms determine the diagnosis. There are fine moist râles, with harsh breathing rather than bronchial breathing, and relatively clear percussion.

The symptoms in a case of deglutition pneumonia are very similar. In the inhalation pneumonia of miners, stone-cutters, and steel-grinders the symptoms are slower in their development and resemble more those of tubercular phthisis.

Physical Signs.—These are by no means as distinctive as those of croupous pneumonia. The association of capillary bronchitis and bronchopneumonia is so close that, given the fine subcrepitant râles of the former, unaccompanied by impairment of resonance, we may infer that bronchopneumonia is at hand. Further signs, however, of actual involvement of the lung-substance are *moderate impairment of resonance* and *harsh* breathing, rather than true bronchial breathing, though more rarely the latter may be present, especially when the bases of the lungs are involved. Inspection may recognize retraction of the cartilages and lower sternum during inspiration, indicating defective expansion of the lung.

Diagnosis.—The diagnosis of bronchopneumonia is usually easy. High fever, cough, mucous expectoration, fine râles, and slight impairment of resonance, following one of the infectious diseases in a child under five years, and developing gradually, admit of but one interpretation. When a number of small foci unite to form a large area corresponding to the whole or a portion of a lobe, the physical signs are more like those of a lobar pneumonia, and the absence of expectoration in children increases the difficulty of diagnosis. Lobar pneumonia develops more suddenly and resolves more rapidly.

The similarity in the morbid anatomy of persistent bronchopneumonia and tuberculosis has been referred to, and the clinical resemblance is even greater, so that it may be impossible to say in a child, whether it is bronchopneumonia or tuberculosis. Signs at the apices are to be sought for, and, if found, tuberculosis may be suspected; but the correct diagnosis is sometimes made only on the autopsy table.

Prognosis.—The prognosis varies with the etiology, but bronchopneumonia is always a serious disease. From 30 to 50 per cent. of all children perish from it.

In fatal cases in children death may occur in 24 hours. When recovery takes place, the disease lasts from five to ten days, and as many more are required for complete restoration to health.

Yet, as mentioned under symptomatology, some remarkable recoveries take place. In adults it is about as serious as croupous pneumonia. The deglutition variety is almost always fatal, and is the usual cause of death in glossopharyngeal palsy.

Treatment.—The indifference of parents and the carelessness of nurses are responsible for many cases of bronchopneumonia occurring during convalescence from measles, diphtheria, and whooping-cough which, with proper care, might have been averted. Among the causes thus responsible are exposure of children with uncovered heads at open doors and windows, insufficient clothing during sleep, overheated rooms, and drafty corridors. Fresh air, rest and food are as important as in the lobar type.

Restorative measures are indicated in this disease from the outset. Nauseating expectorants are rarely demanded and often do harm by lowering the vitality of the young patient. Blood-letting, useful in some cases

of croupous pneumonia, is not called for in catarrhal. Opiates to quiet the cough and relieve the pain are strongly indicated in the earlier stages of the disease and sometimes throughout it. They should be associated with diaphoretics and febrifuges, among which the solution of acetate of ammonium, the solution of citrate of potash, and sweet spirit of niter are the best. The tincture of aconite in small, but often repeated, doses is extremely valuable if the temperature is high and the pulse full and rapid.

When secretions become free and a stimulating expectorant is required, there is none better than the aromatic spirit of ammonium, which fulfills every indication and spares the stomach more than the chlorid or carbonate of ammonoium. At this stage, alcohol, in the shape of whisky or brandy, becomes an important adjuvant. It should be added to the nourishment, of which the best form is milk, although nourishing broths are also indicated. As digestion is likely to be feeble, the milk is better peptonized. Quinin, and especially strychnin as a respiratory stimulant, are useful tonics.

In the way of local treatment counterirritation by mustard and turpentine is especially useful. The former should be used in the shape of a weak plaster, one part of mustard to five or six parts of flour or flaxseed meal. If white of egg and glycerin be used to mix it instead of water, the plaster is less painful and may be kept on continuously. One of the best modes of applying turpentine is by the St John Long liniment, which may be made by mixing thoroughly a teacupful of vinegar, a wineglass of turpentine, and one egg. This may either be rubbed thoroughly on the chest or it may be applied on flannel. It may be that the turpentine is absorbed and acts as an expectorant. Blisters are not recommended.

The poultice is a measure of treatment for catarrhal pneumonia which is variously valued. It is undoubtedly useful in children if properly employed, but great care chould be taken that it does not become cold. It should be lightly made and changed often; and when changed, it should be done rapidly, a fresh hot poultice being at hand to replace the one removed. When poultices are not used, the cotton jacket should be substituted, as it insures a uniform temperature of the body. This is valuable in cold weather in order to protect the child and allow of the freest possible circulation of air in the room.

If the temperature be very high, it may be reduced by sponging or, better, by the wet-pack at a temperature of 75° F. (25° C.). The child does not, however, die of the effects of high temperature, but rather, finally, of a failing right heart. The bath is, nevertheless, very calming to the nervous system, and should be used for this reason.

The same measure may be used with appropriate modifications in the catarrhal pneumonia of adults, and also in the variety known as deglutition pneumonia. As this last form is, however, generally the beginning of the end in some other serious condition, treatment avails little.

TUBERCULOSIS.

I. General Etiology and Invasion. Morbid Anatomy.

Tuberculosis is an infectious and contagious disease due to implantation of the tubercle bacillus of Koch. It may affect any organ or tissue in the

body. Anatomically it gives rise to nodules which may soften, become fibrous or calcify. It is noted clinically by the symptoms characteristic of the organ affected, though irregular fever and emaciation are symptoms common to all forms. It may be acute or chronic depending upon the virulence of the infection and the resistance of the body.

Etiology.—The tubercle bacillus is a short rod-bacterium three to four microns in length, equal to about 1/3 the diameter of a red blood-disk, and 1/6 to 1/5 as broad. When successfully stained and viewed with high power it presents at times a beaded appearance. The organism is spread widely throughout the animal kingdom. Four well-known varieties exist, the human, the bovine, the avian, and the piscium; two rarer forms have been discovered, one in the turtle and one in the blind worm. Of these, the human and the bovine varieties concern us mostly. The bacillus does not proliferate outside the animal body. It is very resistant to ordinary methods of destruction. Direct sunlight, however, will kill it in a few hours, diffuse sunlight will kill it in a few days; corrosive sublimate will not destroy it well in sputum, while 5 per cent. carbolic acid in equal quantity will kill it in 24 hours. It resists the action of the gastric juice, hence the possible infection of the intestinal tract. It contains various toxic substances, among them the so-called tuberculin and a fever-producing substance.

Tuberculin Reaction.—When tuberculin is injected under the skin of a healthy individual, very little, if any, result occurs; if on the other hand the individual be the subject of tuberculosis, malaise, fever, rapid pulse, and general symptoms of intoxication; this, if the dose be small, .5 to 1.0 mg. the reaction will be short lived. This reaction will be spoken of under diagnosis.

Immunity.—Certain animals have been immunized to tuberculosis, and this immunization is the object of tuberculin treatment to be made in man, later to be described.

Bovine Tuberculosis.—Smith, Ravenel, Park and others, have demonstrated beyond question both that cattle may be infected by human tubercle bacilli and that certain cases of tuberculosis in man are due to bovine type of bacilli. Park and Krumwiede found 11.9 per cent. of 436 cases due to this type.

Healed or Healing Tubercular Lesions.—These have been found in human autopsies from 70 to 90 per cent. of all cases. About one-seventh of all deaths are due to tuberculosis.

Methods of Entrance into the Body.—According to Adami, the lungs are perhaps most frequently infected, through the lymph and blood stream, from the mouth, nose and upper respiratory passages, and many authors still believe that the lungs are infected through inhalation of the bacilli. Ravenel has proven that tubercle bacilli may enter the body through the intestinal tract, lodge in the peri-bronchial glands and leave no lesion in the mucous membrane of the intestine.

It is now a well-recognized fact, proven beyond a contravention that tuberculosis is very frequently conveyed by one person directly to another, and also that it may be contracted by healthy individuals living in homes which have been inhabited by tuberculous individuals. Thus, Cornet

studied the records of certain institutions whose immates are devoted to nursing of tuberculous cases, and discovered the fact that a large proportion of these (62.8 per cent. in 25 years) died of phthisis; also that of 100 nurses 63 died of this disease. On the other hand, the statistics of the Brompton Hospital for Consumptives in London is decidedly against any conclusion that contact with patients peculiarly endangers the lives of doctors, nurses, or attendants. This, too, though they cover a period when no precautions were taken to destroy the bacillus.

Flick's studies point to a greater activity of the contagium than was formerly admitted. He examined all of the houses in a ward in Philadelphia where there had been deaths from consumption, and found that 33 per cent. of such houses had more than one case, that 25 per cent. of these houses had been infected prior to 1888, and that more than 33 per cent. of the deaths which occurred since 1888 took place in them. These observations accord with the results of Cornet's experiments, which demonstrated that the scraping from the walls of phthisical wards inoculated into the lower animals produced tuberculosis.

The conditions which favor the growth and multiplication of bacilli in the body have been carefully studied, but have been only partially determined.

Heredity.—Adami says tuberculosis is practially never inherited. How much influence heredity actually plays in the development of tuberculosis is difficult to determine. Even though there be such an influence exerted, certain cases of congenital human tuberculosis are rare. It must be remembered that children born of tuberculous parents and living with them are constantly exposed to the risks of contagion. They may be infected in very early life and not develop the disease until much later consequently such cases are contagious in origin. It cannot be denied, however, that children of tuberculous parents may be feeble, lack resistance and hence fall easy prey to tuberculosis or any other infection late in life.

Defective and *insufficient food*, especially when associated with imperfect ventilation and dark living rooms, privation, grief, and overwork, are also conditions which favor the growth of the bacillus.

Frequently recurring bronchial catarrh by lowering the vitality of the mucous membrane engenders a soil favorable to the growth and multiplication of the tubercle bacillus. Any of the causes that produce such catarrh may be included among predisposing factors. Measles, whooping-cough, and typhoid fever with bronchial complications are sometimes followed by it. Occupations favor it. Particles of dust inhaled in the pursuit of various trades and avocations, as in coal-mining, stone-cutting and steel-grinding, are well known to have this effect. It is said (U. S. Census Report, 1890) that 288 potters die of consumption to 100 farmers from the same cause.

No race is exempt, but the colored race is especially predisposed, as is also the American Indian when brought under the influence of civilization. Tuberculosis appears to be spreading among the Indians, even in districts in the Rocky Mountains where the disease is rare among the whites. The Irish race in this country is also susceptible and many die of it. On the other hand, the Russian-Polish Jews are remarkably exempt, and next to

them are the native American whites. Phipps institute in Philadelphia for 1905 shows 16 per cent. of their patients were Jews. W. A. King, Chief Statistician of the United States Census Bureau, furnishes the following figures as to the nationality and race of victims of this disease:

	Six years 1884–1891	Calendar year 1900
White persons having mothers born in:		
United States,	205.1	151.8
Ireland,	645.7	526.1
Germany,	328.8	214.2
Russia and Poland,	98.2	88.5

Our Health Commissioner Dr. Samuel Dixon reports to us that during 1912, 9,855 persons died of tuberculosis in Pennsylvania. Of these 8,455 died of tuberculosis of the lungs while in the United Kingdom of Great Britian and Ireland 60,000 are said to die annually; and it is probable that at least three times this number are suffering from one form or another of the disease. In Philadelphia in 1908 there were 3,518 deaths from tuberculosis of which 2,065 were from pulmonary tuberculosis.

Climates have little effect upon the development of tuberculosis, except in that inclement weather often forces individuals to live indoors, in rooms that are usually ill ventilated and dark. In the dark rooms tubercle bacilli long retain their virulence.

Age is doubtless a predisposing cause, the susceptible period for pulmonary tuberculosis being between 20 and 35; for meningeal tuberculosis, between two and seven; while the lymphatic glands including the mesenteric and bronchial, are prone to involvement in the first ten years of life. The mesenteric glands are more commonly infected during the first five years of life, including the nursing period and that during which the child is nourished on milk.

The *shape of the chest* has long been regarded as influencing the development of tuberculosis, but except where the chest is so deformed that proper expansion cannot be attained it is doubtful if this has any influence. The "phthisical" chest is flat. At the present day two varieties of chests are described as phthisical, the *alar* and the *flat*. The former is narrow, shallow, and long, the angles of the scapulæ projecting like wings behind, the proper ratio between the antero-posterior and transverse diameters being, however, preserved. The ribs droop or are unduly oblique. The throat is prominent, the neck long, and the head bent forward. In the flat chest the antero-posterior diameter is disproportionately short, owing to the absence of convexity in the cartilages, which are sometimes even depressed, carrying with them the sternum and producing a form of chest which, on section, is kidney-shaped. In this form there is not the increased obliquity of the ribs characteristic of the alar chest.

Traumatism may have the effect of causing a latent tuberculosis to become active. Undoubted cases are cited by Weber. Patients may die of this tuberculosis thus made active. On the other hand a traumatism sustained at the hands of a public corporation is constantly distorted by unscrupulous lawyers to be the cause in cases of death or illness from tuberculosis after the accident, when no such relation exists.

Mode of Invasion and Spread.—The bacillus of tuberculosis derived

from the drying and pulverization of expectorated sputum can doubtless be inhaled and thus become infective either by being absorbed by the blood stream or by local action in the bronchioles. This neglected sputum probably has little affect when it is exposed to fresh air and sunslight, for these factors soon kill the bacillus. but when the sputum is deposited in damp and dark places such as living rooms frequently are, the protected germ lives much longer and can easily become infective. It has been proven, however, that the sputum, when it is first expelled, even in the form of fine spray from the mouth, is highly infective. The entrance into the body in the vast majority of instances is by the *respiratory* tract. Hence the great frequency of tuberculosis in the lungs and bronchial glands, which are the first tissues open to its approach. The comparative studies of George B. Wood[1] go to show that the *tonsillar tissue* of the throat because of its anatomic construction and topographic relations is more liable to become infected by tuberculosis than any other part of the upper respiratory tract. It is possible for the bacillus to enter by the *skin*, causing lupus or skin tuberculosis. It enters more readily by open wounds such as those caused by *circumcision*. Through the *alimentary* canal we have an undoubted route of infection. The work of Ravenel proves that tubercle bacilli may enter through the intestinal tract and leave the mucous membrane of the intestine intact. This may happen in children using the milk of tuberculous cows. It is not necessary that the cow should have tuberculosis of the udder to render her milk tuberculous. This has been conclusively shown by Bollinger and confirmed by Hirschberger and Harold Ernst. The boiling of milk destroys its infective qualities. Tuberculous meat is less frequently the cause of tuberculosis.

The tubercle bacillus having once invaded an organ produces localized tuberculosis, which may or may not become generalized in a manner to be presently described. More rarely, tuberculosis may become general from the onset without any local initial lesion being discoverable. This constitutes one of the varieties of acute tuberculosis. Once established, tuberculosis spreads by contiguity and through the lymphatic system and blood. In the former the tubercle grows by the addition of miliary tubercles at its periphery. Through the lymphatic system tuberculosis spreads to the lymphatic glands, and thence to the adjacent tissues. The barrier of the lymphatic glands once passed, the blood becomes the medium of a general infection. In the vast majority of cases generalization takes place from a focus of tubercle somewhere in the system, as the lungs, or a tubercular lymphatic gland, from which the bacilli start their migration.

The favorite seats of tuberculosis are the lymphatic glands, lungs, liver, kidney, spleen, intestinal canal, urogenital mucous membranes, the brain (especially its membranes and blood-vessels), bones and joints. In fact, no tissue or organ is exempt, the salivary glands and pancreas being least frequently invaded.

A primary tubercle is really in the beginning an infectious granuloma.

The first effect according to Adami of the lodgment of a tubercle bacillus is its absorption by an endothelial cell; but if numerous bacilli are in position

[1] The Significance of Tuberculous Deposits in the Tonsils. "Journal of the Am. Med. Assoc." read at 55th Annual Session, 1904.

at the same time in too great numbers to be absorbed, they are immediately surrounded by swollen, enlarged endothelial cells.

There are thrown out with these endothelial cells a rather small number of leukocytes, first polymorphonuclear and later lymphocytes. This collection of cells, the result of inflammatory reaction, occludes the capillaries in the neighborhood so that the tubercle becomes extravascular. Sometimes the large endothelial cells immediately surrounding the bacilli fuse, forming the so-called giant cell having the bacilli in the center. As the bacilli multiply and as some of their toxins are liberated, they act as intoxicants to the cells in the immediate neighborhood, causing their death. To use Adami's exact words, "The typical giant cell comes to exhibit a central, badly staining, necrosed area, around which other endothelial cells have fused, the typical tuberculous giant cell, which exhibits on section a peripheral circle or crescent of nuclei surrounding a more or less hyaline necrosed mass, the tubercle bacilli being found more particularly at the edges of the necrosed area."

Caseation occurs in the tubercle by the increase in growth of the bacilli, causing a larger and larger central area of necrosis until finally a cheesy mass fills the center.

As the central area of necrosis increases, the giant cells increase, the active cells increase until the mass may be seen by the naked eye.

Several such tubercles as this may occur around a central necrotic area, these give rise to other tubercles, which conglomerate into a mass, the center becoming necrotic. If such a conglomerate mass involved the walls of a vessel or a bronchus, it may rupture forming a so-called *tuberculous ulcer*, which favors the spread of the bacilli. On the other hand the tissues may become surrounded by a definite connective-tissue capsule; under these circumstances, the activity of the bacilli become arrested, and if the tubercles are small, they may undergo absorption, or the larger caseous masses may calcify causing actual calcaseous nodules surrounded by a fibrous capsule.

The bacilli in these fibrous nodules may remain alive for a long while, and if the resistance of the individual is lessened, they may be the focus from which the disease may extend.

II. Acute Tuberculosis.

Synonyms.—*Diffuse General Tuberculosis; Acute Miliary Tuberculosis.*

Definition.—The simultaneous comparatively sudden irruption of miliary tubercles in different parts of the body as the result of the spread of bacilli through the blood and lymphatic systems. It is the most emphatic expression of the infectious nature of tuberculosis. The infection is in almost every instance an auto-inoculation, of which the source is a nodule of softening tubercle in some part of the body. In 300 cases of miliary tuberculosis examined by Buhl such a source was found in all but ten, while Simmonds in 100 cases found the caseating focus in every instance. The most common seat of such a nodule is the lungs, next a tubercular lymphatic gland, especially a tracheobronchial gland. After this there

is less constancy, but tubercular joints, a tubercular pleurisy, tubercular peritonitis, and even a skin tuberculosis may be held responsible. Such a nodule may break directly into a vein, furnishing an instance of true embolic infection.

Acute tuberculosis occurs most frequently in young persons between 12 and 20 years of age, but adults are not exempt. Any tissue or organ may be involved, but very seldom do we find all the organs of the body affected, though it is quite common to find lesions in more than two, as, for example, the lungs, the pleura, the membranes of the brain, and the peritoneum. The first three are favorite locations.

Clinical Varieties.—*Three* principal *clinical forms* of acute tuberculosis are recognized, one presenting easily recognizable *pulmonary* symptoms, another signs of acute general infection *without special localization*, and the third, *cerebral* and *spinal* symptoms.

1. Pulmonary Form of Acute Miliary Tuberculosis.

Symptoms.—This form succeeds in adults on chronic tuberculosis of the lung, on prolonged bronchitis, on whooping-cough or on measles in children. An irruption of miliary tuberculosis the result of infection takes place throughout the lung with or without bronchopneumonia. The tubercles may be scattered throughout the lung, distributed by the blood, and may be found in the walls of the vessels, or radially arranged around the primary focus. It is this event which gave rise to Niemeyer's dictum, "The greatest danger to most phthisical patients is the development of the tubercle." To the previous cough and physical signs are added higher fever, increased cough, and extreme dyspnea associated with marked cyanosis. The last symptom is very striking.

Physical Signs.—The physical signs may not be altered; there may be sonorous and sibilant râles or there may be signs indicating deeper involvement of the lung, including small areas of impaired resonance, crepitant râles, and bronchial or bronchovesicular breathing (bronchopneumonic foci). On this account there may be rusty expectoration, rarely hemoptysis. The dull areas may alternate with areas of hyperresonance—hyperresonance due to relaxation (the Skodaic type)—or it may be due to localized emphysema. On the front of the chest there may be unusual resonance. Occasionally this Skodiac resonance continues to the end. As the disease progresses moist râles become general all over the chest. Again there may be friction crepitation due to tubercular pleurisy.

Diagnosis.—The diagnosis is made by recalling the symptoms detailed. Choroidal tubercle should be looked for. Especially important are the disproportionate dyspnea and cyanosis associated with the signs of diffuse bronchitis. Leukocytosis is here present.

Prognosis and Treatment.—The disease is often rapidly fatal and treatment is of little avail toward cure. It must consist in efforts to make the patient comfortable, but as the diagnosis can perhaps never be made with absolute certainty the treatment to be detailed later for the cure of chronic tuberculosis should be carried out.

2. General or Typhoid Form of Acute Miliary Tuberculosis.

Symptoms.—The general or typhoid form of acute tuberculosis has long been recognized as resembling in a startlingly close manner the symptoms of typhoid fever, and many mistakes have been made in diagnosis because of this resemblance. Since the use of the clinical thermometer in diagnosis, however, such mistakes have been less frequent.

As in typhoid fever, *a prodrome* of several days, and even weeks, of ill-defined sickness often precedes the taking to bed. *Fever*, with its heightened temperature and frequent pulse, is present, as are also the dry tongue, hebetude, and delirium of typhoid. Yet afebrile cases are reported by Reinhold and Eichhost. It differences are sought in the fever of the two diseases, it will be found that the pulse and respiration may be unduly frequent as compared with typhoid fever, but above all, the temperature will be found to differ in its course from that of typhoid fever. There is an absence of the characteristic "tidal wave" rise of temperature of typhoid. There is an evening rise and a morning fall; and an occasional inversion, with lower evening and higher morning temperature, takes place, which is, however, not characteristic. The range is between 101° and 103° F. (38.3° and 39.4° C.), but may reach 104° or 105° F. (40° or 40.5° C.). The countenance is apt to be more dusky than in typhoid.

Excessive sweating is a symptom more characteristic of acute tuberculosis than of typhoid fever, and may result in sudamina, which also characterize the latter disease. *Herpes* is, however, often present, while it is rare in typhoid. These two symptoms—*i. e.*, sweating and herpes, together with the intermitting fever—constitute a resemblance to malarial fever. Waller and Eichhorst have found *rose-colored spots* on the abdomen and breast, but they are certainly infrequent, and they do not occur in crops as in typhoid fever. *Enlargement of the spleen* is often present and even *hemorrhage from the bowels* has been noted. Sleight *albuminuria* is a frequent symptom, not due, as might be expected, to a tubercular involvement of the kidney, but to the fever process.

Repeated examinations of the lungs in early stages fail to discover *physical signs* indicating disease of these organs, and thus the conclusion that there is no lung involvement is apparently confirmed. Later, however, pulmonary symptoms may set in, also meningeal symptoms, the duration of which may lead to a suspicion that the disease is not typhoid fever.

There may be *pleural* or *pericardial friction* and other symptoms of pericarditis and pleurisy, as well as those of *peritonitis* and *meningitis*.

Tuberculosis of the choroid coat of the eye has been frequently met in acute miliary tuberculosis, more particularly in cases where there has been the widest dissemination.

In rare instances bacilli have been found in the blood. Bacilli are rarely found in the sputum in acute general tuberculosis, because in this form of tuberculosis the tubercles are not softened and the bacilli not discharged into the bronchial tubes are situated not in the open air-passages so much as in the interstitial tissue of the lung and in the blood-vessel walls.

Diagnosis.—As stated, acute miliary tuberculosis resembles especially *typhoid fever*, but a carefully kept temperature chart will soon exhibit a

difference in the two diseases from this point of view. If tubercles are found in the choroid the question is settled at once. Examination of a blood culture will frequently show typhoid bacilli in typhoid cases. The duration of the disease, though short, is usually longer than that of typhoid fever. The Widal reaction in typhoid fever and its absence in tuberculosis are valuable aids in the diagnosis.

It is well known that typhoid fever is characterized by a negative leukocytosis, that is, a diminution rather than an increase of leukocytes in the blood. Observations show that in true, uncomplicated miliary tuberculosis, there is also wanting an increase in the colorless corpuscles of the blood over the normal. So soon, however, as there becomes associated with the tuberculosis any catarrhal or suppurative condition of the parts involved; a leukocytosis presents itself.. A Von Pirquet reaction should be always made. When the individual is young, a positive reaction is of the greatest possible importance. A positive reaction here usually means the condition is tuberculous.

The resemblance to intermittent fever has been noted. Here, too, a close study of the temperature will soon show the difference, while a search for the hematozoon of malaria should be made. The failure of quinin to cure will settle the question against a malarial cause for the fever.

Prognosis.—The course is invariably toward an unfavorable issue. Scarcely ever less than four weeks in duration, it is often eight and even longer, although cases are reported to have terminated at the end of two weeks and even twelve days. Such must, however, be extremely rare. The relative shortness of duration, nevertheless, constitutes it one of the forms of *galloping* consumption. Acute miliary tuberculosis always terminates fatally sooner or later, although delusive improvements often raise hopes that are not realized.

Treatment.—Treatment for acute miliary tuberculosis can only be symptomatic. To our present knowledge a cure has never been accomplished. Antipyretics may be used in moderate doses; three to five grains of antipyrin, antifebrin, or phenacetin, the last probably the best, frequently repeated, abate the fever. Anodynes to quiet cough are also necessary. Supporting food and stimulants are indicated.

3. Meningeal Form of Acute Miliary Tuberculosis. Tuberculous Meningitis.

Synonyms.—*Tuberculous Leptomeningitis; Basilar Meningitis; Acute Hydrocephalus; Water on the Brain.*

Definition.—An acute inflammation of the pia mater due to an irruption of miliary tubercles on this membrane and on the blood-vessels proceeding from it, extending also at times to the corresponding membrane of the spinal cord.

Etiology.—The disease consists essentially in an irruption of miliary tubercles on the pia mater, with resulting inflammatory product. To this end there must be somewhere in the body a tubercular focus whence the bacilli start. Tuberculous bones and joints may furnish such a focus, but

it is most frequently located in the bronchial or mesenteric glands. Such focus cannot always be found, even when present. The bare possibility of a primary tubercular meningitis may, however, be admitted, in which event the cribriform plate of the ethmoid is the most likely route of bacilli inhaled from the external atmosphere through the nose to the brain. The disease is most common in children between the second and fifth years, though it is not very rare in adults, long subjects of tuberculosis.

Morbid Anatomy.—The pia mater at the base of the brain is the most frequent seat, whence the common term *basilar meningitis*. Particularly are the neighborhood of the optic chiasm, the Sylvian fissure, the interpeduncular space and pons varolii involved. In addition to the miliary tubercles are seen turbidity of the membrane increasing to opacity, the whole smeared over with fibrin and pus. The medulla oblongata and base of the cerebellum may be covered. More rarely the inflammation may extend to the lateral and convex surfaces of the brain. Especially do we find the adventitia-sheaths of the blood-vessels invaded by the tubercles, which are seen in the bead-like rows when the vessels are withdrawn from the substance of the brain. These vessels are better examined when spread on a dark background, with a low magnifying power. Sections of blood-vessels should be made also, because there may be tubercular infiltration of the intima, causing narrowing and obliteration of the vessel. The cerebral convolutions are softened to a slight depth by the invasion, the blood-vessels dragging a portion of the brain-substance when drawn out. Thus there is really a meningo-encephalitis.

The *lateral ventricles* contain a varying quantity of limpid or turbid fluid, a dram to several ounces, the ependyma is softened and swollen; the septum lucidum and fornix are disrupted. The convolutions may be flattened because of the pressure exerted between the dilated ventricles and unyielding cranium. More rarely there is a chronic process like that described, but slower in its course. As already mentioned, the pia mater of the cord may be involved, resulting in the same turbid picture.

Symptoms.—The symptoms of tubercular meningitis are varied and irregular in their course. At times, the beginning, at least to the superficial observer, is sudden. At others, there are many weeks of ill health with ill-defined symptoms that go to make the child unhappy, restless, and an evident sufferer. In the course of such weeks the child's appetite is poor, its tongue coated, its bowels are constipated or the reverse, and it loses weight. Such a child may have been convalescent from measles, whooping-cough, bronchitis, or other ills of childhood.

An attempt has been made with more or less success to divide the symptoms of the disease into stages, of which the first may be called *irritative;* the second, *that of subsiding irritation;* the third, *paralysis*.

1. *Irritative Stage.*—The symptoms most constant in the irritative stage are *vomiting*, *headache*, and *fever*. As has been stated, convulsions may usher in the attack, and these convulsions may intermit and be separated by periods of some length. Sometimes an accident, as a fall, may be an exciting cause, and the first vomiting may be excited by a meal of food unsuited to the child's age. The three symptoms mentioned as more constant grow in severity, especially the headache, which becomes more

or less incessant and intense, so that the child is never free from it. Yet there may be a lull in the pain as the result of treatment or other cause, followed by an acute exacerbation, which probably causes the peculiar short cry known as the "hydrocephalic cry." In other cases there is constant screaming, which points to the degree of suffering. The child rarely sleeps more than a few minutes at a time, unless under the influence of powerful anodynes. There is always *fever* in this stage, though it may not be very high, 103° F. (39.4° C.) being commonly the maximum. There is more or less delirium. The *pulse* is rapid, even rapid disproportionately to the temperature, while *breathing rate* is little altered, furnishing a symptom of some diagnostic value. Evidences of nervous irritation may occur early, more commonly late in this stage. The convulsion has been alluded to. The pupils may be contracted, irregular of oscillating, there may be strabismus and nystagmus, or twitching of the muscles of the face from involvement of the facial nerve.

2. *Stage of Subsiding Irritation.*—In the *second stage* delirium yields to coma, though convulsions may continue. There may be localized rigidity of the muscles of one limb or of half the body. The head may be retracted and the spine arched, due to rigidity of the muscles of the neck and back. Headache is not complained of, though the child still may occasionally cry out. The pupils are dilated or irregular, and squint is more marked from oculomotor or third-nerve irritation; a purulent conjunctivitis occurs; the bowels are constipated; the abdomen is retracted—scaphoid. The temperature tends to be lower, but is variable. There is often a patchy redness of the skin and *tâche cérébrale* may be brought out by drawing the finger-nail across the skin.

3. *Stage of Paralysis.*—The stupor increases and may be profound. Convulsions, however, still occur. They may be localized in a group of muscles or those of one limb, or they may be unilateral. On the other hand, there may be absolute paralysis of the oculomotor nerves, and even hemiplegia. As a result of the former the pupils are dilated, the eyelids partially closed, and the eye turned upward. Hemiplegia is more apt to occur when the fissure of Sylvius is invaded, when, too, there may be aphasia. Optic neuritis is sometimes present in this stage, usually occurring late, due to invasion of the optic nerve within the skull. The facial nerve may be involved in basilar cases, producing slight facial paralysis; so may the fifth, producing anesthesia, and atrophic changes in the cornea if the Gasserian ganglion be involved. Hyperesthesia of the special senses may also be present, though this is rather a symptom of the first stage. Toward the end a typhoid state may supervene, characterized by dry tongue, muttering delirium, and involuntary discharge of urine and feces. The temperature at this stage may be subnormal, falling as low as 93° F. (33.9° C.), On the other hand, the temperature sometimes rises just before death to 106° F. (41.1° C.) or more. The entire duration of the disease is from two to three weeks. The blood examination fails to find a characteristic leukocytosis.

Diagnosis.—In the diagnosis we have first to recognize the presence of a meningitis, and, second, to separate the tubercular meningitis from meningitis due to other causes. The former is commonly easy, yet mistakes are often made because so many of the head symptoms are simulated by head

symptoms in *dyscrasic conditions*, of which cholera infantum is a type, while retraction of the head may result from rheumatism of the muscles of the back of the neck; but optic neuritis and paralytic symptoms are confined to meningitis. The presence of tuberculosis elsewhere strengthens other signs. Examination of the spinal fluid will show tubercle bacilli.

The *other varieties of meningitis* that may give similar symptoms are meningitis due to internal ear disease, traumatic meningitis due to blows and injuries, syphilitic meningitis and cerebrospinal fever, meningitis due to any infecting organism. In meningitis due to ear disease the history of the case should prevent a mistake. Traumatic meningitis, especially with abscess, might simulate the symptoms described, but here, too, the history of the accident would be helpful, but in absence of a knowledge of the cause there might be confusion. Syphilitic meningitis is usually chronic, involving chiefly the convexity, whence cortical symptoms, especially focal convulsions. It may, however, invade the base of the brain, when it is more apt to be limited in area and confined to one side. Basal headache and signs pointing to localization are then present. Often the history does not help us, because the patient denies the existence of the specific cause. Cerebrospinal fever furnishes sometimes identical symptoms. In cerebrospinal fever retraction of the head and back is more marked and there is more pain in the trunk muscles, in a word, more symptoms of involvement of the spinal membranes. The diagnosis of tuberculous meningitis is most conclusively established by spinal puncture. The fluid is usually clear, contains lymphocytes, does not reduce Fehling's solution and gives a globulin reaction. Tubercle bacilli can be demonstrated. In cerebrospinal fever the fluid is usually turbid with polymorphonuclear cells containing meningococci.

Prognosis.—The prognosis of tuberculous meningitis well established is invariably fatal. On the other hand, the chances of error in diagnosis are so many that it is not wise to be too confident. It has happened more than once that cases in children recover where the disease has been thought present, but where the ultimate result proved the diagnosis erroneous.

Treatment.—Curative treatment is, therefore, futile, but for the same reason should be persevered in. Spinal puncture may be practised every second or third day as a therapeutic measure. It will often relieve convulsions and irregular breathing. Hexamethylenamine should be administered in doses of one to two grains .06 to .12 every two or three hours. In addition to this all supporting measures possible should be used with such treatment of symptoms as will secure the least suffering to the little patient. Where the suffering is great morphin should be used hypodermatically. Bromide of potassium will often control the convulsions.

Acute Tuberculosis of the Lungs.

(*b*) *Pneumonic Phthisis—Bronchopneumonic Phthisis.*

This more unusual form of tuberculosis of the lungs constitutes one variety of "galloping consumption," or *phthisis florida*. In it the tubercular infiltration is by a rapid peripheral invasion inciting to active inflammation. This is manifested as a bronchopneumonia, by which the air-vesicles and

bronchioles are variously blocked with cheesy matter. The result is the dissemination through extensive areas of lung tissue of opaque, white foci one-fifth to one-half inch (5 to 12 mm.) in diameter. These areas are usually separated by others of a more or less congested but still crepitating tissue, contrasting strongly with the white of the tubercular bronchopneumonic foci. These bronchopneumonic foci tend to soften with varying rapidity, resulting sometimes in numerous little abscess cavities throughout the lung. At other times the bronchopneumonic foci are more widely separated or may be limited to the apices. In more rare instances the condition may succeed on croupous pneumonia, forming continuous areas which may also extend throughout a lobe or entire lung. The process is truly pneumonic; the results resemble, indeed, more a lung in the second stage of croupous pneumonia. As in it, too, the lung is heavy and airless, sinking rapidly in water. There is, however, a greater tendency to disintegration than in croupous pneumonia, and cavities form rapidly in the apices and elsewhere.

There may also be enlargement of the bronchial glands in either of these forms, but more particularly in the first—the rapid peripheral extension.

Symptoms.—The *bronchopneumonic* form of consumption occurs most frequently *in children* as a sequel to measles or whooping-cough. In such seemingly ordinary cases of bronchitis, with *fever*, *obstinate cough*, and *shortness of breath*, physical examination will reveal submucous and subcrepitant râles throughout the chest with or without limited areas of consolidation. Tubercle bacilli and elastic tissue appear in the sputum. The fever continues and may become hectic, with sweats. The child emaciates rapidly, and death ensues in from three to eight weeks. Other cases originate more suddenly and with less apparent cause as cases of simple bronchial catarrh, which assume the graver picture described. Such children may inherit a predisposition to phthisis.

In adults the attack begins as an ordinary cold in a person with a predisposition to tuberculosis, though apparently healthy, or run down with overwork. The *cough* is harassing, and soon becomes loose, *expectoration* mucopurulent. There are *high fever* and *rapid wasting*, and *hemorrhage* may set in to the surprise of everyone concerned. Then there may be a lull in the storm, but for a short time only. The symptoms, and especially the burning fever, wear out the patient. Bacilli and elastic tissue will now be found in the sputum and the diagnosis is settled. The patient may perish in three weeks. On the other hand, a reactive effort toward improvement may take place and after a time be followed again by decline and perhaps again by improvement, with the effect of prolonging the disease, but not of altering the termination. The physical signs are the same as in children, submucous and subcrepitant râles throughout the chest with or without limited areas of consolidation.

The pure *pneumonic* form succeeding what seemed to be croupous pneumonia is more an affection of adults. More rare, still, than the bronchopneumonic form, it may be also rapid in its course. It begins with a chill followed by fever, often after exposure to cold, with pain in the side, cough, dyspnea, mucous and rusty sputum, impairment of resonance, bronchial breathing, increased vocal fremitus—in fact, all the symptoms of a pneumonia of the whole or a part of a lung, which may be an upper or lower

lobe. If the lower lobe, it is probably regarded as a pneumonia until the absence of the signs of resolution call attention to the fact that something unusual is going on. Later, softening and the signs of a cavity may present themselves at the apex, and bacilli and elastic tissue be found in the sputum. The case may last for three weeks or three months, or even pass over into a chronic phthisis.

Diagnosis.—In the bronchopneumonic form it is difficult to make the diagnosis early from simple bronchitis and bronchopneumonia. The temperature in phthisis is probably more irregular and higher. Where the disease lasts more than three weeks, the sputum should be examined carefully for bacilli. The diagnosis in the pneumonic form can never be made in the beginning, because the symptoms of the first and second stages of this form are identical with those of the first and second stages of true pneumonia, and it is only when the type of the latter disease is departed from that phthisis can be suspected. The fever in true pneumonia should abate by the ninth day or twelfth day at latest, and if it continue after that time acute tuberculosis should be suspected and the expectoration should be examined for bacilli.

Prognosis.—The prognosis is very unfavorable in this form of consumption, death being frequent in from a few weeks to a few months.

Treatment.—Treatment of the acute stage is symptomatic: fresh air and absolute rest are most important. After the acute stage it is that of chronic phthisis.

Chronic Pulmonary Tuberculosis.

Synonyms.—*Phthisis pulmonalis; Pulmonary Consumption; Consumption of the Lungs.*

(a) *Chronic Tuberculosis of the Lungs.*

Morbid Anatomy.—This most usual form of consumption, beginning with the tubercle and associated with more or less infiltration of the apex, extends thence slowly downward. The deposit in the beginning is not actually in the very apex, but a little below it, and usually the first point at which physical signs are found is on the middle of the clavicle or just below it. Sometimes, however, the extension is rather backward, so that the physical signs are first manifested in the supraspinous fossa, whence the importance of always insisting on the posterior examination.

From this initial focus, usually toward the anterior face of the lung, the disease extends more or less throughout the lobe, or it may pass to another lobe. If the disease be on the right side, from the upper it may extend to the middle lobe, and thence into the lower lobe about an inch below its apex, corresponding also to a point on the surface opposite the fifth dorsal spine. On the left side, the extension is directly from the upper to the lower lobe. From its previous focus the tubercular infiltrate travels centripetally along the bronchi from smaller to larger as a tuberculous peribronchitis. Larger and larger branches become implicated with the intermediate parenchyma, but usually it does not extend beyond the cartilage-

ringed bronchi of the second order, forming tubercular masses of corresponding size.

The infiltration is not limited to peribronchial tissue. It extends also inward toward the lumen of the tube, invading the submucous tissue, where it may be seen as whitish or cloudy patches on slitting up the bronchi and washing off the adherent mucopus. Thus uncovered, the mucous membrane is found also red and inflamed, contrasting strongly with the whitish patches referred to. As we penetrate deeper, these enlarge and intrude upon the lumen of the tube, while the hyperemic areas grow smaller. Such intrusion becomes finally complete invasion, associated, sooner or later, with an excoriation or rupture of the mucous membrane. This is the beginning of ulceration, which assumes an important place in facilitating subsequent destructive process, and is the foundation of the term adopted for this form of phthisis, *chronic ulcerative phthisis*.

The pathological process referred to, and the destructive effects of which they are the cause, give to the lung in a state of chronic phthisis a varied picture that is not always found in a single case, nor, indeed, would the lesions of two or more cases always cover this picture. They include the following:

1. The *caseous tubercular masses*. They embrace single or compound peribronchial foci perforated by the central bronchiole, itself plugged with cheesy matter. Thus constituted they form grayish-yellow masses from a couple millimeters to four or five centimeters (1/12 to 2 inches) in diameter. They have the composition already described. Though usually massed toward the apices of the lung, they may also be disseminated through the remainder of the organ, and around them there may also be found scattered true miliary tubercles.

2. The second anatomical feature of the phthisical lung is the *cavity*. As soon as a tuberculous area reaches a certain size, the tendency to break down is increased, though such tendency does not depend altogether on extent. The bronchial wall, weakened by the tubercular infiltration and the ulceration referred to, is the initial invitation. The wall yields to the pressure which it formerly easily resisted—the inspiratory and expiratory strain incident to coughing—the bronchus dilates, the gap of the ulcer widens and the texture of the bronchus gradually yields. The free access of air to the already necrotic caseous matter causes it to soften, break down, and a cavity results. Small foci unite with others and thus larger cavities form, occupying the greater part of a lobe, or even a whole lung in very rare instances.

Large cavities have usually smooth walls and are lined by the so-called pyogenic membrane, into which, however, often protrude blood-vessels of large size, as thick as a crow-quill, exhibiting also at times and aneurysmal dilatations. Rarely such vessels pass directly across a cavity, and when eroded they may give rise to fatal hemorrhage toward the end of a case of chronic phthisis. On the other hand, these vessels may also become thoroughly occluded by an obliterating endarteritis. The surface of these smooth-walled cavities is constantly producing pus, while muco-pus is being added by communicating bronchi. Such cavities may be more or less completely emptied by expectoration. They are also surrounded by a

consolidated lung tissue, which gives a dull percussion note and thus often prevents the tympany natural to a cavity. Small cavities have rough and ragged walls, from which there is constant breaking down, adding elastic tissue, pus, grandular débris, and bacilli to the matter expectorated. There may be a number of these small cavities, and if under the pleura one may rupture into the pleural sac, producing pneumothorax.

Other cavities form by the softening of the center of a caseous area. Others still may be purely bronchiectatic, being limited by bronchial walls. It is more particularly the bronchi of medium size that are thus involved, weakened also by tubercular infiltration. The form of dilatation may be cylindrical or globular. The small tubes especially may be the seat of cylindrical dilatation.

3. *Pleurisy* is constantly associated with tuberculosis of the lungs. It is found:

(*a*) As an adhesive pleurisy in the immediate neighborhood of tubercular infiltration.

(*b*) There may be perforation from a cavity into the pleural sac, exciting a purulent pleurisy or a pyopneumothorax.

(*c*) Finally, the pleura may be the seat of a tubercular pleurisy, resulting in a thickened membrane, which may be limited or may encase the whole lung and cement the lobes in a continuous inseparable mass.

4. *Pulmonary concretions* are also found in the phthisical lung, usually about half as large as a pea, smooth or lobulated. They represent calcareous infiltration of alveoli[1] of the lung, filled with tubercular bronchopneumonia products. They are a medium of one form of healing of tuberculosis. Those retained in the lung are commonly surrounded by a ring of hyperplastic connective tissue. At times they are expectorated, being released by a sequestrating suppuration into an adjacent bronchus, whence they are brought up by coughing. Sometimes a good many are coughed up. They are something different from *bronchial* calculi, which are always smooth, spherical, or elliptical, and are found in small bronchiectatic cavities.

5. Other evidences of *attempts at healing* seen in the phthisical lungs are of the nature of reactive inflammation. They may occur:

(*a*) In the initial stage as the result of treatment and favorable hygienic surroundings, when the initial granule is replaced by a cicatricial-like puckering of fibrous tissue or a hard cartilaginous mass of connective tissue.

(*b*) There may be a sequestration or encapsulation of a cheesy nodule, which may or may not undergo calcareous infiltration.

(*c*) Even a cavity of moderate size may heal, in which event, the cavity being cleared out, its walls unite by adhesive inflammation and thus a band of cicatricial tissue takes the place of the cavity. Larger cavities may be reduced in size by a contraction of the cicatricial tissue surrounding them, or several small cavities may be thus surrounded. Quite small cavities surrounded by connective tissue and communicating with a bronchus were called *cicatrices fistuleuses* by Laennec.

6. The neighborhood of a tubercular infiltration is often the seat of a pneumonia which may be simply reactive or due to the irritative effect of

[1] If macerated in hydrochloric acid, the lime salt can be dissolved out, and the actual elastic tissue framework of an alveolus, with its infundibula and attached air-vessels, be left.

the bacillus—*i. e.*, a *tubercular bronchopneumonia*. The area is hyperemic, hard, consolidated, and the air-vesicles filled with exfoliated epithelium. The latter may exhibit various stages of fatty degeneration. It may be complete when an appearance indistinguishable from that of tubercular infiltration is present. In fact, it is tubercular infiltration plus catarrhal pneumonia.

7. When a subject dies of tubercular phthisis, *other organs* should be searched for *tubercles*. Tuberculosis of the larynx is common and is not infrequently associated with destruction of the cords and epiglottis. The bronchial glands are usually involved, swollen, inflamed, or tubercular, and when tubercular may become caseous and sometimes calcareous. Other glands are also affected, such as the cervical, mediastinal, and postperitoneal. After the bronchial glands the organs most affected are the intestine; next, the spleen, kidneys, and brain in nearly equal proportion; then the liver and the pericardium.

8. The only remaining morbid states which may be considered as having any essential relation to tuberculosis of the lungs are the *amyloid* and *fatty infiltration*. The former is found affecting the kidneys, liver, spleen, and mucous membrane of the intestines; the latter, especially, the liver and kidney.

Symptoms.—The onset of tuberculosis of the lungs is by no means uniform. Notwithstanding the fact that its insidious nature is well recognized, its initial stage is often overlooked. The victim is scarcely appreciably ill. Yet he may lose flesh and strength continuously. He may even say that he had no cough, while close questioning will ascertain that he has had a *slight hacking cough* for some time, worse in the morning. Soon the symptoms are plainer, there is *evident, wasting* an *intermittent fever*, a *bright eye*, and the *cough with expectoration* is a conspicuous symptom. Yet during all this the patient is cheerful and denies that there is much the matter with him.

In another instance an individual is "subject to cold"; he takes cold repeatedly, and each attack, while passing away, yields more stubbornly than the previous one, and finally one comes that persists. There is *daily fever* which abates to return again, emaciation is evident, and the bright eye and burning cheeks and *night-sweats* again attest the arrival of the dread disease.

Another case may begin with *hoarseness*, due probably to tuberculosis of the larynx.

Again, after a stubborn attack of bronchitis in a person previously healthy a *hemorrhage of the lungs* unexpectedly makes its appearance, or such a hemorrhage may set in without previous warning, although, again, careful inquiry may find that cough has been present for some time. The patient has, perhaps, previously been overworked, or lived under unfavorable hygienic surroundings, or may possess a hereditary tendency.

In still another instance a patient may consult the physician without suspecting that he is very ill, and the signs of advanced disease of the apices will be found present, and there may be but a few more months life remaining to the unsuspecting victim.

A certain number of cases of consumption begin as *tuberculous pleurisy*,

which invades the lung by contiguity or by blood infection. One of the most convincing facts in favor of the infectious theory, which seemed established prior to the discovery of the bacillus, was the frequent occurrence of pleurisy as a forerunner of phthisis. It was held that the caseous product of the pleurisy furnished the infectious virus, which, entering the blood, caused tubercle formations in various parts of the body. Thus, one-third of the 90 cases of pleurisy followed up by Bowditch terminated in phthisis.

Inveterate dyspepsia is associated with many cases and is as often a predisposing cause as a symptom. A great loss of appetite and indisposition to take food are often symptomatic, and their presence does much to diminish the efficiency of remedies and nutriments so essential to successfully combat the disease. Anemia is a constant early symptom. Dyspnea is common as an early sign, usually the result of weakness rather than large involvement of the lungs.

Physical Signs.—Given the suspicion of the existence of tubercular consumption from the presence of the above symptoms, whatever others may be superadded, or whatever modification may occur in them, the diagnosis is completed by a physical examination. An evening rise of temperature with anemia and while it is not always easy to separate the clinical history of a case of consumption into three sets of symptoms corresponding to the three separate stages in the morbid anatomy, the physical signs corresponding with these stages are tolerably definite. They are:

1. The incipient stage, or beginning deposit.
2. Stage of complete consolidation.
3. Stage of softening and cavity formation.

1. *Inspection*, in the *incipient stage*, is as often negative as not. A slightly diminished expansion in the infraclavicular space, as compared with the opposite side, may be present, and more rarely a slight flattening of the same region. The clavicle becomes correspondingly conspicuous. The body may continue well nourished or slightly emaciated, or the heart-beat in the normal position may be somewhat accelerated, while the respirations are likely to be more frequent than in health.

Palpation may recognize increased vocal fremitus in the same situation, although not always, while the physiological difference in favor of the right side is to be remembered. *Percussion* in this stage gives slightly higher pitch and impairment of resonance, which may be noted above, on, or below the clavicle. Usually the resonance on the affected side does not extend as high above the clavicle as it does on the normal side. It sometimes happens that increased resonance is obtained by percussion in the earliest stage of pulmonary tuberculosis. This arises in the following way: The lung is engorged and in consequence relaxed, in which condition the air within the vesicles vibrates more freely, the result being a full clear note (Skodaic reasonance).

To *auscultation* above or below the clavicle, we have the first evidence of abnormality in a prolongation of the expiratory murmur and harshness in the inspiratory sound. Theoretically, this should be preceded by a diminished intensity in the inspiratory sound, owing to the interference of the newly deposited tubercles with the entrance of air into the air-vesicles, but

practically such diminished intensity is rarely encountered, and even if present is not of distinctive significance.

Increased vocal resonance is a constant accompaniment of these modifications in the normal breathing-sounds, but it, as well as the vocal fremitus, may be masked by a pleuritic thickening, and the physiological difference so often referred to must be remembered. J. M. DaCosta also called attention to the fact that in a certain number of cases, at this stage, there is a blowing sound in the subclavian or pulmonary artery, and that a murmur is sometimes present in these vessels before any other physical sign is noted. There are frequently concurrent with these signs those of a bronchitis more or less acute. It must not be forgotten that the signs of early infiltration may begin in any portion of the lung.

2. In the *second stage* the changes discoverable by *inspection* are more easily recognized. There is evident loss of flesh, depression of surface, and impaired range of respiratory movement. The hectic flush is intermittingly present. *Palpation* may even discover an increased warmth of skin. The increased vocal fremitus is now plainly recognized unless obscured by a thickened pleural membrane. Dullness on *percussion* is positive and easily elicited.

To *auscultation* there is increased vocal resonance. The bronchial factor in the breathing now becomes conspicuous, showing itself by the harshness and relative shortening of the inspiratory element, with the decidedly prolonged and blowing expiration; also a gradual diminution of the vesicular factor, until the latter disappears entirely, when we have the typical bronchial breathing of extended areas of tubercular infiltration. This sign will now be found in the supraspinous fossa posteriorly as well as anteriorly. The conduction of the normal heart sounds to the area of infiltration, if at either apex, is a very frequent and significant sign. The high degree of vocal resonance known as bronchophony in also superadded as a valuable confirmation of the presence of complete consolidation. The auscultation signs of a concurrent bronchitis may also be present in this and in the next page.

3. In the *third stage* the information furnished by *inspection* is still more positive. Emaciation is marked, breathing and the pulse are rapid, and the face is often flushed. There is flattening over the affected area, and the excursion of respiratory movement is still more limited. In this stage the superficial veins over the involved area may be prominent, partly from emaciation and partly from obstructed circulation. To *palpation* the vocal fremitus is still more marked, and even remains distinct over cavities, because of the consolidation around them, unless there be some obstruction to the entrance of air into the bronchus leading to the involved area. The skin is hot and dry, unless succeeding one of the sweats that characterize this stage, when it may be moist and clammy.

Dullness on *percussion* is always to be found in the third stage, but to it is often added some one of the varieties of tympanitic note—viz., pure tympany, the "cracked-pot" sound, or amphoric resonance, due to cavities. These require sufficient size and superficial situation on the part of the cavity. On the other hand, resonance may even be normal over a cavity some distance from the surface, especially if the percussion be lightly made,

while the consolidated tissue which almost invariably surrounds a cavity often permits only a dull sound to be elicited. Wintrich's change of note should be sought—a change of note produced during percussion over a cavity on opening and closing the mouth, the pitch being higher when the mouth is open.

Auscultation in this stage may continue to recognize the bronchial breathing of the second, but to it are superadded first small bubbling sounds or subcrepitant râles indicating liquefaction; later, may be added the distinctive signs of a cavity. These signs are cavernous breathing, cavernous voice, pectoriloquy, either whispering or loud speaking, amphoric breathing, and amphoric voice. To these are often added the large bubbling sounds known as gurgling, caused by the air bubbling through fluid in a cavity. Metallic *tinkling* may be added to these phenomena, caused by the bursting of bubbles in a cavity with amphoric conditions.

"Cavernous breathing," generally speaking, is any modification of the normal breathing sounds due to the air passing in and out of a cavity. When high pitched it becomes tubal or amphoric. The amphoric sound is supposed to occur in cavities with firm walls that best secure the "echoing," which is the condition of amphoric breathing and amphoric percussion. Over more yielding walls the breathing is lower pitched, and to this the term "cavernous" is especially applied.

Special Symptoms.—The *cough* of consumption varies greatly. It is at first very slight, and may continue so even in advanced stages. As a rule, however, it grows in severity with the progress of the disease. It is caused by the irritation of intercurrent bronchitis or bronchopneumonia or the accumulated contents of cavities. When a cavity becomes more or less filled with secretion it must be emptied, and a spell of coughing comes on and continues until the cavity is cleared but, whence the paroxysmal character so often assumed by the cough when this stage is reached.

The *expectoration* of tuberculosis varies with the stage of the disease. At first scanty, and in no way characteristic, it grows more copious and becomes puriform as the disease progresses. A more or less circular shape is finally assumed, which is somewhat distinctive, and is called "nummular," from its resemblance to a coin. The quantity of expectoration varies greatly, from 1/2 ounce (15 c.c.) to 1/2 pint (250 c.c.) in the 24 hours. It generally has a sweetish, unpleasent odor, but is rarely offensive. It is sometimes tinged with blood, and may contain Charcot's crystals (p. 297).

Minutely, the expectoration is made up chiefly of pus-corpuscles, among which may, however, be found epithelial cells from the mouth and lung alveoli, elastic tissue from the air-vesicles, more rarely from the bronchial tubes or blood-vessels, bacilli, oil drops, particles of food, generally innumerable tubercle bacilli, and at times blood-disks. The elastic tissue is most easily demonstrated by boiling the sputum in a test-tube with an excess of solution of potash or soda, the effect of which is to thin the sputum and permit the elastic tissue to fall to the bottom of the tube; whence it is easily carried by the pipet to the glass slide and recognized under the microscope by its wreath-like or circular shape, if derived from the air-vesicles. Care must be taken to eliminate fibers of elastic tissue that may be derived from

food. To this end the mouth should be carefully rinsed before collecting sputum for examination, and it is further to be remembered that particles of food containing such tissue may remain in the mouth for two or three days. The elastic tissue from the bronchi occurs in the shape of elongated or reticular fibers. That from blood-vessels is similar; more rarely it is fenestrated membrane. The alveolar epithelial cells are round and oval, mononucleated, highly granular, nearly twice the diameter of a pus-corpuscle.

The *bacilli*, which are an unfailing sign of tuberculosis, are demonstrable only by special staining methods, of which that by carbol-fuchsin, with or without Gabbet's counterstain of methyl-blue is recommended.

One of the most unpleasant consequences of the cough is the *vomiting* which it induces, more especially in the last stages of the disease. It is not unusual to throw up a meal immediately after it is taken. Such vomiting is probably a reflex act, excited by irritation of the pharynx in coughing. Fortunate is the patient who can immediately thereafter take another meal, since this meal is generally retained, because the accumulated muco-pus which caused the coughing spell is also thrown up with the food in the first act of vomiting, and the cough ceases for a while.

Pain is not inherent to tuberculosis—that is, the seat of a tubercular infiltration is not usually a seat of pain. Pain is, however, a frequent secondary symptom. It is most severe as the result of a concurrent pleurisy, when it is usually sharp and cutting at the site of the pleurisy. Pain also results from inveterate cough.

Fever is a symptom of all stages of pulmonary consumption. At the onset there may be fever of an irritative kind, due to deposition of the tubercle and to inflammation. This is a fever of a continued type with slight evening increments, often overlooked, until it becomes associated with hectic fever, which is a septic fever occurring during softening and cavity formation. *Hectic* fever is one of the most interesting symptoms of consumption, adding often a picturesqueness that increases the sadness of the situation. Coming on usually toward the end of the day, the maximum point is reached at no fixed hour, but generally occurs between 2 and 6 P.M., though it may be as late as 10 P.M. The minimum, usually noted between 2 A. M. and 6 A. M., may occur as late as 12 noon. Hence, frequent observations of temperature should be made during the day and night, two in 24 hours being inadequate. Once in four hours is not infrequently desirable, and where careful study is desired, once in three hours may be necessary. The chart (Fig. 95), on page 293, shows extreme range of temperature in hectic fever.

There is, however, no greater mistake than to suppose that every case of tuberculosis of the lungs must have fever throughout. It probably always has fever in the beginning—the fever of onset; but with the disease once established it frequently happens that there is no fever in any part of the 24 hours. Appended is a chart of such a case (Fig. 96).

In the course of a case of consumption it constantly happens that periods occur of various duration, from one to seven days, in which the fever is higher than usual with moderate remissions, say of one degree, and attended with increased localized pain. These are explained by the occur-

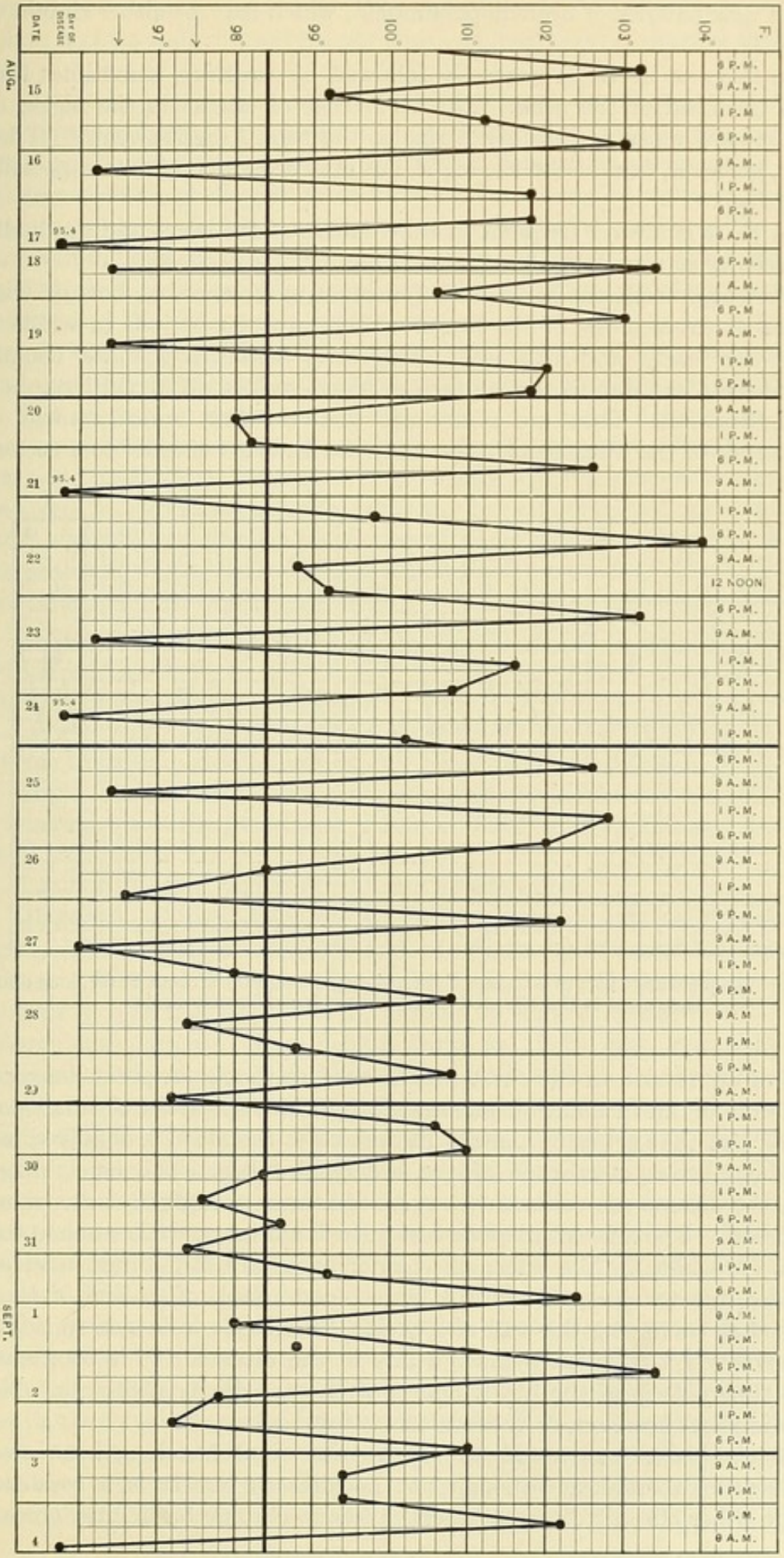


FIG. 95.—Chart Showing Extreme Range of Temperature in Tubercular Phthisis.

rence of new patches of bronchopneumonia, which may be either simple or tubercular.

The fever of hectic is generally followed by *sweating*, sometimes limited to the head or the neck. The occurrence of sweats in the night, or rather toward morning, has given rise to the term "night-sweat." They are not, however, confined to the night, but may occur at any time, especially during sleep.

The *pulse* is always frequent in tuberculosis of the lungs and gradually grows feebler as the disease progresses.

Hemorrhage from the lungs is a symptom everywhere associated with the idea of tuberculosis of the lungs. There are two periods in which it occurs—one early and one late. The early hemorrhages are usually moderate and are due to the rupture of blood-vessels weakened by tubercular infiltration. They are sometimes the very first announcement of the presence of the disease, at others they are a means of relief to a certain

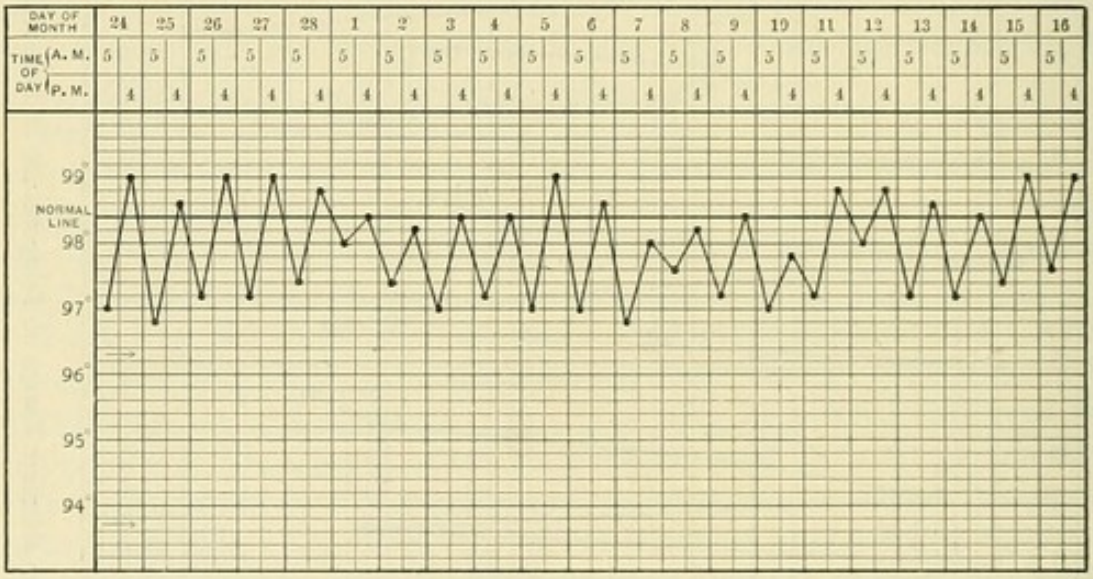


FIG. 96.—Temperature Chart of a Case of Tubercular Consumption without Fever, long under treatment at the Hospital of the University of Pennsylvania.

feeling of oppression in the chest which precedes them. A great danger is production of an insufflation pneumonia by the inspiration of small particles of clot that act as irritants. In such cases the blood probably comes from the mucous membrane of the bronchial tubes. The hemorrhages late in the disease are commonly large, sometimes enough to cause immediate death. The amount of blood lost in such a fatal case has reached four pounds (1.8 kilos). Yet enormous hemorrhages are sometimes survived. They are due to ulceration into a large blood-vessel, often one of those described as traversing the wall of a cavity, bridging it from side to side.

Diarrhea is a frequent symptom late in the disease. It is commonly due to tuberculosis of the bowel and is often exceedingly obstinate. Not every diarrhea, however, in tuberculosis is tubercular.

The *club-finger* was noted by Hippocrates, and has long been associated with tuberculosis—though not peculiar to it. It is a condition found in other chronic diseases, as emphysema, chronic bronchitis, chronic

cardiac disease, and aneurysm. The end of the finger is bulbous, quite like a club, and the nail curves over the end. It may involve some of the fingers only.

Tuberculous meningitis may be added toward the close of the disease. The symptoms vary a good deal with the seat of the involvement, and have been considered in detail when treating of tubercular meningitis. If the inflammation is in the fissure of Sylvius, there may be aphasia and even hemiplegia; if at the base, retraction of the head and palsies of the cranial nerves from pressure, also optic neuritis; if on the convexity, delirium is more decided, and there may be local convulsions with hemiplegic weakness. Ventricular effusion—acute hydrocephalus—adds little to the specialization of symptoms. There may be co-involvement of the membranes of the brain and spinal cord, producing symptoms of cerebrospinal meningitis.

The relation of pulmonary consumption to *cardiac disease* has always been an interesting one. It was formerly thought that affections of the heart and lungs are never concurrent. This is a mistake; such concurrence is observed, but whether such relation is any but an accidental one is doubtful. Osler reports 12 instances of endocarditis in 216 autopsies on cases of consumption. The rarity of lung tuberculosis succeeding chronic valvular heart disease must still be admitted. It has been ascribed to hypertrophy of the unstriped muscular structure about the smaller bronchioles and their acinous terminations, which keeps the alveoli evacuated of such secretions as favor the development of phthisis.

Chronic nephritis and *amyloid kidney* are frequent complications of chronic phthisis. From these causes *albuminuria* may result. There may be simple febrile albuminuria. Or albuminuria may be due to pus, if there is tuberculosis of the bladder or kidney. Tubercle bacilli should be sought for in purulent urine.

The *liver* is often enlarged from fatty infiltration.

Diagnosis.—Early diagnosis is the key-note to successful treatment. Fever, anemia, loss of strength and of weight must never be considered lightly. Tuberculosis must always be kept in mind, and physical signs constantly searched for. The same may be said of neurasthenia. Many tuberculous patients have neurasthenia as their first symptom. The diagnosis of tuberculosis of the lungs may be difficult in the early stages, but later, when the physical signs have developed, it is easy. In any stage the finding of the bacillus removes all doubt. Occasionally, however, the sputum is very scanty and difficult to get or it may be abundant and contain few bacilli. If such an examination is not possible, or furnishes negative results, some time may elapse before a positive diagnosis is obtained, nevertheless every doubtful case should have frequent sputum examinations. A positive finding is worth any trouble; for the physical signs in the early stages cannot always be relied on, while there occur cases in which, even months after bacilli have been found in the sputum, the physical signs are confusing and inconclusive. Due regard must be paid to the fact that in health the expiratory sound below the right clavicle is longer and rougher than in a corresponding position on the opposite side, while the percussion note may also be somewhat higher pitched. The presence of fever more or less constant, the bright eye, and crimson flush in the cheek, the anemia,

with or without emaciation, should excite suspicion and lead to careful physical exploration and examination of the sputum, if not already made. The search of the sputum for elastic tissue is relatively less valuable, because bacilli are usually found much earlier.

The *X*-ray is of the greatest value in locating tubercular lesion especially where the physical signs are meager.

In doubtful cases the tuberculin test may be made. This is of value in cases under the age of puberty, but of little value after that age. The majority of adults react to tuberculin whether they have active tubercles or not. A positive reaction means tuberculosis, but does not necessarily mean the condition from which the patient suffers is tuberculosis. *One milligram* of pure tuberculin is injected hypodermically, and if there be no febrile reaction in 10 to 12 hours, twice this quantity is used two or three days later, and gradually increased at intervals until *five milligrams* have been injected at a dose. If there be no rise in temperature within 10 to 12 hours the patient may be considered free from tuberculosis. The usual rise is from two to four degrees F. The test is valueless when the patient already has fever.

The Tuberculin Ophthalmo-reaction of Tuberculosis.—Suggested by Wolff, Eisner and Vallee, it was left to Calmette[1] to elaborate this test of the presence of tuberculosis. It is an excellent test, but certain accidents have followed its use. It has now fallen out of common practice.

Von Pirquet's tuberculin skin reacuion is much more safe than the opthalmic test and should always be used. It is made in the same way vaccination against smallpox is done with some variation.

The forearm or arm over biceps is carefully cleared, two small drops of Koch's old tuberculin are placed about 5 cm. apart—upon the cleaned skin. A sterile needle is then used, a small area 2 mm. in diameter is then scarified in the sound skin between the drops of tuberculin. Scarifications is then done through the two drops of tuberculin. If the reaction is positive the two spots where the tuberculin was used will become red and swollen in from 24 to 48 hours, no reaction takes place in the spot where there is no tuberculin.

The physical examination must be made early and repeated frequently in the study of a case. Especially is this true of cases in which there is a hereditary tendency. It goes without saying, that the physical signs of incipient tuberculosis may easily escape detection when an examination is made with the clothing on, while they would be easily recognized if the patient were stripped to the skin. Too frequently, also, an examination is deferred because of a fear that the patient will be needlessly alarmed thereby. So-called "hemorrhages from the throat" should be carefully investigated, as should also any continued hacking cough. Many of these coughs are now known to be due to tonsillar trouble, but this should not be taken for granted, and a careful examination of the throat should be associated with a physical examination of the chest. A habitually frequent pulse and rapid breathing should also excite suspicion. We should not omit either to examine the posterior part of the chest in the supraspinous fossæ, for it

[1] Calmette: Académie des Sciences, 17 juin, 1907, vol. cxliv, No. 24, p. 1324. "Presse Médicale," No. 49, 19 juin, 1907, p. 388–389.

sometimes happens that physical signs are here detected before they are recognizable in front.

Prognosis.—The prognosis of chronic tuberculosis of the lungs varies greatly with different cases. Many cases will recover if the diagnosis is made early. Its duration ranges in individual cases from a few months to years.

The modern treatment has been followed by great improvement in results. Not only is the disease arrested in its course in many instances, but in many actual cures result. Indeed, many instances of recovery undoubtedly happened before the modern treatment was instituted. It is difficult, indeed impossible, with the present statistical methods to ascertain the proportions of recoveries, but one can form an idea of the general situation from the number of deaths from this cause as compared with others. Thus in Philadelphia in 1882, there were 3.28 deaths from consumption per 1000 of population. In 1907, 25 years later, the rate was 2.10—a decided falling off. In 1882 there were 2809 deaths from tuberculosis of the lungs to 17,250 deaths from all other causes or 1 to 6.1. In 1907 there were 3157 deaths from tuberculosis and 24,305 deaths from other causes or 1 to 7.6. The per cent. death rate in Pennsylvania is 1.5 per 1000 of population.

(*b*) *Fibroid Phthisis.*

Definition.—This term is applied to a form of pulmonary consumption in which the lung, in addition to being the seat of tuberculosis, is permeated by an overgrowth of fibroid tissue. Its course is much slower, and while it often begins as an inhalation bronchitis in those exposed to the inhalation of fine particles of dust from various sources, it may also begin as an ordinary ulcerative or catarrhal phthisis.

Symptoms.—Its symptoms, on the whole, are less aggravated than those of ordinary phthisis. The cough is less severe, less exhausting, though more apt to be paroxysmal, and the patient has less fever and emaciates less rapidly. He is often able to pursue some occupation. Bacilli are less numerous and are often found with greater difficulty. Expectoration is often, however, as copious, usually arising from cavities or dilated bronchi, and is more frequently fetid. It may contain fat crystals and Charcot's acicular crystals. There may also be hemorrhage. Apart from these symptoms and the presence of bacilli in the sputum, the clinical history is scarcely different from that of simple nonspecific cirrhosis of the lung, from which it is, indeed, often separated with difficulty. As in this affection there may be hypertrophy of the right ventricle, induced by the extra effort demanded of the right heart to move the blood through the fibroid lung. Fibroid phthisis is especially characterized by its prolonged course, which may extend over years.

Physical Signs.—The degree of retraction of the chest wall as noticed by *inspection* is greater than in the ulcerative form, more easily recognized, and not always confined to the vicinity of the apices of the lungs. The heart may be dislocated and its apex correspondingly awry, sometimes to an extreme degree. If on the left side, owing to retraction of the lung, there may sometimes be seen a distinct cardiac pulsation in the second, third, and

fourth interspaces. The intercostal spaces are often narrowed and the diaphragm may be drawn up. Modifications of vocal fremitus as revealed to *palpation* are not nearly so constant, being masked by retraction of the lung and pleuritic complications, and may be absent. There is often little or no elevation of temperature.

Percussion is more constant in its results, there being marked dullness and a wooden-like resistance. The hyperthrophy of the right ventricle referred to may extend the normal cardiac dullness in positive degree beyond the right edge of the sternum.

Auscultation most frequently notes bronchial breathing and exaggerated voice sound, but both of these may be lessened in intensity by a thickened pleura. A dilated bronchus is frequently present, yielding the signs of a cavity, which may be found in the middle or even at the base of the lung.

To the signs of the fibroid state in one part of a lung are frequently added those of emphysema in the remainder or in the other lung.

Prognosis.—This is perhaps no better, so far as cure is concerned than for the chronic ulcerative phthisis, but, as has already been stated, the duration of the disease is much longer, and under favorable circumstances much more can be done for the patient by the same treatment.

Treatment of Chronic Tuberculosis of the Lungs.—There is no disease of like importance in which treatment must for various reasons differ so much in different cases. This is owing partly to the fact that curative measures must be adapted more or less to the circumstances of the patient, and partly to the varying peculiarities of the patient himself. In the following pages we will advise first, regardless of the patient's circumstances, the treatment which experience has shown to be most efficient, then recommend such measures as are useful or necessary under any circumstances.

The fundamental principle of a successful treatment of a case of tubercular consumption is *early diagnosis and corresponding promptness in the application of remedial measures, together with the cooperation of the patient supported by the belief that consumption is a curable disease.*[1] To these necessary points must be added fresh air, rest, food, a certainty of the fact on the part of the patient that he can recover.

Fresh Air.—It is not necessary that a patient be removed from his home in order to get fresh air. Human beings are still afraid of drafts, cold air, hot air, night air, indeed of any kind of air except that to be found in a superheated room. This is slowly being changed for it is a fact that a patient may sleep in a room with wind blowing over him, or outside, in a tent or on a porch, in cold weather or hot weather, dry weather or wet weather, provided he is protected by proper clothing.

A patient in any stage of tuberculosis should spend the entire 24 hours in the open air or in a room which allows a free passage of air from one side to another. He should sleep out of doors or in such a room winter and summer. He should either have an occupation outside or have just as free access of air to his working room as to his sleeping room. Window tents, sleeping porches, tents outside the home any and all should be utilized. These

[1] For evidence of the correctness of this dictum see the annual report of the Adirondack Sanatorium for 1912.

helps can be found in the proper books and advertisements. Sleeping bags and caps can be used in very cold weather.

Rest.—No patient with active tuberculosis should do any sort of work which exhausts him, which causes a rise in his temperature or causes increased cough.

Every patient with a continued evening temperature of 100° or over should be absolutely still in bed in the open air until the temperature remains normal the whole twenty-four hours. Neglect of this precaution is likely to bring disaster.

Work.—Every patient with a normal temperature is the better for some employment. This should be varied for each individual case and should be prescribed by the physician with even more care than the drugs. It should occupy but not exhaust the patient. It must be increased according to the strength of the patient.

Climate.—No climate is a specific for tuberculosis. A patient may get well in any climate if he is early taught to follow the above rules.

No patient should ever be allowed to change to another place of living unless in going to another climate he can have all the comforts of home. Unquestionably a patient who is able to live all the year round in such a climate as southern California, Colorado Spring, or the valleys of Switzerland can spend his time out of doors more comfortably than in the usual changeable climate of the middle and eastern United States, but he must remember that he must be out of doors or he will not recover.

Artificial Pneumothorax.—The injection of nitrogen into the chest cavity is lately coming into vogue. A pneumothorax cannot always be made because of pleural adhesions, but in certain cases especially where there is hemorrhage it can be tried; rather flattering reports are being made. A special apparatus is employed which generates nitrogen. This is introduced between the chest-wall and the lung under a measured pressure.

Food.—Food should be abundant and of the best and most nutritious kind. Eggs, meats, including especially fats, poultry, game, oysters, fish, rich animal broths prepared in the most tempting way should be provided, because the quantity taken should be as large as can be digested and assimilated. Milk and cream, cheeses, and the like are eminently suitable. Koumiss or zoolak may be substituted for milk.

Eggs and milk are the favorite food for consumptives, from six to 12 eggs and two or four quarts of milk daily, with as much additional food as the patient can assimilate. Care must be taken however that the food does not disagree with the patient. Many think they cannot take milk. Actually most persons can take it if it is drank slowly, is taken in moderate quantities at one time, or is charged to suit the taste.

Specific Treatment.—Treatment by tuberculin offers the most scientific method of any system. Unfortunately it cannot as yet be safely used in general practice except under certain conditions. The patient must be carefully watched.

Perhaps the last word so far said is contained in the report of the National Association for Study and Prevention of Tuberculosis for 1912.

Brown says, the tuberculin should be injected under the skin. Koch's original tuberculin, T.R. or B.F. may be used indifferently, a dose of

0.000001 or 0.00001 being given and very slowly increased. A small dose is given which is gradually increased until a tolerance is established. The interval is three or four days. Another method is to give larger doses at longer intervals. He believes that usually incipient cases do better with it than without it.

Sanatorium Treatment.—These resorts are of the greatest value where they can be used. Their chief use is education of the patient by precept, as to what is meant by *fresh air*, *rest*, *work*, and *food*.

Necessarily a patient cannot always live in them, and only a few can ever be accommodated.

They can take the hard-worked, underfed individual, or the hampered patient out of himself and give him time for mental and physical rest, plenty of food and continuous fresh air. When so used they are of the utmost value in many cases.

Medicinal Treatment.—Medicines have long since ceased to be thought specific for tuberculosis. They are now looked upon as materials to be used for symptomatic treatment. As such they are most useful in many instances.

Cod-liver Oil.—When cod-liver oil is well borne it may be administered in tuberculosis of the lungs as a food, but it is perhaps no more valuable than cream.

The various compound preparations and emulsions, consisting of cod-liver oil, other tonic substances, gums, and flavors to cover up the taste, are no better borne than the pure oil, and are frequently harmful.

Creasote is not a specific for consumption, but it relieves the catarrhal symptoms and diminishes the cough and expectoration. It may be used in pill form one or two grains three times a day, or in drops two to three drops three times a day in milk.

Creasotal or Carbonate of Creasote.—It has the great advantage of being unirritating and can therefore be given in larger doses.

Iron is indicated in cases of tuberculosis where there is anemia. It is best given in the form of Blaud's pill. Five grains of the mass being given three times a day.

Arsenic is often useful in tuberculosis and may be combined with iron or alternated with it. Many consider arsenic more beneficial than iron. It is not desirable to give very large doses, and 5 minims of Fowler's solution are a sufficient maximum dose. It is especially useful in small doses where there are gastric symptoms, and may be continued in moderate doses for a long time.

Strychnin is a drug that is very valuable in pulmonary consumption, more especially as a heart tonic. It should also be continued over long periods in doses of 1/30 to 1/20 grains (0.0022 to 0.0032 gm.) three or four times a day. Quinin is also at times very useful, especially when there is fever.

Antitubercular Serum Therapy.—Notwithstanding many attempts to produce an effective antitoxic serum for tuberculosis, no satisfactory experimental or clinical results have been attained in any degree comparable to the success with diphtheria antitoxin. The existence of a true antitoxin in the serum of treated animals is in doubt, although certain antibodies have

been demonstrated. The necessity for repeated injections of sera in such a protracted disease as tuberculosis involves some unpleasent consequences due to the serum itself. The occasional development of some symptoms of "serum disease" (urticaria, joint pains and even collapse), make the subcutaneous use of serum sometimes undesirable.

The principal area for which claims are made at present are those of Maragliano and Marmorek. Of late the latter has found favor in some quarters by rectal administration in doses of 5 to 20 c.c. It is at least unobjectionable when thus given. The dose of Maragliano's serum is from 1 to 5 c.c. subcutaneously. It is also given per os or rectum.

Antistreptococcus or streptolytic serum has been used to combat the supposed mixed infection from this bacterium in certain cases. Its efficiency is doubtful, but Bonney and Pottenger claim good results in some desperate cases. The rectal administration is perferable and safe.

Bacterial Vaccine Therapy.—The inoculation of sterile bacteria prepared according to A. E. Wright's methods from cultures obtained from the sputum is a recent and promising method of treatment for chronic mixed infections in pulmonary tuberculosis. Its application is too recent to warrant an opinion as to its usefulness.

Prophylaxis against Tuberculosis.—Sputum, feces and urine are the chief means by which tuberculosis is spread. If every particle of sputum, all the feces and all the urine of tuberculous individuals were thoroughly destroyed, tuberculosis would disappear as an impotant disease in one generation. Unquestionably certain cases come from the use of milk and meat of tuberculous cattle. Sputum the chief offender is frequently, indeed constantly, expectorated on the floor, on handkerchiefs or clothing in the street, this is full of danger.

Under no circumstances should the patient be allowed to expectorate upon the floor, in cars or other public conveyances, or even, if possible to prevent it, in the street. In order to meet these necessities as well as those of other situations in the house paper cups which can be burned after use should be used, or porous paper can be used to be similarly disposed of. The so-called Japanese handkerchiefs answer the purpose admirably. An important instruction is that no piece of paper should be used twice. The sputum should be deposited in the paper and the paper should then be deposited at *once* in some such a receptacle as an ordinary paper bag. Bag and papers containing the sputum can all be burned at convenient intervals.

The pasteboard spit-cups, supported in a rim of steel, recommended by the New York City Health Department, intended to be burned after use, are correspondingly inexpensive and answer the purpose very well. There is also a paper envelope upon the market which can be easily carried in the pocket. This is also inexpensive.

To the same end, diminution of the possibility of harboring dried bacilli, unwashable curtains and superfluous upholstering should be banished from the rooms occupied by tuberculous patients. There should either be no carpets, or they should be replaced by rugs that can be frequently taken up and shaken. The sleeping-car, with restricted air space *per caput*, its costly upholstery and curtains, used year after year, becomes a possible source of infection, especially in routes toward health resorts, but is less serious than

it might be because of the short time that it is generally occupied by the tuberculous and healthy alike. The state-room of the ocean steamer stands a greater chance of being a medium of infection from its longer occupation. Both these useful means of travel should be disinfected after each trip.

When it is remembered how easy it is with ordinary intelligence and simple means to render completely innocuous the bacillus of tuberculosis, it becomes a question how far the surveillance of boards of health can be helpful. For the well to do who can afford to employ an intelligent physician it would seem unnecessary. For the poor it should be associated with material assistance, and carried out with great tact and consideration. For statistical purposes, at least, every case of tuberculosis should be reported by physicians to the proper authorities in order that intelligible records may be kept by which the disease may be traced and followed from its first recognition to its termination, whenever desired.

Persons affected with tuberculosis should not kiss other individuals. Tuberculous mothers should not nurse their children. When a tuberculosis patient coughs he should shield his mouth with a paper handkerchief which must at once be destroyed as the paper contains sputum.

The second source of infection, the milk of the tuberculous cow is avoided by boiling the milk, which is thus rendered thoroughly sterile. There are, however, objections to boiling milk. In the first place, the taste of boiled milk is not always agreeable, but of greater importance is the fact that it gives rise to certain diseases of childhood—scurvy and the like—especially when it is the only food, as in the case of children. Pasteurization has therefore taken the place of boiling milk. Practically, the use by adults of raw milk mixed with other food cannot be regarded as dangerous, but with children fed exclusively on milk precautions should be taken to render it sterile by cooking or if it must be used uncooked it should be the mixed milk of a number of cows. The milk of a cow known to be tuberculous should be invariably condemned and the animal slaughtered. The products of milk—that is, butter and cheese—are, of course, not amenable to the treatment to which milk can be subjected. Safety from infection from these sources can only be secured by a rigid inspection of cows, and by measures to prevent the development of tuberculosis in these animals.

Infection by tuberculous meat is still rarer. In the first place, the flesh of tuberculous animals may not itself be tuberculous, and, in the second place, the cooking to which meat is subjected must kill bacilli. On the other hand, that the communication of tuberculosis by tuberculous meat when carelessly used is possible is shown by the fact that tuberculosis has been produced in animals by the introduction of the juice of the meat of other tuberculous animals and even from tuberculous human beings. The use of raw or half-cooked meat should therefore be prohibited.

In consequence of what has been said of the experimental production of tuberculosis by the inoculation of sputum as well as the increased possibilities of getting into the mouth portions of tuberculous sputum, no one should sleep with a tuberculous patient. Dishes and utensils used by such patients should not be used by others unless first scrupulously cleaned, and this is best accomplished by thorough boiling. The patient should himself

be taught to prevent his hands, face, and bedding from becoming smeared with sputum.

Precautions against autoinfection are scarcely less important than those against infection of others. It has been said that if it were not for autoinfection most cases of tuberculosis, except those within the cranium, would get well. Be this as it may, it is certain that new foci of tuberculosis are constantly being developed in the same patient, which aggravate his complaint and hasten his death. Such a focus is tuberculosis of the intestine, which probably often has its origin in swallowed sputum. Patients should therefore be enjoined against the practice of swallowing sputum.

The close dependence of tuberculosis upon predisposition, hereditary or acquired, chiefly the former, has long been recognized. Treatment early in life as will correct any possible constitutional taint, to be directed against the dangers of infection. Under the circumstances a due amount of attention paid to both factors cannot be amiss.

Children born of tuberculous parents should follow the following rules.

Outdoor life should be sought under all circumstances. Riding and driving should be practised. Judicious athletics, such as develop all parts of the body in good proportion and especially such as secure expansion of the lungs, should be encouraged. Frequent inflation of the lungs should be practised several times a day. Practice with dumb-bells and clubs of moderate weight is pre-eminently calculated to empty the deeper recesses of the lungs of retained mucus, and to cause the blood to move more rapidly through the more remote parts where the circulation is naturally sluggish.

The treatment of *acute tuberculosis of the lungs* is supporting and stimulant, symptomatic and palliative. There is no advantage to be derived by taking the patient away from home. Food and stimulants are required to combat the exhausting effect of the disease and its fever. The fever itself may be lowered by sponging and the cautious use of such apyretics as phenacetin, acetanilid, and the like, because in this form of the disease it is more apt to be continuous and exhaustive in character. The cough must be controlled by opiates, and such other measures must be taken as will make the patient comfortable and mitigate the sadness with which an inevitable fatal prospect is more or less associated. If it should happen that the disease assumes an unexpected chronicity, it may fall into a class of cases in which the treatment laid down for the more chronic forms of consumption is available.

Treatment of Special Symptoms.—*Cough*, there is no symptom that requires more judgment in its management. A slight cough is often best let alone, because it is an effort to remove secretion, the retention of which may be harmful. If a cough becomes harassing, so as to keep the patient awake or otherwise wear him out, it should be controlled. Fresh air as already advised is the very best remedy for cough at our command.

As to cough medicines, creasote and creasotal may be classed among the curative measures for this symptom, as they diminish secretion and thus relieve cough. Moderate cough is often easily controlled by simple syrupy remedies, such as syrup of wild cherry and syrup of tolu, to which some dilute hydrocyanic acid may be added, 2 to 4 minims (0.12 to 0.24 c.c.) to the dose. If these measures are not sufficient, an opiate becomes indis-

pensable. It does not matter much what preparation is used. A teaspoonful of paregoric in the beginning is often sufficient, acting like a charm, or deodorized tincture of opium, if a stronger preparation be needed, will answer better because of its smaller bulk. For this reason, too, sooner or later, the alkaloids of opium are indicated. Codein is the best of these to start out with in doses of 1/4 grain (0.0165 gm.) increased. Heroin is the most recent and is much commended. It is given in doses of 1/20 grain (0.0033 gm.) or more. Morphin, however, becomes ultimately the best remedy in the majority of cases. When this stage is reached the wiser course is not to order it at stated intervals, but at such times as the cough needs especially to be controlled, as at night on going to bed, or once during the night. The dose essential for the purpose named must vary, anything from 1/24 to 1/4 grain (0.00275 to 0.0165 gm.). Proprietary cough remedies should never be used.

In the morning the patient should be allowed to cough for a time to get up the accumulated mucus. If he has to contend with a cavity full of pus it is better to give him a tablespoonful of whisky or a milk punch, to aid in coughing up the accumulated matter, than to give a sedative cough mixture.

The ammonium preparations, chlorid and carbonate, are rarely useful in the cough of consumptives, while their effect is to derange the stomach and destroy the appetite. Sometimes, however, where there is much loose phlegm, the use of the former for a short time may be beneficial. Under the same circumstances terebene is one of the best medicines given in doses of 5 to 10 minims (0.3 to 0.6 c.c.). It taxes the stomach, however, somewhat severely. Terpin hydrate may be substituted in doses of 3 to 6 grains (0.2 to 0.4 gm.).

The *fever* of tuberculosis rarely demands special measures. Should the temperature exceed 103° F. (39.4° C.) there is no more satisfactory or harmless measure than sponging, allowing to remain on the surface a thin film of water, the evaporation of which produces the refrigerating effect. Or 3 grains of antipyrin or acetanilid or 5 of phenacetin (0.2 to 0.33 gm.) may be given, the effect watched, and the drug repeated two or three times if necessary. The high fever of phthisis rarely lasts long and of itself does little or no harm. It is merely a symptom of a more uncontrollable septic process.

Night-sweats demand special measures. By far the most reliable therapeutic agent is atropin; 1/100 to 1/60 grain (0.00066 to 0.0011 gm.) at bedtime usually suffices. It may be combined with morphin, if the latter is necessary. Sponging at bedtime with a saturated solution of alum in alcohol may be efficient when atropin fails, or sponging with simple hot water may answer.

Agaricin or agaric acid in doses of 1/8 to 1/4 grain (0.0082 to 0.0165 gm.) is a modern remedy for night-sweats. Camphoric acid, 20 to 30 grains (1.32 to 2 gm.) in a capsule at bedtime, is another remedy highly recommended. So are muscarin, 5 minims (0.3 c.c.) of a one per cent. solution, and picrotoxin, 1/60 grain (0.0011 gm.). An old remedy is the aromatic sulphuric acid, and it is certainly a good tonic, which, administered in doses of 10 to 20 drops (0.6 to 1.3 c.c.) before meals, may also aid in checking the sweats. Or the following lotion may be used: Balsam of Peru, 1 part; for-

mic acid, 5 parts; chloral hydrate, 5 parts; trichloracetic acid, 1 part; absolute alcohol, 100 parts.

Hemorrhage is an alarming symptom and must be treated, although it is probable that most hemorrhages stop of their own accord. The patient should be immediately put in bed at rest, with the shoulders raised. Ice suitably encased, may be applied to the chest, or cloths wrung out in cold water. A hypodermic injection of 1/4 grain (0.016 gm.) of morphin to an adult is a useful measure to secure quiet. Indeed, treatment should begin with it.

Strapping is very highly recommended by William Gilman Thompson. He directs that pads of cheese-cloth be placed in the axillæ and over the femoral veins, and buckle-straps drawn over them tight enough to prevent venous return, but not to prevent arterial flow. It is best to strap but three extremities at one time, loosening one strap every 15 minutes and reapplying it to the unstrapped limb. Care should not to loosen all the straps at one time.

Lawrason Brown[1] advises the nitrites and morphin, guiding their use by frequent observation of the blood pressure. If the patient is seen early, while bleeding, amyl nitrite is given by inhalation. If he be nervous 1/8 grain of morphin is injected hypodermically. This effect is maintained by nitroglycerin or sodium nitrite at such intervals as will keep the blood pressure between 115 and 120 mm. of mercury. This is easily done with sodium nitrite, of which is given one grain only at a dose repeated often. In severe hemorrhages of long duration the injection of normal human or horse serum is perhaps the best curative remedy.

The *diarrhea* of consumption does not generally become troublesome until tuberculosis of the bowel develops. Slight degrees seem often to relieve the cough. When there is tuberculosis of the bowel it is exceedingly difficult to control. Sufficient doses of bismuth are on the whole the best remedy—sufficient, because at first the smaller quantities, say 10 grains (0.66 gm.), answer, while later much larger doses are necessary. Opium is, however, often necessary, and sometimes the mineral astringents, as the acetate of lead, nitrate of silver, and oxid of zinc, act well in combination with it. Tannic acid is also efficient in combination with opium. In severe hemorrhages of long duration the injection of normal human or horse serum is the best remedy here as in hemoptysis.

IV. Tuberculosis of Lymphatic Glands.

Synonym.—*Tuberculous Lymphadenitis.*

Etiology.—Even before the discovery of the bacillus of tuberculosis by Koch in 1882, it was generally conceded that what has been known as *scrofula*, or the King's Evil, was a true tuberculosis of lymphatic glands. The minute study of these glands showed the presence of miliary tubercles, and since Koch's announcement the bacillus has been found in them.

Tuberculous lymphadenitis is most common in children and young adults, but may occur at any age.

[1] A Suggestion in the Treatment of Hæmoptysis. "Amer. Jour. Med. Sciences," Aug., 1906.

Symptoms.—The *lymphatic glands* most frequently affected are those of the *neck*, which appear in various degrees swollen and tender, in many instances suppurating and rupturing when not opened by the surgeon's knife. The cervical glands in the anterior triangle are usually the first involved, but those in the posterior cervical triangle are also frequently invaded on one or both sides, though commonly on one side more than the other. The cervical and axillary glands may be conjointly involved, forming a continuous chain behind the clavicle and pectoral muscles. The bacillus usually attacks the glands nearest its point of entrance, and presumably the cervical glands are infected by bacilli, which enter by the way of the nasal or naospharyngeal passages. The vulnerability of these mucous membranes to the bacilli is, of course, increased by any inflammatory state present. As a rule, there is little or no constitutional sympathy in such a degree of invasion. There may, however, be slight fever.

More rarely there is involvement of *all* the *lymphatic glands of the body*. Such cases are sometimes met among negroes. In them are swelling, pain, and tenderness of all the visible glands, including the cervical, submaxillary, inguinal and axillary glands, while autopsy discloses the involvement of bronchial, mesenteric, and retroperitoneal glands. In such cases there is more or less continuous *fever*, but death is usually the result of some intercurrent disease, or of pressure upon the respiratory passages.

In addition to the visible pictures described, the bronchial glands are often involved without visible enlargement, the condition being first found at autopsy, when it may or may not be associated with lung tuberculosis. The enlargements, may however, reach such a size as to form a recognizable mediastinal tumor, which may or may not produce the signs of pressure. The bacilli which invade these glands filter through the respiratory passages.

Tabes Mesenterica.—When the mesenteric or retroperitoneal glands are especially involved the disease is called tabes mesenterica. These cases occur among children. The trunk and limbs are puny, wasted, and anemic, while their little bellies are prominent, partly because of the enlarged glands and partly from tympany, producing a striking picture. The tympanitic distention often predominates, making it difficult to feel the enlarged glands. In these cases, too, there is often *diarrhea*, with thin, offensive stools, yet the bowels are not generally the seat of tuberculosis. There may be tuberculosis of the peritoneum, which may also give rise to an uneven, nodular, tender, and painful enlargement easily recognized by palpation. The disease prevails among poorly fed children in the slums and badly drained and ill-ventilated houses of the poor. There are fever, fretfulness, and a general aspect of abject misery. Death generally takes place through exhaustion; or some acute intercurrent disease, such as enteritis, carries off the little sufferers. More rarely adults may be affected with tabes mesenterica, either as a primary disease or as secondary to pulmonary tuberculosis. Tyson remembers a case associated with peritoneal tuberculosis in which the diagnosis between this condition and carcinoma was difficult, the autopsy determining the question in favor of the former.

While tuberculous glands of the neck, and even of the axilla, tend to suppurate, the retroperitoneal and mesenteric glands more frequently caseate without suppuration, and especially characteristic is a tendency in

the latter to calcify. The bronchial glands are also less prone to suppurate, but caseate and, at times, liquefy. The easier accessibility of the external glands to the pyogenic organisms may explain the greater frequency of suppuration in them.

Diagnosis.—The diagnosis of tuberculous lymphadenitis requires its differentiation from *Hodgkin's disease*, lymphatic leukemia; from sarcoma and carcinoma. The affected glands in tubercular lymphadenitis are usually more tender than those in *Hodgkin's disease*, they are more closely adherent to each other and the adjacent tissues, and are, therefore, more fixed and immovable than the glands in Hodgkin's disease. Again, tuberculosis rarely invades more than one group of glands, is associated with caseation and suppuration, while the lymphadenoid growths do not suppurate. Notwithstanding this, the tubercular process is slower. Tuberculosis affects the young—those of either sex under 20—while Hodgkin's disease occurs at any age, is less frequent in the young, and is more common in males. The examination of an excised gland shows the presence of the typical picture described by Longcope.

From *lymphatic leukemia* tuberculosis of lymph-glands is easily recognized by the absence of leukocytosis characteristic of the former and by the differential blood count.

Sarcoma involves groups of glands, and spreads rapidly, invading also adjacent tissues, while *carcinoma* is always secondary to primary cancer somewhere else.

Prognosis.—The prognosis except in tabes mesenterica is generally favorable unless systemic infection occur. Recovery being sometimes spontaneous. This is favored by suitable conditions to be mentioned under treatment. The condition is a menace because of the danger of systemic infection through it, three-fourths of the cases of acute tuberculosis owing their existence to it. Under the circumstances, we must regard cases of recovery from tubercular lymphadenitis in childhood as instances of a survival of the fittest. Certainly our present knowledge demands a prompter attempt to eradicate the local condition than was formerly practised.

Treatment.—The general management of a case of tuberculosis of the lymphatic glands is similar to that of a case of tuberculosis of the lungs. The patient should be surrounded by the most favorable hygienic conditions, have the best of food, take cod-liver oil and the iodid of iron. At the present day tubercular lymphatic glands are frequently removed by the surgeon. Tuberculin is of value in these cases.

When suppuration has set in it is best to open an exposed abscess with the knife, because if allowed to open itself there is apt to result an unhealthy sinuous ulcer, very slow to heal, and when healed causing marked disfiguration by unsightly cicatrices. The access of air permitted by the opening seems also to be antagonistic to the life of the bacillus, for with the healing of the abscess the tubercular process stops in that particular gland.

V. Tuberculosis of the Serous Membranes.

General tuberculosis of the serous membranes is a rare condition, and is recognized chiefly by the signs of tuberculosis of the peritoneum and,

so far as they exist, of the pleura, these being the two serous membranes of greatest extent and importance.

Tuberculosis of the Pleura.

Tuberculosis of the pleura may be suspected when, along with the physical signs of tuberculosis elsewhere, there appear the signs and symptoms of a dry pleurisy, with or without effusion.

Tuberculosis of the pleura manifests itself—

1. As an acute primary inflammation characterized by a serofibrinous or purulent exudate. The onset of such an inflammation may be like that of ordinary acute pleurisy or it may be insidious in its development, like that of the latent form of pleurisy to be described under diseases of the pleura. It may immediately precede pulmonary tuberculosis, be associated with it, or succeed it.

2. As an acute pleurisy the result of extension from an adjacent tuberculous lung, and as such it may be circumscribed, adhesive, or may constitute an extensive serofibrinous or purulent pleurisy.

3. A chronic, adhesive, proliferative, tuberculous pleurisy characterized by great thickening and adhesion of the pleuræ, with tuberculous infiltration of the thickened product.

The symptoms and physical signs are in no way different from those to be described in connection with the nonspecific forms of pleurisy.

Treatment.—Some time often elapses before an absolute diagnosis is made, after which, if the disease is at all extensive and a purulent effusion exists, its treatment is mainly surgical, consisting in drainage secured by the excision.

In addition all of the restorative and hygienic measures employed in tuberculosis of the lungs should be carried out. These hygienic measures are of even greater value in the pleuritic cases because of their natural tendency toward recovery.

Tuberculosis of the Peritoneum.

SYNONYMS.—*Tuberculous Peritonitis.*

Tuberculosis invades the peritoneum in two ways:

1. As acute miliary tuberculosis.

2. As a tubercular peritonitis when the tubercular deposit is associated with an inflammatory proliferation more or less abundant. In a simpler variety of the latter, the *diffuse adhesive*, the peritoneal cavity is obliterated, the coils of intestine being matted together and adherent to the abdominal walls. In a second variety known as *proliferative* peritonitis, there is marked thickening of the peritoneal layer with less tendency to adhesion and obliteration of the cavity. The omentum is sometimes an inch in thickness and composed of tuberculous tissue in various stages of degeneration. The mesentery is similarly infiltrated and shrunken, drawing the intestines together into a *ball-like mass* or tumor as large as a child's head. The coats of the bowel, especially the large gut, also show localized areas of similar morbid changes. In this condition there is generally much liquid. Tuber-

culous peritonitis is sometimes associated with cirrhosis of the liver, whose capsule and that of the spleen may be infiltrated to enormous thickness. There is often in this form considerable effusion, which may be serous or purulent, at times bloody.

Symptoms.—Acute miliary tuberculosis may begin as an acute peritonitis the origin of which cannot be discovered, or it may be mistaken for a peritonitis due to inflammation of some viscus such as the appendix or the gall-bladder.

Cases are constantly mistaken for typhoid fever because of the abdominal distress and distention with fever. In the latent forms the onset is slow, frequently distention of the abdomen with ill health, is the first symptom which attracts the patient's attention. *Ascites* is frequent, it may be either bloody or purulent. Often, the fluid is sacculated. In practically all the cases, irregular fever is prevent. In other cases a tumor is present due usually to a puckered, thickened omentum.

Diagnosis.—The history of the patient, his appearance, the condition of the lungs and the presence of tuberculosis there and elsewhere, particularly in the pleura and bowel, whence extension to the peritoneum is easy by the lymphatic vessels are of importance. Four-fifths of all cases of tuberculous peritonitis are said to succeed primary tuberculosis of the lungs. In children tuberculous peritonitis is frequent as a part of a general miliary tuberculosis. The character of the liquid and the presence of the tuberculin test will help to distinguish these cases from carcinoma of the peritoneum. The mass may simulate also an ovarian cyst. Tuberculosis in other organs should help to distinguish the cases.

Treatment.—The treatment for tubercular peritonitis is the general treatment for tuberculosis, with such operative interference as may be deemed appropriate after a careful study of each case. The results of operation thus far have been quite sufficiently satisfactory to justify its repetition in suitable cases.

VI. Tuberculosis of the Genito-urinary Organs.

This includes tuberculosis of the kidney and its pelvis tuberculosis of the ureters, bladder, ovaries and testes.

Tuberculosis of the Kidney.

Morbid Anatomy.—Tuberculosis presents itself in the kidney in two forms:

1. In the shape of *miliary tubercles*, which are a part of a *general tuberculosis*, giving rise to no special local symptoms; as secondary invasion confined to the kidney, or rarely as primary in the kidney.

2. As *secondary* foci of localized *tuberculosis*, which in time may fuse to form larger areas that undergo caseation and liquefaction, transforming the whole kidney a series of cysts containing cheesy matter or at times into a sac of purulent or chessy matter or such tuberculosis may begin in the kidney or may start in the prostate gland, bladder, ureter, or pelvis of the kidney, and may extend also into the testicle and epididymis in men and the ovary and Fallopian tubes in women.

Symptoms.—The first form is without special symptoms. In the second class there may be none at all or *they may simulate closely those of nephrolithiasis. Tenderness* to pressure should be especially sought. Frequently, subjective symptoms are reflected to the bladder, and they include frequent micturition, pain, and tenderness in the region of the bladder. There is also purulent urine, but commonly this differs from that of cystitis. It is more uniformly acid in reaction, and contains pus less admixed with mucus. Blood is much more frequent than in simple cystitis, and correspondingly albumin. Tubes casts are very rarely found. Cheesy masses are sometimes present in the urine and with them the tubercle bacillus, which is a pathognomonic sign. It should always be sought. It should not be confounded with the bacillus found in smegma. Hence, a negative result does not, however, exclude tuberculosis. Only the urine from catheteirzed ureters should be examined for bacilli. If bacilli are absent guinea-pigs should be inoculated with the same urine.

A phenolsulphoepthallein test should always be made before any operative interference with the kidneys.

Diagnosis.—In the absence of such conclusive proof as bacilli in the urine and a pig test the presence of tubercle elsewhere, as in the lungs or nearer parts, as the testicles and prostate in men or the ovaries and Fallopian tubes in women, affords suggestive evidence. The latter may be investigated through the vagina and rectum. Catheterization of the ureters may also be practised and stenosis of the ureter due to tubercular infiltration of the pyeloureteral wall thus recognized. In other cases where the lungs are not primarily tuberculous they may be secondarily invaded. Hydronephrosis may result from complete obstruction of the ureter by tubercular infiltration. A cystoscopic examination including catheterization of the ureters should always be made. The urine by these catheters can be examined for tubercle bacilli. Colargol can be injected through the ureters and cavities in the kidney substance demonstrated by X-rays, as shown by Keene and others.

Treatment.—Beyond the general restorative and palliative treatment useful in general tuberculosis there is no medical treatment of tubercular kidney. As soon as the diagnosis is made of tuberculosis of a single kidney and the general condition of the patient justifies an operation, the surgeon should be called and nephrectomy done.

Tuberculosis of the Pelvis of the Kidney, Ureters, and Bladder.

It is difficult to separate tuberculosis of these parts of the urinary tract. So far as symptomatology is concerned, outside of the bacteriological examination, the symptoms of tuberculosis are those of simple inflammation. If the disease is advanced there is tenderness, but this is the case also when there is impacted stone or pyelitis from other causes. The invasion of the bladder produces symptoms like those of cystitis, including frequent micturition and purulent urine in which there may be a small amount of blood. These symptoms, again, are not peculiar to tuberculosis, and the examination for bacilli again becomes necessary. The diagnosis is largely one for a surgeon, though the medical man should see

that the methods are carried out. Cystrocopic examination will show whether the bladder is the seat of lesion or not. Catheterization of one or both ureters will show whether the kidneys and pelves are normal or not.

Tuberculosis of the Ovaries, Fallopian Tubes, and Uterus.

The ovaries may be the seat of miliary tubercles or may contain large cheesy masses. Ovarian tuberculosis is commonly associated with tuberculosis of the Fallopian tubes. The symptoms of the former are in no way different from those of ovaritis from other causes. Fallopian salpingitis produces a hard and thick infiltration of the Fallopian tubes, which may be recognized by the usual methods of examination for the disease of these organs. The uterine ends are commonly closed, while the intervening portion may be dilated and contain mucus, pus, and cheesy material. Tubal tuberculosis is commonly double.

Tuberculosis also invades the uterus, infiltrating it by miliary tubercles, which coalesce, soften, and break down, producing metritis and ulceration, discharges from which may contain the bacilli. The symptoms of the resulting metritis are the same as those of metritis from other causes.

Tuberculosis of the Testes, Prostate Gland, and Seminal Vesicles.

Tuberculosis of the testis and prostate in not infrequent. It presents itself as cheesy infiltration, which more frequently does not liquefy. More rarely, the vesiculæ seminales are invaded. The enlarged vesiculæ seminales may be felt through the rectum. The symptoms of this form of prostatic disease are in no way different from those of other diseases of the prostate with enlargement until rupture takes place.

Tuberculosis of the testis is not such a rare affection. It is commonly secondary to that of the bladder and prostate, whence the bacilli travel along the vas deferens into the epididymis, which may be converted into a cheesy mass surrounding the testicle. With the invasion of the testicle further enlargement results with softening, ulceration, and fistulous burrowing. The walls of these fistulæ are infiltrated with tubercles. This malady is characteristically painless.

The treatment of these conditions is mainly surgical, although the general measures usual in tuberculosis elsewhere are also suitable.

Tuberculosis of the Liver.

In acute miliary tuberculosis, the liver is frequently the seat of miliary tubercles. Large tubercles rarely occur, but they may be present and be multiple. Tuberculosis of the gall-ducts occurs. There are no distinctive symptoms, but in general tuberculosis with hepatic involvement one or the other of these forms may be suspected to exist.

Tuberculosis of the Mammary Glands.

The mammary gland, though rarely invaded by tuberculosis, is nevertheless an occasional seat, it may occur at any age and in both sexes, Warden having collected 58 authentic cases in literature, nearly 90 per cent. of whom were females. Three forms are recognized by Adami, the *acute miliary*,

the *discrete* and the *confluent*. In the *discrete* form the breast is not necessarily enlarged, the nodules may be one or many, and may be immovable. The skin is intact, each nodule on section shows a central grayish material or it may be pyriform in character. The *confluent* form gives rise to considerable enlargement of the breast, there is a single large mass. On section the mass is found to be filled with cavitis of irregular size and shape. Radiating from the central area are rays of fibrous tissue. These cavities may communicate with the exterior by a sinus. Sometimes this form terminates in a *cold abscess*. The bacilli causing the disease are carried by the blood in the acute miliary form.

Tuberculosis of the Heart and Blood-Vessels.

Tuberculosis of the Heart and Pericardium.—Tuberculosis of the myocardium sometimes occurs—usually as extension from a tuberculous affection of the pericardium they are difficult to differentiate from syphilis—presents itself in the shape of *miliary tubercles* scattered throughout the substance of the heart. *Tuberculous pericarditis* may be acute or chonic, more commonly acute, caused by sudden invasion. Both are usually a part of a general tuberculosis. Very rarely the acute form is primary. Tuberculous pericarditis is followed by exudation of fibrin, and sometimes of blood and pus. It is found sometimes in old persons in whom it promptly causes death. Such pericarditis is also commonly adhesive, and is not distinguishable by physical signs and symptoms from the other forms of pericarditis.

In cardiac tuberculosis it is supposed that the bacilli arise from long latent foci of tuberculosis of the bronchial or mediastinal lymphatic glands. The latter, on the other hand, may be secondarily invaded from the cardiac tuberculosis.

Tubercles are sometimes found on the valves of the heart.

Tuberculosis of Blood-vessels.—May arise from infection through the blood or it may also invade the blood-vessels of a part attacked. Both the intima and adventitia may be effected. Sometimes the tubercles caseate and rupture into the vessel and in tuberculosis of the lungs hemorrhages are commonly due to such invasion, which weakens the vessel and ultimately perforates it.

LEPROSY.

Synonym.—*Elephantiasis Græcorum.*

Definition.—Leprosy is an infectious disease, due to the *bacillus lepræ*, characterized by a subcutaneous and submucous nodular infiltrate, or by similar infiltration of nerve-trunks. The former constitutes tubercular leprosy; the latter, anesthetic leprosy.

Etiology.—The bacillus of leprosy was discovered by Hansen in 1871, and subsequently clearly described by Neisser, and is especially characterized by its close resemblance to the tubercle bacillus, both in morphology and in its reaction to stains. Duval has recently been able to cultivate it on mature green bananas. So far as is known it does not exist normally outside

the human body and they are discharged only by secretions of the diseased individuals. They are for the most part found in the interior of cells, rarely outside of them. Some of these cells are of large size and known as lepra cells. In the interior of these cells the bacilli often form clumps. They are exceedingly numerous in leprous tissue.

While the disease is contagious, its spread, even under the most favorable circumstances, is exceedingly slow, the most intimate contact, as that between parent and child, being often unattended by inoculation. Experimental inoculation was, however, successfully performed on a Hawaiian convict by Arning, as well as in rabbits by Melcher and Artmann. According to Morrow, in the majority of cases the disease spreads by sexual intercourse, but cracks and fissures in the skin also favor the lodgment of the bacillus. In certain countries, especially the tropical, its spread is more rapid. Such are India, where there are said to be 250,000 lepers, and the Sandwich Islands, where, in 1889, there were 1100 in the settlement at Molokai.

In this country the cases are for the most part isolated ones that enter by the seaports of the Pacific and Atlantic coasts. In Tracadie, on the Gulf of St. Lawrence, there is, however, a leper settlement, the disease having been brought from Norway in the latter part of the eighteenth century. The number of cases is being gradually reduced, there being in 1896 but 18 as compared to 40 a few years previous. This is apparently the result of segregation, which is now generally practised where possible. A few cases have been reported from Texas and there is a settlement in Louisiana and Florida. The commission appointed in 1899 reported 278 cases in the United States.

All ages and sexes are liable to this disease. Animals are not subject to it, although guinea-pigs have been successfully inoculated. A curious impression has arisen that the disease is caused by eating spoiled fish or vegetables. To this belief Jonathan Hutchinson has given the weight of his opinion. In view, however, of the acknowledged bacillary origin of the disease, this can only be considered as a predisposing cause that lowers vitality by altering nutrition.

Morbid Anatomy.—Tubercular leprosy is characterized by its nodular outgrowths on the skin, especially of the face, the extension surfaces of knees and elbows, and especially of the prepuce and scrotum, the nodules being made up of a small-celled infiltrate together with the "lepra cells," maintaining itself for a considerable time, after which it breaks down and ulcerates. The ulcers may heal, producing cicatrices. The mucous membrane is also invaded, particularly that of the eyelids, the conjunctiva, cornea, and larynx. Lymphatic glands, cartilage, liver, lungs, and spleen are also at times affected. The lepra nodes are vascular, differing in this respect from the nodules of tuberculosis.

The morbid anatomy of the anesthetic variety will be included in the anatomical changes of the skin to be described in the symptomatology of that type of the disease.

Symptoms.—Nothing is known of a *period of incubation*. The outbreak of the disease is apt to be preceded by an *intermittent febrile movement*, by drowsiness, dyspepsia, vertigo, etc., symptoms which have been mistaken for

intermittent fever and which may last for one or two years. Except for the length of time over which they extend they do not differ from the symptoms which may precede many specific diseases. There is often an *erythematous redness* of the skin, which in places becomes pale and in others assumes a brownish tinge, usually those areas are hyperesthetic. From this appearance the name *macular leprosy* has been applied to certain cases which go no farther. From these spots the pigment may also disappear, leaving perfectly white anesthetic areas—*lepra alba*.

In the further development of the disease, in the *tubercular* or more usual form, an *infiltration of the skin* with *tubercular nodules* takes place. These areas usually first appear on the face, remain for a long time intact, without degenerating, but sooner or later, as a rule, though often only after many years, softening and ulceration takes place. Some of them, on the other hand, gradually disappear without ulceration. The number of nodules varies greatly. Some of them are pediculated, others are a simple thickening of the skin, which is conspicuous in such portions as the eyelids, nose, and ears, parts of which may disappear by ulceration. Even the cornea and conjunctiva may be the seat of nodules, and blindness may result.

The same development may take place in mucous membranes producing obstruction of the respiratory passages, including the nose and larynx. There may also be leprous deposits in internal organs, including the liver, spleen, lungs, and lymphatic glands.

In the nervous or *anesthetic* form the peripheral nerves become infiltrated with the leprous growth and are converted into thickened cords that may even be felt under the skin. These are at first painful, but later become anesthetic. Trophic phenomena of a striking character result, producing dryness, smoothness, and tightness of the skin with a total absence of nodules. Atrophy and wasting ensue from the same cause, and toes, fingers, and even larger limbs drop off. Great vesicles also sometimes form. Subsequently are added signs of weakness and exhaustion which gradually increase until the patient succumbs.

Diagnosis.—The diagnosis of the tubercular form when fully developed is not difficult though it might be mistaken for syphilis or tuberculosis.

The anesthetic variety resembles closely certain forms of *scleroderma*, the face has a characteristic canine appearance, but the trophic changes are more extensive. The resemblance of the early stage to intermittent fever has been referred to. The diagnosis may be made absolute by the detection of lepra bacilli in portions cut out for the purpose of study.

The anesthetic or nervous form of leprosy and *syringomyelia* bear a close clinical resemblance. It may also be mistaken for peripheral neuritis, progressive muscular atrophy, or Raynaud's disease, but the leper almost without exception has discolored areas of the skin which are anesthetic and are frequently discolored.

Prognosis.—The course of the disease is almost always prolonged, and the patient may die from intercurrent disease. In some cases death results from the gradual exhaustion of the system, which is more rapid in the ulcerative forms. From the nervous form of leprosy recovery does sometimes take place, though the secondary changes resulting remain permanent.

Prophylaxis.—It is known that the disease is probably spread by the discharges of the patients. Therefore all cases should be at once reported to the authorities and strict measures taken to prevent the patient from spreading the disease by handling food, clothing and the like. It is quite possible also that the disease is spread through the agency of blood-sucking insects and this should be taken into account. Segregation should be practised whenever possible, for such a course is invariably accompanied by a falling off in the number of cases, and the continued practice of this method must ultimately result in the disease being stamped out. In this segregation, sanatoria should be established which would allow the leprous family to live normal lives, and where the segregated individuals should not be made to feel they are undergoing imprisonment.

Treatment.—Hygienic measures should not be neglected. The patients should live in the open as much as possible, should have good nutritious food, exercise which should be regulated must always be used. As yet there is no specific treatment.

Medicines.—Innumerable drugs have been used without the least effect. Chaulmoogra oil is now in the greatest favor. It is used internally and externally. The dose is five to 50 drop doses given in capsules or in milk. A purified form of this oil is used called *antileptrol*. *Nartin* is another preparation now in favor. It is a bacterial fat, produced by a streptothrix isolated from leprous nodules. A diluted mixture of this called Nartin B. is injected subcutaneously in 1 c.c. doses once a week for five or six weeks.

Arsenic has been used as a general tonic.

Serum and vaccine treatment so far have availed nothing.

Salvarsan has been used with good results in a few instances. *Naster's Treatment*, the injection of living cultures of streptothrix leproides, have been reported favorably by Deycke.

Surgical interference may be employed wherever it seems advisable.

Finsen light and Röontgen Ray treatment have both been tried.

INFECTIOUS DISEASES OF DOUBTFUL NATURE.

ACUTE FEBRILE JAUNDICE.

Synonyms.—*Weil's Disease.*

Definition.—An acute infectious disease, characterized by jaundice and fever, described by Weil in 1886.

Etiology.—The cause is as yet undetermined, but it affects males in preference to females, especially butchers, laborers, and brewers, and its subjects are from 25 to 40 years of age. Exposure to cold may be considered an exciting cause. A few cases have occurred in this country, two having been reported from the Philadelphia Hospital by J. H. Musser and John Guitéras. Weiss considers that the symptoms and lesions most resemble the bilious typhoid described by Griesinger, while the latter has been claimed to be identical with the typhoid icterodes of Egypt.

It occurs commonly in the summer months, and nearly always in groups of cases. But for the last fact the disease might well be regarded as catarrhal jaundice.

Symptoms.—The illness sets in suddenly, after exposure to cold, as in a beer vault, most frequently with a *chill* and without prodrome. There is *fever*, with temperature of 102° to 104° F. (38.9° to 40° C.), *headache*, *muscular* and *joint pain*, and *epigastric pain*, which is characteristic. There is especially *tenderness* in the *calf muscles*. *Jaundice* promptly makes its appearance. The fever lasts usually from ten to 14 days, and is characterized by decided remissions. The *liver* and *spleen* are both *enlarged;* the former may be tender. Associated with the jaundice are the usual *clay-colored stools* of obstructive jaundice. Beyond the epigastric pain, which may be hepatic in origin, *gastro-intestinal* symptoms are not marked, though the tongue is coated, and there may be vomiting and diarrhea. There may be dizziness, confusion of mind, and even delirium. The *urine* contains biliary coloring-matter; sometimes albumin with casts and even blood.

After a duration of from eight to fourteen days, convalescence sets in, usually slowly, and it may be prolonged.

Diagnosis.—The conditions with which Weil's disease might be for a time confounded are malaria fever, acute yellow atrophy of the liver, phosphorus poisoning, and catarrhal jaundice. The first would be excluded by the absence of the plasmodium of malaria, while the mildness and favorable termination would exclude the second and third. Catarrhal jaundice is distinguished by the absence of fever, and of muscular, joint, and epigastric pain, which characterize Weil's disease.

Prognosis.—Recovery is usual, but a few autopsies have been made, with the discovery of no definite morbid anatomy. There is cloudy swelling and even fatty degeneration of the cells of the heart, liver, kidney, stomach, and intestines.

Treatment consists in attention to the diet and general symptomatic treatment.

MILIARY FEVER.

Synonyms.—*Febris miliaris; Sudor anglicus; Sweating Sickness; the Sweating Disease of Picardy; the English Sweat.*

Definition.—An infectious fever of unknown cause, characterized by profuse sweats and an eruption of miliary vesicles.

Etiology.—As to the specific cause nothing is known. It is not contagious nor inoculable, and not favored by crowding. Most epidemics occur in summer, fewest in the autumn; second in frequency is the spring; third, the winter. Moist, warm, and unchanging weather favors the disease. Contaminations of the soil, such as arise from neglected drains and collections of refuse, also contribute to its causation. More women are affected than men, and the vulnerable age seems to be between 20 and 50 years. The healthy and strong are as likely to be attacked as the weak, and the rich as well as the poor. Lately it has been confined to Italy and Prussia.

Morbid Anatomy.—No characteristic anatomical changes have been noted in miliary fever. The internal organs are generally hyperemic. The spleen is often enlarged. The most striking feature is the tendency to rapid decomposition, "beginning almost during life," as has been said. The blood is thin and dark in color.

Symptoms.—After an *incubation* of two or three days the patient goes to bed apparently well, and wakes up in the night dripping with *sweat*. With this is a *sense of oppression*, and even *pain*, in the precordial region, *tenderness* and pain in the epigastrium, *palpitation*, *headache*, *dizziness*, and *muscular cramps*. The *temperature* is abnormally high, the *pulse* and *respirations* are frequent; there s even *dyspnea*, sometimes very violent. The perspiration continues, saturating the bed clothing and diffusing an unpleasant odor throughout the room.

On the third or fourth day, as a result of the profuse sweating, miliary *vesicles* make their appearance, at first so minute at to be scarely visible, though they may be felt by passing the hand over the skin. As they become larger they are easily visible by their crystalline contents, which later become turbid and even milky. They appear first on the neck and breast, then over the back and extremities, less frequently on the abdomen and scalp. After two or three days they burst, dry up, and form crusts, which subsequently desquamate. With the appearance of the eruption the other symptoms disappear rather suddenly, but there is often noted a burning and prickling sensation of the skin. There is generally loss of appetite, sometimes nausea, seldom vomiting, scanty urine, and especially constipation.

The *duration* of the disease is usually from six to eight days, although it may be prolonged beyond this, the eruption being sometimes delayed to the seventh, tenth, and even 15th day. Relapses may occur. Sometimes the disease assumes an intermittent character.

Diagnosis.—This is not difficult. The prevalence of an epidemic, profuse sweating, and rash scarcely permit an error.

Prognosis.—The prognosis has varied greatly in different epidemics, the mortality in some of the earlier reaching as much as 50 per cent., while in others none died. The average may be put down at from eight to nine per cent.

Treatment.—The treatment is mainly expectant and symptomatic. Simple febrifuges and acid drinks are indicated. Warm baths and sponging of the skin with warm water are soothing and comforting. The precordial distress and apnea may require anodynes, preferably subcutaneously administered. The sweating itself, if alarming, may be treated by hypodermic injections of atropin, 1/200 to 1/100 grain (0.00033 to 0.00066 gm.), p. r. n., or this drug may be given by the mouth in the same dose.

GLANDULAR FEVER.

SYNONYM.—*Drüsen-Fieber.*

Definition.—An acute infectious fever of children, characterized by inflammation of the lymph glands of the neck, especially those back of the sternocleidomastoid muscle.

Etiology.—No responsible bacterium has been found. The disease may be epidemic, as was that which occurred in Bellaire, Ohio, described by West. It has been observed to prevail more commonly between the months of October and June in the winter season. The infection, whatever it may be, probably enters through the tonsils or the pharyngeal mucous membrane.

Morbid Anatomy.—This includes the enlargement of the glands, which forms so essential a part of the disease. The enlargement may involve not only the cervical glands referred to, but the axillary, inguinal, bronchial, and even the mesenteric. Thus, in West's report of 96 cases occurring between the ages of seven months and 13 years, in three-fourths of them the postcervical, inguinal, and axillary glands were involved, with the mesenteric in 37 cases. The liver and spleen were also enlarged, the former in 87 and the latter in 57 cases.

Symptoms.—The *period of incubation* lasts from five to eight days. The disease is characterized by sudden onset of stiffness with pain on moving the head. Along with this there is fever with a temperature of 101° to 103° F. (56° to 57° C.) with sometimes nausea and vomiting. The enlargement of the glands does not make its appearance until the second or third day, and may attain a size from that of a pea to a hen's egg, but rarely goes on to suppuration. The glands are tender to the touch, but there is not usually redness of the skin. There may also be some hyperemia of the tonsils, or pharyngitis. More rarely there is invasion of the tracheal and bronchial glands which may be the occasion of cough. The swelling persists from two to three weeks, although the fever does not last nearly so long.

Complications.—Among these which may be named as possible are hemorrhagic nephritis, postpharnygeal abscess, and acute otitis media.

Diagnosis.—The disease is to be distinguished from the various forms of infectious sore throat found in scarlet fever and diphtheria which may cause a similar affection of the lymphatic glands.

Prognosis.—Favorable.

Treatment.—Active treatment is scarcely needed. The patient should be put to rest. Cold or warm applications may be made, whichever form is found more comfortable. An aperient, such as a dose of oil or calomel, may be desirable at the very beginning. West recommended small doses of the latter drug.

LEISHMANIASIS.

(*Kala-Azar.*)

Definition.—This is a condition occurring in Asia, and particularly in India, Ceylon, Syria and China. It is caused by a parasite of the Leishmania group, and is supposed to be spread by the bite of a bedbug.

Symptoms.—The symptoms are enlargement of the spleen, irregular fever lasting for a long while, and signs of progressive secondary anemia. According to observers the blood changes vary according to the stage of the disease. According to Leishman there is always an anemia, the red cells numbering from three to four million. Early in the disease there is a slight leukocytosis and then leukopenia is established in the later stages, when from one to two thousand leukocytes are found. Careful examination will show the Leishman body in the leukocyte. Leishman suggests citrating the blood and collecting the corpuscles by centrifugal force. A certain diagnosis can be made by puncturing the spleen, because the Leishman bodies can always be found in the blood from the spleen. But this procedure is not without danger.

Diagnosis.—This may be mistaken for a secondary anemia and for infantile splenic anemia, and possibly for trypanosomiasis. However, only the history of the case as having a chance of infection in southern Asia, and careful repeated examination of the blood will make a diagnosis. Sleeping sickness would show the presence of trypanosomes, which are absent in Leishmaniasis.

ACUTE POLIOMYELITIS.

(Infantile Paralysis.)

Definition.—Acute poliomyelitis is an infectious disease due to a filterable virus. It is characterized by fever, sore throat, pain, prostration, and usually paralysis of one or more limbs. It can be transmitted from one person to another, probably through discharges from the nasopharyngeal mucous membrane.

Etiology.—The disease is often epidemic, and sometimes apparently sporadic. Owing to the difficulty of diagnosing early and abortive cases, this form of the disease is constantly overlooked. Its portal of entrance is probably the mucous membrane of the nasopharynx.

Virus.—This is definitely proven to be filterable, and is therefore not a known bacterium. Flexner in a recent communication reports the cultivation of the organism which is an anaerobic body, but he cannot as yet classify it. It is resistent to glycerization; it resists freezing 2° C. to 4° C. It can be destroyed by heat, by 2 per cent. solution of hydrogen peroxide and by corrosive sublimate.

The serum of recovered individuals and recovered monkeys will destroy the virulence in vitro. It can certainly be transmitted from one human being as proven by Wickham. It spreads along routes of human travel. Flies can be passive distributors of the disease.

Age.—The disease is most frequent in early ages, about 90 per cent. being in the first decade, but 220 cases in the Swedish epidemic were between 10 and over 25. The following age table taken from the Rockefeller report, No. 4, Jan. 24, 1912[1] is of importance.

TABLE OF AGE INCIDENCE.

	Months		Years													
	Under 6	6–12	1–2	2–3	3–4	4–5	5–6	6–7	7–8	8–9	9–10	10–15	15–20	20–25	Over 25	Total
Rockefeller Institute Hospital, 1911.	3	15	62	29	18	13	6	4	0	2	1	3	0	1	0	157
New York epidemic, 1907.	62		221	180	10	663	28	18	11	11	7	14	5	1	2	729
Wickman, Sweden, 1906.	183				214				179			229			220	1,025

[1] The authors are indebted to this same pamphlet for the quotations in this article and many of the statistics.

Season.—It reaches its maximum in the late summer and early fall.

Location.—It is a disease of open country rather than of built up cities. The authors' cases have been largely among the residents of the suburbs.

Morbid Anatomy.—The meninges are edematous and injected. The amount of cerebrospinal fluid is about normal. The brain and cord appear edematous. The gray matter is swollen.

The first change in the meninges most noticeable on the anterior part of the spinal cord, is an acute interstitial meningitis. There is a small celled infiltration about the vessels of the meninges. There are also minute hemorrhages.

"Cellular exudate, hemorrhages and edema, all dependent upon vascular changes, are the first effects of the virus. This is the reason for the nerve changes. The nerve changes are certainly partly vascular, they may also be toxic, and partly due to anemia.

If the hemorrhages and exudate are absorbed soon enough, the cells will ragain their power. The nerve cells either degenerate or recover.

"Of practically constant occurrence are the lesions in the posterior root ganglia. The histological changes are similar to those that take place in the cord itself. There is an infiltration of small round cells in the lymphatic spaces surrounding the vessels which enter the ganglia from the meninges. This has been shown experimentally to be the first step in the process. Then follows a more general, diffuse exudation of cells, degeneration and necrosis of the nerve cells, and finally the entrance of polymorphonuclear leucocytes into the necrotic cells and removal of the disintegrating cells by neurophages. The suggestion has been made that these lesions in the sensory ganglia may in part account for the pain which is such a constant feature of the acute stage of the disease. Another element in the production of pain is the cellular infiltration which is found along the nerve roots."

The lymph glands and the tonsils are enlarged. The spleen often enlarges,

The changes are much like those of typhoid fever. This is particularly marked in the liver. "The disease must be regarded as a generalized process which affects parenchymatous organs, lymphoid tissue, and more especially the nervous system. It is possible that two distinct effects of the disease on the organism should be differentiated."

Symptoms.—The period of incubation is varied and difficult to fix, but it is probably from four to six days. The prodromal symptoms are fever, sore throat, drowsiness, sweating, and muscular pains. Frequently these symptoms are so mild that they are overlooked. Sometimes these symptoms are very severe. The temperature may reach 104°. Drowsiness is common. The pain is increased by flexion of different parts of the body. Sometimes there are symptoms referable to the respiratory tract, coryza, bronchitis, etc.

Gastric and intestinal symptoms are common, vomiting and diarrhœa constantly appearing. These symptoms are followed in the first to the 12th day of the disease by a sudden paralysis. The greatest number develop the paralysis on the first, second or third day of the disease. In certain cases the paralysis is the first sign noticed by the parents.

Other cases have a remission for two or three days and then relapse. In the more severe type the so-called cerebral form, in which the paralysis is

of the bulbo-spinal type, the patients are almost comatose. There may be retraction of the head. Sometimes the patients are delirious, after the acute symptoms disappear. The patient's general condition improves, but the paralysis remains.

Sometimes the paralysis is complete. It varies from this to a mere weakness. Sometimes the paralysis is very transient, lasting from two or three days to several weeks, and entirely disappearing. The parts involved are most frequently the legs, but any nerves, the cranial included, may be involved.

Blood.—In the report above mentioned, there was a constant leucocytosis, sometimes as high as 30,000.

Cerebrospinal Fluid.—It is usually clear. Two hundred and thirty-three fluids were examined from 69 cases. The pressure is usually above normal. They all reduced Fehling's solution. The number of cells is usually increased. The globulin reaction is marked.

Diagnosis.—Until the paralysis occurs, this is most difficult. The prodromal symptoms are exactly like those of many infectious diseases. In the presence of an epidemic, however, fever, drowsiness, pain, and sore throat are very suspicious symptoms. The sudden paralysis is pathognomonic.

Prognosis.—Eight hundred and sixty-eight of Wickham's cases show a death rate of 16.7 per cent. The degree of the paralysis is not a criterion of its permanency. Certain cases recover, but in the Massachusetts cases only 16.7 per cent. made complete recoveries.

Prophylaxis and Treatment.—Early diagnosis is important. Fever, pain, drowsiness at the time of epidemics should be the sign for isolation and the administration of urotropin. The mouth should be gargled with 2 per cent. peroxide of hydrogen solution, and the case at once isolated. When the diagnosis is certain, the case must be isolated, nurse and attendants should wear gowns and gloves.

As the virus may exist in the exudate from throat and nose is certain, all such exudates should be disinfected. The child should be in bed perfectly quiet. Urotropin, six grain doses every three hours should be administered. Pain is often severe in lifting and changing the patient. Care should be observed in moving the patient. The room should be warm but well ventilated.

Salicylates are of value. Bromides and morphine may be demanded. In severe cases where the respiration fails, artificial respiration must be employed. Here the pulmotor should be employed because of the certainty of its action.

Treatment of the Paralysis.—In the early stages absolute rest is imperative. This should last as long as there is pain. On the disappearance of pain, passive movements and massage should be employed twice a day. The patient must be encouraged to attempt voluntary movements. This should be done by tempting the patient to play, putting it on a mattress and rolling over. Electricity should be used, both galvanism and faradism.

Splints, an other orthopedic means, must often be employed, but at all times until contractions begin to occur, the patient must be cajoled into attempting voluntary movements. The hope is that prophylactic serum may be employed. This is not as yet applicable to human subjects.

SECTION II

DISEASES OF THE DIGESTIVE SYSTEM.

DISEASES OF THE MOUTH.

THE COATED TONGUE.

The *natuarl color* of the tongue at its anterior two-thirds is a pale red, on which the *fungiform papillæ* stand out as brighter red points. The epithelium covering the *filiform papillæ*, which are much more numerous and uniformly spread over the dorsal surface of the tongue, is thicker, and they are therefore less distinctly seen. As the base is approached, a grayish color is assumed on account of the greater thickness of the epithelium. At the base are seen the *circumvallate papillæ*, arranged in two rows of red circles. In the *furred* tongue the epithelium is abundant, though it is doubtful whether it is present in increased quantity or is simply raised by hyperemic swelling of the paillæ. The "fur" is also contributed to by various forms of fungi. Too much stress should not be laid on the coated tongue. Some persons have a coated tongue and are perfectly healthy, while others have fair-looking tongues and are ailing seriously with those derangements which are commonly attended with coated tongue, especially gastrointestinal disturbances. Food such as milk, licorice and tobacco also contribute to the coating of the tongue.

The *dry*, brown color of the tongue in low fevers is due to a drying of the exfoliating epithelium, admixed sometimes with mucus or saliva. The tongue may also be coated with dried food and sometimes with dried blood, due to capillary hemorrhage, which imparts to it a black color—the *black tongue* of certain malignant fevers. It is sometimes *pale* and *anemic* in persons whose blood is poor and deficient in red blood-disks. In other cases it is enlarged and flabby, while its edges are easily indented and marked by the teeth. A *bright red* or even a raw appearance of the tongue is met with in certain fevers, particularly in the early stages, when it may also be dry and glazed. It may be coated at the beginning, but later the epithelium desquamates freely and the whole surface may be red; or the fungiform papillæ may be hyperemic, swollen, and unusually distinct, constituting the "strawberry" tongue so characteristic of scarlet fever. The raw-beef appearance of the tongue is often seen toward the close of exhausting diseases, like tuberculosis.

DERANGEMENTS OF DENTITION.

The most serious accident of dentition is what is known as the reflex convulsion, which will be considered among nervous affections. Other gastrointestinal derangements will be discussed under diarrhea of infants.

These are not always reflex. They may be excited by toxic qualities of the saliva, which is not only increased, but altered as well. Other abnormalities including anomalies in the order of eruption of teeth occur, and certain markings on the teeth, ascribed to stomatitis are met.

The order of natural eruption of the *milk teeth* is well shown in the accompanying diagram. The first to appear are the lower central incisors (1,1), at the age of from four to seven months, then a few weeks later the upper central incisors (2, 2), and next the upper lateral incisors (2*a*, 2*a*). Not until the beginning of the second year come the lower lateral incisors (3, 3), and almost simultaneously the four anterior molars (4, 4, 4, 4). In the second half of the second year come the four canines (5, 5, 5, 5), includ-

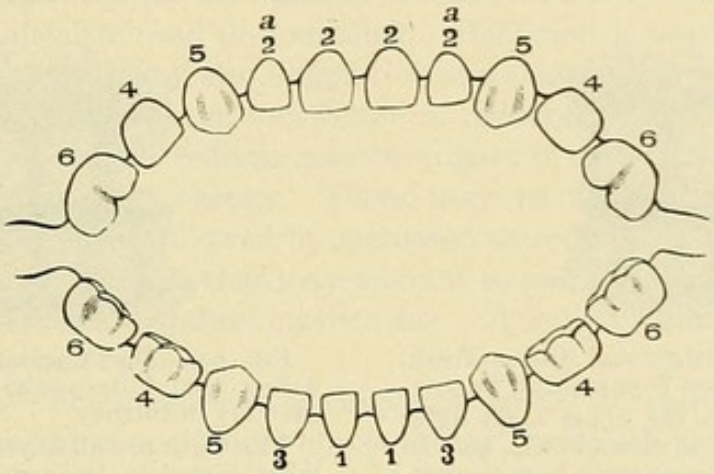


FIG 97.—Diagram Showing Eruption of Milk Teeth.

1, 1. Between the fourth and seventh month, followed by a pause of three to nine weeks. 2, 2, 2*a*, 2*a*. Between the eighth and tenth months; pause of six to twelve weeks. 3, 3, 4, 4, 4, 4. Between the twelfth and fifteenth months; pause until eighteenth month. 5, 5, 5, 5. Between the eighteenth and twenty-fourth months; pause of two to three months. 6, 6, 6, 6. Between the twentieth and thirtieth months—(*from Louis Starr, slightly modified*).

ing the two "eye," two "stomach" teeth; and finally the four posterior molars (6, 6, 6, 6); so that by the end of the second or beginning of the third year the first dentition is completed. The milk teeth begin to be replaced by the *permanent* set in the *fifth* or *sixth* year. Before any of the milk teeth are shed the first molars of the second set are fully developed. Hence they are called the six-year molars. About 12 years are consumed in the cutting of the remaining teeth, but the variations of the date of appearance of each tooth are so great that it is not worth while to attempt to name the dates. Some individuals never develop teeth.

In some children (usually the rachitic, the feeble, and badly nourished) the appearance of the milk teeth is greatly delayed—the lower incisors do not appear until the 11th or 12th month, but the completion of dentition is not much delayed thereby, though under these circumstances dentition is sometimes not completed until the end of the third year. In others they appear earlier—in the third or fourth month—and occasionally children are born with them. It has always seemed that the first appearance of the teeth is more apt to be delayed in blondes and anticipated in brunettes.

The diet of children during dentition should be very carefully watched, as the whole gactrointestinal tract is sensitive and irritable and readily

thrown into inflammation. The mouth is tender, the saliva flows freely, and the child is disposed to bite on anything and to thrust its little fists into its mouth. The term *tooth rash* is applied to certain eczematous eruptions that sometimes appear during teething. Their relation to teething is not established.

Very rarely a purulent conjunctivitis makes its appearance during the eruption of the upper canines or "eye teeth," which is ascribed to dentition and explained by contiguous extension of inflammation through the antrum of Highmore and the lachrymo-nasal duct.

Certain markings are often found on the teeth as a consequence of stomatitis. They include pittings and linear depressions, the result of defects in the development of the enamel. Extreme degrees produce a

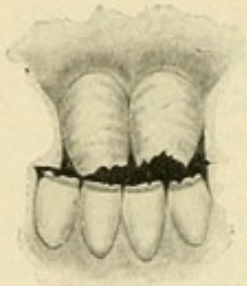


FIG. 98.—Thin-edged and Broken Teeth, not Syphilitic, from a Woman, aged Twenty.

The notches in the upper teeth differ markedly from those shown in Fig. 99. In these they result not so much from the softness and original malformation of the teeth as from their preternatural thinness and brittleness. Near the edges of the lower set a horizontal line of notches is seen to extend—(*after Hutchinson*).

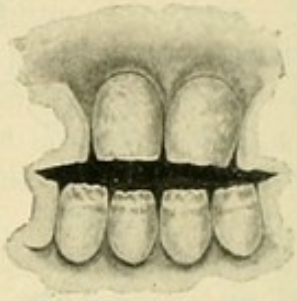


FIG. 99.—The Permanent Front Teeth of a Boy, aged Fifteen, who had Taken Much Mercury in Infancy.

The teeth are all of yellow color, somewhat pitted in their surfaces, and very thickly coated with tartar. Near the edges of the lower set a horizontal line extends similar to that in Fig. 98—(*after Hutchinson*).

honeycombed appearance. These, as well as the syphilitic teeth of children, have been studied by Jonathan Hutchinson, and are not to be confounded with the latter. (Figs. 98 and 99). the "honeycombed" changes are most conspicuous in the permanent teeth, of which the first molars, according to Hutchinson, are the test teeth, though he says the incisors are almost as constantly pitted, eroded, and discolored, often showing a transverse line which crosses all the teeth at the same level. These transverse furrows are also ascribed by Magitot to infantile convulsions or other severe illness in early life.

Care of the Mouth.—That severe toxic conditions may occur from neglected conditions in the mouth is certain. William Hunter was the first to insist upon this. Conditions closely resembling if not identical with acute rheumatic fever occur which are preceded and accompanied by severe septic conditions of the mouth. In our experience the clearing up of the mouth condition has caused an immediate disappearance of the joint symptoms. Pyorrhea alveolaris is constantly the cause of not only the acute joint troubles but of more chronic ones such as arthritis deformans. Severe forms of anemia have resulted caused apparently by neglected mouth conditions.

Fetor oris is frequently the result of carious teeth and pyorrhea. Disturbed digestion may constantly result from poor teeth or no teeth. It therefore

is of the highest importance that great attention be given to the mouth, both as a prophylactic and as a method of treatment.

STOMATITIS.

Simple Acute Catarrhal Stomatitis.

Definition and Etiology.—A simple erythematous inflammation of the mouth, commonly caused by diffuse chemical or mechanical irritants, such as overheated food (very hot drinks), acids, alkalies, stimulating condiments (red pepper, horse-radish, and the like), by excessive smoking and use of alcohol. It occurs in adults and children from the action of such causes, independently of the state of health, but is prolonged when its subjects are unhealthy and ill-nourished. Dentition is also a cause, while stomatitis may accompany also indigestion and the acute fevers.

Symptoms.—The mucous membrane is reddened wherever the irritation has reached, but the redness may be greater in certain situations, as on the tongue, gums, and cheeks. There may be at the very beginning dryness, but it is soon followed by increased secretion and slight swelling. There is always discomfort that may amount to pain, which is increased by the introduction of food and its mastication. A corresponding slight febrile movement may be present.

Treatment.—The treatment of simple catarrhal stomatitis will be considered in connection with that of the other forms of stomatitis to be described.

Herpetic or Aphthous Stomatitis.

Synonyms.—*Vesicular Stomatitis; Aphtha; Canker; Follicular Stomatitis.*

Description and Symptoms.—Some confusion attends the use of this term. The term "aphtha" from the Greek means "an eruption." Aphthous stomatitis is sometimes confounded with thrush, but it is not a parasitic disease, as in thrush, nor is it a follicular disease. Others speak of it as herpetic or vesicular. J. Emmett Holt adopts this name because he agrees with Forcheimer[1] in regarding it as of nervous (neuritic) origin.

The little grayish-white spots which characterize it consist primarily of an exudate of fibrin and wandered-out leukocytes, which pervades the superficial layer of the mucous membrane and is at first covered by epithelium. Hence, an attempt to remove the spots by forceps is futile and followed by bleeding. They are small, round, usually not more than a few millimeters in diameter, and surrounded by a red areola of hyperemia, occurring at times in successive crops. They are most common on the cheeks and lips, especially in the gingival groove at the base of the latter. They also occur on the tip and edges of the tongue, more rarely on the dorsum. The epithelium dies and desquamates, leaving a superficial ulcer, which under favorable circumstances heals up rapidly. Under more unfavorable conditions the ulcer grows deeper and becomes more painful, constituting one of the forms of ulcerative stomatitis. Young children are especially subject to it, but it is common also in adults, especially at times

[1] "Archives of Pediatrics, vol. ix.," p 330.

of temporary physical depression, as in women during menstruation, pregnancy, and lactation.

The aphthæ are commonly associated with a variable amount of simple stomatitis, with increased secretion of saliva, a slight "heaviness" of the breath, but without fetor. There is commonly a stinging sensation, especially when brought in contact with food, and even when the tongue and lips are moved in speaking. There is often some constitutional distrubance, including fever. Relapses may occur.

A similar condition is *Riga's disease*, in which a pearly-colored membrane with induration forms on the frenum of the tongue. It occurs in southern Italy in unhealthy and cachectic children about the time of eruption of the *temporary* teeth, and may be epidemic.

Thrush—Mycotic Stomatitis.

Synonyms.—*Parasitic Stomatitis; Soor; Miguet.*

Definition.—Thrush is characterized by grayish-white deposits in the buccal and pharyngeal mucous membranes, due to the development and interpenetration of the epithelium by a fungus variously known as *oidium albicans* or *saccharomyces albicans*. It is a variety of yeast fungus made up of branching filaments, at the ends of which oval cells develop. It does not grow on the normal mucous membrane. It forms minute white and yellowish spots scattered copiously over the palate, tongue, and cheeks, uniting at times to form large areas. It may extend into the esophagus and even larynx. In severe cases the entire buccal mucous membrane may be covered. Stenosis of the esophagus has resulted from its accumulation. The little areas are commonly surrounded by an inflammatory areola, and may be scraped off, though with some difficulty, leaving the mucous membrane sometimes intact and sometimes slightly excoriated and bleeding.

Thrush is chiefly a disease of nursing children, and is favored by feeble and dyscrasic states and by the want of cleanliness, especially in the care of nursing-bottles and nipples when children are brought up on the bottle. It may be associated with any of the diseases of children or may occur independent of them. It also occurs in adults after long illness or in dyscrasic diseases like diabetes mellitus and tuberculosis. Thrush is often unattended by other symptoms, though the mouth may be sensitive and nursing painful.

Diagnosis.—There should be no difficulty in diagnosis. In thrush the spots are smaller than in aphthous stomatitis, and the microscope at once removes all doubt. The mouth is dry as contrasted with the moist mouth of aphthous sore mouth, where there is free salivation. The secretions are commonly acid.

Koplik's sign in measles is not likely to be mistaken for thrush. It is described on p. 99.

Other Varieties of Stomatitis Due to Fungi.—The mouth is a favorite seat for the development of fungi, because of the warmth, moisture, and organic matters constantly present. Though ordinarily harmless, in certain states of the system they may play an important rôle in producing ulcerative stomatitis, as already suggested. Especially worthy of mention are the

diplococcus of Fränkel and the pneumonia bacillus of Friedländer; also the delicate, thread-like *leptothrix buccalis*, thought to exert a significant part in the production of caries.

Ulcerative Stomatitis.

Synonyms.—*Stomacace; Fetid Stomatitis; Putrid Sore Mouth.*

Definition.—This is a much more serious disease of the mucous membrane of the mouth, attended with necrosis and resulting ulceration.

Etiology.—Any one of the above-named diseases may become ulcerative. It may begin as an aphthous stomatitis, taking on the more serious form in the ill fed and badly cared for, or in those who are indifferent in the care of their mouths. It may begin as thrush. In other cases, an abrasion or laceration by the tooth-brush or a sharp carious tooth, may be the initial lesion. An ulcer may begin, too, in a herpetic vesicle, which, on rupturing, leaves a raw surface that may remain isolated or unite with others. It is a frequent attendant of mercurialization—*mercurial stomatitis*. The ulcer sometimes starts in the mucous follicles of the mouth. In all these cases the stomatitis is probably the result of infection by some organism; it may be the omnipresent streptococcus, staphylococcus or diplococcus, to which the sound mucous membrane in health is invulnerable, but which finds a nidus in the abrasions and conditions referred to.

Symptoms.—The ulcers may occur in any of the situations already named, the lips, cheeks, and, more rarely, the tongue. They vary in size, but are usually of an ashen-gray color, with red areolæ, and often exhibit a tendency to bleed.

Additional symptoms are profuse *secretion*, exquisite *pain* and *tenderness* in the ulcers and vicinity, a *fetid odor* of the breath, which sometimes pervades the apartment. The *gums* become spongy and, in extreme cases, the *teeth* are loosened. There are proportionate constitutional disturbances, *fever*, and often *swelling* of the glands at the angle of the jaw.

Mercurial stomatitis, or mercurial ptyalism. This condition is due to mercury administered as a medicine or absorbed in the course of occupations in which mercury is handled. Acquired in the former way, ptyalism, at the present day, is usually accidental rather than designed, in persons exhibiting a peculiar susceptibility. In such persons even fractional doses frequently repeated sometimes produce salivation in a day or two. The symptom first observed is usually *fetor of the breath*, unless the patient be closely watched during the administration of the drug, when tenderness may be ascertained on closing the jaws with some force. Examination will then discover a swelling of the gums about the teeth. A metallic taste may make its appearance as the first symptom. To these symptoms *salivation* is soon added, and becomes more or less profuse according to the severity of the poisoning. In severe cases, the entire mucous membrane of the mouth becomes swollen, as does also the tongue. In such cases, also, ulceration and loosening of the teeth take place. This form of stomatitis was not infrequent in the older treatment of syphilis, it used to fill a hospital ward with a sickening fetor at once recognizable. Actual loss of teeth was,

perhaps, less common than is supposed even in those days, yet necrosis of the jaw has, in rare instances, resulted.

Parrot's ulceration is a form of ulceration occurring in *new-born children*, consisting of small, symmetrically placed ulcers on the hard palate on both sides of the median line. *Bednar's aphthæ*, two symmetrically placed ulcers, also occurring on the hard palate on either side of the medial line near the velum, are similar, though not regarded as identical. This variety is thought to be traumatic in origin, at least in most cases, either the result of pressure of an artificial nipple against the hard palate, or of undue pressure in washing the mouth. Both are described as usually harmless, but in poorly cared for children may be converted into extensive and deep ulcers. Especially is this the case in the form described by Parrot, which may invade the adjacent bone.

Diagnosis.—This is made by inspection of the mouth and the history of the case. *Scurvy*, though a general disease, happily rare of late, is characterized by local symptoms about the mouth, which include ulceration. There are swelling and bleeding of the gums, which rise up around the teeth. The latter become loosened and ulceration may extend even to the lips and cheeks. The tongue and fauces are not invaded by ulcers, but are subject to ecchymoses. Salivation and fetor of the breath are also symptoms, though less decided than in severe ulcerative stomatitis. On the other hand, in extreme cases deep-seated gangrenous processes are met. Along with these are, however, the general symptoms of scurvy, by which it is commonly easily recognized.

Treatment of Different Forms of Stomatitis.—*Prophylaxis* is exceedingly important in averting these various mouth affections. In the case of infants the mouth should be washed out with antiseptics after each nursing. Nothing is better than a saturated solution of chlorate of potash, boric acid, or bicarbonate of sodium, 15 grains to the ounce (1 gm. to 30 c.c.) of water. So, too, the adult should cleanse the teeth after each meal. Liquor Antisepticus, U. S. P., diluted with twice as much water, is an elegant and efficient wash.

Any of these substances may be used on the tooth-brush as a simple mouth-wash. The tincture of myrrh, a teaspoonful to 4 ounces of water, should not be forgotten, and, though less agreeable, carbolic acid may be used in the same proportion. Permanganate of potassium in the shape of Condy's fluid, a teaspoonful to a tumbler of water, is an excellent wash.

If stomatitis is established, cleanliness is no less important and may be secured by the same antiseptic measures. In addition, the mouths of children may be treated with glycerin and borax. Alum itself is an admirable astringent, too much overlooked of late. A moderately strong solution may be made, 30 grains (2 gm.) to the ounce (30 c.c.), or the powdered alum itself may be applied to the aphthous sore mouth.

For the painful aphthous ulcers of adults and children there is really nothing so efficient as touching with a pointed piece of nitrate of silver. A single application will often suffice, but when healing does not follow, it may be made daily. A very good application also is a solution of equal parts of tincture of the chlorid of iron and glycerin, applied to the ulcers with a brush. Chlorate of potassium in saturated solution is also a very

good mouth-wash, to 8 ounces (240 c.c.) of which 1/2 a fluidram to 1 dram (2 c.c. to 4 c.c.) of tincture of the chlorid of iron may be added.

In the ulcerative stomatitis chlorate of potassium internally in 3-grain doses acts almost as a specific.

Many such cases have an acute or chronic gastritis, as manifested by the symptoms peculiar to that condition, therefore the diet should be carefully regulated. The bowels must be moved each day. Bismuth is, as a rule, an excellent routine remedy. Calomel in divided doses may be used.

General treatment should not be overlooked. Many persons who have stomatitis are much run down, and require iron, quinin, and strychnin, with nutritious food, to build them up. Attention should also be paid to the bowels.

In mercurial stomatitis the use of mercury must at once be stopped. Astringents and disinfectants are especially indicated.

Noma.

Synonyms.—*Gangrenous Stomatitis; Water Cancer; Cancrum Oris.*

Definition.—A rare form of stomatitis, characterized by hard infiltration of the cheek near the angle of the mouth, succeeded by rapid gangrene proceeding outward and inward from the central focus until the cheek is perforated, and the gangrenous mass separates. It may start in the gums and produce necrosis of the jaws. It is confined to one side of the face.

Etiology.—A parasitic origin seems likely, but the specific organism has not been discovered. It occurs in children from two to five years old, affecting more of the former than of the latter. Rarely it affects adults. It is usually confined to those badly fed and surrounded by unsanitary conditions, especially when convalescent from infectious fevers, one-half of all cases having arisen during convalescence from measles, scarlet and typhoid fevers. It may, however, be primary.

Symptoms.—Its approach is insidious, and it is generally well advanced when discovered. It begins with an ulceration of the mucous membrane of the cheek, rapidly infiltrating the underlying tissues, and in a few hours or days causing gangrene. In its extreme severity it may involve the bones of both jaws, the eyelids, and ears; but in its mildest form its results are limited to perforation of the cheek. The dead tissue comes away in dark, offensive shreds.

The constitutional disturbance corresponds to the degree of local involvement, there being high *fever*, reaching often 104° F. (40° C.), with frequent pulse and rapid exhaustion. The adjacent lymphatics are swollen. Inhalation-pneumonia of corresponding virulence often succeeds, while intense irritation of the stomach and bowels follows the swallowing of the ichorous discharge.

Diagnosis.—Noma is rarely mistaken for anything else. *Malignant pustule* is less local in its invasion, furnishes the history of contagion, is even more severe in its constitutional effects, and exhibits the appropriate bacillus. Very bad cases of *ulcerative stomatitis* sometimes suggest cancrum oris, but the devastation is not so rapid, nor is there such a tendency to invasion of the external integument.

Prognosis.—The cases frequently end fatally at the end of three or four days, only the promptest and most energetic treatment saving life.

Treatment.—This consists in the prompt use of surgical means. Time should not be wasted with cauterizing agents. Excision offers the best chance for cure of the patient and limitation of the deformity. The general condition of the patient must be attended to. Every case of measles of severity or in weak ill-nourished individuals should have the cheeks regularly examined, in order to forestall serious results.

Glossitis.

Parenchymatous glossitis, or inflammation of the substance of the tongue is a rare disease, but it occurs as the result of violent injury to the organ, as by accidental biting or poisonous stings. Apparently idiopathic inflammations are probably the result of concealed causes of the kind described.

Symptoms.—The tongue is enormously *swollen* and *painful*, and sometimes extruded from the mouth. There is great difficulty in speech, mastication and deglutition, and in extreme degrees these are scarcely possible. The discomfort is almost indescribable, and there may even be *obstruction to breathing*. If exposed, the tongue becomes dry and fissured. There may be suppuration. There is *fever* corresponding to the amount of local disturbance.

Treatment.—This consists in the constant application of ice, of frequent antiseptic cleansing of the mouth, and sometimes of scarification. Evidence of the presence of pus must be followed by the prompt use of the lancet.

Glossitis desiccans is a more chronic affection of the tongue, characterized by deep fissures and indentations, giving it an uneven, ragged appearance. Associated therewith are excoriations and occasionally superficial ulcers. Severe pain is caused by contact of acids and even the usual food. Its etiology is not known, but it is sometimes associated with gastrointestinal derangements.

Treatment.—This should be directed to the cause, if it can be discovered. Washes of chlorate of potash should be employed, and if there are ulcers, they should be touched with solid silver nitrate.

Epithelial Desquamation; Geographical Tongue.

Synonyms.—*Eczema of the Tongue.*

Definitions and Symptoms.—A localized superficial hyperplasia and desquamation of the epithelium of the tongue, sometimes associated with similar spots on the cheeks and lips. The central parts tend to heal, while the periphery spreads, producing circinate patches. The patches fuse and extensive areas are formed, bounded with sinuous outlines. The appearance has been compared to that of a map—*lingua geographica*. The condition is chronic, sometimes lasts years, but does not usually cause inconvenience save by the itching and burning it occasions and the apprehension of more serious disease. It is occasionally mistaken for syphilitic disease.

Treatment.—It is best treated by solutions of nitrate of silver, which relieves the itching. Weak solutions of iodin may be used, applied with a brush.

Leukoplakia Buccalis.

Synonyms.—*Ichthyosis lingualis; Buccal Psoriasis; Keratosis mucosœoris; Smoker's Patches.*

Definition.—A condition in which there are intense white spots on the mucous membrane of the mouth and tongue, consisting of thickened epidermis.

Etiology.—Some cases of leukoplakia are syphilitic in origin. Some are the result of excessive smoking. Others appear to be the result of irritation of a badly fitting dental plate.

Symptoms.—The patient is first made aware of the condition which appears as white shiny spots on various parts of the mucous membrane of the cheeks and tongue, they may be few or many. Occasionally the whole mucous membrane may be involved.

The spots on the sides of the tongue are often notched, giving them a scar-like appearance, Those on the inner surface of the check are simply flat tabular swellings. They disappear, to be replaced by others; they rarely give rise to inconvenience. Sometimes those on the sides of the tongue become ulcerated, when they are painful if brought into contact with irritants. They occur in adults of both sexes. They sometimes become papillomatous, and are said to have been the starting-point of true epithelioma, as often as once in every three cases.

Treatment.—Before treatment is begun or it is decided to give no treatment a Wassermann reaction should be made to discover if syphilis is the underlying cause. If the patient is the subject of syphilis he should be put upon proper treatment. If syphilis is not present, all sources of irritations such as smoking, ill fitting dental plates, etc., should be corrected. This being done no further interference is necessary unless the spots begin to undergo degeneration. If this occurs they must be at once excised.

Mucous Patches.

The true mucous patches or flat condylomata of syphilis are opaque, white, flat, tabular swellings on the lips, tonsils, tongue, and arches of the palate, and especially at the border-line between skin and mucous membrane. They consist of an irregular imbricated thickening of the superficial layers of the skin; the cells are swollen and the papillæ of the mucous corium hypertrophied.

Treatment.—The treatment of the mucous patches of syphilis is that of syphilis constitutionally, and locally by applications of nitrate of silver.

DISEASES OF THE SALIVARY GLANDS.

FUNCTIONAL DERANGEMENTS.

Ptyalism, or *excessive secretion* of saliva, is a symptom of mercurial poisoning, also of poisoning by gold, copper, and iodin. Some persons are very susceptible to iodin, so that a few grains of iodid of potassium will cause intense salivation, with pain in the salivary glands. Vegetable substances producing the same effect are jaborandi, muscarin, tobacco.

Indeed, almost anything which admits of constant chewing without solution or destruction produces salivation. This is the mechanism of the various agents used in the disgusting practice of chewing gum.

Xerostomia, or *dry mouth*, is the opposite condition of arrest of salivary and buccal secretion, not due to fever—a rather common condition, first described by Jonathan Hutchinson. As a consequence the tongue and mucous membrane are red, dry, and shining. It is more common in women, in whom it follows intense emotion, such as fright, or is associated with hysteria and hypochondriasis. It is probably a neurosis, the result of some cause operating on the center which controls the secretion of salivary and that of other buccal glands.

Treatment.—The treatment of ptyalism and xerostomia is that of the conditions producing them. Xerostomia may be sometimes relieved by the use of pilocarpin.

INFLAMMATION OF THE SALIVARY GLANDS.

Acute Parotitis, or Parotid Bubo.—Apart from mumps, or specific parotitis, considered under infectious diseases, in which any or all of the salivary glands may be involved, the *parotid* is subject to inflammation from the following causes:

1. In the course of infectious diseases, especially typhoid fever, but also scarlet fever, typhus fever, pneumonia, pyemia, and secondary syphilis and dysentery.

2. In connection with diseases or injury of organs in the abdomen or pelvis, including the alimentary canal, urinary tract, abdominal wall, peritoneum, pelvic cellular tissue, or genital organs—a very interesting group of cases, which have been especially studied by Stephen Paget. Sometimes simple transient irritation, such as a blow on the testis or the introduction of a pessary, may produce it.

3. In association with facial neuritis. A fatal case, apparently of such origin, has been reported by Gowers.

In (1) and (2) septic infection is doubtless the cause of the inflammation, which is often intense, going on to suppuration in more than one-half of the cases. Its possible origin through the duct of Steno was considered in treating typhoid fever. In (3) there is probably some vasomotor disturbance which is responsible.

Treatment.—This should consist, at first, in attempts to allay the inflammation by leeches and the application of cold, especially ice. Failing in this, fomentations should be applied, while the lancet should be used at the first indication of suppuration.

Chronic Parotitis sometimes succeeds on acute inflammation, as that of mumps; also on mercurialization or lead poisoning, syphilis, and Bright's disease. Sometimes no cause is discoverable. It may be painful or tender or painless. It may be treated by ointments reputed to promote absorption —ointments of iodin and mercury.

Mikulicz's Disease.—A chronic enlargement of the salivary, lacrymal and buccal glands without discoverable cause. It was first described in 1892 by Mikulicz. It may last many years, but has been known to disappear at least in part.

LUDWIG'S ANGINA.

SYNONYMS.—*Angina Ludovici; Cellulitis of the Neck; Cynanche Gangrænosa.*

Definition and Symptoms.—An infectious inflammation, beginning in the submaxillary gland as a secondary inflammation in the specific fevers, including, especially, typhoid, diphtheria, and scarlet fever, but it may also be primary. It may succeed on a carious tooth. It is probably a streptococcus infection. It spreads rapidly over the floor of the mouth and anterior surface of the throat, sometimes invading the glottis by edema, and sometimes terminating in sloughing of the soft parts—*cynanche gangrænosa*. Or it may go on to abscess, pointing externally or internally. More rarely, resolution takes place.

Further symptoms are *swelling* and extreme *pain*, first in the neighborhood of the submaxillary gland, increased by chewing, swallowing, and talking. The swelling may produce compression of the larynx, with resulting dyspnea, which is suffocative if the glottis becomes involved. Constitutional infection may take place, with its grave array of symptoms and fatal termination. There may be remissions and exacerbations.

Treatment.—This should consist in energetic measures calculated to combat the inflammation, such as the use of ice. If the condition tends to spread immediate dissection of the inflamed area by a competent surgeon should be done. A mere skin opening is worse than useless.

DISEASES OF THE TONSILS AND PHARYNX.

SUPPURATIVE TONSILLITIS.

SYNONYMS.—*Acute Parenchymatous Tonsillitis; Phlegmonous Tonsillitis; Tonsillar Abscess; Cynanche Tonsillaris; Quinsy.*

Definition.—An acute inflammation of the substance of the tonsil and of the peritonsillar tissue.

Etiology.—Quinsy is a disease of later youth and adults, being rarely found in children under ten years of age, and not often in adults over 40. Some persons are much disposed to suppurative tonsillitis, scarcely a season passing for them without an attack, and sometimes more than one attack. In such, almost every cold terminates in quinsy. Others, after a single attack, never have another, and others still are entirely exempt. Suppurative tonsillitis is always the result of infection. Exposure to wet and cold certainly often precedes it. Persons predisposed to tonsillitis are often the subject of chronically enlarged tonsils. Overdistention of the follicles with inspissated secretion may also be a cause of inflammation and suppuration.

Morbid Anatomy.—The tonsil, more frequently on one side, sometimes on both, or on two sides in succession, becomes rapidly enlarged, red, and painful. Sometimes the case begins as one of follicular tonsillitis. It is at first hard and resisting and very tender to the touch, but, if suppuration takes place, it gradually softens until rupture happens or the abscess is

opened by the knife. The lymphoid parenchyma of the gland becomes more and more distended with leukocytes until the entire gland, or a large part of it, is converted into a *pus* sac. Very frequently the peritonsillar tissue is the part involved in the suppurative process, the parenchyma of the tonsil being little affected. When both tonsils are involved, the throat is often almost closed by the swelling.

Symptoms.—The symptoms are *pain* and *difficulty of deglutition*, the latter causing pain, often agonizing. The jaws are stiff and the mouth cannot be opened above half an inch without extreme suffering. The difficulty in opening the mouth is increased by the swelling of the external glands of the neck. The pain is not confined to the interior, but extends to the neighborhood of the angle of the jaw, the front of the ear, and the floor of the mouth. The voice is greatly altered, having the characteristic nasal drawl, and the diagnosis can sometimes be made from the altered speech alone. There is increased salivation, and the saliva dribbles from the mouth because of the pain in swallowing it, while it also often becomes fetid. Respiration may be seriously interfered with.

There is high *fever*, the temperature reaching 104° and 105° F. (40° to 40.5° C.), while the *pulse* is full, bounding, and frequent, 110 to 130 a minute. The *face is anxious* and tells the tale of suffering. From two to six days are occupied in the completion of the process, at the end of which time the abscess begins to point, usually just above the last molar on the side affected, when relief is obtained by spontaneous rupture. But more fortunate is the patient who is relieved early by the lancet. Sometimes the abscess points toward the pharynx. Suffocation has resulted from the discharge of a quinsy passing into the larynx.

Prognosis.—Apart from the rare accident just referred to, the prognosis is favorable, though it must be mentioned also that death from suffocation has occurred where the obstruction by double-sided quinsy was so great as to prevent respiration. This of course should never be allowed to occur.

Treatment.—Free scarification is sometimes useful in shortening an attack, but it is painful, unreliable, and sometimes difficult to do thoroughly. If deferred until about the third day, it will often cooperate with the advancing suppuration and favor an early rupture. Other applications to the tonsils are of a doubtful efficacy, though some relief from pain may be secured by painting the surface with a ten per cent. solution of cocain. Painting with a 40 grain (2.6 gm.) solution of nitrate of silver, after thorough cleansing with a cotton swab, is recommended.

Cold, so soothing in other forms of sore throat, often occasions more discomfort than relief, though in certain cases it is grateful. Sucking of ice is also sometimes valuable. Then poultices and fomentations to the exterior of the throat are apt to be more soothing. And since little can be done to prevent suppuration, these measures are indicated to hasten it. The tonsil should be frequently felt with the finger, and as soon as there is evidence of suppuration, the lancet should be used. A curved bistoury, guarded with adhesive plaster almost to the end, is the best. It must be inserted into the area of pointing, very frequently into the soft palate above the last molar. The incision should be made from above downward,

parallel to the anterior half-arch. If danger of suffocation is imminent, the tonsil must be shaved off.

CHRONIC TONSILLITIS AND HYPERTROPHY OF THE ADENOID TISSUE OF THE PHARYNX.

SYNONYMS.—*Chronic Enlargement of the Tonsils; Chronic Nasopharyngeal Obstruction; Mouth Breathing; Aprosexia.*

Definition.—A chronic inflammatory enlargement of the tonsils or of the adenoid tissue of the pharynx, of the lingual tonsil, or of two or more of these structures.

Etiology.—The most frequent cause is repeated attacks of acute tonsillitis and of inflammatory processes associated with hyperemia of the tonsils and vicinity, including scarlet fever and diphtheria. It is more common in children, in whom it is also sometimes congenital, but it is found usually at the ages of five to fifteen years, and rather more frequently in boys. Adenoid overgrowths of the pharynx and lingual tonsil are due to the same causes.

Morbid Anatomy.—The enlargement of the tonsils is a true lymphoid overgrowth, usually symmetrical. The occasional presence of fibrous stroma produces a harder and smoother tissue. The lumen of the throat is variously encroached upon, sometimes almost closed. The pharyngeal adenoid overgrowths vary in extent from a slight increase in natural unevenness to the formation of actual sessile and pedunculated tumors. The same is true of the tonsillar structures at the base of the tongue, which may encroach upon the glottis.

Symptoms.—Simple chronic enlargement of the tonsils may give rise to no symptoms except when they are the seat of further enlargement due to acute inflammation. Then *obstructed breathing* is immediate, while it is also a permanent symptom in the more advanced forms. It is proportionally contributed to by overgrowth in any of the situations named. The result is *mouth breathing*, which is, perhaps, earlier necessitated by pharyngeal than tonsillar overgrowth, while it may be due altogether to the former, the latter being entirely absent. Tonsillar obstruction is, however, more frequent. The effects are usually first apparent at night, when the child is found to be breathing, more or less noisily, with its mouth open and head thrown back. *Disturbed rest* is an inevitable consequence, the patient often waking up with a start, again relapsing into sleep, or continuing permanently aroused because of the dyspnea, which often only gradually passes away.

As the conditions persist a *changed expression of countenance* is gradually acquired. The face becomes apathetic, staring, and vacant, an appearance chiefly produced by the constantly open mouth. To this may succeed actual mental failure and even stupidity, with sullenness and general bad temper. Further changes in expression are occasioned by contraction of the nostrils and projection of the upper jaw and lip. If the condition is still unrelieved, *deformities of the chest* make their appearance, of which the most conspicuous is the well-known *chicken breast*. In it the upper sternum projects, the manubrio-gladiolar articulation being most prominent, while the lower part is depressed, causing a groove at the gladiolo-xiphoid articu-

lation. There is a cup-like depression of the lower costal cartilages and a horizontal circular depression (Harrison's groove) in the thorax corresponding to the attachment of the diaphragm. The ribs are separated from each other anteriorly and closely approximate posteriorly, especially in the lower thorax. Posteriorly the lower angle of the scapula projects. This is the result of the act of breathing, a study of which during sleep will recognize the retraction of the lower part of the throax during inspiration, caused by the action of the diaphragm.

Another form of chest is the *round or barrel chest*, such as is commonly associated with chronic asthma, due to the same cause. Still another is the *funnel*, or *Trichterbrust* of the Germans, in which there is a deep central depression at the epigastrium and of which the periphery may extend upward as far as the third rib.

Other symptoms are an *altered voice*, nasal in character, in which the letters *m* and *n* are especially badly articulated, the *special senses* of smell, taste, and hearing are deranged, the breath is fetid from decaying secretion, the appetite is impaired, and with it the nutrition of the body. A gradual mental as well as physical deterioration may take place.

Among the symptoms ascribed to this condition are *habit chorea* and *stuttering*. The former will be considered in a later section. There is an almost *constant cough*, which is well termed "throat cough," since it is due to irritation of the respiratory passages by the throat outgrowths and the secretion caused by them. This secretion is generally swallowed by children, but is in part expectorated by adults by the aid of troublesome hawking and coughing, which is stimulated by a sensation as of "something in the throat" or larynx which demands clearing. The absence of discharge from the nose in both children and adults is surprisingly frequent, sometimes misleading the physician as to the true cause.

Defective hearing is another symptom due to obstruction of the Eustachian tube by encroachment of the adenoid growths, or by inflammations, or to retraction of the drum. *Impaired taste* and *smell* are due to involvement of the gustatory papillæ and the terminal distribution of the olfactory nerve. *Extreme fetor* of the breath is sometimes present, due to retention of cheesy masses in the crypts of the tonsils. These are often easily visible, are sometimes expectorated, and can usually be expressed. The odor of these masses when compressed between the fingers is indescribably disagreeable. Sometimes they are found in the tonsils of persons not otherwise affected. The very great susceptibility of the subjects of this disease to "cold" is constantly adding aggravation to the symptoms described.

Diagnosis.—This is not usually delayed at the present day, since the more thorough examination of the throat and nose has become common—thanks to the throat and nose specialists. Most important is it to remember that there may be no tonsillar disease, and all the symptoms may be due to advanced adenoid growths of the pharynx. Digital examination affords the most ready and accurate means of diagnosis. Especially thorough must be the examination behind the pillars of the fauces. In children this can only be done with the finger, but in adults the half-arches may be drawn forward, while the laryngeal mirror is availed of.

The "chicken breast" of mouth breathing in childhood is different from

the "violin" shaped chest of the rickety child. In the latter there is a *prominence* of the *whole sternum* and a vertical flattening of the sides of the thorax, leaving a large curve behind the costo-chondral articulation and a similar one in front, in addition to the horizontal depression of the lower thorax which is common to both kinds of deformity.

Prognosis.—This depends upon the early discovery of the condition, before the secondary effects have established themselves. If the trouble is purely a tonsillar one, it is comparatively easily removed by shaving off the organ. If the overgrowth is pharyngeal, little can be done until children are old enough to submit to the proper treatment. This may be done by the aid of ether as early as the first year. Hypertrophied tonsils begin to atrophy of themselves after puberty, and they have generally disappeared by 30. The face and chest deformity may be outgrown if the cause be removed.

Treatment.—Most important are local measures whose purpose is to reduce the overgrowth or to remove it and to prevent recurrence of acute attacks. The patient should be discouraged from hawking and clearing his throat. If the tonsils manifestly encroach on the faucial lumen, they should be removed by carefully dissecting out the entire tonsil. The same treatment is demanded by the pharyngeal adenoid growths. There is sometimes ment is demanded by the pharyngeal adenoid growths. There is sometimes copious hemorrhage, but it is usually easily controlled. It is certain that one or all of the symptoms above described may result from enlarged tonsils or adenoids. It is equally certain that many enlarged tonsils are unnecessarily removed. The rule to follow should be to remove all tonsils or adenoids which *give symptoms* because of their size or because of their septic nature. If they are not giving symptoms and are not the seat of inflammatory foci they should not be removed. If not requiring this operative treatment, they should receive on alternate days or every third day applications (1) of powdered alum; (2) solution of iodin of the strength of iodin 8 grains (0.5 gm.), iodid of potassium 24 grains (1.5 gm.), glycerin 1/2 ounce (15 c.c.); (3) of tincture of the chlorid of iron and glycerin equal parts; (4) glycerol of tannin or (5) silver nitrate 1 to 20. The solid stick of the latter may be used if there be evident lacunar disease, but far better is electrolysis, by which the crypt is obliterated and the gland may be gradually destroyed. Spraying the nose with antiseptic solutions twice daily is helpful in maintaining cleanliness and purity of breath. Dobell's solution may be thus used; also dilute liquor antisepticus. Tablets containing various proportions of the ingredients therein named are made for solution in the little cup of the spraying apparatus. Great patience and perseverance are required, for the result is but slowly attained.

The general health of the patient should be carefully looked after. Suitable woolen underclothing should be worn, and it should be graduated to temperature and exposure. Cod-liver oil, iron, quinin, and strychnin are the best. It is most important that every effort should be made in the direction of so hardening the patient that he may be able to resist the effects of exposure, a task not easy to accomplish. Cold bathing of the neck and throat, indeed of the whole body is useful, while nourishing food, physical exercise, and outdoor life, with suitable clothing, are means to this end.

SIMPLE CIRCULATORY DERANGEMENTS OF THE PHARYNX.

Hyperemia of the pharynx is a very common condition in smokers. It is also almost always present when there is chronic nasal catarrh. Under these circumstances the mucous membrane is constantly red, angry looking, often streaked with mucous, and is very easily thrown into a state of active inflammation.

In such obstructions to the circulation as are caused by mitral valvular disease, cirrhosis of the liver, or pressure upon the ascending vena cava by aneurysm or tumor, there is venous stasis and the venules may often be seen distended. According to most authors these venules occasionally burst, producing small hemorrhages which stain the mucous secretion and under certain circumstances the bleeding may be mistaken for a pulmonary hemorrhage. Unfortunately the opposite is the rule, pulmonary hemorrhage is constantly diagnosed bleeding from the throat. This latter, in the belief of the writers, is dangerous teaching. It is certainly very rarely the fact that bleeding from the mouth should be thought pharyngeal in origin, never without examination. Almost without exception, any hemorrhage which comes from the mouth other than that which comes from epitaxis, bleeding in purpura, or the mere shreds of blood mixed with mucous, or evident hematemesis, or from the gums, is pulmonary in origin. The same causes may produce edema of the mucous membrane of the pharynx, and especially does this occur in Bright's disease. The edema may extend thence to the uvula, which becomes greatly swollen. In aortic regurgitation the capillary pulse may be seen in the pharynx, and the internal carotid may also be seen to throb strongly.

ACUTE CATARRHAL PHARYNGITIS.

SYNONYMS.—*Sore Throat; Simple Angina.*

Definition.—An acute inflammation of the mucous membrane covering the pharynx and tonsils, sometimes extending upon the palate.

Etiology.—Acute pharyngitis occurs at all ages; a trifling cause lights up an inflammation. Rheumatism and gout are also frequent causes. Pharyngitis and tonsillitis are often associated. Most of the attacks are unquestionably due to infection. The specific organism is not known.

Symptoms.—The first symptom is usually *pain* on swallowing, which is associated at first with a *dryness* and *soreness*, producing a desire to "clear the throat." To this is soon added a *full feeling*, and then pain independent of swallowing. The inflammation may extend into the Eustachian tube, producing partial *deafness*, or into the larynx, producing *hoarseness*. There is a varying degree of constitutional disturbance, and sometimes the *fever* is quite high.

On examining the throat it will be found red and congested, sometimes plainly swollen, especially over the tonsils. There is often considerable mucous secretion. The various forms of ulcer of the tonsils described under tonsillitis may be associated with the pharyngitis, increasing the constitutional disturbance and local discomfort.

Treatment.—Many simple sore throats pass away without treatment.

Astringent washes and gargles are indicated. Even the mild cases had better go to bed. Twenty-four hours in bed is by far the best medicine for an ordinary cold. A spray or a gargle of liquor antisepticus or normal salt solution. Solution of nitrate of silver, 20 grains (1.3 gm.) to the ounce (30 c.c.), may be similarly applied, also the glycerol of tannin.

In severe cases cold cloths wrung out in ice water and applied to the outside of the throat, the clothing being protected by the interposition of a dry towel, make an excellent measure; or the little ice bags referred to in the treatment of acute tonsillitis may be applied to the throat, with a dry towel outside of them. Occasionally counterirritation by mustard is more satisfactory, as every throat does not bear cold equally well.

The fever should be met in the usual way by aconite, sweet spirit of niter, citrate of potash, phenacetin or acetanilid while chlorate of potash and chlorid of iron should also be administered internally. The bichlorid of mercury may be added under the same circumstances as in tonsillitis. There is no advantage in giving large doses of iron. They are not absorbed and the excess remaining in the alimentary canal, locks up the secretions and causes irritation. From 2 to 10 minims (0.12 to 0.6 gm.) every two hours are quite sufficient. The bowels should be kept open, and the treatment may be advantageously commenced with a saline aperient, such as calcined magnesia, the solution of the citrate of magnesium, Hunyadi or any natural aperient water.

Where the disease is traceable to rheumatism or gout, suitable treatment for these diseases should be instituted. The salicylates are the best remedies for both, but guaiacum has some reputation, the tincture or ammoniated tincture being the best preparation, given in doses of 5 to 60 drops (0.35 to 4 gm.).

CHRONIC CATARRHAL PHARYNGITIS.

SYNONYMS.—*Clergyman's Sore Throat; Granular Pharyngitis; Chronic Angina; Chronic Follicular Pharyngitis.*

Definition.—Chronic pharyngitis, when not associated with ulceration, presents much the same appearance as chronic hyperemia, plus the addition of a granular appearance due to enlargement of lymphatic glandules, with which the pharynx is studded.

Etiology.—The disease is rather one of adults than children. Its causes are repeated attacks of acute pharyngitis and excessive smoking and alcohol drinking. Chronic nasal catarrh with its irritating discharges trickling down the fauces is a frequent cause, as is also nasal obstruction and disease of the third or Luschka's tonsil. It also occurs in those who use their voices largely, as hucksters, public speakers, and singers, while the inhalation of dust and irritating gases is also held responsible.

Treatment.—This is very much more unsatisfactory than in the acute type. It is most important to treat the causes or remove them. Postnasal catarrh is responsible for so many cases that the postnasal region should at once be investigated and its diseases treated. Smoking and the use of alcohol, if responsible, should at once be discontinued. The same local

measures useful in the acute disease may be employed in the chronic, but they are less promising as to results. The little granules, which are apparently a source of irritation as well as a result, can be removed only by the galvanocautery needle. Other measures to this end are unsatisfactory and insufficient. The general health of the patient should be carefully looked after, and occupations tending to keep up the irritation should be discontinued.

Ulceration of the Pharnyx.

The ordinary form of chronic pharyngitis rarely produces ulceration. Syphilis, tuberculosis, diphtheria, inflammation, and lowered nutrition, such as is found after the infectious diseases, like typhoid fever and scarlet fever, are frequent causes of sluggish ulcers indisposed to heal. The chief symptom of these various varieties of ulceration is pain, increased during deglutition, with more or less copious mucous secretion, which often adheres firmly to the pharynx. Frequently the small glands which dot the pharynx may be the seat of a follicular inflammation of the same character as that in follicular tonsillitis.

Diagnosis.—It is not always easy to distinguish the different forms of ulceration. The *syphilitic ulcer* is least painful, in fact often painless, and is commonly situated in the posterior wall of the pharynx. It occurs both as a secondary and tertiary symptom. As a secondary symptom it is superficial and associated with mucous patches, while as a tertiary it forms the cavity left by a softened, gummy tumor, and is correspondingly deep. It is associated with the history of syphilis.

The *tubercular ulcer* is more painful—indeed, the most painful of all. It is irregular, not very deep, has a grayish base, and is also seated in the posterior wall of the pharynx, considerable areas of which may be involved, producing an uneven, worm-eaten appearance. It is associated with tuberculosis elsewhere. The *indolent ulcers* of lowered nutrition are also often insidious and occasion few active symptoms. After the separation of the membrane in diphtheritic pharyngitis there are sometimes left ulcers more or less extensive, which are slow to heal.

Treatment.—This consists locally in the application of stimuli and antiseptics, the former represented by nitrate of silver and the latter by thymol and its class, together with general treatment appropriate to the condition, such as tonics of which iron and quinin are the types. Antisyphilitic treatment and treatment of the tuberculosis which always exists elsewhere,

Phlegmonous Pharyngitis.

Definition.—This term is applied to any suppurating inflammation involving the pharynx, however induced, except postpharyngeal abscess, which is a separate condition. It may be a part of the process which constitutes suppurating tonsillitis or quinsy, extending to the adjacent pharyngeal structures. It may include the acute infectious phlegmon of the pharynx described by Senator, in which, along with swelling of the external neck, the pharyngeal mucous membrane is swollen and injected, and becomes

rapidly the seat of suppuration. It may include similar conditions induced by injury, the inhalation of scalding liquids, or the swallowing of corrosive poisons. Or it may be the result of pharyngeal erysipelas or of the lodgment of foreign bodies.

Symptoms.—These are correspondingly intense. There is *painful swelling*, interfering not only with deglutition, but also with respiration. There is high *fever* and rapid *exhaustion*. It may terminate in *gangrene* of the part or gangrenous pharyngitis.

Treatment.—The treatment is locally antiphlogistic, including cold by ice or otherwise, scarification and liberation of pus at the earliest possible moment, together with restorative and stimulating internal measures. If gangrene results, cauterization and antiseptic applications must be added. The aid of the surgeon should be early sought.

Postpharyngeal Abscess.

Definition.—A phlegmonous inflammation behind the proper pharyngeal tissue, subperiosteal in some instances, arising in suppurative inflammation of the postpharyngeal lymphatic glands or caries of the cervical vertebræ. It is a disease of children and adults, more frequently of the former, often a sequel of one of the pharyngeal conditions already considered, favored by bad hygiene and depraved constitutional states, hereditary or acquired.

Symptoms.—Its symptoms are intense *pain*, *swelling*, and interference with deglutition and respiration, with more or less early appearance of a *tumor* in the posterior wall of the pharynx, which can generally be recognized by the finger before it can be seen—a fact which emphasizes the importance of frequent examination of the throat by the finger in diseases of these parts. There is also *stiffness of the neck*, sometimes *nasal voice* or even hoarseness, suggesting croup and edema of the glottis, but there is never absolute loss of the voice, as in the latter, while croup and edema are not associated with painful deglutition.

Treatment.—This consists of incision of the abscess as soon as discovered. It should be made in the median line and the head should be brought forward to avoid the entrance of pus into the larynx. Anodynes are necessary to overcome the intense pain, but it is to be remembered that they may so mask the symptoms as to permit destructive inroads of the disease before it is discovered.

DISEASES OF THE ESOPHAGUS.

EXPLORATION OF THE ESOPHAGUS.

This is a manipulation so frequently necessary that its description is demanded at the outset.

The esophageal bougie is made of flexible whalebone or steel, on the end of which is firmly fixed an olive-shaped piece of ivory or hard rubber. The ends are made of different sizes. The ordinary stomach-tube may also be used for the same purpose, and is the safest instrument at the first exploration.

In introducing the bougie, or tube, the patient should sit on a low chair with his head thrown back. The bougie, or tube, is then passed to the posterior wall of the pharynx and then down into the gullet. Usually a slight resistance is encountered at the level of the cricoid cartilage, but it is easily overcome, and after this the descent is easy. Caution should, however, always be exercised, as the bougie has a few times been pushed through an ulcer of the esophagus into the pleural cavity or lung, while ulceration has been produced by its repeated use in simple nervous spasmodic obstruction. The patient should be instructed to breathe rapidly and to swallow at stated intervals. It is rarely necessary to pass the finger of the operator into the mouth.

ESOPHAGITIS.

Acute Esophagitis.—An acute inflammation of the esophagus is practically limited to inflammation induced by the swallowing of very hot, or corrosive liquids, like strong acids and alkalies, or by the lodgment of foreign bodies. It is true, diphtheritic inflammation sometimes extends from the pharynx downward, and the esophagus has also been invaded by a vesicle of smallpox, but these conditions are not likely to be differentiated from the primary disease. Mycotic esophagitis, producing stenosis of the esophagus in sucklings, has been alluded to as a possibility on page 326.

Morbid Anatomy.—Appearances vary with the cause. In addition to the usual redness, sloughing and disintegration of the tissue may result. Milder degrees of inflammation produce less conspicuous alteration. A granular appearance may succeed desquamation of the epithelium. Diphtheritic false membrane presents the same characters here as elsewhere.

Symptoms.—These are chiefly *pain* beneath the sternum, increased by swallowing, which in extreme degrees of inflammation becomes agonizing and, indeed, renders swallowing impossible. Copious mucous secretion is sometimes present, which may be raised or regurgitated to the fauces and expectorated or passed into the stomach. Milder grades of inflammation may be without symptoms, intermediate grades present corresponding symptoms. If healing results after destructive inflammation, the cicatricial tissue behaves as it does elsewhere contracting and distorting the parts, oftentimes with resulting stenosis.

Treatment.—Little can be done to aid healing. For the most part, therefore, it must be given over to nature. If deglutition is possible, demulcents may be used, while the swallowing of pieces of ice sometimes gives comfort. When deglutition is impossible, the patient must be fed with nutritious enemata. The treatment of resulting stenosis is that of stricture of the esophagus, which see.

Chronic Catarrhal Esophagitis.—This affection is sometimes favored by valvular heart diseases, cirrhosis of the liver, or other cause of venous obstruction. The resulting affection is a catarrhal inflammation associated with mucous secretion. A hemorrhoidal state of the veins may be thus caused, which may proceed to rupture, and an hematemesis which closely resembles the bleeding from a gastric or duodenal ulcer.

PEPTIC ULCER OF THE ESOPHAGUS.

Definition.—An ulceration usually in the lower part of the esophagus, due to the direct solvent action of the gastric juice, favored by certain predisposing causes. The subject has been carefully studied by Tileston and others.

Etiology.—It may occur at any time of life between infancy and old age, but is most frequent in middle life and in men, it having been found in this sex in 28 out of 30 cases. It was found alone and complicating other conditions as ulcer of the stomach and duodenum. Directly caused by the solvent action of the gastric juice, it is favored by some, associated condition always present though not always ascertainable. One of these is insufficiency of the cardiac orifice of the stomach permitting regurgitation. Other associated conditions are repeated vomiting and disease of the various abdominal organs, including peritonitis, nephritis, chronic gastritis, but especially ulcer of the stomach and duodenum; also dilation of the stomach, which is often responsible for insufficiency of the cardiac orifice.

Morbid Anatomy.—The ulcer varies in size from a pin-head to a pea and may be single or multiple. Frequently in the lower part of the esophagus, it is found most frequently in the posterolateral portion. Perforation has taken place into the pleural cavity, the pericardium and the omental cavity. Scars have been found representing healed ulcers.

Symptoms.—The lesion is sometimes found at autopsy when previously unsuspected, and again the symptoms of perforation have been the first indication of it, but pain, tenderness, dysphagia, vomiting and hematemesis are natural consequences. The pain is usually at the xyphoid cartilage with tenderness in the adjoining epigastrium. There is no tenderness in the back corresponding to that of ulcer of the stomach. The dysphagia is characterized by pain and difficulty at the end of the act of swallowing. It may be intermittent. Hematemesis is usually the result of erosion of an artery. It is characterized further by the absence of nausea as contrasted with the vomiting of blood due to ulcer. Perforation is not an infrequent result having been found in six out of 14 cases. More rarely it is associated with evidence of stenosis of the pylorus with consequent dilatation of the stomach, itself the cause of the insufficiency referred to as favoring peptic ulcer.

Diagnosis.—The diagnosis in general is covered by the symptomatology above narrated. Differentially peptic ulcer of the esophagus is distinguished from gastric ulcer by the presence of dysphagia, while the pain follows more closely the act of deglutition than in ulcer of the stomach, and the pain and tenderness due to the latter are generally lower down. In ulcer of the esophagus the tenderness is substernal. Gastric ulcer at the cardiac orifice has symptoms more like those of ulcer of the esophagus.

It is well known that some of the most serious hemorrhages in cirrhosis of the liver arise from varicose ulcers of the esophagus. Such varicosities are characterized by the absence of previous pain and dysphagia, while they are also associated with cirrhosis of the liver with which peptic ulcer is not, as a rule.

Stenosis of the esophagus may succeed upon ulcer and must be distinguished from stenoses due to pressure from without, such as that by medi-

astinal tumors, abscesses, and aortic aneurysm, when the presence of the symptoms of these diseases must come to our rescue. Intermittent dysphagia is a rare characteristic of aneurysm and of the other causes of stenosis; carcinoma is more insidious and is associated with cachexia, but its duration is shorter. The blood in hemorrhage from cancer is scanty and mixed with particles of food and mucus while the painis more constant. The favorite seat of cancer is opposite the bifurcation of the trachea, while ulcer is rarely so high. Enlarged adjacent glands are also present in cancer.

Stenosis by corrosive poisons is readily determined by the history. Tuberculous and syphilitic ulcers are characterized by their indolence and comparatively painless character. The various diverticula due to obstruction by these causes may generally be determined by the sound.

Prognosis.—The prognosis of peptic ulcer is unfavorable as far as cure is concerned, while the difficulty in diagnosis increases the uncertainty.

Treatment.—Treatment is the same as that of gastric ulcer, liquid, cool and demulcent foods being indicated. In extreme cases rectal feeding must substitute all other forms of nourishment. As to drugs, silver and bismuth are given as for ulcer of the stomach. Direct applications may in the future be possible with the esophagoscope. Stenosis must be treated in the usual way.

CANCER OF THE ESOPHAGUS.

Description.—This is usually a hard epithelial tumor, most frequent in the middle third of the esophagus, though it may involve the cardiac orifice of the stomach, and more rarely other portions. E. Rindfleish, especially, describes a softer and more superficial form, which invades larger areas in a diffuse way. It is rather more frequent in men, and appears first as zonular infiltration of the mucous membrance, which ulcerates. The resulting ulcer may also extend around the tube, acquiring a width of two or three inches (5 to 6 cm.). The primary and usually permanent result, unless ulceration does away with it, is a stenosis of the esophagus, followed by dilatation of the tube, with hypertrophy of the walls above the stenosis.

Symptoms.—*Difficult* and *painful deglutition* is usually the first symptom of stenosis, though *pain*, independent of deglutition, may precede. *Swallowing* becomes more and more difficult, and ultimately, even liquids may be regurgitated. *Regurgitation* of food may not be immediate, and the date of its appearance is usually dependent on the seat of the obstruction and extent of dilatation above it. A discharge of *blood* and mucus may attend an effort to introduce the bougie. Death commonly takes place from exhaustion or actual starvation. But before this happens there may be a rupture into the larynx or a bronchus, producing death by suffocation, by gangrene, or by an inhalation pneumonia. There may be ulceration into the aorta or one of its large branches, causing fatal hemorrhage; into the pericardium, producing fatal pericarditis. Ulceration into the mediastinum or erosion of the cervical vertebræ sometimes occurs, with more delayed fatal ending. Emphysema is a sign of rupture into the lung. The adjacent lymphatic glands of the neck are sometimes invaded. Rarely the disease is latent throughout its entire course.

Diagnosis.—This may have to be delayed a short time, but is soon clear. The continued obstruction, the emaciation, and the weakness soon distinguish the case from one of spasmodic stenosis. Compression by adjacent growths should be remembered as a source of obstruction, aneurysm being perhaps the most frequent cause of this kind; but aneurysm may generally be recognized by its other signs. Examination by the esophagoscope with removal of a portion of the growth for microscopic examination is necessary.

Prognosis.—This is always ultimately fatal.

Treatment.—Treatment can only be made to prolong life. The bougie should not be used after the diagnosis of cancer is established, because of the danger of causing perforation. So long as liquid food can pass the obstruction it should be used; after this, nutritious enemas in the manner recommended under cancer of the stomach. Esophagostomy or gastrostomy may be presented for the patient's consideration. The former promises nothing, but life may be prolonged by the latter with much comfort to the patient.

SPASM OF THE ESOPHAGUS.

SYNONYM.—*Esophagismus.*

This is not an unusual affection in hysterical women, and even in male hypochondriacs. These are generally past middle life. It also occurs in hydrophobia, chorea, and epilepsy. The spasm is commonly excited by an effort to swallow solid food, and rarely even liquids act similarly. A possible result of spasm is a dilatation, as shown in a case of my own, to be again referred to.

Diagnosis.—The diagnosis is readily made by the bougie, which, though it may be stayed for a minute at the seat of spasm, ultimately passes it without the application of force. It is also associated with other symptoms of hypochondriasis, while extreme pain, the gradual emaciation, weakness, and ultimate cachexia of cancer are absent. Errors of diagnosis have, however, been made, and death has even occurred when autopsy disclosed no lesion to explain it.

Treatment.—This is that of the hypochondriacal state and the frequent use of the bougie, of which the moral effect is also good. One introduction has sometimes been sufficient. On the other hand, repeated passage of a bougie has produced ulceration, whence the caution already enjoined in the use of the instrument.

STRICTURE OF THE ESOPHAGUS.

Etiology.—Stricture may be produced by any of the conditions just considered, carcinoma, contraction of scar tissue after esophagitis, whether traumatic or syphilitic, or by spasm. Other causes are pressure by external tumors, such as aneurysm, enlarged lymphatic glands or mediastinal tumors. Then there is congenital narrowing, and finally polypoid tumors

projecting from the mucous membrane. If the stenosis be cicatricial, the precise cause is to be determined by the history of the case and its situation by the esophageal bougie.

Symptoms.—These are those of obstruction, described under cancer and spasm, with or without the painful element; to which may be added those of dilatation of the esophagus, to be next considered.

Treatment.—This is altogether by the careful use of the bougie. Dilatation of the cicatricial stenosis is often quite successful. The largest bougie should be first introduced very gently, without force, really as a sound, *as far as the obstruction only*. Then smaller sizes should be tried until one is found which will pass, and from this point, again, larger sizes should be successively employed. At each sitting the bougie originally passed with ease should be started with and followed more rapidly by the larger sizes, as the physician becomes familiar with his patient's case.

In congenital cases less is to be expected, while obstruction by external growths, unless they be removable, is practically irremediable, and grows gradually worse. Even cicatricial stenosis may be such that the smallest bougie cannot pass, in which event nourishment by the rectum may be used. Gastrostomy, however, should be at once performed.

DILATATION OF THE ESOPHAGUS.

Dilatation of the esophagus may involve the whole circumference of tube, when it is known as *diffuse* or *total;* or it may affect only one spot, when it is circumscribed, or constitutes a *diverticulum*.

Diffuse Dilatation.—In every case of organic stenosis of the esophagus, from whatever cause, there is sooner or later dilatation above it, delayed at first by hypertrophy of the muscular coat, which is thus enabled to force the food through the narrowing. Sooner or later this coat becomes paralyzed, the wall yields to the pressure of accumulated food, and dilatation follows. The resulting sac is usually spindle-shaped, but may be cylindrical, and is naturally larger the lower the seat of obstruction.

But dilatation occurs without previous organic stenosis. Repeated spasm is a cause, and dilatation from cardio-spasm has come to be a well-recognized condition. It may be that it is preceded at times by some traumatic cause which weakens the wall of the tube.

Diverticula.—Diverticula or circumscribed pouches in the walls of the esophagus are of two varieties. They have been especially studied by Zenker, who has divided them into *pressure* or *propulsion* diverticula and *traction* diverticula according to their mode of origin.

Traction diverticula are the more frequent, yet clinically are of less interest because often not recognized until their subjects are on the necropsy table. They are small, scarcely ever exceeding a centimeter (0.4 in.) in diameter, and relatively frequent in children. They are ascribed to some traction effect exerted on the wall of the esophagus. This may be, as Rokitansky and Zenker suggested, due to the contraction of a tissue which has formed adhesions to the esophagus. Such a tissue is afforded by the bronchial glands, which become inflamed, caseate, and contract, and as they are situated at the bifurcation of the trachea, the more frequent occurrence

of traction diverticula at this situation in the *anterior* wall of the gullet is thus explained. Such diverticula may be multiple.

Pressure diverticula are much rarer. They occur almost always in men rarely in children. They are found most frequently at the junction of the pharynx and esophagus, on a level with the cricoid cartilage, where the muscular wall, formed chiefly by the inferior constrictor of the pharynx, is weakest, and are caused by pressure from within. This may be exerted by the bolus of food itself, especially if it be habitually large, as in rapid eaters, while its operation may be further facilitated by some traumatic injury to this part of the throat, such as may be caused by the lodgment of a bone.

The sac is found to be bounded by mucous membrane and thickened submucous coat, the muscular coat giving way to let the mucous coat pass through it, as in a hernia. It is found invariably in the *posterior* wall, and hangs in front of the spinal column. One such, reported by Joseph McFarland and John M. Swan was 5 centimeters long and 3 wide.[1]

"Their causation is doubtless due to the fact that the effect of pressure of the wall between a firm bolus of food and a hard organ externally (such as tracheal calcification, a calcified thyroid, or even, it is said, a calcareous artery) is to injure the tissues; they are deprived of physiological rest by constant movements entailed in eating and drinking. When the esophageal wall is thus weakened, the muscular and supportive tissues become insufficient and stretch easily; after each considerable distention the wall fails to contract to its proper degree, and the lumen of the tube remains a little wider at this point than normal, and each successive dilatation increases the size of the pouch. When the wall is weakened, a diverticulum is caused by a stretching of the muscle fibers and a separation of one from another; when an individual group of fibers break, or when this separation occurs, a hernial protrusion of the mucosa and submucosa through the gap follows; rarely these protrusions are still covered by an incomplete muscle layer, but even when this is not the case the thickened submucosa forms a wall that is at times as thick as the normal esophagus. When they result from such a hernial protrusion they often have a narrow slit-like opening; notwithstanding this, they may attain a large size when their liability to rupture constitutes a real menace to the safety of the individual. The increase of intra-esophageal pressure by the passage of food distends yet more the beginning sac, and because of their production thus by pressure from within, these are called "pressure or pulsion diverticula." They occur most frequently in the male sex and in middle or advanced life; it is supposed by some good authorities that pressure diverticula occurring in the continuity of the tube are really the result finally of traction diverticula; if this be true, these would be properly called "pressure traction diverticula" a class described by some authors, which is for our purposes a needless refinement." (*McCrae in Osler's System*).

Symptoms.—In cases of *diffuse* dilatation originating in stenosis, apart from the inference that where there is stenosis there must ultimately be dilatation, the first symptom to attract attention is the *feeling* on the part of the patient that his *food does not enter the stomach*, but lodges higher up.

[1] Reprinted from "Medicine," May, 1903.

though the quantity swallowed is evidently more than would be held by an esophagus of ordinary caliber; usually, sooner or later, follows the *regurgitation*, or gulping up of this accumulation. The same symptoms are said to attend dilatation without stenosis. The latter event can only be explained on the supposition that, in consequence of the paralyzed state of the muscular wall of the esophagus, there is no force to push the food down, while the gradual widening of the tube affords support for its lodgment, which is further favored if the enlargement takes the shape of sacculations or a pocket.

Traction diverticulum rarely causes symptoms. Those arising from pressure diverticulum are first those of *dysphagia*, as the diverticulum grows larger, and the food lodges more and more; *regurgitation*, though the sac is rarely thoroughly emptied, and the retained food sometimes undergoes decomposition, giving rise to fetid breath. "The inflammation so set up may spread to the esophagus itself, or may even proceed to ulceration and rupture, with the formation of peri-esophageal abscess; or the break may occur into the thoracic cavity, setting up gangrene of the lung or empyema; a diverticulum has been known to burst into a tuberculous cavity of the lung (*Schmidt*). When the diverticulum is small the tube will readily pass it, but as it becomes larger the tube is often directed into the sac itself, where the mobility of its lower end and the impossibility of passing it farther may assist the diagnosis (*McCrae in Osler's System*). The difficulties increase until after a while it is almost impossible to get food into the stomach, though extraordinary efforts are made by the patient to do so, with greater or less success. Complete closure results when the diverticulum becomes so large as to flex upon the gullet and compress it.

Diagnosis.—This is suggested by an inability on the part of the patient to swallow well, and regurgitation of food. This is further confirmed when the condition is a diverticulum due to spasm by the inability to pass a soft tube into the esophagus while a solid bougie can be passed with little difficulty. If on the other hand a diverticulum is present, the tip of a solid bougie is likely to catch in the pouch and effectually prevent its passage downward. The best method of diagnosis, however, is the one of X-ray with the administration of bismuth. When the case is thus studied the diagnosis of stricture, dilatation or diverticulum can be certainly made. No excuse now exists for a long delayed diagnosis.

With the prolongation of the condition the proper nourishment of the patient becomes more and more difficult; he emaciates, grows weaker, and ultimately perishes from exhaustion unless carried off by some other disease.

Treatment.—The treatment of diffuse dilatation and diverticula is essentially the same. It consists, first, in measures to maintain the nutrition of the patient. Generally he is able to ingest a certain amount of food by his own efforts, of which those detailed in the case of my own patient are an illustration. After this the stomach-tube becomes the most ready way. This, too, he should be taught to use himself. Rectal alimentation may help somewhat, but is alone inadequate for any length of time, while the inconvenience of any and all of these procedures renders the patient anxious for more complete relief. The treatment of dilatation due to cardio-spasm

will be detailed later and must of course be the treatment of the cardiospasm.

Complete relief may be accomplished by operation, by which diverticula have been successfully removed. The difficulties in the way of operation are, however, great. The operative treatment of dilatations due to stenoses resolves itself into that of the stenoses themselves. In both forms gastrostomy may be the ultimate measure that promises relief for a time.

DISEASES OF THE STOMACH AND INTESTINES.

ACUTE GASTRITIS.

SYNONYMS.—*Acute Gastric Catarrh; Acute Dyspepsia.*

Definition.—Acute inflammation of the stomach, of moderate intensity, due to simple nonspecific irritation or to irritation from the products of decomposing and fermenting foods.

Etiology.—This form of inflammation occurs at all ages, and is often due to the irritant effect of indigestible food or food in a state of incipient decay and fermentation. Simply overloading the stomach, even though the food be wholesome, may be a sufficient cause. The introduction of large quantities of strong alcoholic drinks, as often happens in a debauch, is one of the most common causes of acute gastritis of the simple variety. The susceptibility of different individuals and of different families to the foregoing causes of irritation varies greatly.

Morbid Anatomy.—A more or less uniform coating of the stomach with mucus is the most constant feature of simple acute gastritis, and justifies for it the name, gastric catarrh. The removal of this mucous coating reveals a hyperemic redness, which in the highest degrees may be associated with punctiform hemorrhages and hemorrhagic erosions. The mucous membrane is swollen and edematous, and minute examination recognizes numerous mucous-laden cylinder cells, which have been extruded from the mucus-glands everywhere present, while even the peptic gland cells are cloudy and granular.

Symptoms.—These are a natural sequence of the morbid state. A *want of appetite* and *loathing of food*, *nausea*, more rarely *pain*—these are the more constant subjective symptoms. To them may be added an *unpleasant taste* in the mouth, sometimes bitter, sometimes metallic, a *pasty* sensation of *dryness*, and even *thirst*, a sense of *fullness in the head* rather than headache, and *dizziness*, and often extreme *mental depression*.

Objective symptoms are epigastric *distention*, more rarely *tenderness*, a *coated tongue*, *dryness* of the *lips*, rarely *herpes*, a *heavy breath*, acid or bitter *eructations*, sometimes a scanty secretion, at others an excess of saliva, finally *retching* and *vomiting* with greater or less relief. The *bowels* are constipated, though sometimes there is diarrhea. *Jaundice* is occasionally present, and indicates that the inflammation extends into the duodenum and produces obstruction of the common bile-duct. There may be slight *fever*, sometimes decided, with a temperature of 101° F. (38.3° C.), or slightly more, and a corresponding pulse. On the other hand, the pulse is not infrequently slowed below the normal, being inhibited by the gastric irritation.

The *urine* is "feverish," scanty, and high colored, with a corresponding specific gravity and a tendency to deposit urates. Most cases are without febrile symptoms. Indeed, v. Leube says that in a few instances only is fever the result of acute gastric catarrh, and that when the two are associated, the gastric catarrh is rather the result of some acute febrile process, as, for example, one of the infectious fevers. It has occasionally happened that gastritis has been ushered in with a *chill*.

Gastric Contents.—The vomited matter and gastric contents removed after a test-meal are deficient in hydrochloric acid, but contain an excess of mucus, lactic and fatty acids, and more than the normal residue of undigested food. Digestion is prolonged, the stomach-washings exhibiting a considerable amount of undigested food seven hours after the ingestion of a test-meal. Indeed, it often happens that in from 12 to 24 hours after the beginning of such an attack large quantities of undigested food are vomited in much the same condition in which they are swallowed.

Diagnosis.—This is not usually difficult, except in the case of the febrile form. In this form, especially when the disease has been ushered in with a chill, it is sometimes difficult to decide between it and some one of the infectious fevers, but a few days' waiting will soon remove the doubt by the appearance in the latter of eruptions or other distinctive symptoms. The presence of a cause sufficient to excite gastric inflammation will add to the probability of the presence of acute catarrhal gastritis.

Prognosis.—This is invariably favorable in cases of true simple gastritis.

Treatment.—Many mild cases recover spontaneously, if let alone and if all food is withdrawn for 24 hours. The symptoms gradually subside and the patient recovers. In a few cases where there is evidently retained food, a *lavage* will give relief; in all, a brisk *saline purge* is helpful. A bottle of cold solution of citrate of magnesium in divided doses, a fourth every half hour, is one of the most agreeable and efficient aperients to relieve the congestion and the symptoms. Or some one of the *natural aperient waters*, such as Hunyadi Janos or Friedrichshalle, Apenta, Rubainat, Veronica, or Carlsbad, may be substituted. If there be great sensitiveness of the stomach, small doses of calomel, frequently repeated, 1/6 to 1/4 grain (0.011 to 0.016 gm.) every hour, may be substituted, or 7 1/2 to 10 grains (0.5 to 0.666 gm.) may be given in one dose. In either event a saline should be given sooner or later, as in this way is secured copious depletion of the upper alimentary canal. The *alkaline mineral waters*, represented by Vichy and Vals in France, by Carlsbad and Marienbad in Bohemia, are admirable adjuvants, since they aid in clearing the stomach of mucous secretion and in producing osmosis. The saline mineral waters represented by the well-known Saratoga waters of this country are also efficient, more especially by their aperient qualities.

CHRONIC GASTRITIS.

SYNONYMS.—*Chronic Gastric Catarrh; Chronic Catarrhal Dyspepsia.*

Definition.—A condition of chronic hyperemia, associated with excessive mucous secretion and deranged gastric juice formation and lack of motor power, with ultimate structural changes in the mucosa.

Etiology.—Any cause which will produce continuous moderate irritation of the mucous membrane of the stomach is capable of producing chronic gastritis. The immoderate use of alcohol, constant overeating, rapid eating, eating of improper food, are all common causes of this condition.

Very frequently, too, chronic gastritis is secondary to primary disease elsewhere, and especially mitral disease of the heart and interstitial hepatitis and nephritis. Both of these affections cause a passive congestion of the stomach, which ultimately produces the lesions characteristic of chronic gastritis. Thrombosis of the portal vein acts similarly. Chronic pulmonary disease, and even diseases of the pleura impeding the circulation in the lungs, produce similar effects through stasis. A predisposition exists in certain families to chronic gastric catarrh. All cases of carcinoma and many cases of ulcer and dilatation are causes. General diseases such as anemia, tuberculosis, gout and diabetes are found among the causative factors.

Morbid Anatomy.—The fundamental condition is a hyperemic swelling of the gastric mucosa. This is favored by the superficial situation of the venus plexus about the mouths of the gastric glands as contrased with the deep-seated position of the arterial network around their bases, by the thinness and compressibility of the venous walls, and by the sluggishness of circulation necessitated by the peculiar secretory function of the stomach. The hyperemic surface is, however, more or less obscured by a tough yellowish-white covering, made up of mucus and emigrant pus-cells.

These may constitute the sum of changes, but in more chronic cases minute examination reveals a varying degree of hyperplasia of the connective tissue, and even of the mucous glands, which exhibit in places an atypical branching, like the fingers of a glove. The tubules are distended by secretion in some places, and in others stenosed by the contraction of the overgrown connective tissue surrounding them. The hyperplastic process may result in *plication* of the mucous membrane, such as is natural at the pyloric end, and lead finally to the *mammillated stomach* by atrophy and contraction of certain portions, and to more pronounced swelling of the remaining parts. An ultimate result is sometimes the rare condition known as *polyposis ventriculi*. Atrophy of the mucous membrane may be extensive, and even almost total, such as are being reported by Henry and Osler.

Symptoms.—These naturally result from the morbid state. The mucous membrane is bathed with *mucus*. The *gastric juice* is *imperfect* in quality and quantity. Especially is the hydrochloric acid deficient. Digestion is therefore imperfect, the residue of ingested food undergoes fermentation and decomposition, generating lactic, acetic, butyric acids and alcohol. Peristalsis is delayed because of the absence of its natural stimulus and thence follows a further retention of food in the stomach with eructation of gas and fermented food materials. The natural consequence of such morbid changes is *loss of appetite* and even *disgust for food*, an unpleasant *taste*, a *pasty sensation in the mouth*, a *coated tongue*, and *discomfort after taking food*, including *nausea*, often *vomiting*, sometimes immediately, sometimes an hour or two after taking food. The vomitus consists of undigested food, usually mixed with a large amount of mucus. Its reaction may be neutral or acid, sometimes even acridly so, but the acidity is not

due to hydrochloric acid, which is diminished, but to the organic acids generated in fermentation.

To these symptoms may be added *headache*, or a dull, unpleasant feeling in the head, *vertigo*, *disturbed sleep*, *depression of spirits*, a sense of weariness, disgust with life and nausea. Very disagreeable is the *distention* and sense of fullness in the epigastrium, causing even *pain*, which adds further to existing discomforts. There may be *tenderness*, but it is diffuse, and not circumscribed. There is usually *constipation*, while the urine may be scanty. Reflected symptoms are *palpitation; frequent, slow*, or *irregular pulse;* shortness of breath. There is no fever. *Cough*—the so-called "stomach cough"—is sometimes present, but more frequently what is called by the patient stomach cough is the cough of tuberculosis, which the sanguine patient easily convinces himself is due to stomach derangement.

Gastric Contents.—Analysis of the gastric contents, withdrawn after a test-meal, shows a deficiency of pepsin as well as of hydrochloric acid, while other tests show retarded peristalsis and delayed absorption. Frequently fungi, especially yeast-spores and sarcinæ ventriculi are found.

Should the disease progress to total atrophy, the gastric contents, after a test-meal, may even be devoid of mucus as well as of free and combined hydrochloric acid, of pepsin, and epithelium, and may be made up mainly of undigested food, with bacteria and a few round cells. Repeated examinations of stomach contents, after a test-meal, may be necessary before a sufficient knowledge of its features can be arrived at. These cases are often accompanied by a severe anemia, the gastric disorders, and loss of weight causing the cases to closely resemble gastric carcinoma.

Diagnosis.—With the symptoms detailed, and the altered state of the secretory, absorptive, and motor functions of the stomach ascertained, there is usually no difficulty in diagnosing chronic gastric catarrh. It is to be remembered, however, that chronic gastric catarrh may accompany ulcer and carcinoma of the stomach, in which the otherwise distinctive symptoms of the former are obscured, while with the exception of tumor and occasional coffee-grounds vomit the symptoms of carcinoma may not differ from those of chronic gastric catarrh, hydrochloric acid and pepsin being deficient in both. Dilatation of the stomach is also accompanied with symptoms of gastric catarrh, including even the clinical characters of the gastric juice, and careful examination must always be made for the physical signs of dilatation. The use of bismuth meal and examination by X-ray is advisable. Constantly imperfect peristalsis, a puckering of the wall of the stomach or retention will be indicative of either carcinoma ulcer, or perigastric adhesions which would not otherwise be considered.

Gall-stones and cholecystitis from other sources must always be considered. Chronic appendicitis must also be kept in mind, for it must be remembered that many cases of "indigestion," supposed to be due to a simple chronic gastritis are really symptomatic of cancer, ulcer, gall-stone, appendicitis of pancreatitis.

Prognosis.—The prognosis and treatment will depend upon the etiology. If the chronic gastric catarrh is a result of chronic cardiac or hepatic disease, it is curable only so far as these affections are curable, and is relieved as these are relieved. If it is the result of carcinoma, ulcer, gall-stones or appen-

dicitis the prognosis must depend entirely upon the ability to remove these conditions. Careful physical examination is always necessary in each case, that obscure cases may be recognized.

Chronic gastric catarrh not the result of any organic disease, and which has not already resulted in atrophy of the mucous membrane, may be cured by careful and persevering treatment. If there be extensive atrophy of the gastric mucous membrane, a proper assimilation of food becomes impossible, and the symptoms of anemia are ultimately added. Their close resemblance to those of pernicious anemia has been pointed out, while an essential cause of pernicious anemia has been held to be gastric atrophy, in evidence of which a case of William Osler and Frederick P. Henry is often quoted.

Treatment.—The treatment of chronic gastritis caused by any organic disease resolves itself into the treatment of that disease. If the causative disease is irremediable then symptomatic treatment of the gastritis will frequently give relief. Chronic appendicitis and gall-stones must be removed. Gastric ulcers and carcinomata must be subjected to operation by a skilled surgeon.

A successful treatment of catarrhal dyspepsia requires considerable patience, but if the diagnosis be correctly made and the cause removed, the patient may be promised a cure in time. Of primary importance is the *elimination of the cause*, whether it be alcohol, injudicious eating, or lack of proper exercise and fresh air. Alcohol must be abstained from. Regulated exercise in the open air must be taken, the diet must be regulated. Simple, wholesome, and properly cooked food, thoroughly masticated and slowly taken, should be the rule of every life, and the simple forms of the disease may sometimes be cured by the return to such a habit, especially if a proper action of the bowels is also habitually secured.

Diet.—Each case must be studied separately. No routine diet can be selected. After careful study and inquiry the patient should be given a printed or written list containing the foods he may take. A convenient list is one which contains the printed names of all ordinary foods and drinks. The foods *not* allowed to the patient under treatment may be stricken from the list (pp. 354, 355).

Of course all fat foods, fried foods, pastry and confections must be debarred.

No less important than the kind of food taken in the method of eating. Food should be well chewed, eaten slowly, taken in moderate quantity at regular intervals. Neither just before nor just after violent physical or mental exertion.

The measures by which the *regular habit of bowel movement* is brought about must vary with circumstances. Perhaps the most important factor in obtaining a daily bowel movement is regulation of the time at which a stool is attempted. Too often a stool is only had at a convenient time. If the patient is directed to attempt a stool at a certain time each day this regulation of habit will in itself frequently cause a stool. It is important that this regularity be insisted upon in early life. Foods rich in refuse matter such as fruits, stewed or raw, cereals, etc., are efficacious. Pernicious drugging is harmful. The millions of laxative pills yearly used without reason cause an

FOR ..... **DIET**

SOUPS — DAILY AMOUNT

○ **Broths**
- Beef
- Veal
- Mutton
- Chicken
- Bouillon
- Consommé
- Ox-tail

○ **Purées & Creams**
- Barley
- Rice
- Pea
- Bean
- Potato
- Tomato
- Asparagus
- Onion
- Celery

○ **Thick Soups**
- Vegetable
- Noodle
- Julienne
- Vermicelli
- Calf's Head
- Mock Turtle
- Mulligatawney
- Clam Chowder
- Fish Soups

○ **FISH**
- **Boiled**
- **Broiled**
- **Baked**
- **Salted**
- Sardines in Oil

○ **OYSTERS**
- **Raw**
- **Panned**
- **Broiled**
- **Stewed**
- **Scalloped**

○ Clams
- Crabs
- Lobsters
- Shrimps
- Terrapin

○ **MEATS** — DAILY AMOUNT
- **Boiled**
- **Stewed**
- **Roasted**
- **Broiled**
- **Hashed**
  - Beef
  - Mutton
  - Mutton Chops
  - Lamb
  - Lamb Chops
  - Veal
  - Sweetbreads
  - Brains
  - Liver
  - Kidneys
  - Pork
  - Bacon
  - Ham
  - Dried Beef
  - Corned Beef
  - Sausages
  - Pigs' Feet
  - Tongue
  - Tripe

○ **POULTRY**
- Chicken
  - *White Meat*
- Squab
- Turkey
- Duck
- Goose
- Guinea Fowl

○ **GAME**
- Venison
- Rabbit
- Wild Duck
- Birds

○ **EGGS**
- **Soft Boiled**
- **Poached**
- **Scrambled**
- **Omelet**

○ **MILK**
- Unskimmed
- Skimmed
- Buttermilk
- Cream
- *Boiled Milk*
- *Pasteurized*

○ **BUTTER**

○ **CHEESES**

VEGETABLES — DAILY AMOUNT

○ **Starchy**
- Rice
- Corn
- Peas
- Beans
- Lima Beans
- Lentils
- Potatoes
  - *Baked*
  - *Boiled*
  - *Mashed*
  - *Chipped*
- Sweet Potatoes
- Beets
- Parsnips
- Turnips
- Carrots
- Kohl Rabi
- Artichokes
- Salsify
- Radishes

○ **Green Vegetables**
- Tomatoes
  - *Raw*
  - *Stewed*
  - *Baked*
- Egg Plant
  - *Baked*
- Onions
  - *Boiled*
  - *Baked*
- Leeks
- Cabbage
  - *Sauer Kraut*
  - *Cole Slaw*
- Cauliflower
- Brussels Sprouts
- Sea Kale
- String Beans
- Asparagus
- Pumpkins
- Squashes
- Celery
  - *Raw*
  - *Stewed*
- Rhubarb
- Veg't'le Marrow
- Spinach
- Lettuce
- Water Cress
- Beet Tops
- Okra
- Capers
- Cucumbers
- Endives
- Chiccory
- Sorrel

DIRECTIONS: Any articles are permitted except those crossed out. Individual articles in any list marked thus ○ are not allowed.

GENERAL INSTRUCTIONS:

1. Eat slowly and at regular hours; masticate thoroughly.
2. Take fluids moderately at meals; drink water freely at other times.
3. Avoid anxiety and business cares at table; rest for a while after meals.
4. Use laxative foods and drink an abundance of water to prevent constipation.
5. Do not partake of a great variety of dishes at any one time, nor eat large quantities of anything very hot or cold.

LIST. DATE.....

	DAILY AMOUNT		DAILY AMOUNT		DAILY AMOUNT
○ **BREAD**		○ **ICE CREAM**		○ **NUTS**	
Fresh Baked		Vanilla		Cocoanuts	
Stale		Chocolate		Chestnuts	
Toasted		Fruit Flavors		Walnuts	
Pulled				English Walnuts	
Zwieback		○ **WATER ICES**		Brazil Nuts	
White Flour		Orange		Hazel Nuts	
Graham		Lemon		Pecan Nuts	
Rye		Sherbets		Shellbarks	
Crackers				**Salted**	
Gluten		○ **CAKE**		Almonds	
Almond		Plain		Peanuts	
Inulin					
Soya		Fancy		○ Olives	
Aleuronat				Pickles	
		○ **JELLIES**		Truffles	
○ **HOT CAKES**		Lemon		Mushrooms	
		Wine			
○ **CEREALS**		Fruit		**BEVERAGES**	
Oat Meal					
Corn Meal		○ **SUGARS**		○ **Non-Alcoholic**	
Hominy		Cane Sugar		Coffee	
Arrow-root		Grape Sugar		Tea	
Tapioca		Honey		Cocoa	
Cornstarch		Molasses		Chocolate	
Farina		Confectionery		Lemonade	
Sago		Saccharine		Grape Juice	
Macaroni		Levulose		Ginger Ale	
Spaghetti				Soda Water	
		○ **FRUITS**		Mineral Waters	
○ **SPECIAL**		Oranges		*Label:*	
Beef Juice		Lemons			
Clam Juice		Limes		○ **Alcoholic**	
Scraped Beef		Shaddocks		Beer	
Beef Tea		Grapes		Ale	
Albumen Water		Bananas		Porter	
Milk Toast		Pineapples		Stout	
Toast Water		Melons		Cider	
Barley Water				Sherry	
Gruel		**Fresh**		Port	
Irish Moss		**Dried**		Maderia	
Flaxseed Tea		**Stewed**		Malaga	
Milk Punch		**Preserved**		Tokay	
Egg Nog		Apples		Rhine Wines	
Koumiss		Peaches		*Label:*	
Wine Whey		Pears		Champagnes	
Mulled Wine		Plums		*Label:*	
Panada		Prunes		Clarets	
Caudle		Apricots		*Label:*	
Broth with Egg		Cherries		Burgundies	
Predigested Food		Raisins		*Label:*	
		Dates		Whiskies	
○ **PASTRY**		Figs		*Label:*	
				Brandies	
○ **PUDDINGS**		○ **BERRIES**		*Label:*	
Bread		Strawberries		Gin	
Cornstarch		Blackberries		*Label:*	
Blanc Mange		Raspberries		Liqueurs	
Rice		Huckleberries			
Tapioca		Gooseberries		○ **CONDIMENTS**	
Cup Custard		Cranberries		Pepper	
Junket		Mulberries		Mustard	
Cottage		Currants		Spices	
Hasty				Herbs	
Suet				Vinegar	
Fruit				Olive Oil	
				Horseradish	
				Sauces	
				Caviare	
				Pâté de Foie Gras	
				○ **SALADS**	

6. Rich dishes, fried foods, pastries, sweets, stimulants, and strong condiments, should be used, if at all, only in small amounts.
7. Under-cooked vegetables, overdone or hashed meats, hard-boiled eggs, and any articles habitually found to disagree, are to be avoided.
8. A moderate daily quantity of food for adults should average about—
   10 ounces of animal food (fish, oysters, meat, poultry, eggs, etc.);
   30 ounces of vegetable food (including bread and cereals); and
   50 to 80 ounces of liquids, including tea, coffee or cocoa, but principally water.—THOMPSON.

SPECIAL INSTRUCTIONS:

artificial condition which demands the stimulation. They should not be used.

Drinking of water before meals is useful. A small simple enema or a glycerin suppository may occasionally be used in the beginning of the regulation of the habit of stooling. Massage of the abdomen is of the utmost value in causing a patient to have a normal stool without the aid of drugs. Drugs of the vegetable laxatives cascara sagrada may be used in 30-drop doses of the fluid extract three times a day or a teaspoonful at bedtime. Podophyllin aloin and belladonna make an excellent combination in pill form. When it is remembered that we have to deal with a congested mucous membrane, it is plain why the salines which deplete the upper alimentary canal are so efficient. Among these are the numerous natural aperient waters, such as Friedrichshalle, Hunyadi Janos, Apenta, Carlsbad waters, and our own Saratoga and Bedford waters, all of which deplete the alimentary canal. The useful effects of these waters is so often availed of to remove the uncomfortable effect of a debauch in eating that their use is abused. No remedies are, however, so useful when needed, and the fact that almost any of them can be taken before breakfast, securing an effect after that meal, makes them doubly convenient. A fit substitute for the water, especially when traveling, is the Carlsbad Sprudel Salt, obtained by evaporating the Carlsbad water. Carlsbad salt, of which the dose is usually a teaspoonful, is best taken in a glass of hot water. An artificial Carlsbad salt may be made as follows: Sodium sulphate, 50 parts; sodium bicarbonate, 6; sodium chlorid, 3. The dose is a teaspoonful dissolved in a half a glass to a glass of water. The natural waters are, however, to be preferred, if they can be obtained.

Cascara sagrada is one of the most valuable of aperients. The best preparations are the solid and fluid extracts. The former may be given in 2 to 5 grain doses (0.132 gm.) in a pill after dinner and after supper. The fluid extract, in 15 or 20 minim (1 to 1.3 gm.) doses, can be given in the same manner, but the dose of each must be modified to suit the requirements of individual cases. In lieu of the saline aperients before breakfast, a glass of hot water alone, slowly sipped while dressing, is often useful and tends to relieve the morning sickness that sometimes attends chronic gastric catarrh. It probably liquefies the mucus and washes it away into the duodenum. Phenolpthalein in doses of three grains either alone in pill form or in combination with one of the laxatives mentioned above is of great value. It is of the greatest importance that patients be warned against indiscriminate drugging, either by the various laxative pills or by pills and capsules regularly prescribed.

As to medicines intended to aid indigestion, the most efficient is *hydrochloric acid* or nitromuriatic acid, probably best combined with one of the bitter tonics, tincture of nux vomica or tincture of gentian. It seems now definitely settled that hydrochloric is the acid to which the gastric juice owes its efficiency, and as well settled that it is diminished in chronic gastric catarrh. Another important rôle is assigned to hydrochloric acid, viz., an antiseptic effect, in checking the multiplication of pathogenic bacteria—bacteria of fermentation and decomposition—which are continually introduced with the food into the stomach. The latter has, heretofore, been

administered in too small doses.[1] Not less than 15 minims (1 gm.) of the dilute acid should be given, and from 30 to 60 minims (2 to 4 gm.) are sometimes required. It should be given, further diluted, 15 minutes after a meal, through a glass tube carried back into the fauces, not merely to save the teeth, but also to avoid the unpleasant taste.

It is usual also to employ the *bitter tonics* in the treatment of this form of dyspepsia, including gentian, quassia, columbo, angostura, cardamom, and nux vomica. They are supposed to stimulate the secretion of gastric juice, and should be taken immediately before meals or with food. *Common salt*, on the other hand, is a rational adjuvant, furnishing chlorin for the formation of hydrochloric acid.

Nitrate of silver is also a useful drug in cases of chronic gastric catarrh, in doses of 1/4 grain (0.0165 gm.) 15 minutes to half an hour before meals, dissolved in a quarter of a glass of water. Great care must be exercised that the silver treatment be discontinued after two or three weeks, use to prevent the appearance of argyria. It may be recommenced, however, after another interval of two or three weeks. Where there is acidity bismuth, sodium bicarbonate, and magnesia may be used, but it is better, if possible, to strike at the root of the evil by preventing the fermentations which produce the flatulence and acid. *Beta naphthol* in 3 grain (0.2 gm.) doses after meals is also an efficient remedy where there is fermentation and gaseous distention.

In obstinate cases the *milk treatment* may be resorted to with advantage, and should be carried out with skimmed milk or whole milk diluted with water or Vichy. The efficiency of the milk treatment is largely due to the fact that the quantity of food taken is greatly reduced. Not more than 2 ounces should be given at first, every two hours, the quantity increased only as the hunger of the patient demands more. There will be at first a loss of weight, but this is again recovered with the increase in quantity. Having secured a tolerance for milk, of which from 3 to 5 pints (1 1/2 to 2 1/2 liters) are required in 24 hours, the interval may be prolonged and the other articles of food cautiously added—a little bread and butter, an egg, a chop, or a small piece of steak, broiled. Gradually the simpler vegetables, such as rice and potatoes, may be added, then weak tea and coffee cautiously, the effect of each article being carefully watched. If flatulence is caused by the farinacea and sugars, they should be withdrawn. The same may be said of *ice-cream* and *iced water* with meals, though a moderate amount may be permitted between meals, especially of iced water. Ripe *fruits*, on the other hand, are very desirable foods and should be allowed tentatively.

In bad cases of chronic gastritis with retention *lavage* is one of the most useful measures. Not only does it wash away the coating of mucus which is at once a hindrance to the secretion of the gastric juice and a cause of nauseous discomfort to the patient, but it also stimulates glandular activity. It should be done in the morning before breakfast, with the stomach-tube with funnel attachment. Simple water as hot as can be borne may suffice, or if there be much mucus, a two per cent. solution of sodium bicarbonate

[1] Since 4.5 liters (9 pints) of 0.2 per cent. solution of HCl are required to saturate 100 gm. (about 3 oz., of dry fibrin, and this amount of acid utilized in combining with the albumin leaves none apparent as free HCl, it is plain why the small doses often prescribed are insufficient.

or Carlsbad salt, or a six per cent. solution of sodium chlorid may be used. If antiseptic fluids are indicated, a two per cent. solution of resorcin may be substituted, or a one per cent. solution of salicylic acid. It is extremely unwise to advise the patient to wash out his own stomach. A true habit is frequently formed contributing much to the neurasthenic state often present. The stomach must be previously cleansed and the silver also finally washed out by lavage, using salt solution if a solution of silver nitrate is used.

It is in these cases, too, that a course at Carlsbad is very efficient, and remarkable cures are reported. Here, too, the restricted dietary and depletion of the upper alimentary canal by the natural mineral waters are the beneficial agents. Similar courses are carried out at Kissengen, Wiesbaden, and Ems, but, unfortunately, we have no such places in America. Saratoga fulfills the conditions so far as an aperient water is concerned, but the majority of persons who go to Saratoga continue eating and drinking as at home. Finally, the habitual use between meals of the alkaline mineral waters alluded to—viz., Vichy, Vals, and Contrexville—is undoubtedly useful, relieving and averting gastric catarrh.

PHLEGMONOUS OR SUPPURATIVE GASTRITIS.

Definition.—A rare form of gastritis, in which there is diffuse purulent infiltration of the submucosa, but sometimes also circumscribed abscess, causing a possibly detectable tumor in the gastric region, a tumor which disappears if the abscess ruptures.

Etiology.—Phlegmonous gastritis is a result of infectious processes, among which have been puerperal fever and other forms of pyemia. It has been found associated with peritonitis and trauma. It has been met more frequently in men than in women. It sometimes occurs without discoverable cause in drunkards.

Symptoms and Diagnosis.—Epigastric pain and tenderness, general abdominal pain and tympany, vomiting, diarrhea, fever, delirium, dry tongue, small, frequent pulse, coma, collapse, and death—symptoms that closely resemble those of peritonitis, with which, as has been said, it is sometimes associated—are those met in phlegmonous gastritis. The vomited matter very rarely contains pus. It is plain, therefore, that these symptoms, associated with an infectious process, can only give rise to suspicion that the disease is present, since the same symptoms may be caused by peritonitis. Even the vomiting of pus is not diagnostic, because pus may arise from other sources between the mouth and stomach. The presence of a tumor which subsides after vomiting of pus furnishes better ground for suspicion, though vomited pus may also come from an abscess in the vicinity of the stomach which has ruptured into that organ.

Treatment.—This can only be symptomatic, as nothing can be done to avert a termination which is invariably fatal.

TRAUMATIC AND TOXIC GASTRITIS.

Definition.—An inflammation of the stomach caused by the ingestion of corrosive poisons, such as the strong mineral or organic acids, caustic alkalies, phosphorus, arsenic, corrosive sublimate, and the like.

Morbid Anatomy.—The appearance differs, according to the degree of irritation, and the character of the poison causing the condition. In extreme degrees, such as are produced by the strongest acids and alkalies, the mucous membrane is disintegrated, shreddy, and may be converted into a black eschar, the borders of which are lighted up with intense inflammation. In milder forms, such as are produced by phosphorus, arsenic, and strong alcohol, there are cloudy swelling and fatty degeneration of the gastric gland cells and vessel-walls, producing ulceration and hemorrhagic extravasation.

Symptoms.—These also vary with the degree of irritation, but there are always *intense burning pain, tenderness on pressure, thirst,* and *vomiting* of blood and even of fragments of mucous membrane. To these are added, in severe cases, *small, frequent pulse, cold sweat, and collapse.* Peritonitis often results from extension of the inflammation. If the patient does not perish promptly, *symptoms referable to the systematic action of the drug causing the poison,* supervene. When recovery takes place or death is long delayed, varying areas of mucous membrane may be replaced by *cicatricial tissue,* and there may be subsequent contraction and distortion.

Diagnosis.—This is based on a knowledge that the patient has swallowed a corrosive poison. In the absence of this knowledge the odor of the breath may suggest the cause, and evidences of corrosive action in the mouth and pharynx often disclose unfailing signs.

Prognosis.—This varies with the promptness of treatment and the degree of lesion. The gastritis caused by large doses of the powerful corrosive poisons is always fatal. The lesser degrees may be followed by recovery.

Treatment.—This consists, first, in the use of prompt lavage and immediate use of the chemical antidote to the poison swallowed. (See chapter on Treatment of Poisons.) These should be followed by the free use of diluents and demulcents, of which the various mucilages and milk are examples. (See concluding section of book on the Treatment of Poisons.)

Membranous Gastritis.—This occurs secondarily to typhus or typhoid fever, small-pox, scarlet fever, pneumonia, and sometimes primarily in weak children. There is no way to recognize such condition during life. True diphtheria of the stomach may occur, and according to Adami may extend from the fauces without attacking the esophagus.

Mycotic Gastritis.—It is very doubtful how far fungi can cause inflammation of the stomach. The bacteria which flourish in the mouth are destroyed by the acid gastric juice, while the fungi that thrive in acid fluids, such as the yeast fungus, the penicilium, and the sarcina, are probably accidental results of the retention of the gastric contents beyond the natural time and are not harmful. The possibility of their producing noxious results cannot, however, be denied. Ulceration has even been ascribed to them. On the other hand, the larvæ of certain insects must also be acknowledged as possible causes of inflammation. True thrush has been found.

Syphilis of the stomach is rare, but has been described by Oberndorfer and by Brunner. It may give rise to abnormal shapes of the stomach and to perforation.

Tuberculosis it almost invariably associated with tuberculosis of the

[1]Prepared by precipitating solution of persulphate of iron by ammonia.

lungs in an advanced stage. The treatment of both these local conditions resolves itself into treatment of the infection.

Hair balls may form in the stomach by reason of the patient swallowing her own hair and that of any other individual. The diagnosis must depend upon discovery of the habit and removal of the mass by surgical means.

Gastric Neurosis.

Synonym.—*Nervous Dyspepsia; Gastric Neurasthenia.*

Frequently in nervous individuals severe gastric symptoms arise which are not due to any organic condition of the stomach, but are due wholly to disturbance of the nervous tone of the patient. These so-called gastric neuroses may be so marked in their gastric aspect that the underlying nervous condition of the patient is not observed. Or, it may be the expression of a nervous state which itself is the result of some physical disturbance, tuberculosis, chronic nephritis, locomotor ataxia, etc. Therefore, in the diagnosis of these conditions no examination of the patient should be considered complete which does not include every organ in the body.

HYPERCHLORHYDRIA.

Synonyms.—*Nervous Hypersecretion of Hydrochloric Acid.*

Definition.—Hyperchlorhydria, or hypersecretion of hydrochloric acid in the gastric juice, is a symptom of different morbid conditions of the stomach, notably ulcer and nervous dyspepsia. In a certain number of cases, however, being the chief symptom and apparently independent of any stimulus like the presence of food, it may be studied as an independent neurosis. In normal digestion the total acidity as represented by free and combined HCl may be put down at 1.5 to 2 parts per 1000, requiring 4 to 6 c.c. decinormal solution for neutralization, while in hyperchlorhydria it may reach 3 and 4 parts in 1000, requiring 8 to 10 c.c. decinormal solution to neutralize.

Eliminating the hyperchlorhydria of cholelithiasis, ulcer of the stomach, there remain two varieties:

1. Simple paroxysmal hyperchlorhydria, lasting for an hour or several days.

2. Continuous chronic hypersecretion, which takes place spontaneously during fasting, or, even though excited by food stimulus, continues after the latter has ceased to act. The latter variety is also called Reichmann's disease, after him who first described it.

Etiology.—Both forms of hyperchlorhydria are most frequent in neurasthenics and emotional persons, but occur also in connection with other neuropathies, such as migraine, chlorosis, and tabes.

Symptoms.—In *paroxysmal hyperchlorhydria* there are *pain* and *epigastric discomfort, eructations, heartburn, thirst, nausea,* and even *vomiting, headache,* most common two or three hours after eating. *Constipation* is the rule. The attacks may last for an hour, or may extend over several

days, terminating in vomiting; they may be ended by remedial measures, such as drinking large quantities of water, which dilutes the acids, or by saturation with albuminous food, with which it enters into combination. The *urine, because* of much ingestion of albuminous food, is apt to be highly charged with urea.

In the *continuous form* the same symptoms are present, but without intermission. The pain is even more severe, and is epecially prone to come on at night; there is a *capricious appetite,* which is often excessive. Where the appetite remains, pain may occur several hours after taking food. The *vomiting* is often copious, gaseous, may contain remnants of undigested starchy food, and is of *intensely* acid reaction. It is likely to take place several hours after a meal, also at night. The *urine is scanty* ant there is *constipation.* The patients gradually emaciate and become anemic, even though they may take a good deal of food.

Diagnosis.—A positive diagnosis of hyperchlorhydria can only be made through analysis of the gastric contents. This is done in the sixth hour after a test dinner, with a view to discovering the presence of an excess of hydrochloric acid. The same symptoms may, indeed, be caused by organic acids, while the hydrochloric acid is in normal amount. If the stomach is washed out in the evening and the next morning, no food being ingested in the meantime, the contents are expressed and found to contain an excess of hydrochloric acid, the condition is one of continuous hyperchlorhydria. Microscopic examination of the gastric contents may also aid in the diagnosis. Such examinations made one to one and a half hours after a test breakfast or three to four hours after a test dinner, will often reveal a large number of unaltered starch-corpuscles, instead of only a few as in normal digestion. Great care must be taken not to mistake gastric ulcer and gall-stones for a simple hyperchlorhydria. The diagnostic points will be considered under these two conditions.

Prognosis.—The prognosis of simple hyperchlorhydria is favorable; that of the continuous form is grave, the disease being incurable after a certain stage has been reached. It becomes, therefore, important to treat the simple form promptly and intelligently before it passes over into the continuous form.

Treatment, Hygienic.—Many individuals, the subject of hyperchlorhydria, pass their lives either because of poverty, wealth, overwork or idleness in an entirely artificial manner. Mill workers should be allowed ample time for lunch, which could be economically supplied at a small cost in or about the mill building. They should be taught not to bolt their food, to rest or take rational enjoyment in the evenings, not to remain until midnight in a crowded moving picture show. To sleep with an abundance of air in their rooms, winter and summer. The more wealthy should be warned that dancing until early hours, wine drinking, excitement of society are fertile sources of this condition. Alcoholism should be avoided.

Medicinal measures have two particular objects: (1) To neutralize the excessive acid sceretion, and (2) to restrain its formation.

The first indication is met in two ways:

(*a*) By saturating the acid by nitrogenous food.

(*b*) By the administration of alkalies.

(*a*) The former is fulfilled by the use of *meat and milk diet*. It has, however, its limits, because when the tendency to acid secretion exists, it is often maintained even after that present is combined with any albuminous food that may be in the stomach. Hence it is that the pain is felt some hours after a meal when the albumen is digested. (*b*) Since there is a limitation to the ingestion of meat its use must be supplemented by *antacids*, which further neutralize the effect of the acid. The alkali most frequently employed for this purpose is sodium bicarbonate, though calcined magnesia is in some respects better because of its greater saturating power. The sodium bicarbonate should be administered some time after meals, just before the time the pains are expected. It should be dissolved in water or milk, or put in capsules or cachets. The doses should be sufficient to counteract the acidity—*i. e.*, 10 to 20 grains (0.66 to 1.3 gm.) or more. The quantity of carbonic acid evolved sometimes distends the stomach uncomfortably. Smaller doses of *magnesium oxide* suffice, and it is surprising that its use is not more general. It has the disadvantage of being insoluble in water, but not only are smaller doses sufficient, but there is also absence of carbonic acid evolution. It is indicated especially where there is constipation.

Other alkalies may be used, such as the potassium salts. A mixture of potassium or sodium bromide with Spts. of aromatic ammonia often is of the greatest value, and the officinal *liquor potassæ* in 5 to 10 drops (0.2 to 1.6 c.c.) in milk may be used with benefit. The *benzoate of sodium* may be prescribed in 10 grain (0.66 gm.) doses where antisepsis is required or fermentation is present. *Lime-water* is also useful, but large doses are required, as its neutralizing power is small. One-half ounce to an ounce (15 to 30 c.c.) or more should be given. Lime dissolves more largely in saccharine solution than in pure water, and larger doses may thus be given in smaller bulk. Dilute alkaline mineral waters, such as Vichy, Vals or Contrexville, may be used during a meal. Lavage with nitrate of silver solution may also be used as directed on p. 358.

Of *medicines* other than those intended to meet the symptoms, arsenic, in the shape of Fowler's solution, is sometimes efficient. Long courses of it should be practised, but large doses are not often allowable because of the irritation excited by them. Silver nitrate may also be employed in doses of 1/4 grain (0.0165 gm.), in which dose it is sometimes sedative when given on an empty stomach or by lavage as suggested in gastritis.

(2) Constitutional treatment should be directed to the cause, if it can be ascertained, neurosis by nervines, chlorosis by iron and arsenic. Of course, it is better, if possible, to prevent the excessive secretion of the juice. For this purpose *sodium sulphate* has been recommended, more particularly in the shape of Carlsbad water. Or the sodium sulphate may be dissolved in Vichy, say 45 to 90 grains (3 to 6 gm.) in a glass. It is given in the morning before breakfast, or, if necessary, may be given before the other meals.

Diet.—While the medicinal treatment of hyperchlorhydria is in most cases indispensable, the diet is equally important. It has already been said that theoretically a meat and milk diet is indicated, because meat and milk consume in their digestion the excess of HCl. On the other hand, the starchy foods are but imperfectly digested. Some object to meat diet because of

its overstimulating effect on the acid secretion, and recommend vegetables instead. This is, however, fallacious, and experience sustains the verdict in favor of meat and a minimum of starchy foods. It should be finely cut and well masticated, while meat powder may be substituted. Milk should be the drink, though the alkaline mineral waters may be taken at meals. In extreme cases a pure meat diet, the meat raw or nearly so, finely minced and spread on bread, may be necessary. A meal may consist of about 3 1/2 ounces (100 gm.) of raw meat, a couple of thin slices of stale bread or zwieback, a little butter, and a glass of plain water or weak alkaline water, such as Vals or Vichy. Or an exclusive milk diet may be tried, in which event the milk should be well alkalized or peptonized. To these are added, as the case improves, raw meat, meat powder or meat juice and eggs, and later still starchy foods may be tentatively given, associated with diastasic malt.

In these cases overstimulation of the stomach, induced especially by alcohol, or by pepper, mustard, and other condiments, should be avoided. In like manner coarse food of any kind is contraindicated. On this account constipation is sometimes best treated by enemas, in order to avoid the administration of irritating medicines by the stomach.

The rest cure as originally suggested by S. Weir Mitchell often is a most efficient aid to the successful treatment of hyperchlorhydria.

It not infrequently happens that gastric analyses fail to find any excess of HCl notwithstanding other symptoms point to hyperchlorhydria. In such an event it may be supposed that a previous condition of hyperesthesia of the gastric mucous membrane is present. The treatment would be the same.

GASTRALGIA.

Definition.—A term applied to recurring attacks of gastric pain usually some hours after eating, of great severity without discoverable organic lesion or deranged function.

Etiology.—The disease is confined almost exclusively to women, but does occur occasionally in stalwart men. It is more frequent in weak, anemic women, and those subject to menstrual derangement, in brunettes rather than in blondes. It is especially frequent and severe about the menopause, but does not cease with it. When associated with excessive secretion of gastric juice, or hyperchlorhydria, gastric pain does not come into the category of gastralgia. It is usually independent of exciting cause, such as the taking of food, but it may be induced by food.

Symptoms.—The attack may come on suddenly or with gradually increasing severity first in the neighborhood of the ensiform cartilage, whence it radiates into the back and around the lower ribs. It is a *boring, burning pain* of extreme severity, sometimes causing fainting and collapse, relieved by pressure, such as is produced by boring the fist into the epigastrium or pressing it against some hard substance. On the other hand, it is sometimes excited by pressure. Its most striking feature, after its agonizing severity, is its *intermittent, paroxysmal* character. The pain is usually the sole symptom, but it may be associated with *nausea* and *vomiting* or with nervous symptoms, such as *globus hystericus* and *unnatural hunger*. The attack.

after a variable duration of from a few minutes to an hour or more, may subside gradually or suddenly without other symptoms, though sometimes with vomiting and eructations, at others with the discharge of a large quantity of pale urine. The interval between the attacks varies greatly. It may be a week or it may be months.

Diagnosis.—Essential gastralgia is to be differentiated from intercostal neuralgia and the so-called symptomatic gastralgia due to ulcer, to cancer, from the gastric crises of tabes, and from biliary and intestinal colic; also from the pain of peritoneal adhesions succeeding operation.

In *intercostal neuralgia* the pain is not so severe and the paroxysms are of longer duration, while careful examination will discover a tender spot in an intercostal situation as compared with an epigastric. In *ulcer of the stomach* there is not that total intermission or longer interval of total intermission characteristic of gastralgia, while the general health of the patient with ulcer is commonly more seriously affected. This is, however, not always so, as gastric ulcer may be associated with robustness of appearance. In gastric ulcer pressure increases the pain, while in gastralgia it tends to relieve it. *Carcinoma*, as contrasted with gastralgia, always visibly affects the general health. Careful examination will generally discover a different seat of the pain in *biliary colic*, while the almost invariable presence of jaundice settles the question. In a well-established case of *tabes* there need be no difficulty in diagnosis, but in cases where the diagnosis is not well established there may be much doubt. The history of attacks in comparatively early life and thence throughout life point to gastralgia. Peritoneal adhesions should always be suspected when the pain succeeds abdominal section. Very rarely the pain of appendicitis may resemble that of gastralgia. The greatest care must be taken here as in other gastric neuroses that the organs above spoken of may not be overlooked. Many cases of hyperacidity and gastralgia are in reality gastric ulcer and demand treatment directed to this condition.

Prognosis.—True gastralgia never destroys life, but the attacks may continue to recur at intervals throughout it.

Treatment.—The severest attacks of gastralgia can only be relieved by the use of *morphin*, which is best given hypodermically in the smallest doses which will suffice. Exceeding care must, however, be exercised to avoid a morphin habit. In milder cases chloroform may answer the purpose, or a combination long prescribed in the clinics of the University of Pennsylvania and deservedly popular is, equal parts of chloroform, compound tincture of cardamom, aromatic spirit of ammonia, and brandy, of which a teaspoonful may be given every half hour or 15 minutes until relief comes. If needed, a few drops of deodorized tincture of opium may be added to each dose to increase the anodyne effect.

Anemia should be treated with iron and arsenic, and a change of scene is often beneficial, while sea-bathing is a form of hygiene which is sometimes especially useful. The bowels should receive careful attention. If neurasthenia or hysteria be present, the rest cure, associated with massage, as described under the appropriate section, is often an efficient cure. Gastralgia may be benefited by lavage with nitrate of silver solution as directed on p. 358.

ANOREXIA NERVOSA.

This term is applied to a condition in which absolute loss of appetite is the chief and characteristic symptom. Associated with this are, naturally, great debility, shortness of breath, dizziness, constipation, and sometimes headache; rarely, also, vomiting; sooner or later, emaciation. In women, in whom the symptoms usually occur, there is cessation of the catamenia. The name was suggested by Sir William Gull.

Prognosis.—This is favorable, cases being rarely, if ever, fatal.

Treatment.—The usual tonic measures are likely to fail to excite appetite in these cases, and nourishment must often be given either by the rectum or by forced feeding. The latter is done as follows: A short rubber tube, long enough to reach just below the cricoid cartilage, is introduced as directed on page 342. A bottle or funnel should be attached, and from this liquid nourishment is slowly introduced. This may be milk, plain or peptonized, broths or eggs. Estimating that 3 1/2 ounces (100 gm.) of albumin, 5 ounces (150 gm.) of fat, and 10 ounces (300 gm.) of carbohydrates are a sufficient amount per diem, Wiessner recommends 1 quart (1 liter) of milk, 2 ounces (60 gm.) of butter, 6 eggs, and 3 1/2 ounces (100 gm.) of sugar to be mixed and warmed while stirring. One-third of this amount is introduced three times daily. The food is usually easily digested, for it is not the digestion which is at fault, but the appetite, and the patient, encouraged by the result of forced feeding, is stimulated to eat for herself.

NERVOUS VOMITING.

Definition.—A form of vomiting resulting from direct or reflex irritation of the centers presiding over vomiting, and independent of anatomical lesion in the stomach. Like other gastric neuroses it is probably an expression of a general irritable condition of the gastric nerves—a manifestation of a general neurasthenia. It has been suggested that the exciting cause is some irritating leukomain of unknown nature.

Symptoms.—Especially characteristic of nervous vomiting are the *absence of nausea*, the *suddenness* of the act of *vomiting*, and the absence of the straining. More rarely there is nausea. The *appetite* is good and the vomiting generally follows a meal, but it may also occur at irregular intervals. In the absence of organic nervous disease the patient may be well nourished. There may also be *constipation*, *headache*, *dizziness*, *epigastric pulsation*, and gnawing sensation in the stomach. Intense *acidity of the vomited matter* may be present. To this condition Rosenbach has applied the term *nervous gastroxynsis*. In one of his cases the HCl reached four per cent. In the typical form, however, the vomitus is not abnormally acid, and in this respect it differs from acid dyspepsia and Reichmann's disease. The duration of the vomiting varies. It may be a single act or it may last for 24 hours.

Diagnosis.—This is based, in the first place, on the exclusion of those organic diseases of the stomach which cause vomiting, and, in the second place, on the presence of any one of the affections named as possible causes.

Prognosis.—Except when associated with organic nervous disease, this is ultimately favorable. George M. Garland[1] reported a fatal case of ap-

[1] Garland, G. M., "Trans. of the Assoc. of Am. Physicians," vol. iv., 1889.

parently pure nervous vomiting. At autopsy the mucous membrane of the stomach was found thin, and reddened on its inner surface with minute hemorrhagic points. There was slight interstitial nephritis too insignificant to have any effect, and the gastric changes were probably secondary, so that the case may be regarded as purely neurotic.

Treatment.—When vomiting is the result of disease of the nervous system the fundamental treatment must be that of the disease itself. Temporary relief may be afforded such cases by measures which make a profound nervous impression. Such, pre-eminently, is the *blister* to the epigastrium. The suddenness and irregularity of the vomiting make it almost impossible to provide against a given event. So that *ice*, internal or external, *sinapisms dry cupping*, and similar measures efficient in continuous vomiting or in vomiting preceded by nausea are scarcely available. When, however, circumstances permit their employment, they should be used.

Nerve sedatives, including the bromids and valerian, may be used, but hypodermic injections of *morphin* are often necessary, and are usually very efficient. Hypodermics should be used at the rarest instances. *Rectal alimentation* should be employed when the vomiting is obstinate.

GASTROSUCCORRHEA—REICHMAN.

HYPERSECRETION.

A continuous or intermittent flow of gastric juice causing vomiting and pain.

Symptoms.—Periodical seizures. In the midst of health the patient is seized with headache, nausea, restlessness and vomiting of large quantities of gastric juice. The patient is ill for several days. The clear fluid vomited is highly acid. The patient may then be in entire health for a long period, when he is as suddenly seized with a second attack.

Diagnosis.—Diagnosis is made by the periodical character of the attack, and the exclusion of organic conditions, such as gastric ulcer and tabes.

Treatment.—The patient must be kept strictly quiet and given nothing to eat or drink for 24 hours. Extreme thirst may be allayed by continuous enteroclysis after Murphy method; after that food very gradually resumed.

ACHYLIA GASTRICA—ANACIDITY.

This condition may be a true neurosis with the symptom of lack of normal acidity of the gastric juice, as contrasted with the cases common in cancer of the stomach, and other cases of total absence of gastric juice, common in atrophy of the mucous membrane of the stomach.

Symptoms.—The symptoms vary in intensity and variety. Some have stomach symptoms with loss of appetite, fullness and pain after eating, headache and constipation. Other cases have no stomach symptoms, but intestinal symptoms, diarrhea, alternating with constipation, thirst, weakness and loss of strength. The course is protracted.

Diagnosis.—The diagnosis can only be arrived at by repeated examination of the stomach contents and finding a very small quantity of contents with persistent lack of the normal ingredients of the gastric juice.

It must be separated from gastric cancer, and the achylia which accompanies pernicious anemia.

Treatment.—Most cases, according to Einhorn, do best with little medicine, electricity and bitter tonics and a diet. Well-chosen food is a necessity. Vegetable food is well borne, meats should be given in small quantities.

AEROPHAGIA.

Is characterized by the eructation of large quantities of air. The air is usually swallowed, or sucked in, as in a horse. It is frequent in hysterical girls.

Treatment.—The treatment is that of the hysteria.

RUMINATION.

Rumination is a nervous condition characterized by the regurgitation of food and rechewing it. This is an extremely difficult condition to treat.

CARDIOSPASM.

This is a spasm of the cardiac orifice of the stomach frequently followed by dilatation of the esophagus. Plumner believes the spasm is the primary condition. He described three stages:

1. A spastic cardia with the esophagus able to force food through it.

2. Spastic cardia with the esophagus unable to force food through it, and the food is regurgitated. Here the muscular tissue of the esophagus is hypertrophied.

3. Spasm of the cardia with dilatation of the esophagus, retention of food in the esophagus, and regurgitation at irregular intervals.

Symptoms.—A sudden spasm felt along the line of the esophagus, frequently low down. Soon this sensation is accompanied by immediate regurgitation of food. In the third stage there is the knowledge of inability to swallow food. Liquid food may slowly pass, but solid food is retained.

Diagnosis.—A stomach tube can rarely be passed in these cases, but a solid sound passes easily. When there is dilatation of the esophagus, food may be withdrawn from the dilatation. An X-ray taken after swallowing bismuth shows the bismuth retained in the esophagus. Special sounds have been devised, but the characteristic symptoms with the above findings, will make the diagnosis.

Treatment.—The relief of most cases, and the entire cure of many is accomplished by means of a special dilating apparatus, consisting of a specially devised oblong rubber balloon, connected with a long rubber tube, in turn connected with a water tap or pump, a water gauge is attached to regulate the pressure. The pressure in the tubing is very slowly raised. The pain expressed by the patient is usually used as a guide, but the pressure must paralyze the sphincter. If this cannot be done by this instrument, graded sizes of dilators are used until the object is attained.

Plummer reports 40 cases. The number of treatments varied from two to ten. There were eleven relapses, but no second recurrences.

The general strength of the patient must be attended to, but usually the patient rapidly improves with the ability to swallow.

PYLOROSPASM.

A contraction of the pylorus, without organic lesion. This condition is common in hyperchlorhydria of ulcer. There is pain, and the gastric peristalsis can frequently be seen.

It is difficult to differentiate from organic stenosis of the pylorus. One case under our care, the pylorus could be felt to thicken and become palpable under the fingers. This proved to be due to a gastric ulcer.

GASTRIC AND DUODENAL ULCERS.

SYNONYMS.—*Ulcus ventriculi pepticum; Peptic Ulcer; Simple or Round Ulcer.*

Definition.—A loss of substance in the mucous membrane of the stomach, of more or less progressive character, and frequently extending through the entire thickness of the stomach wall. As there is no practical difference in the clinical course and treatment of gastric and duodenal ulcers, these conditions are consided together. Three-fifths of all gastric and duodenal ulcers are situated in the duodenum.

Etiology.—There is probably more than one mode of origin of gastric ulcer. It may have its origin in *mechanical injury* associated with *feeble nutrition*, which permits the gastric juice to digest out the mucous membrane to various depths, resulting in the formation of an ulcer. Such mechanical injury may be either internal or external. Intestinal injuries are due to corrosive substances, hot foods and hard articles of food. External injuries may be due to trauma, a blow, or to the pressure exerted in the course of one's occupation, such as shoemaking, washing, tailoring, and the like, in which pursuits the costal cartilages are pressed against the stomach. *Anemia*, *chlorosis*, *heart disease*, *Bright's disease*, and the like are frequent precursors of the condition. *Overdistention* of the stomach, it is claimed, may be a predisposing cause by interfering with its proper nutrition and thus favoring the action of the gastric juice.

Thrombosis and *embolism* have been held responsible for a certain number of cases of ulcer since Virchow called attention to such causes. The stasis of circulation thus resulting affords favorable foci for the solvent action of the gastric juice, and certainly no theory explains so satisfactorily the crater shape of many gastric ulcers. Böttcher ascribed ulcer of the stomach to *micrococci*, numbers of which have been found by him in the margins of gastric ulcers. The well-known clinical fact that the gastric juice in ulcer of the stomach exhibits sometimes intense acidity, while traumatic ulcers of the stomach produced under ordinary circumstances tend to heal promptly has led to the suggestion that undue *acidity* plays an important rôle in the causation of ulcer. Increased acidity is not, however, always associated with these conditions. Mayo believes the three great etiological factors are: Excess of hydrochloric acid, traumatism inflicted in the grinding of the pyloric end of the stomach and the upper part of the duodenum. A third essential factor of general character is anemia.

The statements of authors as to the frequency of ulcer of the stomach vary greatly. Thus, Ewald says five per cent. of Germans have ulcer.

Truly, the disease is not nearly so common in America. Yet the discovery at autopsies of unexpected ulceration goes to show that it may be more frequent than is supposed. Fiedler found ulcer or its scar in 20 per cent. of autopsies in women and 1.5 per cent. in men. It is evident, therefore, that women are much more frequent victims than men. While both the very young and the very old are commonly exempt, the period being between 17 and 25, gastric ulcer has been found in infants and in adults as old as 60. In women gastric ulcer usually occurs between the ages of 20 and 30; in men, between 30 and 40.

Duodenal ulcer, on the other hand, is more common in males, in the proportion of 178 to 41, in the combined statistics of Kraus, Chvostek, Lebert, Trier, and William Osler. The last-named observer found it once in a boy of 12. In Mayo's series of 200 cases 77 were male, 23 female—while in true gastric ulcer there were 52 men and 48 women.

Morbid Anatomy.—Gastric ulcer must be distinguished from post-mortem softening or digestion, which is found after death in stomachs in which gastric juice happens to be present at the moment of death. In this there may be erosion of the superficial mucosa, but nothing comparable to ulcer. The seat of postmortem softening is more commonly the fundus and posterior surface, where the gastric juice naturally collects.

The typical gastric ulcer is circular in outline, often with sloping, clean-cut sides, furnishing a crater or truncated cone shape, with the broad end looking toward the cavity of the stomach, a shape corresponding to that of an infarcted area due to embolism or thrombosis. The term "punched out" has long been applied to characterize the appearance of a gastric ulcer. The sides are not always, however, smooth, being sometimes uneven or "terraced." Very rarely ulcer maybe multiple. It is far more frequent, on the posterior wall of the stomach near the lesser curvature. W. H. Welch's extensive studies of hospital records furnish the total of 783 cases, of which 288 or 37 per cent. were in the lesser curvature, 225 or 29 per cent. on the posterior wall, 95 or 12 per cent. at the pylorus, 69 or 9 per cent. on the anterior wall, 50 or 6.75 per cent. at the cardia, 29 or 4 per cent. at the fundus, and 27 in the greater curvature. In Mayo's experience 90 per cent. of all ulcers which exist in the stomach proper are in the pyloric end and at least one-half of gastric and duodenal ulcers are in the upper duodenum.

The floor of the ulcer is usually the muscular coat, but it may be the serous coat, which is sometimes perforated so that the floor may be formed by an adjacent organ to which the stomach has been glued by adhesive inflammation. The ulcer is usually small, not larger than a pea, but it may be 10 or even 15 cm. (4 to 6 inches) in diameter, covering the whole lesser curvature and part of the anterior and posterior walls. Ulcers may heal, leaving a cicatrix, which, if large, causes contraction and deformity, distorting the organ even to an *hour-glass shape* and producing stenosis of the pylorus. It is not unusual to find healed ulcers at autopsies. Or *the ulcer may perforate*, causing fatal peritonitis when in the anterior wall; or, if apposed to neighboring organs, it may cause a local peritonitis which results in adhesion of the various parts, sometimes perforating into the lesser peritoneal cavity resulting as an abscess which may rupture into any of the neighboring organs. The pericardium, the mediastinum and left ventricle, the spleen, the head of

the pancreas, the left lobe of the liver, the gall-bladder, the omental tissues, the pleura, and even the lungs have been invaded, while fistulous communications have been formed with the duodenum, the colon, and even the external air in the neighborhood of the umbilicus.

It is not unusual to see at the bottom of an ulcer an eroded blood-vessel from which there has been a fatal hemorrhage. The vessels invaded may be the gastric artery of the lesser curvature, or the splenic artery in the posterior wall; or, in the case of a duodenal ulcer, the pancreatico-duodenal artery; or it may be the hepatic artery, and even the portal vein. Small aneurysms have been found in the floor of an ulcer.

Gastric ulcer may be multiple, it is said, as often as once in every five cases. Osler records a case in which there were five ulcers and refers to a case, reported by Berthold, in which there were 34.

Symptoms.—Chronicity and periodicity of the attacks are quite characteristic. There is usually a history of months or years of periods of "stomach trouble." The attacks of characteristic pain and distress come on suddenly, last for several days or months then a sudden disappearance of the pain and apparently perfect health for varying periods of time. The milder, less distinctive symptoms of indigestion, viz., a sense of fullness in the epigastrium, acid eructations, loss of appetite and the like may be present in gastric ulcer but the most prominent symptoms are *pain*, *tenderness*, *vomiting*, *hemorrhage*, and sometimes a tumor, but none of these is invariably present.

Pain, with tenderness, is the most constant symptom. According to Mayo, if the ulcer is in the stomach proper the pain is usually most acute from the median line to the left. If the ulcer is in the duodenum proper, the pain and tenderness come on several hours after meals and extend from the midline to the right. According to Graham, in Mayo's clinic, taking of food does not give immediate pain in any form of peptic ulcer but relieves it. The longer pain is relieved by food the farther down the ulcer is found. The pain comes on in definite periods of attacks two to four hours after meals.

The patient will sometimes bend over, pressing his fist into the epigastrium or lean over the back of a chair to secure relief. It may be excited by spasm or by overdistention by gas.

Tenderness on pressure is a characteristic symptom, apart from the paroxysms of pain; and in order to guard against it, the patient may wear the waistband low. Boas has devised an instrument by which circumscribed pressure may be conveniently induced and diagnosis facilitated. The *tendon point is more frequently an inch or two above the umbilicus*. In cases of ulcer of long standing palpation may recognize a *tumor*, the result of inflammatory thickening in the vicinity.

Vomiting is not so frequent a symptom. When present, it occurs usually, about the same time as the pain.

Hemorrhage—from the stomach or intestines—is a most valuable sign of gastric ulcer when it is accompanied or preceded by symptoms of gastric distress. Given a copious hemorrhage of pure red blood from the stomach, with the symptoms described, it can scarcely be due to any other cause. A single large hemorrhage may occur from other causes. In a few instances in ulcer the hemorrhage is small, when, of course, the diagnosis becomes more

difficult. When the hemorrhage is large, blood quite black is found also in the stools. Indeed, sometimes the presence of blood in the stools is the first intimation of gastric hemorrhage. Especially is this the case when the ulcer is duodenal. Hemorrhages from ulcer are also often recurrent, and result at times in intense anemia of the subject. They are not rarely fatal, more frequently syncopal, bringing their subjects to the verge of the grave, from which there are often also surprising recoveries. A hematemesis of ten pounds (4 1/2 kilos) is said to have been followed by recovery. Occult blood in the stools is a valuable sign to be sought for by the various chemical tests, but it must be remembered that there are many other sources of such hemorrhage than an ulcer in the stomach. Hemorrhoids, traumatism and ulceration of any portion of the gastrointestinal tract must be remembered.

Perforation is a rare accident in ulcer of the stomach. It is variously stated at from 6 to 18 per cent. of all cases. Its characteristic symptoms are sudden and violent pain, extreme tenderness, rigid contraction of the abdominal muscles, profound shock, shallow breathing, and absence of the normal hepatic dullness—in a word, the symptoms of peritonitis, followed by those of shock. Perforation is much more frequent when the ulcer is in the anterior wall. Thus, in 13 cases reported by A. B. Mitchell to the "British Medical Journal," March 10, 1890, all were in the anterior wall.

Persons with gastric ulcer lose in weight and become gradually *anemic*, quite independent of hemorrhage, as is evidenced by a blood count. This may be due to the fact that they refrain from taking food because they fear its consequences. From the combined effect of this and actual loss of blood results at times an anemia which is only second to that of pernicious anemia. The hemoglobin is correspondingly reduced.

Chemical examination of the *stomach-contents* after a test-meal frequently shows an increase of HCl, in fact the symptoms are, at least in part, those of hyperchlorhydria, in some part of the course in almost every case of gastric ulcers. Mayo clinic statistics are valuable in showing the condition of the stomach contents in cases which came to operation, 250 cases of gastric and duodenal ulcers. Free hydrochloric acid present 237 times. Below normal, 23 cases; normal 102 cases, above normal in 112 cases. Exceptionally hyperacidity is absent, possibly due to associated chronic, gastric catarrh.

Finally, it is to be remembered of gastric ulcer that it is often *latent* throughout, quite without symptoms during life, and recognized for the first time at necropsy, when, also, as already stated, healed ulcers are sometimes found, or the first intimation of an ulcer may be a perforation.

Course and Termination.—The course of ulcer is usually slow, sometimes very protracted. A few cases are acute and rapidly fatal. The symptoms of gastric ulcer quite frequently disappear, and after a time, even considerable time, recur giving rise to the so-called recurrence. This very recurrence as stated above is one of the characteristics of the disease.

Hour-glass contraction is one of the terminations of gastric ulcer when a large ulcer in the middle belt of the stomach has healed. It is best recognized by the Roentgen ray, the stomach being previously partially filled with a mixture of bismuth and water introduced after a meal. But its existence may be suspected in the presence of the following signs named

by Moynihan: 1. In washing out the stomach a part of the fluid may be lost. 2. If the stomach is washed clean a sudden reappearance of gastric contents may take place. 3. When the stomach has apparently been emptied a splashing sound may be elicited by palpation of the pylôric end (paradoxical dilatation). 4. After distending the stomach a change in the situation of the distention tumor may be seen. 5. A gushing, bubbling or sizzling sound heard on dilatations by CO_2 may be heard at a point distant from the pyloris. 6. In some cases when both parts are dilated, two tumors separated by a notch or sulcus may be seen and felt.

Diagnosis.—Is frequently difficult. Given the characteristic symptoms of paroxysms of pain, relieved by eating and occurring several hours after food, and these symptoms coming on in periods after intervals of good health, particularly in the fall and spring, the diagnosis is extremely likely. Moynihan says the anamnesis is everything in making a diagnosis. This chronicity and periodicity is almost characteristic when the pain comes long after eating and is relieved by food.

Gall-stone colic and appendicitis may both give rise to a chronic periodical pain accompanied or preceded by the symptoms of indigestion accompanying gastric ulcer. Hyperacidity and pylorospasm may also closely resemble the attacks of gastric ulcer, but in all these conditions, there is lacking the machine-like periodicity, the occurrence in spells and the complete relief by food. It is true the distinction becomes less marked as the ulcer approaches the cardia in position.

In *gastralgia*, as in ulcer, hydrochloric acid may be increased, and the question often becomes a most difficult one to settle. In gastralgia, however, the general health of the patient is less severely affected, there is less chlorosis or menstrual derangement, and the pain has a less definite relation to taking food—while in ulcer the symptoms of dyspepsia are more constant. Above all, in ulcer there is tenderness on pressure between the attacks of pain, a symptom absent from gastralgia, while pressure always relieves the pain of the latter. Indeed, in gastralgia dyspeptic symptoms between the attacks are generally absent. We may look for assistance from the standpoint of etiology. Given the causes of ulcer, especially valvular heart disease with possible embolism, the vomiting which produces thrombosis, the occupations which favor gastric ulcer, their import should be recognized. Gastralgia occurs in neurotic individuals—those subject to hysteria and uterine disease.

In *tabes* the gastric crises are almost identical with the severe gastralgic attacks of ulcer. But in tabes the appearance of gold health is preserved, while it is not long before the distinctive symptoms of the disease show themselves, if they are not already present—viz., lightning pains, ocular symptoms, and absence of knee-jerks.

In rare cases *intercostal neuritis* may be mistaken for ulcer, if there be pain in the epigastrium associated with accidental dyspeptic symptoms. But in this affection painful points will also be found in the course of the affected nerve.

From *cancer* of the stomach ulcer sometimes is distinguished with difficulty in the absence of the more distinctive symptoms of the former disease. Heretofore much reliance has been placed on the absence or extreme di-

minution of free hydrochloric acid in cancer as contrasted with its excess in ulcer. It has, however, happened that the association of chronic catarrhal gastritis with ulcer has caused a relative diminution of HCl. The researches of Boas have added a very much more reliable diagnostic sign in the presence of lactic acid in cancer and its constant absence in ulcer. Other facts to be weighed in the balance as to the existence of cancer are a palpable tumor, the greater age of the patient, with rare exceptions over 30, the extreme emaciation and cachectic appearance, and the intermittent vomiting of large quantities of accumulated ingesta, sometimes of blood mixed with mucus, or blood presenting the "coffee-grounds" character as contrasted with the bright clear blood of ulcer. The presence of Oppler Boas bacillus in the gastric contents.

The prognosis depends largely upon the type of ulcer and the treatment. Acute ulcers, those giving symptoms in the very beginning of the lesion are amenable to the medical treatment described below. Latent ulcers may end in perforation without premonitory symptoms. Chronic ulcers rarely end in complete recovery without surgical interference, though long periods of absence of symptoms occur. Perforation is almost universally fatal without operation. Hemorrhage is a bad prognostic sign unless the case be treated surgically after recovery from the hemorrhage.

Prophylaxis.—Anemias should be corrected. All cases of chronic nephritis should be properly treated. Patients, especially young female mill workers, should be taught the gospel of proper hygiene, regular meals, proper food, fresh air, rest and exercise.

Irritating food should be avoided that the mass which grinds the pylorus may be as little irritating as possible.

Treatment.—The treatment of ulcer of the stomach or duodenum resolves itself into medical and surgical methods. The last word has not been said as to which cases must be operated. It is certain that many ulcers heal either spontaneously or as the result of treatment. The following advice, however, seems correct. Cases to be treated *medically* are the acute ones. When a case presents itself with acute symptoms, pain relieved by eating, tenderness, vomiting with stomach contents suggesting the condition, the first indication is REST. Rest both for the stomach itself and for the entire organism. The patient should be still in bed, take no food for eight to ten days, be given rectal enemata of eight ounces of peptonized milk every eight hours. While fasting they may have a small amount of water, and should be given bismuth subcarbonate or subnitrate in 1 gram (15 grain) doses every four hours. After eight or ten days food may be resumed; first one-ounce doses of milk, either peptonized or with lime water, and gradually increased until on the third day of food taking they get three or four ounces every three hours; then soft toast, potatoes, chicken, etc., may be slowly added. All the time bismuth may be taken.

Some cases do well with lavage of nitrate of silver 1 to 1000 during the entire time. Some do not insist upon total fasting but give small amounts of milk during the entire time. Cases thus treated usually speedily lose all their symptoms, they either remain permanently well or relapse. Once a diagnosis of gastric ulcer is made a patient should be given this form of treatment or some modification of it or the Lenhartz treatment detailed

below. If the case relapses in a short or long time a retrial of the treatment should be given, but in our opinion the case should be turned over to the surgeon if more than one relapse occurs.

Surgical treatment is demanded in all relapsing cases, in all cases of a chronic course whether relapsing or not. Leaving out of consideration the complications, the well-proven fact that 50 per cent. of carcinomata of the stomach have their origin in an ulcer whose base gives ample justification for the employment of a skillful surgeon when a simple chronic ulcer is considered. All cases which have had one or more hemorrhages should be operated after recovery from the hemorrhage, it being remembered that certain cases in young girls have bleeding from points in the mucous membrane which cannot be discovered.

All cases of obstruction of the pylorus, hour-glass stomach, demand operation. All chronic and acute perforations must be operated on at the earliest possible moment.

Care must be taken that a surgeon of skill and experience be employed.

According to the Mayo clinics, decided food remnants in the stomach contents of ulcer cases, demand operation. In the Mayo clinic a full meal is given the night before, the contents are withdrawn. In the morning an Ewald test meal is given.

During hemorrhage rectal alimentation should be relied upon. For this purpose peptonized milk is also the best nutrient. Great care should be exercised in the use of enemas not to exhaust the toleration of the bowel. To this end they should be given at first tentatively, never oftener than once in eight hours, and should not exceed at first at most, six ounces. This quantity if well borne may be increased to eight ounces. The various meat peptones, bouillon or beef juice may be substituted for or alternated with peptonized milk; or an egg may be beaten up with milk, though such addition is not often necessary. A nutrient injection which has given great satisfaction at the Hospital of the University of Pennsylvania consists of four ounces of milk (130 c.c.), to which are added two eggs, a pinch of salt and 3 drops of laudanum, the whole being predigested with pancreatin. The enema should be given very slowly through a long rectal tube, the patient having the hips elevated and the position maintained for an hour after the injection. In this way patients may be nourished for weeks with peptonized food, but it is rarely necessary to continue the rectal alimentation for more than a week or ten days. As the hemorrhage and vomiting cease the stomach may be tested, first with small amounts of milk or albumen water, gradually increased; and for a time the two methods may be pursued jointly, feeding by the mouth being increased, while that by the rectum is gradually withdrawn. Plain milk and beef-juice may be substituted for peptonized milk, and various thin gruels made with flour may be used as a change is demanded.

The hemorrhage requires also to be met by remedies. For the present all astringent remedies have given place to suprarenal extract or its active principle adrenalin, of which one dram (5 c.c.) may be given at one dose by the mouth. It has no effect on the blood pressure unless it is injected into the blood thus large doses may be used.

The Lenhartz Treatment of Gastric Ulcer.—This treatment, to which

especial attention has been called in this country by Samuel W. Lambert,[1] has for its object first to furnish nourishment and improve the patients' general condition; to continue to nourish them by feeding by the stomach; to use foods rich in albumen, in small amounts at short intervals, of one hour for the first ten days from 7 A. M. to 9 P. M., and insisting on slow eating, best accomplished by feeding in teaspoonful amounts; to insist on a three or four weeks' rest cure in bed. Other medical procedures are allowed if indicated; for example, an ice-bag to the epigastrium and bismuth subnitrate internally for hemorrhage; enteroclysis for the effects of hemorrhage, and iron and arsenic for the anemia. The course of treatment covers two weeks' time and includes the following articles of diet: fresh milk, iced; whole raw egg beaten up and iced. Both the milk and the egg are prepared in a covered glass surrounded with cracked ice; the feeding spoon is also kept iced. Changes suggested by Lambert are mixing of the eggs and milk and feeding the mixtures instead of hourly alteration; the addition of granulated sugar after the third day.

The following table from Lenhartz, modified by Lambert, gives the details and the method of feeding:

LENHARTZ TREATMENT OF GASTRIC ULCER.

Day	Eggs	Milk	Sugar	Scraped beef
1	2 drams each dose, total, 2 eggs.	4 drams each dose, total, 6 ounces.		
2	3 drams per dose, total, 3 eggs.	6 drams per dose, total, 10 ounces.		
3	½ oz. per dose, total, 4 eggs.	1 ounce per dose, total 13 ounces.	20 grams added to eggs.	
4	5 drams per dose, total, 5 eggs.	1½ ounces per dose, total 1 pint.	20 grams added to eggs.	
5	6 drams per dose, total, 6 eggs.	14 drams per dose, total 19 ounces.	30 grams.	
6	7 drams per dose, total, 7 eggs.	2 ounces per dose, total, 22 ounces.	40 grams.	36 grams in 3 doses.
7	4 drams per dose, total, 4 eggs. also, 1 soft boiled egg every 4 hours, total, 4 eggs.	2 ounces per dose, total, 25 ounces.	40 grams.	70 grams with boiled rice 100 grams in 3 doses.
8	4 drams per dose, total, 4 eggs, also, 1 soft boiled egg every 4 hours, total, 4 eggs.	2½ ounces per dose, total 28 ounces.	40 grams.	Beef same.
9	4 drams per dose, total, 4 eggs, also, 1 soft boiled egg every 4 hours, total, 4 eggs.	3 ounces per dose, total 1 quart.		Beef same. Rice 200 grams, Zwieback 40 grams in two portions.
10	4 drams per dose, total, 4 eggs, also, 1 soft boiled egg every 4 hours, total, 4 eggs.	Add cooked chopped chicken 50 grams, also butter 20 grams.	40 grams.	Beef same. Rice 200 grams, Zwieback 40 grams in two portions.

11 to 14. Intervals of feeding made 2 hours. Milk given in 6 oz. = doses with ½ oz. of raw egg. Butter increased to 40 grams and various additions made as detailed above.

[1] The Lenhartz Treatment of Gastric Ulcer. "Am. Jour. of the Medical Sciences," January, 1908, p. 18.

The resulting chlorosis or anemia may be treated with *iron* and *arsenic*. Of the former, the neutral preparations are to be preferred; of the latter, Fowler's solution, because of the easy regulation of the dose. Large doses of iron should not be given, since the excess of such doses remains unabsorbed, astringing and irritating the alimentary canal. The tincture of the chlorid, so valuable usually, is especially contraindicated, because it increases the acidity of the gastric juice and thus favors the solution of the gastric wall.

CANCER OF THE STOMACH.

SYNONYMS—*Carcinoma ventriculi; Gastric Cancer.*

Etiology.—Little definite is known of the etiology of cancer. Heredity is an acknowledged factor, though it is less potent than is commonly supposed. W. H. Welch[1] was able to trace cancer, or at least a family history of cancer, in 242 out of 1744 cases, or 14 per cent. Dieulafoy found such a family history in 16 per cent. and Musser in 8 per cent. of cases of gastric cancer. There is some evidence to show that abuse of the stomach by eating and drinking may be influential in causing the disease, though it is not conclusive. The same has been claimed for the depressing emotions. As mentioned under ulcer, there is better reason to believe that ulcer is a predisposing cause, since autopsies have disclosed cancer developing in the floor of ulcers and in cicatrices. Fifty-four per cent. of cancers of stomach are found to have the base of an old ulcer as their starting place. Mention should be made of the fact that a parasitic origin of cancer is claimed by some, but the subject is altogether too unsettled to justify more than reference in a text-book.

Gastric cancer is a disease of mature life, three-fourths of all cases occurring between the 40th and 70th year. One of our patients was 32 when he first consulted us, and died just one year later. Adolf Strümpell has seen cases between 22 and 25. George Dock[2] reports three cases occurring in his own practice, where the patients were 20, 21, and 24 years of age, confirmed by autopsy, and Marc Mathieu published in 1884 a monograph, "Du cancer précose de l'estomac." The disease is slightly more frequent in men than in women.

Pathology and Morbid Anatomy.—After the uterus, the stomach is the organ most frequently attacked by cancer, a little more than one-fifth of all cases of *primary* cancer being found in this organ—according to Welch, 21.4 per cent., from an analysis of the very large number of 30,000 cases. It is far more common in the pyloric end and on the lesser curvature, 1300 cases collected by Welch being distributed as follows: pyloric region, 791; lesser curvature, 148; cardia, 104; posterior wall, 68; whole or greater part of the stomach, 61; multiple, 45; greater curvature, 34; anterior wall, 30; fundus, 19. Every variety of cancer is found in the stomach, in the following order of frequency:

1. Cylinder-celled epithelioma, most frequent at the pylorus.
2. Medullary or soft cancer, most frequent in the smaller curvature.

[1] "System of Medicine by American Authors," vol. ii., Philadelphia, 1886.
[2] "Transactions of the Association of American Physicians," vol. xii., 1897.

3. Scirrhus, at the pylorus and the smaller curvature, causing, especially, stenosis of the pyloric orifice.

4. Colloid, diffuse infiltration with a tendency to spread to the peritoneum and adjacent organs.

5. Melanotic.

6. Squamous epithelioma, near the cardia.

All the forms start from the gland cells of the mucous membrane.

The medullary variety is prone to ulcerate and to form extensive fungoid ulcerated surfaces, from which there may or may not be hemorrhage. It may be associated with scirrhus. While nodular outgrowths are usual, the cancerous tissue may infiltrate the walls, producing diffuse thickening.

Secondary cancer of the stomach is an occasional event: in 17 out of 37 cases, according to Welch, secondary to primary cancer of the breast. We have met one case succeeding epithelioma of the lip. Much more frequently primary cancer of the stomach is a cause of secondary cancer elsewhere, most often in the adjacent lymphatic glands, which were the secondary foci in 551 out of 1574 cases collected by Welch; the liver was involved secondarily 475 times; the peritoneum, omentum, and intestine, 357; pancreas, 122; pleura and lung, 98; spleen, 26; brain and meninges, 9; other localities, 92; among the latter is to be included adjacent integument, especially about the navel.

Marked changes in the size, shape, and position of the organ occur as a result. Most common is dilatation, sometimes due to pyloric obstruction. Medullary cancer, on the other hand, is apt to produce a reduction in the size of the stomach and its cavity. A reduction in size may attend obstruction at the cardiac orifice, because of disuse of the organ, while the esophagus itself may be dilated. The same effect may be produced by cancerous infiltration of the stomach walls, by which the capacity of the organ is greatly reduced—in one instance, a case of Fussell's,[1] to 10 ounces. Further reference will be made to extraordinary dislocation of parts of the organ in treating of symptoms. Adhesions may also form between the stomach and adjacent organs, and between it and the anterior abdominal wall. Peritonitis may occur; also perforation into an adjacent organ, as the transverse colon, and even the small intestine.

Symptoms.—The initial symptoms in almost every form of cancer of the stomach are those of indigestion, including *anorexia*, *eructations*, *vomiting*, *constipation*, *discomfort*, and *pain*, more rarely *acidity*. These are present for a variable time before a more serious condition is suspected. Occasionally paroxysmal pain in the epigastrium is the only symptom. Increase in the severity of symptoms despite the use of remedies, progressive *debility*, *emaciation*, and *cachexia* invite closer examination, which may or may not result in the discovery of a *tumor*. Before a tumor is recognized there is often tenderness, which follows sooner or later, if it does not precede tumor. Cachexia and wasting may also be present a long time before the tumor is discovered.

A *chemical examination* of the *gastric contents* after a test-meal may disclose the absence of free and combined hydrochloric acid or a minimum of it. The persistent presence of lactic acid in decided quantity, to which, as

[1] Report Philadelphia Pathological Society.

well as to the absence of hydrochloric acid, attention was originally called by von der Velden, is held by Boas to be confirmatory. As to hydrochloric acid, it must be remembered that it is also diminished in gastric catarrh, in atrophy of the mucous membrane, in amyloid degeneration, and even in nervous dyspepsia at times, while in rare instances it happens that hydrochloric acid is increased in cancer. The motor as well as the secretory and absorbing functions will be found impaired, undigested food being found long after the seven hours' limit. Such motor delay characterizes

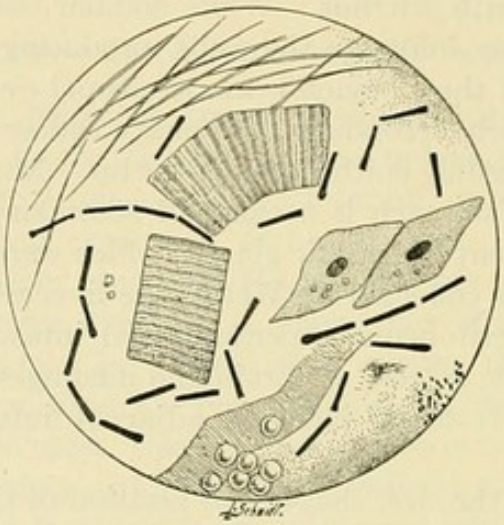


Fig. 101.—Oppler-Boas Bacillus, from Contents of a Carcinomatous Stomach—(*Hemmeter*).

more particularly the pyloric situation of cancer, with its resulting obstruction. The following are the finding of the stomach contents of 150 of Mayo's cases of cancer of the stomach.

Free Hydrochloric acid present in	70	cases
Average age	48	years
Duration of symptoms	41/2	years
Absent in	80	cases
Average age	54	years
Duration of symptoms	9	years

Free hydrochloric acid present without blood, lactic acid or food remnants in 24 cases.

Free hydrochloric acid present without blood, lactic acid, or food remnants in 46 cases, in 36 of these cases no palpable tumor present; 33 of these cases had palpable tumor present (blood alone 15 cases).

Blood present in 80 cases; blood and lactic acid 20 cases; blood and food remnants, 15 cases; blood, lactic acid, and food remnants, 30 cases.

Lactic acid present in 64 cases; lactic acid and food, 3 cases.

Food remnants present in 63 cases.

Food remnants present without blood or lactic acid in 15 cases.

Palpable tumor present in 79 cases.

From this summary it will be noted that a large number of cases of gastric cancer must be diagnosticated independently of the test meal findings, yet, on the other hand, there are a few cases in which the subjective symptoms are indefinite, and where the test meal throws the first light upon the real pathologic condition present.

The Oppler-Boas bacillus was first described by Oppler in 1895, as an unusually long and thread-like bacillus, nonmotile, found in the contents of carcinomatous stomachs.[1] The bacilli lie either end to end, in long thread-like chains, or at right angles to one another. The stain readily with anilin dyes. They prefer a medium containing lactic acid; indeed, Kauffmann ascribes to the bacillus the power of forming lactic acid from various kinds of sugar. Hydrochloric acid in any large proportion causes it to disappear. Schlesinger and Kauffmann declare the presence of large numbers of the bacilli in association with pyloric stenosis to be an indication of carcinoma, and their absence, associated with the absence of lactic acid, to be evidence against carcinoma. Riegel does not consider the organism pathognomonic of carcinoma, but very important in its diagnosis. Stockton says it is often present in carcinoma, and has not been found in other diseases of the stomach. The Oppler-Boas bacillus and sarcinæ do not coexist for any length of time in carcinomatous stomachs. The sarcina thrives in the presence of hydrochloric acid, and disappears with it, being replaced by the Oppler-Boas bacillus and lactic acid. Even when introduced into the stomach in cases of obstruction due to carcinoma, the sarcinæ disappeared in twenty-four hours, the Oppler-Boas bacillus seeming to replace them.

In evidence of the value of the Oppler-Boas bacillus in diagnosis of gastric carcinoma it may be said that Kauffmann[2] found it in 19 out of 20 cases, and in the one in which it was absent there was no lactic acid. John C. Hemmeter informs us that he found the bacillus in 52 out of 55 cases, that he regards it "an important diagnostic sign in carcinoma of the stomach, within limitations, and though it is by no means pathognomonic." He has found it in a case of benign pyloric stenosis, and also in such cases when HCl was still present. Ullman, of Buffalo, N. Y., found it in all of ten cases.

At a later stage periodic *vomiting* of large quantities of fluid containing the ingesta of hours and even days previous is a characteristic symptom, and a dilated stomach may now be easily demonstrated. The vomitus may also contain blood, and that peculiar mixture of blood and gastric juice which is called "coffee-grounds," vomit. If, owing to their disintegration, the microscope does not recognize blood disks, Teichmann's hemin crystals may be easily prepared. Occult blood may also be found in the stools and gastric contents. The vomited matter is sometimes very foul-smelling, as are also at times the eructations. Vomiting is by no means an invariable symptom, though even when there is no vomiting, nausea is commonly present. The absence of vomiting generally means that the cancer is not at the pylorus. It may be at the middle belt, at the fundus, or at the cardiac end. When at the latter point, there is almost always difficult and painful deglutition.

By this time the patient is emaciated, anemic, *cachectic*, with a peculiar yellowish, sallow, swollen appearance, and now a *tumor* is commonly easily recognized by palpation. Very interesting is the varying situation of the tumor, as well as at times its great mobility. The tumor of pyloric cancer

[1] Boas, "Specielle Diagnostik und Therapie der Magenkrankheiten." Oppler, "Deutsche medicinische Wochenschrift," 1895, No. 5.
[2] Kauffmann and Schlesinger, "Wiener klinische Rundschau," 1895, No. 5.

is commonly found near the normal situation of the pylorus, in the neighborhood of the umbilicus, a little to the right or left. At other times the weight of the tumor drags it out of the normal position, and it may be found lower down, toward the symphysis pubis. The tumor itself may be fixed in the position it assumes, or it may be freely movable. Its location is usually influenced by breathing. It rarely gives a positive dull note on percussion—rather a muffled note. In a certain number of cases no tumor can be detected throughout the whole course of the disease, it is said in 20 per cent. Especially is this the case when the disease is toward the cardiac end.

Toward the end of life *edema of the legs* and *ankles* appears, and an intensity of cachexia, which simulates pernicious anemia—in fact, even furnishes the blood changes characteristic of this affection—with extreme weakness and death. The *urine* is often scanty, and may give a decided reaction for indican. In a few cases a *febrile movement* makes its appearance with *chills* and *sweating* at intervals, probably due to intercurrent inflammation. To these symptoms are often added those of *secondary cancer*, especially of the liver, including enlargement of this organ and jaundice. The signs of secondary cancer elsewhere than in the liver should be sought. The duration of cases of gastric cancer is from one to two years; it may be less, especially, if the cancer is ingrafted on a pre-existing ulcer. Slow development is said to be characteristic of cases in younger persons.

Diagnosis.—This is generally easy if time and opportunity be allowed for the study of a given case, but one should not waste time in waiting for all of the following symptoms. *Ulcer* is perhaps the disease which furnishes most difficulty, especially as cancer may succeed it. On the other hand, the earliest symptoms of gastric cancer are also those of *gastric catarrh*, which in many cases is mistaken for cancer. The pain and the peculiar intermittent vomiting are the first distinctive signs, and while coffee-grounds vomit may occur whenever moderate quantities of blood are poured into the stomach and mixed with gastric juice, the causes other than cancer are rare. The copious hemorrhage of ulcer gives bright red blood. Bloody vomiting is by no means always present in cancer. To the symptoms described are soon added the emaciation and cachexia, and the palpable tumor more evident after the stomach has been emptied out by vomiting or washing. In the meantime, however, the gastric contents will have been examined, and furnish their quota of information, not pathognomonic, but contributory. Very rarely does it happen that in the vomitus or washings of the stomach we obtain particles of morbid growth whose examination will disclose the structure of cancer. Most frequently cancer for a long while is considered simply a chronic indigestion. It should always be remembered that a chronic indigestion may be cancer, gallstones, ulcer, or appendicitis.

There is not usually much difficulty in fixing the location of the tumor supposed to be in the stomach. If there is doubt, it may be eliminated in part or altogether by filling the stomach with air and noting the effect upon the tumor. Expert examination of the stomach by means of X-ray and bismuth will often lead to an early diagnosis. There will be seen loss of motility, notching of one or the other, curvature and other signs of in-

terference with the stomach wall. There is usually less interference with digestion in cancer of the gall-bladder, no mobility of the tumor, and often suppuration with incident fever.

The distinction of gastric from pancreatic cancer demands some consideration. The tumor may be in the same position, but in a large proportion of cases of cancer of the pancreas there is jaundice. The tumor of a pancreatic cancer is often inaccessible. In the latter there are also symptoms of indigestion, like those of gastric cancer, but there is often also diarrhea, and frequently the liquid stools contain oil. Such diarrhea may be checked for a time by ordinary remedies, but in a few days the liquid discharges seem to burst through a barrier which held them temporarily in check. The pancreatic tumor, if felt, is also more immovable.

Tumors of the liver and spleen are continuous with these organs, while the gastric tumor is generally easily distinguished from them by palpation or by an intervening tympanic area. A *cancer of the transverse colon* may occupy much the same position in the abdomen as one of the stomach, and be also quite movable. The filling of the colon and stomach with air may also be availed of in diagnosis. As the growth in the intestine increases, obstruction may result and the tumor increase by the accumulation of fecal matter behind the stenosed portion. A rare complication, increasing the difficulty in diagnosis, is adhesion between the bowel and stomach, restricting motion and possibly causing perforation, through which fecal matter may enter the stomach. Still more difficult, nay, even impossible, in most instances, is the distinction between *duodenal* and gastric cancer. The absence of hydrochloric acid would point to gastric cancer, though such absence, being due to atrophy of the gastric tubules caused by dilatation, may also occur in obstructive duodenal cancer. The acid might also be neutralized by regurgitated bile, regurgitation being favored by the stenosis of the gut. The presence of jaundice would point to duodenal cancer.

Gastric tumors may be confused with *omental tumors*, which may also cause dyspeptic symptoms. But the omental tumor is usually a more nodular, uneven tumor, and is sooner or later associated with peritoneal effusion.

Moreover, every tumor of the stomach is not a cancerous tumor, although most of them are. A thickened pylorus may be associated with gastric ulcer. Such a circumscribed thickening and induration are always possible. We may have the same pyloric stenosis and secondary dilatation. Similar *noncancerous thickening* may even occur without ulcer. Other forms of morbid growths, such as fibroma, sarcoma, and the like, are too rare to demand notice from the clinical standpoint.

Finally, the gastric tumor is not always demonstrable, and may not be throughout the whole course of its existence. It is said to be absent in about 20 per cent. of cases. Then the diagnosis must be made from the symptoms, especially the rapid wasting and cachexia, which are rarely simulated, even in ulcer. The age of the patient, generally past 40, the deficiency in HCl, and the presence of lactic acid must be allowed due weight. The cachexia of *pernicious anemia* resembles very closely that of cancer of the stomach, and, in the absence of appreciable tumor in the latter, may occasion difficulty. But a study of the blood will in most cases clear up a

doubt. The number of red blood-cells in cancer of the stomach is rarely below 2,000,000 while in pernicious anemia it is often below 1,000,000 per cubic millimeter. This difference exists even while the cancerous subject exhibits more emaciation and weakness than that of pernicious anemia. As F. P. Henry well puts it: "In cancer of the stomach the reduction in the number of red corpuscles does not keep pace with cachexia; in anemia the cachexia does not keep pace with the destruction of red corpuscles." Cancer of the stomach may be latent throughout.

Prognosis.—This is inevitably fatal when one waits long before making a diagnosis, but something may be done toward prolonging life and even saving it by early diagnosis and prompt surgical treatment, the proper cleansing out of the stomach, the selection and regulation of food, and measures to aid its digestion.

Treatment.—An active treatment is summed up in very early diagnosis and an immediate surgical interference. The old method of waiting for a positive diagnosis and a tumor must be supplanted by a careful diagnosis by modern methods and the performance of an exploratory operation in every suspicious case. Many, very many, cases of carcinoma in their early and possibly curable stage masquerade as chronic indigestion. The practitioner never suspects cancer until a tumor stares him in the face. Frequently to wait for a positive diagnosis is to wait for death. Since many cases of cancer of the stomach come to us late, treatment must be directed toward prolonging the patient's life. An operation in indicated at any stage when there is not secondary involvement of some other organ, and it is quite sure that a great deal more can be done than is commonly thought possible. The limit of life of the victim of established gastric cancer does not exceed two years without surgery.

The stomach has no purpose other than the preparation of the food for absorption. It is not a vital organ in the sense that the heart and the lungs are vital organs. It is important so far as it prepares the food, but if the food can be prepared for absorption outside of the body, its importance is diminished. So it is if we introduce artificially digested food by the rectum. Or we may use both of these methods. We can, by the use of prepared food, diminish the labor of the stomach, and by using the rectum we can, while doing so, relieve the stomach of all labor. This is rendered easier at the present day by the use of peptonized foods of various kinds.

However careful the preparation of food, when taken into the stomach in these cases, only a part is used up, and there accumulates gradually a quantity of unabsorbed material which does not pass the pylorus, and to this a copious mucous secretion is added. Gastro-enterectomy under these conditions gives much relief and often prolongs life. When for one reason or another this operation cannot be performed it is desirable, once a day or every other day, to wash out the stomach with water as hot as can be borne, or alkaline waters, as described in the treatment of gastric catarrh. The free use of hydrochloric acid as a medicine also aids not only in the solution of the food ingested, but prevents the fermentations, which contribute irritating acids to the gastric contents and cause further mischief and discomfort. Iron, arsenic, and strychnin may be used to help sustain the habit.

DILATATION OF THE STOMACH.

SYNONYM.—*Gastrectasia.*

Definition.—Two forms exist—a chronic and an acute dilatation.

CHRONIC DILATATION.

Etiology.—(1) The nervo-muscular atony causing dilatation may be the result of habitual overdistention, especially by food of defective quality, resulting in stasis and fermentation; of excessive drinking, as in beer-drinking by employees of breweries; of chronic gastritis; of diseases producing general nervo-muscular atony, such as disease of the spinal cord, pulmonary consumption, anemia, chlorosis, acute fevers, affections of the heart, liver, and kidneys, and other diseases of like import. (2) Mechanical or obstructive dilatation is most frequently due to obstruction by cancer to cicatricial contraction, or to hypertrophic thickening at the pylorus or in the duodenum. Such obstruction may also be due to pressure from without, as by cicatricial adhesion or tumor of an external organ or a floating right kidney. It is most frequent in middle-aged persons, but may occur even in children. Tight lacing, by producing dislocation of the stomach and obstruction to the onward movement of its contents, may also be a cause of dilatation.

Morbid Anatomy.—In addition to the increase of volume the coats of the stomach may be thinned and the glandular structure more or less atrophied. The average normal stomach of an adult holds about 1 1/2 liters (three pints), while the abnormally dilated organ may attain a capacity of three or four liters (six or eight pints), and even more. Where the dilatation is mechanical, there is added the lesion which is responsible for the obstruction.

Symptoms.—The symptoms arising from dilatation are a *sense of fullness in the epigastrium*, *eructations*, *flatulence*, and *vomiting*, often of enormous quantities. The *appetite* is sometimes poor, at others quite good, and the patient is hungry and thirsty. The *vomited matters* are largely water, but include also remnants of food and every variety of fungus—viz., bacteria, sarcinæ, yeast fungi, etc. Their reaction usually exhibits *lessened acidity*, because of diminished hydrochloric acid secretion, but it may be normal or even abnormally acid. Such abnormal acidity is the result of fermentations producing lactic, butyric, and acetic acids. Various gases are thus produced, including carbonic acid and hydrogen. The latter may also arise from decomposition of albuminoid substances, whence too arises sulphuretted hydrogen. These fermentations are favored by the absence of HCl, the importance of which in preventing fermentation has been referred to, and by a stasis of the contents in the stomach; for not only is absorption delayed, but the transit of gastric contents into the intestine is also hindered. Indeed, in some cases the stomach is never emptied unless by the tube. Nay, more; it would seem that at times it contains more liquid than was ingested—a possible condition, since the endosmosis of crystalloids (viz., sugar, dextrin, alcohol, and peptones) is attended with the exosmosis of water. From such causes, too, occur *torpor of the bowel*, *scantiness of urine*, and *dryness of the skin*.

Anemia, *emaciation*, and *debility* sooner or later succeed, and in fatal

cases death is commonly preceded by a *drowsiness*, which may be due to the absorption of toxic substances arising in the decompositions going on in the stomach. Dilatation of the stomach is also one of the acknowledged causes of *tetany*, as first pointed out by Kussmaul. The cramps, though often quite severe, are of short duration. They occur chiefly in the muscles of the hands, arms, and legs. von Leube suggests that this tetany may be due to a "drying out" of the nerves and muscles, but it may also be the result of autointoxication. *Unconsciousness may precede death.*

Physical Signs.—These may be elicited by inspection, palpation, and percussion. *Inspection* does not always afford information, but in emaciated cases the greater curvature of the distended organ may be recognized as low as the navel and below, instead of from 1.2 to 2.8 inches above it (3 to 7 cm.). When the stomach is very low, even the smaller curvature may be recognized about two inches (5 cm.) below the ensiform cartilage, uncovering the pancreas. In obstruction of the pylorus the peristalsis from left to right may even be recognized stopping short at the pylorus, where the tumor-like thickening may sometimes be seen. In rare instances a reverse peristalsis, from right to left, takes place.

Palpation may confirm inspection, recognizing the contour of the stomach by its peculiar consistence, which has been compared to that of an air-cushion, but affords little additional information unless there be a tumor at the pylorus which may be felt. Peristalsis, if present, may also be felt, and may be stimulated by filliping the abdominal walls with the fingers, by which also a splashing sound may be produced in the water-laden dilated stomach down as low as the greater curvature. This is to be distinguished from a similar splashing which may be obtained in the normal stomach and adjacent colon, the latter being less constant and less intense.

If a stiff *sound* is passed into the stomach—its end may be felt through the abdominal walls, while the unusual extent to which it may be carried before meeting resistance will attract attention.

Percussion affords the most valuable evidence as to the presence of a dilated stomach, and in the majority of instances such evidence is conclusive. Auscultatory percussion is especially satisfactory in determining the outlines of the stomach, and the stethoscope may be used with advantage, made from above downward, beginning at the edge of the ribs in the neighborhood of the right parasternal line. The note is tympanitic until the upper curvature is reached, when it is substituted by dullness due to the liquid contents, to be succeeded again by tympany of the bowel when the lower border of the stomach is passed. If the patient lies on his back, the dullness disappears and is replaced by tympany. If there is no liquid in the stomach, a change in the pitch of the tympanitic note will indicate the transition from the stomach to the intestine. Further information can be gained by means of the tube, by which the stomach can be emptied and refilled with air and its borders determined by percussion. This is more satisfactory than filling the stomach with carbonic acid gas, and even such procedure is not always necessary. If the larger curvature be found by percussion at the navel or below, the stomach is certainly dilated. The X-ray may be used with the greatest advantage. A meal of bismuth is given the patient X-rayed while standing. In this way better than any

other a perfectly correct idea may be had not only of the size but of the shape of the stomach.

Diagnosis.—This is usually readily made by attention to the symptoms and physical signs described. Dilated stomach has, however, been mistaken for an ovarian cyst, and abdominal section has been made for its relief.

Dilatation differs from falling, or gastroptosis, though descent and dilatation are often present in the same organ. Different also is enteroptosis, or visceroptosis, which will be considered later.

Prognosis.—Depends upon the measures used in the treatment and upon the promptness of the diagnosis.

Surgical Treatment.—All care of dilatatiom due to obstruction of the pylorus, whether the obstructing cause is benign or malignant should be given into the hands of a competent surgeon. A pyloroplasty, a pylorectomy or a gastric jujenostomy will frequently save lives, and practically always will give much comfort. Cases of dilatation due to atony may be permanently relieved by gastro-jujenostomy.

Medical Treatment.—The most important part of the medical treatment is *washing out the stomach*. This may be done daily, but sometimes it is sufficient to do it on alternate days, occasionally even twice daily. When practised once a day, it is usually best done on retiring at night, as the stomach is thus freed for the night of irritating material which, if retained, disturbs rest and aggravates the local condition. The patient soon learns the most suitable time for lavage, and when it is often necessary, he should be taught to perform it. But this only if surgery cannot be employed.

Of drugs, *hydrochloric acid* is the most likely to be useful, not only because of its importance as a digestive agent, but also as a preventive of fermentation. *Nitro-muriatic acid* may sometimes be substituted with advantage, especially when a stimulating effect is desired on the liver. It should be freshly prepared, and from three to five drops of a pure acid should be given to an adult at a dose. *Strychnin* is a drug which has much to recommend it from the theoretical standpoint as a muscular tonic, and has the further advantage of easy absorption. It should be administered in full doses from a small beginning, 1/30 grain (0.002 gm.) three times a day, increased to 1/20 grain (0.003 gm.) and even more. Extract of nux vomica may be substituted, but it is less easily absorbed. Tincture of nux vomica is better. It may be given in gradually increasing doses until 30 drops, or 15 minims, are given three times a day.

In addition to the hydrochloric acid as an antiferment, other remedies for this purpose are *charcoal* and *creasote*. The power possessed by charcoal of absorbing gases cannot be utilized, because it possesses this property only in the dry state. Yet it does relieve flatulence and is antiseptic. Such antisepsis may be extended to the intestine. Doses of charcoal of 5 to 10 grains (0.33 to 0.06 gm.) and even more may be given conveniently in concils.

Creasote is a useful antiseptic, and may be given in pill form, in doses of 1/2 grain to a grain (0.03 to 0.0 ogm.), or it may be given in sherry wine, whisky, brandy, or tincture of gentian. The following one per cent. solution of creasote is a modification, by George Herschell, of Bouchard's well-known formula:

℞ Creasoti, 10
Tr. gentianæ, 20
Vin. xerici, 800
Sp. vini gallici, 170
M. et Sig. One hundred minims contain one minim or one grain of creasote.

When the condition is part of the morbid anatomy of cancer of the stomach, only palliation may be expected, unless an operation can be performed, either of excision of the cancer or drainage of the stomach.

Dietetic Treatment.—Most important is the selection of food in these cases. Solids should be almost totally prohibited, while the typical nourishment is the various kinds of artificially digested food, such as peptonized milk. Beef-juice and rare beef scraped are also easily assimilated, while fatty, and especially starchy, foods are to be used sparingly, if at all.

Acute Dilatation[1]

Definition.—A condition of sudden enlargement of the stomach caused by surgical operations; by acute infections, or by errors in diet.

Etiology.—By far the greater number of cases of acute dilatation occur after operation. Whatever be the primary cause of the condition here, Conner in his noteworthy work had proven beyond question that certainly in the later stages a real obstruction exists, due to constriction of the duodenum where it crosses the spinal column, under the attachment of the mesentery.

Operations need not necessarily be upon the stomach to cause acute dilatation. Any abdominal operation may be followed by this accident, indeed, operations in remote parts of the body have been followed by it.

Pathological Anatomy.—The stomach is always greatly enlarged, often filling the entire abdominal cavity, and filled to a large extent with fetid, fecal-like matter. The stomach wall may be thickened and not thinned. This thickening is due to infiltration of the tissue.

As Conner has pointed out, when one has met and recognized a case of acute dilatation of the stomach, he is not likely to be misled by any case which occurs. The diagnosis in postoperative cases and in cases which arise with no apparent cause is more difficult than the condition with pneumonia, though here, too, the diagnosis must remain in doubt unless the fact of a possible gastric dilatation is remembered, and a careful physical examination of the stomach is made.

The **symptoms**[1] and **physical signs** of the condition are: Vomiting, abdominal pain, abdominal distention (due to enlarged stomach), constipation (diarrhea in a few cases), collapse, splashing sounds, peristaltic movement over the stomach.

1. *Vomiting.*—This is the most frequent symptom. It occurred in all but one of the cases cited by Fussell and was present in 90 per cent. of Conner's cases from all causes. The vomiting in two instances in Fussell's series was yellowish. In two it had a fecal odor, in the remainder it was dark greenish or blackish in appearance. The quantity is usually large, one pint or more, though rarely it is small. The act of vomiting is painless, and has much the character of that of general peritonitis. The vomitus is suddenly

[1] Acute Dilatation of the Stomach in Pneumonic, Amer. Jour. Med. Sc., Dec., 1911.

and violently expelled from the mouth, without effort on the part of the patient.

2. *Pain was complained of* in 42 of Conner's cases. In this series of pneumonia cases, it occurred twice. In one of my cases it was so severe that morphin was required.

3. *Abdominal distention* usually occurs quickly, is frequently severe, and is almost without exception in the epigastrium, causing a tumor in that position, but on account of the distention being due to the large stomach, and the stomach occupying an abnormal position, the whole abdomen is distended. In one of this series the outline of the greatly distended stomach could be plainly seen. This is exactly in accordance with one of Fagge's cases. This abdominal distention completely disappeared after lavage.

4. *Constipation is the rule.* In two of my own cases the first thought was that probably the symptoms were due to intestinal obstruction. In two of the pneumonia cases, however, there was diarrhea. This constipation adds color to the picture of intestinal obstruction.

5. *Collapse.*—The patient is frequently almost totally collapsed. The face is pinched and anxious. The eyes are sunken. The breathing is rapid. The patient gives every indication of almost immediate dissolution.

6. *The Splashing Sounds.*—By placing ones hands upon the lower abdomen and making a quick percussion of the portion of the abdomen, occupied by the tumor usually a splashing sound can be detected, which is so characteristic of dilatation of the stomach. Peristaltic movement of the stomach area can occasionally be seen. This has been noted in only a few instances, and is apparently not as marked in cases of acute dilatation as it is in cases of chronic dilatation.

Diagnosis.—The condition must be diagnosticated from general intestinal distention not due to obstruction or peritonitis; from peritonitis due to perforation or to extension of inflammation; from intestinal obstruction; from pancreatic cyst; from uremia; from postanesthesia vomiting; and from acute hemorrhagic pancreatitis.

General abdominal distention is common in pneumonia and is more frequently than not unaccompanied by gastric dilatation; that they may occur simultaneously is certain. In simple distention peristalsis may be heard over the entire abdomen. The outline of the stomach cannot be made out, and the stomach tube introduced will not remove the distention; frequently in these cases a rectal tube will relieve the tympany. There is no vomiting, there is often diarrhea.

General Peritonitis.—Here there is the same rapid distention as in gastric dilatation, but the stomach cannot be seen outlined. There is much more tenderness than in dilatation of the stomach, there is no splash, and above all, there is the same collapsed condition of the patient. The stomach tube does not dissipate the distention.

Intestinal Obstruction.—Three of the cases which occurred in pneumonia, were believed to be due to intestinal obstruction, and, indeed, the picture was very like it. Abdominal pain, vomiting, in two instances almost fecal in character, great distention, and constipation. Indeed, the later view that there is always an obstruction where the mesentery crosses the duodenum, gives real reason for the likeness of the pictures. If there is a stricture high

up not due to gastric dilatation, the differential diagnosis would be impossible. In intestinal obstruction, however, the distention is originally general over the entire abdomen, whereas in acute dilatation of the stomach the distention is likely to be in the epigastrium, or at least greater in that position. Sometimes a marked epigastric tumor is seen, occupying the entire epigastrium and the left hypochondrium, also the lower epigastric region, and in rarer instances, the shape of the distended stomach can be made out through the abdominal walls. Careful passage of the stomach tube will cause the immediate disappearance of the abdominal distention where this is due to stomach dilatation.

Pancreatic Cyst.—Dilatation of the stomach has been mistaken for this condition, but in cyst there is the evidence of a true mass. This mass is dull to percussion. The stomach tube will not cause its disappearance. There is no collapse in a cyst.

Uremia.—The dull unconscious condition of uremia is not like the rather active delirium of dilatation of the stomach. There is no collapse. There is no distention.

Acute Hemorrhagic Pancreatitis.—In this condition there is the same sudden onset, with collapse, but the distention is general and not confined to the stomach. It is easily differentiated by the stomach tube.

Prognosis.—Conner gives a death rate of 72.5 per cent. in 102 cases. Laffer a death rate of 62.5 per cent. in 217 cases. Judging from the result in pneumonia cases, six deaths in eleven cases, 55.5 per cent., and in operative cases where the proper treatment has been instituted early, this terrific death rate is probably largely the result of the true nature of the case being unrecognized, or improper treatment (surgical) being applied.

Treatment.—It would appear that acute dilatation of the stomach is one of the rare abdominal conditions in which medical men can both advise and administer the treatment to the exclusion of the surgeon. The first requisite is early diagnosis. Sudden abdominal distention occurring in the course of pneumonia must bring the thought of acute gastric dilatation at once to the practitioner's mind. Distention, collapse, increased gastric tympany, pain, vomiting, are the suspicious signs. Before the patient is moribund, often before the diagnosis is definitely determined, and, as a diagnostic step a stomach tube must be introduced and lavage practised. If the contents of the stomach are foul and copious, or if there is much flatus, relief will be almost instantaneous, and if the dilatation occur after the crisis, recovery may be confidently expected. The lavage must be practised as often as the distention occurs. When a patient is collapsed with running pulse, it is often feared that the passage of the stomach tube may be fatal. This is a mistake. On the contrary, the tube is easily passed and relief is marked even in most desperate cases in the midst of an attack of pneumonia. The position of the patient is of some importance. In distention of the stomach, as had been stated, there is constriction of the duodenum, under the root of the mesentery, and the collapsed small intestines are far down in the pelvis, making the mechanical obstruction still more marked. By turning the patient on the right side or on the face, this element is probably largely removed. All food and drink by the mouth

must be interdicted. Strychnin and eserin hypodermically have seemed of value in two of my cases.

VISCEROPTOSIS.

SYNONYMS.—*Splanchnoptosis; Enteroptosis; Gastroptosis; Glénard's Disease.*

Definition.—A condition in which, as a consequence of relaxation of the ligaments of the abdominal viscera, especially those of the stomach, large intestine, kidneys, spleen, and liver, these organs fall below their normal position. The organs more decidedly involved are the stomach and the transverse colon, especially the right half, and the hepatic flexure.

Etiology.—A satisfactory explanation of the phenomena of visceroptosis has not as yet been offered, though several, more or less applicable, have been suggested. First, there are certain predisposing or favoring conditions, among which are debilitating and emaciating diseases or loss of elasticity of the abdominal muscles due to repeated pregnancies, to gastro-intestinal auto-intoxication, to exhausting hemorrhages, or to damage to abdominal muscles by pressure of clothing. The loss of fat in emaciation, however caused, undoubtedly favors viscero loptosis.

Glénard, whose name is so closely identified with the subject that the affection is called Glénard's disease, holds that a descent of the right or hepatic flexure of the colon followed by dislocation of the transverse colon is the primary disturbance in enteroptosis. The hepato-colic ligament, which is the name he applies to the portion of the mesocolon that approaches the right flexure of the colon, he says is naturally very weak, and can be loosened and stretched by the weight of the transverse colon, particularly when this is loaded with feces. When the hepatic flexure of the colon has sunk, the right half of the transverse colon also descends stretching the gastro-colic ligament which is attached to the pyloric end of the stomach. At this point the colon becomes kinked, causing stagnation of its contents, followed by dilatation of the colon in front of the constriction. Beyond this it contracts, and, according to Glénard, can be felt as a tense cord. As their ligaments become loosened, the remaining abdominal viscera follow the descent of the transverse colon, the stomach being drawn down by traction on the gastro-colic ligament, the liver and kidney following. Ewald confirms Glénard except that what Glénard regards as the contracted portion of the colon beyond the constriction, and calls "*corde colique transverse*," Ewald believes to be the pancreas. He denies also that simple kinking of the colon, uncomplicated by peritoneal adhesions or by stenosing neoplasms, can cause stagnation of feces. Without assigning a distinct cause, Ewald emphasizes the fact that long-standing dyspepsias and bodily overexertion may create altered relations of pressure and tension, and thus lead to the condition. Landau especially emphasizes relaxation of the abdominal walls as the primary cause, though cases are reported in which there is no such relaxation. Recent studies are disposed to call into play a congenital factor the action of which may be intensified by any of the various causes named. In late fetal life and early extrauterine life the position of the abdominal viscera is quite like that characteristic of the disease. This is especially shown by Joseph Rosengart, although Henle and

other earlier anatomists described these positions of the viscera in young children. Kussmaul[1] and Leichtenstern are among those who regard the vertical position of the stomach and colon in adults as a congenital anomaly. The influence of adhesions in producing displacements of the abdominal viscera must not be overlooked, but these are not included in the condition being described.

Visceroptosis is far more frequent in women than in men, 306 out of 404 cases collected by Glénard being women, tight lacing and pregnancy being regarded as the chief causes of this difference in the two sexes. While it is true that the majority of cases met in practice are true visceroptoses, yet it must be admitted that there are instances in which one organ only—as, for example, the stomach, the kidneys, the spleen, or the liver—may be dislocated in the manner referred to.

Symptoms.—First of all it must be stated that such a state of affairs as that described may exist without producing any symptoms. The symptoms which are characteristic are, in a word, those of nervous dyspepsia, including derangement of appetite, and especially anorexia, more rarely false sensation of hunger, a sense of fullness in the epigastrium, noisy belching, various bad tastes, and dryness of the mouth. To the fullness in the epigastrium may be added various sorts of pain—shooting, burning, etc.—after eating. There may be constipation or an opposite condition of diarrhea. Hard, scybalous masses may be removed by purgatives or enemas, also mucus in varying amounts, including casts like those in membranous enteritis. The lower portion of the abdomen is distended, and sometimes, in persons with thin-walled abdomens, the dislocated viscera may be recognized by their outlines. Especially is this true if they be dilated with air or gas. By palpation or percussion, displacements may be recognized with more or less ease. The transition from stomach to colon can often be recognized by change of note on percussion, while the kidneys, spleen, and liver may be recognized by palpation. Among nervous symptoms may be named general weakness, depression of spirits, headache and fullness of the head, vertigo, cold feet and hands. There may be palpation of the heart and disturbed sleep or insomnia. As the result of all this disturbance the patient may become so emaciated as to suggest malignant disease. Chlorosis is often present, and by Meinert is regarded as a constant symptom of the disease; indeed, he holds that gastroptosis is the chief cause of chlorosis in women.

Treatment.—When there are no symptoms produced by this unusual state of affairs, of course no treatment is indicated. When the symptoms are due to displacement, it is evident that mechanical measures or operation are alone likely to be useful in restoring the organs to their normal situation. The former includes trusses and pads, which must be adapted to each case after a study by the instrument-maker with the aid of the physician. In the absence of more elaborate appliances a simple broad bandage may be of service in relieving the symptom. Various degrees of success have been attained by these measures. It is reasonable to suppose that permanent relief can alone be obtained by operation. Treves early reported a case of complete cure by laparotomy and stitching the stomach. At the

[1] "Zeitschrift für diätetische und physikasliche Therapie," Bd., i, 1898, S. 220.

present day operation especially for gastroptosis is not infrequent and is commonly successful. Our colleagues John G. Clark, and Alfred C. Wood have each devised an operation.

In a stomach thus dislocated there are apt to be atony and sluggish peristalsis, which may result in the accumulation of undigested matters, which are better removed by lavage. Other measures useful in dilated stomach may also be expected to be useful as well as those indicated for nervous dyspepsia.

DISEASES OF THE INTESTINES.

SIMPLE ACUTE CATARRHAL ENTERITIS.

SYNONYMS.—*Acute Intestinal Catarrh; Acute Diarrhea; Acute Ileocolitis.*

Definition.—The term employed is applied to a diffuse inflammation which generally pervades more or less of the small intestine and the upper part of the large bowel. More circumscribed inflammations are described, and doubtless sometimes occur, but is it not easy to localize them.

Etiology.—The usual causes of simple intestinal catarrh are overeating or eating improper food, and excessive drinking, or the swallowing of acid or mineral substances of an irritating character. Impurities in drinking-water and in the summer and autumn, unripe fruit are frequent causes. The toxic products of fermented and decomposed food are also causes. These sometimes arise from substances commonly harmless, such as milk or preparations thereof. Cream-puffs, and even ice-cream, are among these. Irritating minerals are corrosive sublimate and arsenic. Although hot weather favors intestinal catarrhs, especially in infants and older children, they are not so much the direct result of the heat as of its effect in weakening the resisting powers of the child and favoring the decompositions and fermentations referred to. The effect of heat on the nervous system of the very young may reasonably be regarded as a factor in increasing irritability of the gastrointestinal tract or in so diminishing its functional power as to render the ingesta irritating. Cold, or rather a chilling of the body by a fall in temperature, is often followed by enteritis.

Secretion altered in quantity or quality has already been mentioned as a cause of simple noninfectious intestinal inflammation. Much spoken of, but of inferred, rather than of demonstrated, import, is excessive biliary secretion, producing what is known as bilious diarrhea. When such diarrhea is associated with a burning sensation at the anus and with the recognized presence of bile in the stools, the term may be justified, but it is to be remembered that an acid reaction of the alvine dejecta produces a similar sensation. A scanty supply of bile to the intestine, by deprivng the gut of the imporant antiseptic property of this secretion, may also favor the fermentations and decompositions mentioned.

Hyperemia, however induced, favors catarrhal enteritis. Such is the hyperemia secondary to hepatic and cardiac disease, and to inflammation, whether traumatic or infectious, in adjacent tissues, whence it extends by contiguity. Such is the inflammation occasioned by peritonitis, by intestinal obstruction, and the like. Cachectic and anemic states, such as are

secondary to cancer, to Addison's disease, and to the last stages of Bright's disease and of tuberculosis, are also favoring causes. Enteritis is also a symptom of certain infectious diseases through their specific poisons, which act directly on the mucous membranes, as in the case of cholera, dysentery, and typhoid fever.

Apart from the effect of nervous influence already mentioned, this cannot be said to cause simple enteritis. It is not unusual for fright and other causes of nervous excitement to produce diarrhea; but this is not the result of an enteritis, but of an increased peristalsis and disturbed vasomotor regulations, and is properly called nervous diarrhea.

Morbid Anatomy.—The morbid changes of simple intestinal catarrh are variously distinct. A hyperemia is naturally to be expected, and in the more decided cases may be manifested by a diffuse redness and injection. It is not often, however, that these are demonstrable. A layer of mucus covering the mucous membrane of the bowel more or less interruptedly is more frequently present. Nor is swelling often evident. At times the solitary follicles are unnaturally distinct, surrounded by a hyperemic circlet. Such enlargements, commonly as distinct as a pin's head, may be as large as a pea, and, becoming filled with pus, form little abscesses, which may rupture leaving an ulcer. They may extend to Peyer's patches. More rarely chronic ulceration results.

Symptoms.—*Diarrhea* is the most constant symptom of enteritis, involving the part of the intestinal tract named in the definition. The resulting stools consist of, first, ordinary fecal contents of the small and large intestine, often offensive; but, as they continue, they become more and more watery, almost colorless. There may be but two or three, or there may be 20 or more. They contain more or less mucus, and are often frothy and associated with flatus. With diminished consistence the odor may grow less obnoxious, until totally absent. At other times it is persistently offensive. Minute examination recognizes in these discharges columnar epithelium variously altered, enlarged, granular, and fragmentary, with nuclei obscured or absent, also various nonpathogenic bacilli and cocci, including the *bacterium coli commune*, yeast fungus, crystals of triple phosphate, oxalate of lime, cholesterin, and undissolved food matters. The reaction of the discharge may be neutral or acid.

Next to diarrhea is *pain*, usually colicky, varying greatly in degree; often, indeed, in the milder forms, absent. There is rarely *tenderness*, but palpation may elicit gurgling and the signs of gaseous distention. *Thirst* and *oliguria* are natural consequences of the free discharge of water. There is usually little *fever*, the rise of temperature rarely exceeding one or two degrees, and the higher grades suggest tubercular inflammation of the bowel. The *appetite*, at first little altered, ultimately fails. Very rarely do the ordinary diarrheas in children and adults terminate in collapse.

It is reasonable to expect modification of the foregoing symptoms as the result of localized inflammation, as contrasted with those of the more diffuse form just described. Thus, the presence of *jaundice* suggests the probability that the *duodenum* is especially involved. In such cases the urine may also be jaundiced, and there may be added other symptoms commonly associated with jaundice. In the absence of this symptom there

is no sign that points to the duodenum as the special seat of the inflammation. On the other hand, jaundice is by no means always present, even if the duodenum is involved. Duodenitis is often associated with acute gastritis, spreading from the stomach—*gastroduodenitis*. Inflammation limited to the duodenum is unattended with diarrhea.

When there is also involvement of the whole of the large intestine—*ileocolitis*. When this is the case, while the lower down the inflammation, the purer the mucus and the more there is of tenesmus, the mucus remains separate and unmixed with the fecal matter, which may contain undigested particles of food, such as muscular fibers, starch, and fat corpuscles. A diarrhea in which these undigested portions of food are visible to the naked eye is known as *lienteric*. Gmelin's nitric acid test for the biliary coloring-matters ceases in health at the sigmoid flexure, so that if this reaction is obtainable in the liquid discharges, it implies that the excessive peristalsis has affected also the large bowel, by which the bile is carried through with abnormal rapidity. The green stools of children, and more rarely of adults, also indicate a large quantity of bile. Simple feverish states, however, may have the effect also of interfering with the proper digestion of food matters, which may appear in the discharges in consequence. Some information—not, however, too much to be relied upon—may be derived from the seat of tenderness and colicky pains. When these are in the middle or inferior part of the abdomen, they point to the small intestine; when in the upper and lateral parts, to the large. A form of acute gastroenteric colitis, characterized by profuse vomiting, purging, and painful cramp, formerly called *cholera morbus* demands special notice.

Etiology.—The intensity of the symptoms and their similarity to those of true cholera justify a suspicion that a specific organism is responsible for *cholera nostras* as well as for true cholera. No single bacillus has, however, been settled upon. Especially frequent are these attacks in the hot weather of July and August, though cold and dampness are also regarded as predisposing causes. So are fatigue and debilitated state of the system. Young adults and persons in the prime of life are more frequently victims than either the very old or very young.

Symptoms.—The victim of this form of enteritis is commonly seized suddenly, often at night, with *severe cramp*, *vomiting*, and *purging*. The first vomitus is the food last ingested, but this is rapidly succeeded by bilious matter, and still later by almost pure water. The same may be said of the bowel discharges, which follow each other in rapid succession—in fact, become at times almost continuous. They present ultimately all the physical characters of the rice-water discharges of true cholera.

Diagnosis.—This diagnosis of acute intestinal catarrh is ordinarily easy, by attention to the symptoms previously detailed, including those more or less peculiar to the more circumscribed localities referred to. From *typhoid fever* acute enteritis is usually easily distinguished by its short duration, minor fever, and the absence of the characterisic course the fever takes in the infectious disease, and absence of the spots which so invariably make their appearance on the eighth day in typhoid. The Widal test in the latter disease also aids the diagnosis.

During *cholera* epidemics mild cases of this disease are not recognizable

symptomatically from the severer colliquative forms of diarrhea. Under these circumstances, bacteriological examination should be made. The importance of a correct diagnosis will be appreciated when it is remembered that indifference in the treatment of simple diarrhea may not seriously affect the result, while such treatment of a case of cholera, however mild, may result disastrously.

Prognosis.—This is always favorable with prompt and judicious treatment, recovery taking place in from one to three days, as a rule, rarely longer.

Treatment.—Many cases of acute catarrhal enteritis recover under rest and restricted diet, the degree of which necessarily depends on the severity of the case. The simple withdrawal of all food, the substitution of plain milk, or, in severe cases, of boiled milk, for the usual food, generally suffices.

No attempt should be made to lock up the bowel until all irritating matters are removed, and it is often desirable to give an aperient, castor oil being the best, though the unpleasantness of the dose often precludes this valuable remedy. In such event calomel in divided doses, the solution of the citrate of magnesium, Rochelle salts, or Hunyadi water may be substituted. When there is much pain, larger doses of opium may be necessary, especially if hot fomentations, mustard plasters, or turpentine stupes fail to produce the desired effect. When there is elevation of temperature, no better means than the local application of ice can be found to relieve pain. Astringents are rarely necessary, but in the absence of other measures may be used. Tannic or gallic acid in 5 grain doses (0.33 gm.) may be given separately or combined with opium.

A few grains of bismuth subnitrate every two or three hours, fortified with 1/8, 1/4, 1/2 grain (0.0082, 0.015, 0.033 gm.) of opium, or 1/2 ounce (15.5 gm.) of chalk mixture with a fluidram (4 c.c.) more or less of paregoric may be added. Salol in doses of 3 to 5 grains is an admirable adjuvant to bismuth through its antiseptic properties.

Treatment of Choleraic Forms of Enteritis.—Opium is almost indispensable to the successful treatment of an attack of cholera morbus. The best method of exhibition is by the hypodermic needle, more especially because everything given by the mouth is apt to be promptly ejected. For an adult less than 1/4 grain (0.0165 gm.) of morphin is hardly to be thought of. On the other hand, such a dose will often act magically. It should be associated with diffuse counterirritation over the abdomen by mustard, while the hot bath may be added, if the symptoms do not yield.

In the absence of the hypodermic needle, remedies must be given by the mouth. The association with morphin of the hot aromatics, such as ginger and cloves, seems to aid its retention.

The nausea may be controlled by ice, by cold carbonated waters, by pieces of ice swallowed whole, or by champagne. The latter is particularly appropriate when stimulants are needed, as constantly happens. When there is a tendency to collapse, whisky and ether may be injected under the skin, while enteroclysis and hypodermoclysis may be needed for the same reasons as in true chlorea—the restoration of the water lost from the system.

For the nausea counterirritation by mustard plasters should be used, pieces of ice swallowed entire, while too much water should be disallowed.

Champagne and cold carbonated waters may be used for this purpose. The latter may be combined with milk, while the old reliable remedy of qual parts of milk and lime-water should not be forgotten.

CHRONIC CATARRHAL ENTERITIS.

SYNONYMS.—*Chronic Enterocolitis; Ulcerative Colitis; Mucous Colitis; Chronic Diarrhea.*

Definition.—A chronic inflammation of more or less of the large and small intestine, with or without ulceration.

Etiology.—Chronic enteritis may remain after repeated attacks of the acute form, or it may arise *de novo*, however induced, favored by whatever occasions passive congestion. Such favoring causes are diseases of the liver or heart, feeble and anemic states, and the defective nutrition consequent theron. Chronic exhausting diseases, such as tuberculosis and Bright's disease, may act in this way also. Dysentery is a frequent cause of chronic intestinal catarrh, a remnant of the acute process.

Morbid Anatomy.—The primary condition is that of acute catarrh, and in many cases the morbid changes do not exceed those of acute catarrh, being simply permanent, or later more pronounced. In others, still more decided changes are found, chiefly in the lower part of the ileum and colon. These are mainly ulcerative, but include also discolorations due to hyperemia, blood extravasation and pigmentation succeeding it, thickening of the coats of the bowel, and contraction of partly healed ulcers. There may be stenosis or the opposite condition of dilatation. Such ulceration is distinct from that of tuberculosis, typhoid fever, and syphilis. It may be follicular, as often seen in the diarrheal affections of children, more rarely in adults, or there may be large ulcers or large areas of ulceration. The remnant of mucous membrane is often pigmented and slate-colored, and a pseudo-polyposis sometimes results from contraction. In the small intestine the pigment is apt to be deposited on the ends of the villi and in rings around the solitary follicles, or in their centers, producing the "shaven-beard appearance." The surface of the bowel is more or less covered with mucous and purulent secretion incident to the inflammation. Still another sort of ulceration, from the etiological standpoint, is found at the bottom of saccules of the large intestine in which scybala or hard fecal masses have lain a long time. Ulceration, too, may result though rarely, from encroachment from without by various kinds of disease of the peritoneum, including cancer, tuberculosis, and the like. Atrophy of the mucous membrane of the bowel is also one of the results of chronic enteritis, not usually recognizable before death.

There may even be atrophy not only of the mucous membrane, with destruction of the glands, but also of all the coats of the small and large intestines.

Symptoms.—These are not uniform. While there is often more or less *diarrhea*, this may be absent, or substituted by *constipation*, while constipation and diarrhea frequently alternate. More characteristic of the stools is the large amount of *mucous matter* contained in them. This may be present

in the shape of "sago"-like masses or "mucous" granules, yellow or brownish-yellow, bile-stained also from the small intestine. Bile-stained mucus is present only when there is abnormally rapid peristalsis of the large bowel, which causes the mucus to pass rapidly beyond the sigmoid before the bile is decomposed. Ulceration may cause the presence of blood in the stools.

Mucous Colitis.—A variety of chronic colitis known as *mucous colitis* or membranous enteritis is characterized by the discharge of large masses of mucus, forming at times complete casts of the bowel. It is more frequent in women, this sex including 80 per cent. of recorded cases, according to W. A. Edwards. It may occur also in children. Its subjects are usually women of the nervous type, and most of the symptoms are nervous in character. It is commonly associated with constipation. At intervals, however, occur attacks of abdominal pain and tenderness, sometimes accompanied by tenesmus and followed by discharges of the mucoid matter referred to. Such attacks may be excited by mental emotion of various kinds. The mucoid material itself seems to be the direct result of an increased activity of the mucous glands, which, with the mucous membrane, are, however, commonly intact after the separation of the large mucous casts. These casts are constantly mistaken for worms by the laity. Minute examination recognizes more or less numerous cells, round and columnar, entangled in the mucus, sometimes also cholesterin plates and triple phosphate crystals.

Throughout the numerous attacks nutrition is commonly well maintained, and the woman subject appears plump and well nourished. At other times there is gradual emaciation.

Diagnosis.—This is always easy, except as to the determination of the portion of the bowel involved or the presence of ulceration. Differences in the character of the mucus, as previously noted, will aid in the diagnosis, while the constant or intermittent presence of blood and pus or fragments of tissue in the stools point to the ulcerative condition. Ulceration is sometimes found postmortem where no symptoms were present before death. In the rectum, and, indeed, as high as the sigmoid flexure, ulcer may be recognized by specular examination. Deep-seated ulceration may cause circumscribed peritonitis or may produce abscess. The presence of scybala, surrounded with mucus, points to inflammation of the rectum or colon as far up as its transverse portion. It is not possible to diagnose the presence of atrophy of either bowel.

Prognosis.—The prognosis in all forms of chronic intestinal catarrh is grave so far as recovery is concerned, and treatment avails little in many cases. The disease, however, extends over months, and even years, before the patient succumbs, and recovery is sometimes complete, quite independent of treatment.

Treatment.—As in the case of acute catarrhal enteritis, rest is an important condition of success in the treatment of this disease. Next, we must select a diet with a minimum of waste, so that there may be as little irritating residue as possible. Milk, beef juice and the albuminous type of foods are the chief of these. Still less irritating are they if partly digested before being taken into the stomach. Thus, milk may be peptonized, and meat also, but the beef peptonoids of the manufacturers should not be employed. It is difficult to ascertain the relative nourishing power of these

peptonoids with the thought that they in any adequate degree can replace milk or meat in the diet. This, then, should be a fundamental principle of treatment—to furnish a diet with a minimum of waste.

When it is remembered that chronic intestinal catarrh is seated mainly in the large intestine, it is manifest that to reach it with remedies administered in the ordinary way is difficult, and that it is more than likely that such remedies are absorbed or decomposed before they arrive at the seat of the disease. It is barely possible that after prolonged administration certain drugs, as nitrate of silver, will ultimately reach the seat of ulceration and stimulate it to heal. Prolonged use must, therefore, be pursued with any remedies thus administered. Nitrate of silver and the sulphate of copper are the two which possess most reputation. The doses are 1/4 grain (0.0155 gm.) of each three times a day, or a smaller quantity more frequently. The acetate of lead may be substituted in doses of 2 grains (0.132 gm.). The latter is more astringent, but is less likely to excite healing. All these remedies are commonly combined with opium in suitable doses. Subnitrate of bismuth in large doses, 1/2 to 1 dram (2 to 4 gm.), is strongly recommended by some. It undoubtedly diminishes the discharges, but how far it is curative is uncertain.

The natural astringent waters, such as the Rockbridge alum and other alum waters in this country, have earned some reputation in the treatment of chronic intestinal catarrh, but improvement under their use is always more marked at the springs themselves, showing that some effect must be ascribed to the change of scene and air and to the salubrious climate of the locality.

Should these measures fail, and there is evident involvement of the colon, irrigation of the bowel may be practised. This is done by means of a fountain syringe, or a funnel in connection with a tube, which is carried high up into the bowel, the patient being placed on his back with a pillow under his hips. The fluids used are solutions of nitrate of silver, sulphate of zinc, and boric acid. At first warm water, say at 100° F. (37.7° C.), should be run in very slowly to the amount of two to three pints (1 to 1 1/2 liters). Then solutions of any of the foregoing substances, of silver nitrate and zinc sulphate, strength of 3 to 4 1/2 parts to 1000 or 1 1/2 to 2 grains to the oz. (0.1 gm. to 0.13 gm. to 30 c.c.); beginning with the weaker solutions. Salicylic acid may be used in two per cent. solution, boric acid in one per cent. solution, or a one per cent. solution of salicylic and boric acids combined. A one per cent. solution of tannic acid is also recommended, as well as of corrosive sublimate, but the latter is exceedingly irritating and the strength of the solutions should not exceed, at first, 1 : 15,000, which may be increased, if well borne. The nitrate of silver has, on the whole, the best reputation. A preliminary anodyne enema of 30 minims (1 gm.) of laudanum may be given, if needed, or a suppository of extract of opium, say one grain (0.066 gm.). Great care must be taken to allow the solutions to pass in slowly, the hips being elevated by a pad or pillow. To be effectual, the treatment must be patiently prolonged, especially the dietetic part, and not weeks, but months, of patient perseverance insisted upon. It has been suggested that these irrigations should be made in intractable cases through the appendix whose opened tip has been sewed to the abdominal wall. (Weir's operation of appendicostomy.)

Mention should be made of the opposite treatment of chronic *mucous colitis* by a coarse diet containing a large proportion of indigestible residue, especially cellulose derived from bread made from flour containing a large proportion of husks (Graham bread); all varieties of leguminous vegetables, peas, beans, etc., with their husks, vegetables containing much cellulose, fruits with small seeds and thick skins, like currants, gooseberries and grapes, together with large quantities of fat, especially butter and bacon; also weak saline mineral waters, of which Saratoga water is the type in this country and Kissingen, Homburg, and Racocsky abroad. This diet is recommended by von Leube, von Noorden, Boas, Einhorn, Westphallen, and Hemmeter. Some, however, especially v. Noorden would make the change suddenly, others gradually.

The effect is claimed to be to give the stools after two to four days a normal consistence and normal appearance. No purgatives are required. It is claimed that the results of this treatment are much more satisfactory than the older light diet composed of easily digested foods, such as milk foods, broths, white bread, and the like. Von Noorden calls it the "dietetic exercise treatment of the intestine" as contrasted with the older "protective rest treatment."

The treatment of the nervous state which accompanies *mucous colitis* deserves especial mention. The patients whether men or women keep a close watch upon each and every stool passed, when mucus is present they are much depressed, they are elated when it is absent. Consequently all patients should be warned against looking at the stool, the warning must be constantly repeated. In addition the patients should keep mentally employed; to this end they thould have some light physical work which is not merely automatic. If they are financially able, travel in new and interesting regions is advisable. Camp life with its slight hardships may be tried.

DIARRHEAS OF CHILDREN.

The importance of these, and some specialization in their symptomatology, demand a separate consideration. Three forms, more or less distinct, are recognizable—viz., acute dyspeptic enteritis, cholera infantum, and acute enterocolitis.

Acute Dyspeptic Enteritis.

Synonym.—*Acute Dyspeptic Diarrhea.*

Definition.—An acute inflammation of the small intestine due to diet unsuited to the infant.

Etiology.—The commonest cause by all means is artificial food in infants and improper food in older children, but the errors in diet referred to do not necessarily consist in unnatural foods substituted for the mother's milk. The latter itself may be altered in quality by emotional causes, by improper food, and by improper hygiene; or the child may be too liberally supplied by overfrequent nursing.

Milk used for artificial feeding, the most carefully selected, is unnatural, and is probably the most frequent cause of dyspeptic diarrhea in children

otherwise well cared for. Two factors in this are active: first, the relatively greater indigestibility of the food thus applied; and, second, the bacteria and their toxic products which develop in it before or after ingestion. In infantile diarrhea the number of species of bacteria is greatly increased, but no one or more species has as yet been shown to possess a specific causal effect.

There are also predisposing influences which facilitate the action of the essential causes. These are, especially, dentition and the extreme heat of summer. The effect of the former is learned in the experience of every mother, while the extraordinary frequency of infantile diarrhea in summer attests the latter. It is evident, too, that constitutional weakness and bad hygiene must also cooperate to diminish the resisting power of infants to other causes. Hence it is that the children of the delicate, the poor, and the unclean suffer most.

Morbid Anatomy.—This seldom exceeds the stage of catarrhal swelling, already described when treating of the enteritis of adults.

Symptoms.—Usually there is an intoxication shown in the beginning *restlessness*, with slight *fever*, which seldom becomes high. Such restlessness may be due to *nausea* or to colicky *pain*. The nausea may go on to *vomiting* or not, but *purging* soon occurs. Sudden onset is characteristic. Sometimes the first symptom is diarrhea. The stools are at first copious and offensive, often yeasty and sour, and generally contain particles of coagulated milk or other undigested food, such as unripe fruit, if the child is old enough to eat it. At first infrequent, they become more numerous, more scanty, acquire sometimes a green color and sometimes contain mucus, rarely blood. In other words, the condition passes over into enterocolitis. There may be but three or four stools or there may be 20 or more in the 24 hours.

In other cases fever is more decided, and the temperature may rise rapidly to 104° F. (40° C.); there are great *thirst* and *scanty urine*. Even when there is no fever, *emaciation* is rapid, and the child falls away amazingly in a few days.

Diagnosis.—The sudden onset and the character of the stools are distinctive and scarcely mistakable. The small amount of mucus distinguishes them from those of ileo-colitis, and the absence of serous discharge from those of cholera infantum.

Prognosis.—This, among the better classes, is commonly favorable, but among the weak, puny, and half-starved children of the poor large numbers perish, especially in hot weather. The disease may pass over into the much more serious affection of entero-colitis.

Prophylaxis.—Proper food is the keynote to both prevention and treatment of all the diarrheal diseases of childhood. Infants should be nursed whenever practicable, either by the mother or a wet nurse. Holt found only 2% of 1945 cases summer diarrhea had been totally breast fed. This failing, some proper modification of cows milk should be used in children under 12 months of age (the reader is referred to the books on Diseases of Children by Holt and by Rotch for the details of these measures). Older children should also be fed rationally and rules governing this feeding can also be found in the volumes referred to. Especial care is directed to summer-time. The researches carried on under the auspices of Rockerfeller

Institute show that mortality from these diseases was 2.5% in winter and 10.5% in summer. Avoidance of patent infant foods is an important point in preventing these diseases.

Treatment.—The regulation of diet is of the utmost importance. It is better to give the child nothing except a little cold water or barley water than unsuitable food, while any food that is given should be very much diluted, and should be scanty rather than overabundant. Too much food is often given. Nothing is better than milk properly modified. This modified milk may be peptonized under certain circumstances, if the mother's milk or that of a wet-nurse be unobtainable. Plain fresh cow's milk well diluted may do as well in older children. *In fact, all milk foods should be diluted* with Vichy water, lime-water, or plain water. Animal broths, however dilute, are not advised, though occasionally beef juice is well borne when milk has not been, especially in children two or more years old. Albumen water, made by mixing the albumen of one or two eggs with 1 pint (1/2 liter) of sterilized water, is much more suitable.

The principles of treatment are similar to those of enteritis in adults. A primary purge is commonly indicated. Calomel and calcined magnesia are very suitable, though castor oil is here also useful. After the purge, bismuth subnitrate or prepared chalk, in doses of 2 1/2 grains (0.165 gm.) for a child a year old, with 1/2 grain (0.033 gm.) of salol may be given every two or three hours. If there is pain, 1/24 to 1/12 grain (0.0027 to 0.0054 gm.) of opium may be added each time or every other dose, as may be demanded by circumstances. An attempt should first be made to relieve pain by gentle counterirritation, as by weak mustard plasters or a plaster of mixed spices, wet in whishy or alcohol, and known as a "spice plaster," and worn continuously. Deodorized tincture of opium or paregoric may be substituted for the whisky. Astringents are seldom necessary in children's diarrhea. Chalk mixture, to which a few drops of paregoric may be added, is an efficient remedy.

The hygienic surroundings of the child are important. Frequent bathing; light, cool dressing in warm weather; and fresh air at all times are indispensable. The patient should be removed from city air to the country or seaside, when possible; and when this is not possible, frequent excursions should be made to the country or on an adjacent river. It is not desirable to keep the child on the lap any more than is necessary.

Acute Ileo-colitis.

Synonyms.—*Acute Entero-colitis; Follicular Enteritis; Follicular Dysentery.*

Definition.—An inflammation more severe than dyspeptic enteritis, chiefly of the ileum and colon, affecting especially the lymph follicles.

Etiology.—Entero-colitis is also a disease of the hot months and of teething. It is met, however, in the cooler seasons. It is produced by the same causes as dyspeptic diarrhea. It is more frequent between the ages of six and eighteen months—second summer—and is not infrequent in the third and fourth years. It may be a termination of dyspeptic diarrhea or of cholera infantum.

Morbid Anatomy.—The morbid changes are more positive than in acute dyspeptic diarrhea, and are found chiefly in the ileum and colon. In the first stage the mucous membrane is congested and swollen, while the solitary follicles and Peyer's patches are more distinct. The epithelium is exfoliated in places. As the disease continues into the second stage, say after the first week, the enlarged follicles and Peyer's patches become ulcerated. The changes may end here or may become more extensive, constituting the third stage, the ulcers enlarging and deepening to the muscular coat, with the separation of a slough. Or there may be a diffuse infiltration of the bowel with small cells, producing a decided thickening of the same, with more or less obliteration of its distinctive structure. The process may be so intense as to cause coagulation-necrosis—false membrane.

Symptoms.—The disease may begin as a *dyspeptic diarrhea*, also as a *cholera infantum*. It is much more serious than dyspeptic diarrhea, as evidenced by the higher fever, which rises rapidly to 104° F. (40° C.), but still remains lower than in cholera infantum. *Vomiting* is less common than in dyspeptic diarrhea or cholera infantum. There are decided abdominal *pain* and a *tense, swollen belly*. The fecal discharges, which are at first painless, are small in quantity and contain much mucus and even a little blood. They vary in frequency from 15 to 30 in the 24 hours, and occur more frequently during the day. The disease may abate at this stage and convalescence be established, though recovery remains slow. Or the symptoms may increase in severity, the fever persist, and the *stools be painful* and *small*, consisting mainly of mucus and blood. Commonly odorless, they may also be extremely fetid. The *urine* is scanty, of high specific gravity, and deposits mixed urates. The child wastes almost to a skeleton, the skin becomes loose and flabby, and the "old man" appearance is assumed. Such a case may last five or six weeks, terminating fatally, yet may, on the other hand, get well. A few fatal cases are much more rapid in their course, being ushered in with convulsions and ending in from 48 hours to five or six days. Relapses after convalescence are not uncommon, and should be guarded against.

Diagnosis.—Acute ileo-colitis is characterized by a greater severity than *dyspeptic diarrhea*, by the high fever, the large amount of mucus in the stools, the greater pain, and the more rapid prostration. From *cholera infantum* it differs in its lower hyperpyrexia, and in the absence of vomiting, of colliquative diarrhea, and of collapse.

Prognosis.—This is more unfavorable than in acute dyspeptic diarrhea; more favorable than in cholera infantum. Recovery is not infrequent after a lengthy illness of four to six weeks, while the severe dysenteric form is apt to be early fatal. Much depends upon the promptness with which treatment is instituted and the ability of the parents to carry it out, and upon the previous vigor of the child, its hygiene, and its food.

Treatment.—The general hygienic and dietetic treatment of acute entero-colitis is similar to that of acute dyspeptic diarrhea; the medicinal treatment is somewhat different. Anodynes are more imperatively demanded, because there is greater suffering. Otherwise, drugs are not of much use, though bismuth, in full doses, may be given with advantage.

The colon may be flushed with a one per cent. cold salt solution, or

cold water or pieces of ice may be introduced into the rectum, which may also be used for medication, more particularly by opium. If used, they should be very weak. Solutions of nitrate of silver, 1 grain to the ounce (0.066 gm. to 30 c.c.), and tannic acid, 5 grains to the ounce (0.33 gm. to 30 c.c.), are suitable. The mouth should be often examined and, when necessary, the coming teeth scarified, not once only, but as often as necessary.

Cholera Infantum.

Definition.—A variety of acute catarrhal enteritis of intense severity, corresponding in symptoms and course to cholera morbus in the adult, but much more serious in termination.

Etiology.—The same reasons that lead us to expect a specific cause of cholera morbus would suggest one also for cholera infantum. None has, however, been found. It may reasonably be ascribed to toxins generated in the decomposition and fermentation of foods, since some error of diet is almost always the apparent exciting cause. There are also predisposing causes, of which hot weather, dentition, or both, bad hygiene, the previous presence of dyspeptic diarrhea or entero-colitis, are instances. It is less frequent than either of the last-named affections, including only a small proportion of the summer complaints of children—according to Holt not more than two or three per cent.

Morbid Anatomy.—There is little, if any, deviation from the normal appearance in the affected bowel.

Symptoms.—These consist in copious *serous stools*, at first containing some offensive fecal matter, later a few particles of greenish matter; but ultimately they are almost aqueous, being ejected also with great force. They contain numerous bacteria, but no constant organism has been found. There is crampy *pain*, and the *limbs are drawn up* or *rigidly extended*. There is decided *fever*, more than in either of the two other forms, the temperature reaching 105° F. (40.5° C.); the *pulse* is frequent and feeble, while *restlessness* is a characteristic symptom. The temperature should be taken in the rectum, as that of the axilla may be misleading. Indeed, the skin sometimes feels cool when the internal temperature is high. There is intense *thirst*, and the child eagerly drinks water. The purging may come on suddenly or may succeed dyspeptic diarrhea or ileo-colitis. Simultaneously there is severe and obstinate *vomiting*, including bile at first; but later the vomited matter is also serous. The *tongue is coated* in the beginning, but later becomes dry and red. The child rapidly loses strength and as rapidly emaciates. The restlessness is succeeded by *apathy* and *indifference*, and the condition passes into collapse. The eyes become sunken, the fontanels depressed, the skin gray or ashen and closely applied to the frame, producing an appearance which, once seen, is rarely forgotten. Or the more severe symptoms may subside, and a condition of *torpor* or *semi-consciousness* may supervene. The head is retracted, and there may be *convulsions;* the breathing is interrupted and of the Cheyne-Stokes type; the pupils are irregular; there is clutching of the fingers—in a word, the "hydrencephaloid" state, so called by Marshall Hall, is present. These "brain symptoms" have often misled the inexperienced, but they are not

associated with changes in the brain or in its meninges. They may be due to the toxins developed in the intestine by bacteria.

Diagnosis.—This is not difficult. The serous vomiting and purging, rapid emaciation and prostration, and the hyperpyrexia are significant, while the nervous symptoms described as succeeding them confirm the nature of the disease.

Prognosis.—Unless the last-described symptoms supervene, the course is rapid to a fatal termination by collapse in from a few to 24 or 48 hours. If the hydrencephaloid state is added, the disease may be prolonged a few days more. Recovery is not impossible, and begins with abatement of the more serious symptoms within the first 24 hours, followed by tedious convalescence. Or there may be a delusive improvement, followed by a return of the choleraic symptoms, or the disease may pass into entero-colitis.

Treatment.—All that has been said about food in dyspeptic diarrhea and entero-colitis applies here, but the opportunity for its application cannot, indeed, be availed of unless convalescence sets in. The symptoms must be met with the greatest promptness by the same measures described in the treatment of adults, but adapted to the age of the child. The stomach must be emptied by thorough washing by a stomach tube. The bowel must be flushed with normal salt solution. Here, too, opiates are indispensable. Even morphin may be used hypodermically with great caution, but never after the diarrhea has ceased. One two-hundredth of a grain (0.00033 gm.) is about the proper dose for a child a year old, and it may be associated with 1/1000 grain (0.00005 gm.) of atropin. This may be repeated in an hour if the symptoms do not subside, at a longer interval if they do. Laudanum or deodorized tincture of opium may be substituted and administered by the rectum in doses of from 2 to 4 drops (0.133 to 0.264 gm.) in 2 drams of starch-water. Minute doses of Dover's powder, say 1/20 grain (0.008 gm.), may be given in combination with bismuth in doses of 2 grains (0.12 gm.). For the diarrhea that may continue after abatement of the acute symptoms preparations of silver, preferably, the oxid, are sometimes of value. They may be combined with opium, the dose of the silver being 1/12 grain (0.0056 gm.), of the opium 1/24 to 1/12 grain (0.00275 gm. to 0.0056 gm.).

The hyperpyrexia must be combated by hydrotherapy—the bath at 80° F., rapidly reduced to 70° F. (26.6° to 21.1° C.); or, if this cannot be done, the child should be wrapped in sheets wrung out in cold water. Sponging is a feeble substitute. Hyperpyrexia is one of the dangers.

Brandy is of value, though iced champagne may be given in small doses often repeated, while the prompt rejection of liquids should not discourage their readministration. Irrigation of the large bowel, spoken of above, may be added, using a flexible catheter, which is introduced six or eight inches (2.3 to 2.7 cm.). A pint (0.5 liter) will suffice for a child six months old, and a quart (1 liter) for one of two years. The water may be tepid, or cold if the temperature is high. The 0.6 per cent. salt solution may be administered by enteroclysis, and even by hypodermoclysis in extreme cases of collapse. The hot bath should be substituted for the cold in collapse, and strychnin may be administered hypodermically in doses of 1/100 grain (0.00066 gm.) to a child one year old.

Should convalescence set in or entero-colitis supervene, great caution in the giving of food should be observed. Only peptonized milk should be used, substituted occasionally by raw beef juice, increased, if well borne, a teaspoonful at a time; or dilute egg albumen may be tried if there are not retained.

PSEUDOMEMBRANOUS ENTERITIS.

SYNONYMS.—*Croupous Enteritis; Diphtheritic Enteritis.*

Definition.—A rare variety of intense inflammation affecting either bowel, and characterized by the formation of false membrane.

Etiology.—Pseudomembranous enteritis occurs in connection with such infectious diseases as pyemia, pneumonia, scarlet fever, and even typhoid fever; also from the toxic effect of mineral poisons, such as lead, mercury, and arsenic, and during the cachexias which develop toward the close of cancer, Bright's disease, cirrhosis of the liver, and the like, as a terminal infection.

Morbid Anatomy.—The false membrane present varies in extent and depth. It may be limited so as simply to tip the villi and valulæ conniventes or other folds with a grayish-yellow film, or the coagulation-necrosis may infiltrate a greater depth in flake-like patches, or it may invade the follicles and solitary glands, which may suppurate. To the false membrane is commonly added a hyperemic basis. The deep-seated diphtheritic inflammation found in diphtheritic dysentery is elsewhere described.

Symptoms.—These may be so slight as to be unnoticeable. At other times there are *diarrhea* and *abdominal pain*, but nothing distinctive.

Treatment.—This is symptomatic, and that of the attending and causing disease.

PHLEGMONOUS ENTERITIS.

This is a rare disease, consisting in a diffuse suppurative infiltration of the submucosa, analogous to phlegmonous inflammation of the stomach. It has been found after intussusception and strangulated hernia, and may cause symptoms of peritonitis by invasion of this coat of the bowel, but there are no symptoms by which it can be recognized before death. It has been met in the duodenum.

HEMORRHAGIC INFARCT OF THE BOWEL.

Definition.—Hemorrhagic extravasation in the wall of the small intestine, due to embolism or thrombosis of one or other of the mesenteric arteries.

Etiology.—A warty vegetation from coexisting valvular heart disease may become the embolus, or the latter may arise from the clot in an aneurysm of the aorta or in the tip of the left auricle.

Morbid Anatomy.—There are congestion, infiltration, and swelling of the jejunum and ileum, and the superior mesenteric artery will generally be found plugged with a clot, which may be preceded by an embolus. The mesentery may also be the seat of congestion and infiltration.

Symptoms.—There may be sudden *nausea, vomiting, faintness, abdominal tympany,* and *pain.* There may be symptoms of *obstruction,* or *diarrhea* with *blood-stained stools.*

Diagnosis.—The condition is so rare that infarction is not apt to be thought of. But should there be valvular heart disease or aneurysm, the sudden occurrence of the symptoms mentioned might suggest this cause.

Prognosis and Treatment.—The prognosis is invariably fatal in severe cases, and though the occlusion of a small vessel may be followed by recovery, there is no treatment which will avail further than to abate the symptoms.

APPENDICITIS.

SYNONYMS.—*Typhlitis; Perityphlitis; Paratyphlitis.*

Definition.—An inflammation of the veriform appendix, catarrhal, ulcerative, or interstitial, commonly extending to the peritoneum adjacent to it, producing:

1. A local adhesive peritonitis limited to the region of the appendix associated with an exudate of plastic material which may be absorbed or become organized periappendicitis.

2. Circumscribed suppuration or abscess—paraappendicitis, or perityphlitic abscess.

3. General septic peritonitis.

Perforation and gangrene are often intermediate incidents.

Catarrhal appendicitis may not extend beyond the mucous coat of the appendix in which event the peritoneal coat may be intact at operation.

The term typhlitis, so long employed, was adopted because it was thought that the disease began in the cecum, or typhlon. Modern studies go to show that true appendicitis never begins in the cecum, but that in essentially all cases the appendix is the root of the evil.

Etiology.—*Exciting Causes.*—All stages of appendicitis are probably due to the invasion of microorganisms, while the foreign bodies, concretions, and other agencies to be mentioned are to be regarded as predisposing causes, furnishing the conditions favorable to the operation of the pathogenic bacteria.

While in most instances the *bacillus coli communis* has been found in pure cultures, pyogenic bacteria have been found associated with it. The most important of these is the *streptococcus pyogenes;* after this the *staphylococcus pyogenes aureus* and the *bacillus pyocaneus;* so that the existence of more than one possibly infecting species may be admitted. The bacilli of typhoid fever and influenza are infective agents causing appendicitis. In these cases the appendicitis is part of a general infection.

Predisposing Causes.—The most important predisposing cause of appendicitis is the appendix itself. An organ without function, and therefore undeveloped and feebly nourished, is correspondingly feebly resistant to all disease. Its anatomy is such that the entrance of irritating matters is easier than their exit, while inflammatory products are not easily evacuated. As predisposing causes, too, must be considered certain influences which formerly were regarded as exciting causes, such as overeating, especially of

unwholesome and indigestible food, acute indigestion from any cause, in addition to the foreign bodies and concretions already mentioned. It cannot be said that the precise mode of operation of such cause is certainly known. It may be that a hyperemia or deranged circulation thus induced produces a condition favorable to the action of incessantly present bacteria. Similar is the effect of fatigue, cold, and traumatic causes, such as blows and contusions.

Appendicitis is a disease of children and young adults, but may occur at any age. From 50 to 55 per cent. of cases occur under the age of 20, 30 per cent. between 20 and 30, 15 per cent, under 15. In 3000 cases operated by Deaver, 61.87 per cent. were in males and 38.13 per cent. in females. It has been suggested that this is because the lumen of the appendix is larger in males, and therefore more liable to receive fecal or foreign matters. Attacks have occurred in the first year of life and as late as the 76th. More cases occur in summer than in winter. Occupation has no effect in exciting it, but after a first attack recurring attacks of appendicitis are more frequently in men who do heavy work, such as porters and carriers, or men who stand on their feet long each day.

Pathology and Morbid Anatomy.—Appendicitis may be divided into acute and chronic forms:

Acute Catarrhal Appendicitis.—Our knowledge of this is based upon the systematic, minute study of cases which come from operation. In the first or acute stage there is a shedding of the epithelium of the mucous membrane, with detachment, partial destruction, and extrusion of the follicles of Lieberkühn, and some cellular infiltration of the retiform tissue at their base. The lumen of the appendix contains mucus, leukocytes, exfoliated cells, and casts more or less perfect, of the crypts, with granular débris from the same sources. In the second stage the basement membrane is broken and dislocated, and retiform tissue more closely infiltrated with leukocytes, and the internal surface ragged and uneven. In the third or still more advanced degree the mucous membrane is thickened by infiltration with cells. *The most important fact as to catarrhal appendicitis is that all three stages offer vulnerable foci for the attacks of pathogenic bacteria, and starting-points of an infectious peritonitis.* On the other hand, by the union of the opposing surfaces, *obliteration of the lumen* of the tube may take place, by which it is rendered immune against further attacks, but this occurs very rarely. The obliteration may be partial, producing stricture, beyond which a cystic distention of the tube in the end nearest the cecum is not infrequent and beyond wihch a severe inflammatory condition may take place.

Acute Ulcerative Appendicitis.—In this stage the mucous membrane and submucous tissue are destroyed to various depths, while it may even culminate in perforation. It is often associated with a concretion or a foreign body. The latter is now acknowledged to be much more rare than was formerly supposed. The error was a natural one, owing to the close resemblance of fecal concretions to seeds, grains of wheat, cherry stones, and even date stones, as the result of a gradual molding of shape and loss of water. The concretions are sometimes also the seat of a deposit of lime salts. They may be multiple and may be in the appendix a long time with-

out producing harmful effect, the patient dying of other causes. The same is true of foreign bodies, which do, of course, occur and include the objects already mentioned. Fecal concretions are found in from 35 to 50 per cent. of cases; foreign bodies in a much smaller number—say 7 to 12 per cent.

Acute Interstitial or Parietal Appendicitis.—This stage may succeed upon either of the two stages just described, but occasionally it may arise *de novo* by infection along the lymphatics. In the former event it starts in the abraded or ulcerated surface described; in the latter, in the substance of the appendix wall. It is commonly associated with necrosis or gangrene of the wall, but may prove fatal before the necrosis sets in. The appearances vary greatly. They may be limited to a mere point, scarcely visible, and between this and sphacelation of the entire organ there is every intermediate degree. The gangrenous organ is usually enlarged and distorted. The virulence of the appendicular peritonitis is, however, just as great when there is no necrosis. The peritonitis which ensues on perforation of the appendix is virulent, resulting from the invasion of the peritoneum by myriads of bacteria in the fecal matter set free at the time of rupture of the bowel.

The *minute changes* in interstitial appendicitis are as varied as the macroscopic. The cases which succeed on the catarrhal or ulcerative form are, of course, characterized by the loss of tissue corresponding to the extent of the disease. To these succeed destructive necrotic processes in the deeper structures of the wall. In the first stage of the latter the inflammation is characterized by necrosis of the muscular coats; in the second by suppuration in them; and in the third by their infiltration with leukocytes and inflammatory exudation. The first is, by far, the most common. In all three bacteria are found in the mucous and muscular coats, and all three are followed alike by virulent peritonitis. There is no sharp demarcation between these varieties, the condition evidently depending upon the degree of the inflammation and the virulence of the infecting organism.

The appendix may also be the seat of *tubercular ulceration*, followed, too, by perforation. So, too, a *typhoid ulcer* may form in the appendix and perforate, with the formation of a tumor mass in the right iliac region. *Follicular abscess* may exist and occasion the usual symptoms of appendicitis. Actinomycosis has also occured in the appendix, with the formation of retrocecal abscess and metastatic abscess of the liver. Primary carcinoma of the appendix occurs frequently.

Superadded to these conditions is often a *localized* or *general peritonitis*. In lesser degrees of the localized peritonitis the adhesions which form are limited to the appendix and adjacent serous tissues, limiting the inflammation and acting as a barrier against general peritoneal infection. In higher degrees the inflammation attacks as well the tissues more remote from the appendix (paraappendicitis), and forms the iliac phlegmon or tumor. This occupies the right iliac fossa and is variously constituted. It may consist of serous and cellular exudation, which mats together coils of small intestine and cecum, or there may be a massive accumulation of cells and liquid exudate, constituting abscess. This appendicular or perityphlitic abscess may rupture into the peritoneum, not infrequently producing fatal general peritonitis. The amount of pus varies. There may be a dram or two (4 to 8 c.c.), or a pint (a half liter) or more. The pus is usually thin and

very fetid; at times it is thick, yellow, and odorless. It may be mixed with fecal matter. The pus may have escaped into the bowel, bladder, or vagina, or externally at some point in the abdominal wall—as the navel or groin, as in a case of my own—or through the obturator foramen into the hip or thigh. The iliac muscle may be destroyed and the ilium bared. The abscess, usually in the iliac region, may be in the lumbar region, or perinephric, in the true pelvis, or under the liver. These very diverse sites are commonly determined by erratic situations of the appendix. There may be secondary abscesses of the liver by pylephlebitis or portal embolism. These may have all the terminations possible to hepatic abscess.

If general peritonitis supervene, there are added the usual anatomical appearances incident to this condition—flakes of lymph scattered over the intestines, binding the latter together, with pus-cells in varying numbers in the flakes.

Symptoms.—The onset is usually sudden.

The first symptom is invariably pain—*sudden pain.* Its location at first is not constant; it may be anywhere in the abdomen. Most frequently, perhaps, it is in the neighborhood of the umbilicus. At other times it is in the epigastrium; at others, diffuse. It is intermittent, or at least remittent. Usually, within the first 24 hours, it settles itself in the right iliac region, where it remains. It may then be mild or severe; more frequently it is moderately severe. Even at this stage—end of 24 hours—its location is not always in the right iliac fossa. It has even been most severe in the *left* iliac fossa, under the liver, or beneath the spleen, anomalous situations for the appendix. This pain is increased by coughing or taking a long breath, or by turning over on the side.

As constant as pain is *tenderness* in the right iliac region, or if the appendix happens to be placed in one of the unusual situations named, it will be in that situation. Rather strong pressure may at times be necessary to elicit it, but usually moderate pressure suffices. Its extent varies. It may occupy the whole lower quadrant of the abdomen, or may extend up to the costal margin and around into the flank, but the seat of maximum tenderness is oftenest a point known as McBurney's—a point at the intersection of a line drawn from the anterior superior spinous process of the ilium to the umbilicus and another along the right edge of the rectus muscle. It is from one and one-half to two inches from the anterior superior spinous process of the ilium. The patient almost invariably assumes the *dorsal decubitus*, often with the right leg drawn up, because of the relief thus afforded.

The third cardinal symptom, is *rigidity of the right rectus abdominis muscle* and other muscles overlying the focus of inflammation. This may be associated with a slight distention of the entire abdomen. In explanation of the tenseness it may be said that the rectus and other abdominal muscles receive their nerve supply from the seven lower intercostal nerves, while the superior mesenteric plexus gets its splanchnic branches from the same nerves. This primary tenseness, after two or three days, may be substituted by a *tumor*. The latter varies in size and shape, but is more commonly oval and about as large as a hen's egg, with its longer axis parallel with the upper part of Poupart's ligament. It may be much larger, occupy-

ing also the whole lower left quadrant and extending upward and backward into the flank, while its shape may be quadrilateral or triangular. It varies in consistence. Its composition has been described in considering the morbid anatomy of the disease.

There is usually *impairment of resonance* to percussion over such a tumor, though less than might at first be expected. This is because we are really percussing over hollow organs, though matted together by exudation. At times, however, there is a duller note, while at others, it may be natural. In the latter event the tumor is small. Indeed, tumor may be altogether absent, but this can never be said of tenderness.

Vomiting is a symptom more or less frequent. It is commonly regarded as reflex and is variously severe. The matter vomited is first the gastric contents, with the evacuation of which the vomiting usually ceases, though it may recur in the event of perforation or rupture of the abscess. If the symptom is more prolonged, the vomited matter becomes greenish. Many so-called "bilious attacks" of past times have really been attacks of appendicitis.

Constipation is present in a decided majority of cases from the beginning of the attack. It is due to paralysis of the bowel, and may be so obstinate as to simulate obstruction of the bowel, being even attended at times with stercoraceous vomiting. Indeed, appendicitis has often been confounded with obstruction. On the other hand, there may be diarrhea, recurring with each successive attack. There is *loss of appetite*. The *tongue* at first may be natural, but later becomes more or less coated, and in advanced stages dry.

There is usually *fever* at the outset, the temperature 102°, 103° F. (38.9°, 39.4° C.), and even 104° F. (40° C.), rarely higher, after which it gradually falls, reaching the normal in from five to seven days in favorable cases, which terminate in resolution. The pulse-rate corresponds with the degree of fever, but the force and volume of the pulse vary with the patient's strength. Should suppuration take place, the temperature continues with but slight fall, or may even rise higher. *Suppuration may, be unattended with fever.*

A sudden fall of temperature does not always mean the establishment of convalescence. Not very rarely the event has a widely different meaning. It means that, instead of convalescence, perforation has taken place. It is extremely important that this fact should be realized. A high fever means continued inflammation, but a normal temperature may not mean convalesence. Another even more unusual explanation of sudden fall of temperature is the rupture of a small abscess into the bowel. *Finally, too much stress cannot be laid upon the fact that there may be gangrenous appendicitis in the presence of normal temperature.*

Leukocytosis is present in a large number of cases, the white cells often amounting to 16,000 to 20,000. It is an important diagnostic sign. On the other hand, the absence of leukocytosis, like the absence of fever, should not inspire overconfidence that appendicitis does not exist, as a lowering blood count is sometimes evidence that nature has given up the struggle.

The *urine* is *scanty*, as is usual in fever, and quite frequently contains an abnormal quantity of indican. It is rarely albuminous, unless there be

high fever, when there may be the slight albuminuria characteristic of fever. There are often irritable bladder and frequent micturition.

The *expression* of the patient varies with the severity of the symptoms, but seldom exhibits the anxiousness characteristic of peritonitis, unless the latter actually is present in consequence of perforation or rupture of abscess.

Rapid growth of the tumor and the attainment of large size in a short time point to suppuration, but the most valuable sign is *the presence of extreme tenderness over the focus of inflammation*. Continued high temperature is significant, though it may be wanting. Fully formed abscess has been found as early as the third day. More commonly six to eight days elapse before a diminished tenderness and slight decline of swelling point to this formation. Appendicitis allowed to go on to suppuration—*i. e.*, not relieved by operation—usually terminates by rupture of the abscess into the peritoneum, followed by general peritonitis and death. The event is variously delayed by the extent and toughness of the protective adhesions which may have formed about the abscess. A few abscesses rupture into the bowel, thus saving the patient's life. Two or three cases in a hundred are thus saved. The fecal fistula incident in this termination usually closes eventually, though not always. In rare instances the abscess, especially if deeply situated in the pelvis, ruptures into the bladder. The termination in these cases is less favorable, 50 per cent. being fatal. A few also break through the groin, and are followed by recovery. Lumbar abscess and perinephric abscess must be mentioned as possible terminations, also infiltration of the abdominal walls and tissues of the thigh, pylephlebitis, and hepatic abscess.

General peritonitis may also ensue after perforation of the appendix. The symptoms of the resulting general peritonitis are those characteristic of this disease when suddenly induced by other causes, viz.:

1. Diffuse pain, as contrasted with pain localized in the right iliac region—pain of extreme severity.
2. Generally distended and tender abdomen.
3. Moderate fever, succeeded by normal or subnormal temperature, already alluded to as often misleading the physician.
4. Rapid and feeble pulse.
5. Dry and coated tongue.
6. The phenomena of collapse—*i. e.*, cold, clammy skin, feeble pulse, anxious expression, death.

Complications and Sequelæ.—The most important complication is obstruction of the bowels, by which is not meant the obstinate constipation so often met as an early symptom of appendicitis, but a true obstruction, the direct consequence of constriction by adhesions developed in the course of the peritonitis. It is one of the causes of death, as determined by autopsy, while operation frequently discloses conditions which could easily have produced obstruction.

Another complication is hepatic abscess from pylephlebitis, due to thrombosis and even embolism of branches of the portal vein; another is phlebitis of the right iliac vein causing milk-leg. In abscess of the liver it has happened that the diaphragm has been perforated, producing empyema and pyopericardium. Pyemic abscesses elsewhere in the system, including

the brain and lungs, have also been found in rare instances. Fecal, vesical, and umbilical fistulæ have been referred to. Fatal hemorrhage has also resulted from necrosis of the walls of the iliac vessels. Appendicitis may occur in a hernial sac.

Recurring and Relapsing Appendicitis. Chronic Appendicitis.—These terms are applied to cases of appendicitis which recur after a first attack. The terms are sometimes used interchangeably, but, strictly speaking, cases are *recurring* which repeat themselves at considerable intervals, as some months or a year or more; *relapsing*, when the attacks are very close—at intervals, say, of one or two weeks, so as to make them almost continuous. In the former, to which attention was first called by William Pepper in 1883, it is reasonable to believe that the patient has recovered in the interval or there exists a cystic appendix as an exciting cause. In the relapsing form it seems likely that there has not been complete recovery in the interval. Certain it is that one attack predisposes to another, so that, in at least 23 per cent. of cases observed, according to Hawkins, and 44 per cent. according to Fitz, it is found that there have been previous attacks. The symptoms of a recurrent attack are the same as those of a primary one. In many cases the interval between the attacks is passed in comparative comfort; in others, there is no small amount of pain or discomfort in the situation of the appendix. The term chronic appendicitis may also be applied to such cases. It must be remembered as pointed out by Deaver and by other surgeons who do much abdominal work, that chronic appendicitis may simulate almost any abdominal condition. Many cases of indigestion are in reality cases of appendicitis. Chrohic appendicitis constantly simulates gall stones, gastric ulcer.

The lesson to be learned is, that in all cases of disease of the upper abdomen, chronic appendicitis must be thought of, and in operation the appendix should be explored.

Diagnosis.—The diagnosis of many cases of appendicitis is easy, and becomes more so as experience grows. A certain number of cases must be carefully weighed, and in a few diagnosis is extremely difficult. *Sudden pain*, becoming localized, *tenderness*, and *rigidity* in the right iliac region are three symptoms, which, if present, point almost unmistakably to appendicitis, particularly if they are accompanied by leukocytosis, care must always be taken to exclude pneumonia and pericarditis which may have the same set of symptoms. A tumor in the vicinity of McBurney's point is less frequently present, though it is found in many cases, and greatly aids the diagnosis. The cases difficult of diagnosis are those in which these symptoms are vague or are in unusual situations. But, in truth, they are less often absent than has been supposed. More frequently they are not looked for, because there is very little to draw attention to them. A rule should, therefore, be made to search for them carefully in any persons subject to gastro-intestinal attacks, however induced and however manifested. It is certain that some cases of so-called catarrhal enteritis and chronic indigestion are really cases of appendicitis as stated above.

Differential Diagnosis.—Intestinal obstruction is a condition with which appendicitis has sometimes been confounded. The special symptoms of the various causes of obstruction, whether those of fecal impaction, of strangula-

tion by bands or twists, by intussusception, or by tumor or foreign body, should be recalled. Especially characteristic of obstruction is the absence of fever, unless the patient lives long enough to permit peritonitis to set up. In appendicitis there is almost always fever at the outset though it may abate later. The pain in obstruction is more intermittent at first, and though, like that of appendicitis, it may be anywhere in the abdomen, it is not likely to localize itself in the right iliac region. The constipation is more complete in obstruction, and even the passage of flatus is usually absent. The vomiting, also, is more severe and persistent, and is more likely to be stercoraceous. There is more general distention of the abdomen, and limited tenderness is less easily differentiated. *Intussusception* occurs more frequently in children younger than those subject to appendicitis, and is often attended with bloody discharges, which seldom occur in appendicitis, while a tumor may often be felt on examination *per rectum*. *Strangulation* by bands or twists is more common in adults. *Malignant growths* causing obstruction are usually in the left iliac region, although cancer of the cecum is to be remembered as a disease of the right. Its slower development distinguishes it from appendicitis. (See, also, Obstruction of the Bowels.)

Typhoid fever may be confounded with appendicitis, especially when there is tympany and prolonged tenderness in the right iliac region; but one has, as a rule, only to recall the mode of beginning of the illness, the gradual development of the fever, its greater intensity and peculiar diurnal variation, the spots at the eighth day, to say nothing of the Widal test, to be reassured in the majority of instances. On the other hand, there is nothing to prevent typhoid fever and appendicitis from accidentally coinciding.

Diaphragmatic pleurisy and especially pneumonia in children have been ushered in with symptoms identical with those of appendicitis, viz., abdominal pain, constipation, nausea and vomiting. Careful examination of the thorax for the physical signs of these affections may avert an unnecessary operation, and the local physical signs in the abdomen are frequently wanting.

Pericarditis may exactly resemble an attack of appendicitis, there may be sudden pain referred to the right iliac fossa.

A question which one would naturally expect to give rise to difficulty is that differentiating between appendicitis and the *pelvic affections of women* when on the right side, such as a suppurating ovarian cyst around a Fallopian tube, or a pyosalpinx. There can be no doubt that before our present accurate knowledge of appendicitis was acquired, numerous mistakes of diagnosis were made.[1] Many smpytoms are identical, but usually the location of the original pain in the appendicitis is not in the pelvic cavity or in close proximity of the uterus, even though it be not at McBurney's point or the right iliac fossa. The appendiceal abscess itself is usually limited to the neighborhood of the normal appendix and cannot be recognized per vaginam, while the pelvic abscess can. Should the appendix rupture, as it rarely does, into the vagina, the pus may be recognized by its stercoraceous odor. It should be remembered that appendicitis and pregnancy may be associated. The onset of suppurating ovarian cyst is much more gradual, and the pain

[1] For evidence of this, see an excellent paper by the late Paul F. Munde entitled, "Perityphlitis and Appendicitis in their Relations to Obstetrics and Gynecology," published in "Medical News," May 15, 1897.

more constant and duller. Pyosalpinx is in more intimate relation with the uterus, while the history differs from that of appendicitis.

Many cases of acute appendicitis were formerly mistaken for *acute indigestion*, but indigestion is unaccompanied by tumor or tenderness, while the vomiting is more persistent and the vomited matter differs. Gastro-enteritis may cause mistake. Persistent fever is more characteristic of gastro-enteritis. There is pain and tenderness but no rigidity or tumor. *Enterocolitis* occasions colicky pains, but there is no hardness or localization, while there is diarrhea with mucous stools. It will be remembered, however, that these symptoms sometimes attend appendicitis, and it should be remembered, too, that gastro-enteritis may be a favoring cause of infection of the appendix, indeed may be an actual cause, the result of an afferent wave of bacterial invasion from an irritated intestinal tract as suggested by Arthur J. Patek.[1]

Ptomain poisoning or food infection may closely simulate the symptoms of appendicitis, by abdominal pain, nausea and vomiting. The patient will, however, have taken food of the kind known to produce such illness, namely, lobster, sausage, ham, canned meats, cream puffs, old ice-cream and the like.

Acute Epididymitis (Right-sided).—Right-sided epididymitis sometimes gives rise to abdominal pain so severe, to fever and leukocytosis, that an operation may be contemplated. One glance at the testicle will make the diagnosis. We have seen a case prepared for appendiceal operation which was suffering from epididymitis.

In *hepatic colic* the pain is higher up, in the region of the gall-bladder, while jaundice is often present, and sometimes there is pain under the left shoulder; there is no fever. In *nephritic colic* the pain extends from the lumar region into the groin and testicle and blood or pus is found in the urine. A *floating kidney* with twisted ureter is movable, as contrasted with the iliac tumor of appendicitis; there is sometimes flattening of the corresponding lumbar region, while sudden relief of symptoms, which characterizes the untwist, is altogether peculiar. The presence of blood in the urine under these circumstances is confirmative of renal origin. In *pyonephrosis* there is tenderness in the region of the kidney, as well as pus in the urine. *Perinephric abscess* occasions tenderness in the lumbar region while the pain radiates into the groin, as in nephritic colic. It is to be remembered that perinephric abscess may be occasioned by suppurating perityphlitis, when the position of the appendix is posterior to the cecum.

Gastric or duodenal ulcer have usually a symptomatology of their own, hyperacidity, pain recurring long after eating, just when the gastric juice begins to flow over the ulcerated area. Relief from taking food. However, the first attack of pain may be due to a perforating ulcer. The pain is not usually severe as appendicitis but may be. The resistance and tenderness are usually above the umbilicus. There may be occult blood in the stool (this cannot be ascertained at the first visit). There is no leukocytosis.

Hyperacidity.—This is mentioned in this place because many of the supposed cases of simple hyperacidity are in reality gastric or duodenal ulcers, or appendicitis, or gall stones, and should not be confounded with these diseases.

[1] "American Medicine," April 1, 1902.

Appendicular colic is a vague condition of pain in this region, which has been ascribed to peristaltic contraction of the appendix, constituting an effort to expel fecal pellets and certainly occurs. A case has been described by one of us in which the pain occurred as the result of the attempt to expel some links of tapeworm.

It is sufficient for the physician to diagnose the existence of appendicitis without attempting to point out the particular variety of appendicitis, and while the possibility of such diagnosis cannot be denied serious errors have been made by those claiming such ability that little confidence can be placed in their claims.

Mention should be made of carcinoma of the cecum or appendix as presenting identical symptoms with appendicitis. It has occurred to us to make the diagnosis of appendicitis where operation showed the presence of cancer of the cecum.[1]

Prognosis.—It is a difficult matter to consider fairly the prognosis of appendicitis, or rather of the periappendicitis growing out of disease of ths appendix. For if we separate the cases which do not go on to suppuration, recovery from the immediate attack is apparently the rule. But a relapse is very likely to occur.

It is impossible to say of any case, however mild, that if left alone it will not terminate in suppuration, and a large number of cases still perish because of imperfect diagnosis and delayed operation. Chronic cases cause prolonged ill health.

Treatment.—*Appendicitis is a surgical disease.* As soon as the diagnosis is established—indeed, pending its settlement—a competent surgeon should be associated with the physician, for the reason that in practically all cases operative treatment is demanded. *The diagnosis being thoroughly established,* operation should be immediately performed in all cases in which any operation is permissible. The sooner an operation is performed after the onset of symptoms the less the mortality. During 1912 in Mayo's clinic 977 cases were operated and 3 died.

When to Operate.—The habit of waiting to observe whether a case will subside without operation is pernicious in the extreme. Operate at once. No one can by any single sign or set of symptoms tell the gravity of every case. There is no *sure* sign which distinguishes a catarrhal from a gangrenous appendicitis. Delay is much more dangerous than operation. All advanced thinkers of experience are agreed to these statements in very early cases.

There has within the last few years, however, arisen among surgeons, some difference of opinion as to whether immediate operation should be performed in late cases. In the hands of some expert surgeons this would appear to be safe, but the only safe rule for the general practitioner is to look upon every case not ill with some intercurrent disease, such as pneumonia, or any acute infection, alcoholism, etc., as an operative case. Do not meddle with so-called medical treatment. Operate at once.

It must be remembered that operation for appendicitis is a major operation and should be performed only by a surgeon of experience and training.

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[1] See also a paper on "Primary Cancer of the Tip of the Appendix," by J. Riddle Goffe," Medical Record," July 6, 1901.

The habit of young men doing operations of this character, before they have been surgically trained, is reprehensible.

Medical Treatment should be subject to the following rules. Do not use medical treatment for the purpose of reaching an interval, there may be no interval. Cases must occur, however, in which, from various complications, medical treatment is necessary. Operation may be declined even if urgently advised. A surgeon may not be immediately at hand, or some acute infection, advanced lung conditions, alcoholism, or heart disease will make the risks of an operation greater than the risks of waiting.

First of all, *absolute rest* in bed must be insisted upon as the first essential condition of abatement of the inflammation. Many a fatal case would have been saved had this been carried out. The patient should take nothing by the mouth, should be placed in Fowler's position and receive normal salt solution by the drop method or by the interrupted method. Purgatives should *not* be given. Cases which are doing well may become extremely severe by the use of laxatives or purgatives. Ice may be applied to the appendiceal region.

Next, *relief of pain* is demanded. Only when relief cannot be secured by the ice-bag or by hot fomentations should opium be given in moderate doses. One-eighth of a grain (.008) of morphin may be given hypodermically. There is no question here of masking symptoms or the diagnosis.

INTESTINAL OBSTRUCTION.[1]

Definition.—The words intestinal obstruction explain themselves. Obstruction to the descent of fecal matter is the fundamental idea, but the absence of alvine discharges, though common, is not essential. For in the course of our studies it will be found that in intussusception, for example, frequent loose bowel movements occur, and that in fecal obstruction they may be present throughout the whole course of the disease, while in other forms of obstruction they are not infrequent at the beginning. Intestinal obstruction is further divided into *acute* and *chronic*, according to the rate of development of its symptoms, the same causes at times producing acute, and at others chronic forms.

Acute obstruction is produced by strangulation, intussusception, foreign bodies, twists and knots, strictures, and morbid growths.

Chronic obstruction is produced also by stricture, intussusception, morbid growths, and fecal impaction.

I. Obstruction by Internal Strangulation.

Synonyms.—*Constriction of the Bowel; Hernia within the Abdomen.*

Definition.—By internal strangulation is meant stricture of the bowel by inflammatory bands or adhesions, by vitelline remains, omental or mesenteric slits, adherent appendix, and the like.

Occurrence.—This is probably the most frequent cause of acute intes-

[1] Reginald H. Fitz's able paper in the "Transactions of the Congress of American Physicians and Surgeons," 1899, Leichtenstern's article in Ziemssen's "Cyclopædia of Practical Medicine," and Frederick Treves' book on "Intestinal Obstruction," 1884, are important modern papers to which I am indebted for much of the matter in this section.

tinal obstruction, though intussusception closely approaches it in frequency. Thus, Reginald H. Fitz found it in 35 per cent. of 295 cases of obstruction, as against 32 per cent. of intussusception.

Etiology.—The causes of strangulation have been carefully worked out by Fitz in his 101 cases, collected from reports since 1880. Of these in 84 the strangulation was caused by *bands and cords*, of which 63 were simple inflammatory bands or adhesions and 21 were vitelline remains, represented by Meckel's diverticulum,[1] or by the persistent remains of vitelline blood-vessels. Meckel's diverticulum is usually attached by these remains to some part of the abdominal wall near the navel or to the mesentery, or it may be adherent because of peritonitis. The persistent vitelline vessels may themselves be the strangulating cord in the absence of Meckel's diverticulum. Other causes were *adherent appendix*, *mesenteric*, *omental slits*, *peritoneal pouches*, *adherent Fallopian tube*, and *pedunculated tumor*. To these must be added *diaphragmatic hernia*. This was the cause of strangulation in ten per cent. of Leichtenstern's cases, but Fitz found none reported between 1880 and 1888. Two cases of diaphragmatic hernia were reported by Tyson in 1893,[2] both of some standing, the immediate cause of death being acute strangulation.

The seat of the strangulation is in the small bowel in a decided majority of cases—nearly 90 per cent. In 83 per cent. the strangulated part lay in the lower abdomen, and in 67 per cent. in the right iliac fossa.

Seventy per cent. of cases occur in males, and at least 40 per cent. between the ages of 15 and 20, the causes in these being inflammatory adhesions twice as often as vitelline remains. Strangulation in early youth is relatively uncommon, and when it does occur, it is usually caused by vitelline remains.

II. Intussusception—Invagination.

Definition.—In this condition one part of the bowel has slipped into another, always from above downward, and may readily be illustrated by slipping one part of a coat sleeve into another.

The external or receiving portion, known as the *intussuscipiens*, has its mucous surface in contact with the mucous surface of the middle or intermediate portion, whose peritoneal surface is in contact with the peritoneal surface of the internal or returning portion, while the two mucous surfaces of the returning portion are apposed. The internal and middle part are called the *intussusceptum*. The resultant is a cylindrical tumor which varies from half an inch to a foot or more in length. The annexed diagram gives a very good idea of the different parts of the tumor.

Intussusceptions may occur in any part of the bowel from the duodenum to the rectum, and are named in accordance with the part of the bowel involved. According to Leichtenstern, 52 per cent. are ileo-cecal and ileo-colic, 30 per cent. are enteric, and 18 per cent. rectal and colico-rectal.

It will be remembered that intussusception is almost, if not quite, as

[1] Meckel's diverticulum, a remnant of the omphalo-mesenteric duct, through which, in the early embryo, the intestine communicates with the yolk-sac, is a finger-like projection from the ileum, usually within 18 inches of the ileo-cecal valve. The length of this tube is on an average three inches, while it has attained at times a length of ten inches.

[2] "Transactions of the Association of American Physicians," 1893.

frequent a cause of obstruction as strangulation, under which the percentages were given.

Etiology.—Diarrhea and habitual constipation are probable exciting causes, having preceded in 13 and 12 cases respectively out of 51. Other possible causes are so infrequent as to be unworthy of mention. Experiments with faradism would seem to show, however, that spasm plays a more important rôle than relaxation.

As to sex, two-thirds are found in males and one-third in females. It is especially an accident of the young, occurring in 34 per cent. under one year and 56 per cent. under ten years.

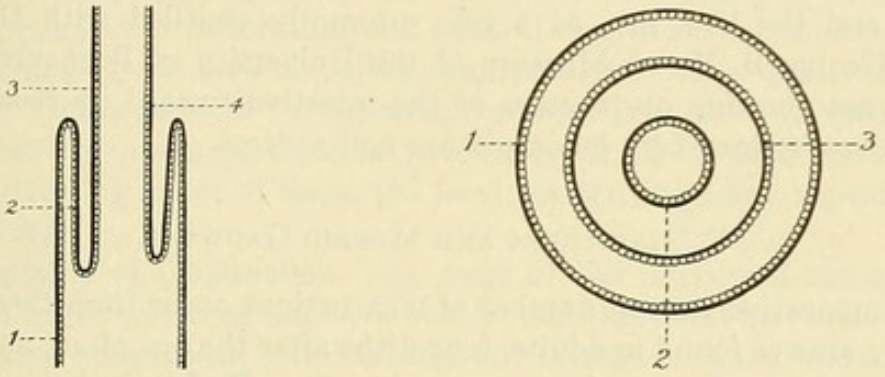


FIG. 101.—Vertical and transverse Sections of an Intussusception.
1, The Sheath, or Intussuscipiens; 2, The Entering, or inner layer; 3, The Returning or middle layer.

Intussusception of the dying should be mentioned in passing, as a form of intussusception which often takes place a short time before death, more frequently in children, and is probably caused by certain irregular peristaltic movements toward the end of life. It produces no symptoms during life.

III. Twists and Knots—Volvulus.

The majority of cases are axial twists—*i. e.*, the bowel is twisted on its mesenteric axis—this being the case in 40 out of Fitz's 42 cases, two only being knots. Eighty-seven per cent. of cases occur in the large intestine, the remainder in the small intestine, one-half are in the neighborhood of the sigmoid flexure, and nearly one-third in the ileo-cecal and cecal region.

It is more frequent in males in the proportion of two to one. Most cases occur between the ages of 30 and 40.

IV. Obstruction by Abnormal Contents or Foreign Bodies.

The majority of these are *gall-stones*—23 cases out of 44 of obstruction by foreign bodies collected by R. H. Fitz; 19 were fecal impactions and two enteroliths. Obstruction by gall-stones appears to be three times as common in females as in males. They enter the bowel usually by ulcerating through the gall-bladder, commonly into the small intestine, more rarely into the colon.

The seat of obstruction by gall-stones is most frequently the ileo-cecal region; after this lodgments are in the small intestine, with diminishing

frequency as we ascend. The ages are pretty uniformly distributed from eight to eighty. One of the enteroliths was made up of shellac, found in a man who had been in the habit of drinking alcoholic solution of shellac. Usually, enteroliths are made up of triple phosphate of lime and magnesia, about a nucleus which may be a mass of hair or other foreign body. Courvoisier collected 131 cases, in 70 of which the stone was spontaneously passed *per anum*. Some were very large. Six were found in diverticula or in the appendix. A coil of lumbricoid worms has caused obstruction, as has the accumulation of certain medicines, such as magnesia and bismuth.

In a few instances obstruction is caused by substances introduced by the mouth, but the objects thus introduced, as pennies, buttons, pins, fruit-stones, and the like, are, as a rule, promptly expelled with the stools. In the George B. Wood Museum of the University of Pennsylvania is a plaster cast showing obstruction of the intestine toward its cecal end by plum-stones, followed by inflammation and abscess.

V. Strictures and Morbid Growths.

A comparatively small number of obstructions occur from these causes. They are always found in adults, four-fifths after the age of 40, and are apparently twice as common in women as in men. By far the largest number is met in the large intestine and lower abdomen, the majority being in the left iliac fossa.

Strictures may be (1) Congenital, illustrated by imperforate anus and defective union between the pylorus and duodenum.

(2) Cicatricial, from healed ulcers. Tubercular ulcers in their healing have produced decided and fatal obstruction, especially in the rectum. Syphilis is also though to produce stricture in the same locality.

Of morbid growths, the most frequent is the cylinder-celled epithelioma, which may form a ring in the vicinity of the sigmoid flexure, where colloid cancer is also met. Any of the varieties of benign tumors may produce obstruction, while inflammatory processes external to the bowel, especially in the pelvis, may cause obstruction by pressure from without.

VI. Fecal Obstruction.

Synonym.—*Ileus paralyticus vel nervosus.*

Occurrence.—Fecal obstruction occurred 19 times in Fitz's 44 cases of obstruction by foreign bodies. It is more frequent in females and in adults, especially in the aged. It occurs more frequently in the large intestine, especially in the cecum, and in the lower part of the bowel. The fecal tumors found in appendicitis are now regarded as the result of the inflamed appendix, rather than as the cause of the cecal inflammation.

A local peritonitis may also be developed about the paralyzed and distended intestine. Mention is made under chronic constipation of the enormous masses of fecal matter thus accumulated. The wall of the intestine above the accumulation may also be hypertrophied because of the propulsive efforts of the muscular coat.

Etiology of Fecal Impaction.—Fecal impaction is favored by constipation and its causes, although a tendency to fecal obstruction is sometimes congenital. Nervous influence is not to be ignored; the tendency to constipation is seen in the chronic insane, in the hysterical and hypochondriacal, and in affections of the spinal cord. Chronic enteritis and chronic peritonitis favor it; so may anatomical peculiarities of the colon. These causes weaken the muscular coat which moves the contents of the bowel onward, resulting ultimately in an absolute paralysis of a segment of the bowel, arrest of motion of contents, and finally obstruction. The plug of fecal matter grows harder and larger, and compresses and stenoses the adjacent bowel, resisting any further onward movement, and increasing the impediment to the restoration of a natural condition, culminating, finally, in stretching of the muscular fibers and paralysis—*ileus paralyticus*. The so-called "stercoral ulcer" of the cecum, on which the older writers laid much stress, and which was ascribed partly to gangrene, due to pressure, and partly to the irritating effect of impacted fecal matter, is to-day regarded as extremely rare.

Symptoms of Obstruction.—As most of the important symptoms are common to the different causes of obstruction, they will be considered from the general standpoint, emphasizing any special relation which a given symptom may bear to a special cause. In addition to the usual absence of bowel movement there is:

First, *abdominal pain*. This is the most constant of all symptoms, being present in a decided majority of cases of obstruction from whatever cause. The pain is one of the earliest symptoms in every form of acute obstruction. It is usually sudden and very severe, and may be intermittent or constant with exacerbations. It may occur in any part of the abdomen, regardless of cause, though most frequent in the neighborhood of the umbilicus, so that its location is of no diagnostic value.

Nausea and *vomiting* are almost as frequent. The vomitus at the onset consists of the food last taken, but soon becomes bilious, yellow, and finally fecal. Vomiting is relatively infrequent in strangulation and intussusception, while it is relatively frequent in volvulus, stricture, and tumor. The vomitus is especially apt to become fecal when caused by strangulation—usually from the third to the fifth day.

Tympany is next in frequency. It is a symptom of later occurrence than pain and vomiting, presenting itself usually from the second to the sixth day. It varies greatly in degree, increasing as a rule with the duration of the obstruction and being sometimes enormous. It is of least importance in obstruction by intussusception, and most marked in volvulus. It is sometimes, but not always, accompanied by tenderness.

Inability to pass flatus is as constant as the absence of bowel movement.

Tenesmus is a frequent symptom when there is obstruction in the large bowel, as in 15 per cent. of cases of volvulus and 55 per cent. of acute intussusception. *Fecal vomiting* succeeds in some cases.

Tumor, under which are included circumscribed visible intestinal coils as well as swelling characterized by absolute dullness, is a rare symptom except in intussusception, when it is characteristic, having been present in

69 per cent. of Fitz's cases, more particularly when in the large intestine, where it is also sometimes associated with a relaxed sphincter. The tumor of intussusception is more frequently found in the left iliac region in the descending part of the large bowel, because the invagination extends in that direction and often does not form an appreciable tumor till that part of the bowel is reached. Tumor occurs sometimes in obstruction by foreign bodies. In strictures, morbid growths, and invagination it may be recognized by rectal examination. Tumors with dullness on percussion are not seen in twist, though visible coils are sometimes present.

Fever is infrequent—in fact, its absence is rather characteristic, especially in the beginning. Records of elevated temperature are, however, found in from 22 to 28 per cent. of all cases, the maximum record being 102° F. (38.9° C.).

Hiccough is an occasional symptom, and appears to be more frequent in volvulus. *Jaundice* is often found in obstruction by gall-stones.

The *urine* has been irregularly studied in acute obstruction. It is not infrequently spoken of as scanty and containing an increased amount of indican, especially in obstruction in the small intestine, not, it is said, of the large. Albumin is rarely present. It is to be remembered that peritonitis causes an increased indican reaction.

Tumultuous peristalsis is frequent above the seat of obstruction.

Bloody stools and *tenesmus* may be present when there is intussusception. They are important in the diagnosis, the former occurring in three-fifths of the cases, the latter in two-fifths. They may occur early or late. Blood-stained stools also occur in connection with cancer of the lower bowel.

Local *peritonitis* is caused by *volvulus* and *obstructing gall-stones*, caused, in the latter case, rather by the destructive results incident to the passage of the stone into the bowel from the common duct.

Collapse is the terminal symptom in fatal cases, due to the profound impression on the nervous system, and presents the lowered temperature, leaking skin, and feeble pulse characteristic of collapse from other causes. Cases are reported in which operation during collapse was followed by recovery.

The same train of symptoms may succeed *stricture* and *tumors*, to be followed at times by partial relief, which is a gain succeeded by similar symptoms leading to ultimate total obstruction and death. Such symptoms will, of course, be associated with the anemic dyscrasia and emaciation which belong to the causing diseases, and which more frequently lead to death without obstruction than with it. Meteorism in the right inguinal region is said to be more or less characteristic of obstruction by Meckel's diverticulum.

In *chronic obstruction* due to fecal impaction, more rarely to stricture, cancerous disease, or foreign bodies, these symptoms are less marked and succeed each other more slowly. Though initial symptoms may occur suddenly. *In fecal impaction*, what appears to be simple constipation at first is succeeded by permanent retention, which may last for weeks without causing inconvenience. Examination *per rectum* will often disclose this tube filled with hard fecal matter which may be cleaned out with the finger or a spoon-handle. There may even be *diarrhea*, due to irritation of the

bowel above the impaction, when the catarrhal secretion may channel out the mass and carry a portion with it. Gradually, however, the impaction becomes impregnable to all remedies, natural and artificial, the abdomen *swells*, there are *fullness* and *weight* within, and *pain* in the genitals or thigh from pressure on the sacrolumbar nerves; the *appetite* fails, the *tongue* is coated, and the *breath offensive;* sometimes a condition of *lethargy* and *indifference* supervenes along with *great weakness*, and the patient dies of exhaustion. At any time, on the other hand, may follow with suddenness the train of symptoms already described—pain, tympany, nausea and vomiting, ultimately of fecal matter, with collapse and death.

In many cases of impaction sooner or later, a *fecal tumor* presents itself —a tumor formed by the mass of retained feces, chiefly in the right iliac fossa, the region of the cecum, corresponding to the outer half or Poupart's ligament. It is sometimes hard, at others soft and yielding, and sometimes tender and painful, probably because of a mild local peritonitis. In the ascending colon the tumor is soft, and in the hepatic flexure it may give rise to the notion of an enlarged liver. It may move in the more loosely attached parts of the colon, and may drag the transverse colon down toward the pubis. In the descending colon and sigmoid flexure it is usually harder, and may be subdivided into scybala. It is, of course, easier of detection in persons with thin abdominal walls, and may be obscured by flatulent distention. When recognized, it is of great diagnostic value. Such tumors have been mistaken for tumors of the stomach, liver, spleen and kidneys, and for pregnancy.

Diagnosis.—The importance of early and correct diagnosis is intensified at the present day by the fact that operative interference promises by far the best results, while to be effectual it must be early. The diagnosis has three principal objects: first, the existence of *obstruction per se;* second, its seat, and third, its *cause*. The first is by far the most important, as operation is indicated in one variety or situation almost as much as in another.

First, as to the *presence of obstruction* in general, the absence of bowel movements, the presence of abdominal pain and tympany are suggestive symptoms. In the beginning of obstruction increased peristalsis is marked, later it disappears. As to differential diagnosis, it has happened that a case of intense *enteritis* has presented all the symptoms of obstruction. Fever is commonly present in such enteritis, while it is absent in chronic obstruction, at least at first. Enteritis is characterized by diarrhea, obstruction by constipation.

Acute poisoning associated with vomiting, such as is caused by poisonous mushrooms, biliary, renal, and intestinal colic, the pain caused by twisting of the ureter in a movable kidney, all present symptoms more or less like those of obstruction; but the combination of signs necessary to the picture of obstruction is still wanting.

Much more common is the mistaking of *appendicitis* for obstruction. In this there are pain, vomiting, and constipation, as well as tumor in the neighborhood of the cecum, but the differentiation between these two conditions was considered when treating of acute appendicitis. *Peritonitis* itself presents symptoms common to it and obstruction, including abdominal pain, distention, constipation, and collapse, with increase of indican. But

the presence of fever, the absence of tumor and of fecal vomiting, point to peritonitis.

Acute dilatation of the stomach is frequently taken for obstruction of the bowels. The large area of tympany over the stomach will make the diagnosis and passage of a stomach tube will be of more certain value. If the distention is in the stomach it will disappear by the passage of a stomach tube.

The symptoms of incarcerated *hernia* are also those of intestinal obstruction, and in all cases careful search should be made for a concealed hernia. Such herniæ have been found in the external ring and in the obturator foramen at autopsy by William Osler, who has also met a case of acute *hemorrhagic pancreatitis* presenting the symptoms of acute obstruction.

Having excluded hernia by a careful examination for a seat of strangulation, examination *per rectum* should be made, also *per vaginam*. By either method a tumor may sometimes be recognized. Especially is this true of a tumor caused by intussusception. A stricture may also be detected by digital examination of the rectum, as may obstruction by foreign bodies.

On the other hand, the rectum may be totally empty of feces and continue so, whence it is probable that the obstruction is in the small intestine or high up in the large. The position and size of the uterus and ovaries may also be ascertained by rectal examination. The rectum can be more thoroughly explored by suitable specula in the knee-elbow position. The hard rectal tube has produced perforation, while the flexible tube so coils itself up as to be valueless in diagnosis. Sometimes, if the distended intestine is filled with hard fecal matter, it can be felt as an uneven mass in the course of the bowel.

Moderate distention in the upper part of the abdomen, with flatness below and in the sides, rapid collapse and oliguria, point to obstruction in the duodenum and jejunum. Such distention is temporarily diminished by vomiting, but is uninfluenced by fecal discharges secured by enemas. Nor is the vomiting always fecal in duodenal and jejunal obstruction; when the obstruction is in the ileum and cecum the distention is more central, the region of the colon being flatter until covered in by the extending tympany, and the vomiting is more likely to be fecal.

When obstruction is seated in the colon tympanitic distention is greatest, yet the difference between it and that of obstruction in the ileum is not so great as to possess much diagnostic value. If in the lower colon, there may be tenesmus and discharge of blood and mucus. Measuring the capacity of the large bowel by air, gas, or water has been recommended as an aid to diagnosis. These substances may be made to pass the ileo-cecal valve.

Third, the presence of obstruction being recognized, the *nature of the obstructing cause* may sometimes be determined with a degree of probability. First to be considered is the relative frequency of the different morbid states. Adopting Fitz's figures, strangulation and intussusception together make up 70 per cent. of *all* cases, the two being nearly equal. After that come volvulus with 15 per cent., gall-stones with eight per cent., and stricture or tumor six per cent.—that is, the twists about equal obstruction from gall-stones and tumor and stricture together.

Again, if the obstruction be found in the *large intestine*, it is more likely

to be intussusception, twist, or stricture and tumor, since of the obstructions in the large bowel 51 per cent. are intussusception, 30 per cent, twists, and 12 per cent. stricture and tumor. If in the *small* intestine, it is most likely strangulation or gall-stone obstruction, since 72 per cent. of obstructions in the small intestine are strangulations and 14 per cent. gall-stones, leaving eight per cent. only for intussusception, five per cent. for twists, and one per cent. for stricture and tumor. If the attack has been preceded by one of jaundice or by other liver symptoms, as hepatic colic, it is almost certain to be gall-stone, especially if the patient be over 50 years old.

If the patient is under 30, particularly if a child, it is more likely to be *intussusception* than twist, while if there are palpable abdominal tumor, bloody stools, and rectal tenesmus, the case is almost sure to be intussusception, rendered still more likely if the rectum has a large capacity for water, since intussusception is found near the cecum in 75 per cent., while twist is found near the sigmoid flexure in 50 per cent. Of all forms, intussusception presents the clearest clinical picture and is most easily recognized.

Twist, cancer, and stricture are more apt to be below the sigmoid, and the last two may sometimes be felt by the finger. In point of fact, twist in the large bowel is not often recognized. It is a disease of the adult, rarely occurring under 40; vomiting is less early and less severe than in strangulation by bands. Pain is often severe in twist. Some degree of local peritonitis almost invariably results, causing rigidity of the abdomen, while meteorism appears early and is extreme, the distended intestine often displacing the solid viscera.

If there is a history of previous peritonitis, *strangulation* becomes more likely, since such inflammation precedes in 68 per cent., while there is also a history of previous attacks in 12 per cent. The pain in strangulation is early, sudden, and severe, and the same may be said of vomiting. It becomes stercoraceous in 60 per cent., while the vomiting affords no relief. There is little or no distention unless peritonitis supervene. There is great prostration, and no tenesmus or discharge of blood. The average duration is about five days. The presence of *diaphragmatic hernia* as a cause of internal strangulation must not be overlooked; it is almost always the result of severe injuries. The half of the thorax containing the viscera is distended and tympanitic on percussion, while breathing movement is restricted, the breath-sounds are feeble, the vocal fremitus and vocal resonance diminished or absent—signs shared with pneumothorax. The pitch and intensity of the percussion note vary also with the degree of distention and the position of the viscera invading the thorax, while there may be metallic tinkling of fluid in the intestine, due to peristalsis.

Obstruction by Meckel's diverticulum is said to be indicated by meteorism in the right inguinal region.

Fecal obstruction is recognized by the symptoms already described under chronic obstruction, and such recognition is not very difficult, especially if the fecal tumor is found. Sometimes, however, on account of its insidiousness, fecal obstruction is overlooked when presenting only the more chronic symptoms, and the patient dies of supposedly unknown cause when accurate and careful study would have led to its discovery.

Prognosis.—In fecal tumors alone, of all the causes of obstruction considered, is the prognosis favorable if the condition is recognized sufficiently early, while a considerable latitude of duration may also be allowed.

Treatment.—Obstruction of the intestine is a surgical disease. Early diagnosis, best made in consultation with an abdominal surgeon and early operation before the patient is exhausted offer the only measures for relief.

Treatment of Intussusception.—It is dangerous to attempt to reduce an intussusception by inflation or irrigation, but the method may be tried in very recent cases. If irrigation is decided upon, the fluid—salt solution at 100° F. (37.8° C.)—is best allowed to pass into the large intestine slowly by its own weight through a long tube carried high up, the reservoir being raised no more than 2 1/2 feet (0.75 meter) above the etherized patient. A higher level than this for the reservoir may result in rupture of the bowel, while the bowel may also kink if the fluid be allowed to enter too rapidly. Some place the patient head downward over the back of an inverted chair, suitably covered with a bolster and quilts. Others hold that inversion is unnecessary. The nozzle should be closely fitted to the anus, accomplished by simply compressing the buttocks. One hand should be kept flat on the abdomen, while variations of pressure should be avoided. If success is not attained after a short trial laparotomy should be done. Long manipulation may rupture the bowel or allow gangrene to occur at the seat of intussusception.

Give *no aperients*. To relieve the excessive vomiting after the simpler remedies have been tried, the *stomach* may be *washed out* as suggested by Kussmaul and described under gastritis. This is at once a harmless measure and may be efficient for the purpose intended. It may be done three or four times a day. It may be expected to be of service in the vomiting of any variety of obstruction. *Opium* may be administered hypodermically to allay the intense pain, and may also relieve the vomiting. Or it may be given with a view to cure, which there is reason to believe it has accomplished in cases of intussusception and even strangulation. Opium may, on the other hand, be harmful, by obscuring diagnosis and producing an appearance of relief, while the local condition of the bowel is really growing worse. This should never be given before a diagnosis is made.

The *nourishment* of the cases demands careful thought. It is irrational to continue the administration of nutriment by the mouth when it is rapidly rejected. If the obstruction is in the small bowel the rectum should be the only route employed, while ice should be administered freely by the mouth. On the other hand, when the obstruction is in the colon, when tenesmus and diarrhea are symptoms, and when vomiting is a less prominent symptom, small amounts of liquid nourishment may be introduced by the mouth.

Treatment of Fecal Tumor.—The situation is altered when a diagnosis of fecal tumor has been correctly made. Here nothing is so efficient as *repeated large injections* of warm water, high up and retained for from 10 to 15 minutes if the patient can retain them, as he should be encouraged to do. Good results are sometimes obtained from the coincident use of small doses of calomel, 1/8 to 1/5 grain (0.008 to 0.013 gm.), given hourly.

If the fecal impaction is low enough down in the rectum, in most cases the finger covered with a rubber glove or some mechanical appliance, as a spoon-handle, can be used to loosen it.

Twist, strangulation by bands and *obstruction by gall-stones* can only be relieved by operation, and a surgeon should be associated in the treatment from the outset.

For treatment of *cancer of the bowel* see section on that affection.

CONSTIPATION.

SYNONYM.—*Costiveness.*

Definition.—Unnatural retardation or delay in the natural evacuation of the bowels.

Though there may be some exceptions, an evacuation of the bowels once in 24 hours seems to be nature's law in the case of the adult human being, and any prolongation of this interval may be said to constitute costiveness. A popular application of the term is also, however, to a condition in which, though there may not be infrequency of stools, the dejecta are dryer and harder than natural and are discharged with more or less difficulty and pain. The physician should appreciate this, otherwise misunderstanding may arise as to the exact meaning of the patient. Constipation is also something different from retention due to obstruction by various causes. The interval between bowel movements in constipation varies greatly, ranging between a couple of days and weeks. Many constipated persons have no dejections unless aperient medicine is taken.

Morbid Anatomy.—There are no morbid changes characteristic of constipation. Dilatation of the colon in various degrees is present, sometimes enormous, as shown in Fig. 102, and there may be found the remnants of inflammatory or other local lesions which may be responsible for the obstruction. The large accumulations of fecal matter found in these cases are known as *coprostasis*.

Etiology.—The immediate causes of constipation are:

1. Atony of the colon, whence results a slow peristalsis. Perhaps the most common cause of atony is a habit, engendered through indifference or necessity, of disregarding nature's call for relief. Repeated disregard of such call results sooner or later in disappearance of inclination. Sedentary habits cooperate to produce such disinclination. Atony may also be the result of disease of the bowel and of general disease causing debility, such as anemia, chlorosis, and protracted illness, like typhoid fever.

2. Overdrugging. Nothing is more common than the pernicious habit of drug taking for constipation. It assures a chronic condition.

3. A deficiency of the natural stimuli to peristalsis afforded by various secretions, especially the bile.

4. A loss of muscular power in the abdominal walls from overdistention or obesity.

5. Improper food. The foods which most stimulate peristalsis are vegetables, especially those with an insoluble residue, such as is afforded by the outer coatings of grain. Foods of an opposite kind are represented by milk and the farinacea.

6. Finally, stricture and displaced organs—such as the uterus—and the colon tumors, and foreign bodies impinging on the bowel and delaying the descent of the feces, become causes.

Among consequences of fecal impaction are hemorrhoids, which result from pressure on the hemorrhoidal veins.

Treatment.—Every case of constipation should be carefully studied with a view to determining its cause, and to removing it when possible. If such cause is not found, the first injunction in the management of constipation is the observance of *regularity in going to stool* at a fixed hour of the day, whether inclination prompts or not. The usual hour for this purpose is immediately after breakfast, though it matters not much when it is, so that it is regularly observed. Especially harmful is it to disregard any inclination which may appear at this time, or, indeed, at any time. Next is the use of *food* of the kind referred to under the head of etiology, such as fresh green vegetables of all kinds and succulent fruits.

Of breads, the so-called "brown" or bran bread, or glutten bread, is to be preferred. With such food should be conjoined massage of the abdomen or compression, either by the patient himself or by another. Preferably by a trained masseur. This is a most valuable measure. A very excellent daily practice is to flex the body forward and as far as possible backward, a number of times while in the standing position. This has the effect of compressing the bowels and stimulating peristalsis, and is one of the most useful aids. It should be practised once or twice a day: in the morning and at bedtime. Rising to a sitting posture while lying on the back with the feet fixed is another exercise helpful in the correction of constipation; so is twisting of the body while standing. Daily exercise, including horseback riding, golf, and tennis, has an important influence in correcting constipation. We have known dancing also to be serviceable. The free use of plain water is sometimes sufficient to overcome the milder cases. Thus, a glass of water may be taken before breakfast and another at bedtime.

Last of all should aperients be employed. Unfortunately, these are often necessary. The simplest and least irritating should be employed. A simple tonic pill composed of 1/3 to 1/2 grain (0.022 to 0.033 gm.) of the extract of nux vomica and 1/12 to 1/8 grain (0.005 to 0.008 gm.) of the extract of belladonna, three times a day, and kept up for some time in connection with the dietetic measures alluded to, is often sufficient.

But of actual aperients, the natural mineral waters are deserving favorites, especially Friedrichshalle, Apenta, Hunyadi János, and Carlsbad. and, when less active waters are required, the American Saratoga waters. The Saratoga waters are saline waters which present quite a range of proportion in their constituents, chiefly sodium chlorid, at the various springs, The waters of the Bedford Springs, of Bedford, Pa., are also very efficient, stimulating, as does the Saratoga water, the secretion of bile. The doses of all of these waters vary so much with circumstances that it is impossible to indicate them with definiteness. The minimum dose of the foreign aperient waters mentioned is 2 fluidounces (60 c.c.), increased to 8 fluidounces (240 c.c.). Less than the latter quantity of the American waters is seldom used at a dose.

Of drugs, cascara sagrada has become deservedly popular. The best

preparation is the fluid extract, as its dose can be readily regulated. From 10 to 30 minims (0.6 to 2 c.c.) may be given after the evening meal, and if this should prove insufficient, the same dose after the midday meal is to be preferred before increasing the evening dose. The solid extract is, however, also efficient, and a grain or 2 (0.066 to 0.13 gm.) more may be added to the laxative pill already mentioned, or, if a more active aperient is desired, as many grains of extract of colocynth may be substituted.

An old favorite, a pill composed of extracts of aloes, nux vomica, and belladonna, in varying proportions, to be taken at bedtime, has been largely substituted of late by another made by the manufacturers and pharmacists, of aloin 1/5 grain (0.013 gm.), strychnin 1/60 grain (0.0011 gm.), and belladonna 1/8 grain (0.008 gm.), of which one or two are a dose. To such a pill podophyllin, in doses of 1/4 to 1/2 grain (0.0165 to 0.033 gm.), may be added with advantage, or blue mass in doses of 1/2 grain to 2 grains (0.033 to 0.132 gm.), or rhubard 1 to 2 grains (0.066 to 0.013). The belladonna may be substituted by the extract of hyoscyamus, of which 1 to 2 grains (0.066 to 0.132 gm.) may be given. The compound licorice powder in which senna and sulphur are the active ingredients is a favorite aperient with some, but is bulky, and has a tendency to cause griping. The dose is a dram (3.8 gm.) or more. Phenolphthalein in doses of 1 1/2 to 7 1/2 grains (0.1 to 0.5 gm.) in powder or tablet form is a good aperient.

A glycerin suppository or 1/2 dram (2 c.c.) of glycerin injected has become a favorite means of securing an evacuation. It should be remembered as a possible remedy, but it acts by irritating the lower bowel and soon loses its effect. The enema of plain water, 1 to 2 pints (500 to 1000 c.c.), though less convenient, is to be preferred, and some persons use it regularly. None of these measures is curative. They simply empty the bowel at the time, and systematic effort should be made to reduce them gradually, while the hygienic treatment is kept up.

It sometimes happens that an impacted fecal mass becomes channeled, and fecal matter may descend from above through it, and thus lead to the belief that normal passages are being secured. The physician should explore the rectum with the finger, and by means of it or the handle of a spoon clear out the mass. This is often absolutely necessary before an evacuation can be secured.

Treatment of the Constipation of Infants.—This is best overcome, when possible, by simple small enemas repeated until an effect is produced, and carried out at a fixed hour each day, preferably in the evening. The child is best held on the mother's lap, properly protected by a mackintosh and a small quantity, say 2 ounces (60 c.c.), of tepid water is thrown into the rectum. If it returns unchanged, after a few minutes' delay, another syringeful is thrown in, and, if necessary, another. Ultimately, a fecal discharge is usually thus obtained. Regularity of this performance is important. It may be necessary to add a little soap to the hot water. Sometimes slight titillation of the anus by twisted pieces of paper answers every purpose. At the same time, the belly of the child should be massaged by the mother. Small suppositories of soap or of glycerin may be used if the measures mentioned are inefficient. For simple constipation in infants it is preferable to administer nothing by the mouth if it can be dispensed with.

Dilatation of the Colon.—This is one of the consequences of chronic constipation, though it may also occur as an acute condition, the result of sudden obstruction, as by a twist in the meso-colon. It may involve the whole colon, but the vicinity of the sigmoid flexure is its usual seat. Two

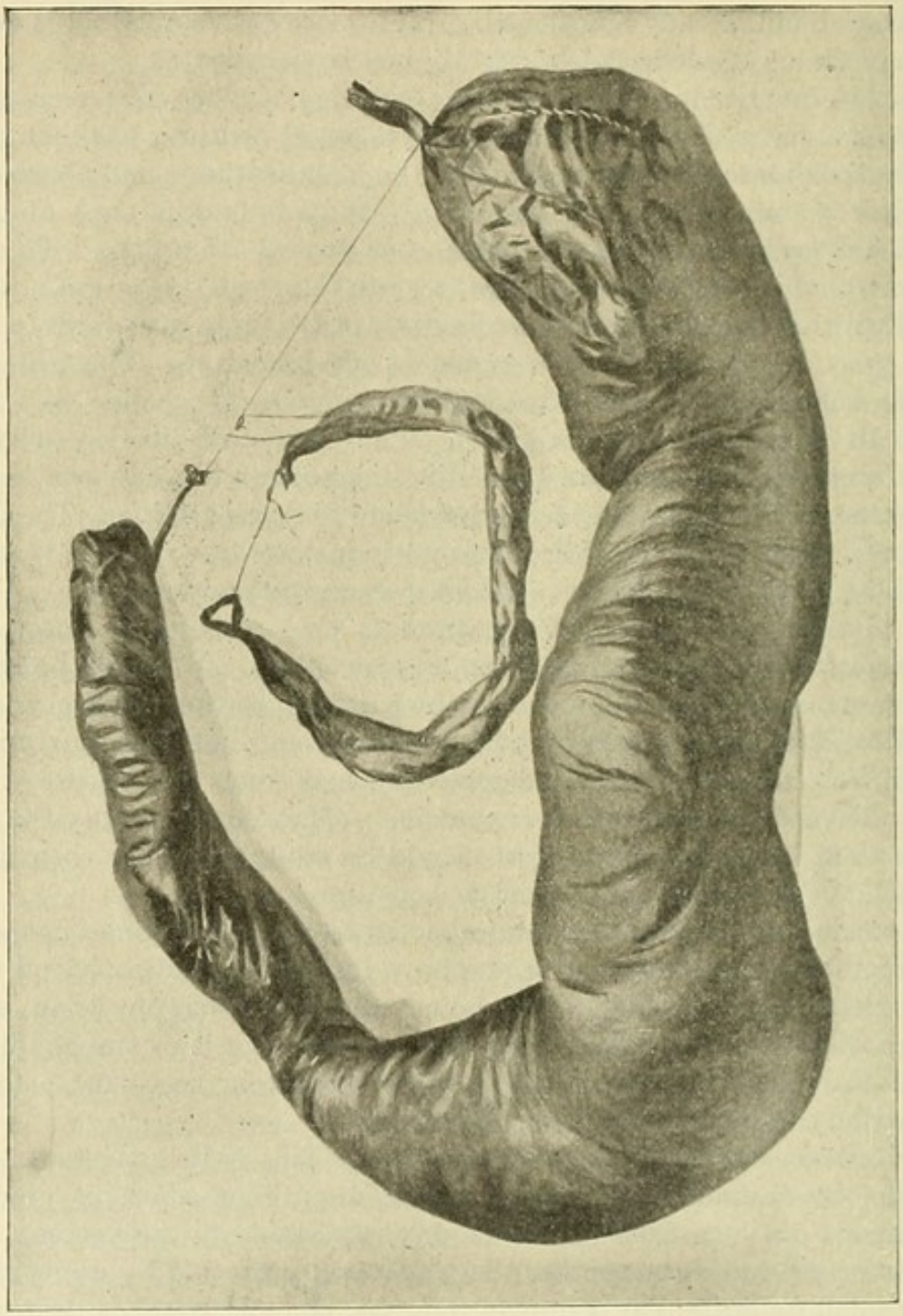


FIG. 102.—Giant Congenital Dilatation of Human Colon.

The more distended end is the sigmoid flexure. The narrow part taking exit from it is part of the rectum, which was normal. The narrow distal end of the preparation represents the head of the colon with the string attached to a fragment of the small intestines. The arched part of the specimen represents a normal human colon photographed simultaneously for comparison of dimensions. Both were dried preparations.

classes of cases of idiopathic dilatation are met—first, that of adult males, generally over 50 years of age; second, that of children in whom abdominal symptoms have been present more or less since birth. In the former it is thought that the overloaded sigmoid hanging into the pelvis and bent on

itself becomes occluded and responsible for dilatation. The form met in children is usually congenital and involves the lower portion of the colon, which is also hypertrophied. The congenital form becomes the direct cause of chronic constipation or coprostasis, which in turn increases the dilatation. Such is a remarkable specimen in the museum of the University of Pennsylvania, secured by the late Henry F. Formad[1] in the course of his work as coroner's physician. Two and a half pailsful of feces, weighing 40 pounds (20 kilograms), were removed at autopsy.

Symptoms.—They are the same as those of obstinate constipation extending over weeks, in addition to enormous *distention* and *tympany* of the abdomen. Physical examination in extreme cases recognizes dislocation of the adjacent abdominal and thoracic viscera, especially the liver, spleen, heart, and lungs.

Treatment.—The treatment is that of the resulting constipation, which, in cases of this kind, is by enemas carried high up into the bowel, together with remedies which simulate secretion into the upper bowel, of which calomel is one of the best. It should be given in doses of not less than 1/4 grain (0.016 gm.) hourly, until an effect is produced in association with that of the enemas. Dilatation probably results, at times, from the gradual accumulation of fecal matter, while frequent small discharges are being obtained which do not clear out the bowel. Hence the rectum should unhesitatingly be explored by the finger in doubtful cases. Complete evacuation of the bowels is sometimes extremely difficult, but if the exact state of affairs is appreciated, perseverance will ultimately conquer. Operation with exsection of large portions of the bowel has been done with excellent results and is the only rational treatment.

Diverticulitis.

Inflammation of the diverticula, commonly in the lower portion of the colon, although it may occur in the cecum.

The patients are usually over forty years of age. The patients are otherwise well, and usually obese. Cases reported by Mayo occurred in the sigmoid; either an abscess forms intraperitoneally, obstruction occurs, or the case is slight and receives no attention. Some of the cases begin suddenly with pain low down on the left side; all had tumors in that region.

The only treatment is resection of the gut and removal of the tumor, or if an abscess be present, opening and draining of the abscess.

CARCINOMA OF THE INTESTINE.

All parts of the intestine are subject to *carcinoma*, which occurs in growing frequency as the gut is descended. Thus, of all cases of intestinal cancer, barley 5 per cent. are found in the small intestine, 15 per cent. in the cecum and colon, while 80 per cent. are met in the rectum.

In the small intestine, in the neighborhood of the orifice of bile-duct, we meet most frequently the cylinder-celled epithelioma or adenocarcinoma.

[1] "Transactions of the Pathological Society of Philadelphia," vol. xvi., 1891–93, p. 23. Formad gives in his paper a summary of other cases reported.

In the *large intestine* there is:

1. Cylinder-celled epithelioma, the most common form of cancer, in the cecum and sigmoid flexure.
2. Colloid cancer
3. Scirrhus
4. Soft cancer } in the rectum.
5. Squamous epithelioma just above the anus
6. Scarcoma, including the melanotic variety

Benign tumors of the bowel, which may present symptoms similar to those of malignant tumors or no symptoms at all, include mucous polypi and fibromata, more rarely lipoma, myoma, angioma, and lymphoma.

Symptoms.—There are no symptoms distinctive of cancer of the bowel. The most constant local effect is more or less obstruction of the bowel, and we have already seen in our study of obstruction how far it is contributed to by cancer. There are, however, other symptoms which, added to those of obstruction, aid in the diagnosis. Particularly is this true in the case of the rectum.

The symptoms of obstruction met with in cancer of the bowel, already considered in treating of obstruction, include, especially, *constipation*, *pain*, *tumor*, *anorexia*, *nausea*, and, more rarely, *vomiting*. The added symptoms are *cachexia* and *altered fecal discharges*, which may include pus, blood, occult blood and, in few instances, fragments of cancerous tissue. Of the symptoms of obstruction named, *tumor* alone demands further consideration, being the most important of all the symptoms of cancer. In fact, without it a certain diagnosis is scarcely possible. On the other hand, given a case of obstruction, the presence of tumor points more to cancer than to any other cause except intussusception and fecal impaction. As contrasted with intussusception, the tumor of cancer is of long duration and found in adults; as with impaction, it is tender and movable, usually harder and more irregular. While the tumor may give a dull note to light percussion, to a hard stroke it is tympanitic. It may pulsate also if it lie over one of the large blood-vessels. Fecal tumors never do this. The difficulty of distinguishing from a fecal tumor is increased when a fecal mass is added to the cancerous tumor, but some of it may be cleared up by the use of purgatives and injections.

Cachexia, added to other signs of chronic obstruction, points to cancer. *Change in the shape of the formed feces*, especially a band-like flattening, is much spoken of. It may be produced by any cause which protrudes into the lumen of the large bowel, characterizes rather disease of the lower part, and, to be of value in diagnosis, it must be constant. The more or less constant presence of sanious pus, particularly of fetid character, is important evidence in favor of cancer.

Sudden obstruction of the intestine following a long-standing constipation occurring in those past middle life, with or without tumor, is strongly suggestive of carcinoma (schirrus) in the lower intestine. Volvulus, obstructing bands and other common causes must of course be considered.

Diagnosis.—1. *Diagnosis of the Part of Bowel Involved.*—As to the part of the bowel involved, once assured that the tumor is of the bowel, some indication of its more exact location may be obtained by noting its position,

which, if in the right upper abdominal region, suggests the duodenum; in the vicinity of the umbilicus, the transverse colon; in the right iliac fossa, the cecum, and in the left, the sigmoid flexure. It should be remembered, however, that serious dislocation of the tumor from its natural site may occur as the result of inflammatory adhesions formed while the tumor is temporarily in a position remote from its natural site. Often, too, a cancer of the sigmoid flexures gives no indication of its presence to abdominal examination. Persistent occult blood in the stools is very significant of carcinoma of the intestinal tract. Allusion has been made to the presence of jaundice as characteristic of duodenal cancer; also to the retained natural acidity of the gastric contents removed after a test-meal as compared with gastric cancer. Cancer of the rectum can generally be reached by the finger or some by the aid of the speculum.

2. Carcinoma of the duodenum is not easily distinguished from *tumor of the pylorus;* indeed, it is sometimes impossible to separate them. Both are movable tumors. With pyloric tumor are associated symptoms of obstruction and dilatation of the stomach. More rarely cancer of the duodenum has the same effect. The presence of jaundice points to cancer of the duodenum, as does also the continued natural acidity of the gastric contents removed after a test-meal, but neither of these symptoms is pathognomonic of duodenal cancer. In cancer of the stomach dyspeptic symptoms occur earlier and are more serious. Carcinoma of the duodenum may terminate suddenly by fatal hemorrhage. *Cancer* of the head of the *pancreas* also produces jaundice, but the tumor arising from it is fixed and immovable, and much more deep-seated than tumors of any portion of the bowel, being behind the pylorus and the transverse colon, between the right sternal border and parasternal line.

With the other abdominal tumors intestinal cancer is not likely to be confounded. The *floating kidney* is movable, but when sufficiently so to be compared in this respect with a cancerous tumor, is more movable, and may be generally returned to its natural seat. The kidney shape may not infrequently be recognized. Compression of the kidney often produces a peculiar sickening pain. The presence of nervous symptoms is especially characteristic of floating kidney, but there is no cachexia. A *movable spleen* is even less likely to be confounded, for similar reasons. It is, moreover, less sensitive. A *laced-off lobe of the liver*, often quite movable, can generally be traced to its normal attachment.

An actual *tumor of the kidney*, being behind the peritoneum, pushes the bowel and the ascending or descending colon before it, and must attain considerable size before it shows itself to the usual examination from the front. Such tumor very rarely compresses the bowel so as to produce symptoms of obstruction. The same may be said of tumors of postperitoneal lymphatic glands. An *ovarian tumor* is characterized by its deep-seated origin, its ascending development, and its relation to the uterus, as determined by joint vaginal and abdominal examination.

A *circumscribed peritoneal exudate* might be mistaken for a cancer of the bowel, but the history of its development, its flat percussion note, and the presence of some temperature, which characterizes it, are wanting in cancer of the bowel.

Carcinoma of the cecum is frequently mistaken for appendicitis. Pain may be the first symptom attracting the attention of the patient. Examination will show tenderness, resistence and a tumor in the appendiceal region. Operation only revealing the fact that the tumor is a new growth. If a history of long-standing tumor can be obtained a carcinoma would be suspected, but chronic appendicitis may likewise give rise to a tumor lasting some time.

Chronic inflammatory thickening of the bowel may, however, be a serious stumbling-block. Especially apt to occur about the sigmoid flexure, it produces also obstructive symptoms, and careful and prolonged study may be necessary to the making of a correct diagnosis. Cachexia remains absent in simple inflammatory stenosis for a longer time at least than cancer.

Diverticulitis lately described by Mayo gives sometimes all the rational symptoms and signs of carcinoma.

Cancer of the rectum exhibits a somewhat special train of symptoms. The rectum is subject to the same forms of cancer as the pylorus, and in somewhat the same order of frequency, the columnar-celled epithelioma being most ccmmon.

The early symptoms of cancer of the rectum are those of *irritation*, including *pain*, *tenesmus*, the *discharge of mucus and blood*, and, probably, most cases of carcinoma of the rectum are mistaken at first for dysentery. In the cases of colloid cancer, the colloid material may be discharged from the bowel and reasonably mistaken for mucus. Early examination of the rectum by the finger should always be made for generally the disease can be felt, either as an ulcerated mass infiltrating the wall of the bowel, thus intruding upon the lumen, or as one or more nodular growths under the mucous membrane and adherent to it. If ulceration has occurred, bloody and mucoid matter, characterized by extreme and persistent fetor, is apt to adhere to the finger. Von Leube especially calls attention to hemorrhoids as a symptom of cancer of the rectum, and says they are seldom absent, because of the resistance opposed to the return of the venous blood. He claims he has discovered rectal cancer in examination suggested by hemorrhoids when no other symptoms were present. So, too, the presence of secondary cancer of the liver should suggest examination of the rectum, since marked instances of the former have been found associated with cancer of the rectum, otherwise latent.

Almost all morbid growths affecting the rectum of adults are cancerous Polypi, mucous and fibromatous, occasionally found in children, produce dysenteric symptoms, including bloody discharges, while they may project from the rectum during stool. Lipomata and other histioid tumors have been found at autopsy without having caused symptoms.

Prognosis and Treatment of Cancer of the Intestine.—The prognosis of cancer of the bowel is always unfavorable. Occasionally operative procedures have prolonged the life of the patient at the expense of an artificial anus in the lumbar or abdominal region, while resection has even been made with success. Especially favorable have been the results in some cases of excission of the tumor.

The propriety of operation should, therefore, always be considered. Should it be decided against, the patient must be nourished by easily assimil-

able foods, such as peptonized milk, by the mouth or bowel, as circumstances may determine. A regular and sufficient evacuation of the bowels should be carefully looked after, lest impaction add its inconveniences to the others present.

INTESTINAL SAND.

Intestinal sand occurs in women. Not infrequently it is a true mineral gritty substance, in one case seen by us exactly resembling the red sand supplied for the care of canary birds. A false intestinal sand occurs, due to remains of vegetable materials which have been partially digested and incrusted with mineral salts.

DISEASES OF THE LIVER.

ABNORMALITIES IN THE SHAPE AND POSITION OF THE LIVER.

Altered Shape.—The only abnormality in the shape of the liver requiring special mention is the "laced-off" or "corset" liver. In this the right lobe is divided by a transverse furrow, more or less deep, into two nearly equal parts. In extreme cases the connecting furrow is a mere fibrous band, and the liver can be folded on itself; in others it contains more or less liver parenchyma. It is said to be caused by the pressure of a tight waistband or corset, and accordingly is more frequent in women, but it is met also in men.

It seldom gives rise to any symptoms, but sometimes leads to confusion in diagnosis, being frequently mistaken for a movable kidney or an abdominal tumor, for the inferior portion may extend as low as the crest of the ilium. This confusion is increased if, as occasionally happens, a loop of intestine lies in the furrow and gives a tympanitic note on percussion; whence the inference that the lower portion is a separate organ. Skillful palpation is a valuable means for determining the true nature of such a condition. The edge of the liver should be followed around from the epigastrium into the right lumbar and iliac regions. If the continuity with the supposed tumor is uninterrupted, the latter must be a portion of liver laced off. It is not unlikely that such a condition may occasion symptoms of dragging and weight, with the nervous strain frequently incident to them, like that which is so characteristic of floating kidney. The corset-liver is said to be one of the favoring causes of cholelithiasis, by reason of its interference with the natural onward movement of the bile.

Abnormality of Position.—The liver in cases of transposed viscera is found on the left side. More frequently it is simply turned downward or upward, anteverted or retroverted as it may be on its transverse axis, chiefly as a consequence of tight lacing in women. It may be pushed upward above its normal site by ascitic fluid or abdominal tumors, and downward by pleuritic effusion on the right side or by emphysema of the right lung.

The *floating liver* is by far the most interesting of these conditions. When it occurs, the natural site of the liver is vacant, especially when the

patient is in the upright position, occupied usually by hollow viscera, or, in rare instances, by morbid growths. The condition of such mobility is a long suspensory ligament and a coronary ligament so stretched as to form a sort of mesohepar, which permits the liver to fall out of its normal position. It occurs usually in women past middle life, with loose abdominal walls, and is favored by tight lacing. It has been met with in men. It is sometimes responsible for the condition known as the pendent belly. It is a rare condition.

The organ itself is usually easily recognized as a large, hard, but movable tumor, below the normal place, and having also the shape and size of the liver, while the normal site is tympanitic on percussion or occupied by organs which do not give the same outline on percussion. The suspensory ligament may also be felt. The organ may generally be restored to its normal position when the patient is recumbent. A deceptive form of movable liver occurs in the late stages of portal cirrhosis. The liver is tilted on its transverse axis, the lower edge extending far below the margin of the ribs, the upper margin being the posterior border of the liver is found about at the normal upper limit of the liver dullness, namely, at the fifth rib. This closely resembles a large liver and is constantly mistaken for such.

The same dragging symptoms mentioned as characteristic of the constricted liver, with the usual contingent of nervous symptoms which succeed upon it and the movable kidney, may be present here.

Treatment.—The treatment for both of these conditions—the constricted and the displaced liver—must consist in some instrumental means by which the organ or constricted portion can be held in position.

Diseases of the Bile Passages and Gall Bladder.

JAUNDICE OR ICTERUS.

Jaundice is not a disease, but a symptom, consisting in a yellowish discoloration of the skin and other tissues and fluids of the body by coloring-matters derived from the bile. The shades of coloring range from a very pale, scarcely appreciable, yellow to a brown-olive hue. It is a symptom present in so many different diseases and so associated with other symptoms more or less constant that its separate consideration is justified.

Obstructive Jaundice.—Reabsorption of bile takes place when there is obstruction to its onward movement, such as results, for example, from impaction of a gall-stone in the hepatic duct or common bile-duct; from closure of the duodenal end of the common bile-duct by inflamed and swollen intestinal mucous membrane; from complete or partial obliteration of the duct by adhesive inflammation; and from pressure from without by morbid growths. These growths may be enlarged glands in the fissure of the liver, or tumor in the gall-bladder, in the liver itself, in the pancreas, and in the stomach, and especially cancer of the pylorus and duodenum. More rarely tumors of the kidney or omentum, abdominal aneurysm of the celiac axis or aorta, or enlargement of the uterus may occasion obstruction. So may fecal accumulation. The morbid states in the liver which may

produce jaundice are cancer, abscess, hydatid cysts, and cicatrices, all of which will be referred to again. It is reasonable to suppose that the bile is absorbed from the overdistended biliary vessels by the adjacent capillary vessles of either portal or hepatic vein system facilitated by pressure. Reduced pressure in the blood-vessels of the liver, as contrasted with that in the biliary vessels and ducts, also favors reabsorption of bile from the latter.

This is the usual form of jaundice. All ages are subject to it. In addition to the discoloration described there is often an annoying *itching of the skin*, due to irritation of the deposited bile pigment. Further evidence of the irritation thus caused is seen in occasional *eruptions*, such as urticaria, lichen, and even furuncles. A *bright yellow discoloration of the sclerotic coat of the eye* is as constant as the staining of the skin, while the mucous membranes are often similarly tinged.

After the skin, the *urine* exhibits the most conspicuous alteration, even in mild cases. Indeed, this is sometimes the first symptom. The color may be slightly yellow or deep brown, like that of porter. The presence of bile pigment in the urine is readily shown by Gmelin's nitrous acid test, though ordinary nitric acid answers nearly as well. A few drops of the urine and half as many of the acid are placed on a procelain plate and gradually allowed to approach and fuse, when a brilliant play of colors appears, in which green, yellow, red, and violet are most easily recognized. The reaction is due to the exidation of the bilirubin by the acid. One of the most reliable ways of recognizing bile in the urine is by the stained cellular elements which it contains. Under no other circumstances are the bright yellow stained cells found, and they are even met with when the quantity of coloring-matter is insufficient to react by Gmelin's test. In a few cases the bilirubin reaction is not obtainable, when the urine contains in increased amount its normal coloring-matters, urobilin or hydrobilirubin—*i. e.*, reduced bilirubin.

Of the *remaining secretions*, the perspiration is often stained, the milk, the tears, and saliva, are rarely stained. There is sometimes a bitter taste in the mouth, showing an elimination of some constituent of the bile by the buccal glands probably the salivary.

On the other hand, the *feces* are often devoid of biliary coloring-matter, and their pale-gray or pipe-clay color has long been significant of the absence of bile. For the same reason the bowels are usually constipated and the discharges pasty, ill-smelling, and acid. Occasionally there is *diarrhea*, which may be caused by irritating effect of the feces disposed to rapid decomposition, because of the absence of their natural antiseptic ingredient. For the same reason, too, the absorption of fats is hindered. There may be other signs of gastrointestinal derangement, such as *loss of appetite*, *nausea*, *fetid breath*, and *fullness in the epigastrium* after eating. *Hemorrhage* is common in chronic jaundice. The clotting time of the blood is much prolonged, often being as long as ten to twelve minutes. Hemorrhages occur from many of the mucous membranes and into the skin in the form of purpura. Operations in chronic jaundice are dangerous on account of bleeding which follows. In cases of long standing there may be *albuminuria* as well, with bile-stained tube casts.

Very characteristic of simple obstructive jaundice is a *slow pulse*, which may be as infrequent as 50, 40, or 30. The breathing rate, on the other hand, is normal.

The chief subjective symptom of jaundice is *depression of spirits*, which may even amount to melancholia. *Irritability* is also prominent. *Headache and vertigo* are frequent. *Vision* is variously affected: to some, objects appear yellow; some see better by obscure light—nyctalopia; to others, the approach of darkness is associated with more than usually difficult vision—hemeralopia. Grave nervous symptoms, rarely manifested, are *sudden coma*, *acute delirium*, and *convulsions*. These usually supervene in cases of long standing, and are attended by *fever*, *rapid pulse*, and *dry tongue*—the symptoms, in a word, of the typhoid state. The term cholemia is applied to the sum of these symptoms, and the condition is regarded as due to the presence in the blood of the constituents of bile, of which cholesterin is the most important; whence also the name cholesteremia.

The *liver* is more or less altered, in accordance with the disease which may be present in it and responsible for the jaundice. These changes will be considered in treating of the diseases in which jaundice is a conspicuous symptom. It may also be bile-stained, as are other internal organs, especially the kidneys.

The *duration* of this form of jaundice depends upon the disease which is reponsible for it, and it may be a few days or many months. In chronic cases remission and exacerbations occur, but the longer the duration, the more likely is there to be some organic change in the liver.

2. *Toxic Hemolytic or Hematogenous Jaundice.*—This form of jaundice is now recognized as in reality obstructive in character.

Stadelman has shown that the jaundice is in reality due to the plugging of the small bile ducts with viscid bile, swollen epithelial cells and products of blood destruction. The pressure in the hepatic vessels being very low the bile is absorbed into the vessels and jaundice occurs. The symptoms of this form are those of the diseases which are responsible for the hemolysis—viz., acute yellow atrophy, phosphorus-poisoning, yellow fever, bilious fever, typhoid, typhus, and relapsing fevers, pyemia, pernicious anemia, snake poison, chloroform, and other poisons. In all of these there is some toxic agent working destruction of the blood. It should be added that in this form of jaundice the *stools are not clay-colored*. The urine also is less bile-stained, though the true urinary pigments, notably urobilin, are often very much increased. The patients are frequently delirious, with rapid running pulse, cutaneous hemorrhages, and are in a typical typhoid state.

Recognition of Jaundice.—One of the most frequent errors of the inexperienced, and a constant one of the laity, is to mistake for jaundice a dirty yellowish discoloration of the skin, known as sallowness, which is symptomatic of general ill health. It is probably an anemia and may be distinguished from jaundice by the fact that it is not associated with staining of the conjunctiva and secretions. It is, moreover, not a yellow, but a dirty brown. One needs only to have his attention aroused to avoid error.

Much more closely does the discoloration of the skin in *Addison's disease* resemble that of some cases of jaundice. In the former there is no

discoloration of the sclerotic coat nor of the urine, while the feces remain natural. In Addison's disease the exposed portion of the body and its flexures are more deeply stained. Great exhaustion is pressed in Addison's disease.

The purpose of diagnosis includes the discovery of the cause and seat of obstruction. In the first place, most cases of acute jaundice are due to catarrhal inflammation of the common bile-duct. If associated with fever, it may be assumed that the smaller ducts are involved. After this, obstruction by gall-stones causes many cases; then follow hypertrophic cirrhosis and the various malignant diseases of the liver, hydatid disease, abscess, pressure by enlarged glands in the fissure of the liver; also obstruction, the result of kinking of the common duct.

Icterus Neonatorum.

Synonym.—*Jaundice of the New-born.*

Jaundice occurs in new-born children in a simple and *harmless* form, with symptoms comparable to obstructive jaundice, and in a *grave* form comparable to hemolytic jaundice. The first is probably a form of obstructive jaundice due to like causes especially congestion of the liver. It is much the more frequent, and disappears in from a few days to several weeks. This form occurs 300 times in 900 births reported by Holt.

A patulous ductus venosus has been suggested as an avenue through which the portal blood which contains bile enters the circulation.

The grave form, usually fatal, has been found associated with absence of the hepatic duct or common duct, with congenital syphilitic hepatitis, and with septic phlebitis of the umbilical vein.

Treatment.—The simple form of jaundice of new-born infants demands no treatment. In the graver forms when the condition can be traced to syphilis, it demands the treatment of that disease in its tertiary form. In certain forms accompanied by grave hemorrhage the injection of large doses of human blood serum effects a cure. This will be described under melena.

Hereditary jaundice occurs, numerous cases being reported where all the children of a mother were jaundiced at or shortly after birth.

Duodeno-cholangitis or Simple Catarrhal Jaundice.

Synonym.—*Inflammation of the Common Bile-duct.*

Definition.—The term catarrhal jaundice is applied to jaundice due to any inflammation of the common duct not the result of impacted gall-stone.

Etiology.—The most frequent cause of such inflammation is the extension of a gastro-duodenitis into the common duct. To the same cause is ascribed the jaundice sometimes occurring with passive congestion of the liver due to mitral valvular heart disease, also that found in association with the infectious diseases, especially pneumonia, or with mental emotion. Catarrhal jaundice may also be epidemic.

Morbid Anatomy.—Opportunities of studying postmortem conditions

after catarrhal jaundice are not often afforded, but when they occur, the duodenal end of the duct—the *pars intestinalis*—has been mostly involved. In it the mucous membrane is swollen, while its orifice and the diverticulum of Vater may be filled with mucus. The inflammation may extend up into the cystic duct, and even higher, into the hepatic duct and branches. Suppuration does not take place in this form of cholangitis.

Symptoms. – Excepting the jaundice, there may be no symptoms, the first indication of disturbance being the yellow color of the skin. There may be pain and *tenderness*, due to gastro-intestinal derangement rather than to the hepatic state, though this may cause it, while such derangement may also lead to *general malaise*, *loss of appetite*, *coated tongue*, *fetid breath*, *nausea*, *vomiting*, a sense of *fullness*, *constipation*, or irregular action of the bowels. There may be also slight *fever*, particularly if the smaller biliary passages are involved. If the gall-bladder is distended and can be felt at the edge of the liver, there is probably obstruction of the common duct. The paler the feces, the more complete must be the obstruction, and the more likely it is to be in the common duct. Obstruction of the hepatic duct is unassociated with distention of the gall-bladder, while there will be jaundice. Obstruction of the cystic duct may still be associated with distention of the gall-bladder, either through transudation or pus-formation, but there may be no jaundice, and the feces may remain colored. In the hemolytic form the jaundice is usually so plainly secondary to other symptoms that there is little difficulty in recognizing its cause.

Diagnosis.—The presence of acute jaundice without pain or other symptoms points almost invariably to catarrhal jaundice. The same diagnosis is justified by the presence of the symptoms of gastro-intestinal catarrh, of associated mitral disease, or of any of the infectious diseases. If, however, jaundice is prolonged into weeks there is likely an organic lesion of grave character.

Prognosis.—Unless associated with infectious diseases or with hypertrophic cirrhosis, the prognosis of catarrhal jaundice is favorable. In the diseases referred to the danger is not from the jaundice, but from the diseases with which it is associated.

Treatment.—The treatment of catarrhal jaundice resolves itself into two parts: first, that for the catarrhal state; second, that demanded by the absence of bile in the small intestine. Careful search must be made for a cause such as heart disease and Bright's disease, the treatment of which must be the treatment of the jaundice.

For the catarrhal inflammation, either of the duodenum adjacent to the duct or of the duct itself, local depletion is indicated. This is accomplished by the use of saline aperients and the natural mineral waters which act similarly—*i. e.*, produce watery stools. Of the former, Rochelle salts, Epsom salts, or the solution of the citrate of magnesium are representative; while the Saratoga, Apenta, Hunyadi János, Friedrichshalle, or Rubinat and Carlsbad waters represent the latter. These should be taken daily in aperient doses. These drugs, however, should be used only as aperients not as *purgatives*. The Bedford Springs waters, near Bedford, Pa., are also useful, but not nearly so efficient as the Saratoga waters. Of foreign waters, those of Carlsbad are especially valuable, and in Europe these springs may be

resorted to. Their use may also be associated between meals with that of the alkaline mineral waters, of which those of Vichy and Vals are the type. These waters are largely employed in this country, and may be availed of at home. Calomel may be given for its laxative effect. Podophyllin and colocynth may be used for the same purpose. Sodium salicylate or salicylic acid in some form may be given in doses of ten grains (0.6) for its antiseptic effect. Hexamethylenamine may be given in five grain doses (0.5) for the same purpose. Irrigation of the large bowel with cold water has been recommended as a means of stimulating the descent of the stone.

The second indication should be met by the use of such food as does not require the bile to facilitate its digestion or absorption or to prevent its decomposition, and which will not irritate the intestinal mucous membrane of the intestine. Fats and oils should, therefore, be avoided; hence skimmed milk, animal broths, and egg-albumen, with an abundance of liquids, are indicated. The liquids may be some one or more of the mineral waters previously named, or, in their absence, plain water. Warm bathing is especially indicated, as it causes elimination by the skin and relieves the itching. Lotions of carbolic acids and glycerin are also useful for the same purpose.

CHOLELITHIASIS.

SYNONYMS.—*Hepatic calculus, Biliary calculus.*

Etiology.—Since the great bulk of the gall-stone is cholesterin, an evident condition of its formation is a precipitation of this chief constituent of the bile. The thicker the bile, the more likely it is to throw down sediment. Moreover, studies, especially by Naunyn, have shown that micro-organisms play an important part in the production of gall-stones, primarily by exciting a catarrhal inflammation which modifies the chemical composition of the bile and favors the precipitation of cholesterin and of lime salts, in combination with epithelial débris and bacteria, the epithelial débris and bacteria frequently being the nidus of the stone. The typhoid fever bacillus is an especially frequent cause of inflammation of the gall-bladder. Naunyn also showed that cholesterin and lime salts are a secretion of the mucous membrane of the gall-bladder and bile-ducts that this is especially active when the mucosa is in a state of inflammation. If, as is supposed, the cholate salts of sodium hold cholesterin in solution, it is plain that their decomposition or destruction may cause precipitation, which may also be further favored by micro-organisms.

Occurrence.—Gall-stones have been met in infants and in the new-born, but practically are found in adults only, while their tendency to form appear to increase from the age of 30 upward. Most patients who consult us for the effects of gall-stones are over 40 and under 50. Cholelithiasis is also very much more frequent in women than in men; according to Naunyn four times as frequent, and especially so in women who have borne children or have had abdominal tumors. He says that 90 per cent. of women who have gall-stones have borne children; also that 25 per cent. of all women who die have calculi in the gall-bladder.

Lack of exercise, sedentary habits, and tight lacing are held partly

responsible for this, and with some reason, since all of these conditions are calculated to impede the movement of bile. Cholelithiasis has been found associated with the habit of free eating of starchy and saccharine foods and in stout persons; cases occur among the lean also. The movable liver and the movable right kidney are likewise said to predispose to cholelithiasis. Constipation and a tendency to depression of spirits are apt to be associated, probably as effects rather than causes.

Morbid Anatomy.—The gall-stone itself is a brown object, nearly spherical, oval or faceted, and even polygonal in shape, usually the size of a pea, or as small as a millet-seed, producing in aggregation "gall sand." The faceted shape is produced by close packing of a large number of stones in a gall-bladder, as frequently happens. More rarely the stone is irregular—mulberry-shaped, single and very large. In addition to cholesterin, which makes up from 70 to 80 per cent. of most stones, they contain varying small amounts of bile pigment, calcium carbonate, and organic matter. A few are made up almost entirely of bilirubin and lime. On section, the stone exhibits either a concentric or homogeneous appearance, with or without a nucleus of bile pigment or organic matter, and very rarely of some foreign body. The cholesterin stones are almost completely soluble in etherized alcohol, whence beautiful crystals of cholesterin may be obtained after evaporation.

In addition to their enormous accumulation in the gall-bladder, where they may be counted sometimes by hundreds, they are found anywhere in the biliary tract between the duodenal end of the common duct and the ultimate ramification of the bile vessels. Outside of the gall-bladder the cystic duct and the common duct are the situations in which lodgment most frequently occurs. If in the common duct, it is usually at the orifice of the papilla in the diverticulum of Vater, and from the duodenal side the stone feels as though it were directly under the nucous membrane. Two or even more stones may be found in the duct. The common duct under these circumstances may attain a diameter of an inch (2.5 cm.) or more. Permanent obstruction of the cystic duct causes dilatation of the gall-bladder —*hydrops vesicæ felleæ*. Such dilatation may be enormous, filling the entire abdominal cavity, and has been mistaken for ovarian tumor; usually it is more moderate, but the contents frequently amount to a pint (500 c.c.) or more. The contents are a colorless, viscid, or watery fluid, more or less albuminous, and neutral or alkaline in reaction; the greater the dilatation, the more aqueous and unlike bile do its contents become. In any situation the stone may produce ulceration and even suppuration, with perforation into the peritoneal cavity or adjacent organs, the duodenum, stomach, transverse colon, right renal pelvis, ureter, through the diaphragm into a bronchus, and into the abdominal wall.

Acute Impaction.

Synonym.—*Biliary Colic.*

Symptoms.—The characteristic symptom of impacted gall-stones is *biliary colic*, but biliary colic is by no means always present in every case of cholelithiasis. The pain is the result of infection, distention of the ducts

during the attempted passage of the stone and contraction of the muscular elements of the gall-bladder or gall ducts. The gall-bladder is often found full of calculi without the suggestion of a symptom. Small stones even pass into the duodenum without producing symptoms. Commonly, however, they lodge while in this transit, and give rise to attacks of *pain* which are known as biliary colic. This pain is usually sudden, very severe, often excruciating, and the patient writhes in agony and sometimes faints in consequence. It is usually referred to the epigastrium, whence it radiates in all directions over the abdomen and at times into the right shoulder and arm. As a rule, however, it is localized on the right side, under the liver. It is a sharp and cutting pain. There is always *tenderness* in this region, which varies in degree. It is sometimes associated with a more or less rigid state of the abdominal muscles of that side. The duration of the pain is that of the lodgment of the stone, and it may be from a few hours to weeks, ceasing rather suddenly when the stone is discharged into the bowel. There may, however, be remissions. *Nausea* and *vomiting* are almost invariable symptoms of biliary colic. They often bring temporary relief through the resulting relaxation. *Fever* is soon added to the pain, while a *chill* is not infrequent. The temperature is usually 102° F. to 103° F. (38.8° C. to 39.5° C.). It may be intermittent, but such intermission is more apt to be associated with prolonged obstruction, constituting with a chill a part of the symptoms of so-called *hepatic fever*, to be next considered. Gall-stone crepitus may sometimes be detected when the gall-bladder is packed with calculi. Jaundice occurs when the stone is in the common and hepatic duct; rarely occurs when the stones are in the gall-bladder. Hence, jaundice is not a necessary symptom of gall-stones. Indeed, it frequently is absent.

If the stone is likely to be in the common duct, having probably started in the cystic duct. Three or four days may elapse between the beginning of obstruction and the supervention of jaundice, the degree of which increases with the completeness and duration of obstruction. The entrance of the stone into the common duct may be attended by one of the remissions alluded to, though the jaundice grows even deeper on account of the more thorough obstruction to the descent of the bile.

David Riesman has called attention to a cardiac systolic murmur sometimes developed in the course of an attack of biliary colic. He ascribes it to a dilatation of the heart and relative insufficiency of the mitral valve, caused by pain, anemia or cachexia.

The liver is sometimes slightly enlarged, as determined by percussion. A rare symptom is *collapse* with fatal syncope, due to perforation at the seat of lodgment, with consequent *peritonitis* and *shock*.

Diagnosis.—This is commonly easy. While the pain may be more or less diffuse, it is for the most part localized in the right lower thoracic and upper abdominal regions, and the tenderness is always there, while, if jaundice and biliary urine are present, all doubt is removed. *Nephritic colic* and biliary colic are confounded with surprising and unjustified frequency. In the former condition the pain starts in the lumbar region and radiates down ward into the groin, the testicle, and the inside of the thigh. Such error is fortified by the fact that bilious urine is too often confounded with bloody urine. It should be necessary only to mention this to guard against error.

Examination of the urine will show blood and leucocytes in nephritic colic which are absent in hepatic colic.

Cholelithiasis has been mistaken for acute pleurisy in the vicinity of the gall-bladder and the reverse mistake has been made. The friction râle of pleurisy should preclude an error, but the friction râle may not be present at the particular stage.

Our growing knowledge of *appendicitis* has led to the discovery that the pain characteristic of this disease is sometimes localized in the right hypochondrium, where, indeed, the appendix has been found at operation. Jaundice and bile-stained urine do not, however, attend appendicitis. *Gastralgia* has been confounded with biliary colic, but attention to the symptoms described when treating that affection should prevent mistake. The term hepatic neuralgia has been applied to an apparently causeless pain, sometimes felt in the neighborhood of the liver, but it is less severe than biliary colic and unaccompanied by any of the other symptoms. This is allied to *pseudo-biliary colic* which is to be remembered as a possible event in nervous women. Both are characterized by the absence of jaundice. The pain of ulcer of the stomach resembles gall-stone colic to a certain degree. It is, however, rarely severe, and usually is attended by relief by taking food.

It is very important, immediately after an attack of supposed biliary colic, to *search for a stone* in the fecal discharges. For this purpose the fecal mass should be placed on a sieve, and water passed over it until all soluble parts are run out. Such examination should be kept up for several days after the attack, for the stone is not always passed immediately.

Prognosis.—The termination of an ordinary attack of biliary colic is, in the vast majority of instances, favorable. It is only in the rare cases, where perforation takes place, that a fatal ending follows. Surgery of the gall-bladder has come to be an important division of surgery, and many lives have been saved by operations. The surgeon should, therefore, be promptly sent for if the symptoms persist.

Chronic Impacted Gall-stone.

Symptoms.—These vary somewhat with the seat of the impaction and its duration. From this standpoint they may be divided into certain groups:

1. *Symptoms Due to Stones Retained in the Gall-bladder.*—There are practically always the direct result of infection. For without acute infections stones in the gall-bladder are frequently without symptoms. There is pain, tenderness over the gall-bladder, fever, and often leucocytosis. If the cystic duct is obstructed there is usually distention of the gall-bladder. On the other hand the gall-bladder may be atrophied and drawn tightly about the stones.

2. *Symptoms Due to Chronic Calculous Obstruction of the Cystic Duct.*—In addition to more or less of the symptoms detailed under acute impaction, the immediate result of such obstruction is dilatation of the gall-bladder, or *hydrops vesicæ fellæ*, already referred to. Dilatation of the gall-bladder is more frequently caused by obstruction of the cystic, than of the common duct. The source of the accumulation is not, however, the bile, which, as might be expected, cannot get into the gall-bladder through the obstructed duct any

more than it can get out of it. It is the products of inflammation of the mucosa, added to the bile previously present, which cause the dilatation. The occasional enormous dilatation has more than once been mistaken for ovarian disease, an error the more excusable when we remember that *jaundice is usually absent.* More frequently the dilatation is moderate, and can be felt below the edge of the liver as a round or ovoid elastic tumor, in which fluctuation may sometimes be obtained.

3. *Symptoms Due to Chronic Calculous Obstruction of the Common Duct.*—If the common duct is obstructed by a calculus, dilatation of the gall-bladder is generally absent, and if it does occur, the dilatation is moderate; whereas in obstruction of this duct by new growth the gall-bladder forms a palpable tumor, although not invariably. This is Courvoisier's law. Such obstruction is commonly associated with cholangitis, catarrhal or suppurative. (*a*) *In simple chronic catarrhal cholangitis* the common duct is dilated; at times also the branches of the hepatic duct extending into the liver. This condition has been especially studied by Charcot and Murchison abroad and William Osler in this country. It may be intermittent or remittent. Very interesting among the causes of intermittent obstruction is the movable or ball-valve stone in the diverticulum of Vater. A stone in this position is characterized, in addition to the persistent jaundice and paroxysmal pain, by *ague-like attacks*, consisting of chills, fever, and sweats. These occur at surprisingly regular intervals, resembling in this respect the quotidian, tertian, or quartan spells of intermittent fever, with which the condition has been confounded. They may occur for weeks at a time and then remit. *Pain* is commonly associated with the ague-like spells, but is not always present. The chills may be extremely severe, the sweats also, and the *fever* correspondingly high, the temperature sometimes reaching 105° F. (40.5° C.). The *jaundice* usually deepens after an attack. There may be *nausea* and *vomiting*. The duration may be indefinite from a few months to years, and the patient may yet recover; or he may perish, although the exhaustion is extremely slow and the effect on the general health barely appreciable from week to week. The fever is probably irritative, although it has been ascribed to the omnipresent organism—*bacterium coli commune*. There is no sign of suppuration in these cases. There is sometimes slight enlargement of the liver, appreciable to physical examination, and in long-protracted cases some fibroid induration may be expected to take place. The stools are sometimes bile-stained, at others not. There is occasionally enlargement of the spleen.

The following are Naunyn's distinguishing signs of stone in the *common duct:* "(1) The continuous or occasional absence of bile from the feces; (2) distinct variations in the intensity of the jaundice; (3) normal size or only slight enlargement of the liver; (4) absence of distention of the gall-bladder; (5) enlargement of the spleen; (6) absence of ascites; (7) presence of febrile disturbance, and (8) duration of the jaundice for more than a year."

Thus, Ecklin found that of 172 cases of obstruction of the common duct by calculus, the gall-bladder was contracted in 110, normal in 34, and dilated in 28. Of 139 cases of occlusion of the common duct from other causes the gall-bladder was contracted in 9, normal in 9, and dilated in 121.

(*b*) *Suppurative cholangitis* is marked symptomatically by a fever which

is more of the septic type, with remissions rather than intermissions. The jaundice is less marked, the liver is tender and enlarged, the duration of the disease shorter, and termination fatal. The inflammation involves more or less the ducts of the liver, whence it may extend into the liver substance or gall-bladder, causing abscess of the liver and empyema of the gall-bladder.

Other Remote Results of Gall-stone Impaction.—Rarer terminations of impacted gall-stones are the various forms and situations of biliary fistulæ, mentioned when treating of the morbid anatomy. Some more detailed reference to these fistulæ should be made. Much has been added to our knowledge of the subject by the industry of Prof. L. G. Courvoisier, of Basle.[1] Courvoisier collected 499 cases of ulcerative perforation of the biliary passages of which 70 occurred directly into the peritoneum, while in 49 cases there was encapsulated abscess, and in three there was retro-peritoneal perforation. Between the biliary passages themselves were eight cases; this perforation was found directly from the gall-bladder into the substance of the liver (four cases); into the hepatic duct (two cases), into a diverticulum of the common duct (one case), or between the intestinal and hepatic parts of the common duct (one case). Perforation between the biliary passages and portal vein was found in five cases. Openings between the biliary passages and gastro-intestinal canal are not uncommon (137 cases); most frequently between the bile passages and duodenum, of which there were 83 cases, of which 73 were between the gall-bladder and the duodenum, while ten were between the common duct and duodenum. From the biliary passages into the stomach there were 13 perforations; into the jejunum one, ileum one, colon 39. As might be expected, perforation takes place most frequently from the intestinal part of the duct, the stone first lodging in the diverticulum of Vater. Perforation into the urinary passages was found in seven cases and into the pleura and lungs in 24 cases. To these last J. E. Graham[2] added ten cases of broncho-biliary fistula. Finally, there may be fistulous communication between the biliary passages and the external integument, Courvoisier having collected 196 cases, in 49 of which the communication was in the right hypochondrium, 36 at the border of the ribs, 49 at the navel or in its vicinity, 17 in the right meso-gastrium, ten in the right iliac region, and six in the epigastrium. Very interesting in this connection is the fact that out of 169 cases in which the sex was noted, 126 were women and 43 men. Among other remote results are septic cholecystitis, associated with high fever, intense prostration, and death from fatal peritonitis; empyema of the gall-bladder, already alluded to as a result of suppurative cholangitis; the latter is commonly associated with gall-stones. Calcification of the gall-bladder is a frequent termination of purulent inflammation. It is present in two forms: first, as a simple incrustation of the mucosa with lime salts, and, second, as a true infiltration of the whole thickness of the wall.

Atrophy of the gall-bladder is not infrequent and may succeed on *hydrops vesicæ felleæ*. Many gall-bladders do not hold more than a dram (4 c.c.) or two of bile, and sometimes there is a mere remnant left in the shape of a fibroid mass; at other times the shrunken bladder closely em-

[1] Casuistisch-Statistische Beiträge zur Pathologie und Chirurgie der Gallenwege, Leipzig, 1890.
[2] Transactions of the Association of American Physicians, vol. xii., 1897.

braces a gall-stone of large size. Gall-stones are occasionally found in diverticula of the gall-bladder. Suppurative phlebitis and abscess of the liver may also be due to gall-stone, causing a puriform thrombus in an adjacent branch of the portal vein.

In other instances the gall-stone is of such size as to obstruct the bowel when discharged into it, although it may have passed through the natural channel, as evidenced by dilatation of the common duct. But for the most part such discharge is by ulceration into the intestinal tract. This subject has been sufficiently considered when treating of obstruction of the bowels.

Diagnosis and Prognosis.—Chronic impaction of gall-stone must be diagnosed from obstruction due to other causes. Dilatation of the gall-bladder when accompanied by jaundice is usually due to pressure from a new growth in the common duct. Extremely large gall-bladders are usually due to obstruction of cystic duct from adhesions, stone or new growth. The jaundice accompanying obstruction of the common duct by a stone is usually accompanied by intermittent pain and fever without emaciation which occurs in carcinoma. The perforations which may occur are usually preceded by a history of gall-stones. There may be some difficulty at first in the diagnosis of hepatic fever, but the persistent jaundice, the ague-like paroxysms of chills, fever, sweats, and pain are a combination of symptoms belonging to no other condition than that of a ball-valve stone. A *cancer of the gall-bladder*, which will form a tumor in the same locality, is much more tender; it is harder and more uneven, and jaundice is frequently associated with it, while the patient is much more seriously ill and declines more rapidly. There should be no confusion with a *movable kidney*, which furnishes a different physical condition. The suppurative form of cholangitis or cholecystitis is characterized by the more continuous fever and the more serious aspect of the septic state, its shorter course, and its ultimate fatal termination. The catarrhal form is less serious and quite often terminates favorably.

Treatment of Impacted Gall-stone and its Complications.—The first indication in an acute attack is the relief of pain. This is best accomplished by the hypodermic injection of morphin, the action of which is favored by combination with atropin. Scarcely less than 1/4 grain (0.0165 gm.) with 1/150 grain (0.0005 gm.) of atropin suffices, and this must often be repeated. The use of anodynes must be kept up as long as needed. The atropin favors the relaxation needed to release the calculus. The severest cases may require the inhalation of a few drops of chloroform pending the action of the morphin.

Whether anything else can be done toward releasing the stone is not established. The nausea and vomiting, which are so often symptoms, sometimes relieve the pain by the relaxation they produce, such relaxation being at times sufficient to favor the onward movement of the stone. Anesthesia by ether or chloroform may act similarly, and the inhalation above suggested while waiting for the morphin to act favors such relaxation. Hot baths or fomentations applied to the region of the liver may also be similarly effective.

Some solvent for the stone is constantly inquired after. *No known solvent for a gall-stone in the living human being is known.*

The free use of alkaline mineral waters does seem to favor the dislodgment of the stone, especially if the authorities at Carlsbad are to be relied on, who claim the discharge of immense numbers of biliary calculi under the use of Carlsbad water. Certainly no harm can attend its use, and when within the power of the patient to get it, it may be freely taken. The same is claimed by the physicians at Vichy for the Vichy waters—true alkaline waters. In this country, however, the Saratoga waters may be used instead. These waters are saline and not alkaline waters, but they seem to fulfill much the same indications. Those containing the largest proportion of alkaline carbonate are to be preferred. The waters of Vals—also true alkaline waters—are recommended for the same purpose.

To relieve the itching caused by the deposit of pigment in the skin, which is sometimes very annoying in chronic cases, the hot pack on alternate days or even every day is serviceable. A very efficient local application for this purpose is a mixture of 7 1/2 minims (0.5 gm.) of carbolic acid, 2 fluidrams (8 c.c.) of glycerin, and 6 fluidrams (24 c.c.) of water. It should be applied with a sponge and allowed to dry on the skin.

Surgical Treatment.—In acute suppurative cholangitis and cholecystitis immediate drainage of the gall-bladder and ducts should be performed. In acute attacks of gall-stone colic, where the condition lasts for days, and perforation of the bladder or ducts is threatened, operation should be performed. Chronic cholelithiasis is a surgical condition. All of the symptoms described above will be relieved and the patient cured by a skillful operator. The patient will suffer and perhaps become mortally ill by long-continued treatment by drugs and hygiene.

The *preventive treatment* is important. To this end diet is important. The patient should eat sparingly of hydrocarbons and carbohydrates, omitting every form of fat, alcohol, sugar, and starch. Meat, cheese, and glutens, on the other hand, are allowable.

The alkaline and saline mineral waters are more especially indicated between the attacks than during them, and their more or less continued use is advisable, especially in the morning, when their efficiency is also increased by their being taken hot. The sodium salts have considerable reputation for their efficiency in preventing the concentration of bile and formation of gall-stones, having been long ago recommended by Prout. The phosphate is the modern favorite, in dram doses in the morning, or more frequently, but the sulphate is more constant and more potent in its results, and little, if any, more unpleasant. The sodium salicylate has a similar reputation, and may be used when no effect on the bowels is desired. By either of the former or by the aperient mineral waters a daily action of the bowels should be secured, while a proper hygiene of the body, in which daily exercise, bathing, and friction play a conspicuous part, is to be constantly maintained.

Salicylic acid in some form, in ten grain (0.12) doses every four hours, or one of the soda salts, succinate in 5 grains (0.30), or hexamethylenamin in the same dose.

ACUTE INFECTIOUS CHOLECYSTITIS.

SYNONYM.—*Acute inflammation of the gall-bladder.*

Definition.—Inflammation of the gall-bladder due to infection by pathogenic bacteria.

Etiology.—The most frequent predisposing condition which leads to infection of the gall-bladder is probably biliary calculus, the stone being lodged either in the gall-bladder or some one of the biliary ducts, the vulnerability of the mucous membrane of the gall-bladder being thus increased. But any obstructive cause, such as inflammatory adhesion, or even inflammatory swelling of the mucous membrane of the cystic duct, may be such cause—facilitating bacterial infection. Adhesive inflammation between the gall-bladder and intestines, however induced, is a rare cause, the process extending inward through the peritoneum. Lithiasis is not, however, necessary to produce infection. Pathogenic bacilli may act independently of predisposing cause. Indeed, gall-stones themselves are a result of bacterial invasion. The infecting bacterium may be any one of the pathogenic bacteria infesting the small intestine, but recent observations have shown the bacillus of typhoid fever and the colon bacillus to be probably the most frequent, although the pneumococcus, staphylococcus, and streptococcus have also been found to be the infecting agents.

Morbid Anatomy.—This varies with the virulence of the inflammation. In the severer cases there is distention of the gall-bladder with mucus, muco-pus, or pus; at times the contents may be hemorrhagic. Perforation and gangrene have been the first indications of the presence of the disease. There may be adhesions between the gall-bladder and colon or omentum.

Symptoms.—The most invariable symptom is *pain*, which is commonly sudden and sometimes paroxysmal. It is situated to the right of the median line at the border of the thorax; is attended by *fever*, sometimes preceded by *chills* and followed by sweats. So many abdominal conditions, however, cause pain that it alone is not distinctive. A *chill* is often the first symptom. *Tenderness*, less circumscribed than might be expected, is invariably present. *Jaundice* is not a frequent symptom, never unless the infection involves the hepatic duct or common duct. *Vomiting*, on the other hand, is very common and often severe. It, too, may be paroxysmal. Certain cases are fulminating, and it may be impossible to get the surgeon soon enough to avert perforation and a fatal termination. On the other hand, many mild cases occur, like one seen with Thomas Potter, of Germantown, succeeding a relapse of typhoid fever after a normal temperature had been maintained for several days. After recovery from this relapse, there occurred suddenly a chill, sharp pain in the region of the gall-bladder, and rise of temperature. These symptoms subsided in four or five days, to be followed by another attack in which, instead of a chill, there was simply chilliness with pain and fever less marked; again, after a couple of days, a return of pain with sudden rise of temperature, but no chill, again disappearing in a few days. *The distended gall-bladder may sometimes be felt.* The pulse is sometimes very slow, as in a case reported by Frederick A. Packard, where the rate fell to 48, and another seen with Markley, of Cam-

den, N. J., in which it fell to 40. It is seldom over 100. In Packard's case there was no fever, in that of Markley the temperature rose to 103° F. (39.4°C.).

Symptoms may arise from *adhesions* with adjacent organs, chiefly pain, but sometimes also a dragging sensation. These are commonly part of a chronic condition. *Constipation* is also a symptom to be expected. In fact, some cases have been treated for obstruction of the bowel, for appendicitis, and more rarely for pancreatitis.

Diagnosis.—Since attention has been directed to the subject, the diagnosis in many cases has become easy. In others it still remains difficult or impossible. Given a case of typhoid fever in which, especially during convalescence, a chill, fever, and sweat make their appearance and there is pain in the region of the gall-bladder, we may infer reasonably the presence of cholecystitis. The same inference may be made if these symptoms occur in a case of chronic cholelithiasis. The presence of an actual tumor at the seat of the gall-bladder is even more confirmatory. Circumscribed tenderness is more frequent. The severity of the attack cannot always be inferred from the early symptoms, but as there are a good many mild cases, a diagnosis of cholecystitis need not necessarily cause alarm. It should be remembered that jaundice is not a frequent symptom, indeed, it is a rare symptom.

As to differential diagnosis, the conditions with which it has been confounded are *appendicitis*, *pancreatitis*, *localized peritonitis*, *pyonephrosis* and *inflammatory thickening* about the pyloric orifice of the stomach and the duodenum. In the absence of the predisposing conditions referred to, these lesions are sometimes difficult to differentiate. Disease of the head of the pancreas is much more frequently associated with jaundice than is cholecystitis. If a tumor is present in pancreatitis, it is fixed and immovable. It is not usually movable in cholecystitis. An exploratory operation should not be long delayed as perforation of the gall-bladder may precipitate a fatal issue. In cases like three narrated by Maurice H. Richardson,[1] in none of which was there history suggesting gall-stones and where the symptoms, including pain, vomiting, fever, and tenderness over the appendix, were so suggestive that an incision was made in that quarter, a diagnosis of cholecystitis is impossible. It is difficult to see how anything but appendicitis could be expected in such cases.

Prognosis.—This depends, of course, upon the severity of the case and the promptness of operative interference. There appear to be a good many mild cases which seemingly do not go beyond catarrhal inflammation.

Treatment.—There is really no medical treatment except the symptomatic, and the patient recovers through inherent tendencies, or his life is saved by operation and drainage. In gangrenous cases even operation fails to save some, but all cases demanding operation have the chances of recovery increased by promptness. Richardson says that acute cholecystitis demands interference even more strongly than appendicitis. If an operation is denied, or the condition of the patient will not allow of an operation, an ice-bag or mustard or hot fomentations may be applied to the region of the gall-bladder to relieve pain. Nausea and vomiting are among the most

[1] "Acute Inflammation of the Gall-bladder," "Am. Jour. Med. Sci.," June, 1898.

difficult symptoms to relieve. It is a reflected nausea like that of appendicitis. Local applications of ice, or at times the opposite treatment by heat, pieces of ice swallowed, champagne, cold effervescing waters may all be tried. Calomel in hourly doses of 1/10 gr. (0.0066 gm.) to 1/5 gr. (0.0132 gm.), applied dry on the tongue, should be given in connection with other remedies. None of these remedies should be used except as palliatives.

CANCER OF THE GALL-BLADDER.

Etiology and Morbid Anatomy.—John H. Musser, in a study of 100 cases, found it three times as frequent in women as in men.

When primary, it commonly begins in the fundus. It may occur by contiguous invasion, either from the liver or adjacent abdominal organs. Cancer may also extend from the gall-bladder to adjacent parts. The primary form is associated in at least 87 per cent. of all cases with biliary calculi, and there has been much discussion as to which is primary, the gall-stone or the cancer. Zenker and others regard the cancer as secondary, starting in the ulcerative and cicatricial tissue caused by the stones, as is thought to be the case in some instances of cancer of the stomach. This, too, may account for the greater frequency of the disease in women, if such is the case, since women are much more commonly the subjects of gall-stone. More recent experience proves that gall-stones are often the exciting cause of carcinoma.

A more or less hard, solid, irregular, and fixed mass is the form assumed by the cancer.

Symptoms.—*Jaundice* is absent so long as the disease is limited to the gall-bladder, but as soon as the biliary duct or the common duct is involved it ensues, so that jaundice is present in 69 per cent., gradually increasing in intensity. There is great *tenderness*, with *pain; vomiting*, sometimes of blood, *bloody stools*, and *dropsy*, at times succeeded by the *cancerous cachexia*. But none of these is distinctive, being found in cancer of the pylorus, duodenum, and transverse colon. The presence of a hard, uneven, and tender tumor in the neighborhood of the gall-bladder, which moves with the liver in respiration, confirms the suspicion. This has, in fact, been found in about 69 per cent. If the disease is seated in the cystic duct, the enlargement of the gall-bladder is comparable to that due to obstruction in that duct from other causes, and may be marked.

Diagnosis.—This is difficult. Pain and tenderness are more marked than in most other affections of the liver, except cholecystitis. Fever and rigors are exceptional and point rather to infectious disease of the gall-bladder or ducts.

Treatment.—All recognized gall-stones should be removed before they give serious symptoms. The treatment can only be palliative after the carcinoma has developed.

Carcinoma of the Biliary Passages.

Cancer of the bile-ducts may be primary or secondary. In either event the first symptom is usually *jaundice*, which grows deeper and deeper until the skin may assume an almost bronze-like hue. A cachexia rapidly de-

velops. There are pain and tenderness and moderate enlargement of the gall-bladder. Enlargement of the gall-bladder is characteristic of cancer of the common bile-duct as contrasted with obstruction of the common duct by gall-stones, according to Courvoisier's law. Moreover the jaundice keeps progressively increasing and never grows better, while in calculous obstruction it may not be very deep or progressive. The disease often escapes recognition until an autopsy reveals it. Cancer may invade the bile-ducts from the gall-bladder and possibly from primary or secondary cancers in the parenchyma of the organ.

The relation of the morbid growth to gall-stones in its vicinity is governed by the same laws as that between gall-stones and cancer of the gall-bladder.

Stenosis of the Biliary Ducts.

Stenosis, or more or less incomplete occlusion of the common duct, may be due to inflammatory adhesion or to compression from without. Sometimes it follows the ulceration attending the passage of a gall-stone. External pressure may be produced by morbid growths and other causes alluded to on p. 434. Notably, cancer of the pancreas is one.

Cicatricial contraction the result of perihepatitis, syphilitic disease, perforating duodenal ulcer, and cholelithiasis should also be mentioned as a cause of external compression of biliary passages, to be recognized, if at all, by aid of the associated symptoms of the disease causing it. In the first there may be a peritoneal friction in the neighborhood of the liver, audible and palpable.

Parasites.

Parasites may enter the larger biliary passages and produce obstruction. Such are echinococci which may enter the ducts primarily in the larval state and develop there the *hydatid* cyst with resulting obstruction; or, as is more frequent, the sac perforates or compresses a duct in the course of its growth. The other symptoms of echinococcus disease are added to those of obstruction thus produced, or the cysts may appear in the stools, vomited matter, or expectoration. Cases are reported in which the *distoma hepaticum* has been found lodged in the hepatic duct, and *round worms* in the common and hepatic ducts. A remarkable specimen, containing a number of lumbricoids lodged in these ducts, is in the Wistar and Horner Museum of the University of Pennsylvania. The symptoms of these last conditions would be undistinguishable from hepatic obstruction from other causes.

DISEASES OF THE BLOOD-VESSELS OF THE LIVER.

Hyperemia.

Passive Hyperemia—Red Atrophy.

The hyperemia of the liver which is of chief clinical importance is passive hyperemia.

Etiology.—It is always due to obstruction to the movement of the

blood toward or through the heart. Valvular heart disease is the most frequent cause, though diseases of the lungs, such as emphysema or cirrhosis, intrathoracic growths, diseases of the pleura, compression of the vena cava, or other cause resisting the movement of the blood through the organ and thus cardiac decompensation are all competent to produce passive hyperemia of the liver.

Morbid Anatomy.—The appearances of the organ after death are determined by the duration of the congestion. If it has been of short duration, the liver rapidly assumes its natural size and appearance after death. Even in long-continued passive congestion the liver after death becomes very much smaller than during life, by reason of the emptying of the blood-vessels which rapidly succeeds death. In other respects, however, after prolonged hyperemia it presents decided changes. It is dark in color, and the vessels still contain an excess of blood, but the *intra*lobular vein—*i. e.*, the central vein of each lobule—and its adjacent capillaries contain most blood, contrasting strongly with the peripheral or *inter*lobular vessel and its adjacent capillaries. There is thus produced in one way that alternation of dark and light tint which constitutes the *nutmeg* liver and which is particularly conspicuous on section. It becomes even more marked at a later stage, when the organ, in its ultimate atrophy, becomes reduced in size, constituting the so-called *red* or *cyanotic atrophy* of the liver—the atrophied nutmeg liver—the histology of which exhibits a destruction of the cells and capillaries in the center of each lobule and a deposit of dark pigment in their places. In the liver thus atrophied the blood-vessels also share in the destruction, and short cuts are established between the branches of the portal vein and hepatic vein, while the latter may also become dilated. The exterior of the liver is smooth, and the organ differs in this respect from the cirrhotic liver, though there is sometimes a slight overgrowth of the interlobular connective tissue.

Symptoms.—The liver at first is *enlarged* and *tender*. The lower border, as determined by palpation, may be as low as the umbilicus and even lower. It may be the seat of expansile *pulsation*, due to regurgitation of blood into it from the right heart. This pulsation is to be distinguished from a motion communicated to the liver by the action of the heart. In the true pulsation the whole liver seems to dilate, and does dilate as the blood flows back into it, as contrasted with the downward movement communicated by the heart. Very characteristic of this enlargement is the changing size of the organ *pari passu* with the degree of congestion, whether spontaneous or the result of treatment.

Ascites is also a symptom. It does not occur, however, until a marked degree of passive hyperemia or *secondary* contraction is attained. The ascites is partly the result of the general stagnation always present, and partly of the congestion of the portal system due to the backing of the blood of the hepatic vein into it. *Jaundice* occurs. It is due to the compression exerted on the fine interlobular gall-ducts by the overdistended interlobular capillaries, thus producing an obstructive jaundice.

Scanty urine of *high specific gravity* is also a symptom, while *hyperemia with enlargement of the spleen* and *hyperemia* of the *mucous membrane of the stomach* are constant, as a result of the same cause.

All of these symptoms must be accompanied by a failing heart to make the diagnosis.

Treatment.—The treatment of passive hyperemia is the treatment of the condition causing it. Rest is most important because it gives the heart an opportunity to regain its tone. All the treatment which is described in that of failing cardiac compensation must be used. Simultaneously the urine is increased, and the general dropsy, ascites, and hydrothorax disappear. Such treatment is aided also by depletion from the portal side by purgatives. Blue mass is the type of these, but colocynth, elaterium, and compound jalap powder, or the simple salts, are also efficient. It sometimes happens that the general dropsy in these cases is dispersed by treatment, but the ascites remains, in which event we must suppose the simple passive congestion to be combined with some degree of atrophy, when the dropsy is more likely to remain. Treatment should now be supplemented by hydragogue cathartics, or, still better by tapping, followed by dry diet and the hydragogues. A dram (4 gm.) or more of compound jalap powder may be given each morning fasting, or elaterium, 1/6 grain (0.01 gm.) every three hours, until the bowels are moved.

Active Hyperemia.

Definition.—This is a much less important condition than passive hyperemia, and, indeed, is rarely recognized. A physiological hyperemia of the liver takes place after each meal, which may be exaggerated and even continuous in those who overeat and overdrink habitually. Such hyperemia may lead to structural change, consisting ultimately in interstitial growth. Like this, also, is the hyperemia which is associated with diabetes mellitus, and which is the associated condition of many glycosurias, whether experimental or the result of disease affecting the diabetic center. Such is a vicarious hyperemia said to take place during suppressed menstruation and after cutting off a hemorrhoidal flux.

Active hyperemia does not, however, present any symptoms referable to it, unless it be that the dull ache and full feeling sometimes felt in the right hypochondrium be caused by such condition.

Treatment.—The treatment must consist of measures which tend to diminish this, mainly the substitution of a scanty for an overabundant diet, simple and easily digested foods, dilute milk, and thin broths, and the avoidance of fats, alcohol, and sugar.

Thrombosis and Embolism.

The portal vein is the seat of thrombosis and of inflammation, constituting pylethrombosis and pylephlebitis. The hepatic artery also becomes rarely the seat of aneurysms.

Pylethrombosis.

Thrombosis takes place in the smaller branches of the *portal vein*, which are constantly being obliterated in the course of cirrhosis of the liver.

Larger branches are sometimes invaded by cancer, or a gall-stone may be admitted into one of them by ulceration, or the lodgment of a parasite may be the focus about which a coagulum may form, while thrombosis may also be favored by the pressure incident to the encroachment of a neighboring tumor. Rarely a thrombus may extend into the portal vein from one of its branches in the intestine or mesentery.

Symptoms.—These include those to be detailed when treating of cirrhosis—viz., ascites, hyperemia in the parts behind the obstructed vessel, with this difference, that the symptoms appear more or less suddenly and severely. It is mainly by the suddenness and intensity of the symptoms that we are led to suspect thrombosis, especially if it be associated with any of the previously named conditions capable of producing it. In such an event the symptoms would come about in the course of a few days, instead of weeks and months. A *caput medusæ* thus rapidly produced would mean that the thrombus had formed, not in the portal vein itself, but more peripherally, causing the para-umbilical veins to be filled from the peripheral branches. These come off the portal vein in the suspensory ligament, and pass out to the neighborhood of the umbilicus by two branches communicating with the epigastric and internal mammary vein.

When pylethrombosis occurs, it sometimes happens that a complete collateral circulation is established, the thrombus undergoing the usual changes, while the portal vein may be ultimately converted into a fibrous cord. Osler reports such a case, in which compensation finally failed, and the usual symptoms, including hematemesis, supervened, and the patient died.

Pylephlebitis.

Mild grades of pylephlebitis probably succeed the thrombosis referred to, but they are of no consequence unless the thrombus is septic. Hemorrhagic infarct does not usually succeed the lodgment of an embolus in a branch of the portal vein, because of the free anastomosis of its branches with those of the hepatic artery, by which the lobular capillaries are supplied. It does, however, sometimes occur. Here again the results are not serious, so long as the embolus is not septic. Much more serious is suppurative phlebitis, the result of septic embolism, or septic thrombosis arising from an inflammatory focus somewhere in the portal area, as in the case of the bowel, dysentery or in the territory of the umbilical vein of the newborn child or in suppurative appendicitis. Pylephlebitis is one of the causes of abscess of the liver. It is associated with the usual signs of septic infection—viz., *chills*, *remittent fever*, and *sweats*, while the symptoms which point to the liver are *pain* in that neighborhood, *jaundice* in most cases, and the signs of *portal vein obstruction* more or less pronounced. Suppurative peritonitis is also sometimes added. Such phlebitis does not always proceed to the degree of abscess formation before death supervenes. The symptoms of abscess will be considered, when treating of that subject, when, too, attention will be called to the diagnosis between it and suppurative phlebitis, so far as it can be made out.

Other Changes in the Hepatic Artery and Vein.

The *artery* is sometimes dilated in cirrhosis of the liver; it may be the seat of endarteritis and sclerosis. Aneurysm of the artery is a rare condition. The symptom is a pulsating tumor, which may be the seat of a murmur. In the cases reported there have been hematemesis, bloody stools, jaundice from compression of the biliary ducts, and pain in the neighborhood of the liver due to compression of adjacent nerves.

The hepatic *vein* is subject to dilatation, alluded to in treating of passive hyperemia; to stenosis, and to thrombosis extending backward from the right auricle.

FATTY LIVER.

Definition.—The term fatty liver is applied to a condition in which the cells of the liver are more or less completely converted into fat. This is accomplished, however, by two distinct processes. In one there is an infiltration of the liver cells with fat drops, which simply push aside the protoplasm and cause its ultimate disappearance by interfering with its nutrition. In the other there is a disintegration or metamorphosis of the protoplasm of the cell into various products, of which one is oil. In the former, fatty infiltration, the cell maintains its integrity, being simply filled with the fat drops, in the latter the cell disintegrates and leaves a residue of which fat is the chief representative. It should be mentioned that some use the term "fatty liver" as synonymous with "fatty metamorphosis."

Fatty Infiltration.

Etiology.—Abnormal fatty infiltration occurs in two ways:

1. In case of overingestion of fat-producing substances, resulting in obesity, of which it is a part, and as the result of which the liver becomes a storehouse for fat. Excessive consumption of alcohol is attended by fatty infiltration, because more carbohydrate is introduced than can be burned up. It is, therefore, stored in the liver cells.

2. In a series of cachectic states, in which oxidation is interfered with and the fat which is ingested is not oxidized, but accumulates in the liver. Such a condition is pulmonary tuberculosis, which is the most common cause of fat-infiltrated liver, except alcoholism.

Morbid Anatomy.—The liver of fatty infiltration is uniformly large, soft, and smooth. Its appearance varies somewhat at different stages. Since the infiltration begins at the periphery of the lobule, we have, in the first stage, a simple distinctness of the line of demarcation between the adjacent acini. In the second stage this has become more marked, contrasting strongly with the darker color of the center of the lobule, and producing one form of *nutmeg* liver—as contrasted with the liver of red atrophy, already described in treating of passive congestion. In the third stage the entire acinus is infiltrated, and the whole organ assumes a uniform yellow or brownish-yellow appearance, from complete fatty infiltration of the cells. The organ is also anemic. In this last stage it is that we have the macro-

scopic changes complete—the softness, the broadened edges, and increase in size, with, however, a decided reduction in specific gravity.

Symptoms.—Outside the physical condition, determined by palpation and percussion, and the causing disease or state, there are no distinctive symptoms. There is no jaundice, and the bile-forming function of the liver seems little interfered with, though the stools are pale. There is no obstruction to the portal circulation, and, therefore, no abdominal dropsy. Percussion recognizes *enlargement of the liver*, which is, however, moderate compared with that of amyloid liver and cancer, extending, as it does, but a short distance below the normal site, where its edge can be felt even through abdominal walls of some thickness. There is no enlargement of the spleen.

Diagnosis.—It becomes necessary to differentiate the enlarged fatty liver from the amyloid liver, which is harder and larger and associated with enlarged spleen and albuminuria. With the hyperemic enlargement of the first stage of cirrhosis it is not likely to be confounded. Such enlargement would be trifling, accompanied by tenderness, and sooner or later succeeded by contraction, while the fatty liver continues to enlarge. From the enlargement due to the cloudy swelling characteristic of the infectious diseases, typhoid and typhus, it is distinguished by the absence of fever and other symptoms of these diseases.

Prognosis.—This depends upon that of the causing disease.

Treatment.—The treatment is that of the disease causing it.

Fatty Metamorphosis.

Definition.—This is a much more serious condition, in which the cell protoplasm is directly converted into fat, or rather, perhaps, into a number of products of which fat is one, while the cell undergoes disintegration. It is the effect of some poison, which has its type in phosphorus-poisoning and in the cause, whatever it may be, of acute yellow atrophy of the liver.

Morbid Anatomy.—The liver, instead of enlarging, undergoes rapid reduction in size, or at least, if there is enlargement, it is of such short duration that it is never recognized. The appearance and condition of the liver, to be described under acute yellow atrophy, are those of the liver which is the seat of rapidly progressing fatty metamorphosis.

Symptoms.—They are those of the diseases causing it, and will be described under Acute Yellow Atrophy.

The **prognosis** is fatal and **treatment** is unavailing.

THE AMYLOID LIVER.

Synonyms.—*Lardaceous Liver; Waxy Liver; Albuminoid Liver.*

Definition.—In the amyloid liver there is an infiltration, in various degrees, of all the tissues of the organ by the so-called amyloid substance. The blood-vessel walls are the first affected, and by preference those of the

intermediate area of the lobule—*i. e.*, that supplied by the hepatic artery, then the central or hepatic vein zone, and finally the peripheral or portal zone. The infiltration begins in the smaller arteries, then invades the cells and capillaries, and in extreme cases pervades all the liver tissue, including connective tissue.

Etiology.—The most usual cause of amyloid liver is prolonged suppuration, especially in connection with tubercular disease of the bones. Hence it is found in children who have had hip disease. For the same reason it is found associated, though less frequently than might be expected, with prolonged tuberculosis of the lungs. Syphilis is one of the recognized causes, whence it may arise as a tertiary manifestation or as the result of bone disease incident to it. Rickets likewise produces some cases, and it is also associated, though rarely, with leukemia, the cancerous cachexia and the infectious diseases.

Morbid Anatomy.—The liver is much enlarged, reaching sometimes enormous dimensions, scarcely exceeded by the largest cancers. Its appearance is waxy or resembles bacon, especially in thin sections. This appearance is partly due to the anemic state. The amyloid parts strike a mahogany-red color with weak solutions of iodin. In addition to the change in size and translucency, the amyloid liver is hard and smooth, its border usually, though not always, rounded, and its fissure exaggerated. In certain syphilitic forms its surface is beset with nodules. Instead of being general, the amyloid change is sometimes circumscribed, when it may be associated with red atrophy. It is occasionally combined with fatty infiltration.

Symptoms.—Beyond the *enlargement*, which is usually manifest, the organ extending sometimes as low as the umbilicus, and, in addition to the symptoms of its causing state, there are none peculiar to the amyloid liver. There is no pain, unless it be the result of an associated syphilitic hepatitis, but there may be a *dragging sensation*, induced by the weight of the organ. There is no jaundice, though the stools may be light-hued, because the secretion of bile is diminished. There is no ascites, except in extreme cases, when it is a consequence of the general hydremia, and not of obstruction in the portal circulation. It is usually associated with *amyloid spleen*, which is enlarged, and with the *amyloid kidney*, which secretes albuminous urine.

Diagnosis.—This is usually easy. The large, smooth, hard organ, the history of the presence of the primary disease, the absence of jaundice and of dropsy, the association of enlarged spleen and albuminuria, admit of scarcely any other interpretation. It is to be remembered, however, that amyloid spleen is not invariably present, and, when present, may be overshadowed and compressed by the large liver. The enlarged liver of leukemia, the result of lymphoid infiltration, may at once be distinguished by the blood examination.

Prognosis and Treatment.—They are those of the primary disease. We have never seen an amyloid liver reduced to the normal size, yet the absence of symptoms growing out of moderate degrees of it makes practical recovery not impossible.

THE CIRRHOSES OF THE LIVER.

SYNONYMS.—*Chronic Interstitial Hepatitis; Gin Liver, Granular Liver; Hob-nail Liver.*

Definition.—Cirrhosis of the liver is a disease characterized by an overgrowth of connective tissue with more or less destruction of the parenchyma of the organ, commonly attended by a harder consistence, sometimes by a reduction of size, at others by enlargement, and at others by no changes in size. Too much stress has been laid in the past on shrinking of the organ as a necessary feature of the disease. Hypertrophic and atrophic as applied to cirrhosis should be abolished. As Adami remarks, they are only relative.

There are various types of cirrhosis which are found affecting the liver. The capsular cirrhosis, where the whole organ is enveloped in a thick fibrous membrane; the cirrhosis due to cardiac disease which has been described, syphilitic cirrhosis, which will be considered; and small cirrhotic patches due to various causes. Besides these there are two forms of diffuse cirrhosis which especially concern the clinician: portal cirrhosis (atrophic), and biliary cirrhosis (hypertrophic).

PORTAL CIRRHOSIS

Laennec's Cirrhosis. Atrophic Cirrhosis.

This form is popularly known as atrophic cirrhosis, but as stated before this term applies only to the terminal stages of the disease.

Etiology.—In many cases it follows the overuse of alcohol, but overeating, especially of highly seasoned food is also a well-recognized cause; syphilis, tuberculosis, and malaria have been given as causes. It occurs in men twice as frequently as in women. It has been reported in children frequently. Alcohol and syphilis seem to be the causes here.

Pathology.—The liver varies much in size. There is, however, always an overgrowth of the connective tissue. It can be very small, one of our cases it weighed a little over one pound. This is the true atrophic cirrhosis, the hob-nailed liver—small, deformed, rough, the surface being covered with small prominent areas, whence the name hob-nailed. In the early stages the liver is enlarged, the surface is slightly granular.

A third form is the fatty, cirrhotic liver.

In all these forms there is increase in the connective tissue. There is hyperplasia and hypertrophy of the liver cells.

In the atrophic form the hyperplasia of the liver cells is most marked. Here great islands of hyperplastic cells are cut off from other parts of the liver, causing the yellow-tawny appearance of the cut surface of the liver. The depressions on the surface are found to correspond to fibrous bands which are of a pinkish color. Histologically the fibrous bands are found to surround several lobules or around single lobules.

Finally the liver cells are destroyed and replaced by the contracting connective tissue.

In the fatty cirrhosis there is more or less fatty infiltration in addition to the usual changes.

Symptoms.—(*a*) *Of Portal Cirrhosis.*—Clinicians have sought with increased effort for symptoms caused by cirrhosis of the liver in its early stages. None are distinctive, but given an enlarged liver otherwise inexplicable in an alcoholic subject, the presence of chronic gastric catarrh manifested by anorexia, nausea and sense of distention; a tendency to gastro-intestinal hemorrhage, recurring slight jaundice, high colored urine and growing anemia, a strong suspicion of the presence of a first stage is justified. The same symtpoms continue as the result of more advanced degrees. The gastric catarrh is the consequence of chronic passive hyperemia, due to obstructed movement of the portal blood. As a result of the hyperemia the mucous membrane of the stomach is more or less constantly covered with mucus, which excites nausea and interferes with secretion of gastric juice. A similar condition exists in the small intestine, causing *constipation*, which is increased by the deficient biliary secretion. This is further shown by the paleness of the stools. The well-known comforting effect of the early morning "dram" upon the inebriate may be due to some action of the alcohol upon this mucus. The disease is usually afebrile. Occasionally there is slight fever with temperature of 100° to 102° F. (37.7° to 38.8° C.).

The late symptoms are mainly the result of the ligature-like effect of the connective tissue on the portal vessels. *Nasal hemorrhage*, often very obstinate, is one of these. So are *gastric*, *intestinal* and *esophageal hemorrhages*, these hemorrhages being often enormous and alarming, but really beneficial, by removing the gastro-intestinal congestion. Either one of these forms of hemmorrhage may be the very first symptom to attract attention. Uterine flooding also sometimes occurs, and even hematuria. Similarly caused is the *abdominal dropsy*, which is often enormous. Four gallons (15 liters) and more are not infrequently removed at one tapping, and sometimes the fluid, from its weight, bursts through the feeble barrier at the abdominal ring, distending the tunica vaginalis. The navel is often pushed out by the enormous distention.

The surface of the upper abdomen and lower thorax, anteriorly, is marked by *overdistended veins*. This is directly due to the backing of the blood into these veins, rendered possible by the anastomotic communication between the portal and caval circulations. Such anastomosis between the rudimentary veins in the round ligament (branches of the portal vein) and the epigastric and mammary veins leads to enlargement of the superficial branches of these veins, and in extreme cases to the formation of a *caput medusæ* about the navel. Communication between the superior hemorrhoidal vein (a branch of the portal vein) and the middle and inferior hemorrhoidal, and through them with the hypogastric veins and vena cava, produces *hemorrhoids*, a characteristic symptom of cirrhosis. Anastomosis between the superior gastric vein (a branch of the portal) and the inferior esophageal, whose blood goes to the cava through the azygos and hemi-azygos, causes a varicose condition of the veins of the lower end of the esophagus which has resulted in fatal hemorrhage. The overfilling of the esophageal and azygos veins may also obstruct the movement of the blood through the intercostal and pleural vessels of the right side, causing rightsided hydrothorax. These dilatations, which have been characterized

as "attempts at compensation," are to be distinguished from the more diffuse dilatation of the abdominal veins seen in the flanks, which are due to the pressure on the cava by extreme abdominal dropsy, preventing the return of the blood of the lower extremities by the cava and causing the effort to return through the more superficial vessels. *Edema of the legs* may occur but is much more uncommon than abdominal dropsy, and, when present, depends upon the further pressure exercised by the enormous accumulation of fluid in the abdominal sac upon the returning blood of the lower extremities.

Jaundice is slight in atrophic cirrhosis, and less frequent than might be expected. It may be because comparatively little bile is secreted. A *sallowness of complexion* is also sometimes present, while a *ruddiness of face* is not uncommon.

Physical examination by palpation and percussion discovers a *diminished* area of *hepatic dullness* when the liver is atrophic or increase in dullness when the liver is enlarged. Occasionally an atrophied liver will tilt on its axis and appear in the examination as an enlarged liver. On the other hand, *splenic dullness* is often *enlarged*, the latter because of resisted return of blood from the spleen through the liver, through the spleen may be enlarged simultaneously through other causes. According to Frerichs, the spleen is enlarged in about one-half of the cases; some even say in three-fourths. In alcoholic cirrhosis especially enlarged spleen is considered evidence of an advanced stage of the disease. It is often impossible to outline either liver or spleen because of the extreme abdominal distention, and tapping must first be resorted to before physical exploration is satisfactory.

The *urine* in atrophic cirrhosis of the liver is generally scanty, of high specific gravity, highly colored, and often loaded with urates, which subside on standing, forming a bulky sediment. The proportion of urea is often diminished, a natural result of the deranged function of the liver, to which modern physiology assigns an important rlôe in urea formation. The urine also contains at times bile pigment, but less frequently than in hypertrophic cirrhosis. *Blood* is also sometimes found in the urine.

In atrophic cirrhosis the *feces* are often wanting in bile and consequently are gray or the color of pipe clay.

Drowsiness and *coma* and even *delirium* are sometimes terminal symptoms, especially in cases where there is jaundice, but also where there is ascites without jaundice. They have been ascribed to cholesteremia.

Diagnosis.—The diagnosis of cirrhosis of the liver is not usually difficult in advanced stage. If one is satisfied that there is a reduction or enlargement of the organ, and there are associated with this no symptoms of acute disease and no history of starvation, if enlarged spleen, acsites and hemorrhages from the stomach are present, we may infer scarcely anything else but cirrhosis.

Tuberculous peritonitis, with its liquid effusion, has been mistaken for cirrhosis, and the wasting which attends advanced stages of the former affection closely resembles that in the latter, but the abdominal tenderness in peritonitis is characteristic, there is fever, and the effusion is never very large. The effusion of the two conditions differs.

BILIARY CIRRHOSIS.

Hanot's Cirrhosis. Hypertrophic Cirrhosis.

The French clinicians, headed by Requin (1846) and Hanot (1875), have studied this form most thoroughly. The subjects are young, more frequently males. The liver is enlarged, weighs from four to eight pounds (2 to 4 kilograms).

The surface of the liver is smooth, the cut surface is smooth of green or greenish-yellow color due to stains with bile, the connective tissue invades the lobules and divides off small groups of liver cells.

The disease apparently begins as a catarrh of the fine biliary capillaries, the portal system remaining free. The spleen is enlarged.

(*b*) *Symptoms.*—The symptoms which distinguish this form from the atrophic variety are:

1. The jaundice, which begins with the first vague symptoms of the disease and gradually deepens as the disease progresses.

2. The absence of hyperemia of the stomach and bowels, of hemorrhoids, enlargement of the spleen, and preeminently of ascites; or the presence at least of only mild degrees of these symptoms.

3. The presence of tenderness in the liver, in addition to its evident enlargement and smoothness.

4. Certain differences in the urine in the two forms.

It is a well recognized fact that when there is jaundice the urine is also colored. In *portal* cirrhosis jaundice is more infrequent, and when present, say in about one-fourth the cases, it is very slight. The same is true to a less degree of the urine, for while the latter is scanty and highly colored, it less frequently contains bile pigment. In *biliary* cirrhosis, on the other hand, bile-stained urine is more common. Blood is never found in the urine of biliary cirrhosis, while in atrophic cirrhosis it sometimes is in advanced stages, as is also albumen. In portal cirrhosis the urea is diminished; in biliary, it is normal in quantity. In biliary cirrhosis the feces are sometimes devoid of bile; at others bile is present.

Rosenstein has made a study of the blood in hypertrophic cirrhosis, and has found the red corpuscles diminished one-half and the leukocytes relatively increased. He also found it associated in certain cases with the hemorrhagic diathesis. The disease is of unknown toxic origin.

The course of hypertrophic cirrhosis is of usually chronic course. It may be put down at two to six years, yet in some cases it is shorter. Osler mentions a case which proved fatal in ten days; another in three weeks. It may be questioned whether these very short cases were not cases of acute exacerbations. All cases terminate more or less acutely. Delirium sets in, the tongue becomes dry, the pulse rapid, and the temperature rises from 102° to 104° F. (38.9° to 40° C.).

(*b*) *Diagnosis.*—Hypertrophic cirrhosis is to be distinguished from cancer of the liver, amyloid liver, multilocular echinococcus disease, and the liver of obstructive jaundice. In *cancer* there is no splenic enlargement, ascites is more frequent, the liver is more uneven, and the patient is older, while in hypertrophic cirrhosis we may have the history of alcoholism.

In *amyloid* liver there is also splenic enlargement, but there is no pain, no jaundice, and we have the etiological history peculiar to amyloid disease.

Multilocular hydatid disease in the liver may present almost identical symptoms, including jaundice and splenic tumor, but in addition there are the nodules on its surface which soften with time.

The liver which is associated with *chronic biliary obstruction* and secondary cirrhosis, while somewhat enlarged, is not nearly so much so as in hypertrophic cirrhosis. Hepatic colic has been present at some time in the course of the disease. The liver is also hard, and the condition is accompanied by marked jaundice and other evidence of hepatic obstruction. Its course, while slow, is more rapid as a rule than that of hypertrophic cirrhosis, while the liver also after a time diminishes in size.

The prognosis and treatment of the two forms will be considered together.

Prognosis.—The prognosis of cirrhosis of the liver is unfavorable if restoration of the normal organ be the object. A liver once the seat of interstitial hepatitis can probably never resume its normal histology. Yet the liver has a good deal of elasticity of function, and if the cause of the condition, supposing it to be alcoholism, is removed and the contraction be not too far advanced, the patient may be restored to comparative health. Generally, however, the course of cirrhosis is from bad to worse, although it may be a slow course, and the patient finally dies of exhaustion and cholesteremia.

It only rarely happens that death is caused by the copious hemorrhages from the esophagus which sometimes occur. We have already referred to two cases in our practice. On the other hand, they frequently relieve the portal congestion, thus giving to the patient a new lease of life. The patient may live many years in comparative comfort.

Treatment.—The treatment of cirrhosis of the liver resolves itself into two parts—first, prophylaxis; second, the relief of the symptoms, and, third, the restoration of the organ to its normal state.

Prophylaxis.—Overindulgence in alcohol, syphilis, and overeating are fertile sources of cirrhosis of the portal type. They should be avoided.

Toward the relief of symptoms the removal of the cause is indispensable. The alcoholic must stop drinking. This, after some temporary inconvenience, of itself brings alleviation. But the effect of gastric congestion remains in part, and sufficiently to cause want of appetite, nausea, unpleasant taste in the mouth, and a general disgust of one's self and everyone else. The mucous membrane of the stomach is swollen, and probably bathed with mucus. The latter can be removed by free drinking of alkaline mineral waters before meals, such as those of Vichy, Vals, and Carlsbad, the effect of all of which is increased when hot. Here, too, as in gastric catarrh—it is really gastric catarrh we are treating—the hot-water treatment is often highly useful by ridding the stomach of mucus. A tumblerful, as hot as it can be borne, is taken slowly before breakfast, or before each meal. Its effect is often highly beneficial. We know no additional explanation of its action unless it be that it may likewise stimulate the secretion of gastric juice. Lavage also relieves this condition and its consequent symptoms.

The congestion which is responsible for this secretion must be removed.

This is best done by the saline and mercurial purgatives. Five to 10 grains of blue mass at bedtime, followed by a dose of sulphate of magnesium in the morning or of Hunyadi or Friedrichshalle water, will deplete the engorged veins and relieve the symptoms for the time being. The mineral waters of Saratoga, in this country, some of which are also purgative, are very useful for the same purpose. A course at Saratoga is greatly appreciated by the confirmed free drinker, and he is always better for some time after it. The hot saline and sulphur waters at Greenwood, Colo., are similar in their effects.

Finally, foods which make the least demand upon the stomach are to be used. Fatty matters are especially contraindicated. In advanced stages milk and Vichy, peptonized milk, and buttermilk may be assimilated when other foods cannot be managed by the feeble digestion, but even these are absorbed with difficulty as long as the mucous membrane of the bowels is much congested.

The abdominal effusion is combated by the purgatives alluded to, and diuretics may be added; of these the acetate of potassium seems more efficient than the bicarbonates and citrates where dropsy is due to hepatic derangements. Perhaps this is because in large doses it has also some laxative effect. Theobromin is often an efficient diuretic in these cases, especially when the heart is in good condition. When the abdominal effusion becomes large, it must be removed by tapping, although the reaccumulation may be very rapid and it may have to be repeated many times. Recently operation has been suggested for permanent cure of abdominal effusion due to this cause. It consists, in a word, in the production of vascular adhesions between the parietal peritoneum of the abdominal walls and the omentum, providing a short cut for part of the blood which must otherwise pass through the liver, the so-called Talma operation to be of value it must be done early.

Nothing can be done to remove the growth of the connective tissue.

General tonic treatment must be instituted. Stychnine and iron are the drugs to use.

SUPPURATIVE HEPATITIS.

Synonym.—*Abscess of the Liver.*

Etiology.—Abscess of the liver is traceable to causes which, in one way or another, are associated with microbic origin. Even traumatic abscess, which it is admitted may occur, is ascribed to an associated infectious agent, although the possibility of abscess excited by simple chemical, as contrasted with bacterial cause should at least be mentioned. Abscess of the liver may be solitary or multiple. The *solitary* or tropical abscess is due to a preexisting amœbic dysentery. Amœbæ coli have been found in the pus of the abscess and in the abscess walls.

The multiple or pyemic or thrombotic abscesses are caused by infectious thrombus, which, starting in the venules of an area drained by the portal vein, extends thence to the branches of the portal vein in the liver, where it gives rise to a suppurative pylephlebitis. This area may be the colon, rectum, the neck of the bladder or the appendiceal region.

Abscesses of the liver may also be caused by infectious emboli arising in the *left heart*, the *pulmonic* or *systemic circulation*, reaching the liver *via* the hepatic artery. Even a noninfectious embolus may excite an abscess if brought into association with pyogenic organisms entering the liver in another way. Such organisms may enter the liver through the common duct from the alimentary canal. This is probably the route of the organism causing suppurative cholangitis, and of that causing the abscess often associated with hydatid cyst of the liver..

Morbid Anatomy. MULTIPLE ABSCESS.—The right lobe of the liver in its thickest part is the most frequent seat of abscess—in two-thirds of all cases. In the multiple, the abscess varies in size from that of a mere point to that of a child's head, the whole right lobe being sometimes converted into one abscess cavity. It may be single or multiple. Rarely, the abscesses intercommunicate. The liver is, of course, correspondingly enlarged. Notwithstanding this, the external appearance of the organ may not be changed. On the other hand, if the abscess is near the surface, there may be a prominence under which fluctuation may be recognized, or the liver may become adherent to the abdominal wall or adjacent viscera. The abscess cavity, if of any size, is usually ragged, and not sharply defined from the surrounding hyperemic liver tissue. Such hyperemia may involve two or three rows of acini.

The contents of the abscess may be pus, or a puriform fluid consisting of the granular débris of cells, oil drops, a few leukocytes, cholesterin and other fat crystals, and numerous crystals of bilirubin. Should the abscess accompany hydatid disease, echinococcus hooklets may be found. The contents of such abscesses is generally a true pus. Any form of abscess may perforate the diaphragm and lung, producing abscess or empyema; or the pus with echinococcus hooklets may be expectorated; or the abscess may burrow into the peritoneum, setting up fatal peritonitis, or into the pericardium, causing fatal pericarditis; into any adjacent hollow organs or into the abdominal wall, discharging externally by fistulous openings.

The thrombotic and embolic forms of abscess always begin as a phlebitis, which rapidly invades the adjacent tissue. Contrary to what is usual in embolism elsewhere, the lodgment of an embolus in the liver is not followed by hemorrhagic infarct.

Symptoms. MULTIPLE OR PYEMIC ABSCESS.—Abscess of the liver is generally associated with *pain* in the hepatic region, with *fever*, very often with *chills*, *sweats*, and sometimes with *jaundice*. The pain is almost invariably accompanied with *tenderness*. It may be deep or superficial, and in the latter event it may be sharp and cutting, because involving the peritoneum. The characteristic *shoulder pain* of hepatic disease may also be present.

Fever is, perhaps, the most invariable symptom, and in no other affection of the liver does it rise so high. Indeed, except acute yellow atrophy and the so-called hepatic fever, there are no other diseases of the liver associated with fever. In the former it is of comparatively short duration, and in the latter it is moderate. The temperature reached in abscess is very high—104° to 105° F. (40° to 40.5° C.)—and may be preceded by chills of corresponding severity, while the fever, in turn, is succeeded by sweats, profuse and exhausting. *Jaundice* is present, but varies in degree.

Solitary Abscess.—The case is one of pyemic symptoms with jaundice, solitary, or amœbic abscess. Fever, pain and septic symptoms are the symptoms found in this form also. There is often cough, sometimes accompanied by expectoration of pus due to rupture of the abscess into the bronchus. Amœbæ coli may be found in the expectoration. The liver is enlarged upward mostly in the right lobe. The enlargement is sharply upward in the nipple line. The face is sallow, pale and possibly jaundiced.

Diagnosis.—This may be difficult at first, but as time passes doubts clear up. *Intermittent fever* very naturally is first thought of in many instances, but it will not be long before this disease can be eliminated. There is no enlargement of the spleen, no history of malarial exposure, no malarial organism is found in the blood, and, above all, antiperiodic therapeutics, so efficient in malarial disease, fails of its purpose. In the absence of malaria and in the presence of the causes usually responsible for abscess of the liver there is little else left to mistake for it. A *pleuritic effusion* on the right side gives dullness on percussion in the same locality, but along with this are the diminished fremitus and diminished vocal resonance characteristic of fluid in the pleural sac, while there may also be the bronchial breathing brought on by compressed lung. A *suppurating echinococcus* cyst may give rise to similar symptoms, but in view of its rarity in this country, is scarcely likely to be recognized until aspiration discovers the elements characteristic of it. The needle should be tried early if abscess be suspected, yet it is evident that in so large an organ an abscess of moderate size may easily elude it.

Hepatic intermittent fever, due to chronically impacted calculus, resembles abscess by its fever, chills, and sweats, and by tenderness over the liver, but the history of hepatic colic is present, jaundice is more marked and obstinate.

Prognosis is fairly good when the abscess is of the solitary type due to amœba coli. Here operation is of value. In the multiple abscess prognosis is fatal.

Treatment.—This is palliative and supporting, except in those cases where surgical interference is possible. The usual measures to relieve pain, nourishing and easily assimilable food, quinin, iron, and stimulants are indicated. Surgical interference in the suppurative type will save many.

PERIHEPATITIS.

Definition.—An inflammation of the peritoneal covering of the liver.

Etiology.—Perihepatitis occurs in a circumscribed area—(1) as the result of extension by continuity from some one of the various diseases of the liver, such as abscess or hydatid cyst; (2) as a part of a general peritonitis, and (3) rarely by the spread of a pleurisy through the diaphragm; (4) it may be also caused by direct violence, as by a blow; (5) it may be the result of a perforating ulcer of the stomach, duodenum or gall-bladder.

Morbid Anatomy.—In the more acute forms there is a fibrinous or puriform product with more or less adhesion. These adhesions may lace off areas between the liver and the diaphragm which may be filled with pus, sometimes large quantities, constituting *subphrenic abscess*, or if there be

perforation of the diaphragm, *subphrenic pyopneumothorax*, more common over the right lobe. In the more chronic form the capsule of the liver is thickened, especially near the portal fissure, and adhesions may take place with adjacent organs, as the diaphragm, stomach, colon, or abdominal wall. The organ may be shrunken and lobulated, and the portal or hepatic vein and bile-ducts may be stenosed. The capsule of the liver is often found thickened at autopsies when no symptoms were present during life to indicate it.

Symptoms.—The *pain* and *tenderness* which, naturally, are attached to this condition, while often exceedingly severe, like those of peritonitis from other cause, are not distinctive of it. Nor is the *jaundice* resulting from compression of the bile-ducts; nor the symptoms of portal *engorgement* due to compression of the portal vein by the inflammatory products. Physical examination sometimes gives more definite results. Thus, a *friction rub* may sometimes be heard in the mammillary line from the seventh rib downward, and in the axillary line from the ninth rib downward; also sometimes in the epigastrium. It is, however, of short duration. If there is a purulent collection, fever is likely to be present, while the right hypochondrium may be distended and the intercostal spaces motionless. The dullness on percussion may extend as high as the angle of the scapula, and all the signs of a pleuritic effusion may be present. On the other hand, the lower border of the liver may be much lowered—as far down as the navel.

The course of perihepatitis may be acute, or it may be much prolonged, when all the symptoms of chronic suppurative processes are added—fever, high temperature, sweats, fistulous communications with other organs, including the lungs, intestines, and abdominal wall.

Diagnosis.—This lies chiefly between that form of the condition under consideration, attended with pus accumulation between the liver and diaphragm, and an *empyema* or *pneumothorax*. The physical signs and later symptoms are very similar, and it is chiefly in the initial symptoms that the two conditions differ, the one beginning with cough and pleuritic pain associated with cardiac displacement; the other with symptoms more abdominal in situation. The liver in pleuritic effusion and empyema is never so much pushed down as in the peri hepatic disease. Aspiration may also be availed of in diagnosis, but will not differentate it for a pleural collection. X-ray examination will often make a diagnosis possible.

Prognosis.—This is grave in the severer forms terminating in suppuration. A protracted illness, with gradual exhaustion of the patient's strength, is prone to occur, which skillful surgical measures may nevertheless turn to recovery. Milder attacks terminate favorably in a few days.

Treatment.—Treatment in the early stage must consist of measures to relieve pain, local and general. Counterirritation by cupping operates to check the disease and also shorten the attack. Sinapisms and fomentations contribute in a less degree to the same end. If suppuration occur, the counsel and aid of a surgeon should be early sought, as it is by his efforts that a cure becomes possible.

Glissonian Cirrhosis.—This is a term applied to a form of perihepatitis in which the capsule is thickened, assuming a semicartilaginous

appearance. It is associated with reduction in size and some degree of interstitial overgrowth and distortion. The capsule may attain a thickness of from 4/10 to 6/10 of an inch (1 to 1.5 cm.).

ACUTE YELLOW ATROPHY OF THE LIVER.

SYNONYMS.—*Icterus gravis; Acute Parenchymatous Hepatitis; Malignant Jaundice.*

Definition.—A rapidly destructive disease of the liver, resulting in fatty degeneration and atrophy of the organ, associated with toxic symptoms and death.

Etiology.—This remarkable and fortunately rare disease is probably due to the action of some virulent poison, autogenetic perhaps, but the nature of which is as yet undiscovered. Pregnancy is one of the conditions acknowledged to produce it, and more cases occur among women than men. It occurs in the sesond half of pregnancy. It has occurred in the course of the infectious diseases, and the usual microbic origin has been held responsible for it, as have been alcoholism and mental excitement. It is also found in phosphorus-poisoning and also in chloroform poisoning. Bacteria have been found in the organ after death. Autodigestive processes have been suggested. Beyond this we know nothing of its cause.

Pathology and Morbid Anatomy.—The destructive process in the liver is almost identical with that of phosphorus-poisoning, and consists essentially in a very rapid destruction of the liver cells. Opinions are divided as to whether this is the result of an acute inflammatory process, or whether the cells are destroyed by some solvent or digestive action.

The liver at necropsy is found very much reduced in size, often to half and even quarter its normal volume. This may take place in three or four days, and even less. A stage of primary enlargement is said to be sometimes present, but is never seen at autopsy. The organ is flattened, flabby, and can be folded over on itself, and the usual lobular markings are either very indistinct or altogether absent. The capsule is loose and wrinkled, and the organ is of a dirty yellow color.

On section, the surface is either uniformly yellow or it exhibits an alternation of yellow and red. The yellow appears, for the most part, in islets, which are surrounded by the red. The yellow represents an earlier stage of the disease. It is soft and spongy, and rises cushion-like above the surface. The red is tougher, more leathery, and sinks below the level of the cut surface. When the organ is uniformly yellow, this later stage, represented by the red, has not been reached before death.

Histologically, the *yellow areas* exhibit softening and apparent solution of the cell network, very few liver cells remaining which retain their own contour. Instead are found disintegrating cells with fat drops of all sizes, the cells being in places still united by their connecting substance so as to maintain the original network. Sometimes crystals of bilirubin, leucin, and tyrosin are met with. The *red areas* consist of a network of capillaries whose meshes contain fat drops and biliary coloring-matter, representing the softened liver parenchyma bereft of its cells. In places there may be seen a

slight degree of cell infiltration of the interstitial tissue, in others irregular branching bands and apparently blind-ending tubes of cells resembling biliary epithelium. The atrophy usually takes place more rapidly in the left lobe.

The skin and organs are generally intensely bile-stained. There may be small extravasations of blood in various parts. The spleen is enlarged and hyperplastic, the renal epithelium and heart muscles are fatty, while the serous cavities contain more than the normal amount of fluid.

Symptoms.—There are no symptoms distinctive of the beginning of acute yellow atrophy. For several days there may be signs of gastro-intestinal catarrh, promptly followed by jaundice. The former include *headache, malaise, loss of appetite, nausea, vomiting, eructations*, and *epigastric discomfort*. Then there suddenly supervene serious symptoms—*delirium, abdominal pain, convulsions*, local or general *drowsiness*, and *coma*. Sometimes the symptoms of this stage are delayed—in extreme cases as long as three weeks.

The *liver rapidly diminishes in size*. Three or four days may see its disappearance to percussion and palpation, favored by further obscuration by distended air-holding viscera. W. von Leube calls attention to a symptom elicited by palpation which he thinks may be of diagnostic value—a more or less permanent "*pitting*" *to pressure in the epigastric region*. He ascribes this to an impression made upon the relaxed liver, to which the abdominal wall fits itself. The *spleen*, on the other hand, is enlarged, the *jaundice* is intense, the *vomiting* obstinate, while there may be *epistaxis, hematemesis, hematuria, menorrhagia*, and hemorrhagic extravasations, while the stools are devoid of bile. The pregnant woman aborts. There is little fever, and in the worst stage there is but moderate rise of temperature—rarely above 101° F. (38.2° C.). The pulse, a first infrequent, increases toward the end to 120 or more.

The *changes in the urine* are very characteristic and have been thoroughly studied. It is deeply bile-stained, is concentrated, the specific gravity often reaching 1030. It is slightly albuminous, and may contain the bile acids, bile-stained fatty casts, and bile-stained renal epithelium. The quantity of urea is diminished, even totally absent. The characteristic feature is the presence of *leucin spheres* and *tyrosin needles* in most cases. These crystals may appear without treatment of the urine or they may come down after slight concentration. In addition are found also aromatic oxyacids, especially oxymandelic acid, all representing products of albumin disintegration.

Diagnosis.—The symptoms of acute yellow atrophy in the first stage do not admit of a diagnosis. This is the more true because there is no symptom, even atrophy, which may not be wanting. Thus, cases have perished from hemorrhage before the disease was recognized or before jaundice appeared in the rapidly terminating cases. In the second stage, on the other hand, the symptoms are so distinctive that it seems almost impossible for one familiar with them to fail to recognize them. It is, however, so rare a disease in this country that the opportunity does not often present itself; hence it is sometimes overlooked because not suspected, the more excusably because grave nervous symptoms may occur even in catarrhal jaundice and in the infectious diseases—as, for example,

in pneumonia, where jaundice is sometimes a symptom. A case of pneumococcicæmia with rapid hemolysis came under our care with all the symptoms of acute yellow atrophy of the liver. *Acute phosphorus-poisoning* so closely resembles acute yellow atrophy that the diagnosis depends largely upon the possible recognition of the cause. There are, however, some differences. The reduction in size of the liver is not so rapid, the nervous symptoms are not so grave, and leucin and tyrosin are not usually found in the urine of phosphorus-poisoning. *Hypertrophic cirrhosis* also sometimes resembles acute yellow atrophy clinically, but the enlarged liver is the distinctive feature of the former.

Prognosis.—This is so unfavorable that recovery may be said to imply an error of diagnosis.

Treatment.—There is no curative treatment. Symptoms should be relieved by the usual palliatives. Headache should be relieved by phenacetin and acetanilid, rather than morphin. An ice-bag may give great relief.

MORBID GROWTHS OF THE LIVER.

The only morbid growths of the liver which are of clinical importance are *cancer* and *sarcoma*. An *angioma* is an interesting new formation of small size, which presents no recognizable symptoms before death. It is composed of vascular tissue and is distinctly capsulated. The large sizes may be as big as a walnut, more rarely still larger. Some pathologists describe an *adenoma*, which others class among the cancers as a trabecular variety. *Myoma* is another form of histioid tumor rarely found in the liver. *Cysts*, represented by the dilatation cyst and the hydatid cyst, are of occasional occurrence.

Carcinoma of the Liver.

Etiology.—Cancer of the liver is a comparatively common disease; it is next in frequency to that of the uterus and stomach. It is, moreover, in the vast majority of cases secondary—in full three-fourths of cases, and of these two-thirds are secondary to primary cancer of the portal area, one-third to primary cancer elsewhere. The stomach is, the most frequent primary focus. Cancer of the liver is most common in male adults between the 40th and 60th year.

Morbid Anatomy.—There are two chief forms in which cancer of the liver presents itself—the *nodular* and the *massive*. Rare forms are radiating, colloid, and cancer with cirrhosis.

1. In the *nodular form* nodules of various sizes are scattered throughout the organ. The nodules vary in diameter from one-fifth of an inch to two inches (0.5 cm. to 5 cm.) or more. They are usually opaque, white, or yellowish-white, and may be very numerous. The superficial nodules project above the surface, and may even be felt through the abdominal wall in the emaciated subject, giving rise to the oft-described "bosselated" feel. These superficial nodules are often umbilicated, because of the disintegration and absorption of the older central cells, leaving a residue of connective

tissue and partially obliterated blood-vessels. The umbilication is confined to the superficial nodules, which also received the name of Farre's tubercles. This variety of nodular cancer may be both primary and secondary. The nodules usually reach a large size in the secondary, and are apt to be more numerous.

2. The *massive form*, in which there is one large cancerous mass, greatly increasing the bulk of the organ. It is grayish-white in color, and may reach four or six inches (10 or 15 cm.) in diameter. This form is primary.

3. The *radiating form*, usually pigmented, in which the nodules may also be multiple, but smaller and less numerous than in the nodular form. It is a form of secondary cancer.

4. A *colloid form*, rare and only secondary.

5. A rare form is *cancer with cirrhosis*, in which the liver is but slightly enlarged, weighing 4.5 to 6.5 pounds (*circa* 2 or 3 kilograms), and presents a greenish yellow appearance, studded over with *small white nodules* not unlike those of the hob-nail liver, the same appearing in large numbers when the organ is cut.

All varieties of cancer are subject to *degeneration*, but the secondary forms degenerate more rapidly. The change is a fatty metamorphosis of the cells, associated sometimes with rupture of blood-vessels and large extravasations of blood, which may even burst into the peritoneum and gall bladder. There may be occasional *suppuration* around the nodule.

As to the histological origin of cancer, the primary forms start in the liver cells; they are true epitheliomata, the capillary network forming the primary stroma, to which an independent growth of stroma is subsequently added. The secondary forms are embolic in origin, chiefly through the branches of the portal vein, but possibly by the hepatic artery, with or without intermediate involvement of the lung, the first new cancer cell in this form being an infected cell of the capillary wall, whence the parenchymal liver cells are in turn affected. The stamp of the pigmented radiating cancer is, perhaps, thus derived, and illustrates this mode of invasion. The secondary forms repeat the type of the primary varieties. The cells are mainly epithelioid, but may be polygonal and even cylindrical. They exhibit various grades of fatty degeneration.

The liver is variously enlarged by these different forms of cancer, the maximum product being the largest produced by any disease of the liver (see Fig. 103).

SARCOMA.—Of the remaining morbid growths of the liver, sarcoma alone demands a few words. It is almost invariably secondary, very few cases of primary sarcoma of the liver having ever been found. Secondary sarcoma of the liver includes melanosarcoma, lymphosarcoma, and myxosarcoma. The melanosarcoma is the most frequent and interesting. It is always secondary and usually multiple, though a diffusely infiltrated variety exists, giving the liver on section a granitic appearance. Melanotic sarcoma of the orbit often precedes it, and it is sometimes a part of a general melanotic distribution over the body, including the skin. *Sarcoma of the liver is said to be never associated with ascites.*

Symptoms.—Very rarely cancer of the liver may be latent, except as to a vague ill health explained by the findings of the autopsy. In most in-

stances such ill health grows worse more or less rapidly, and examination of the liver shows *enlargement*, to which may or may not be added recognizable *nodules*. The enlargement may extend beyond the umbilicus, but it is not usually so great, and in some cases there is none whatever. To inspection the enlargement is first seen in the upper zone of the abdomen, and produces a change of configuration which involves commonly the whole upper abdomen. Rarely, the nodules may be seen. The *superficial veins* are enlarged.

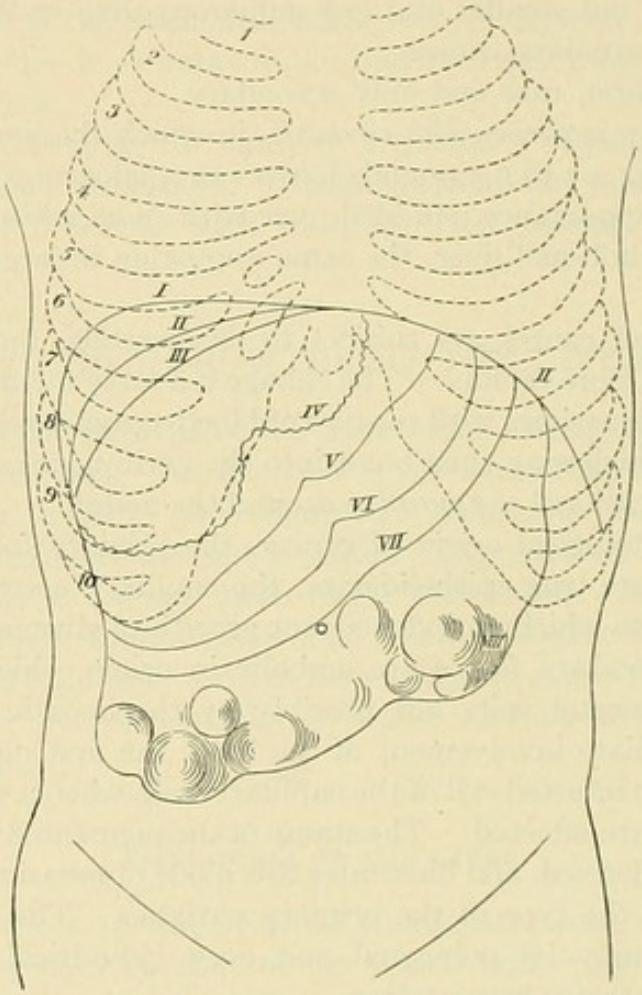


FIG. 103.—Showing Appropriate Enlargement of the Liver Corresponding to the Different Diseases Described in the Text—(*after Rindfleisch*).

1. Position of the diaphragm to the maximum enlargement (carcinoma and in abscess). *II. II.* Normal situation of the diaphragm. *II, III.* Relative dullness. *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 cancer, leukemia, and adenoma.

The other signs of ill health alluded to, apart from those of a primary cancer elsewhere, are *loss of appetite*, *nausea*, a *sense of epigastric fullness*, *pain* in the epigastric or hypochondriac region or in both simultaneously. The pain may be lancinating and extend to the right shoulder. To this *tenderness* is sooner or later added. Indeed, perhaps tenderness precedes. *Emaciation* may have preceded the more striking degree of these symptoms and increase rapidly, while the characteristic *cachexia* develops *pari passu*. An examination of the blood shows a *reduction of hemoglobin and corpuscles*, and as the blood degenerates, *edema* develops. In some cases there is *fever*, especially toward the end, with a temperature of 100° to 102° F.

(37.8° to 38.9° C.), more or less intermittent, but rarely associated with rigors.

Obstructive *jaundice* is a frequent symptom in *carcinoma hepatis*—it may be said in fully half the cases. It is due to compression of the smaller billiary passages, and does not usually reach a high degree. Nor are the feces usually devoid of bile. If the latter event occurs, and the jaundice is intense, it means that some of the larger ducts are obstructed, while involvement of the gall-bladder or the portal lymphatics may be suspected. *Jaundiced urine* is about as constant as jaundice itself. The presence of melanin is said to point especially to the presence of the pigmented varieties of cancer. Albuminuria is, on the other hand, unusual.

Ascites is a rather infrequent symptom, and can only occur when the portal vein or branches become involved either by compression or invasion. This is common in carcinoma with cirrhosis. Should, however, a bloody fluid be obtained by tapping, and a tumor of the liver be present, the indications are that the tumor is cancer. *Enlargement of the spleen is rarely present* in cancer of the liver.

The duration of the disease ranges from *three* to 15 *months*.

Diagnosis.—This is not always easy, even if there is enlargement. It is simplified if the nodules can be felt, or if there is recognized primary cancer elsewhere.

The smooth, enlarged liver of cancer is distinguished from that of the more benignant conditions of fatty liver and amyloid liver by the absence in these two of grave symptoms and of jaundice. The *fatty liver* is softer than the liver of cancer, the *amyloid* is harder, more often smoother, while its rounded border can sometimes be felt. It is also accompanied by enlarged spleen. In *abscess* of the liver the organ may be soft or doughy in consistence, and the same may be true of the abdominal walls over it. There are also the causes of abscess of the liver, and among symptoms the characteristic chills, high fever, and sweats.

Multiple *echinococcus cysts* may furnish similar local signs, even the "bosselated" feel, but hydatid disease is rare in temperate climates; the nodules are softer, the disease is of longer duration, and is less rapidly followed by wasting. Enlargement of the spleen is quite common in hydatid disease, present, it is said, in nine-tenths of all cases. It is rarely present in cancer. Jaundice is even more frequent in this disease than in cancer—in four-fifths, as contrasted with a little more than one-half. Aspiration may aid in the solution. Of other affections attended by uneven surface of the liver the *amyloid organ beset with nodules* offers difficulties, but the lesser gravity, the longer duration, and, especially, the syphilitic history solve the question. Cancer, as a rule, is not associated with enlarged spleen, but the rapid enlargement of the liver in amyloid disease sometimes obscures the enlarged spleen and even interferes with its development.

Doubt sometimes arises in the presence of *certain stubborn forms of jaundice* as to whether cancer may not be the cause, especially as in some of these there is rapid loss of weight. If there is enlargement of the liver, the solution is less difficult, because in simple jaundice there is no enlargement; but in its absence time alone can settle the question; for stubborn as these rare cases of jaundice are, they are less so than cancer, while even

if they are not followed by ultimate recovery, their course is much longer than that of cancer. Should ascites arise, the question is settled in favor of cancer. It may sometimes be difficult to decide between cancer and *hypertrophic cirrhosis*, which also furnishes an enlarged, hard, more rarely nodular liver, with jaundice. Carcinoma occurs in persons over 40 years of age, hypertrophic cirrhosis in those younger. Carcinoma produces cachexia, hypertrophic cirrhosis does not. Carcinoma produces marked tenderness, hypertrophic cirrhosis but slight. A possible cause in either case must be sought, primary cancer elsewhere pointing to cancer, and the alcoholic habit to cirrhosis, to which also the enlarged spleen and the absence of cachexia point. A family history of cancer, if present, adds weight to other signs of cancer of the liver. Many cases of syphilis of the liver are mistaken for carcinoma. Therefore in every doubtful case of enlargement of the liver, a Wassermann, reaction should be done, and inunctions of mercury should be given.

There is no special reason why cancer of the liver should be distinguished from *sarcoma* or *adenoma*, as the clinical significance of the various conditions is about the same. But if, along with a primary sarcoma elsewhere, as in the orbit, there appears enlargement of the liver, then the inference is reasonable that a secondary sarcoma is there established. Melanosarcoma is more likely to invade other organs, as the lungs, kidneys, spleen, and even the skin.

There is no sign by which secondary cancer can be distinguished from primary, except by the presence of primary cancer elsewhere, notably in the stomach, breast, large intestine, uterus and appendages, and the presumption based on the fact that the majority of all cases of cancer of the liver are secondary. Careful search should, however, be made for cancer in all organs in which primary cancer is likely to occur. The gastric secretion should be investigated chemically, the rectum explored by the finger and speculum, the uterus by the finger, speculum, and sound. Such investigation is further useful in the settlement of the diagnosis of cancer of the liver, for a doubtful case becomes confirmed if a primary focus can be found.

Prognosis.—This disease is invariably fatal—usually in from three to 15 months.

Treatment.—This must consist in attempts to relieve the discomfort and prolong the life of the patient.

SYPHILIS OF THE LIVER.

Definition.—Syphilis of the liver includes several morbid conditions due to this specific poison, which are best considered under a single title.

Etiology.—Syphilis of the liver may be the result of acquired or inherited syphilis.

Morbid Anatomy.—1. The product in the liver of *inherited* syphilis is always a cellular infiltrate, which may be diffuse or localized. (1) The diffuse infiltrate produces an enlargement and hardening of the organ, which gives place to a reduction in size and unevenness due to contraction of the newly formed connective tissue. (2) The circumscribed product, more rare as the result of inherited syphilis, is the gumma. The gumma is rather a

product of acquired syphilis, but rarely also it is found in connection with hereditary syphilis.

2. The changes in the liver due to *acquired* syphilis are regarded as one of its tertiary manifestations, and do not show themselves until some time after the primary infection—it may not be for several years. They are represented by an interstitial hepatitis, by the syphilitic gumma or syphiloma, by amyloid disease, and occasionally by endarteritis. Diffuse interstitial hepatitis does not differ essentially from the more usual forms of nonspecific cirrhosis. The ultimate product is sometimes very irregular, and the lobules preserve a palpable distinctness. The gumma is the most characteristic lesion of tertiary syphilis. It is a nodular growth, which may be as small as a pea or smaller, or as large as an orange—from 1/5 to four inches (five millimeters to ten centimeters) in diameter. A favorite seat is the convexity of the organ near the suspensory ligament; another, on the under surface in the connective tissue embracing the portal vessels; while it is also found in the substance of the organ. The tendency is to cheesy change in the center of the nodule, and to contraction, which distorts the liver and reduces its size, with the formation of cicatricial markings and furrows. These cicatrix-like puckerings and fibrous bands are found also on section of the syphilitic liver. Endarteritis sometimes invades the smaller, and even the larger, branches of the hepatic artery and portal vein.

Symptoms.—Syphilitic changes in the liver are often first discovered at autopsy. When symptoms are produced during life, they are commonly those due to portal obstruction, as already detailed in treating ordinary cirrhosis. *Jaundice* may be thus caused. It is not a frequent symptom, yet it was early made a matter of record, A. D. 1493–1541. Gubler collected seven cases in which jaundice followed syphilitic infection. It accompanied a syphilitic exanthem, and was also preceded by *digestive disorders, loss of appetite, nausea, diarrhea, bitter taste in the mouth*, and *pain in the epigastrium*. There has been under observation one case precisely fulfilling these conditions pointed out by Gubler. The jaundice may be slight, moderate, or severe. It rapidly attains its maximum intensity, lasting a variable time, seldom more than a fortnight. Though the explanation may not be immediately easy, Gubler gives sufficient reasons for justifying a relation of cause and effect. It is possible that the poison may act like certain other poisons which produce grave icterus, as phosphorus. On the other hand, it is quite as likely that it may arise from a duodenal and biliary catarrh, the result of the general disturbance, especially as it is so often associated with other symptoms of this condition—viz., loss of appetite and nausea. Or it may be the result of biliary obstruction by the contracting processes of the syphilitic liver as already stated.

Enlargement of the spleen is an associated symptom when there is amyloid disease, to which ascites may also be added. Sometimes the larger nodules of gummy growth can be felt through the abdominal walls, when the diagnosis must be made between syphilis of the liver and carcinoma, a differentiation greatly aided by the history of the case.

Diagnosis.—This depends most largely upon the history of the case, which must be carefully sought. Nor should the physician be satisfied with

a negative history, in view of the fact that it is so common for syphilitic subjects to deny infection, even though they know it is to their interest to tell the truth. Careful examination should, therefore, be made for secondary symptoms, such as glandular enlargement or cicatrics and markings left by syphilids. A Wassermann reaction should be made in every suspicious case.

Prognosis and Treatment.—Patients should be subjected to the usual syphilitic treatment by iodid of potassium and inunctions of mercury as soon as the diagnosis is established, and even when it is doubtful. For while early treatment may be efficient in preventing new growths, it is less certain that when present they can be removed by antisyphilitic treatment. Certain cases clear up in a remarkable manner. We have seen a tumor of the liver as large as a grapefruit presenting in the epigastrium, which disappeared in *one week* under mercurial inunctions.

PARASITES OF THE LIVER.

Echinococcus Disease, or Hydatid Cyst of the Liver.

Etiology and Pathogenesis.—The most important and interesting of the parasitic diseases of the liver is the echinococcus or hydatid cyst, caused by the embryo or larva of the *tænia echinococcus*, a minute tape-worm, consisting of three or four links, and about 1/5 inch (four to five mm.) long. Its natural habitat is the upper part of the intestine of the dog, the wolf and jackal. The worm is not often found in this country, (see Fig. 104) and can be easily overlooked, appearing as minute, thread-like body, adhering to the villi of the intestine of the dog. Hydatid disease, though not very common, is still, nevertheless, more so than would be expected from the seeming rarity of this worm. In Australia and Iceland, where the intercourse between men and dogs is more intimate, hydatid disease is comparatively frequent. In the latter country 28 per cent. of all dogs are said to be infected; in Copenhagen, four per cent.; in Zurich, 3.9 per cent.; in Lyons, 7.1 per cent.; in Berlin, one per cent.; and in Leipzig, none, as far as investigated.

The ovum of this tape-worm, entering the human intestine with food or drink, has its shell dissolved by the digestive fluids; the larva is liberated, and bores its way by its stilettos and hooklets into a branch of the portal vein, through which it is carried to the liver. Lodging there, the hooklets disappear, and the embryo becomes a small cyst, called the *proscolex*, possessed of two layers—an external cuticle of laminated structure, the ectocyst, and an internal parenchymatous or germinal layer, the endocyst. Within the cyst is a clear fluid. Surrounding the cyst is gradually developed a capsule of connective tissue, due to reactive inflammation.

At the earliest stage at which these bladders or resting embryos have been with certainty observed—by Leuckart in the pig four weeks after feeding with ripe proglottides—they form solid, spherical bodies, 25/100 to 35/100 of a millimeter in diameter, and are called *proscolices*. At this stage they resemble a mammalian egg, and are subsequently differentiated into the bladders.

Development from Proscolex.—When from 15 to 20 millimeters in diameter

this proscolex, or bladder-worm, proceeds to development of numerous heads or scolices. It may give rise, first, to a single head, arising from the germinal layer producing a *cysticercus;* second, to many heads, each of which is termed a *cenurus;* third, to numerous heads produced, not directly from the germinal layer, but indirectly from special delicate sacs called *brood capsules*, which arise as minute elevations from the cells of the germinal layer. In these elevations a small spheroidal cavity appears, gradually increases in size, and becomes lined internally with a delicate cuticular membrane, outside of which is a layer of cellular structure. Thus, the wall of the brood capsule consists of two layers like those of the mother bladder, but inverted as to relative position, as if the brood capsule were an invagination of the mother bladder. These brood capsules exhibit active movements.[1] From the internal wall of the brood capsule arises the head, first as a discoidal thickening, growing into an externally situated club-shaped process, perforated longitudinally by a tube-like continuation of the cavity. While an external protrusion of the brood capsule, it may be temporarily inverted. At the distal end of this protrusion, furthest from the point of attachment, the suckers and hooks of the head or scolex are formed. The hooklets appear as a thick fringe of prickles, all of which, except the foremost rows, subsequently drop off. Thus, in different stages of development, heads to the number of 10, 15 or 20 may live within one capsule, and in large bladders the included capsules may number thousands. From these, should they reach the intestine of a suitable host, the proglottides of the strobile, or sexual worm, are formed by lengthening and transverse segmentation. The period of development from the scolex condition to that of the adult worm varies from four to eight weeks.

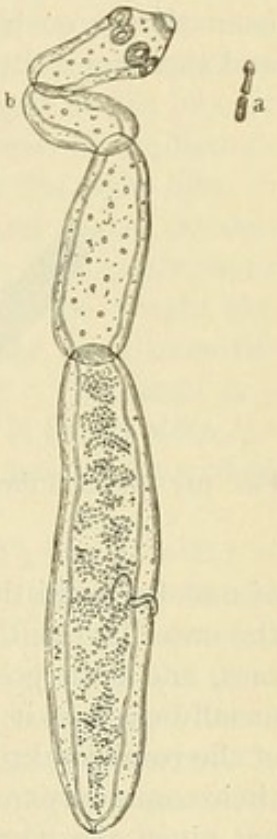


FIG. 104.—Tænia Echinococcus, from the Dog—(*after Heller*). At *a*, natural size; at *b*, magnified.

In the liver the hydatid bladder thus described consists of a single sac, which may attain an enormous size, bearing on its surface brood capsules containing scolices in varying number and stages of development. This is the form of cyst known as *echinococcus veterinorum*, because common in the domestic animals, though frequently also found in man.[2]

Development by Daughter Cysts.—In another method of development secondary and completely separated bladders may be formed, either inside or outside the primary or mother cyst, constituting daughter cysts. The former, or endogenous type, is that usually met in man—*echinococcus hydatidosus* of Leuckart, *echinococcus endogenus* of Kuhn—and arises either by vesicular transformation of the scolices of the brood capsules, or by in-

[1] They are easily ruptured and may escape observation altogether, whence it has been inferred that connection between the heads and brood capsules is temporary, and that, after separation, the living scolices float free in the fluid of the mother bladder. According to Leuckart, however, all parts of the echinococcus—mother bladder, brood capsules, and heads—are throughout life in direct continuity with one another. According to Verco and Stirling, it may be that the scolices are also formed directly from the germinal membrane, in evidence of which they state that they have examined a specimen which shows four heads sprouting directly from the germinal membrane of an exogenously developed daughter cyst.

[2] J. C. Verco and E. C. Stirling in Allbutt's "System of Medicine," vol. ii., 1897, p. 1110.

foldings of the parenchymal layer. The daughter cysts thus formed and lying within the parent cyst, with which they correspond in structure and behavior, also give rise to brood capsules and scolices. These daughter bladders may also bud endogenously and exogenously, and produce a third or fourth generation within or without themselves, the whole brood being contained within the mother bladder.

The exogenous type—*echinococcus exogenus* of Kuhn—is less common in man, but is frequently met in domestic animals, especially the pig. In this form the secondary bladders arise from small granular masses in the deeper layer of the cuticle of the mother cyst, probably ultimately derived from the parenchymal layer. They assume a special cuticular covering, and their central parts clear up and liquefy. As the centripetal formation

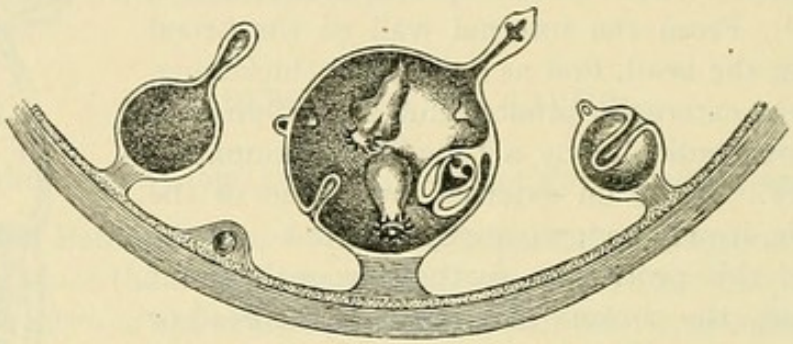


FIG. 105.—Section through an Echinococcus Cyst with Blood Capsules—(*from Braun, after Wax Model*).

of new layers in the cuticle of the mother bladder goes on, with rupture of the outer layers, the new formations make their way externally as separate sacs, and undergo subsequent development outside of the mother bladder, usually close to it, though at times, as in hydatids of bone, the individuals of the resulting broods may lie at some distance from one another and from their common parent. It is to a special variety of this latter that Virchow has given the name *echinococcus multilocularis*, wherein the cysts, becoming surrounded and joined together by thick capsules of connective tissue, form a hard tumor composed of vesicles the size of a pea, often resembling, *en masse*, colloid cancer. In the spaces are found remnants of the echinococcus cyst, at times hooklets or scolices, by the discovery of which their true nature is determined. At other times they are barren. Most cases of this form of disease have been met in Bavaria and Switzerland, but one case being reported in this country—by Delafield and Prudden, in their "Pathological Anatomy," third edition, page 372. The subject was, however, a German, who had been in the country five years.

The *fluid contents* of the young cyst are clear and limpid, have a specific gravity of 1005 to 1009, are nonalbuminous, but contain a small quantity of chlorid of sodium, occasionally a trace of sugar, succinic acid, or hematoidin. Scolices and booklets are almost always present, and are of great diagnostic value.

The hydatid cyst ranges in size from that of a pin's head to a child's head. It grows very slowly, and may be in the liver for many years—some say as many as 20. Ultimately it dies, the walls contract, their contents become inspissated and walls themselves calcified. Sometimes they

suppurate, the cysts forming large abscesses; or they may rupture in various directions with corresponding mischief, including sudden death from collapse. The bile passages and inferior cava have been seats of rupture.

Symptoms.—Small cysts may occasion no symptoms, being often unexpectedly found at necropsy, and under any circumstances the failure of health is very gradual at first. As cysts become large they produce a *sense of weight or dragging* in the region of the liver, and other symptoms, depending on their size and situation; *jaundice*, if they cause obstruction of the biliary passages; *dyspnea* and *cardiac disturbance*, if they encroach on the lungs or heart; *pyemic symptoms*—that is, fever, sweat, and sometimes chills, with rapid exhaustion—if they suppurate. The liver may become very much enlarged, demonstrable by inspection, palpation, and percussion. If there is a single superficial cyst, either in the right or left lobe, it may be felt as an elastic or even fluctuating tumor; or there may be the distinct feel of a nodular growth over the liver. If posterior in the right lobe, it may encroach on the inferior part of the lung and pleural space, causing dullness on percussion posteriorly and postero-laterally, and other signs of pleuritic effusion. *Hydatid thrill* or *fremitus* is always to be sought for. It may be found, if the cyst is superficial, by placing one hand over the tumor and tapping lightly with the fingers of the other. The result is a vibrating or trembling movement felt for a short time. It is not often obtainable, and is possible only with superficial cysts. It has been ascribed by Briancon to the collision of the daughter cysts.

If rupture occurs, other symptoms are added. The pleural cavity is often invaded, or the lungs, as evidenced by the expectoration of cysts and hooklets; the bile passages, by the production of jaundice or increased jaundice; and the subsequent appearance of hooklets and cysts in the fecal discharges. Rupture into the stomach is manifested by vomiting of hooklets and cysts; into the vena cava, by embarrassment of right cardiac action and pulmonary thrombosis from lodgment of cysts; into the pericardium, by fatal pericarditis; into the peritoneum, by fatal peritonitis; and into the abdominal wall, by outward discharge.

The spleen is commonly enlarged.

Diagnosis.—The differential diagnosis depends on the recognition of hydatid fremitus or on some of the pathognomonic features just mentioned, and the history of the case in connection with the slowness of development of the symptoms. The resemblance to *cancer* is sometimes very close, in consequence of the presence of nodular swellings over the liver, and to *syphilis of the liver* for the same reason. In cancer the health fails very much more rapidly, but in syphilis scarcely more so, and the history and blood examination must here again come to our assistance. When suppuration takes place, we have the symptoms of *abscess* of the liver. The recognition of sugar in the fluid obtained by tapping is presumptive evidence of its hydatid nature.

Prognosis.—When the disease develops sufficiently to manifest symptoms, the chance of spontaneous recovery is very slight. It is possible when external rupture takes place, but this should be anticipated by operative interference, which is often successful.

Treatment.—No medicinal treatment avails, while spontaneous cure is

not infrequent, by reason of the death of the parasite before the development of the disease to a recognizable degree. A surgeon should be consulted as soon as the diagnosis is made. A preliminary tapping is justified under strict antiseptic precautions, and, in fact, has been succeeded by permanent recovery. Australian surgeons have had the largest experience, and it appears of justify the bolder course of incision and evacuation of the cysts rather than the more conservative method of first securing adhesion of the sac to the abdominal walls and then laying open the cyst and evacuating the contents. The former practice of injecting the sac with iodin has also been discontinued. Should suppuration take place, the treatment becomes that of abscess of the liver.

Other Parasites of the Liver.

The remaining parasites of the liver are of pathological rather than of clinical interest.

The arthropoda are presented by the *pentastomes*, of which the *pentastomum denticulatum*—larval form of the *pentastomum* or *linguatula tænioides*—has been found in the liver. The adult worm is lancet-shaped and marked with numerous rings. The female is from three to five inches (8 to 13 cm.) long, the male little less than one inch (1.8 to 2.5 cm.). The adult worm has been found in the nostril of man.

The *cystercus cellulosæ* and *psorosperma* are rare parasites. Of the latter, the *coccidium oviforme*, which is very common in the liver of the rabbit, produces whitish nodules, as in other organs, ranging in size from that of a pin to that of a split pea, and even larger. They may produce fever of an intermittent type, diarrhea, nausea, and tenderness over the liver or other organ invaded with enlargement.

In examining a case of suspected hepatic disease the following questions should be raised with a view to eliciting important facts which bear upon the diagnosis: First, whether there has been or is syphilis; second, suppurative disease or rickets; third, alcoholism; fourth, enlargement of the spleen; fifth, elevation of temperature; sixth, jaundice; seventh, what has been the duration of the symptoms?

DISEASES OF THE PANCREAS.

Almost the only diseases of the pancreas which possess much clinical interest are cancer and pancreatitis. Reginald H. Fitz has invested the subject of pancreatitis with increased interest by his masterly Middleton Goldsmith lecture, and more are now recognized antemortem than previous to its publication. The remaining diseases are, however, of great pathological interest.

ACUTE PANCREATITIS.

Definition.—Acute pancreatitis is an acute inflammation, affecting primarily the fibrous and fatty interstitial tissue of the organ. It is a rare

affection. Fitz divides it into hemorrhagic, suppurative and gangrenous, but as suppuration and gangrene are terminations rather than initial features, and hemorrhage is at least a very frequent primary etiological feature, It will be treated under the single heading of *acute pancreatitis*.

Etiology.—It may begin with hemorrhage, which may be traumatic. Most subjects are between 26 and 70 years old. The majority are men. A few are alcoholics. James M. Anders[1] collected 40 cases of pancreatic hemorrhage, in 34 of whom the sex was given. Twenty-five of these were males and nine females. The ages of 30 were stated, of whom 13, or 43.3 per cent., were over 45. Many had been previously subject to gastric and gastro-intestinal derangements, often inflammatory. The causative gastro-duodenitis extends probably from the bowel to the pancreatic duct. Pathogenic organisms play an undoubled rôle.

Morbid Anatomy.—This varies with the stages or varieties, which, as seen at necropsy, are *hemorrhagic*, *gangrenous*, and *suppurative*. In the *hemorrhagic* stage the pancreas is enlarged throughout or at its head, and is infiltrated with blood, which imparts its color in different shades and may invade the pancreatic duct. The hemorrhagic foci may alternate with white spots of *fat-necrosis*. The hemorrhage may extend into the peripancreatic tissue or the mesentery, mesocolon, omentum, and beyond to the brim of the pelvis. On minute examination round cells and red blood disks are found in the ducts and acini. Many lobules are in a state of coagulation-necrosis, while bacteria are present in large numbers.

If the patient survive the first few days—say the fourth day—the condition passes on either to *gangrene* or *suppuration*. If to *gangrene*, the tip or the entire gland may be converted into an offensive, dark, slate-colored mass, which softens and becomes shreddy. Gangrene may set in almost simultaneously with hemorrhage. The organ may become completely sequestrated in the smaller omental cavity, attached only by a few shreds. The adjacent parts exhibit the appearance of peritonitis, with dirty, purulent extravasate. Disseminated *fat-necrosis* may be present. The spleen may be enlarged and its veins thrombosed, as may be also the portal vein.

In the *suppurative* termination the organ is enlarged, and contains numerous small abscesses, intervening parts being hyperemic. There may be peritonitis of adjacent areas of the peritoneum. There may be diffuse suppuration or small abscesses disseminated throughout the organ. In the chronic form there may be a solitary abscess as large as a hen's egg, with cheesy contents. The lesser omental cavity and peripancreatic tissue may be invaded; rarely, also, the liver. Fat necrosis in this form is a rare condition, while thrombosis of the splenic and portal veins may still occur.

Symptoms.—The disease begins suddenly with *abdominal pain*, sometimes succeeding attacks of indigestion. It is severe and in the upper left quadrant of the abdomen and in the course of the pancreas, but it may extend throughout the abdomen. It is ascribed to stretching of the celiac plexus of nerves. There is also tenderness. The pain is usually followed by *vomiting*, rarely by nausea alone. The vomited matter may be bilious or black. There may be diarrhea. The upper abdomen becomes swollen and tympanitic, or the *tympany* may be general. The *temperature* is *sub-*

[1] "Pancreatic Hemorrhage," "Journal of the American Med. Assoc.," December 2, 1899.

normal or slightly *elevated*. Death occurs in untreated cases usually within three days, but may be delayed a week. If the patient lives longer, the case becomes one of gangrenous, or suppurative pancreatitis. Recovery may occur, though rarely.

If the gangrenous or suppurating termination succeeds, *chills*, *fever*, abdominal *swelling*, *tympanites*, *tenderness*, *jaundice*, collapse, and death ensue.

If suppuration occurs, life may be prolonged for three or four weeks, and there may be added *high temperature* and *irregular chills*, with exacerbations and remissions and signs of deep-seated peritonitis in the epigastric region.

Diagnosis.—This is based upon the foregoing symptoms and their suddenness, especially the *circumscribed tympany*. The disease is to be differentiated from the effects of *irritant poison*, *perforation of the stomach* or *biliary tract*, and *acute intestinal obstruction*. The history eliminates corrosive poison. Perforation of the stomach is preceded by symptoms of ulcer, and of the biliary passages by symptoms of gall-stones. There is no tenderness localized in the region of the pancreas in intestinal obstruction, which is rare in the upper part of the small intestine. Obstruction in the large intestine must be eliminated by measures calculated to determine the patulousness of the bowel. Laparotomy has been done for intestinal obstruction, and pancreatitis was found.

Prognosis and Treatment.—The former is almost always unfavorable. If recovery takes place, it is accidental rather than the result of medical treatment, which, in the main, can only be palliative, and such as is demanded by peritonitis. Surgical treatment is of great value. A surgeon should at once be consulted in all suspected cases and operation should be done early.

Chronic Pancreatitis.—This consists of an interstitial overgrowth, by which the organ is hardened and slightly enlarged. The secreting structure is compressed and degenerated. It has frequently been found in diabetes. There may be pigmentary deposits, and pancreatic calculi may be found in the ducts.

Symptoms.—There are those above mentioned, in a milder form. Fat and sugar digestion are poor and large quantities of undigested matter especially sugar and fat are found in the feces. Jaundice may be especially present in this form where the pancreatitis is due to an extension of an infection from the duodenum. The relation of chronic pancreatitis has been pointed out of late particularly by Deaver. The only available treatment is surgical.

CANCER OF THE PANCREAS.

Morbid Anatomy.—Though a rare disease, it is not infrequently correctly diagnosed. It is usually primary and situated in the head of the organ. It is commonly scirrhous, but it may also be colloid. It may arise by contiguity from cancer of the stomach or intestines. It occurs in those past middle life. It is especially apt to invade adjacent parts and more distant ones by metastasis, especially the liver and lymph glands.

Symptoms.—These are not distinctive. The most valuable symptom is *jaundice*, which occurs when the head of the organ is involved. It is caused by obstruction of the common bile-duct. A *fixed tumor* may be felt in the pancreatic region, and if it be associated with jaundice, the pancreas may be justly suspected to be its seat. If we add to these symptoms *fatty* or *pancreatic stools*, the suspicion is fortified. There are symptoms of *indigestion* and a *dull pain* in the epigastrium, but these are not distinctive. *Emaciation* and *loss of strength* proceed irresistibly. As the former advances the *aortic pulse* is transmitted with great distinctness through the transverse colon and pancreas. There may be *ascites* and *diabetes mellitus*.

Diagnosis.—Cancer of the pancreas must be differentiated from cancer of the pylorus, of the transverse colon, of the glands in the hilus of the liver, and from aortic aneurysm. In case of *cancer of the pylorus* there should not be much difficulty, for the pyloric tumor is movable in a decided majority of cases, and the pancreatic is fixed; the pyloric cancer is rarely associated with jaundice, the pancreatic is almost always so; pyloric cancer produces dilatation of the stomach, pancreatic cancer does not.

Cancer of the transverse colon is rare. It is also more movable than pancreatic cancer, and sooner or later obstruction of the bowel results. *Cancer in the hepatic fissure* is difficult to distinguish, but it is higher up and more superficial. The tumor is also tender. It is accompanied by jaundice and by this symptom resembles cancer of the pancreas.

The pulsation communicated to the pancreas is very different from the expansitle dilatation of *aneurysm*. Fatty stools are of great assistance in diagnosis, but they are by no means always present.

Sarcoma is a possible tumor of the pancreas, but it is not distinguishable from cancer. Tuberculosis and syphiloma may occur and present similar difficulties. The Cammidge reaction is of value in differentiating obstruction of the pancreatic duct. Fatty stools are also of value both in chronic pancreatitis and in cancer of pancreas.

The **prognosis** of cancer is unfavorable, and the **treatment** only symptomatic.

CYSTS OF THE PANCREAS.

Definition.—These are retention cysts, due to closure of Wirsung's duct by concretions or cicatricial contraction. They may become very large, and may even occupy the entire abdominal cavity. They may be slow or rapid in development.

Symptoms.—In none of the 53 cases collected—35 by W. W. Johnston and 18 by N. Senn—was there fatty diarrhea, a condition regarded as symptomatic of suspended function of the pancreas. On the other hand, the *stools* may be *clay colored* and *putrescent*, probably because there is a simultaneous obstruction to the descent of bile. A resulting *tumor* presents itself usually in the left part of the epigastrium, between the costal cartilages and the median line. More rarely it is in the neighborhood of the navel. It is globular, resisting, and inelastic, changes its position slightly with the movements of the diaphragm, and possesses some lateral motion.

The differentiation of such a tumor, in the absence of more definite

symptoms, cannot be said to be easy, yet the diagnosis was made in seven out of Senn's 18 cases. Exploratory laparotomy should be done. The fluid is usually brown or chocolate colored, but sometimes it is transparent. It presents some of the characteristics of pancreatic fluid, emulsifying fats and converting starch into sugar.

Treatment.—The treatment is surgical.

PANCREATIC CALCULI.

Etiology.—Pancreatic calculi can only be regarded as a precipitation from an inspissated pancreatic juice determined by some unknown cause.

Morbid Anatomy.—The calculi, commonly about as large as a pea, are contained in the pancreatic duct and its branches. They are usually numerous. They may be smooth, round, faceted, or irregular and rough of surface. They are composed of carbonate and phosphate of lime.

Symptoms.—Pancreatic calculi are often unattended by symptoms, but deep-seated colicky pain may be present. The difficulty in distinguishing this from the pain of biliary colic is increased by the fact that jaundice may be associated with either. Theoretically, the pain of pancreatic colic should be more deep-seated, more central, and more to the left. Practically, this is not often found to be the case. If fatty diarrhea and diabetes are associated with the colic, pancreatic calculus may be inferred. Rarely stones are passed by the bowel, and if such stones are found to be made up of phosphate and carbonate of lime, they probably come from the pancreas.

Treatment is mainly palliative by morphin or other anodynes. Eichhorst has recommended hypodermic injections of pilocarpin to stimulate the pancreatic secretion. Surgery is indicated.

DISEASES OF THE PERITONEUM.

ASCITES.

SYNONYM.—*Hydroperitoneum.*

Definition.—Any freely movable collection of fluid in the abdominal cavity sufficiently copious to be recognizable by the physical signs present.

Etiology.—Ascites is a symptom of any one of a number of diseases causing venous engorgement of the vessels draining the peritoneum, but a symptom of such importance as to demand separate consideration. Its causes are *in situ* and remote. The most frequent local cause is obstruction to the portal circulation, commonly by some disease of the liver, especially hepatic cirrhosis. Any growth or inflammatory new formation in the gastrohepatic omentum or hepatic fissure exerting pressure on the portal vein may have the same effect. Abdominal tumors outside of the liver large enough to exert the requisite pressure may also produce ascites. Such are enlarged spleen and tumor of the ovary and even of the uterus. Chronic inflammation of the peritoneum is also a cause, whether tubercular, cancerous, or simple. More rarely cirrhosis and emphysema of the lungs and chronic pleurisy cause it. This is the result of dilatation of the right heart.

Remote causes include, first of all, valvular heart disease, the general obstruction due to which causes ascites as a part of a general anasarca, the peritoneal cavity being the last invaded. Rarely, it is the only dropsical symptom of heart disease, in which event there must be associated some intermediate obstruction of the liver. Bright's disease is also a cause of abdominal dropsy, in which disease, too, the peritoneum is, as a rule, is last invaded. More rarely it occurs as a consequence of intense cachectic states, such as the gravest forms of anemia.

Symptoms.—A rather large amount is required before all the physical signs to be described are developed. The abdominal cavity thus occupied is more or less distended, pendent when the patient is upright and widened when the patient is on his back, the flanks dropping down and outward. The fluid also flows from one side to the other when the patient turns on his side. If the distention is excessive, "Lineal albicentes" such as extend across the abdomen in pregnancy, make their appearance, and the umbilicus is obliterated or protuberant. The superficial veins—branches of the epigastric—are distended and distinctly visible due to pressure by the fluid on the vena cava, such pressure obstructing the return of blood from the lower extremities. Sometimes these superficial veins from below are seen to join those of the mammary from above. Such distention, however, is often contributed to by coincident portal obstruction (see p. 458). There may also be edema of the lower extremities. There is no caput medusæ about the navel unless the portal circulation is also obstructed.

As intimated in the definition, the physical examination affords the most reliable evidence. To palpation there is the succussion wave, which is elicited by placing the palm of one hand on the side of the abdomen and tapping with the fingers on the opposite side. A false succussion wave is sometimes produced by this procedure in persons with fat, flabby belly-walls, but error may be avoided by having an assistant place the edge of his hand vertically on the median line and pressing firmly downward while the tapping is done, as in this way the false wave, which travels around through the abdominal wall, is obliterated. It is always difficult, and sometimes impossible, to palpate solid organs when the abdomen is distended with fluid. Such palpation is, however, facilitated by a modification of the ordinary method—viz., first applying lightly only the ends of the fingers, then suddenly depressing them, and so displacing the fluid that the solid organ can be felt.

Percussion elicits absolute dullness over the fluid, while over the bowels, which are floated upward, a tympanitic note is produced, which changes with the position of the patient. If there is considerable effusion and the patient lies on his back, there is a small oval area of tympany in the middle of the abdomen. If a small amount of fluid is present, the flanks only are filled in this position, and there is a large superficial area of tympany in front, which will be substituted by dullness if he be placed in the knee-elbow position.

The statement that in ascites there is dullness in the flanks must be taken with some allowance, for it sometimes happens that a tympanitic note may be produced by percussion far back in the flank behind the mid-axillary line, because in this situation lie the ascending and the descending

colon, with the posterior aspect uncovered by peritoneum and therefore inaccessible to the fluid.

Differential Diagnosis.—The morbid condition which the physician is most frequently called upon to distinguish from ascites is probably the *ovarian cyst*. The ovarian cyst, especially when large, furnishes some points of resemblance, yet there are striking differences. It begins in one side and rises from the pelvis toward the center of the abdomen, which soon becomes the most prominent portion, while the dropsical effusion spreads out into both flanks. The ovarian cyst distends one side more than the other at first, and continues to do this even when large and fully developed. It produces no obliteration or projection of the naval, as does abdominal dropsy. Palpation also recognizes fluctuation in the ovarian cyst, but it is usually less distinct and more circumscribed, while in ascites the wave passes all the way across the abdomen. To percussion, the latter condition affords a central tympany and dullness in the flanks, while in ovarian cyst the flanks are resonant because the bowels are pushed into them. This is at least true of one flank, even if the other is completely occupied by a large tumor. If there is tympany in the upper abdomen, with an ovarian tumor, it is bounded below by a convex line, while in ascites its lower border is concave.

A change of position has less influence on the dullness in ovarian tumor than in ascites. Vaginal examination affords some information. In ascites the vaginal vault is obliterated, the uterus prolapsed, but freely movable, while in ovarian tumor the vagina is less encroached upon, the uterus being sometimes drawn up and less movable.

The characters of the contained fluid are, as a rule, widely different. The fluid of a simple ascites is usually transparent, has a low specific gravity, commonly below 1012, and contains a small quantity only of albumin and a few leukocytes. The ovarian fluid is usually dark and grumous in appearance, highly albuminous, with a specific gravity of 1020 or more, and reveals to microscopic examination numerous *granular fatty cells* (compound granule cells), cholesterin plates, and small, *pale granular cells*. These last are round or slightly oval, about the size of a white blood-corpuscle, and are by some regarded as pathognomonic of ovarian cyst contents, and therefore called "ovarian cells." They are found in pleuritic fluids, pus, and even ascitic fluids, but they are much less numerous in these. The cell is probably a degenerated endothelial cell from the peritoneum. The presence of these cells in large numbers is certainly a help to the identification of ovarian fluids.

In rare instances the fluid of ascites is milk-white. This occurs when from any cause there is leaking of chyle into the peritoneal cavity—*ascites chylosus* and the same condition due, not to chyle but to fat drops, is present in certain malignant cases. In the effusion associated with morbid growths, such as cancer and tuberculosis, the fluid is also sometimes white in color, from the presence of an unusual number of fattily degenerated cells from these sources or from the peritoneal endothelium.

The *overdistended bladder* has been more than once punctured by mistake for ascitic fluid, but this accident can never occur if the patient is directed to empty his bladder or the catheter is used before tapping.

Hydronephrosis has been confounded with ascites, and this is less excusable than the confounding of hydronephrosis and ovarian cyst. In advanced hydronephrosis the fluid may be almost identical with that of ascites, but its mode of development is from one side and exceedingly slow, while there are pain and tenderness in the region of the kidney. W. von Leube relates a case in which he mistook an enormously dilated stomach filled with fluid, for ascites, and points out how easily the mistake could have been avoided by the previous use of a stomach-tube.

A *cyst of the omentum* is a rare condition, but should be remembered as a possible one to be distinguished from ascites.

Chronic peritonitis is also attended by effusion, which is, however, more limited than in ascites, and the change in the area of dullness on change of position is less complete because of the peritoneal adhesions, which interfere with the ready movement of the fluid. In tubercular peritonitis, where there is less limitation by adhesions, there is also tenderness. The withdrawn fluid is more highly albuminous and of higher specific gravity than the ascitic fluid and is likely to be purulent.

Treatment.—The treatment of ascites is that of the primary disease. Paracentesis is often necessary to relieve the discomfort of the patient. In any case of ascites in which the origin is obscure a laparotomy should be performed instead of tapping. The fluid may accumulate with rapidity and the tapping require to be repeated quite frequently, but it is not true, as commonly supposed by the laity, that a first tapping necessitates a second *per se*. When frequent tapping is necessary, it is sometimes better to keep the orifice open and allow the fluid to drain away continuously, rigid antiseptic precautions being taken. Under these circumstances the patient sometimes improves rapidly, as he is relieved from the exhausting effect of the pressure and weight of the large amount of liquid and of the constant dread of repeated tappings.

ACUTE PERITONITIS.

Definition.—An acute inflammation of the peritoneal membrane.

Etiology.—1. *Of Primary Peritonitis.*—Primary peritonitis, or that form which originates independently of inflammation of adjacent structures, is spoken of as idiopathic in origin. It is a disease of such rarity that its existence may reasonably be questioned, and there are those who deny its occurrence *in toto*.

2. *Of Secondary Peritonitis.*—By this is meant an inflammation the result of invasion of the peritoneum from a primary focus of disease somewhere in the vicinity, or traumatic agencies, like blows or punctures involving the peritoneum. Formerly, operations involving the peritoneum were fruitful causes of peritonitis, but since aseptic surgery has become general, such operations are done with an immunity previously undreamed of. There are two chief foci whence such inflammation originates. One of these is the digestive tract; the other, the genito-urinary system, more particularly of women. Inflammation may also invade the peritoneum from the liver, gall-bladder, spleen, or perinephritic region, or from Pott's

disease or psoas abscess. Perforation of the appendix, stomach in ulcer or cancer, of the intestine in typhoid fever, and dysentery, are the commonest causes originating in the gastro-intestinal tract. The second focus is purulent inflammation of the Fallopian tubes and the genito-urinary tract. Endometritis and metritis may be the starting-point of such inflammation, which may extend up the Fallopian tube, or there may be parametritis with suppuration, the abscess arising from which may rupture into the peritoneal cavity. All of the different forms of secondary peritonitis are infectious, and caused either by organisms responsible for the primary disease or by such as are set free with the gastric or intestinal contents by perforation. The organisms found under these circumstances are the *streptococcus pyogenes*, the *staphylococcus pyogenes aureus* or *albus*, and the *bacterium coli commune*, the latter especially after perforation of the appendix, also the *tubercule bacillus*. The *ameba coli* has been found in the peritoneal fluid in amebic dysentery. Peritonitis may also occur from infection from more distant foci of suppuration, when it is also called pyemic peritonitis. Tubercular peritonitis is rather common, and when of the miliary type, the symptoms are very acute.

Finally, peritonitis not infrequently becomes a complication of pleurisy, also articular rheumatism, and nephritis by a process not thoroughly determined. The first is probably the result of extension by continuity, since the two cavities communicate by the lymph-vessels of the diaphragm.

Morbid Anatomy.—This varies somewhat with the extent of the peritonitis and the duration of the attack. First, there may be a "general" or "diffuse" peritonitis, or it may be "circumscribed." In general peritonitis the peritoneal surface of the intestinal coils is hyperemic and covered more or less continuously with flakes of yellow lymph made up of fibrin and leukocytes. This is especially abundant in the sulci between the coils, while it also covers the convexity. In an earlier stage, before the exudate appears, the surface of the peritoneum is dull and rough, owing to a desquamation of the epithelium. In the flanks is found a variable amount of fluid, which may be serous, sero-fibrinous, or purulent, which, increasing, produces an appreciable ascites. In prolonged cases organization and vascularization from the capillaries of the peritoneum take place, the solid contingent being formed from the epithelium or wandering cells, resulting in adhesions between the coils of intestine and adjacent organs. These are at first soft and easily ruptured, but later become firm bands. These latter are, however, more common in the circumscribed form.

In circumscribed peritonitis limited areas of lymph formation occur and adhesions are more pronounced. Copious fibrinoserous exudate is less frequent, though sometimes quite large circumscribed collections of pus occur, laced off from the remainder of the peritoneal cavity by organized tissue. Such abscesses sometimes rupture into the general peritoneal cavity, producing general inflammation, collapse, and death.

Symptoms.—1. *Of an Acute Diffuse Peritonitis.*—The most decided symptom is *pain*, usually of extreme severity, which is commensurate in extent with that of the inflammation. There is also extreme *tenderness*, which is similarly limited. So great is this that any tension on the abdominal walls excites pain; hence the legs are drawn up to relieve this, and we

have the well-known position almost characteristic of diffuse peritonitis—dorsal decubitus, with the thighs flexed on the abdomen. Any motion such as straining, even the act of breathing and the emptying of the bladder, increases pain. From the nature of the causes this pain is usually sudden in occurrence, succeeding, as it does, on perforation, on abscess rupture, and the like. Sometimes, indeed, it is the first intimation of any illness whatever. *Abdominal distention* is a third characteristic symptom of peritonitis, ascribed to a paralysis of the muscular coat of the bowel, and continues throughout the attack. Rarely, however, the abdomen is flat, hard, and board-like. As rarely, too, pain is altogether absent.

Among the symptoms which may usher in the attack is *vomiting*. It is regarded as reflex in origin, excited by the inflammation of the peritoneum. The effort is sometimes ineffectual, and sometimes a perforation of the stomach permits the more ready discharge of its contents into the abdominal cavity. The vomitus consists of what happens to be in the stomach at the time, or of mucus and, if the symptom is prolonged, of green, bilious matter. The primary vomiting is followed by abatement or exacerbation.

The symptoms which are associated with these or succeed upon them vary with the nature of the cause and extent of the disease. In fulminating cases due to perforation of the bowel, as in typhoid and appendicitis, they are the symptoms of *collapse*—viz., *extreme weakness, cold, clammy skin, frequent, small,* and *feeble pulse*. The pulse exceeds 120 and often reaches 160 and even more. The breathing-rate is from 30 to 40. The temperature is slightly raised, remains about normal, or may be subnormal. Rarely, it is high—104° to 105° F. (40° to 40.6° C.)—though the skin may feel cool and clammy. The expression is characteristic—Hippocratic. The eyes are sunken, the cheeks and temples are collapsed, and the nose is pinched. The urine is scanty and contains indican.

If the patient survive, the *physical signs* of effusion make their appearance. There is dullness on percussion, first in the flanks, whence it ascends as the fluid increases. If sufficiently abundant, the dullness becomes general and fluctuation may be recognized. Palpation and percussion both occasion pain. A change of position from the back to the side causes a change in the position of the fluid, and corresponding alterations in the physical signs. In severe cases the diaphragm is raised, the apex of the heart dislocated, and the liver dullness may be obliterated in the mammillary line by combined effusion and extreme tympany. Similar obliteration may happen to the splenic dullness. Both may be restored by turning the patient on his side. Such obliteration is, however, far more characteristic in what is known as *pneumo-peritonitis*, a form of peritonitis caused by perforation from an air-containing organ into the peritoneal cavity, and of intense severity, excited by the pathogenic bacteria thus admitted. Acute pain, rapidly developing collapse, scarcely appreciable pulse, icy coldness of the skin, and great distention of the abdomen are the symptoms. The air, of course, occupies the highest part of the abdominal cavity, covering the liver and spleen, causing the obliteration referred to. The distinctive point in the diagnosis between pneumo-peritonitis and the extreme degrees of the ordinary form is the fact that in the former hepatic dullness is absent even in the midaxillary line when the patient is on his left side, whereas, in simple

peritonitis, hepatic dullness may be elicited when the patient is in this position, though it may not be if he is on his back.

Throughout all, the intellect is clear, and while there is often a total lack of realization of the inevitable and usually dreaded end, it is as often thoroughly appreciated by the patient and is viewed with a calmness which increases the awe which always attaches to the presence of the shadow of death. Rarely, in the course of his experience, is the physician called upon to witness a more painful scene. Toward the very end, however, a somnolence commonly supervenes which obscures the expiring moment, or a slight delirium the visions of which may be interpreted by surrounding friends as the first glimpses into another world.

The course of such a case is steadily downward, reaching its end in from two to six days.

2. *Of Acute Circumscribed Peritonitis.*—The symptoms include those of the general form in a very much milder degree. The pain is less severe and more circumscribed, the tenderness proportionate, while neither is sharply defined. Vomiting may also usher in the attack, and may be similarly modified. There may likewise be the signs of collapse, and the patient is often very weak. There is, however, more decided and constant fever though remittent, as in septic fever generally, and the cases run a longer course, ending not rarely in recovery, but more frequently in death from exhaustion.

As already mentioned, circumscribed abscesses are more frequently recognized by fluctuation, and may even point toward the surface, though they are as liable to rupture into the general peritoneal cavity, producing there the symptoms and more usual fatal termination of general peritonitis. This serious termination, at the present day, is often prevented by the timely interference of the surgeon. As varieties of such abscess may be mentioned the perinephric abscess, the pelvic abscess, the subdiaphragmatic abscess, arising from perforation of the stomach or colon or disease of the liver or spleen, and the periappendicial abscess. The results of circumscribed peritonitis in children are sometimes seen in the shape of a painful, fluctuating tumor in the groin. Circumscribed peritonitis is also more or less associated with the symptoms of the disease which causes it.

Diagnosis.—That of general peritonitis is seldom difficult, especially in the fulminating variety. Some days may, however, elapse before the question is settled, for sometimes the symptoms are closely simulated by those of other conditions. Particularly is this the case with extreme tympany and tenderness which are sometimes associated with *typhoid fever*, especially when there is deep-seated ulceration. It not rarely happens that on these symptoms is based the diagnosis of a peritonitis, which is not found at necropsy. *Entero-colitis* may give rise to similar symptoms. On the other hand, it has happened that grave and fatal peritonitis has eluded detection, having been found for the first time at autopsy. Obstruction of the bowel has been spoken of in a former chapter.

Acute hemorrhagic peritonitis should be mentioned as a variety, the symptoms of which sometimes are the same as those of the ordinary form.

Circumscribed peritonitis is more frequently difficult of detection, and its diagnosis often requires a knowledge of the presence of the causative

disease to suggest it. Fluctuation is only available in diagnosis when there is superficial abscess.

Prognosis.—This, in general peritonitis, is almost invariably fatal, only the mildest cases offering the possibility of recovery under medical treatment. Modern surgery has many times saved life even in peritonitis which succeeds perforation in typhoid fever, gastric ulcer, and perforated gall-bladder. The duration of untreated cases is from two to six days.

Localized peritonitis is a more promising malady. A few cases get well by spontaneous discharge of resulting abscesses, more with the assistance of the surgeon, and some neglected cases doubtless perish when timely aid from this source would have saved life.

Treatment.—The treatment of general peritonitis succeeding perforation consists in immediate surgical interference, repair of the ruptured organ and drainage of the cavity. If this for any reason fails an operation cannot be done or will not be accepted, or if a peritonitis is present or develops with an operation the best treatment is as follows: Put the patient upright in bed, Fowler's position, give absolutely nothing by the mouth, introduce by the drop method into the rectum a normal saline solution. Control pain with morphine. Hot or cold applications to the abdomen. When doubt as to diagnosis exists—as to whether there is true peritonitis or painful distention of the bowel—turpentine may be administered with full doses of strychnin, say 1/30 to 1/20 grain (0.002 to 0.003 gm.), while turpentine may be applied locally. Iced turpentine stupes are often exceptionally grateful. Turpentine enemas under these circumstances are of doubtful utility, in fact, may do more harm than good, and should be discouraged.

Special symptoms, such as nausea, faintness, and exhaustion, require the treatment usually appropriate to control them. For failing strength, stimulants, local heat, hypodermic injections of ether, digitalis, brandy, and strychnin are available, as the case improves small quantities of peptonized milk, albumin water or beef juice may be given.

The treatment of circumscribed peritonitis permits the use of local measures not admissible in the general form. The ice bag is sometimes of signal service in relieving symptoms and may even effect a cure if the cause is removed. Blisters and leeches should not be used as they interfere with the expected field of operation. The surgeon and the gynecologist should be early summoned, as it is most frequently through their assistance that a cure is accomplished.

CHRONIC PERITONITIS.

Etiology.—By far the largest majority of cases of chronic peritonitis are tubercular in origin. Some cases are caused by cancer and other morbid growths in the abdomen, while there are also others of simpler origin. Thus originating, we have both a *circumscribed* adhesive peritonitis and a *diffuse* form of the same disease. See also Section on Tuberculosis of the Peritoneum, p. 308.

Chronic Adhesive Peritonitis.

Synonyms.—*Chronic Adhesive Sclerosive Peritonitis; Chronic Circumscribed Peritonitis.*

This occurs between adjacent organs, such as the spleen and diaphragm, liver and diaphragm, stomach and liver, and organs in similar relation, as the result of chronic disease in one or the other. The spaces about the gall-bladder, the flexures of the bowels, posterior peritoneum and the omentum may be sites. These adhesive connections are not always close, but sometimes consist of bands of considerable length, such as have already been referred to as occasional causes of obstruction of the bowel.

Morbid Anatomy.—The primary result is a thickening of the peritoneum with subsequent contraction and adhesion. The condition may begin as a subperitoneal fibroid infiltration.

Symptoms.—Constipation or symptoms of obstruction of the bowel are often the first evidence of the existence of such adhesive bands. Other symptoms are a sense of *restriction in the motion* of organs involved, with pain when such motion occurs; also *colicky pains*, and pains resulting from traction exerted in peristalsis. Other vague symptoms occur which go to make the patient uncomfortable, but are not distinctive. Should a peritoneal friction, however, be felt, more conclusive evidence is thus furnished. Should suppuration attend chronic inflammation, more distinctive symptoms also arise. In addition to the pain and tenderness a *hectic fever* may be present, which may guide to a correct conclusion, or eventual rupture into one of the hollow abdominal organs may occur.

Diffuse Chronic Peritonitis.

This may succeed upon acute diffuse inflammation of mild degree, which is followed by an abatement in all the symptoms. It may occur in connection with chronic cardiac disease including pericarditis or hepatic disease where there has been long-continued venous stasis; or it may succeed the punctures of numerous tappings and, most rarely, chronic intestinal disease.

Morbid Anatomy.—The peritoneum is thickened. The intestinal coils may be cemented to one another and to neighboring organs. The liver and spleen are sometimes covered by thick, tough, gristly capsules. The omentum and mesentery may be thickened and shrunken. There may be thickened nodules, not tubercular. There is in these cases rarely considerable effusion. A hemorrhagic form, suggesting hemorrhagic pachymeningitis, was described by Virchow. It is more commonly situated in the pelvis and characterized by bloody effusion.

Symptoms.—These exhibit for the most part a diminished degree of those characteristics of acute peritonitis, to which may be added *tumor-like swellings and thickenings* and swelling difficult to interpret. Other vague symptoms are engendered by them as the result of contraction and pressure, including *pain, edema, albuminuria, irregularity of the bowel action*, and sometimes *feverishness*. There is little that is characteristic unless it be the occasional presence of recognizable effusion. The very slow forms

attended with extensive effusion are not separable from ascites, the result of hepatic disease, although there are differences in the effusion. In peritonitis the effusion is more turbid, contains abundant albumin, and has a specific gravity rather higher than the fluid of an ascites: 1018 as compared with 1012.

A chronic peritonitis not unusual in children from two to ten years old is described by Strümpell and others. It is associated with decided ascites, debility, and other symptoms of ill health more or less marked, while recovery is the usual termination. Such a cause for the ascites should not be assigned without careful search for others, especially disease of the liver.

Treatment.—The treatment must be determined by circumstances. It is chiefly palliative, unless operative interference promises more.

MULTIPLE SEROSITIS.

SYNONYMS.—*Multiple hyaloserositis; Zuckergussleber (Iced liver); Hyperplastic perihepatitis; Pericarditic pseudocirrhosis of the liver; Indurative mediastinopericarditis; Polyorrhomenitis.*

Definition.—An inflammatory affection invading extensive areas of serous membrane, beginning in the pericardium, the pleura, or peritoneum and further characterized by ascites and more rarely by edema of the extremities.

Etiology.—No special pathogenic organism has been found associated with the disease, but as suggested by Nicholls,[1] some germ of relatively low virulence with a penchant for serous membrane is likely to be the cause of the proliferative inflammation. Such a bacillus may be the pneumococcus, the bacillus of typhoid fever, the bacillus coli and the bacillus tuberculosis.

Morbid Anatomy.—The morbid anatomy of the process varies with the seat of the membrane first invaded and the organs covered or embraced by it. Thus where perihepatitis is primary the primary perihepatitis of Nicholls, ascites appears early and is prone to recur after tapping. The liver itself is large, smooth, subsequently contracted and the Glisson's capsule may take on the appearance of "icing" which has given rise to one of the names of the affection. After this, the pleura is usually invaded, and finally the pericardium. In the cases where pericarditis (primary pericarditis of Nicholls) is primary, an adhesive pericarditis sets in which may be obliterative, and even invade the entire mediastinum. Occasionally only there is a little pericardial effusion. From the pericardium the inflammation extends to the pleura and finally the peritoneum and capsule of the liver. Pleuritic adhesions may be marked as the result of the pleurisy. The spleen is sometimes enclosed in the "icing" capsule, so striking in the case of the liver.

Symptoms.—These also vary with the seat of beginning. In the pericardial variety, the symptoms and physical signs, more or less pronounced, of pericarditis, usher in the disease. It is in this form that edema of the

[1] Nicholls: Stu lies from the Royal Victoria Hospital, vol. i., No. 3, April, 1902.

extremities and face appears early as the result of cardiac weakness, but may disappear later and be replaced by ascites. Previous to the latter, however, the signs of pleurisy on one or both sides have probably appeared. In the perihepatitic form, enlarged liver, often palpable, with subsequent contraction and ascites, is first noted, followed by symptoms of pleurisy with or without effusion.

Fullness of the abdomen, oppression and dragging weight are also symptoms depending upon the abdominal contingent of the disease. Pulsation of the liver may be present and distinguish the associated disease of this organ from cirrhosis of the liver.

Diagnosis.—The chief cause of errors in diagnosis and of late diagnosis is the latency of the affection and the slowness of its development. But this very slowness in association with the signs and symptoms named should be a valuable aid to diagnosis. Emphasis should be laid on the fact that this disease is something different from cirrhosis of the liver which is also associated with ascites but not with pericarditis and pleurisy.

Prognosis.—Slowness of development and gradually failing physical strength, with ultimate death from exhaustion are characteristic, but occasional interruptions in the progress of the disease, during which the patient may even resume work for a time, should be added as a feature.

Treatment.—There is no treatment except that of the symptoms, including especially effort to build up the strength of the patient.[1]

CANCER OF THE PERITONEUM.

Primary cancer of the peritoneum is an event of extreme rarity. Its occurrence as a true epithelial cancer must, however, be admitted. Colloid cancer also occurs as a diffuse and extensive growth, relatively firm, and without fluctuation. More frequently peritoneal cancer is secondary to cancer of the stomach, bowel, pancreas, uterus, or other organ; most frequently, perhaps, as an extension by contiguity, though also by metastasis. It occurs in the shape of *small* or *large nodules* scattered over the peritoneum. The former constitutes what is known as *miliary* carcinoma. The larger nodules are found in the omentum, in Douglas' culdesac, around the navel and elsewhere, while the retroperitoneal glands may be simultaneously involved.

Symptoms.—These are those of chronic peritonitis, including effusion, with the added cachexia, and a diagnosis must be based on these, the antecedent history, and the possible presence of cancer elsewhere. The investigation must include the uterus and the rectum. The physical resemblance of the miliary form to tuberculosis is very marked, and in primary carcinoma

[1] This disease is exhaustively discussed in a valuable paper "On Multiple Serositis," read before the College of Physicians of Philadelphia by A. O. J. Kelly, in March, 1902, and published in the Transactions of the College for that year; also in the "American Journal of Medical Sciences," Jan., 1903, p. 116. Other papers on the same subject are by:

Curschmann, "Zur differential Diagnostik der mit Ascites verbundenen Erkrankungen der Leber und der Pfortadersystem." "Deutsche medicinische Wochenschrift," 1884, vol. x., p. 564.

Harris, "Indurative Mediastino-pericarditis." "Medical Chronicle," 1895, vol. ii., pp. 1, 87, 178, 250.

Nicholls, "Studies from the Royal Victoria Hospital," vol. i., No. 3, April, 1902.

Pick, "Ueber chronischer, unter dem Bilde der Lebercirrhose verlaufende Pericarditis" (pericarditische Pseudolebercirrhose). "Zeitschrift für klinische Medicin," 1896, vol. xxix., p. 385.

Siegert, "Ueber die Zuckergussleber (Curschmann) und die pericarditische Pseudolebercirrhose" (Pick), "Virchow's Archiv. für path. Anatomie," 1898, vol. cliii., p. 251.

Howard Fussell and Henry D. Jump, A Case of Probable Multiple Serositis.

"Transactions of the College of Physicians of Philadelphia," vol. xxv., p. 55, 1903.

Kelly's paper also contains a large number of references to which the interested reader is referred.

the distinction is difficult. Palpation may recognize friction in both. In both the effusion may be bloody, but is more apt to be so in cancer than in tuberculosis. The test injection of tuberculin should be availed of. The cancerous patient is past middle life, the tubercular younger, tubercular peritonitis being especially frequent in children.

HYDATID DISEASE OF THE PERITONEUM.

The possible presence of *echinococci* in the peritoneum is to be remembered. The local symptoms may resemble those of cancer very closely. The presence of hydatid tumors elsewhere, as in the liver, of course suggests the true nature of the simulating disease.

SECTION III.

DISEASES OF THE RESPIRATORY SYSTEM.

ACUTE RHINITIS.

SYNONYM.—*Coryza.*

Definition.—Simple acute inflammation of the nasal passages.

Etiology.—Simple acute rhinitis in many instances is unquestionably infectious. In many victims there is a tendency to recurring attacks. The exudative forms, including simple fibrinous rhinitis and nasal diphtheria, are, of course, of a specific infectious nature.

Symptoms.—The well-known uncomfortable full feeling which all have experienced under the name of "cold in the head," is a frequent event. There may be previous sneezing. The fullness is due to swelling of the mucous membrane, the result of inflammation, and is sooner or later followed by a discharge which, at first watery, may or may not become mucopurulent. With it comes relief of the most uncomfortable symptom, the nasal obstruction. This is most serious in nursing children, in whom it renders sucking often very difficult. There may be slight fever, but the constitutional disturbance is seldom decided, and the elevation of temperature is correspondingly trifling, rarely exceeding a degree. There is sometimes dullness in hearing and perverted sense of taste and smell.

Treatment.—Prophylaxis is important. Unquestionably most, if not all, common colds including a "cold in the head" are infectious. Living in shut-in rooms, frequently unventilated places where others congregate are further sources of the condition. Fresh air and avoidance of close rooms is important. Sleeping in well-ventilated rooms or even protected places in the open will do more to cure a cold in the head than any other remedy. When this condition is associated with inflammation of the adjacent mucous membrane of the respiratory passages and of the throat, its treatment is that of the concurrent affection. A promptly acting saline aperient, such as citrate of magnesium of Epsom salts, may with advantage precede other treatment. The discomfort is partially due to the dryness, and this is overcome by the application of any simple ointment, as liqiud petrolatum, cold cream or vaselin, applied by means of a brush or the end of the finger. The same result is better accomplished by the oil spray, for which liquid paraffin may be used. Such applications to the adjacent parts are also useful when the discharge is irritating. Dobell's solution may also be sprayed into the nose, and when dry discharges accumulate, they should be washed out by gentle injections of tepid normal salt solution. Dobell's solution is composed of sodium borate 1 dram (4 gm.), sodium bicarbonate 1 dram (4 gm.), glycerite of carbolic acid (U. S. P.) 2 drams (8 gm.), and water 1 pint (0.5 liter).

The liquor antisepticus of U. S. P. may be used. Hexamethylenamine in doses of from 3 to 5 grains (0. 18–0.3) may be used every 3 hours. A spray of adrenalin chloride 1–4000 may be used with benefit. *Cocaine should not be used* for fear of causing the drug habit.

CHRONIC NASAL CATARRH.

SYNONYMS.—*Chronic Rhinitis; Ozena.*

Definition.—Chronic inflammation of the nasal mucous membrane, associated with increased secretion and loss of the sense of smell.

Etiology.—Chronic catarrh of the nasal passages may be the result of acute inflammation frequently recurring, but it may arise from speical causes. A few cases of rhinitis are the result of tuberculosis of the mucous membrane of the nose. A frequent cause is syphilis. In consequence of the offensive odor frequently associated with one form of chronic nasal catarrh—the atrophic—it has been termed ozena.

Morbid Anatomy.—Two broad divisions of chronic nasal catarrh are made from the anatomical standpoint—the hypertrophic and the atrophic. In the *hypertrophic* there is a thickening of the mucous membrane, while in the *atrophic*, a thinning or atrophy is present. In the hypertrophic catarrh, the membrane is red, swollen, and spongy. The cavernous tissue over the turbinated bones shares in the process, and the nasal cavities may be encroached upon from all sides. The protrusion becomes more marked as the disease progresses, and to it is added a greater or less hypersecretion of mucus.

In the *atrophic* or fetid form, the nasal mucous membrane is thinned, the cavities are enlarged, and within them are found the thick, yellowish-green crusts which, in decomposing, give rise to the characteristic offensive odor of this form of rhinitis. The atrophic process involves all the tissues, from the epithelium down to and including the underlying bone. The accessory sinuses connected with the nose—the frontal, ethmoidal, and maxillary—may all become implicated in this disease by extension from the nasal chambers, and may become the seats of chronic purulent inflammation.

Symptoms.—The two principal forms of nasal catarrh have certain symptoms in common. In both there is more or less *marked obstruction to nasal respiration.* In the hypertrophic form, however, this is due to actual narrowing of the nasal chambers by the overgrowth of the contained structures, while in the atrophic form it is due to the choking of the passages by the large masses of inspissated mucus and mucopus. There is generally some slight impairment *of the sense of smell* in the hypertrophic form, while in the atrophic it is more often completely abolished. Both forms are usually accompanied by *disturbances of secretion* in the nasopharynx, and these lead to those noisy efforts at clearing the throat termed "hawking." The ozena, or fetid odor, is symptomatic only of the atrophic variety. No odor is produced by simple hypertrophic catarrh.

Hypertrophic nasal catarrh is apparently much more common in the United States of America than in Europe—indeed, the observations of the specialists go to show that almost every person is more or less the subject

of these hypertrophic processes, of which, in many instances, he is quite ignorant until examination has shown their presence.

Treatment.—The proper local treatment of chronic nasal catarrh, which is by far the most important, demands such special measures as in the main can only be carried out by accomplished specialists. This treatment, therefore, so far as can be taken up in this book, can only be palliative, or, if curative, limited to the early stage of the disease. In all forms of chronic catarrh the most important measures to be employed by the physician, as distinguished from the specialist, are those which have for their purpose the most thorough cleanliness of the affected regions. The simplest means for accomplishing this purpose is sniffing from the palm of the hand simple salt solution of the strength of a teaspoonful of sodium chlorid to a pint (0.5 liter) of water, or some one of the substitutes named below.

As ordinarily used, it is, however, much less efficient. We may use with it varying proportions of Dobell's solution or liquor antisepticus and water, say from 1 to 4 up to equal parts; also an alkaline solution composed of liquor alkalinis compositus 1 part, water 4 parts, and 1/2 dram (2 gm.) each of sodium bicarbonate and sodium borate to the pint of this mixture. When large quantities are required to wash out the nasal cavities, the postnasal syringe may be used instead of the nasal douche. Liquor alkalinis antisepticus is disinfectant and deoderizing. A plug of borated or salicylated cotton may be used for a like purpose.

General treatment, although not so important as the local, is still of great value, and the health of the patient should be carefully looked after. In view of the fact that atrophic rhinitis is very apt to occur in tuberculous persons, cod-liver oil is a tonic always indicated, and should be given for a long time, intermitting occasionally to avoid derangement of the stomach. It should be associated with iron, and even with arsenic. Other tonics should be given as indicated, and the best food should be prescribed, including an abundance of meat, eggs, and cream. Wholesome ventilation should be secured for the indoor life, while as much time should be spent in the open air as possible. The air indoors is especially apt to be contaminated by the breathing of the patient with atrophic rhinitis, and on this account good ventilation is imperative. If syphilis is present, it should receive appropriate treatment at once.

HAY FEVER.

Synonyms.—*Catarrhus æstivus; Hay Asthma; Autumnal Catarrh; Rose Cold; Pollen Catarrh; Vasomotor Coryza.*

Definition.—A catarrhal affection of the upper air-passages, associated with asthmatic dyspnea, occurring in the spring, late summer or autumn, ascribed to the pollen of plants and grasses as exciting causes.

Etiology.—In a large proportion of cases, hay fever has as its fundamental condition an anatomical change in the nasal passages, such as hypertrophy of the mucous membrane, a polypoid growth, a deflection of the septum, or a lowered position of the inferior turbinated bones so that they rest upon the floor of the nose. These conditions are not always demon-

strable, but they, or some allied source of reflex irritation, produce an irritability. This may be increased by a neurotic constitution, though the latter may not manifest itself until after the attacks have become habitual, so that at times, at least, it is more likely that the neurosis is a result, rather than a cause of the disease. A third necessary etiological factor is an irritant. This irritant, whatever it is, originates usually in the spring or the late summer. In the spring, it has been regarded as due to a pollen coexistent with the fragrance of roses; hence the term, "rose cold" or "June cold." In August and September the pollen of flowering plants is commonly regarded as the exciting cause, and in certain early instances this seems to have been conclusively demonstrated, as by Blakely in his own case. These suppositions have been shown by the studies of Dunbar to be facts. He has *proven* that it is caused by the pollen of certain grasses and plants. Thus far there have been isolated about 25 grasses and seven plants whose pollen is active. The pollen of rye is one of these, and in this country the pollen of rag weed and golden rod are conspicuous in the fall of the year. Almost infinitestinal quantities 0.000025 of a milligram of an albuminous substance isolated from pollen is capable of producing the required conjunctival irritation in susceptible persons. On the other hand, the pollen of roses, linden flowers and other plants reputed to cause hay fever are without effect. This form of hay fever is unquestionably the result of sensitization a true example of anaphylaxis, cases exactly resembling the hay fever caused by the pollen of grasses occur from the inhalation of effluvia from horses, guinea pigs and rabbits. In the case of a famous medical man every time he enters the rabbit house of the laboratory he is seized with coryza, lachrymation and more or less asthma. These attacks are certainly due to anaphylaxis, as vaccination with the serum of the rabbit is followed by a prompt skin reaction of Von Pirquet.

Changes of temperature may excite attacks and in warm countries, as in the Southern United States, it may prevail the year round. Emotional causes, imaginary odors, and the like may cause it. Heredity is an important factor in its causation, successive generations being attacked with astonishing regularity.

Localities variously favor it. Generally, cities furnish more cases than the country, and low countries more than elevated ones, yet certain seaside places are absolute cures for many cases. Such a place is Long Beach, N. J., where is located Beach Haven, a seaside resort, 50 miles from Philadelphia, which has long been a resort for the victims of hay fever. The disease is more common in the United States than in Europe, and in the United States than elsewhere in America. It is more common in men than in women, there being three cases of the former to every two of the latter.

Morbid Anatomy.—There is no morbid anatomy other than that referred to in the remarks on the etiology of the disease.

Symptoms.—The onset of hay fever may be quite sudden, coming on with remarkable regularity often on the same day of the month each year. At other times it is more gradual in its onset. It frequently begins with *sneezing*, and indeed may consist entirely of inveterate sneezing. At other times there are *asthmatic attacks* of great severity, closely resembling those

of bronchial asthma, constituting the "asthmatic type" of the disease. Again, there may be *obstinate cough*, with or without expectoration; or there may be an alternation of the two symptoms, but generally there is more or less persistent *shortness of breath*. There is also often great *depression of spirits*, and victims have even been impelled to suicide. The *eyes* are suffused with redness, and there may be conjunctivitis.

Diagnosis.—The diagnosis furnishes no difficulty. The reason of the year and the periodical recurrence of the cough coryza and asthma combine to make the recognition easy.

Prognosis.—Patients seldom die of hay asthma, yet we have known cases which seemed to be very ill when they reached the haven which afforded them relief.

Treatment.—The complete *cure* of an individual attack is seldom accomplished except by removal from the district in which the patient resides. The White Mountains and the Adirondack Mountains are favorite resorts in the eastern part of the United States, and Bethlehem, N. H., is the Mecca of American hay-fever victims, though other places in the same neighborhood are equally exempt. The Catskills and Alleghanies are less celebrated. Certain seaside resorts have also a deserved reputation; Beach Haven, N. J., has already been mentioned; Fire Island, on the Atlantic Coast outside of New York Bay; the Isles of Shoals, Nantucket, and Mount Desert, on the New England coast, are others. Sometimes a sea voyage will abort a threatened attack, and some persons are quite exempt while at sea.

A few cases have been totally cured by operations on the nasal cavities such as correcting deviations, and the removal of hypertrophic processes by the knife or actual cautery, but the confident expectation which followed some of the earlier of these operations has not been realized.

Home treatment, at best, has been uncertain and but partially successful, and, as is always the case with a malady so difficult to cure, the number of remedies is legion.

Dunbar's serum is based upon proper series of observation. Its use as claimed by Dunbar is certain to result in cure, but this is not the rule. Both a liquid serum and a dry powder are prepared and are applied to the nasal or conjunctival mucous membrane two or three times a day, while the prophylactic treatment is far more satisfactory than the curative. In using this treatment doors and windows should be kept closed at night during the hay-fever season.

Irrigation of the nasal passages by the nasal douche or spray with simple salt solution or weak solutions of quinin, 1 grain (0.065 gm.) to the ounce (15 c.c.), has been used, with varying results. Helmholtz was the first to suggest quinin solution, and thought it efficient. The oil spray is another efficient measure of this kind. A strong solution of cocain—four to ten per cent.—applied with a brush affords temporary relief, but it should not be used because of danger of forming the cocain habit. Subnitrate of bismuth and boric acid, 1/2 dram (2 gm.) to the ounce (15 gm.) of vaselin of simple ointment, will sometimes allay the itching. Solution of suprarenal extract will be considered later. Boric acid, 10 grains (0.65 gm.) to the ounce (30 c.c.) of water, may be used for the conjunctivitis.

Sodium iodide is given in small doses, frequently repeated, as 1 grain (0.2 gm.) every two hours. Fowler's solution has some reputation. Morphin is undoubtedly a useful palliative, but its employment is extremely dangerous. From 1/8 to 1/2 grain (0.008 to 0.03 gm.) may be required, and the smaller doses should be tried first. Chloral is also of undoubted use as a palliative, and is much safer than morphin, with which it may be combined. It renders smaller doses of the anodyne more efficient, and may be given in combination with 1/24 to 1/12 grain (0.0027 to 0.0055 gm.) of morphin at short intervals. Suprarenal extract has acquired considerable reputation in the treatment of hay fever. S. Solis Cohen and Beamon Douglass were among the first to report favorably on its effect. It acts by reducing turgescence of the turbinated tissue. It is used externally and internally. For local applications solution of adrenalin chloride, in the shape of a spray is valuable the solution may be used every two hours until the symptoms have subsided, repeating the treatment on the appearance of obstruction, coryza, and sneezing. Adrenalin solution, strength 1 to 1000, is of late the usual proportion, applied with a brush; or a mixture of the same solution of adrenalin with equal parts of four per cent. solution of cocain may be used. Internally suprarenal extract may be given in the tablet form or in a capsule. Five to 10 grains are administered, day and night, every two hours until an examination of the nasal membrane shows that the vasomotor paralysis is under control, or until giddiness or palpitation is noticed. After this improvement the same dose may be given every three hours, then every six hours, and finally, only twice daily, which is continued during the hay-fever season. If the dose is too rapidly diminished and the symptoms reappear, one tablet should again be given every two hours until the symptoms are controlled. Adrenalin solution is also administered internally in doses of 10 minims (0.3 c.c., of the 1 to 1000 solution. Mild cases may be comparatively comfortable during the season when the extract is used in this way. If the pure dried extract is used 1 to 3 grains may be given in a capsule.

ACUTE CATARRHAL LARYNGITIS.

Etiology.—The most common cause of catarrhal laryngitis is some infection, but predisposition plays a most important part. Such predisposition may be the result of previous attacks of laryngitis, or it may be brought about by constant use of the organ in speaking and singing; whence it is common with persons thus engaged. In these occupations the larynx is hyperemic from overuse, and this hyperemia is ever ready to be fanned into active inflammation. Exposure to cold is constantly at hand to furnish the exciting cause. Laryngitis is also brought about by the inhalation of irritating vapors or gases, while intemperate smoking and the use of strong alcoholic drinks are also causes of the hyperemia so readily converted into an inflammation. Catarrhal laryngitis is frequently associated with catarrh of the adjacent parts, as of the nose and pharynx, trachea, and bronchi.

Morbid Anatomy.—It is characteristic of the mucous membrane of the larynx, and, indeed, of the trachea and larger bronchi below it, that it loses,

postmortem, the anatomical characters of the inflammatory process as they appear during life. It is only by the image in the laryngeal mirror, therefore, that we can obtain an idea of these appearances as they present themselves during active inflammation. The picture thus obtained by the laryngeal mirror is one of intense redness, with swelling. These changes involve the true and false vocal cords and the trachea below, as well as the epiglottis above. The latter appears in strong contrast to the yellowish-pink of health. Even greater is the contrast between the appearance of the vocal cords and the pearly white of health. If secretion has set in, streaks of mucus may be seen in places. Escessive swelling of these parts may occur in edema of the glottis, but it is not frequent in simple acute laryngitis and will be described separately.

Symptoms.—The most constant symptoms of acute laryngitis are *hoarseness* and *cough*, which vary with the degree of the swelling and hyperemia, and which also give rise to a sense as of something present in the larynx and a constant desire to clear the throat. In high degrees of inflammation there may be *aphonia*. To these there is sometimes added *pain during deglutition;* with higher degree of inflammation there is a feeling of *constriction* or *oppression*. The cough is more or less *husky* and often stridulous. It is further characterized by its *dryness* and sometimes the act is *painful*. Both these features disappear with the establishment of secretion. There is generally a *slight febrile movement*, seldom very high. All of these symptoms are aggravated as the disease becomes more severe, culminating in the intense distress and impending suffocation accompanying *edema* of the glottis.

Treatment.—The cases due to infection of a common cold are best treated by abundance of fresh air in a protected room or porch, while special *inhalations* of such air are extremely useful both in giving the patient comfort and in abating the inflammation. They require no complicated apparatus. A piece of rubber tubing may be attached to the spout of a teapot or kettle, or the steam may be collected by an ordinary funnel and carried thence to the mouth. For obvious reasons, care should be taken that the funnel be not allowed to become too hot. Special appliances in the shape of a steam atomizer, more costly and scarcely more useful, may be used instead of the simple measures. *Cold applications* may be made to the outside of the throat. More rarely counterirritation by mustard may answer better. The irritative cough may require to be relieved by *anodynes*, which may consist of small doses of opium or some one of its preparations or derivatives. *Expectorants* are of doubtful value, and certainly are not nearly so useful as the simple measures which have been mentioned.

SPASMODIC CATARRHAL LARYNGITIS OR FALSE CROUP.

Definition and Symptoms.—What is known as spasmodic croup in children of from one to five years is acute catarrhal laryngitis, to which is added a *spasm of the glottis*, producing the hard, stridulous breathing, with *croupy cough* characteristic of this affection, which, once heard, is never forgotten. It is produced by the same causes. To the croupy cough are added *extreme restlessness* and an *anxious expression*. The attacks generally

come on suddenly at night, the child waking from a sound sleep, although warning is often given by some disturbance of respiration while the child still sleeps. There is little fever. The next day the child may appear almost or quite well, or there may be a slight croupy cough, yet there may occur another attack on the following night and even the third, while in very severe cases the recurrences continue for a week.

Diagnosis.—The only condition with which spasmodic croup can be confounded in diphtheritic croup, and then only if no membrane is visible in the latter. The throat should always be examined; the nose as well. In diphtheria, suddenness of onset seldom occurs, but the patient usually is hoarse several hours before much attention is paid to it. The hoarseness gradually increases, and as a rule the patient cannot speak above a whisper and cannot make a high note. The cough is smothered and sometimes harsh. In doubtful cases a bacteriological examination should be made.

Prognosis.—The prognosis in all forms of acute laryngitis not diphtheritic is generally favorable, and death is very rare from spasmodic croup. Carelessness may, however, prolong an attack.

Treatment.—The favorite measure to break the paroxysm of croup in children is an emetic. The simplest of emetics is ipecacuanha, which may be given in the shape of the wine or syrup in the dose of 1/2 dram to a dram (2 to 4 c.c.) every few minutes until vomiting is produced. The mineral emetics are more prompt, but more depressing in their action. While waiting for the action of the emetic the little patient may be put into a hot bath —temperature 98° to 112° F. (36.7° to 44.4° C.)—and some mustard may be added. The temperature is kept up by the addition of hot water, as required. The majority of attacks of spasmodic croup may be broken up in this way without further treatment. Between the paroxysms the child should receive small doses of syrup or wine of ipecac, say 5 to 10 minms (0.33 to 0.66 c.c.), until nausea is produced, or small doses of powder of ipecac conveniently in the shape of triturates containing 1/20 grain (0.003 gm.) every two hours, for an infant a year old. An opiate is particularly useful at bedtime, and by means of it a child may often be tided through a night without an attack.

Just as early as possible in the treatment an aperient should be given, than which none is better than castor oil, but calomel is also an admirable remedy for children, given in doses of from 1 to 3 grains (0.06 to 0.2 gm.). When there is fever, aconite and sweet spirits of niter in appropriate doses should be given. Special pains should be taken to maintain a uniform temperature and avoid drafts, especially when the child is perspiring freely, and it is on this account that bed is the safest place.

Counterritation by weak mustard plasters is an adjunct to treatment which should never be omitted, while gentle permanent irritation is very useful. It may be secured by any of the rubber-spread plasters now sold, known as porous plasters or capcine plasters. In severe cases, ice to the exterior of the throat, or clothes wrung out of iced water should be used, especially when there is much fever.

Parents are naturally anxious to secure some treatment by which the recurrence of attacks is prevented. It is to be remembered that a gradually increasing immunity comes with added years. Certainly no medicine can

produce immunity. It is possible, however, to do something by care and judicious outdoor life, by which is secured a "hardening" or protection against the more usual causes of laryngitis. Children who are housed are much more susceptible to croup than those who spend a portion of each day in the open air because they are more subjected to the infection causing it.

SIMPLE CHRONIC CATARRHAL LARYNGITIS.

Etiology.—The causes of nonspecific chronic catarrhal laryngitis are chiefly those which have been already mentioned as producing the predisposition to acute laryngitis—that is, the constant use of the voice in speaking and singing, excessive smoking, and the use of strong alcoholic drinks. Laryngitis occasioned by smoking and whisky drinking is often accompanied by chronic granular pharyngitis. So, too, frequently recurring attacks of acute catarrhal laryngitis independent of predisposing cause, and the long-continued inhalation of slightly irritating substances are to be included among the causes of chronic inflammation.

Morbid Anatomy.—The morbid anatomy of simple chronic catarrhal laryngitis is commonly not widely different from that of the acute form. There are the same redness and swelling, but the former is less vivid. The chronic hoarseness which is so constantly associated with it is due to a permanent thickening of the parts concerned in the production of the voice. Ulceration is not common, although there may be superficial erosions. The follicular glands are often distended, and, if the inflammation is long kept up, a hyperplasia of the squamous epithelium may result in a moderate villous outgrowth on the cords. Nodular swellings on the vocal cords are also a recognized, but rare condition, known as *chorditis tuberosa* or *pachydermia laryngis*. Relaxation of one or both cords is often present, and maintains the voice symptoms as long as it continues.

Symptoms.—The most prominent symptom of chronic laryngitis is *hoarseness*, which is found in every degree from a simple roughness of the voice to almost entire loss of it. There are also more or less *pain* and *discomfort*, but these are not ordinarily conspicuous symptoms, except when an attempt is made to use the voice. There is a decided disposition to *cough*, with a view of getting rid of some foreign substance which seems to be in the larynx. The cough also varies in degree. It may be a mere hack, or it may be scraping or ringing. It is also variously effectual in bringing up a secretion of mucus and mucopus, scanty for the most part.

Prognosis.—The prognosis of chronic catarrhal laryngitis is not encouraging for total recovery, largely, perhaps, because it is so difficult to induce the patient to comply with the conditions essential to his cure. Could this entire cooperation be secured, sometimes withheld through no fault of his own, it is not unlikely that better results would follow treatment.

Treatment.—The treatment of chronic catarrhal laryngitis requires, first, the removal of its causes, whatever they may be. The public speaker cannot expect to be cured of his malady while he continues the use of his voice, nor can the singer, or he who works among irritating vapors, nor

the *bon vivant* who will not give up his alcohol. Next to the removal of the cause comes the use of local measures because internal medication is not promising. Of course, the patient's general condition must be looked after and his strength maintained, but *local treatment is the most important.* This must be done by one versed in laryngeal applications.

TUBERCULOUS LARYNGITIS.

Etiology.—The occurrence of primary tuberculous laryngitis, long denied, has come to be generally conceded as possible, though rare. With the accepted view of the etiology of tuberculosis, tuberculosis laryngitis of the primary kind ought to be of frequent occurrence, for if the tubercle bacillus reaches the respiratory passages from without, the first point of attack would naturally be the larynx. The fact that such is not the case can only be explained on the ground that the bacillus fails to find in the mucous membrane of the larynx conditions as favorable for its growth and multiplication as it finds in the deeper portions of the lung. Since tuberculosis of the larynx is commonly secondary to the same affection of the lungs, the bacillus probably invades the larynx from the expectoration inoculation being favored by the greater or less friction between the vocal cords. Tuberculous larnygitis occurs as a complication of 20 to 25 per cent. of all cases of pulmonary tuberculosis.

Morbid Anatomy.—To the essential morbid anatomy of tuberculous laryngitis is always added that of simple catarrhal laryngitis. The latter has been described. The first stage of miliary tuberculosis without ulceration is sometimes recognized by the laryngoscope, appearing sometimes as pearly granulations in the mucous membrane, more frequently as a less distinctive, close, small-celled infiltrate. The tuberculous ulcer is more easily discovered, yet it possesses no one anatomical character by which it can be infallibly recognized. Nor are all the ulcers in the larynx associated with tuberculosis of the lungs necessarily tuberculous. The larynx is more vulnerable to the ordinary causes of simple laryngitis under these circumstances, while the constant coughing and gagging in consumption may of themselves cause laryngitis. The true tuberculous ulcer results from the caseation and disintegration of the miliary tubercle. The ulcer thus produced by the fusion of adjacent miliary tubercles is at one stage more or less characteristic by its racemose or sinuous edge, resembling in this respect the conglomerate tuberculous ulcer elsewhere. Its favorite seat is the posterior part of the larynx, *i. e.*, the posterior part of the vocal cords, the interarytenoid fold and the laryngeal surface of the arytenoid cartilages. The epiglottis is less commonly invaded, and the ventricular bands more seldom. In the case of the epiglottis there is swelling, succeeded by ulceration.

Symptoms.—The early symptoms of tuberculous laryngitis differ in no way from those of simple catarrhal laryngitis, and it is the intractability of the disease which often gives the first intimation of its tuberculous nature. The stage of simple *hoarseness* with which it is always ushered in varies also in duration, but sooner or later it is succeeded by the *aphonia* and the *painful whispering voice* which are so characteristic of ulceration of the vocal

cords or the other parts intimately concerned in the production of the voice. Sooner or later, too, *painful deglutition* sets in as a result of the extension of the ulcerative process to the more exposed portions of the larynx. The pain on deglutition is often agonizing, and is due to the fact that during the act the constrictor muscles of the pharynx squeeze the sensitive epiglottis and arytenoids. The pain is frequenty referred to the ear in the same side. Inanition and emaciation characteristic of the latter stages of the disease now rapidly increase, and death is often a welcome relief to the sufferer.

Diagnosis.—Just suspicion attaches to an obstinate laryngitis associated with acknowledged tuberculosis of the lung. With obstruction of the larynx the auscultatory signs of tuberculosis are sometimes wanting, so that we must depend on the percussion sounds entirely. As has been intimated, the distinctive features of the ulceration are scarcely sufficiently well marked to enable us to recognize the tuberculous ulcer by the laryngoscope, and to distinguish it either from the *ulceration of syphilis* or that of certain stages of *malignant disease*. To distinguish it from the former, tuberculosis elsewhere and the history of the case may help to a conclusion, therapeutic test by iodids and mercurials may be used. Syphilitic laryngitis, even when it is ulcerative, quickly yields to these remedies, as a rule, while the tuberculous condition is quite unaffected by them. With the healing of the former comes also the tendency to contraction so characteristic of all cicatrization, and especially of that of syphilitic ulcers. It is also to be remembered that syphilitic ulceration and tuberculous ulceration are sometimes associated. The involvement of the tongue in the infiltrating and ulcerating process is more characteristic of tuberculosis.

Prognosis.—The prognosis of tuberculous laryngitis is unfavorable at best. It is true that of late years the reported cures of laryngeal tuberculosis have become much more numerous, but these still bear a very small proportion to the cases that progress from bad to worse, in spite of the most skilled treatment. It is to be expected that primary tuberculous laryngitis is much more easily curable than the form secondary to consumption of the lungs. Severe pain and signs of stenosis of the larynx are unfavorable symptoms.

Treatment.—All measures which have been mentioned as useful in the treatment of chronic catarrhal laryngitis are also more or less so in tuberculous disease, with, however, less complete and less permanent results. Marvelous effects have been reported as following the use of lactic acid, while iodoform and even alkaline inhalations are also said to have healed tuberculous ulcers. All local treatment must be, of course, associated with that for general tuberculosis of the lungs. The painful deglutition, which is at once so characteristic and so distressing, has been relieved by the use of cocain applied directly to the larynx by the brush or by the spraying apparatus. The latter is the more convenient, because it can be used by the patient himself. For this purpose a two per cent. solution is suitable. A stronger—10 or 20 per cent.—may be necessary, but this must be applied with a brush by a second person. They should be used some minutes before the taking of food, as deglutition is rendered less painful for the time being by their successful application. Solutions of morphin may be sprayed for

the same purpose, or the morphin, either pure or mixed with powder or starch, may be insufflated upon the painful larynx. Iodoform is used for the same purpose in the same manner. When the pain is persistent and frequent applications are necessary, we have found none more satisfactory than the official solution of morphin sprayed into the larynx. Food may be introduced by Wolfenden's method, placing the patient on his back with the head lower than the body. General treatment by fresh air and an abundance of food as directed in the article on tuberculosis are imperative in this condition.

SYPHILITIC LARYNGITIS.

Etiology and Morbid Anatomy.—It is not necessary to dwell on the etiology of syphilitic laryngitis, as there is but one cause—the virus of syphilis. Syphilitic laryngitis may be either secondary or tertiary, and may occur at any time in the course of the disease subsequent to the second or third month following infection. Like tuberculous laryngitis, the morbid anatomy of the syphilitic form is associated with that of simple catarrhal laryngitis of the chronic kind. Excessive mucous and muco-purulent secretions cover the surface of the epiglottis and the vocal cords, while the ulcer of syphilitic laryngitis is usually more distinctive in its characters than is that of tuberculous laryngitis. The milder forms of syphilitic laryngitis are not accompanied by ulceration and are in no way peculiar, from the anatomical standpoint. The most distinctive anatomical manifestation of syphilis in the larynx is the mucous patch, like that on mucous membranes elsewhere. It is found on the epiglottis, in the laryngeal wall, and on the epiglottidean folds; rarely, on the vocal cords. The patches are rarely replaced by ulceration. The breaking down of the syphilitic gumma gives rise to another form of syphilitic ulcer, often of greater depth. The ulcer may come to a standstill at any stage, and cicatrization take place with deformity and permanent change of voice. In addition, necrosis of the laryngeal cartilages is not infrequent, portions of these being at times expectorated. Among the results of cicatrization are stenosis, resulting sometimes in complete obstruction, necessitating even tracheotomy for their relief.

Symptoms.—These are essentially the same as in tuberculous laryngitis, hoarseness, cough, aphonia, pain in deglutition.

Diagnosis.—The diagnosis of syphilitic laryngitis is justified in the absence of tuberculosis elsewhere, especially when the history of primary syphilis is present. A Wasserman reaction will make the diagnosis.

Prognosis.—The prognosis of this form of laryngitis is much more favorable than that of tuberculous disease, especially if the diagnosis be made early. The effect of contraction after healing is, however, often serious in producing stenosis, or, at least, a permanent impairment of the voice.

Treatment.—The treatment of syphilitic laryngitis is the treatment for the general affection plus the topical treatment. The latter includes the use of measures to free larynx of mucus and muco-pus, these being followed by applications of strong solutions of nitrate of silver to the ulcers,

or even the solid stick. An insufflation of iodoform, in combination with bismuth and a little morphin, is an excellent addition to the treatment.

EDEMA OF THE GLOTTIS.

Definition.—By edema of the glottis is meant edema of those parts which immediately surround that opening.

Symptoms.—It commonly results from the other conditions which have just been described, or accomapnies them. Thus, it may occur in connection with acute laryngitis, though rarely, and occasionally with the tubercu.ous and syphilitic form of chronic laryngitis. It not infrequently, also, is a complication of general diseases attended with dropsy, especially Bright's disease, small-pox, and even diseases of the heart. It may occur as a part of the symptomatology of perchondrites of the larynx accompanying typhoid fever. In any of the latter conditions it may come on quite suddenly. The more precise situation is the submucous tissues of the arytenoepiglottic folds or of the ventricular bands. The edema may also involve the epiglottis. It occurs most frequently in middle life, but it also happens in the toung.

An additional symptom of this condition is a feeling of *intense oppression* or *suffocation*. The *breathing is stridulous*, and the efforts of the patient to obtain air may bring into play all the extraordinary muscles of respiration, the whole expression being in extreme cases one of great anguish.

Treatment.—For the mild degrees of edema of the glottis the prompt application of a blister to the larynx is often sufficient to relieve the symptoms. Another remedy of some value in the milder cases is a direct spray, frequently repeated, of a solution of alum, 20 grains (1.3 gm.) to the fluid ounce (30 c.c.). Spraying with solution of adrenalin chloride is of value. In the treatment of the severer cases, cold plays an important rôle. Ice should be constantly kept in the mouth, as well as applied externally by means of ice-bags. If obtainable, the Leiter coil may be used. When danger is imminent, and time is too limited to wait for the tardy action of blisters, a half dozen or more leeches may be applied over the region of the larynx. These failing to afford relief, scarification of the edematous tissues is to be promptly performed, and, as *dernier ressorts*, either intubation or tracheotomy.

The hypodermic administration of pilocarpin has been remarkably successful in some cases, and particularly when the symptoms are of a sthenic nature this should never be omitted. One-quarter of a grain (0.0165 gm.) is the proper dose thus administered.

DISEASES OF THE TRACHEA AND BRONCHIAL TUBES.

ACUTE BRONCHITIS.

Synonyms.—*Acute Bronchial Catarrh; Acute Tracheobronchitis.*

Definition.—An acute inflammation of the tracheal and bronchial mucous membrane. It is essentially a symmetrical disease, the bronchial tree in both lungs being more or less uniformly invaded.

Etiology.—The most frequent cause of acute bronchitis is some infection due either to some organism already *in situ* or to one introduced from without (the specific organism if one exists is not yet identified). Exposure to wet and cold may be and frequently is a predisposing factor, the resistance of the body being thereby lowered and the infection then becoming active. "Catching cold" is such an old and common expression that it may be retained, but it should be well understood, that cold *per se* does not cause the symptoms. This is amply proven by the results in the treatment of tuberculosis, pneumonia, etc., in the open air, and by those who live out doors night and day. Such individuals do not "catch cold" however low the temperature, or however many drafts in the room, provided they are not exposed to infections and are well *protected*. Chilling of the body due to lack of protection may bring about the infection and all its symptoms. It often succeeds an ordinary coryza or cold in the head or a laryngitis, the inflammation extending from the upper air-passages downward. It is naturally more prevalent in the winter than in the summer. It is usually a symptom of influenza, whether epidemic or sporadic. Invariably too, it accompanies measles, of which it is the most annoying symptom. More rarely it is caused by irritating fumes.

Morbid Anatomy.—The mucous membrane of the trachea and large bronchi is congested and more or less covered with a tough mucus, rich in cells, the hyperemia being especially marked about the glands whence comes the secretion. Decided *cellular* infiltration of the mucosa does not occur in ordinary cases, because of the almost tendinous basement membrane which intervenes between the blood-vessels and the mucosa.

Symptoms.—*Cough* is the most constant and conspicuous symptom. At the beginning it is hard and dry, without expectoration; sometimes it is painful. As the disease advances it gradually becomes looser. In the milder degrees there is no shortness of breath, but in the severe there is a varying degree of *dyspnea* with a sense of *oppression* or *constriction in the front of the chest*, caused by stenosis of the bronchial lumina, due to the swelling of the mucous membrane and the presence of secretion. *Fever* in mild degree is commonly present, but the temperature rarely exceeds 101° F. (38.3° C.). If it does, there is reason to suspect a more deep-seated involvement of the smallest or capillary tubules, whence the name capillary bronchitis, referred to in considering bronchopneumonia. This extension is particularly apt to take place in children and old persons, in whom the physicians should always be on the lookout for it. With the access of fever the *pulse* is correspondingly accelerated. *Rarely*, a *chill* may usher in the disease. It cannot be too strongly impressed that a continued high temperature indicates a more serious infection than that affecting the bronchila tubes.

The scanty *expectoration* of acute bronchitis is at first glairy or mucoid, and later mucopurulent. With the appearance of the latter the cellular element, composed of pus-cells and desquamated epithelium, becomes more abundant. With the abatement of the disease the pus-cells become again less numerous and finally disappear.

Physical Signs.—There may be absolutely no physical signs—inspection, palpation, percussion, and auscultation being alike negative. When the trachea alone is involved there may be roughened breathing over the

trachea. In other cases *inspection* may reveal increased frequency of breathing, and possibly increased rate of the cardiac apex-beat if there be fever. *Palpation* may appreciate a rhonchal fremitus if there be sufficient narrowing of the breathing tubes. It may be found anywhere on either side, and is usually transient. *Percussion* continues invariably clear so long as the bronchitis is uncomplicated. *Auscultation* furnishes the most distinctive and constant physical sign, the presence of dry râles, the sonorous and sibilant which may invade either or both lungs, and may also be transient, coming and going. To these may be added harshness of breathing sounds. When resolution sets in, bubbling râles may take the place of the sonorous and sibilant, in consequence of the presence of liquid secretion. For physical signs of capillary bronchitis see Bronchopneumonia.

Diagnosis.—This is generally easy. The presence of the dry râles and a clear percussion note belong to no other condition than acute bronchitis and bronchial asthma, but to the latter are added the signs of spasmodic contraction of the bronchi, notably the panting breathing. The same clearness of percussion note continues with the appearance of moist râles, unless there be the complication of capillary bronchitis or pneumonia.

Prognosis.—Very often the symptoms subside without treatment in the course of two or three days. The cough becomes loose, expectoration is easy, fever and other unpleasant symptoms disappear, and in a week the patient is well. Suitable treatment may hasten such an issue. In other instances, especially in persons who are weak and debilitated, no such speedy termination takes place, but even in many of these after a long interval the patient recovers. More rarely, particularly in the very young and old, the inflammation travels down into the smallest tubes, producing the capillary bronchitis alluded to. In other instances still, especially after several attacks, and in the old particularly, chronic bronchitis may supervene with the symptoms and physical signs which will be described when considering it.

Treatment.—The best treatment for a case of ordinary acute bronchitis is "the bed." Twenty-four to 48 hours in bed with the windows wide open and the patient well protected by clothing will go farther to cure such a case promptly than all the cough medicines ever prescribed. Such a course is not, however, always possible, and the physician is often expected to cure acute bronchitis while the patient is on his feet and even attending to business. Every patient, however, can sleep at night with his windows open much to his benefit. The patient should, however, be put to bed if possible. Next to rest in bed is counterirritation. Turpentine and mustard are the best agents. A turpentine stupe or weak mustard-plaster applied to the front and back of the chest will aid greatly in allaying cough and relieving the sense of oppression.

Cough medicines are, of course, expected, and are useful. In the ordinary simple bronchitis, especially when there is moderate fever, there are few remedies more efficient than the simple solution of citrate of potash of the United States Pharmacopeia, in doses of 1/2 ounce (15 c.c.) every two hours. It may be desirable to add a few drops of wine of ipecac or wine of antimony to each dose to increase the relaxing effect, while, if the fever is decided, 1 or 2 minims (0.06 or 0.12 c.c.) of the tincture of aconite will aid

in breaking it. A diaphoretic effect is further encouraged by adding 30 minims (2 c.c.) of the spirit of nitrous ether. By such measures the cough is usually loosened in 24 hours, the dry râles are substituted by moist ones, and convalescence progresses. If there is decided oppression, it may be relieved by inhaling the steam from a hot saturated solution of chlorid of ammonium, or the compound tincture of benzoin floated on hot water, while in children an emetic dose of ipecac may produce the desired relaxation.

The cough may, however, be so constant as to harass the patient and keep him awake in spite of the measures suggested. In this event an opiate is necessary, and a small quantity of morphin or heroin, 1/16 to 1/12 grain (0.004 to 0.0055 gm.) for an adult, may be added to the combination previously recommended. It is, perhaps, on the whole better to administer the opium separately, and of all the preparations, Dover's powder is probably the best. Indeed, Dover's powder alone is one of the best medicines in acute cough in doses of 2 1/2 grains (0.16 gm.) every two hours, preferably in a pill or capsule; or if it be at night and a prompt effect be desired, 5 (0.32 gm.) or even 10 grains (0.65 gm.) in one dose will often act like a charm. Codein is a good preparation of opium, and has the advantage of disturbing the system less than some others. It may be given in doses of 1/4 to 1/2 grain (0.016 to 0.032 gm.) as often as necessary to quiet cough. Heroin is a popular modern remedy of this class, given in doses of 1/20 to 1/12 of a grain (0.003 to 0.0055 gm.). This must be given tentatively, the writers have seen 0.005 of heroin give rise to toxic symptoms when used every three hours.

Should convalescence be slow and expectoration prolonged, the ammonium chlorid in 5 to 10 grain (0.32 to 0.65 gm.) doses with syrup or tincture of squills may be substituted for the sedative mixture, and quinin and restorative measures added to the treatment. If the cough is paroxysmal, the preparations of belladonna may be given, and are often efficient in controlling the paroxysms where opium is contraindicated or deemed unnecessary. So, too, when secretion is copious and cannot be expectorated, belladonna tends to diminish it, and may be given with expectation of relief. Copious secretion in children may be removed by an emetic. To this end alum and honey may be given or syrup of ipecacuanha in teaspoonful doses. All such measures are, however, depressing and may be succeeded by recurrence of secretion, and should be used only when necessary and in very severe cases.

CHRONIC BRONCHITIS.

SYNONYM.—*Chronic Bronchial Catarrh.*

Definition.—A chronic inflammation of the mucous lining of the large and medium-sized bronchial tubes, commonly symmetrical.

Etiology.—Uncomplicated and primary chronic bronchitis usually develops gradually, representing the accumulating remnants of frequently recurring "colds," each of which leaves something behind it until the chronic condition is established. A bronchitis that is associated with or consequent upon another disease may continue and become chronic after the disease

has disappeared. This may happen with measles or influenza, or even, rarely, pneumonia.

Chronic bronchitis constantly attends other affections as a consequence. The most common of these causes is tuberculosis, but it is also the result of diseases which favor congestion of the air-tubes by reason of the obstruction to the circulation which they cause, such as cardiac valvular disease. Especially is this true of mitral valve disease and Bright's disease.

Morbid Anatomy.—The bronchial mucous membrane is bathed with a dirty gray secretion derived from the mucous glands, which are sometimes hypertrophied. The darker color is due to inhaled "blacks," exfoliated degenerated cells, and sometimes to decomposed blood. On scraping this mucus away, there may be little or no change of appearance; at other times there may be a decided hyperemia. In places the mucous membrane may be thickened by cellular infiltration; at others it may be thinned, producing sometimes a lattice-like appearance, because of the prominence of the bands of elastic tissue which resist the atrophic process. In old cases there is often dilatation, which may be saccular, fusiform, or cylindrical, and sometimes cavernous dilatations are present, usually about the center of the lung. It is in the latter more particularly that the mucous membrane is found thinned and the mucous glands atrophied; at others, ulcerated. In other old cases there are ulceration and necrosis of the cartilaginous rings.

Symptoms and Course.—The chief symptom of chronic bronchitis is *cough*, which is troublesome in various degrees, and is apt to be worse at night or in the morning. Frequently it is paroxysmal, the spells terminating in free expectoration of the secretion which has excited the coughing.

Chronic bronchitis is commonly attended with *free expectoration*, either in the manner just described or more uniformly distributed through the day. The expectorated matter is usually mucopurulent or purulent, the color deepening to yellow as the proportion of pus corpuscles increases, and becoming darker in hue with the admixture of dead epithelium and "black" inhaled. The quantity is sometimes very large, amounting to 1/2 liter (a pint) or more in the 24 hours. As the quantity increases, however, the consistence diminishes, and it may be thin and watery. To such copious expectoration the name of *bronchorrhea* is applied, giving the name to one of three varieties of chronic bronchitis. It is probably the *asthma humidum* of the older authors, the *catarrhe pituiteux* of Laennec.

More commonly the expectoration is purulent, containing greenish-yellow masses which are coughed up easily. The bronchi are usually more or less dilated in these cases. The more copious secretion of bronchorrhea usually separates, on standing, into two portions—a superficial seromucous portion, which may be frothy, and a lower thick portion made up more largely of pus-cells. In addition to such pus-cells the microscope discovers squamous epithelium from the mouth, columnar cells from the deeper air-passages, bacteria, and sometimes a few blood-corpuscles, as well as the delicate whetstone-shaped crystals known as Charcot's crystals.

Respiration is accelerated in various degrees, but except in the rare forms to be described and on exertion, dyspnea is never so marked as even in mild cases of tuberculosis. The *absence of fever* is characteristic as contrasted with tuberculosis of the lungs which chronic bronchitis

so often resembles in other respects, tuberculossis being constantly diagnosed bronchitis. Again after chronic bronchitis has existed for a long time in the old, especially when secretion continues copious while expectoration becomes difficult, there sometimes supervenes a condition of *low fever*, probably septic, from absorption of putrid matters, and unless expectoration can be reestablished, the patient sinks and the fatal end is not very remote.

The appetite and digestion commonly remain good, and the patient maintains his weight for a long time. After a while, however, these may fail, especially if there is much expectoration, and then the patient loses weight. On the other hand, some subjects of chronic bronchial catarrh remain quite corpulent and well nourished throughout a long illness, and, except for the cough, the amount of disturbance is often remarkably slight. There is no pain, except sometimes about the attachment of the diaphragm in the lower thorax caused by the harassing cough.

In a second variety the cough is "dry," without expectoration except small, tough, tenacious masses of mucoid matter. These are raised after paroxysms of coughing, often of great severity. This dry variety—the *catarrhe sec* of Laennec—is commonly associated with emphysema, and is a very troublesome form.

A third variety of chronic bronchitis is well called *putrid* or *fetid* bronchitis, in which the secretions decompose in the air-passages and acquire a sweetish, sickening, and disgusting odor, which may pervade an entire apartment and make the patient a nuisance to himself and others. In these cases there is apt to be a communication with a cavity due to bronchiectasis or tuberculosis. The decomposition is due to the bacteria of decomposition, the action of which is doubtless favored by retention of secretion in dilated bronchi and phthisical cavities, and in a decided majority of cases it succeeds an ordinary chronic bronchitis. It also sometimes follows an empyema which perforates into the lung. At times it is said to be primary. The expectoration is copious and correspondingly thin. It is also separated into layers: an upper one of frothy, mucopurulent matter in which occur separate masses, and a lower of thicker, greasy, purulent matter. In the latter the naked eye often recognizes dirty gray masses about as large as a pea, known as Dittrich's plugs, which on microscopic examination are found to contain pus, bacteria, and detritus of uncertain origin, together with delicate acicular fat crystals. Among other fungi are found also leptothrix filaments, which must not be mistaken for elastic tissue.

The chief additional symptoms are *fever*—it may be septic—with increase of cough and pain in the side. There is also sometimes a *chill*. These symptoms may again abate and those of the more usual form of chronic bronchitis prevail, subject to exacerbation and improvement. The effect of the fetid form, as might be expected, is more severe on the constitution, and there are loss of appetite, indigestion, and failing health. The fingers may be clubbed, as in phthisis. Secondary purulent meningitis and abscess have appeared from the transfer of pus germs. The physical signs do not differ from those of chronic bronchitis and bronchiectasis, to be described.

Physical Signs.—Physical signs of a decided character more constantly attend chronic bronchitis than acute. They present, however, no unchanging picture. There may be nothing apparent to inspection, or the frequently associated complication of emphysema of the lungs may be the cause of a diminished excursion of respiratory motion, and the roundness or barrel shape of the chest characteristic of that disease may be seen. Such emphysema may give diminution of the normal tactile fremitus and to percussion a hyperresonance. In the vicinity of a superficial dilated bronchus filled with secretion there may be impairment of resonance. The resonance is, however, restored after copious expectoration, or the percussion signs of a cavity may be substituted, though in the middle or lower part of a lung instead of the apex, as in consumption. Vesiculo-tympanitic or even tympanitic resonance may be present from relaxation of lung tissue, especially in the lower posterior part of the lungs.

Auscultation may also be negative, but much more frequently recognizes an alternation or combination of harsh and feeble breathing, sonorous and sibilant râles, with moist râles of all sizes, variously modified by different distances from the ear and varying consistence of the secretion. The moist râle is the most constant sign of chronic bronchitis.

Diagnosis.—This is not usually difficult, for while the symptoms, including coarser appearance of the sputum, sometimes closely resemble those of tuberculosis of the lungs, the physical signs do not, except when a dilated bronchus presents the same signs as a cavity. Such a dilatation is, however, found in the middle of the lung, and furnishes its signs in the neighborhood of the angle of the scapula, rather than at the apex. The absence of fever and especially of tubercle bacilli from the sputum after careful examination is presumptive evidence of the absence of tuberculosis. The tuberculin test is of little value because of its appearance in adults as the result of non-active tuberculosis. A great danger is the habit of considering tuberculosis of the lungs as chronic bronchitis.

Prognosis.—This is unfavorable as to recovery, but favorable as to fermination. The patient rarely dies of the direct effect of the disease, being generally carried off by some intercurrent affection, often croupous pneumonia. In the old, however, a condition described on page 268 may intervene, or a bronchopneumonia may supervene and terminate fatally. On the other hand, many patients the subject of chronic bronchitis live for years in comparative comfort, getting almost well in the summer and relapsing in the winter.

Treatment.—If it were possible to remove every person with simple chronic bronchitis uncomplicated by heart or kidney disease to a warm *climate* where they could live continuously in unbreathed air, they would probably get well. Certainly is this true of the earlier stages. Much may, however, be done at home to prevent the exacerbations due to exposure, each of which adds a little to the previous chronic condition. This consists mainly in dressing warmly, living day and night in thoroughly ventilated rooms, and in avoiding exposure to rapid changes of temperature without adequate protection to the body. Exercise under careful supervision is very necessary, lack of exercise may lead to weakness of heart and skeletal muscles which may be ruinous. It is especially important that

the old should be warmly clad with wool next the skin, and precautions against cold feet should be especially secured. When bronchitis complicates other diseases, as heart disease and kidney disease, the treatment of these is important.

In the way of medicine, much can be done by the *stimulating expectorants*. It must be remembered that constant dosing with an excess of any of the following drugs which are recommended, may lead to two things—impairment of digestion, which in turn will lead to loss of general health and strength; and second, depending upon drugs and omitting rational hygiene rules. The terebinthinates are the best, and of these one of the best is terebene. Five to 10 minims (0.3 to 0.6 c.c.) in a capsule every three hours is a proper dose. Terpene hydrate another derivative of turpentine, may be given in doses of 2 to 6 grains (0.13 to 0.40 gm.) in pill as often, or it may be given in mixture with enough alcohol to dissolve it. Creasote is an admirable remedy in chronic bronchitis; 1 grain or minim (0.06 c.c.) or 2 minims (0.12 c.c.) three times a day, increased gradually to 5 grains (0.3 c.c.), or even more than three times a day, will after a while diminish the secretion and the cough. Creasotal, or the carbonate of creasote, is a much more pleasant remedy, and may be given in doses of 10 minims (0.6 c.c.), which may be increased. Sandalwood oil or balsam of tolu or Peru may be given. The compound tincture of benzoin is another old but good remedy. The oil of eucalyptus is another remedy of the same class in 5 minim doses in a capsule with an equal quantity of oil of sweet almonds. Yerba santa, in the shape of the syrup or fluid extract, is a similar and very agreeable remedy. Other stimulating expectorants, like the carbonate of ammonium or the aromatic spirit of ammonium, are often useful, but they lose their effect after a time. The carbonate of ammonium, to be useful, must be given often—5 to 10 grains (0.32 to 0.65 gm.) every two hours. The ammonium chlorid is indicated where less of a stimulating effect is necessary —5 to 15 grains (0.32 to 1 gm.) four times a day in combination with the syrup of squill in 15 minim 1 c.c.) doses, both in the compound licorice mixture. In some cases the iodid of potassium is very useful, especially when secretion is scanty. It should be kept up for some time.

Inhalations of medicated vapors are sometimes useful. The compound tincture of benzoin may be thus used, or the oil of eucalyptus, also turpentine. They may be placed on the surface of boiling-hot water, the vapor from which will carry the medicated preparation with it, and may be conducted to the air-passages through a cone of paper placed over the vessel containing the medicament. These vapors are more efficient than atomized fluids. Simple steam or vapor from a two per cent. solution of common salt or of sodium bicarbonate may be used. If there is fetor, carbolic acid may be used in the atomizer, a two per cent. solution, or thymol one part in 1000.

Alkalinity is an essential condition of easy secretion from the air-passages, so that both inhalations and internal remedies should fulfill this condition. Hence, simple liquor potassæ, U. S. P., in 15 to 20 minim (1 c.c. to 1.25 c.c.) doses in milk is a good remedy. To this end the free use of alkaline mineral waters, as those of Vals, Vichy, and Ems, is useful.

Digitalis and strychnin are excellent medicines, especially the latter. Both stimulate the cardiac action and aid in pumping the blood through

the lungs with increased force, thus causing relief to the congested mucous surfaces. Strychnin in ascending doses may be given with advantage.

As to *health resorts* suitable for cases of chronic bronchitis, those with a dry climate, not too cold, should be selected for cases with copious secretion, such as southern Georgia and the Carolinas or New Mexico in this country, or for stronger persons the cooler climate of Colorado. For cases of dry bronchitis the warmer moist climates of Florida are very suitable. In this, as in all other diseases, the factor of complete bodily and mental rest enters largely into the cure.

Chronic bronchial catarrhs always improve in summer, and it is generally sufficient if the patient be directed to leave the hot and noisome city and spend his summers either in the mountains or at the seaside, where the air is pure and bracing. Surely this is due to living out of doors and not breathing vitiated air.

Of foreign resorts, those of southern Europe, especially Italy, the western Riviera, San Remo, Mentone, and Cannes, are suitable, while still better, if they can be availed of, are Egypt, Algiers, and the island of Madeira.

BRONCHIECTASIS, OR BRONCHIAL DILATATION.

Etiology.—The most common cause of bronchiectasis is chronic bronchitis, either simple or tubercular, the effect of the inflammation being to weaken the bronchial walls so that they yield to the inspiratory and expiratory strain to which they are subjected in the act of coughing. It is, therefore, often associated with emphysema. The same cause contributes to the bronchial dilatation following bronchopneumonia, measles, and whooping-cough in children. Accumulated secretion is also a factor, as seen in the dilated bronchi which succeed obstruction of a bronchial tube by a foreign body, or compression by aneurysm or mediastinal tumor. The traction associated with fibroid induration is also a cause of bronchial dilatation; hence we find it in association with interstitial pneumonia and sometimes in chronic pleurisy. Finally, bronchial dilatation is rarely a congenital defect, in which event it is also commonly unilateral and general—*bronchiectasis universalis* of Grawitz.

Morbid Anatomy.—Bronchial dilatation is cylindrical and sacculated. The terms explain themselves. Both forms may occur in the same lung.

In the *cylindrical* form, which is the more common, dilated tubes of nearly equal caliber may run through the substance of the lung, from the root to the pleural surface, producing an appearance not unlike the fingers of a glove. More frequently the smaller tubes only are affected, dilatation being recognized at autopsy by the inequality of lumen, rather than by *ante-mortem* physical signs. It may, however, be suspected in any case of chronic bronchitis with copious expectoration.

The *saccular* bronchiectases are spherical or oval dilatations, into which the tube merges gradually or suddenly. They may attain a diameter of from two to three inches (5 to 8 cm.), more or less. The lung tissue around a saccular dilatation is rarely normal. Commonly, the dilatations, single or multiple, are surrounded by indurated and contracted lung tissue, the trac-

tion of which on the bronchial wall produces the dilatation. Adhesion of the lung to the costal pleura also contributes, and large subpleural cysts are at times thus formed by the contracting tissue. The cavities thus produced are commonly at the base of the lung, while in chronic phthisis they are found at the apex. Cylindrical and saccular dilatation may also be associated under these circumstances. In universal bronchiectasis the entire bronchial tree is converted into a series of sacs communicating one with the other. Many cavities in pulmonary consumption are primarily bronchiectatic cavities.

In all forms there is decided change in the bronchial wall, the principal feature of which is atrophy. This atrophy not only attacks the mucous coat, but also the muscular, and sometimes the elastic tissue and cartilage, reducing the wall to a thin, smooth membrane, lined with pavement epithelium, instead of the usual cylindrical form. At times overgrowth, involving particularly the connective tissue, takes place, forming lattice-like projections on the inner surface of the tube already referred to in treating of chronic bronchitis. At other times ulcerative processes develop, perforating the bronchus and invading the lung parenchyma, converting the bronchiectasis into an ulcerating cavity.

Symptoms.—These, in addition to those of the disease with which the bronchiectasis is associated, are the peculiar *sputum* and *paroxysmal cough.* The sputum furnishes the most distinctive feature, from which alone the diagnosis can sometimes be made. It is mucopurulent, of a dirty yellowish-green color and unpleasant, stale, and sweetish odor, though not exactly fetid, as in fetid bronchitis. It is often raised in mouthfuls—another characteristic. It also separates into layers, usually three, as in chronic bronchitis, of which the upper is frothy and thin, the middle mucoid, and the lowest made up of pus and epithelium in various stages of fatty degeneration, acicular fat crystals, and sometimes red blood disks and hematoidin crystals sufficient to color it. Elastic tissue of the lung is not present; nor are tubercle bacilli, unless there is associated tuberculosis with ulceration of the bronchial walls.

The cough is paroxysmal, because it is not usually excited until the sac, which is often insensitive, becomes full enough to irritate the healthy mucous membrane, when cough is at once excited and continues until the cavity is empty. The paroxysms are usually in the morning, when they may be excited by a change in position. After their termination there is commonly a long period of rest until the sac is again filled. The more paroxysmal the cough and copious the expectoration in chronic bronchitis, the more likely is there to be a dilated bronchus. Very characteristic is the *absence of fever.*

Physical Signs.—When distinctively present, they are those of a cavity in the lung, readily recognizable when near enough to the surface. They include tympanitic percussion note, bronchial and even amphoric breathing, bronchophony or pectoriloquy if the cavity is empty. If it contains liquid, gurgling may be heard and the percussion note is dull. To palpation there is usually increased vocal fremitus, caused by surrounding consolidation. All signs vary according as the cavity is filled or emptied of secretion. A restricted breathing excursion may also be present, uninfluenced by the state of the cavity, whether full or empty.

Diagnosis.—A bronchiectatic cavity is usually distinguished from a *phthisical cavity* by the absence of tubercle bacilli and elastic tissue from the sputum of the former, the situation of the cavity in the center instead of at the apex of the lung, although a cavity from tuberculosis may occur anywhere in the lung, the history of its development, the absence of cachexia and fever. Hypertrophy of the right ventricle is more frequent in bronchiectasis, but may also be present in fibroid phthisis with or without bronchiectasis.

A *circumscribed empyema* which has ruptured into the lung is much more sudden in its development than bronchiectasis, while the history of a previous pleurisy is superadded. A coincident external perforation of an empyema would clear up all doubt. A true *abscess* of the lung which has found its way into a bronchus has also a different history of origin, succeeding, as it usually does, a pneumonia, a massive hemorrhage, or traumatic cause. The same is true of gangrene of the lung, which is, however, disclosed by the extreme fetor of the breath and expectoration.

Treatment.—This includes that of chronic and fetid bronchitis, to which may be added, under favorable circumstances, the injection of sacs and their drainage. It is to be remembered, however, that physical signs are sometimes misleading, and that what seems to be the clearest evidence as to the exact site of a sac is not always to be relied upon. The newer methods of pneumosurgery allow the exploration of the chest cavity with relative ease and safety, hence, suspected areas can thus be explored. The location of such cavities can sometimes be done by X-ray.

The cure of well-established bronchiectasis is impossible with medical treatment except, perhaps, in young persons. Something may be done to prolong life and make the patient more comfortable and less disagreeable to others. To this end we must aim at the evacuation and disinfection of the offensive purulent secretion, and as far as possible the obliteration of cavities. For the first of these, the inhalation of crude creasote vapor was recommended, first by Arnold Chaplin, and indorsed by Theodore Dyke Acland in an exhaustive paper on this subject. The method is as follows: The patient is placed in a small air-tight room with cotton pads over the eyes and ears, the nostrils stuffed with cotton-wool. A teaspoonful of creasote is then poured upon water in a suitable vessel and vaporized over a spirit lamp. The fumes are at first very irritating and provoke violent coughing, which causes the offensive material to be entirely expelled. The treatment should at first be kept up for 10 to 15 minutes only every other day. As the patient becomes accustomed to it, the exposure may be lengthened to an hour daily. A simpler method, though not so effectual, is to have the patient inhale through an inverted funnel the fumes of creasote laid upon hot water. Intralaryngeal injections of oily and antiseptic substances have been employed, with doubtful results. The difficulties in the way of operation are shown in the first paragraph on treatment.

BRONCHIAL ASTHMA.

Definition.—Bronchial or spasmodic asthma is a paroxysmal dyspnea, which is the direct result of a stenosis of bronchi.

Etiology.—There is some diversity of opinion as to the etiology of bronchial asthma. This much, however, is admitted, that in some way there is produced a narrowing of the smaller bronchi.

Various explanations of the narrowing are suggested. Some allege a simple swelling of mucous membrane to be a cause. Such swelling is variously spoken of as "fluctionary" (Traube), "vasomotor turgescence" (Weber), "diffuse hyperemic swelling," or "exudative" inflammatory swelling (Curschmann). On the whole, the older view of Trousseau, that the narrowing is due to a spasmodic contraction of the muscular coat, seems the most likely one, and has recently received the support of Biermer and still later of Meltzer based upon experiments of Auer and Lewis.

Accepting Trousseau's view of a primary spasmodic contraction of the bronchi, it becomes necessarily a reflex act, the causes of which are various. It implies, first, a hyperexcitability of the reflex center. Hence bronchial asthma is not infrequent in neurotic persons, and has even been classed as a functional nervous disease with neuralgia and epilepsy, with which it is said to alternate at times. Such hyperexcitability is sometimes inherited, so that bronchial asthma often runs in families. Presupposing such excitability, numerous peripheral causes may supervene, the most frequent of which is bronchitis. It very often happens that an asthmatic subject has an attack of asthma, brought on by "taking cold," the incident bronchitis being the exciting event.

Comparatively modern studies have demonstrated the association of some affections of the throat and nasal passages with bronchial asthma, and that their removal has resulted in its cure. Among these have been enlarged tonsils, chronic catarrh, nasal polypi, and the like. Other causes in susceptible persons are impressions of certain odors, pleasant and unpleasant, notably that of flowers or plants in early summer, whence the term "rose" asthma and hay asthma, both of which are allied affections. Similar attacks come from inhalation of emanations from cats, horses and rabbits. A change of air, as from town to country, or the reverse, or from mountain to lowland, acts similarly. Causes more remote than those of the nasal passages, such as gastric derangement, intestinal worms, uterine disease, may be admitted. Purely emotional causes, as fright and emotion, may also act. The frequency of bronchial asthma in children has already been mentioned. It is more common in the male sex. Meltzer in 1910 draws attention to the fact that many cases of asthma resemble in every particular except that of termination, anaphylactic shock. He therefore makes the suggestion that "asthma is an anaphylactic phenomenom. That is, that asthamatics are individuals who are 'sensitized' to a specific substance and the attack of asthma sets in whenever they are 'intoxicated' by that substance.

"It was proved that anaphylactic shock was peripheral and not of central origin. It is therefore suggested that also the so-called nervous asthma is due to a peripheral and not a central cause."

This theory is so attractive as to possibilities of treatment, that we accept it.

Morbid Anatomy.—Whatever may be the morbid state of the tubular structure of the lung during an attack of asthma, there are no postmortem

appearances which are distinctive of it. In the first place, the chance is seldom offered at the opportune moment, and we know of no report of a necropsy made on a person dying during an attack of asthma. In the case of the asthmatic dying at other times, there may be found the morbid states peculiar to chronic bronchitis and emphysema, but nothing more. However, autopsies made upon animals dead of anaphylactic shock show the alveoli dilated as the result of spasm of the bronchioles.

Symptoms.—The symptoms of an attack of spasmodic asthma are unmistakable. The typical asthmatic is apparently in good health between the attacks, and often is so up to the time of the attack, which then comes on suddenly, often at night. At other times there is a prodromal stage, a feeling of thoracic discomfort, or *"tightness" in the chest*, or an anxious, nervous, restless feeling, the import of which is well understood by the victim.

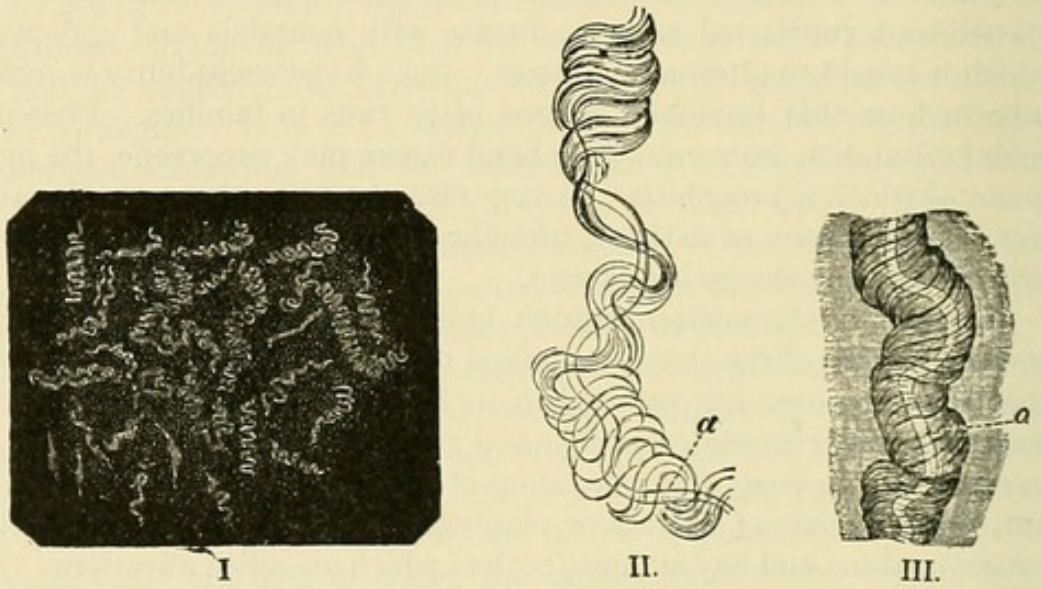


FIG. 107.—Curschmann's Spirals—(*after Curschmann*).
I, Natural size; II and III, enlarged; *a*. *a*, central thread.

The attack consists of a long-drawn-out inspiratory act, in which it is evident the air cannot get into the lung fast enough to meet the demands of the *besoin de respirer*. The auxiliary muscles of respiration, the sterno-cleidomastoid, and the scaleni, do their best to enlarge the thorax, but that is not the difficulty. It is the contracted tubes which resist the entrance of the air. Even more marked are the effort and the duration of expiration; hence the dyspnea is spoken of as an expiratory dyspnea. The abdominal muscles are the auxiliaries here, and they contract strongly and assume a board-like hardness. The air is heard to whistle as it enters and passes out of the chest. The patient sits in an upright position, or leans slightly forward, and often astride of a chair grasps the back with his hands, for it is by fixing the shoulders that he can bring the extraordinary muscles of respiration into play. His face is anxious, pale, or it may be cyanotic, and few more distressing pictures are seen. Notwithstanding his efforts, they fail of their purpose and comparatively little air enters the lungs. With all these efforts, the *breathing is not accelerated*—at least accelerated to any marked degree—while in a few instances the breathing-rate is diminished.

The *temperature* is normal or subnormal, and the pulse is accelerated and small.

The attacks last for a variable period, rarely less than an hour, and unless broken up, sometimes several hours. They may *terminate* as *suddenly* as they began, sometimes with a spell of coughing. On the other hand, *cough is not a marked symptom*, and in brief paroxysms of asthma may be altogether wanting. In severe ones, however, it is present, accompanied by a tough and scanty expectoration, containing rounded masses of matter, either yellowish or grayish translucent—the "perles" of Laennec. On minute examination, these are found to be made up of the so-called Curschmann's spirals, together with numerous swollen and fatty degenerated pus-cells and cells shed by the bronchial mucous membrane and alveoli. The spirals have long been recognized, but were first studied by Ungar and Curschmann. Two shapes are found. The first appears to be made up of *mucin* spirally arranged, entangling pus-cells and alveolar epithelium. A second form consists of a tightly wound spiral of mucin fibrils, containing in its center another bright, clear filament. The spirals are believed by Curschmann to be formed in the finer bronchioles, and to be a product of bronchiolitis. Their spiral form is unexplained. The sputum also sometimes contains *crystals of calcium oxalate* and *calcium phosphate*. The yellow masses contain, in addition to the cells named, various numbers of acicular crystals, which were first found by Leyden in the sputum of asthmatic patients, and therefore called Leyden's crystals. They are identical with the so-called Charcot's crystals, found in leukemic spleen, bone-marrow, and semen.

In the *blood* of cases of bronchial asthma an increase of eosinophiles is noted at about the time of the paroxysm, amounting from 10 to 53.6 per cent., according to J. S. Billings, Jr. This fact is of value in diagnosis as such leukocytosis is said not to occur in renal and cardiac asthma. In like manner, eosinophilic, leukocytes and granules are found in the *sputum* of asthmatic attacks. The latter are often attached to the Curschmann's spirals.

In addition to the cases of typical asthma in patients perfectly comfortable between attacks, and for which the foregoing description is intended, patients with chronic bronchitis and emphysema are subject to attacks which may be called symptomatic asthma. The symptoms are, however, similar and need not be repeated. It is to be remembered, too, that emphysema is caused by asthma, as well as that chronic bronchitis and emphysema may cause asthma.

Physical Signs.—These are also characteristic. *Inspection* notes the most labored effort in breathing, yet the chest moves but slightly. It is in a state of permanent inflation. The spaces above and below the clavicle and above the sternum, the intercostal spaces, and the pit of the stomach are drawn in from the same cause—that is, the thoracic cavity not being filled from within, the external atmospheric pressure forces the yielding portions inward. Rhonchal fremitus is recognized by *palpation*, while vocal fremitus, obscured by the rhoncus, is further diminished by a frequently associated emphysema. *Percussion* may produce abnormal resonance which is due to temporary emphysema. This disappears after

the attack unless the emphysema is long standing. *Auscultation* discovers the most striking and easiest recognized of the physical signs. All over the chest are heard sonorous and sibilant râles, inspiratory and expiratory, the latter longer and more marked. In fact, for the most part, they do not require the ear to be placed close to the chest. They may be heard at a distance. The vesicular murmur, on the other hand, is inaudible. Later in the attack, as secretion increases, the râles become moist. It is to be remembered that chronic bronchitis, emphysema, and asthma may also complicate one another and render correspondingly complex the physical signs.

Diagnosis.—This can usually be made at a glance. *Spasm of the glottis* and *paralysis of the abductors of the glottis* produce similar efforts at breathing, but the dyspnea is inspiratory and unattended by the lung sounds characteristic of asthma, while the history will be found different. *Hysterical dyspnea* furnishes no physical signs, while in *cardiac asthma* also the breathing sounds are normal, or there are many moist râles. (See also Cardiac Asthma.)

Prognosis.—Bronchial asthma, though a distressing disease, is not a fatal one. Very often the attacks grow more infrequent and milder as the patient grows older, and they may disappear altogether, while in some cases they increase in severity and frequency with age. In other cases a cure is effected by discovering and removing the cause. In still other cases a grave emphysema develops which may give rise to all the distressing symptoms of that condition.

Treatment.—The first object in the treatment of asthma is to *relieve* the *paroxysm*. This is best accomplished by a hypodermic injection of morphin, 1/4 grain (0.0165 gm.), with 1/150 grain (0.00044 gm.) of atropin, which may be repeated in an hour if ineffectual. If morphin and atropin are not at hand, nitrite of amyl may be inhaled from a handkerchief on which a few drops have been placed, or a pearl may be broken, if one of these be at hand. In the absence of amyl nitrite, chloroform or ether may be similarly used. Nitro-glycerine may be used hypodermically. Adrenalin chloride in 10 to 15 minims hypodermically is sometimes useful. After the paroxysm is broken, every effort should be made to *discover* a *cause* for the recurring attacks. The nose may be responsible, and should be carefully examined for any one of the causes referred to. Possible peripheral irritation, whether by error of diet, gastric derangement, uterine or other distant reflex cause, should be sought and corrected. These are not always easily found, but sometimes they are. Bronchitis, when present also requires treatment by the usual remedies.

It is needless to say that when special external causes, such as odors or exhalations, or undiscoverable peculiarities of location are responsible, they should be eliminated. With all our efforts, however, the cause remains in perhaps a decided majority of cases undiscovered, but the theory of asthma being an anaphylactic phenomenon gives hope that the cause in individual instances may be discovered and its simple avoidance be all the necessary treatment. But even under these circumstances we have in the iodid of *potassium* and *belladonna* two drugs which possess undoubted power to relieve bronchial asthma and even to avert attacks. A certain measure

of relief is almost always secured by these drugs, and in many cases the effect is magical. From 5 to 10 grains (0.33 to 0.65 gm.) of the iodid, and 3 to 7 minims (0.2 to 0.5 c.c.) of the tincture of belladonna should be given every three hours until relief is permanent. A combination of tincture of belladonna, tincture of hyoscyamus, tincture of cannabis indica and deodorized tincture of opium, equal parts, given in 20 drop doses every two hours, signally efficient in cases of asthma. The hypodermic injection of a combination of hyoscin and atropin, 1/200 of the former and 1/300 of the latter, has been found useful in the wards of the University Hospital.

The *fumes* of burning *paper impregnated with nitrate of potash* and *stramonium* are also useful adjuvants, and cigarettes and pastiles made out of such paper are constantly employed for their effect. These substances form the basis of most of the advertised remedies for asthma.

The *diet* of asthmatics should be exceedingly simple, as indiscretions in it are often the exciting causes of attacks. No fixed rules for climatic treatment can be laid down, as conditions favorable to different cases are exceedingly capricious. Certain patients can live comfortably in one part of the same country and not in another, possibly because in the "bad" climate they are "intoxicated" by the substance to which they are sensitized. On the whole, high, dry *climates* are most suitable for pure asthmatics—*i. e.*, those cases uncomplicated with emphysema—though moist, warm climates, such as those of Florida and Madeira and the Canary Islands, are also serviceable, especially when there are catarrhal symptoms; or southern California, where the climate is also warm and equable, but drier. When there is emphysema, high altitudes are not well borne. Cold and moist climates are harmful. Oxygen breathing is often helpful, as is also inhalation of compressed air in the pneumatic cabinet. The final treatment of cases eventuating in emphysema is that of emphysema.

CHRONIC INTERSTITIAL PNEUMONIA.

SYNONYM.—*Cirrhosis of the Lung.*

Definition.—A chronic inflammatory disease consisting in a gradual invasion of a lung by fibroid tissue, with a corresponding reduction in the vesicular structure of the lung. According as it involves limited or more extensive areas it is local or diffuse.

Etiology.—Interstitial pneumonia is mainly a secondary affection. There are few chronic affections of the lung which do not cause a certain amount of fibroid overgrowth. Especially is this true of tuberculosis and bronchopneumonia. A form of the latter is the so-called *pneumoconiosis*, a fibroid induration succeeding a bronchopneumonia due to the irritating effects of minute particles arising in the occupations of coal-mining, stone-cutting, steel-grinding, and iron-working in general. To the form associated with tuberculosis the term fibroid phthisis is applied, and it will receive separate consideration. The seat of a healed tuberculosis is also occupied by fibroid tissue, which may be regarded as an example of interstitial pneumonia. Less frequently it succeeds croupous pneumonia as fibroid induration, which has been considered on page 253 and constitutes an important product in pleurogenic pneumonia mentioned

on page 253. Even abscesses of the lung may excite it, while the various forms of morbid growths, as sarcoma, carcinoma, chondroma and hydatid cysts, are causes of it, and are surrounded by fibroid growths. Especially does the fibroid change occur in a lung that has been long in a state of compression, as by a pleuritic effusion. Since the majority of cases of chronic interstitial pneumonia are directly or indirectly the result of microbic agents, it has appeared to us best to retain its consideration in this section, even though some cases may be due to other causes.

Morbid Anatomy. *Pathological Histology.*—In bronchopneumonia the fibrosis usually starts from the outer sheath of the bronchi, invading the alveolar walls and converting the entire lobule into grayish fibroid tissue, in which no lung structure is distinguishable. This form is frequently associated with dilated bronchus, of which the fibrosis is probably the direct cause, its contraction drawing the walls apart.

The line of demarcation between interstitial pneumonia on the one hand and tuberculosis on the other is often not very sharp. In interstitial pneumonia after croupous pneumonia a gradual organization takes place of the fibrinous plugs in the air-vesicles; the alveolar walls themselves become thickened by a new formation, at first cellular and subsequently fibrillated. Death usually occurs in these cases in one to three months after the onset of the disease. The whole of the part primarily invaded may become thus altered.

Macroscopic Morbid Anatomy.—The chest-walls of the side affected are often depressed, and on opening the thorax, the lung, or as much of it as is involved, is found retracted; it may be drawn back into the spinal gutter. If on the left side, the heart may be retracted with it. Commonly the two pleuræ are found united, but not always. On section the lung is hard and tough. It is gray, fibrous, and the alveolar structure has, to a varying extent, disappeared. The bronchi and the blood-vessels, however, remain, the former being often dilated, to produce the so-called bronchiectatic cavity, of which there may be a number. The pulmonary artery may be atheromatous. In the phthisical variety there may also be a cavity at the apex, and a recognition of this before death will be an aid to diagnosis. Otherwise a careful study is often necessary to distinguish the two varieties, unless the tubercle bacillus has been found.

The uninvolved lung is usually enlarged and emphysematous in proportion to the degree of contraction of the affected lung. The right ventricle of the heart, which has increased work imposed upon it in forcing the blood through the contracted lung, becomes hypertrophied and may become ultimately dilated.

Symptoms.—The principal symptom is *cough*, which starts with the condition causing the fibrosis and continues to the end. It varies greatly in its severity, being sometimes trifling, at others very troublesome. The *expectoration* is as variable as the cough; more copious as the cough is more troublesome. Persons thus affected have the appearance of delicate health, and are commonly regarded as phthisical, although they have often considerable strength and can pursue some occupation. In nontubercular interstitial pneumonia there is less *fever* than is present as a rule in phthisis, but the recognition of the tubercle bacillus is the crucial test, for otherwise

the symptoms are very similar. In both conditions there is paroxysmal cough, with copious expectoration of mucopurulent matter. The resemblance is still more close if there is bronchiectasis, when the usual emptying of the cavity by cough takes place, commonly in the morning, sometimes twice a day, and even oftener. The expectorated matter of the bronchiectatic cavities may be fetid from decomposition. There is usually less dyspnea than in true phthisis, and except where the disease is the sequel of true pneumonia, the fatal termination is longer deferred than in tuberculosis—it may be for years.

Physical Signs.—The chest is more or less retracted, its circumference diminished. Its movements are restricted and its topography altered. When the left lung is extensively affected, the heart is pulled far to the left. Retraction of the right lung causes the heart to be drawn over the right side of the chest. In high degrees of the disease the shoulder is drawn down and the spinal column laterally curved, just as in recovery after empyemic pleurisy. The unaffected side is more prominent than in health, due to compensatory emphysema. The tactile fremitus may be diminished or increased according as the pleural membrane is thickened or not. The same is true of vocal resonance. *Percussion* generally elicits impairment of resonance over the affected lung, though there may be high-pitched tympany and even amphoric resonance over a dilated bronchus. The lung on the sound side furnishes hyperresonance. To *auscultation* the breathing sounds may be feeble, but there may be bronchovesicular or bronchial and even amphoric breathing of the most intense kind on the affected side. The other side gives the signs of compensatory emphysema.

There is usually sharp accentuation of the second pulmonic sound because of the forcible effort of the right ventricle to push the blood through the contracted lung; and when the right ventricle begins to yield, cardiac murmurs may develop at the tricuspid valve.

Diagnosis.—Chronic interstitial pneumonia is mainly to be distinguished from fibroid phthisis, which is often impossible without an examination of that sputum for bacilli. The history and duration of the case may be of assistance. The fact that the right lung is contracted may be assumed by the physical.

Prognosis.—Recovery is impossible, yet cases last many years—ten, fifteen, and even longer.

Treatment.—As intimated, treatment for the fibrosis is unavailing, though lung gymnastics should be practiced with a view to developing lung expansion. Intercurrent bronchitis may be helped by the usual remedies for that disease. Antispasmodics, belladonna, and hyoscyamus are often useful adjuvants to the cough medicines. Patients are generally better in summer and in a warm climate, where they should dwell, if possible. They should be fed with an abundance of rich, nutritious food, and surrounded by the most favorable hygienic conditions.

EMBOLIC PNEUMONIA.

Definition.—An embolic pneumonia is a pneumonia caused by an embolus or, more rarely, by a thrombus in smaller branches of the pulmonary artery. Embolic pneumonia is either nonseptic or septic.

Embolic Nonseptic Pneumonia.

Synonym.—*Hemorrhagic Infarct of the Lung.*

Etiology.—The nonseptic hemorrhagic infarct of the lung is the result of nonseptic embolism, or thrombosis. The emboli come from the right side of the heart, where they either originate as fragments of thrombi, often found in the tips of the auricles, or have entered from the systemic veins. Emboli usually lodge at the bifurcation of the branches of the pulmonary artery. The usual transudation of blood takes place in a cone-shaped area. Not every embolus is followed by an infarct. An embolus may be so large as to cause death before an infarct can be formed. Nor is every hemorrhagic infarct followed by a pneumonia. The ultimate consequences of non-infectious emboli depend on their size. A large embolus and a corresponding infarct with free extravasation of blood are liable to be followed by gangrene of the lung, which may excite intense reactive inflammation in its neighborhood, and the aspirated blood may cause pneumonia. When the lodged particle is small, the hemorrhagic infarct is small, and the transudate is a diapedesis rather than a hemorrhage. From this, true embolic pneumonia results only when there is no collateral circulation—that is, when it is supplied by an end-artery.[1] When hemorrhagic infarct is caused by thromboses, the thrombus is commonly preceded by pulmonary endarteritis. They are most common in diseases of the heart especially the myocardial conditions where there is an extremely weak heart muscle.

Morbid Anatomy.—The infarct thus caused is conical in shape with its base toward the pleura, and varies in size from that of a cherry-stone to that of a hen's egg. The pleura over the infarct at first projects above the surrounding surface, and is at first smooth, but later is roughened by a film of lymph. The infarct when recent is dark reddish-brown in color, and on section rises also above the surrounding surface.

This transudation is the preliminary of a peculiar reactive inflammation—the embolic pneumonia under consideration. Succeeding a slight preliminary contraction there takes place an immigration of leukocytes from the contiguous vessels which accelerates the reabsorption of the blood. To the disintegration and absorption of the red blood-disks succeed a more rapid paling and contraction, until no color remains, or there may be a hardening of the pulmonary tissue, with a cicatricial-like contraction, into which the pleural membrane is drawn, producing fibroid thickening with radiated prolongations. Such hardening is partly due to a condensation of the lung and partly to an organization of the cells in the infiltrated alveolio and alveolar walls. Such remnant is slate-gray from the residue of hematin derived from the extravasated blood, or it may be dark red, owing to hematoidin crystals throughout it. If the infarct is large, a part may break down into reddish inodorous pulp, which may be absorbed, or a part may make its way into a bronchus and may be expectorated. In the event of so large an infarct the residue of cicatricial tissue is larger. Caseation and calcification of the remains are possible results.

The embolus itself is in like manner removed, a few filaments or slight wrinkles in the walls of the vessel being the sole residue.

[1] All the large branches of the pulmonary artery are end-arteries, and many of the smaller branches also. The reader is referred to W. H. Welch's article in Clifford Allbutt's System of Medicine, vol. vi.

Symptoms.—There may be no symptoms, or these may be confined to a transient *pleuritic pain* in the pleura covering the embolus. With increase in size of the infarct such pain increases, and may be associated with some *shortness of breath*, due to destruction of the aerating surface. There is often marked collapse. To this may be added *expectoration of blood* if the effused blood gets into the bronchus. If the infarcted area be sufficiently large, there may be dullness on percussion, increased vocal fremitus and resonance, crepitant and subcrepitant râles, bronchial breathing, and bronchophony. Further characteristics are the absence of fever and suddenness of onset and the presence of intravascular disease. It has been mentioned that the embolus may be so large, and cut off so large a supply of blood to the lung, that death will take place before an infarct can form. Jaundice has sometimes been noted, probably hematogenetic in origin, a consequence of the extensive blood destruction.

Diagnosis.—Embolic nonseptic pneumonia is often overlooked. The foregoing symptoms, suddenly occurring in connection with states leading to thromboses in the veins or the right heart, may be suspected to be due to nonseptic embolic pneumonia. Infarcts that form in the lung from non-infectious emboli arising in the *left* heart or arterial system must be so small as to escape detection, since the emboli themselves must be so small as to pass through capillaries into the veins, thence into the right heart, and thence to the lung.

Prognosis.—The prognosis of nonseptic embolic pneumonia is favorable unless the embolus is so large as to stop up a large vessel, producing a correspondingly large infarct. An embolus plugging one of the largest branches of the pulmonary artery is fatal before an infarct can form.

Treatment.—Nothing can be done actively to relieve an embolic pneumonia of this kind. A patient in whom it is suspected must, of course, be kept absolutely at rest. Counterirritation may be applied to the chest-wall over the area involved. Anodynes should be used to a degree required to relieve pain.

Embolic Septic Pneumonia.

Synonym.—*Metastatic Abscess.*

Etiology.—The cause of septic pneumonia or metastatic abscess of the lung is a septic embolus. Such a septic embolus may originate in a thrombus at a seat of putrid inflammation or suppuration, such as the wound of an operation or a compound fracture, or in the uterus after child-birth. The veins of such a focus are filled with thrombi, which extend into the larger branches, where they soften and break up into fragments, some of which may pass into the right heart, thence into the pulmonary artery and its branches, until one is reached small enough to resist its further transit. Such an embolus, which is probably swarming with bacteria, is an intense irritant, and inflammation sets in that invariably terminates in abscess, as contrasted with the simple indurative irritation caused by a nonseptic embolus. Thus caused, septic pneumonia is one of the anatomical features of pyemia.

Morbid Anatomy.—Should it be our fortune to see this form of pneumonia in its first stage, the same dark-red color as that seen in the hemorrhagic infarct of nonseptic pneumonia may be noted except that the blood extravasation is more copious. Such extravasation is a further irritant, and soon an intense inflammation sets in, which may also be divided into two stages. In the first stage the alveolar spaces and the connective tissue of the alveolar and infundibular walls are infiltrated with pus-cells. The latter furnish a white-gray ground, on which may be seen, with the naked eye, delicate red lines and circles, which represent infundibula whose vessels are still pervious to blood. In the next stage abscess-formation rapidly succeeds when the hepatized area melts into a creamy pus, in which float a few fragments of elastic tissue representing broken-down alveolar walls and blood-vessels. The abscesses thus produced may be multiple, but are mostly of small size. If the abscess is subpleural, there will be suppurative pleuritis with empyema, and possibly perforation of the lung.

In case a very large vessel is obstructed and a corresponding part of lung cut off, say a fifth of a lobe, the area thus deprived of pulmonary arterial blood is rapidly filled from the veins, and a condition analogous to a hemorrhagic infarct occurs, to the border of which the inflammation is confined, where finally the necrotic mass is dissected loose.

Symptoms.—The symptoms are those of pyemia (see p. 161), of which the lung abscesses form a part. A *chill* succeeding a surgical operation, or occurring during the lying-in state, followed by *sweating* and *high fever*, are significant symptoms. Where there are symptoms as pain, dyspnea, etc., referable to the lung itself, successions of these are even more conclusive.

PLASTIC OR FIBRINOUS BRONCHITIS.

Definition.—This is a rare form of inflammation of a part of the bronchial tree, commonly chronic, but occasionally acute, in which a fibrinous mold of the bronchus and its branches is formed and expelled. It does not include those instances which occur in croup or diphtheria as an extension downward, or in pneumonia by centripetal extension.

Etiology.—No definite cause for this bronchitis is known, though it is frequently associated with tuberculosis—in 10 out of 21 cases studied by Model. It occurs at all ages, and though more common between 10 and 30, has occurred at 72. It has happened in more than one member of a family. It is found more commonly in males and in the spring months. Other associations named are probably accidental, as with skin diseases. In the chronic form, which consists in recurring attacks extending over many years, the same part of the bronchial tree is apparently attacked each time.

Morbid Anatomy.—As primarily expectorated, the exudate is a round mass mixed with blood and mucus. This mass, sometimes, quite large, may be unrolled, when it is found to be a true cast, of dendritic shape and hollow interior, of the trunk and branches. The latter may even terminate in bulbous ends corresponding to the infundibula of the lung. The mold is true fibrillated fibrin, in which are embedded numerous leukocytes. It is whitish or yellowish-gray in color, and concentrically laminated. In the

latter feature it differs from the branching clots, which occasionally form in a bronchus and branches after hemorrhage into the lungs. These are solid and homogeneous. A fine specimen of one of these is in the pathological museum of the University of Pennsylvania. The true fibrinous casts are usually 1 1/2 to 2 inches (3.75 to 5 cm.) long, but may be five or six inches (12.5 to 15 cm.) long. The tubes whence the casts come are not superficially changed, but on minute examination have been found bereft of epithelium. The submucous tissue may be swollen and infiltrated with serum. Charcot's crystals and Curschmann's spirals have occasionally been found.

Symptoms.—These are those of an ordinary bronchitis of severe form. There are aggravated *cough* and *dyspnea*. Sometimes this is preceded by a stage in which there is, for a variable time, prolonged, bronchial *catarrh* of ordinary severity. At times the attack is ushered in by *rigor*, and there are *high fever*, *pain* in the side, and *soreness*. There is *slight expectoration* until the cast is loosened and expelled. The cough preceding the expulsion does not usually last more than a few hours, though it does sometimes continue for days. With the expulsion of the cast comes prompt relief for the time being. It is sometimes followed by slight *hemoptysis*, which may also rarely precede the expulsion. The expectoration of a single cast does not, however, terminate the attack. After 24 to 48 hours the cough and dyspnea return, and another cast is expelled. This may be kept up for several days, after which the attacks cease to recur. Smaller pieces may be expelled. The attacks may occur but once in a lifetime, or they may be repeated at intervals for years.

Physical Signs.—These are usually those of bronchitis. There is no dullness or percussion, unless it be from consolidation due to collapse of the lung. There may be, according to Walshe, circumscribed pneumonia with crepitant râle and rusty sputum. The effort at breathing is labored, and if there is obstruction of a large tubule, there may be retraction of the lower ribs during inspiration. The cast then begins to be loosened, and moist râles make their appearance.

Diagnosis.—The rarity of the disease is so great that in the absence of distinctive physical signs the true condition is rarely suspected. In recurring attacks the true nature of so severe an attack of bronchitis may be suspected.

Prognosis.—This is usually favorable, although the symptoms are often alarming. N. S. Davis has reported two fatal cases of the acute form.

Treatment.—The disease, so long as its true nature is undetermined, is treated as an ordinary bronchitis. If its true nature is suspected, the vapor from alkaline solutions should be inhaled, or these should be sprayed into the larynx. Lime-water is one of those commonly employed. Alkaline solutions may be of the strength of 30 grains (2 gm.) of sodium bicarbonate to the fluidounce (30 c.c.) of water. Jaborandi or its active principle, pilocarpin, may be tried. Emetics should also be employed when the breathing is much embarrassed. They sometimes have the effect of discharging the cast. Iodid of potassium is recommended, and should certainly be used when the attack is protracted.

EMPHYSEMA OF THE LUNGS.

SYNONYMS.—*Alveolar Ectasia; Increase of Volume of the Lung.*

Definition.—There are *three* applications of the term emphysema, and they have very different significations. In the *first* place, there is *interlobular* or *interstitial* emphysema, in which, in consequence of rupture of air vesicles deep in the lung structure, the air escapes into the interlobular tissue and may collect there like rows of beads outlining the lobules, while under the pleura larger vesicles may form. This form occurs after wounds of the lung, and in severe and persistent whooping-cough, and in cough of bronchial asthma, in both of which the expiratory strain is very great. It is also termed acute emphysema. It is not, however, demonstrable clinically, except in those cases in which it takes place at the root of the lung and the air travels along the trachea until it reaches the subcutaneous tissue of the neck and chest-walls. It gives rise to a peculiar crepitation to the touch. A similar condition of the subcutaneous tissue may be due to infiltration of the tissues, with gas arising from decomposition. It is found in the neighborhood of wounds which take on an unhealthy action, and where decomposition leads to the generation of gas. This form of emphysema is, of course, more circumscribed than that due to a wound of the lung.

The *second* form, *vesicular emphysema*, is an overdistention followed by atrophy of air vesicles, either symmetrical, involving both lungs, or localized. It occurs in certain portions of a lung adjacent to another which cannot, from some cause, expand fully in inspiration. Such are portions of the lung adjoining tuberculous areas, or areas of collapsed lung, or adjacent to parts whose expansion is prevented by pleuritic adhesions. It is particularly the anterior parts of the lung that are the seat of localized emphysema in the latter case. When such complemental dilatation is impossible, as is often the case in extensive pleuritic adhesions, the chest-wall must sink in to occupy the space. Perhaps all emphysema is more or less localized, but in general or symmetrical emphysema very much larger areas of both lungs are involved. The distended air vesicles are useless, while many of them are also atrophied. The former is also called hypertrophic, but *pseudo-hypertrophic* would be a much more suitable term, because there is no true hypertrophic enlargement.

The term "compensatory" is also applied to localized emphysema, but this term should not be applied unless the dilatation is truly compensatory—that is, is the result of an effort on the part of a lung or portion of it to supplement the office of another more or less useless part, when the condition is really developmental, and not degenerate.

A *third* form of emphysema of the lungs is known as *atrophic emphysema;* it is called also by Sir William Jenner *small-lunged emphysema*. In it the whole lung and thorax may be reduced in size, and even the respiratory muscles may be atrophied. It is a disease of old persons, and is to be regarded as an involution process. There is a true atrophy of air vesicles, and bullæ of various sizes are formed by the wasting of intermediate vesicles.

The section is limited to the consideration of

VESICULAR EMPHYSEMA—PSEUDOHYPERTROPHIC EMPHYSEMA.

Etiology.—By far the larger number of cases of emphysema are the result of chronic bronchitis. This bronchitis may begin in childhood. It may begin as whooping-cough, from which the child has not completely recovered, or succeeding which it has been subject to constantly recurring attacks of acute bronchitis. It is scarcely likely, if the lung-tissue preserved its proper integrity, that even under the forced inspiratory strain of coughing the air vesicles would undergo the dilatation and destruction characteristic of emphysema. With chronic bronchitis there is sooner or later an impairment in the nutrition of the air vesicles, which makes them more yielding and more likely to give way under the strain. Blowing on wind instruments and glassblowing, as well as occupations requiring muscular strain and the lifting of heavy weights, are assigned as causes. Bronchial asthma is another cause. In all these cases both inspiration and expiration cooperate to produce the strain, but it is probable that expiration is the more potent factor. The severe cough of chronic bronchitis begins with a deep inspiration which, while harmless to a healthy air vesicle, may overdistend a weak one. Then follow closure of the glottis and a forcible contraction of the muscles of expiration—abdominal muscles. The latter compress especially the lower part of the lung, and as the air cannot escape, it is forced into the peripheral parts, overdistending the air vesicles there. So, also, in horn-blowing and muscular strain we have the effect of deep inspiration, and especially the increased pressure during expiration, with the glottis closed. We may admit also a valve-like effect of certain plugs of mucus, which permit the entrance of air during inspiration, but do not allow its exit. Thus, the vesicles become filled with air which cannot get out. Since the air is forced in the direction of least resistance, it is the air vesicles in the apices and edges of the lungs which dilate first. This is probably one way in which expiratory strain acts in producing dilatation. The valve-like action may also be in the opposite direction, permitting the air to get out of the vesicles, but preventing it from getting into them, and thus finally a portion of the lung becomes collapsed. The inspired air must go somewhere else, and produces what may be called a collateral dilatation.

The vesicles thus overdistended finally lose their elasticity, like an overdistended india-rubber air balloon, which, after repeated distentions, loses its power to recoil. Succeeding the overdilatation comes atrophy of the vesicles, and with this the blood-vessels surrounding them are destroyed. Although under these circumstances the lung occupies more space, its blood-aerating power is diminished. The circulation is cut down to the larger trunks, and the blood takes a short cut, as it were, from the pulmonary arteries to the pulmonary veins. The aeration of the blood is thus rendered difficult or impossible, accounting in part for the dyspnea.

There is also reason to believe that heredity plays a decided rôle in the causation of emphysema, and that congenital defect often takes the place of acquired nutritive retrogression. This was first shown by the late James Jackson, of Boston, who found that in 18 out of 28 cases one or both parents were affected. Accordingly, too, emphysema is surprisingly common in children, and in adults may often be traced back to childhood.

Morbid Anatomy.—The emphysematous chest is often highly characteristic, in that the anteroposterior diameter is greatly increased, making the two diameters nearly or quite equal, producing the "barrel shape." On opening the thorax in an adult the cartilages are found calcified, and on raising the sternum the greater volume of the lungs at once shows itself. They are in a state of permanent distention, meeting by their edges in the mediastinal space and almost or entirely covering the pericardium. Nor do they collapse when removed from the chest.

The individual air vesicles are not only dilated, but large numbers of them are atrophied, producing bullæ of various sizes, from the walls of which extend inward partition which are the remnants of vesicles, so that the large vesicle has been aptly compared to a frog's lung with its semipartitions. The pleura is pale and the lungs are especially so, partly from atrophy of the pulmonary capillaries which accompanies the destruction of the vesicles, associated with diminution in the natural pigment. The lung surface pits readily on pressure. The distention and destruction are not limited to the periphery of the lungs, but are also found in the center and toward the root, where large bullæ, two to three inches (5 to 8 cm.) in diameter, may be found.

The bronchi exhibit the changes already described under Chronic Bronchitis and Bronchiectasis. An important anatomical change is *hypertrophy and dilatation of the right ventricle of the heart*, due to the extra effort required to drive the blood through the diminished vascular area in the lungs. In the later stages the hypertrophy has given way to dilatation, and there may be relative insufficiency of the tricuspid valve with dilatation also of the right auricle. In a few cases there is hypertrophy of the whole heart. There is sometimes, also, atheroma of the pulmonary artery and of the other blood-vessels, or there may be associated pulmonary tuberculosis of the fibroid variety, as well as Bright's disease.

Symptoms.—The typical emphysematous subject may often be recognized by his peculiar *round-shouldered stoop* and *barrel-shaped chest* and *short neck*. Rarely, this form of emphysema is an acute or comparatively rapid development, succeeding whooping-cough; but the approach of the disease is mostly gradual, the first symptom to develop and remain constant being *shortness of breath*, which is partly due to the fact that the air in the vesicles does not undergo the usual interchange. In health the intercostal muscles, the diaphragm, and auxiliary muscles of respiration enlarge the thoracic box, and the lungs expand to fill it partly by their own resiliency, but chiefly to fill the vacuum, producing the act of inspiration, while the air is expelled in expiration partly by the recoil of the elastic tissue and partly by the pressure of the contracting thorax. This natural resiliency is absent in a large degree, while the thoracic box also remains in a state of "rigid dilatation." The lung is always filled with air, but it is air charged with carbonic acid and does not change. As a consequence the patient makes increased efforts to draw the air into the lungs, but as the air vesicles are already filled, these efforts are ineffectual. The *dyspnea*, which is but slight at first and is brought about only by exertion, soon becomes decided and constant. The *pulse-rate* is also accelerated, but the temperature is usually

normal. *Cyanosis* is a characteristic symptom in established cases, owing to the universal presence of unaerated blood.

Aside from these symptoms are mainly those of the associated bronchitis—viz., *cough*, *expectoration*, and sometimes *oppression*—while variations in these add to or abate his discomfort. With failure of the right heart come venous engorgement, dropsy, and effusions into the serous sacs. Tuberculosis of the fibroid type sometimes develops.

Physical Signs.—The physical signs are not always distinctive. *Inspection* reveals a rounded chest, with increased circumference and wide intercostal spaces in the hypochondriac regions, but narrow above. The epigastric angle is obtuse. The result is the well-known "barrel-shaped" chest. More rarely the emphysema may be so circumscribed as to produce local bulging, by preference over the upper lobe of the right and lower lobe of the left lung. Expansion of the chest-wall is diminished, while the scaleni and sterno-cleido-mastoid muscles stand out distinctly. The chest does not expand, but is raised up by these muscles, which are hypertrophied; the apex-beat is not visible, but may be felt displaced downward and to the right, and is often difficult to find, because covered up by the enlarged lung. The breathing is rapid. There may be retraction of the lower intercostal spaces and the upper abdomen instead of swelling out during inspiration, because of failure of the diaphragm to descend. Vocal fremitus is diminished, while the natural resiliency of the chest-walls is substituted by increased resistance.

Percussion produces resonance exaggerated in various degrees, sometimes amounting almost to tympany, the result of the overdistention of the air vesicles, whose elasticity is spent. To *auscultation* vocal resonance is decreased because of the diminished vibration in the air columns. Expiration is much prolonged and of low pitch, being difficult to hear unless there are accompanying râles. Feeble crackling is sometimes heard. Strumpell says the vesicular murmur is at times exaggerated and "shuffling," at others "rougher and more indefinite." Roughness and exaggeration seem impossible in true emphysematous areas. They may be present in adjacent supplementally acting areas. If bronchitis is present, its sounds are associated, and often obscure all else. The pulmonary second sound at the second left interspace is accentuated on account of the hypertrophy of the right ventricle, but the heart-sounds are usually obscured by the extra covering of the lung. With dilatation of the right ventricle, which sooner or later succeeds, the increased accentuation disappears.

Interlobular emphysema, in which the connective tissue between the lobules is infiltrated with air as the result of rupture of air vesicles due to violent acts of coughing or by wounds of the lung, affords no physical signs, indeed rarely any symptoms. The shape of the chest in such cases is not altered. Suddenness of onset is characteristic of this form of emphysema, and it is apt to be associated with a similar infiltration of the tissues of the neck, which gives rise to a very distinctive crepitation on palpation.

Diagnosis.—This is not usually difficult, at least in true symmetrical emphysema. In *pneumothorax* there is some simulation of the symptoms of emphysema. There is the same shortness of breath, and there is some resemblance in the physical signs. There is bulging of the chest, which is,

however, more marked on one side than on the other. Pneumothorax is unilateral, emphysema usually bilateral. The parts of the chest more likely to be affected with emphysema are the upper part of the right lung and the lower part of the left. Also in the matter of percussion, emphysema gives hyperresonance. Pneumothorax gives more marked tympany. The hyperresonance, although often marked, is not always so. The unyielding chest-walls modify it. The sound in pneumothorax is a real tympany, comparable to that obtained over the distended abdomen. In both, the thickness of the chest-walls exerts a modifying effect. Maybe effusion, which gives flatness on percussion, and a line of separation between tympany and flatness is demonstrable. Pneumothorax is sudden in its occurrence, whereas emphysema develops gradually. It is, however, not impossible for the two affections to be combined. The heart is displaced in pneumothorax and not in emphysema.

There is still another condition with which emphysema may be confounded, though it is of rare occurrence; I refer to *diaphragmatic hernia*, in which tympanitic resonance is a striking symptom.

Prognosis.—This, except in cases of acute emphysema, which heals spontaneously, is unfavorable as to cure. The course is always a chronic one, and much may be done for the comfort of the patient. No classes of cases are so benefited by admission into hospitals as members of the laboring class afflicted with emphysema.

Treatment.—It is impossible, to restore destroyed lung texture. If a number of air vesicles have been converted into one sac or bladder-like cavity, there are no means by which these vesicles can be restored. At the same time, when the patient is young, there is some hope of cure if the structural loss is not too great. Effort must be directed mainly to averting those conditions which complicate and increase the emphysema. As has been said, chronic bronchitis is its most frequent cause, and, therefore, we to relieve this condition by every means in our power. As the general health must try is often impaired, it is as important that this should be reestablished as that the bronchitis should be relieved. The blood is to be restored to a proper composition by tonic remedies, like cod-liver oil and iron, and the very best food that the patient can procure. To the cod-liver oil and iron should be added strychnin in full doses, 1/30 to 1/32 grain (0.0022 to 0.0055 gm.), while arsenic is an admirable tonic either in the shape of Fowler's solution, 5 drops at a dose for an adult, or of arsenious acid, 1/30 grain (0.0022 gm.).

While the bronchitis is treated by the usual remedies, it is of the utmost importance that the stomach should be kept in good condition, and that digestion should not be interfered with, while more than ordinary care is required in the selection of remedies for the bronchitis.

Strychnin is an admirable remedy, not only as a tonic, but it may also be regarded as an expectorant, and secretions in the lungs are often disposed of by its use. It has also the effect of improving the nutrition of the muscular tissue of the walls of the bronchi, as it has of improving the muscular tissue in general. Full doses should be given—not less than 1/60 grain (0.001 gm.), three times a day, increased gradually to 1/30 grain (0.002 gm.). This is to be kept up for a long time.

Bronchial asthma is one of the most serious and frequent complications, and often overshadows all else. The treatment recommended on previous pages must be employed.

To relieve the constant dyspnea, the treatment suggested some years ago by Waldenburg is one the usefulness of which is only limited by its relative difficulty in application and the costliness of the necessary apparatus. It consists in the inspiration of compressed air and the expiration into rarefied air. It is evident that if compressed air can be introduced into the vesicles, the aeration of the blood will be more perfect, and that if the patient breathe into rarefied air, the residual air, which it is so difficult to get rid of, will be more effectually forced out. The compressed-air chamber has a similar purpose.

Expiration may also be aided by compression of the chest, intermittently applied so as to coincide with natural breathing. This must usually be practised by a nurse or an attendant, but Strümpell describes in his text-book a simple contrivance devised by a patient of his own for self-treatment. It consists of two boards fastened behind and allowed to project forward on each side in front, so that the patient himself, taking hold of the projecting ends, can compress his own chest with each act of expiration. Removal to other climates suggested in chronic bronchitis should be carried out where possible. As the heart begins to fail in emphysema before the end in practically all cases, great care must be taken that overexertion be not indulged in. All the care must be given to cardiac decompensation in these cases as is advised under diseases of the heart.

TUMORS OF THE LUNG.

The lungs are subject to morbid growths classified as tumors, though, owing to their situation, they rarely present the macroscopic, tumor-like qualities.

They include carcinoma, and many of the histoid tumors.

Etiology and Morbid Anatomy.—*Carcinoma* occurs rarely as a primary growth, but is not infrequent as a secondary new formation. Primary cancer presents itself usually in the shape of a white or yellowish nodule two to four inches (5 to 10 cm.) in diameter. It is found in the upper lobe of one lung, posteriorly and externally; more seldom in other parts. It probably originates in the alveolar epithelium, and causes secondary infiltration of the bronchial glands and pleura. It may be represented by any of the three principal forms, scirrhous, encephaloid, or epithelioma, also by the colloid and melanotic. It occasions a reactive pneumonia in the lung tissue about it, and often furnishes the physical signs of this affection.

There also occurs in the lung a primary *peribronchial* cancer, disseminated in nodules throughout the lung along the bronchi, smaller nodules on the smaller bronchi, and larger, irregular masses on the larger, varying in size from that of a pea to a walnut. It produces also infiltration of the lymph glands at the root of the lung. *Sarcoma* is also a rare form of primary tumor of the lung.

More frequently both carcinoma and sarcoma are found in the shape of secondary nodules invading both lungs. From three to 20 opaque

white nodules, 1/2 inch (1.25 cm.), more or less, in diameter, are found irregularly scattered through each lung. Every variety of primary cancer may be thus represented secondarily in the lung. Its origin is probably embolic, and it may be secondary to cancer elsewhere, most frequently in the breast.

As elsewhere, these growths generally present themselves after middle life, primary cancer affecting either sex about equally, while secondary is more common in women, consistently with the more frequent occurrence of cancer elsewhere in women.

The histioid tumors are represented by a subpleural enchondroma, occurring, rarely, primarily as large as a walnut; more frequently, secondary to occurrence elsewhere, when it may attain a large size. Other histioid tumors are myxoma, adenosarcoma, dermoid cysts, fibromata, osteomata, and gummy tumors.

Symptoms.—Carcinoma and sarcoma may both be latent, or at most produce such vague symptoms that it does not occur to physician or patient to locate them. There may, however, be *pain*, *oppression*, *cough*, *expectoration*, and superficial signs of *vascular obstruction*, such as lividity of the face and swelling of the upper extremities. The encroachment of the larger cancerous masses upon the pleural cavity may be marked. Pressure on the trachea and bronchi may occur and occasion great *dyspnea*, while the heart may be dislocated. The pneumogastric and recurrent laryngeal nerves are sometimes involved, occasioning the various forms of paralysis of the vocal cords and aphonia. The reactive pneumonia referred to may present the physical signs distinctive of this disease, and it is probably thus that the prune-juice expectoration, though to be quite characteristic of cancer of the lung—10 times out of 18, as elaborated by Stokes many years ago—originates. This complication, too, may occasion the fever which is sometimes present.

The external lymphatic glands, as those in the neighborhood of the clavicle, may be involved and exhibit enlargement.

Sooner or later, if the patient lives long enough—that is, if his life is not destroyed by some encroachment on the breathing or vascular function—he emaciates, and becomes cachectic and debilitated. The more usual duration of the disease is from six to eight months, but death is liable to occur suddenly from the causes named.

Physical Signs.—These, of course, are indefinite, and it is probably their indefinite and irregular manifestation, with the symptoms named, which will suggest the nature of their cause. Physical signs of pneumonia and pleurisy, either alone or combined, may be present, the voice and breathing sounds and percussion note being affected accordingly.

Diagnosis.—Secondary cancer, where primary cancer is present elsewhere, is suggested whenever any of the symptoms named occur in a pronounced degree and are sufficiently long continued. In the case of primary growths, the diagnosis must longer remain doubtful, and we must study and await the development of the more distinctive symptoms.

The nonmalignant tumors present no signs by which they can be distinguished from the malignant, except that their course is less rapid and they develop no cachexia.

Treatment.—This consists only in measures calculated to relieve symptoms and to make the patient comfortable.

DISEASES OF THE PLEURA.

ACUTE PLEURISY.

Definition.—Acute inflammation of the serous investment of the lung or of its reflection on the ribs and diaphragm.

Etiology.—Simple fibrinous pleurisy may be caused by an infection called forth by simple chilling of the body during exposure to cold.

Morbid Anatomy.—The morbid anatomy of pleurisy will be best understood by supposing every pleurisy to begin, as it probably does, with a dry stage, a *pleuritis sicca*, whatever may be its subsequent course. Thus considered, the earliest stage of all pleurisies has a hyperemic basis, succeeded immediately by a roughness of surface due to loosening and detachment of the epithelium, a roughness increased by the addition of fresh inflammatory lymph composed of transuded fibrin and wandered-out leukocytes from the subpleural blood-vessels. Further progress of such pleurisy is—

First, toward *resolution*, in the course of which the product described liquefies and is reabsorbed.

Second, toward *primary organization* and *adhesion*, and two surfaces of the pleura are more or less permanently glued together over an area corresponding to that of inflammation. This is the probable explanation of the little patches of adhesion so frequently found at autopsies, some of which may have formed without the consciousness or discomfort of the patient, while others have succeeded upon a "stitch" in the side which has been passed by as of little consequence. Other instances of this primary adhesive inflammation are found between the opposed surfaces of pleural membrane covering tuberculous deposits in the lung, or limited pneumonic areas, or morbid growths, such as gumma, cancers, and sarcomata.

Third, toward *serous accumulation* constituting *sero-fibrinous exudative pleurisy*, in which varying quantities of fluid are transuded into the pleural cavity. In this usually clear, straw-colored exudate may be suspended shreds of the yellowish plastic lymph already described, which accumulates also most abundantly where the movement of the pleural surfaces is least, as in the chinks and corners of the pleural cavity. This effusion also, in a large number of cases, is absorbed, allowing the pleural surfaces to approach each other and again unite by what is known as secondary adhesive inflammation, organization taking place as before, producing either continuous fusion or bands of new tissue attaching different parts of the pleural surface. The question as to how the process of exudation is stopped is an interesting one, which cannot be satisfactorily answered, though it is probable that pressure of accumulated fluid and contraction incident to organization, as well as cessation of the cause, may be a part.

The ordinary serous fluid which commonly fills the sac in serofibrinous pleurisy is a highly albuminous liquid, sometimes coagulating spontaneously, in which may be found a few leukocytes, exfoliated endothelial

cells, shreds of fibrin, and sometimes a few red blood disks. Modifications are those in which the red blood-corpuscles are much more numerous, producing a bloody fluid, or in which leukocytes are variously numerous, short of a number sufficient to justify the term pus. Urea, uric acid, and sugar are sometimes found in pleural exudates. The quantity of fluid ranges from half a liter to four liters (1 pint to 4 quarts).

Fourth, toward *pus-formation*, in which either primarily, from the outset, or secondarily—that is, some time after the process has commenced—the microbes of suppuration become active, and produce a purulent product or an empyema. The pleural surfaces thus apposed are, however, comparable to an ulcer, and the union and repair take place by formation of cicatricial tissue. This is subject to the contraction usual to such tissue, dragging not only the heart and lungs out of place, but also in extreme cases the ribs and vertebræ, producing slight lateral curvature of the spine.

Various displacements of adjacent organs are caused by the liquid effusion. In the right-sided pleurisies the liver is depressed. The heart is pushed to the left. In left-sided pleurisies the heart may be displaced so far that the apex will be to the right of the sternum. The displacements from traction after organization are difficult to describe, but the heart may be dragged so that its apex is much higher than is normal or further to the right, while the parts of lung adherent are drawn in various directions, with the production, at times, of bronchiectatic cavities. If the patient die while large liquid effusions are present, the lung is also found compressed into the back part of the pleural sac.

Symptoms.—The initial symptom of pleurisy is usually *pain*—at first in the side. It may, however, be preceded by a *chill*, and at times there may be a short *prodrome of discomfort* in no way peculiar. The pain in bad cases is of the severest kind, and among the pains most difficult to relieve. It is sharp and cutting in character, aggravated by breathing, so that the patient takes the shortest breath possible, and the breathing is made up of short, hurried gasps. Cough likewise causes agonizing pain, and it is accordingly restrained. Nor is the pain in these cases always confined to the chest, but may shoot down into the abdomen and back. The latter probably implies that the diaphragmatic pleura is involved. *Fever* is also a constant symptom, but is not, as a rule, so high as in pneumonia. At the beginning the temperature may be 102° or 103° F. (38.9° or 39.4° C.), but as a rule it subsides early, even though the other symptoms abate but partially, and under any circumstances it falls much lower after a week or ten days unless there is purulent exudate, when the fever assumes a hectic type. The *cough* is peculiar enough to require special mention. It is a short cough, attended with little expectoration, and is a much less conspicuous feature than in pneumonia. Its characteristic shortness is due to the pain caused by the act of coughing, on account of which the act is cut short. The *decubitus* of pleurisy is quite constantly on the affected side, in order that the unaffected side may be free to expand. This pertains to pleurisies associated with copious effusions, as well as dry pleurisies.

While the majority of pleurisies begin in this way, a certain number also begin insidiously. For days and even weeks the patient, while feeling uncomfortable and doubtless feverish and slightly dyspneic, continues

his occupation, and even when the physician is called, scarcely mentions symptoms which suggest an examination of the thorax. Such pleurisies are known as *latent* pleurisies. They are latent only to superficial observation. Closer investigation promptly reveals the physical signs of a pleural effusion.

It has already been mentioned that purulent pleurisies may be primary or secondary. In any event, they are most frequently tubercular, and an examination of the pus from such a pleurisy not infrequently discovers the tubercle bacillus in it.

Physical Signs.—Acute pleurisy is also resolvable clinically into three stages, each of which is characterized by physical signs more or less distinctive. They include a dry stage, a stage of effusion, and a stage of resolution or absorption.

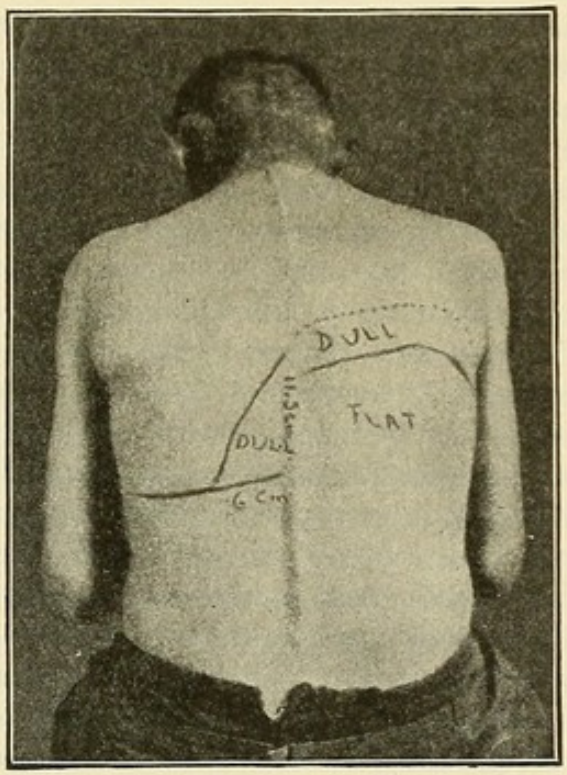


FIG. 107.—Grocco's Sign. Paravertebral Triangle of Dullness on the Left. (*After Thayer and Fabyan.*)

The *first* or *dry stage* is characterized anatomically by the presence of the so-called lymph or exudate on the pleural surfaces. During this is revealed to inspection a restrained expansion of the affected side, often thrown into jerks or catches because of pain suffered in a continuous inspiration. The expansion on the opposite side is full and unhampered. The patient lies on the affected side. Palpation may recognize a fremitus corresponding to the friction of the two pleural surfaces. Percussion in this stage is negative, except that it may cause pain, but auscultation recognizes the friction sound, which will be further characterized in treating diagnosis. It may be at a single spot in the inframammary or infraaxillary space, and hence be overlooked. At other times it may be noted over a considerable area. According as the inflammatory process stops here with resolution or continues into the second or stage of effusion, there may or may not be other signs.

The signs of the *second stage* vary with the amount of liquid in the sac; with a small amount the lung is slightly floated up, and there may be no signs, unless it be a vesiculotympany above the line of the fluid, a Skodaic resonance by mediate relaxation of the air vesicles. The effusion, however, rarely remains so trifling, but commonly rises to the midchest. In the upright position of the patient inspection recognizes in a spare person shallowness and perhaps obliteration of the lower intercostal spaces, and bulging of the chest on the side affected. The motion of the chest-wall is lessened both in the vertical and transverse directions.

To palpation vocal fremitus is diminished over the area of effusion, but may be increased in the lung above it. To percussion there is absolute flatness over the area of effusion, but the line of demarcation is not everywhere at the same level, being higher behind than in front. Calvin Ellis first called attention to an S-like curve in the line of demarcation which is said to be diagnostic. Very important in the diagnosis is the fact that the fluid changes its level when the position of the patient is changed, and correspondingly the line of dullness is altered. This change of level does not occur when the chest is completely full of liquid. There is also an abnormal sense of resistance to the finger in percussing over the area of effusion. Above the effusion, especially anteriorly, there is again Skodaic resonance by mediate relaxation, and even rarely a "cracked-pot" sound. Tympany may also be due to the proximity of a distended stomach. When the effusion is in the left side the stomach tympany is abolished or diminished. Measurement discovers that the circumference of the affected side is a centimeter (0.4 in.) or more greater than that of the other side.

To auscultation the breathing sounds are inaudible or very feeble, as compared to the corresponding portion of the opposite side, but vocal resonance, though diminished, is still distinctly heard where the collection of fluid is moderate. Baccelli called attention to the fact that the whispered voice is transmitted through a serous but not through a purulent exudate. He advises direct auscultation in the antero-lateral region of the chest. This transmission of voice and breath sounds is often confusing, but where they are present over an effusion there is *no* fremitus and the heart is dislocated. Above the line of dullness there is occasionally a friction sound, and close to the root of the lung bronchial breathing may be heard. This is, however, more apt to be the case when the effusion is larger and the lung is further compressed. Egophony is also sometimes heard over a thin layer of effusion.

When the effusion is larger, filling up two-thirds or three-fourths of the pleural sac, the effects described are increased, while new ones are added. Inspection notes that respiratory movement is still more hampered, that the intercostal spaces are widened and even bulging, while fluctuation may sometimes be recognized through them. The heart is displaced by the accumulated fluid, and if the fluid be in the left sac, the apex is often found far over to the right of the median line, and if in the right, the apex may be pushed further to the left. The heart sounds are not, however, altered. On the opposite side the breathing movements are supplementally increased. There is complete absence of vocal fremitus on the affected side.

Percussion is absolutely flat all over the effusion, and Skodaic resonance is now not obtainable, because the lung is too thoroughly compressed up into the apex of the sac. Resistance to pressure is marked.

Paravertebral Triangle of Dullness in Pleural Effusion (Koranyi-Grocco's Sign).—In 1897, Koranyi of Buda Pesth pointed out a valuable sign of pleuritic effusions, a triangular paravertebral area of dullness on the side opposite that of the pleuritic effusion. In 1902 Grocco described the same sign as shown in the adjacent figure. It is caused by an intrusion of the pleuritic effusion across the vertebral column pushing the movable mediastinal contents, viz., the aorta, esophagus and azygos vein before it.

The presence and significance of this sign is variously estimated. It was found by Thayer and Fabyan in 30 out of 32 cases; by Frankenheimer in every one of 31; by Hermon C. Gordonier[1] in 27 out of 29, in all cases in which there was actual pleural effusion. It is not found, as a rule, in the paravertebral gutter opposite pneumonic consolidation or when there is empyema on the affected side. The strongest proof of its relation to pleural effusions is its disappearance after the fluid is removed from the affected side by tapping. It is sometimes influenced by changes of position. Thayer gives the following method of determining the triangle:

After determining by percussion the boundaries of the supposed effusion, the lower limit of pulmonary resonance on the opposite side should be marked out. One should then percuss downward directly over the spine, marking the spot at which relative dullness begins. This will be found to correspond approximately with the beginning of relative dullness on the side of the effusion or is a little higher than the limit of flatness. Then percuss downward along lines parallel with the spine and inward along lines parallel to the lower limit of pulmonary resonance. Thus one can mark out usually the mesial and inferior angle on the healthy side a triangle of dullness. The vertical side of the right-angled triangle corresponding to the line of the spinous processes reaches a point somewhat higher than the upper limit of flatness on the affected side, the base from the mesial line outward on the unaffected side ranges according to the extent of the effusion to 2 cm. to 7 cm. (0.8 in. to 2.8 in.).

The third side of the dullness corresponds with a line joining the extremities of these two lines. Thayer[2] and Fabuyan noted that this line sometimes showed a slight outward convexity.

On auscultation over large pleuritic effusions bronchial breathing may be heard at the upper posterior portion of the lung, because the large tubes are still pervious to air, and the compressed lung intensifies the sound. Sometimes bronchial breathing is heard in more peripheral parts of the chest, probably conducted hither along a band of adhesion or along a rib. Elsewhere there is absence of breath sounds. Vocal resonance and whispering voice are alike absent, or the former is very feeble. In certain situations, too, high up, where there is but a thin film between the chest-wall and the lung, there may be egophony, but this is more likely to be present as the fluid is being absorbed.

[1] Albany Medical Journal, Ap., 1909.

[2] For a thorough discussion of this sign based on a story of the literature and of 32 cases, see a paper by Thayer and Marshall Fabuyan "Paravertebral Triangle of Dullness in Pleural Effusion," Amer. Jour. Med. Sc., vol. cxxxiii, p. 14, 1907.

In the *third stage*, if resolution takes place with a gradual retrocession of the fluid and the reexpansion of the lung, we have a return to normal physical signs. There may be, too, a *friction redux*. A considerable time is, however, required for absorption, and it is often many days before the normal breathing sounds are heard with their usual intensity or the natural fremitus is felt. Often, on the other hand, resolution is not complete, and the two surfaces become glued together, constituting a *plastic pleurisy*, and the feebly heard breathing sounds and diminished fremitus and vocal resonance remain more or less permanent (chronic pleurisy). There then remain the symptoms and sequelæ of a chronic pleurisy. In cases of purulent pleurisies, if recovery takes place it is always by adhesion of the apposed surfaces. (See Chronic Pleurisy.)

In connection with the heart, *pleuropericardial friction* may be heard if the pleura covering the portion of the lung adjacent to the pericardium is involved. The apex-beat may not be discoverable if it is so dislocated as to be covered by the sternum, and it often happens that the heart must be located by its signs. In many cases the use of an aspirating needle is necessary for a diagnosis.

Varieties of Acute Pleurisy.—*Tubercular pleurisy* is a pleurisy due to the invasion of the pleura by the tubercle bacillus, and has been considered when treating of tuberculosis.

Diaphragmatic pleurisy is a painful form of pleurisy, in which the pleural covering of the diaphragm is involved, either alone or along with the remaining pleura. It is usually dry, plastic, but may also be exudative, with a serofibrinous or purulent product. The pain is low down in the thorax in the zone of the diaphragm, and is often aggravated by deglutition as well as by breathing and may be transmitted to the appendiceal area. Because of the pain in breathing, the diaphragm is fixed and the patient breathes by the upper thorax. Of diagnostic value is the fact that the pain may be increased by pressure at the insertion of the diaphragm at the *tenth rib*.

Hemorrhagic pleurisy, characterized by bloody effusion, is found in asthenic states, however induced, in tubercular pleurisy, in which event the hemorrhage occurs from the young blood-vessels, and in cancerous pleurisy; also sometimes in persons otherwise healthy. It is, of course, not to be confounded with blood-stained serum, caused by wounding a blood-vessel in the act of tapping or with a hematothorax from rupture of an aneurysm.

Encysted or *circumscribed pleurisy* is a form of purulent pleurisy in which adhesions form so as to produce loculi, or spaces which are filled with pus. They are quite difficult to recognize during life—in fact, they are commonly found when exploring the chest with the needle. More rarely they are revealed to physical examination, dull percussion areas being found in alternation with clear areas. Such physical signs should suggest the use of the needle to clear up the diagnosis. These collections sometimes pulsate and become *pulsating pleurisies*. Pulsating pleurisies are almost invariably on the left side and receive in some way the impulse of the heart, which in turn is communicated to the eye or hand of the observer. The possible confounding of these with aneurysm will be again referred to.

In *interlobular pleurisy* the apposed surfaces of two lobes of the lung

are agglutinated, and sometimes a sac of pus is pent up between them, forming a variety of encysted pleurisy. Such an abscess may break into a bronchus. It should be recognized especially when a hectic fever follows pneumonia.

Diagnosis.—The certain diagnosis of pleurisy depends almost entirely upon the physical signs, for, however severe the other symptoms, there is nothing in them by which the disease can be surely recognized. In the majority of cases of pleurisy the diagnosis is made easy by the aid of these signs. It is true there is a certain resemblance between pleurisy and *pneumonia* in the first stage of each, and in that stage a diagnosis is often difficult, especially when the physical signs are not distinct. The resemblance of the friction sound to the crepitant râle is well recognized. Indeed it is often impossible to distinguish between a pulmonary and a pleural adventitious sound. The usual distinctive features are the superficial situation and the intermittent character of the friction sound, its presence during expiration as well as inspiration, and if confined to one of these acts, rather to expiration, while the crepitant râle is heard only during inspiration. The friction sound is also usually rougher and more circumscribed, while it may sometimes be heard better with the stethoscope. Pain is very apt to be elicited in pleurisy if the stethoscope is pressed hard upon the chest. As the pleurisy becomes dry and adhesions form, the friction sound resembles more closely that of creaking leather.

In the second stage of pleurisy, too, furnishing as it does a dullness on percussion like that of the same stage of pneumonia, and frequently bronchial breathing, we have also a resemblance in the physical signs. But it is true of the bronchial breathing of pleurisy that it is commonly best heard at the upper border of the dullness and least where the dullness is most marked; whereas, in pneumonia the bronchial breathing is most intense where the consolidation is greatest. *Above all, in pleurisy with effusion there are diminished vocal fremitus and diminished vocal resonance; in pneumonia, increased vocal fremitus and increased vocal resonance.* There is commonly, further, in pleurisy with effusion, a change of level of the dullness with a change of the position of the patient, which is not the case in pneumonia. The egophonic voice is also often here present in pleurisy; whereas we have only bronchophony in pneumonia. Finally, in the differential diagnosis between acute pleurisy and pneumonia, the trifling cough and absence of expectoration in the former are valuable signs, though it must not be forgotten that in old persons there is sometimes very little cough in pneumonia.

The X-ray to be of service in the recognition of pleuritic effusion. The dense fluid cutting off the rays about as much as an organ as dense as the liver; whereas the outline of the ribs may be obliterated on the side of the effusion. The fluoroscope also enables us to recognize displacement of the heart caused by effusions, especially to the right of the sternum, when sometimes percussion does not show it. The effect of changes of position on the pleuritic effusion may also be seen by the fluoroscope as well as movements in the fluid caused by the movement of the diaphragm.

As to further differential diagnosis, pleurisy in the dry stage has been mistaken for *muscular rheumatism, intercostal neuralgia, periostitis,* and

caries of the ribs, and even *gastralgia* and *ulcer of the stomach*. The absence of fever in the first two, the circumscribed situation of disease of the ribs, and the associated history of gastralgia and ulcer of the stomach, serve to differentiate them.

The confusion of *mediastinal tumors* arising from the pleura itself with pleurisy is a natural error, especially since such tumors in their turn produce pleurisy. In pleurisy, the physical signs are commonly limited to one side, while in mediastinal tumor the fremitus is less diminished, the dullness extends upward, is more irregular, and more circumscribed; while symptoms of compression of nerves and vessels, and of encroachment on the esophagus sooner or later make their appearance. Repeated exploratory punctures may be necessary to settle the diagnosis, which, may require some time.

The impulse of a *pulsating empyema* sometimes very strongly suggests an aneurysm, but the empyema furnishes no murmurs or pressure symptoms while the location is usually different from that of aneurysm.

Prognosis.—The prognosis of acute pleurisy depends largely upon its cause. The simple pleurisies which are the result of infection due to exposure always get well, and recovery is the termination in most cases even when there is large effusion, if the exudate remains serous. It has already been said that a purulent pleurisy is often tubercular. We have learned, however, that a tubercular pleurisy is not necessarily fatal, and it is more than likely that some of the cases of healed empyema with which we are familiar are instances of such recovery. In children it is usually due to pneumococci and the outlook is favorable. The sacculated empyemas are also frequently pneumococcic. Others are cured by the introduction of drainage-tubes and exsection of ribs, but often the patient slowly succumbs to the exhausting effect of the illness or to tuberculosis of the lungs. Not a very rare event in badly neglected cases is the spontaneous rupture of such a pleurisy outward, an event better anticipated by paracentesis. Very stubborn, too, are the somewhat rarer cases in which perforation takes place from the pleural sac into the lungs, adding the symptoms of a pneumothorax to those of the pleurisy. Yet even these sometimes heal spontaneously.

Though not a frequent event, sudden death, when least expected, is sufficiently so to make it important that one should be on his guard for it. It is not alone when the chest is full, or during a tapping, that it occurs, but it may happen several days after a large part of an effusion has been removed. Pulmonary thrombosis is probably the most frequent cause. A case of Tyson's terminated thus, when convalescence was thought to be established, and the patient expressed himself better than on any day during his illness. At the necropsy, a white "chicken-fat" clot was found in the right ventricle, extending as a red clot into the pulmonary artery. The chest was partly filled with serofibrinous fluid. Edema of the opposite lung and degeneration of the heart muscle are probably causes, suggested by Wiel. Obstruction to the circulation by dislocation of the heart or twisting of the great vessels has also been suggested as a cause.

Treatment.—Many simple pleurisies doubtless get well of themselves, with, perhaps, more or less adhesion of the lung, which may be the cause of certain unexplained restrictions in expanding the chest. For very severe

cases of pleurisy, local blood-letting in the form of wet or dry cups is the promptest measure of relief, and there is no condition in which so delightful an effect comes to the suffering patient gasping for breath and racked with pain. The duration of many pleurisies is shortened by such a treatment. Strapping the chest after the manner of a dressing for fractured rib is of the greatest value. Anodynes—morphin hypodermically is the best—are often necessary to relieve the pain, and must sometimes be repeated, while We have even known repetition to be inefficient and unsatisfactory, when a blood-letting produced prompt relief.

Even where the effusion is considerable, it often passes away without any very active measures. But a fairly large pleural effusion needs tapping to prevent serious collapse of the lung. The salicylates in full doses sometimes bring about a large polyuria and rapid disappearance of the effusion. If there is much delay, however, in the absorption of fluid, *paracentesis thoracis* should be practiced as soon as the fever has subsided. It is an operation every physician should be ready to do without calling on the surgeon.

This simple operation should be done under the strictest aseptic precautions. The chest should be cleansed with alcohol, and the spot thoroughly sprayed with iodin. The instruments should be sterile, as should the hands of the operator. A trochar and canula attached to an aspirating apparatus should be used. The trochar and canula is better than the sharp needle. The latter may injure the lung. The best point for tapping is the sixth or seventh interspace in the midaxillary line. The interspaces are made wider and the operation easier if the arm of the side to be operated is carried over to grasp the opposite shoulder. After a small skin incision is made the needle should be introduced close to the uppe-margin of the rib, so as to avoid wounding the intercostal artery. Local anesthesia should be obtained by the application of ice and salt, or by chlorid of ethyl. It is particularly in the insidious forms of pleurisy that the tapping to the chest becomes necessary, because they seem to be as slow to disappear as they are slow to make their presence known. A further indication for paracentesis is aggravated dyspnea. The operation is usually well borne, though sometimes faintness results. It is, therefore, well to fortify the patient in advance with an ounce of whisky, and if faintness or cough results, to desist. Sudden death during the operation has happened in rare instances. On the other hand, sudden death has occurred more frequently in cases of full pleura without operation. When this accident occurs, it is more than likely that the heart was previously damaged.

Repeated tappings are sometimes necessary.

Empyemas almost never get well after a simple tapping. The pus reaccumulates, and the symptoms and physical signs are renewed. These cases are for the surgeon—resection of a rib is best done—though in children the insertion of a large drainage tube may be practiced.

CHRONIC PLEURISY.

Definition and Pathogeny.—Under the term chronic pleurisy are included several morbid states, the result of inflammatory processes of

longer duration than a few weeks. These include both *exudative* and *dry* or *plastic* pleurisies.

1. *Exudative pleurisies*, characterized by liquid product, include—

(*a*) The condition already spoken of as *latent pleurisy* associated with effusion.

(*b*) *Suppurative pleurisies*, all of which, though they may originate acutely, are of long duration, and may therefore be appropriately classified as chronic.

2. *Plastic pleurisies*, characterized by a dry product. These originate in two ways: First, they are plastic from the beginning—that is, the so-called lymph first deposited becomes permanently organized as a more or less thick layer uniting the pleural surfaces. Such primary adhesions are more usual in circumscribed areas of pleural surface. Second, the same result follows when the surfaces separated by the more copious seropurulent transudate reapproach each other as the latter is absorbed, producing secondary adhesions. Third, we have a most distinctive product of chronic pleurisy in the cicatricial tissue, which succeeds the healing of the extensive suppurative surfaces forming the walls of an empyema and which also closely cements the lung to the costal pleura.

Mention should also be made of the form of chronic pleurisy resulting in a thick, pleural and sub-pleural deposit slowly formed, tuberculous in origin, which grows from the pleura into the interlobular tissue of the lung, dividing it or dissecting it in extreme cases into distinct areas, well shown upon section, which has given rise to the name *pneumonia dissecans*, or pleurogenous pneumonia. This form of pneumonia has its type in the pleuropneumonia of cattle. Tyson met one striking instance of this form of chronic pleurisy of tuberculous origin in man. Any one of these varieties of chronic pleurisy may originate as a tuberculous pleurisy, and probably most of them are of this kind.

The morbid product of chronic pleurisy requires no further description than has just been given, and in the description of the morbid anatomy of acute pleurisy, which necessarily included to some extent that of its frequent termination in the chronic form. The adhesion between the lungs and the ribs is variously close and the product variously thick, insomuch that while usually the two surfaces are easily dragged apart, sometimes it is impossible to do this without lacerating the lung. Attention may again, however, be called to the displacement of viscera, the retraction of the chest-wall and curvature of the spinal column, which sometimes take place as a consequence of the extreme contraction of the plastic product of chronic pleurisy in its most aggravated form—that with empyema.

Treatment.—It need only be added to what has already been said in the treatment of acute pleurisy that, in chronic pleurisy especially, chest gymnastics, consisting in systematic inspiratory efforts and massage of the thoracic walls, must be availed of. Operative procedures must be considered in conjunction with the surgeon. Mild local measures, such as counter-irritation by iodin and counterirritating ointments, may be useful to relieve pain, which sometimes annoys the subjects of chronic pleurisy. Blisters should not be used.

HYDROTHORAX AND HEMATO-THORAX.

Definition.—The term hydrothorax is applied to any accumulation of clear serum in the pleural sacs, not due to inflammation of the pleura.

Etiology.—It is the result mainly of resistance to the free circulation of the blood through the vascular basis of the pleural membrane. It occurs as a part of general dropsy, however caused, but Bright's disease or valvular heart disease are the most frequent causes. Hence the chest should be frequently examined in these diseases, as hydrothorax may be the first symptom of dropsy. Hydrothorax is frequently bilateral in both renal and heart affections. In a careful study of this subject by J. Dutton Steele,[1] based upon a large number of autopsies with cardiac hydrothorax, in about 83 per cent. of cases the effusion was bilateral, and in 17 per cent. unilateral. Of the bilateral, 70 per cent. were unequal in distribution, and of these, three-fourths were greater on the right side. Of the 13 unilateral cases, ten were right-sided and three left-sided. The usual explanation of this preference of pleural effusion for the right side in cardiac hydrothorax is that more frequently pressure is exerted by a dilated right auricle upon the root of the right lung, interfering with the return circulation from the pleural sacs. Left unilateral effusion occurs as the result of pressure upon the root of the left lung and left superior intercostal vein. Unequal bilateral pleural effusion must, therefore, be due to unequal pressure on the roots of the two lungs. The serous fluid in hydrothorax is characterized by the small amount of albumin as compared with that exuded in pleurisy.

Symptoms.—The symptoms are those of pleuritic effusion, both as to subjective symptoms and physical signs. Crepitant râles are sometimes heard in the lung above the effusion, due to its retraction and to partial atelectasis.

Treatment.—This is considered under that of the diseases causing the hydrothorax.

Hemato-thorax is a term applied to any accumulation of blood in the thorax, however caused. It may be due to the wounding of vessels, malignant disease, or aneurysmal rupture. The symptoms and physical signs and treatment are those of pleural effusion.

PNEUMOTHORAX.

Synonyms.—*Hydropneumothorax; Pyopneumothorax.*

Definition.—Pneumothorax means air in the thorax, but the term is limited to the condition in which there is air in a pleural sac. It is almost always accompanied by a liquid inflammatory exudate, usually purulent or seropurulent, whence the terms pyopneumothorax and seropneumothorax, though a form of spontaneous pneumothorax occurs when there is no formation of liquid. The effects of pneumothorax are compression of the lung, almost always dislocation of the heart toward the opposite side, and in some instances displacement of the liver and spleen. Pneumothorax is almost without exception one-sided, though it is not impossible for it to be double.

[1] "Distribution and Etiology of Cardiac Hydrothorax," "University Medical Magazine," vol. ix., 1897 p. 563.

Etiology.—The most frequent cause is perforation of the pleura over a phthisical cavity or a hemorrhagic infarct, or over a septic bronchopneumonic focus, or gangrene of the lung. Other causes are perforating wounds of the lung, perforation of the diaphragm due to malignant disease in the abdomen, especially cancer of the stomach or colon, or of the esophagus. Perforation into the lung from the pleural side may occur in empyema. Rupture of the lung due to straining has caused it. The opening may be valvular, so as to admit air intermittently. Rupture of emphysematous blebs gives rise to the so-called spontaneous pneumothorax.

Symptoms.—*Sudden pain* and *increased dyspnea* usually usher in a perforation causing pneumothorax, though the effect may be more gradual. Sometimes the symptoms are more severe, constituting those of *collapse—faintness, frequent pulse,* and *lowered temperature.* Later, at least slight fever, corresponding acceleration of pulse and breathing rate, continue while the condition lasts. Pneumothoraces have also been found postmortem when unsuspected before death, having occurred without producing symptoms. The patient may be orthopneic, or may lie upon the affected side, for the same reason as in pleurisy. Pleurisy is a frequent, but not invariable, consequence, and superadds its own symptoms, most palpably effusion.

Physical Signs.—These are the most distinctive symptoms. *Inspection* recognizes commonly a bulging half-chest, with the intercostal spaces obliterated or prominent as compared with the opposite side, the apex of the heart displaced. The breathing is frequent and short. *Palpation* recognizes absent or very indistinct vocal fremitus, the lungs being no longer in contact with the chest-wall, which is also in a state of tension interfering with vibration. The *percussion* note is resonant, often ringing and amphoric over the upper part of the side. If a liquid is present over the area below, containing the fluid, there is absolute dullness. On the other hand, there may be *dullness* over the air-containing space, instead of tympany, on account of the extreme high tension checking all vibration. We may also meet here that interesting modification of tympany known as Biemer's change of note, based upon the fact that with a given tension the larger an air-containing cavity, the lower the pitch of the percussion note. If the patient with pyopneumothorax sits, or especially stands, in the upright position, the pleural air-containing space is enlarged, because the weight of the fluid pushes the diaphragm downward, whereas in the horizontal position the fluid flows into the gutter between the ribs and spinal column, the diaphragm rises, the cavity becomes smaller, and the pitch of the percussion note is raised. There is also the usual change of level of the dullness corresponding with change of position, as in pleurisy with effusion.

Auscultation recognizes feeble or absent vesicular murmur in the situation where it is present in health, while amphoric breathing may be substituted—bronchial breathing of a metallic character. Ringing amphoric bronchophony is also heard when the patient speaks. An interesting auscultation sign is the so-called "metallic tinkling," a sound ascribed to the dropping of liquid from the seat of perforation into the fluid below. Here also is produced in its typical expression the "coin-clinking" sound conveyed to the ear of the auscultator listening at the back of the chest, while a coin placed upon the chest in front is tapped by another coin.

This is a sign usually limited to pyo- or hydro-pneumothorax, though it may also be produced over bronchiectatic cavities. Here, too, may be produced the well-known Hippocratic succussion sound by shaking the body of the patient, the splashing being intensified in the air-distended cavity. It should be remembered that tinkling, splashing and coin sound occur only when there are both air and liquid in the chest.

Diagnosis.—Almost the only condition with which pneumothorax may be confounded is *diaphragmatic hernia*, the physical signs of which very closely resemble those of pneumothorax. The causes of diaphragmatic hernia are usually severe traumatic agencies, such as compression between cars or under masses of earth, yet occasionally more trifling causes produce it, as in the case of severe cough. A *distended stomach* itself is named as a source of confusion with pneumothorax, and it is true that succession and metallic tinkling can be elicited in it in great perfection. The absence of distention of the thorax itself, the limitation of the physical signs to the neighborhood of the stomach, their association with movements of the stomach quite independently of breathing, point to the proper source. Pneumothorax is scarcely likely to be confounded with large *tubercular cavities*, for while the latter furnish amphoric signs over them, vocal fremitus is increased, or at least remains distinct, while with pneumothorax vocal fremitus is diminished or absent. Further, there is at least no prominence over cavities, while there is often depression, and succussion signs cannot be elicited. Finally, cavities are circumscribed. *Bronchiectatic cavities* furnish signs behind and below the scapula, and therefore more in the situation of those of pneumothorax, but there is dullness instead of tympany, no bulging, and vocal fremitus probably remains distinct, while there is often pectoriloquy, never present in pneumothorax. X-ray gives excellent diagnostic results.

Treatment.—This is mainly symptomatic. Sudden pain and extreme dyspnea must be treated by morphin, preferably subcutaneously; embarrassing accumulation of fluid, by thoracentesis and draining of the sac, and in extreme cases the air may be liberated in a similar manner. Often pneumothorax gives surprisingly little inconvenience, and it is by no means impossible for spontaneous healing to take place. Potain suggested replacing the air and fluid by sterilized air, but such air would soon be substituted by impure air. Operative interference has been carried out with more or less success.[1] The cases of spontaneous pneumothorax got well by simple rest. If, however, there is prolonged dyspnea the air may be with drawn by an aspirator with no untoward results following.

MORBID GROWTHS OF THE PLEURA

These are rare and will be considered to some extent in treating mediastinal disease. The pleura is subject to *carcinoma* and to *sarcoma*, the clinical phenomena of which are identical. Most cases of carcinoma of the pleura arise by contiguous growth from primary cancer of the lung. Secondary cancer of the pleura occasionally arises by metastasis from the mammary gland or lungs.

[1] See a paper on the "Operative Treatment of Pneumothorax," by Samuel West, "British Medical Journal," November 27, 1897, p. 1568.

Sarcoma occurs as a primary growth in the shape of the so-called endothelial carcinoma of Wagner, which starts from the endothelial cells of the lymphatics and connective tissue. It also gives rise to secondary deposits in the lungs, lymphatic glands, the liver, and muscles.

The **symptoms** of any one of these forms of growth are those of chronic pleurisy, varying in intensity with the extent of the growth, single secondary nodules often giving rise to no symptoms, while the diffuse forms, spreading from the lungs, cause all the symptoms described as belonging to chronic pleurisy, the lung symptoms being relatively insignificant. In the meantime the true nature of the disease may long remain unknown, its real nature being determined with the development of cachexia toward the end, the decline of strength, and probably secondary deposits in discoverable localities. The bloody character of the effusion is a sign pointing to malignant disease of the sarcomatous or carcinomatous type. Occasionally a sarcoma of the pleura gives rise to signs identical with pleural effusion.

The **prognosis** is altogether unfavorable, and **treatment** is palliative only.

There are also sometimes found in connection with the pleura *chondroma* and *lipoma*, while *calcification* sometimes takes place in chronic inflammatory products.

Echinococcus or *hydatid disease* is occasionally found in the pleural cavity. Of this, the first clinical symptom is hydrothorax, the fluid from which is nonalbuminous, differing in this respect from that of pleurisy and to a less degree from that of ordinary hydrothorax. The only unmistakable evidence of hydatid disease is the presence of hooklets and fragments of the hydatid cysts in the aspirated fluid. Here, also, the product may be purulent.

MEDIASTINAL DISEASE.

Definition.—Under mediastinal disease are included all anatomically morbid conditions situated in the mediastinal space, except diseases of the heart, aorta, trachea, and esophagus. By far the greater number of these are tumors, but simple lymphadenitis, abscess, and hemorrhage and fibrinous mediastinitis are also included.

Anatomical.—In consequence of the difficulty attending the conception of the mediastinum and its contents, the consideration of mediastinal disease is preceded by a brief anatomical description of the mediastinum and its spaces.

The mediastinum is bounded in front by the sternum, posteriorly by the vertebral column from the lower edge of the fourth dorsal vertebra downward, and laterally by the two pleuræ. Clinicians are in the habit of subdividing this space into the *superior*, *anterior*, *middle*, and *posterior* mediastinum or mediastinal spaces.

The *superior* mediastinum is that portion of the interpleural space above the upper level of the pericardium, between the manubrium sterni in front and the upper dorsal vertebræ behind, and bounded below by a plane passing from the junction of the manubrium with the body of the

sternum backward to the lower border of the fourth dorsal vertebra. It contains the origins of the sternohyoid and sternothyroid muscles, and the lower end of the longus colli; the transverse portion of the arch of the aorta; the innominate, the left carotid, and left subclavian arteries; the superior vena cava and the innominate veins, and the left superior intercostal vein;

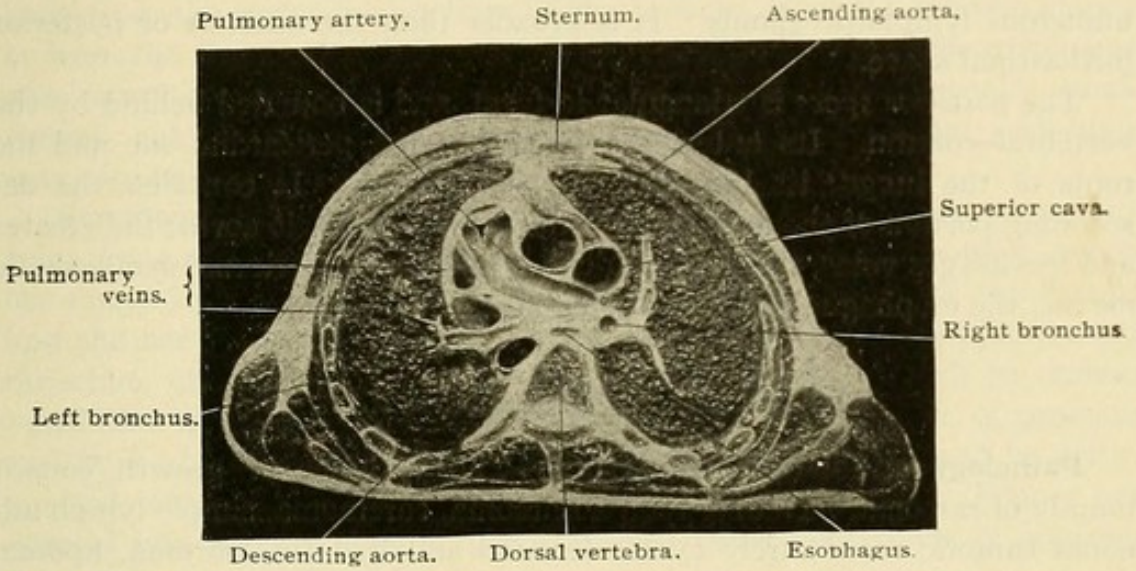


FIG. 108.—Section through Frozen Thorax at Second Interspace in Front, Looking from above downward, Showing Mediastinal Space.

the pneumogastric, cardiac, phrenic, and left recurrent laryngeal nerves; the trachea, esophagus, and thoracic duct, and the remains of the *thymus gland* with lymphatics.

The *anterior space* of the lower or clinical mediastinum is bounded in front by the sternum, posteriorly by the pericardium, and laterally by

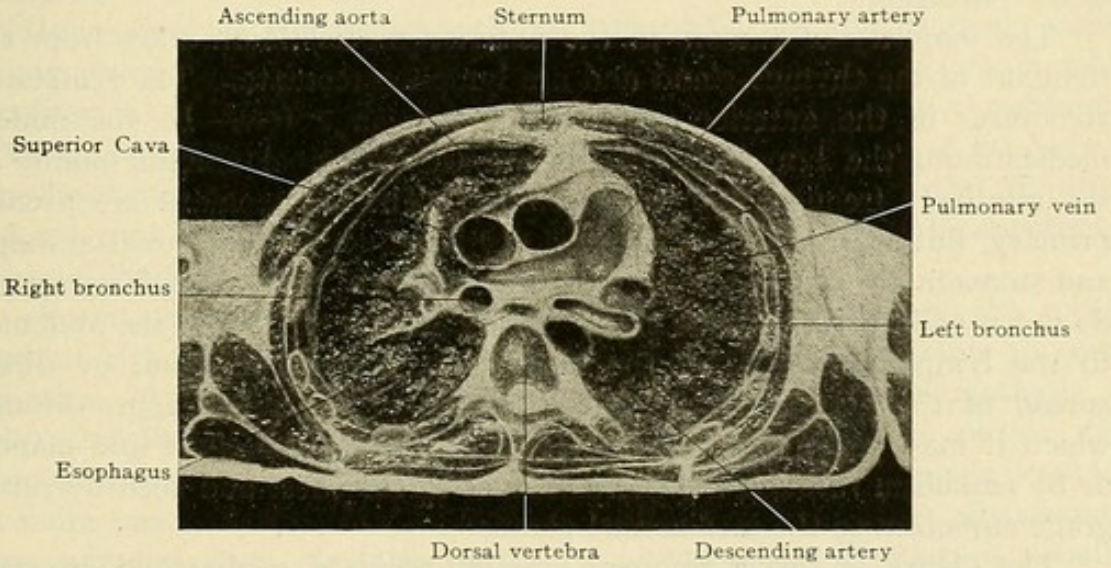


FIG. 109.—Section through Frozen Thorax at Second Interspace in Front, Looking from below upward, Showing Mediastinal Spaces.

the pleuræ. It is wider below than above, and is narrowest in the middle, since at this point the two pleural edges approach each other, while in some instances they are actually in contact. The anterior mediastinum contains the origins of the triangularis sterni muscles; the intearnl mam-

mary vessels of the left side; a quantity of loose areolar tissue; a few lymph atic glands, with lymphatics from the upper surface of the liver and two or three lymphatic glands called anterior mediastinal glands.

The *middle space* contains the heart in its pericardial sac, the ascending aorta, the superior vena cava, the pulmonary artery and veins, the phrenic nerves, the bifurcation of the trachea, and the roots of the lungs, with numerous lymphatic glands. It is broader than the anterior or posterior mediastinal space.

The *posterior space* is triangular in form, and is bounded behind by the vertebral column. Its anterior boundary is the pericardial sac and the roots of the lungs; its lateral walls, the pleuræ. It contains the descending portion of the arch and the descending thoracic aorta; the greater and less azygos veins, the thoracic duct, the pneumogastric and sympathetic nerves, the esophagus, and some lymphatics.

MEDIASTINAL TUMORS.

Pathology and Morbid Anatomy.—The varieties of growth consist mainly of sarcoma, including lymphosarcoma, carcinoma, simple lymph adenoid tumors; more rarely cysts, dermoid and hydatid, fibroma, lipoma, gumma, and chondroma; also the teratoma myomatoids of Virchow. Sarcoma and carcinoma and lymphadenoid tumors make up the larger number. Most observers have found more carcinomata than sarcomata, but in the light of the fact that many tumors formerly described as cancerous are at the present day acknowledged to be sarcomata, it is more than likely that the latter have always predominated. Hilton Fagge and Douglas Powell were the first to announce this, and William Pepper and Alfred Stengel, in their monograph published in 1895, came to the same conclusion.

The majority of tumors in the anterior mediastinum start from the remnant of the thymus gland and are lymphosarcomata. The lymphatic structures in the anterior mediastinum furnish a few. In the middle mediastinum the lymphatic glands are the principal starting-points of the relatively frequent lymphosarcomata. The carcinomata are usually primary, but secondary carcinoma is not infrequent. The breasts, lungs, and stomach are among the primary seats named. The secondary cancers do not usually attain a large size. Cancer may extend from the abdomen to the lymphatic glands of the chest by vascular embolism, by direct spread of the disease to the under surface of the diaphragm, through which it may penetrate along the lymphatics into the chest and glands, or by embolism through the thoracic duct to the chest and then by retrograde embolism to the mediastinal glands.

The pleura is also a frequent starting-point of mediastinal growths. Among these are the so-called endotheliomata of Wagner and Schulz, starting in the endothelium of lymphatic vessels and sometimes the surface endothelium. They are sarcomata or carcinomata according as the endothelium is counted mesoblastic or endodermic in origin. The cases of primary cancer of the pleura are probably endothelioma. Fibrous, fatty, and calcareous tumors of the pleura are of rare occurrence. The

lungs also contribute tumors to this locality—carcinoma, primary and secondary, and sarcoma, primary and secondary. Of the primary tumors, carcinoma is the more common, but primary sarcoma of the lymphatic glands surrounding the bronchi and within the lungs near the root is not very rare. The clinical symptoms are the same as when the glands around the bronchi outside of the mediastinum are affected. The cancers may start from the surface epithelium of the bronchi, from the mucous glands, or from the alveolar epithelium of the lung. Finally, from the esophagus, also, start cancerous tumors invading the mediastinum, usually small, though not always. From these the posterior mediastinum and lungs may also be invaded.

Symptoms.—Mediastinal tumors may be *latent*. Their symptoms when present are, in a word, those of *pressure*. Such pressure may involve the lungs, the trachea, the bronchi, the esophagus, the heart, the vessels, and the nerves of this locality. They include symptoms, subjective and objective, of the usual kind, and also physical signs. It will be remembered that the symptoms of aneurysm are also largely those of pressure, and it is chiefly from aneurysm that mediastinal tumor is to be distinguished, often a matter of some difficulty. The division by Pepper and Stengel into three groups affords the most convenient mode of studying these symptoms. These groups are:

1. Those in which the anterior mediastinum is the seat of the growth.
2. Those involving the middle and posterior spaces.
3. Those in which the pleura or superficial portion of the lung is involved.

1. *Intrathoracic Tumors Situated in the Anterior Mediastinum, and in which the Physical Signs are easily Observed.*—The symptoms are mainly those arising from pressure exerted on the venous trunks, the superior vena cava, and the right and left innominate veins. The yielding walls of these vessels as contrasted with the firmer adjacent arteries easily suffer compression, and may even be penetrated by the growths which may proliferate within them, sometimes causing occlusion by thrombosis. The consequence is *distention* of the *veins* of the *upper part of the body*—the head, neck and upper chest, sometimes the arms. Coldness, lividity, edema, and clubbing of the ends of the fingers result, while the superficial venous channels may be dilated and tortuous.

From pressure on the arteries may result *inequality* of the *radial pulses*. Of the nerves, the inferior laryngeal is especially liable to compression, with resulting *hoarseness* and *aphonia*. The sympathetic is also sometimes compressed, with consequent *inequality of pupils*, the pneumogastric being less frequently involved than when the tumor occupies a more posterior situation. As the tumor enlarges and the air-passages are intruded upon, dyspnea makes its appearance. *Dyspnea* is usually of the inspiratory kind. *Pericarditis* and *pleurisy*, with pain, hydropericardium, and pleural effusion may be present. With the prolongation of the disease the patient wastes, but it is said that *cachexia is less apt* to develop than in malignant growths of the posterior mediastinum. *Pain* is not always present—indeed, it is said to be *less marked* than in aneurysm.

Physical Signs of Growths in the Anterior Mediastinum.—To *inspec-*

tion the sternum is frequently pushed forward, and in a few instances eroded. Vocal fremitus may be either increased or diminished. *Percussion* elicits abnormal dullness, characterized by more or less irregular shape. Pulsation may occur, but is rare, while the sharp diastolic shock of aneurysm is wanting. If the tumor extends upward sufficiently, it may be felt in the suprasternal notch. *Auscultation* over the area of dullness may be negative, but sometimes the breath-sounds and heart-sounds are well transmitted, while a distinct systolic bruit may be produced by pressure on the aorta or the pulmonary artery. Eustice Smith's sign may be elicited. It is a murmur heard over the upper part of the sternum when the head is bent far backward, caused by pressure of enlarged bronchial glands on the aorta. Secondary enlargement in the cervical lymphatic glands sometimes makes its appearance.

Intrathoracic Tumors in the Middle and Posterior Portions of the Spaces around the Bronchi, Esophagus, Aorta, and Nerves, and in which the symptoms predominate over the physical signs.—The first effect is likely to be pressure on the trachea and bronchi. Hence dyspnea is an important and early symptom of tumors in this situation, and the inspiratory effort is extreme. Pressure here is also exerted upon the vena cava ascendens, whence result edema of the *abdominal* walls and *lower extremities*. The effect of pressure on the arteries is not serious. From pressure on the vagus nerve arises *peculiar cough*, paroxysmal and whooping. Sometimes it is loud and ringing, at other times constant and hacking. This cough is said to be due to the joint involvement of one vagus and the pulmonary plexus; whereas experimentally two pneumogastrics are required to be cut to produce it. The explanation is in the involvement of the pulmonary plexus. Mucopurulent and even *blood-stained sputa* may attend the cough. The latter is sometimes a sign of perforation of the bronchial wall. *Dysphagia* from pressure on the esophagus is a symptom in this group, sometimes, indeed, the only one. It is not, however, invariably present. Vomiting, cardiac palpitation with irregularity, and syncope, when present, are also ascribed to the pneumogastric involvement. *Pressure upon the azygos veins* may cause edema of the upper part of the abdomen and serous effusion in the chest, while pleural effusions are also due to complicating inflammations or neoplasms of the pleura. *Fever* may be a symptom of tumor of the posterior mediastinum. It is usually moderate but is sometimes high and irregular, followed by sweating. On the other hand, there may be lowered temperature, as in tumor of the anterior mediastinum from impeded circulation.

Cachexia is much more frequent with this group of symptoms, as might be expected from the greater severity and disturbing effect of the disease. including, as it does, destructive process involving bone and lung structure, as well as severe and deep-seated pain.

Physical Signs of Growth in the Middle and Posterior Mediastinal Space.—It is evident that in this group the physicial signs play a secondary rôle, and except as a result of modified breathing by pressure and impairment of resonance to percussion, have little significance.

3. *Tumors Originating in the Pleura and Lung*, and in which the symptoms and physical signs are of equal prominence. The former is the

more frequent starting-point, but the underlying lung is usually soon invaded and may be more frequently the actual starting-point than is commonly supposed. Naturally, the *symptoms first produced are those of pleurisy*, and the disease is generally so regarded at first, being characterized by the comparatively sudden onset, sharp pain, cough, embarrassed breathing, and pleuritic effusion. Instead of abating ultimately, as is the course in pleurisy, these symptoms grow worse, especially the pain, which extends along the intercostal nerves and their distribution and to the neck and arms. The cough also persists, while the expectoration may become bloody and include sometimes cells from the morbid growth.

Paracentesis, too, is successful, and often furnishes in the peculiarity of its product valuable aid in the diagnosis, because, instead of being clear or nearly so, it is apt to be bloody or slightly chyliform from the presence of fatty matter. This fatty character has been found where there were cancer and sarcoma. The diagnostic importance of certain large, swollen cells of endothelial nature, which seem to become detached and transformed only in case of pleuritic disease of malignant character, is insisted upon by Fraenkel. To the information gained from the fluid obtained by tapping are added also unusual resistance to the trocar and imperfect relief to the dyspnea. Rapid emaciation, anemia, and cachexia complete the picture, while all doubt is removed if secondary growths make their appearance in the lungs, as not infrequently happens.

Physical Signs of Mediastinal Growths Originating in the Pleura and Lung.—These are those caused by pleurisy, pleuritic effusion and consolidation of the lung plus the signs of a growth in one or the other part of the mediastinum.

Diagnosis.—In view of the similarity of symptoms to *aneurysm*, the history of the case in mediastinal disease becomes of the utmost importance, but shortness of breath, the bulging of the thorax, irregular outline of percussion dullness, the feebleness of breathing sounds, the dislocation of the heart and sometimes of the abdominal organs, the symptoms of venous engorgement, which are usually more marked in mediastinal disease, the more rapid course, and secondary metastatic deposits are strong points in favor of the latter as contrasted with aneurysm. Laryngoscopic examination with a view to discovering any constriction of the trachea from pressure by the tumor may be availed of. The subjects of mediastinal disease are usually younger than those of aneurysm. Bony erosion and pain are less frequent. Constitutional disturbance and emaciation are more marked. Diastolic shock is never present in mediastinal disease, while pulsation, if present, is not expansile.

Confusion with *pleurisy* and *pericarditis* is a natural error when the symptoms involving the pleura and pericardium are recalled, and here the slower development of the symptoms associated with those of compression of the various mediastinal tissues and absence of tendency to improve should lead to suspicion of the true nature of the disease.

The nature of the tumor may even be suspected from certain features. Thus, rapid growth, metastatic deposits in the glands of the neck and apices of the lungs, cachexia, tumors and in other situations point to malignancy. Especially may sarcoma be suspected if the subject be a

youthful one. Abscess may be suspected if there is a history of injury, caries, or pyemia, or if there is abscess of the lung or empyema attended by the supervention of pressure symptoms. Hemorrhage may be suspected also when there is trauma and the symptoms develop very rapidly.

Treatment.—There is no treatment for mediastinal disease, except such as may suggest itself for the palliation of symptoms.

Mediastinal Abscess.—Separate mention should be made of mediastinal abscess, since it is relatively not a very rare disease. Out of Hare's 520 cases of disease of the mediastinum 115 were abscesses, as contrasted with 134 cases of cancer and 98 of sarcoma, 21 cases of lymphoma, 7 of fibroma, 11 of dermoid cyst, 8 of hydatid cyst, with isolated cases of gumma, chondroma, and lipoma.

The abscesses were found in the majority of instances in males, most often in the anterior mediastinum, and most could be traced to traumatic causes. Other causes were tuberculosis, the eruptive fevers, and erysipelas. A few cases of mediastinal abscess also originate in the bronchial and tracheal lymphatic glands, as tubercular lymphadenitis. In 54 cases the abscess was acute.

Of **symptoms,** *substernal pain*, sometimes throbbing, was the most conspicuous. To this was added *fever* in acute cases; sometimes *chills* and *sweats*. Erosion of the sternum and burrowing along a rib into the abdomen were noted, also rupture into the trachea and esophagus. In chronic abscess the pus may become inspissated—cheesy. Suppurative lymphadenitis has been known to terminate thus, previous symptoms having been masked by the lung affection. Rarely are we able to detect fluctuation at the edge of the sternum and in the suprasternal notch, where there may be pulsation. Only as the abscess becomes large enough to encroach upon the air-passages does it cause dyspnea.

The *physical signs* are not distinctive. They are essentially those described in the general description of mediastinal disease. Fever, throbbing pain, fluctuation, and the history of trauma are symptoms which, if added, aid the diagnosis.

As to **treatment,** given a correct diagnosis, operative interference is justified, and likely to afford relief if the pus is reached.

Simple Lymphadenitis.—This probably occurs to a degree, in all inflammatory affections of the bronchi and of the lungs, but is rarely recognizable. The glands are mostly in the posterior mediastinum, and their enlargement may be appreciable to percussion in the upper interscapular region behind, though lymphatic enlargement may contribute also to dullness in the region of the manubrium. Tuberculosis may effect these glands, and great masses form both from tuberculosis and from leukemia and pseudo-leukemia.

SECTION IV.

DISEASES OF THE HEART AND BLOOD-VESSELS.

GENERAL SYMPTOMATOLOGY OF CARDIAC DISEASE.

Serious cardiac disease may be present without giving rise to any symptoms though the physical signs are unmistakable. Under these circumstances the heart is capable of normal action and is said to be fully compensated. When the heart's force is no longer sufficient for efficient work the following symptoms occur as this result of failure and there is said to be loss of compensation, or *decompensation* of the heart. Whatever the original lesion causing the weakness of the heart the symptoms are practically the same. If the cardiac muscle is weakened as the result of some condition outside the heart itself, emphysema, high arterial tension, etc,, the symptoms may be identical with those arising from disease of the heart muscle or its valves.

1. **Shortness of Breath, Cardiac Asthma.**—Dyspnea or shortness of breath is commonly the first symptom of cardiac disease. At first it is very slight, being felt only on exertion. As the disease advances it is induced by slighter effort, and finally it is more or less permanent. The higher degrees are commonly characterized as *cardiac asthma*.

2. **Palpitation.**—The second symptom characteristic of heart disease and commonly concurrent with shortness of breath is palpitation. By palpitation is meant undue frequency of the heart's action, with or without irregularity. It succeeds very early upon shortness of breath, or is coincident with it, and is more common in mitral disease than in aortic disease. It varies greatly in degree, being at times scarcely noticeable by the patient, and at others exceedingly distressing. The rate attained by the heart under these circumstances is sometimes as great as 200 in a minute, more frequently 120 to 150.

3. **Slow-pulse.**—Unnaturally slow action of the heart as a symptom of organic heart disease is not infrequent. The number of heart-beats is reduced to 40, 20, or even less. It is more frequently associated with degenerative, fatty or fibroid disease of the muscular substance of the heart and of the coronary arteries. Such diseased state of the muscle, often due to scleroses of the coronaries, may interfere with prompt contractile response to the stimulus of the endocardial blood on the ventricles.

Slow pulse may also be caused by resistance (scleroses and high tension) in the peripheral vessels, to overcome which the diastole is prolonged and the pulse thus slowed. In other cases there is deranged innervation.

4. **Pain.**—Pain is not so frequent in heart disease as is palpitation or dyspnea. It is of two kinds—a dull, aching pain and a sharp pain of great severity, radiating through the heart and down the arms, especially the left arm. Sometimes the patient complains of a sensation as if the heart was being compressed, or grasped in a vise. This pain is associated with an anxious expression and feeling, including a sense of impending death, which

is characteristic of the severer forms of angina pectoris. Pain of this kind is apt to be associated with disease of the muscular substance of the heart, of its blood-vessels, and of the aortic valves. Pain is less common in mitral-valve disease, and when present is more likely to be of a dull, aching character.

5. **Dropsy.**—Dropsy is another symptom of heart disease. It does not occur with every form, being for the most part absent in disease of the aortic valves and is most common in mitral disease. Not every case of mitral disease is associated with dropsy, but it occurs sooner or later in the vast majority of cases. It is sometimes the earliest symptom noticed, and makes its appearance first almost invariably in the lower extremities. It is the direct consequence of backing of the blood into the venous side of the circulation, and is due to the transudation or filtration of its watery element. The serum is, as it were, strained out. When unchecked, the swelling extends from the feet to the legs, thighs, the trunk, abdominal walls, and, last of all, serous cavities and especially the peritoneal cavity, producing ascites. The pleural sacs may, in rare instances, be the first seats of transudation in heart disease. (See remarks on Hydrothorax, page 545.) These simple transudates are usually free of albumin, as contrasted with inflammatory exudates.

6. **Hypertrophy and Dilatation of the Heart.**—These conditions will be frequently mentioned in the following pages and will be considered at greater length on pp. 600, 601, at present briefly, that a correct application of the terms may be learned. By hypertrophy is meant enlargement of the heart associated with physiological thickening of the muscular wall with or without enlargement of the cavities. When the cardiac cavity remains unchanged in size the hypertrophy is called *simple;* when there is enlargement of the cavity it is called *eccentric* hypertrophy or hypertrophy with dilatation.

When the left or right ventricle alone is affected, the hypertrophy may be simple or eccentric; when there is general hypertrophy, it is always eccentric. All true hypertrophies are numerical—that is, there is an actual increase in the number of muscular fasciculi, due partly to a fission of previously existing fibers and partly to a new formation of fibers.

The word dilatation is applied to conditions in which the cavities are enlarged without corresponding thickening of the walls. Usually there is attenuation of the walls. The latter is the typical condition. Dilatation implies degeneration, for it is through intermediate degeneration that the muscular fasciculi waste and ultimately disappear, producing thinning.

Hypertrophy more frequently affects the left ventricle, dilatation the left and right ventricles, but the whole heart may be involved by one or the other condition.

Morbid Anatomy.—The hypertrophied and dilated heart is altered in its weight, dimensions, and shape. The adult heart weighs in health, in the male 50 to 60 years old, about 335 grams (11.8 ounces); in the female, 295 grams (10.44 ounces). The average thickness of the wall of the left ventricle in health is from 5/8 to 2/3 inch (1.6 to 1.7 cm.); of the right ventricle, 1/6 to 1/4 inch (0.4 to 0.6 cm.); of the left auricle, 1/8 inch (3 mm.); the right auricle, 1/12 inch (2 mm.).

Hearts exceeding these weights and measurements are, therefore,

hypertrophied. Measurements should be made before rigor mortis sets in or after it has passed away. Relaxations may be favored by soaking the heart in water. Commonly, the hypertrophied heart does not exceed 25 ounces (750 gm.), though hearts weighing 48 and 53 ounces (144 to 1590 gm.) have been found.

The shape of the heart varies; in left ventricular hypertrophy it is elongated to the left and lies more horizontally, while the conical shape is less marked; when both ventricles are hypertrophied, the heart is round. In mitral stenosis with hypertrophy of the left auricle and right ventricle it is also quadrate, the right ventricle occupying the chief bulk of the organ, while the left ventricle recedes behind it.

DISEASES OF THE PERICARDIUM.

ACUTE PERICARDITIS.

Definition.—An inflammation of the serous covering of the heart and of its reflection on the inner surface of the pericardial sac.

Etiology.—By far the larger number of cases of pericarditis are due to some toxic substance in the blood, such as is developed in the infectious diseases, or to some excrementitious matters which accumulate in the blood because of deficient elimination. Pathogenic organisms may be the direct cause in certain cases. Other cases arise *per contiguum*, a few cases are traumatic, and those that cannot be accounted for are called idiopathic. *Acute articular rheumatism* or its cause is by far the most frequent etiological factor, from 30 *to* 70 *per cent.* of all cases being ascribed to it. The greater the severity of the primary disease, the more likely is it that the complication, pericarditis, will occur; yet it arises also in the mildest cases, and has sometimes even preceded the rheumatic attack. It may be that certain seeming idiopathic cases are due to the toxin of rheumatism spending itself on the pericardium instead of on the joints. Other infectious diseases causing it are pyemia, scarlet fever, typhoid fever, diphtheria, and even measles. Bright's disease is one of the best recognized causes of pericarditis, though not a very frequent one, and it may be the toxic matters which accumulate in the blood in this disease which are responsible for it. Such dyscrasic states of the blood as are represented by scurvy and purpura hemorrhagica may cause it. Tuberculosis of the pericardium is a common cause of pericarditis. Tubercular pericarditis may be part of a general tuberculosis or a secondary infection from the lungs.

Diseases of adjacent organs which cause pericarditis are pneumonia, pleurisy, especially tubercular pleurisy, morbid growths in the vicinity, ulcerative disease of the esophagus, disease of the bronchial glands and bronchi, disease of the vertebræ, ruptured aneurysm, abscess of the heart, or invasion of the pericardium by suppuration through the diaphragm.

Morbid Anatomy.—The appearances vary with the stage of the disease. Ordinary acute pericarditis is met with in one of three stages. The *first stage* is represented by hyperemia and its consequences. The initial events are hyperemia followed by roughness due first to loosening and detachment of the epithelium, and further increased by deposits of fresh inflammatory

lymph. This lymph is spread at first in yellow flakes over the surface of the pericardium.

From this point onward morbid appearances vary with the mode of termination. This may be by resolution, when the products described undergo fatty degeneration and are absorbed, restoring the normal state. Or there may be organization and union between the visceral and reflected pericardium (primary adhesive inflammation).

Frequently there supervenes the *second stage*, in which the liquid transudate increases, separating the two surfaces of the pericardium and distending the sac. This transudate is a clear, straw-colored fluid in which may be found floating flakes of lymph above described. The quantity of fluid varies greatly, amounting sometimes to a liter (2 pints) or more. In favorable cases it, too, is reabsorbed, and the two pericardial surfaces are reapposed with or without union of the apposing surfaces. Sometimes this union is complete and firm, so that the two surfaces are separated with difficulty, or it may be partial, by bands of varying length.

The term *third stage* is usually applied to the phenomena succeeding the transudation described. They include organization or suppuration. The former may be adhesive in various degrees or villous. The latter occurs when, union being prevented by the constant motion to which the two surfaces are subjected, organization takes place without attachment of the opposing surfaces, and a peculiar villous product results, characterized by numerous projections, uniform in size and shape, resembling closely the papillæ on a sheep's tongue. These papillæ, composed of vascular connective tissue, originate in the usual way by an outgrowth and vascularization of the connective tissue of the serous membrane, and not by organization of the exuded lymph, as was formerly supposed. This lymph undergoes fatty degeneration and absorption.

The more unfavorable cases terminate in suppuration, which may also be primary or secondary. In the former instances there is at once a rapid outwandering of leukocytes and the formation of a purulent fluid in the pericardium—pyo-pericardium. In the secondary form the clear, serous transudate is substituted by pus, an event which is usually ushered in by a chill and is followed by hectic fever. The cause of the suppuration in either case is the access of the usual pus organisms, the streptococci, the staphylococci and pneumococci. It may also be caused by the pneumococcus. The contents of the pericardium may become cheesy, especially if the inflammation is tubercular.

Symptoms.—Clinically, as well as anatomically, we seek to separate the stages, first of roughening, second of effusion, and third of absorption or organization, chiefly by aid of the physical signs.

Pericarditis is sometimes ushered in by a *chill*. More frequently a sharp *pain* in the region of the heart initiates the attack, previous to which there may, however, have been a sense of *discomfort* or *distress* about the organ, which may, indeed, be the only subjective symptom. The pain and discomfort may be referred to the epigastrium and to the appendiceal region. Attacks of pain in the region of the appendix are most confusing when they are due to a pericarditis. MacKenzie says the pain is always indicative of some myocardial involvement. To these symptoms may be added *dyspnea*

or orthopnea. There is also *fever*, which is not very high—temperature 102° F. (39.9° C.)—unless there be previous disease with fever, when the pericardial complication adds an increment. The *pulse* is frequent and the patient restless and uncomfortable. There is often *tenderness* over the region of the heart, which may be brought out by percussion or pressure with the stethoscope. The *position* assumed by the patient varies; sometimes he may prefer to lie on the left side, at other times on his back or on the right side, or he may prefer to sit up. Finally, there may be no subjective symptoms added to those of the primary disease, in which case the pericarditis can be discovered only by the physical examination, or it may escape detection altogether until the necropsy reveals it.

As the effusion distends the pericardium and encroaches on the lung, the difficulty in breathing increases, dyspnea becomes more marked, the action of the heart more disturbed, frequent, and irregular. When large it may press upon the left lung producing changes in the percussion note which will be discussed when considering the physical signs of the disease.

Still larger effusions produce *dysphagia* in consequence of encroachment on the esophagus. *Aphonia* may occur from pressure on the recurrent laryngeal nerve. The *pulsus paradoxus* of Griesinger and Kussmaul, in which the pulse beat is weakened and accelerated during inspiration, is common. (Fig. 110.)

A certain degree of *prominence of the epigastrium* may result from the encroachment of distended pericardium, while the excursion of *breathing movement* may be noticeably *greater on the right side*.

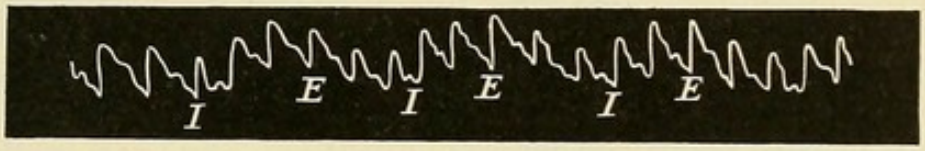


FIG. 110.—Pulsus Paradoxus.
Influence of Respiration upon the sphygmogram (after Riegel); I, During inspiration; E, During expiration.

Physical Signs.—In the *first stage* there may be pain in response to pressure, but the physical sign characteristic of the stage is the *friction sound*. It may be associated with an impulse stronger than natural. The friction sound, is of the greatest importance in diagnosis. It is a superficial to-and-fro sound synchronous with the heart beat, heard directly under the ear, commonly loud and rasping, never blowing, sometimes creaking. *It is loudest over the middle of the heart.* It is not conducted as are the murmurs at the valves in the direction of the blood current. It is often influenced by changes of position or by breathing. The rub may sometimes be felt by the hand placed over the heart. In the first stage, at least, it lasts a short time—a day or two at most and sometimes only a few hours—and disappears with the filling of the pericardium by effusion. It may sometimes be brought out or intensified by pressure with the stethoscope. It may occur suddenly long after the beginning of the inflammation, and disappear almost as suddenly.

The *second stage*, or that of effusion, exhibits usually, but not always, signs discoverable to *inspection*, or *palpation*, or to both. They depend on the amount of effusion. If large, the precordium may be bulging, the interspaces obliterated, and the impulse undulating, tumultuous, and indistinct. As the effusion increases the heart is pushed further and further away from the chest-wall and assumes a more horizontal position, while the impulse, feebler and feebler to vision and touch, may disappear altogether. The friction sound so characteristic of the first stage is found high up and becomes less marked. The site of the apex beat is raised. *Percussion* furnishes the most striking change. The area of dullness is enlarged—peculiarly enlarged. It becomes rudely triangular or truncated pyramidal with the apex toward the inner end of the left clavicle and the base as low as the seventh rib, and extending in extreme cases from nipple to nipple, even pushing the diaphragm and liver downward. The absence of resonance in the fifth intercostal space, to the right of the sternum, is known as Rotch's sign in pericarditis, and has been assigned considerable value in the early diagnosis of pericardial effusion.

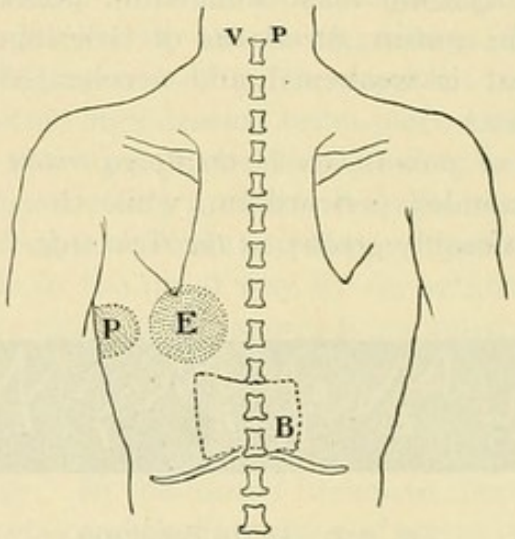


FIG. 111.—E, Ewart's posterior pericardial patch. P, Pins' sign. B, Broadbent's sign within dotted lines.—(*After Ewart modified.*)

It is not impossible, however, that a similar dullness may be caused by a circumscribed pleuritic effusion or even great enlargement of the heart The cardiohepatic angle as determined by percussion, normally an acute or a right angle, may become *obtuse*.

Auscultation confirms palpation. The conditions of the friction sound are removed more or less by separation of the opposed pericardial surfaces. Yet the sound does not always disappear. The heart-sounds are indistinct and best heard at the top of the sternum. Sometimes there is a basic systolic murmur.

The *third stage* represents a return to the normal state of affairs, which may come about with the intermediation of a *friction redux* or not; or adhesions may form between the heart and the sac, embarrassing its movements permanently, and producing retraction of the chest-wall with systole. On the other hand, necropsy has often revealed close adhesions between the heart and the pericardium which were not suspected during life. Permanent roughening, represented by the "sheep's tongue" surface or other roughen-

ing or adhesions, may produce permanent friction sound, and the pericarditis is chronic.

Secondary Physical Signs in the Lungs.—The enlarged distended pericardium protruding upward toward the left clavicle may produce there Skodaic resonance to percussion in the adjacent lung by indirect relaxation, or it may compress the lung producing dull percussion. More frequently the lower lobe is encroached upon, sometimes completely emptied of air, whence the percussion note over the lung in the lower axilla and about the angle of the scapula may be Skodaic or even dull if the lung is completely emptied of air. Correspondingly the breathing sounds may be feeble, broncho-vesicular and rarely bronchial, and there may be egophony. These sounds are not to be confounded with those due to a possible associated pleuritic effusion which gives diminished or absent tactile fremitus as contrasted with increased fremitus of the compressed lung. Attention was called to these symptoms as far back as 1857 by Bamberger[1] whence they are known as Bamberger's sign, though the names of Ewart and Sansom have also become associated with his. The normal state of the lung may be in part restored by changing the position of the patient, causing him to lean forward, to lie on his right side, or assume the knee-elbow position. To this attention was especially called by Pins.

Ewart has also called attention to an area of dullness below the ninth rib, on the left side between the spine and a line drawn through the posterior edge of the scapula, and to a less degree to the right of the spine. In this area known as "Ewart's posterior pericardial patch of dullness" the respiratory sounds are also absent and the voice sounds are feeble. He ascribes this sign to an altered dorsal relation of the liver due to pressure of the pericardial effusion. Ewart has also called attention to what he calls the "first rib sign," also recognized by palpation. The upper edge of the first rib may be followed round by the finger tip because the clavicle is apparently raised above its normal position by the effusion which must of course be large.

Physical Signs of Chronic Adhesive Pericarditis or Adherent Pericardium.—These differ materially. They are most easily studied in children, in whom the condition is especially apt to occur after rheumatism. Their study is further facilitated by dividing the condition into two groups:

1. *Simple adhesion of the pericardial and epicardial layers.* These are the cases more frequently overlooked, sometimes giving rise to no symptoms and first found at necropsy. There may, however, be friction and creaking sounds with indistinct apex beat on the one hand, or retraction of the chest-wall below described.

2. *Adherent pericardium, with chronic mediastinitis and fusion of the outer layer of the pericardium with the pleura and to the chest-walls*, a serious form, leading to marked hypertrophy and dilatation, especially in children. To *inspection* and *palpation* the precordiun is bulging, the impulse is more diffuse, extending sometimes from the third to the sixth interspace, and from the right parasternal line to outside the left nipple. The *apex* may be *displaced* in various degrees from its natural site; it may be to the right of its normal position and above it or down toward the epigastrium. It is some-

[1] "Lehrbuch der Krankheiten des Herzens," von H. Bamberger, Wien, 1857.

times multiple, or spreads in a wave-like manner over the area named. At other times the systole is associated with a *tugging retraction of the chest-wall*, which is especially evident in thin persons and is regarded by some as the most valuable sign of adhesion of the pericardium. It is most frequently noted between the seventh and eighth ribs in the left parasternal line. This may be followed by a rapid rebound of the chest-wall, known as the *diastolic shock*. It may be associated with a coincident collapse—the *diastolic collapse* of the *cervical veins*, due to a sudden emptying of these vessels consequent on the expansion of the chest-wall, a sign first described by Friedreich.

Broadbent's diaphragm sign has attracted much attention—a systolic tug which is communicated through the adherent diaphragm to its points of attachment, especially on the left side behind, between the eleventh and twelfth ribs. It is distinct and apart from the tugging in the left parasternal line, between the seventh and eighth ribs, to which attention had been previously called. Furthermore, owing to the attachment of the pericardium to the central tendon of the diaphragm this muscle does not descend with inspiration, and consequently the usually visible movement of the epigastrium during this act does not take place.

It is in adhesive pericarditis, too, that we sometimes have the *pulsus paradoxus*, referred to on page 559. First, Griesinger, and later Kussmaul, called attention to it as a constant symptom of cicatricial mediastinitis, due to the dragging of the cicatricial tissue on the great vessels during inspiration. It happens, too, when the great vessels, already compressed by the exudate, are further encroached upon by the expanding lung, making the pulse smaller and more frequent. This is more frequently demonstrable by the sphygmograph, but in extreme cases may be appreciated by the finger. It is not a pathognomonic sign of either event, but if associated with an inspiratory distention of the cervical veins, it points strongly to adhesive pericarditis.

To *percussion* there is usually a large increase in the normal area of cardiac dullness, commonly upward and to the left, sometimes as high as the first interspace. Adherent pericarditis may give rise to extremely large hearts. Often the pericardium is adherent to the adjacent pleura, in which event the area of cardiac dullness is not influenced by deep breathing, a sign pointed out by C. J. B. Williams as of great value in diagnosis.

Auscultation may be entirely negative, or there may be a modification of the usual friction sound which closely resembles the creaking of leather. A galloping or fetal rhythm may be present, or there may be a loud systolic murmur at the apex due to relative insufficiency which has often given rise to the erroneous diagnosis of mitral valve disease. Endocardial disease may, however, coexist, especially in children.

The possible association of chronic adhesive pericarditis and mediastinitis with proliferating peritonitis, perihepatitis, and splenitis should be remembered. Rarely ascitis may be one of the early signs of this condition and lead to the mistaken diagnosis of cirrhosis of the liver. This condition has been described as multiple serositis.

Diagnosis.—In all cases of acute articular rheumatism the heart should be frequently examined, because pericarditis often supervenes with feebly

pronounced subjective symptoms. At the outset the distinction is to be made between pericarditis and *acute endocarditis*, which as frequently succeeds rheumatism with subjective symptoms no more distinctive. There is usually not much difficulty in acute cases. The to-and-fro rhythm, heard directly under the ear, usually most distinct over the center of the heart, and the absence of sounds transmitted in accordance with the laws of transmission of the valvular abnormal sounds, are distinctive features of the cardiac friction. If, however, one of the to-and-fro elements is wanting, the difficulty is greater and errors do occur. Close study must be made as to transmission. It is further characteristic of the friction sound that it is increased in loudness by pressing the chest-wall with the stethoscope, while this is not the case in endocardial murmurs. Such pressure is, however, often painful to the patient. In *chronic valvular defects* there are changes in the size and position of the heart which are not present in the first stage of acute pericarditis. When both acute endocarditis and pericarditis are present, the difficulty is greatly increased and one or the other condition is likely to be overlooked.

The "pleuropericardial" friction sound or "extrapericardial" friction sound is to be distinguished from pericardial friction sound. It is a sound similar in rhythm to the pericardial sound, but the primary condition of its causation is a pleuritis involving the opposed surface of the mediastinocostal sinus of the left side. It is more commonly heard, therefore, over the left border of the heart. It is the combined product of the respiratory and cardiac action, being usually louder during expiration. It generally ceases during a deep inspiration, because at this time the cardiac action cannot produce the required rubbing. On the other hand, this is sometimes the very condition under which the friction sound is loudest. Simply holding the breath may also stop it, though not necessarily, because the heart motion produces it. This influence of the breathing one way or the other is, however, of importance in diagnosis, while other symptoms must also be taken into consideration. Thus, if it be a pleurisy, the pleural friction sound is probably heard elsewhere, and there are the other symptoms of a pleurisy present, while those of a pericarditis are absent. Unlike the true pericardial friction sound the pleuropericardial friction sound is uninfluenced by bending the body forward, but is heard with equal distinctness with the body in any position. Difficulties again increase when it is associated, as it sometimes is in a pleuropneumonia, with endocarditis. It also occurs in tuberculosis, where it is sometimes associated with a systolic click due to the simultaneous expulsion of a bubble of air from a portion of softened lung.

For diagnosis between pericarditis with effusion and dilatation of the heart see page 604. It is in this differential diagnosis particularly that Rotch's sign and the difference as determined by percussion of the cardiohepatic angle become valuable. It must be remembered, however, that Rotch's sign is not always present, even when there is considerable effusion. The possibility of a circumscribed pleuritic effusion must also not be overlooked. Bamberger's sign—Skodaic resonance and dullness in the lower axilla and region of the angle of the left scapula—should be sought; also Ewart's posterior pericardial patch.

Prognosis.—The course of pericarditis varies with different cases. In an ordinary uncomplicated case passing to recovery, the duration is one to three weeks, even when there is considerable effusion, which is often absorbed with surprising rapidity. In other cases, especially in cachectic subjects, the duration is longer. Relapses occur. When adhesion results, convalescence is greatly prolonged, and in the majority of cases the heart is permanently crippled. On the other hand, extensive adhesions are sometimes found at necropsy where no lesion was suspected. The pyo-pericardial cases are usually fatal.

Treatment.—Prompt treatment is of the greatest importance in pericarditis. Rest is an absolutely essential condition. Measures to relieve pain are indicated. Nothing is so satisfactory as moderate doses of morphin administered hypodermically, associated with atropin in the proportion of 1/150 grain (0.00044 gm.) of the latter to 1/4 grain (0.0165 gm.) of the former. Cold applications to the pericardium by Leiter's coil or the ice-bag are sometimes useful. At other times hot applications are more comforting. Blisters of 1 to 2 inches in diameter may be useful in the stage of effusion.

Digitalis may be used when one is certain that the strength of the heart muscle is failing. It should not be used as a routine measure. The aromatic spirit of ammonia is indicated. Strychnin is a valuable heart tonic. Liquid food, including milk and broths, should be adhered to until convalescence is established. Eggs may, however, be early allowed.

If the effusion is very large, tapping the pericardium may be necessary to relieve the patient, although practically the relief which first follows a successful operation is rarely followed by complete recovery. The aid of the surgeon should be secured if possible, but if not, puncture may be made in the fourth interspace, an inch (2.5 cm.) to the left of the edge of the sternum. If made in the fifth interspace, the puncture should be made a little further out—say 1 1/2 inches (3.5 cm.). A safe point which may be used in large effusions is the left xiphocostal angle, at which the needle should be pushed upward and backward. Still another site is the left fifth interspace between the apex impulse and the outer margin of dullness. When the pericardial fluid is pus, a simple tapping is insufficient. Free incision should be made, and free drainage should be established with aseptic precautions. John B. Roberts[1] collected 35 cases of suppurative pericarditis treated by incision, of which 15 recovered and 20 died. It is not impossible that if operation were done earlier, better results would follow.

The treatment of chronic adhesive pericarditis is mainly symptomatic, and when a condition of dilatation arises is that of dilatation of the heart from any other reason.

OTHER PERICARDIAL AFFECTIONS.

Other affections of the pericardium are hydropericardium, hemopericardium, pneumopericardium, and tuberculous pericarditis, rarely morbid growths.

Hydropericardium.—This term is applied to a large accumulation

[1] "American Journal of the Medical Sciences," December, 1897.

of serous fluid in the pericardium. In health the pericardium is simply lubricated by this fluid. It occurs sometimes as a part of a general dropsy, most frequently cardiac dropsy, more rarely in renal dropsy. The accumulation is seldom large in these cases. It is not common, but is sufficiently so to demand frequent examination of the heart, as it is often overlooked. Its signs are the same as those of the inflammatory effusion.

HEMOPERICARDIUM, or blood in the pericardium, occurs only as a result of rupture of an aneurysm in the first part of the aorta into the pericardial sac, from rupture of the heart itself or a wound of the heart. It is rapidly followed by shock and death. The physical signs are those of effusion. It may also be caused by tuberculosis of the pericardium. Cancer of the pericardium may be associated with blood effusion.

PNEUMOPERICARDIUM is a rare condition in which gas is present in the pericardial sac. It is analogous to the much more common one of pneumothorax. As in pneumothorax, the presence of air implies also the presence of liquid and that, usually, pus. It is produced by similar causes, such as perforation into an air-containing space like the lungs or esophagus. Such perforation is usually traumatic. Decomposition of pericardial exudate or morbid growth, it is said, may also produce it.

Symptoms.—Its symptoms are pain and pericardial embarrassment, but the physical signs are most distinctive, especially those of auscultation. To inspection there is prominence of the precordium, with indistinctness or obliteration of apex-beat, restored by the patient's bending forward. Percussion furnishes dullness over the lower portion of the cardiac area and tympany above it, the position of both being altered by change in position of the body. To auscultation the heart-sounds assume a striking metallic character, being audible even at a distance from the body. A similar metallic character is given even to a friction sound, if it is present, as it often is.

Diagnosis.—The diagnosis of this condition requires differentiation from the effect of an *air-dilated stomach* on the heart-sounds, or rarely of a *phthisical cavity* or *pneumothorax*. All doubt in the case of the stomach is removed by filling it with water. The associated symptoms of the other conditions make a mistake unlikely.

Treatment is scarcely available, except in case of external injury, when operation may be of service.

TUBERCULOUS PERICARDITIS presents nothing peculiar in its symptoms or signs as already described.

MORBID GROWTHS OF THE PERICARDIUM are rarely diagnosticated before death.

DISEASES OF THE ENDOCARDIUM.

ACUTE ENDOCARDITIS.

SYNONYM.—*Valvulitis*.

Definition.—Endocarditis in both its acute and chronic forms is an inflammation for the most part confined to the valves; for such inflammation, therefore, *valvulitis* is a more correct term. The lining of the cavity of

the heart is, however, sometimes affected in acute endocarditis, especially in the more severe cases, when it is known as *mural endocarditis*. It is usually in the apex of the left ventricle that such inflammation occurs. It is, in reality, only a complication of some infectious disease.

There is, however, a great difference in the severity of different cases of acute endocarditis, and the disease varies from those in which recovery almost always takes place up to a certain point, leaving often a degree of valvular defect known as chronic endocarditis, to those which are fatal. It is impossible, however, in the beginning to definitely state when a case will be mild and when severe.

In attempting to explain why at one time the simple form and at another the virulent form of endocarditis arises, it may be stated that the toxins generated by the less virulent bacteria may pave the way for the operation of the virulent streptococcus and staphylococcus pyogenes, the pneumococcus, the gonococcus, and other organisms which are found in the morbid products of malignant endocarditis. It is not unreasonable to suppose that the former produce the simple form of endocarditis, while the cooperation of the septic bacteria named is necessary to produce the malignant variety. On the other hand, it may be not so much the specific organism as the constitutional or local peculiarities of the individual on whom the disease is engrafted—the nature of the soil, as it were.

Etiology.—Almost any one of the recognized infectious diseases may become a cause of simple endocarditis. Acute articular rheumatism is, however, the most frequent cause, about 30 per cent. of all cases being ascribed to it. After this comes chorea. Indeed, William Osler, who has made the subject a special study, says: There is no disease in which, at necropsy, acute endocarditis has been so frequently found. Vegetations were found on the valves in 62 out of 73 fatal cases of chorea collected by him, chorea probably being a form of rheumatism. The complications may accompany a very mild form of rheumatism, the "growing pains" of the laity. Tonsillitis, gonorrhea, scarlet fever, pneumonia and tuberculosis, are not infrequent predisposing causes; less frequently are diphtheria, erysipelas, smallpox, and typhoid fever. Endocarditis also supervenes as a complication of Bright's disease. Even in these cases bacteria are found in the vegetations. Cachectic states, such as are caused by tuberculosis and cancer, also seem to favor the development of acute endocarditis. Finally, chronic valvulitis is a predisposing condition of both mild and severe forms of endocarditis, whence the term "recurring" endocarditis. This latter form has been fairly well proven to be due to a special organism.

Morbid Anatomy.—The left side of the heart is more frequently involved, and in this the mitral leaflets first, in at least half of all cases; next the aortic cusps; then, in the right heart, the tricuspid valve, and finally the pulmonary valve. In embryonic life, in which acute endocarditis also occurs, the right side of the heart and the tricuspid valve are most frequently affected, accounting thus for certain congenital valvular defects.

The type of the morbid change on the valves in simple endocarditis is so constantly a product warty or fungous in appearance that the term *warty* or *verrucose* endocarditis is often applied to this form. On the auricular sur-

face of the mitral, and the ventricular surface of the aortic valves, at the line of their contact during closure—*i. e.*, 1/25 to 1/12 inch (1 to 2 mm.) back of the valve edge—granular and warty excrescences make their appearance. These rise 1/12 to 1/8 inch (2 to 3 mm.) above the surface and extend a variable extend along the valve. They soon become capped with fibrin, often abundantly, and thus a vegetation is formed. The vegetation begins in a proliferation of the cells of the adventitia and of the connective tissue of the external laminæ of the endocardium. Thus formed, it is a friable product, liable to be broken off at any time and carried into the general circulation to a point of lodgment, where it plays the rôle of an embolus. In point of fact, this accident does not often happen in the simple acute endocarditis succeeding febrile diseases. It occurs more frequently in the acute endocarditis engrafted on chronic valvular disease, and in the malignant form. More frequently the vegetation undergoes organization and contraction, and the valve is restored partially to its natural condition, leaving a simple sclerotic thickening, which is especially prone to become the starting-point of new processes. Unless there has been previous valvular disease, or unless a myocarditis accompanies the condition, there is no enlargement of the heart in the beginning of acute endocarditis. In the severe and malignant forms the vegetations vary in size from that of a pin's head to that of a pea, and are reddish-yellow in color. The seat of this vegetation becomes rapidly necrotic and breaks down into an ulcer which may perforate the valve, with or without previous protrusion—the so-called *valvular aneurysm*.

Symptoms.—These are often masked by those of the previous disease, and sometimes overlooked, the autopsy first disclosing the lesion. There is frequently noticed, however, greater or less *embarrassment of breathing*, orthopnea being not infrequent; the *pulse* is much more rapid and may be *irregular*, the patient is restless, the countenance dusky, while the *temperature* is a degree or two higher than normal. Altogether, it is plain that he is sicker. Yet there is rarely actual pain, as in pericarditis. Leukocytosis is present. A blood culture will frequently show the infecting organism in the blood stream. Fever and leucytosis may be the first to attract attention.

Physical Signs.—As already stated, in the first attack of endocarditis there is no notable enlargement of the cardiac area as determined by percussion or inspection of the seat of apex-beat. *Auscultation* may recognize a murmur, the situation varying with the valve involved. If the mitral, a murmur is heard in this area, usually systolic, soft, and blowing, at times quite harsh. Very rarely is there a presystolic murmur, though its more frequent occurrence might be expected from the nature and situation of the lesions described. When the lesion is at the aortic orifice, the murmur is heard in the aortic area at the second interspace at the right edge of the sternum. It is usually also systolic, but may be diastolic. But not every aortic murmur heard in acute endocarditis is due to a valvular lesion, as the condition of the blood predisposes to a hemic murmur. Basic murmurs also occur in the pulmonary area to the left of the sternum, which are functional in nature.

Nor is a systolic murmur in the mitral area always due to organic change in the valves because the state of the muscle predisposes to imperfect

closure of the auriculoventricular orifice. Mitral regurgitation may also occur in rheumatism and in other acute febrile diseases from myocardial changes, as the result of which the basal part of the cardiac muscle is enfeebled and unable to do its part of the work of closing the mitral orifice, and the valve leaflets are insufficient to complete it. Some of the cases of murmur which disappear with recovery may belong to this category. The same excrescences which form on the valve leaflets may also attach to the papillary muscles and *chordæ tendineæ* as well. It is characteristic of endocardial murmurs to come and go. In the severe forms there are *chills*, followed by *fever* and *sweats*. In this form, particularly, the resemblance to intermittent fever seems at first close, but a careful study of the temperature chart from day to day, and, above all, the leucocytosis show that the malarial disease is not present. The absence of the *plasmodium malariæ* serves, however, to distinguish it from malarial disease.

It always greatly aids the diagnosis when to chills and fever are added other symptoms suggesting *embolism*, which so frequently occurs. The occurrence of a hemiplegia, pain in the region of the spleen, with increased dullness on percussion, pain in the region of the kidney with hematuria, or a *sudden blotch in the skin*, of the kind described, is of inestimable value. Unfortunately for diagnosis, these symptoms are not often present. Rarer symptoms of similar origin are *impaired vision* from retinal hemorrhage, *parotitis*, and *abscess of the parotid gland*. The symptoms are not always so pointed as detailed, while they may include others not mentioned. The fever may not be so high, but it is always present; again, it may not be remittent, but continuous. There may be *jaundice*, *precordial oppression*, *shortness of breath*, while heart symptoms may be altogether absent, when it is almost impossible to distinguish the disease from a septic fever of the ordinary kind. The *pulse* and *respirations* are invariably accelerated. Extreme embarrassment of breathing is very characteristic. *Albuminuria* and *casts* occur in all forms, either as the result of acute nephritis or of renal embolism.

A further study of the symptoms of malignant endocarditis permits their classifications into three groups, known as the *septic* or *pyemic*, the *typhoid*, and the *cerebral*.

The *septic* type occurs in connection with such septic processes as external wounds, the puerperal process, or acute bone disease with necrosis. The symptoms added are rigor, irregular fever, sweats, and exhaustion. Yet these are only the symptoms characteristic of pyemia. In fact, it is a pyemia; and the term *arterial pyemia*, suggested by Wilks, is a good one, because the pyemic abscesses result from emboli, starting in the left heart and lodging in arteries. The endocarditis constitutes the distinctive feature of the disease. The resemblance to intermittent fever here exists also, and a quotidian or double tertian type may be simulated.

The symptoms of the *typhoid* type are even more characteristic. We meet here, too, the same prostration, irregular temperature, and sweating; rigor is less frequent, and the onset is more gradual. There are delirium, drowsiness, often diarrhea, with distention of the abdomen and tenderness in the right iliac region, to which a rash may also be added, which, though not identical with that of typhoid fever, is, nevertheless, similar to it. The

tongue is dry and brown, and sordes collects about the teeth. The temperature is remittent, like that of typhoid, reaching 103° to 104° F. (39.4° to 40° C.) and even higher. Here again the heart symptoms may be overlooked.

Still another group is the *cerebral*, in which the symptoms simulate meningitis, basilar or cerebrospinal, with acute delirium as the distinctive feature.

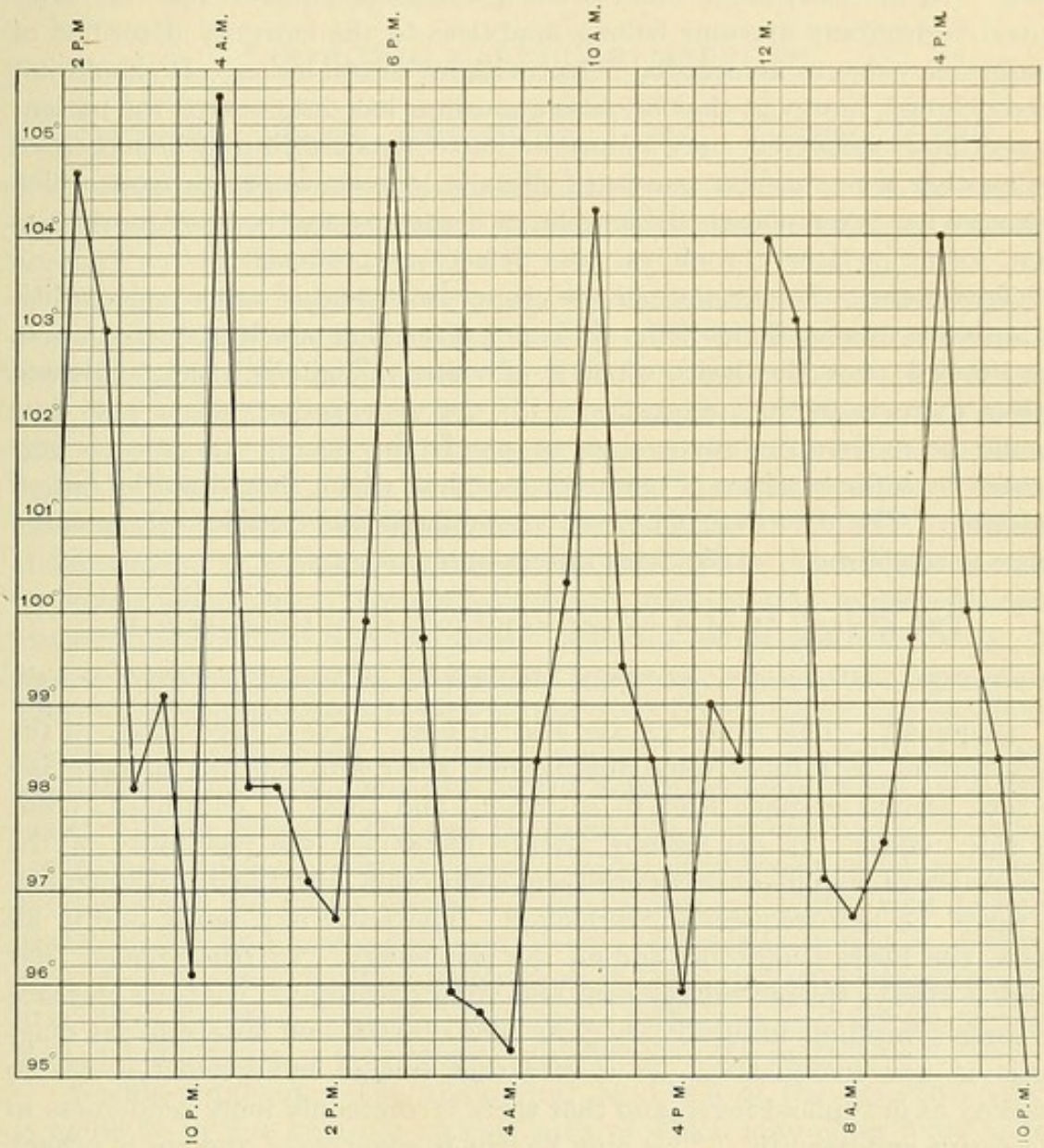


FIG. 112.—Temperature Chart, Malignant Endocarditis.

Diagnosis.—This is based almost entirely on the physical signs, as no one of the symptoms is pathognomonic. Nor are the murmurs always to be relied upon, for the reasons assigned.

The distinction of the endocardial from the pericardial murmur was considered in treating of *pericarditis*. The more superficial situation of the latter over the body of the heart, its to-and-fro rhythm, not connected with the heart-sounds, its failure to follow the usual laws of conduction, and the fact that it is made more pronounced by pressure—all serve to distinguish it. A. E. Sansom calls attention to a possible source of error in a pericardial roughening at or about the apex, especially in children, which causes a sys-

tolic apical murmur. This should be remembered as a possible, but rare, occurrence. In the malignant forms this is difficult.

Prognosis.—The subject of the simple form of acute endocarditis rarely dies, but he is likely to recover with a damaged heart—in other words, chronic valvular disease results, while the severer forms are rapidly fatal. This is not, however, always the case, for complete recovery is not impossible. On the other hand, some of the instances of complete recovery after mitral regurgitant murmur belong doubtless to the category described of insufficiency due to myocardial defect without mitral lesion. It should not be concluded, however, that because a murmur has disappeared the patient has certainly recovered, since a murmur due to myocarditis may be succeeded by another true valvular murmur. Finally, one acute attack from which recovery has taken place is liable to be succeeded by another and another, so that, sooner or later, chronic valvular defects are produced.

Treatment.—The keynote of the proper treatment of acute endocarditis is absolute quiet rest in bed. It is not often that much else is required. Ice applied over the heart often is of value. Digitalis is not indicated unless there is distinct dilatation with decompensation, when the dose should be moderate—only enough to steady the heart. Dyspnea is best treated by sufficientdoses of opium or morphin, which should not be put off too long. The diet should be easily assimilable and liquid until convalescence is established. Salicylates are probably of value.

The Severe or Malignant Form of Acute Endocarditis.

Synonyms.—*Ulcerative, Infectious, Mycotitic, or Diphtheric Endocarditis.*

Diagnosis.—This is not always easy at first. A few days' study of the temperature, with its extreme fluctuations, the rigors, and the supervening sweats, should at once lead to suspicion, and these, if continued, point to this disease. If one would always remember the possibility of the occurrence of malignant endocarditis in connection with the diseases named, it would be less frequently overlooked. The fever is a septic one in all cases, the heart symptoms adding the peculiarity. In true *typhoid fever* there is always splenic enlargement and often parotitis, so that the presence of these symptoms naturally suggests that disease, and an erroneous diagnosis is not inexcusable. It is said that splenic enlargement is not so marked as in typhoid fever, and that there is commonly more tenderness in ulcerative endocarditis. This may be true in some cases, and not in others. At the present day the Widal test should, of course, be made in all doubtful cases of fever and may afford important assistance in diagnosis. The blood culture furnishes the most conclusive evidence of the presence of the disease.

Rheumatic fever often more closely resembles malignant endocarditis, with its high, irregular fever, and copious sweats, while confusion is further contributed to by the fact that endocarditis is one of the most frequent complications of rheumatism, the malignant form being, however, more infrequent than the simple. But recurring rigors are not usual in rheumatism. The joint symptoms of rheumatism are conspicuous at an early stage of the disease; there is no enlargement of the spleen, nor symptom ascribable to

embolism, unless secondary to endocarditis. The essential identity of ordinary *pyemia* and malignant endocarditis has been mentioned, and only the endocarditis and its consequences distinguish the disease from ordinary septic fever. The presence of leukocytosis in endocarditis.

It must not be forgotten that the simple and severe forms are not separated by any sharp line.

CHRONIC VALVULAR DEFECTS.

SYNONYMS.—*Chronic Endocarditis; Chronic Valvular Disease.*

Definition.—Permanent alterations in the structures about the cardiac orifices, producing incompetency, narrowing, or other deviations from the normal.

Etiology.—The majority of chronic valvular defects are the consequence of endocarditis, acute or chronic. It may be that the very first attack of acute inflammation has left the valve leaflets in so sclerotic a condition that they readily become the seat of the subsequent changes which constitute the chronic disease, or it may be that several attacks are necessary before a permanent effect is produced. On the other hand, we must acknowledge, too, a chronic valvulitis, in which valvular defect is brought about gradually without the intervention of acute inflammation. This process is analogous to chronic endarteritis, consisting in hyperplasia with fatty (atheromatous) and calcareous degeneration of the new tissue. In fact, a chronic endarteritis may spread from the aorta to the aortic valves. These slowly induced inflammations are variously caused. The rheumatic poison may cause them, as it does the acute forms. Alcoholic indulgence and intemperate eating, whether by the direct irritation of the substances taken into the blood or through the poison of gout engendered by them, are frequent causes. Another cause is prolonged muscular strain, producing overtension of the valve leaflets. This operates in laborers who do much heavy lifting, and sometimes in athletes. Especially potent is it when, as is often the case, hard muscular work is associated with overeating and drinking. To these, syphilis also often contributes. Under all of these latter circumstances it is the aortic cusps which suffer most.

Morbid Anatomy.—The anatomical condition of the defective valves is made up of five separate factors, each of which may enter more or less into the lesion. This is true both of the auriculo-ventricular and semilunar valves. These conditions are:

(1) Thickening. (2) Retraction. (3) Adhesion. (4) Atheroma, either alone or associated with calcification. (5) Calcification.

1. *Thickening* is the immediate result of an overgrowth of connective tissue. The slighter degrees are seen along the bases of the aortic cusps and at the line of contact in closure of the mitral leaflets. Such degrees do not necessarily impair the function of the valves. More advanced stages produce a distinct thickening and sclerosis of the whole of each aortic cusp and mitral leaflet.

2. *Retraction* or curling is the result of shrinkage of this hyperplastic tissue. The three aortic cusps are often reefed back and fixed, although

the very edge of the valve may still remain movable. In the case of the mitral valve, the tendinous attachments of the papillary muscles often contract and draw the valves into the left ventricle, producing a permanent funnel-like extension analogous to that which takes place in physiological closure of the mitral orifice.

3. *Adhesions* unite the valve leaflets, increasing their immobility and rigidity, interfering with complete opening and closure. The right and posterior aortic cusps are most frequently united. Most serious is the effect of union of the mitral leaflets, which sometimes results in a reduction of the orifice to a mere slit or buttonhole-like opening—the buttonhole mitral orifice.

4. *Atheroma*, or fatty degeneration, is also often found in the shape of yellow spots on the surface of the valves and at the marginal attachments of the aortic cusp, without producing insufficiency.

5. *Calcification* or limy infiltration of the valves thus united may succeed in various degrees, producing in extreme cases firm, calcareous rings which further diminish the mobility of the valves. In less degrees there are splinter-like projections into the substance of the valve which also interfere with complete closure and opening; at other times there may be simple marginal deposits which impede the function of the valves only slightly or not at all.

Still another form of lesion found at necropsy is *rupture of a leaflet*, the result of strain. This is perhaps not possible with a sound valve, while one weakened by the morbid states described may give way. The physiological result is insufficiency, while the lumen of the orifice during systole is not encroached upon. Such an accident is not infrequent in acute ulcerative endocarditis in consequence of erosion and partial destruction of the valve.

Congenital defects are relatively common in the right side of the heart, which is the subject also of inflammation during intra-uterine life. The changes resulting from the latter are of the nature of fusions. Such defects also occur on the left side; most rarely in the mitral valve.

The term relative insufficiency is applied when a valve is insufficient or incompetent because of dilatation of the ventricular cavities or vessels which it guards. (See below.)

Mitral Insufficiency or Incompetency.

Occurrence and Mechanism.—This is the most frequent of the uncombined forms of valvular disease. The valve leaks. The blood flows backward during systole from the left ventricle to the left auricle. The distended auricle, first attempting to resist the backward flow, hypertrophies but eventually dilates, and the blood is crowded backward into the lungs, which become engorged. The right ventricle, in its efforts to push the blood through the engorged lungs, hypertrophies, and the pulmonary factor of the second sound becomes louder and sharply accentuated. The compensating effect of the hypertrophied right ventricle for a time arrests the mischief. At this stage, perhaps, begins the hypertrophy of the left ventricle, which in all cases of mitral insufficiency presents itself sooner or later, although at first the double outlet for the blood from the ventricle would seem to demand

less strength of the left ventricle. The right ventricle, however, in its hypertrophied state, delivers more blood through the lungs to the left ventricle, which demands more power to drive it on, hypertrophy results, and thus compensation is for a time longer maintained. Sooner or later the right ventricle dilates, the tricuspid valve becomes insufficient, the blood regurgitates into the right auricle and thence into the great veins of the neck. The valves of these ultimately yield, the jugular vein dilates with each systole of the right ventricle producing the so-called jugular pulse and the general venous system is engorged.

Incompetency of the cardiac valves is often brought about by dilatation of the ventricles and the great vessels leading from the heart, the valve leaflets themselves remaining intact. Such *relative insufficiency* affects most frequently the auriculo-ventricular valves, and, as a consequence, the latter are not "sufficient" to stretch across their respective orifices and close them. Less commonly the semilunar valves are similarly deficient; more frequently the aortic in dilatation of the aorta; and more rarely also the pulmonary valve when that vessel is dilated.

Etiology.—Endocarditis, acute or chronic, is the most frequent initial cause of mitral insufficiency.

Symptoms.—Often there are no symptoms, because for a considerable length of time compensation keeps pace with the development of the disease unless the latter be sudden, as by rupture of a valve leaflet. The first thing noticeable is usually *shortness of breath* on exertion, followed by severe attacks of dyspnea, the so-called *cardiac asthma*. With this is soon associated *palpitation*, or "beating" of the heart, which increases and abates *pari passu* with the dyspnea. Next is *irregularity of the heart's action*. This is the beginning of waning compensation, of which the immediate result is congestion of the lungs. Dyspnea is now permanent. Thence the engorgement extends to the right ventricle and venous side of the circulation, the pressure in the arteries being proportionately less. The lung engorgement invites frequent attacks of bronchitis, excites *cough* and increases dyspnea. *Orthopnea* is frequent at this stage, and the patient can only rest sitting in a chair. There is sometimes *blood-stained expectoration*, in which may be found alveolar epithelium dotted with pigment granules.

Along with this, or before it, the *liver becomes congested*, enlarged, and tender; the mucous membrane of the stomach also becomes congested, causing *nausea* and *indigestion*. The hepatic enlargement is sometimes very great, and it has been mistaken for cancer of the organ. The liver is often the seat of pulsation, and as often a jugular pulse is seen. Both signs are pathognomonic of tricuspid regurgitation usually the result of mitral regurgitation. Later, this enlarged liver may return to its normal state or contract still further, constituting the so-called *red atrophy*. *Jaundice* is sometimes present due to compression on biliary capillaries by engorged blood-vessels. In advanced stages the *kidneys* also *become passively congested*, the urine is scanty and its specific gravity high, while there are copious deposits of urates. It contains a small quantity of albumin and there may be hyaline tube-casts, rarely even a few blood-disks. As a secondary result of hepatic engorgement only there may also be enlargement of the spleen.

Concurrent, or succeeding on failing compensation, comes *edema* or dropsy, the direct result of venous engorgement and the filtration of the liquid elements of the blood into the subcutaneous connective tissue of the body—first of the feet and legs, then of the trunk, face, and upper extremities, and, finally, into the pleural and peritoneal cavities, causing various degrees of inconvenience. Effusion into the pleural sacs may occur before there is any tendency to dropsy elsewhere. This can be observed by careful and repeated examination of the chest.

Nose-bleed is a symptom sometimes seen in this disease. It is a natural result of the venous congestion. Among the later and rarer symptoms in children especially, is clubbing of the finger-ends.

Physical Signs.—In the early stage before compensation is broken the systolic murmur may be the only physical sign, later *inspection* discovers increased frequency of breathing movements. The impulse is to the left of its normal position in the fifth interspace, or perhaps a little lower down. It may be in the line of the nipple or even beyond it, more forcible and diffuse than in health. The outward dislocation of the apex is due to the enlargement of the two ventricles. In thin persons an auricular impulse may be seen to the left of the pulmonic area in the *second interspace*, and may be presystolic and active for the auricle—that is, produced when the auricle contracts; or systolic and passive for the auricle—that is, caused by a filling of the auricle by regurgitation from the ventricle during the latter's systole. In young persons a bulging precordium may be looked for in the second and third interspaces to the left of the sternum; also to the left of the lower part of the sternum from hypertrophy of the right ventricle. In advanced stages there is a jugular pulse, which is also pathognomonic of tricuspid regurgitation. The jugular pulse must be distinguished from the *false* jugular pulse which occurs when the venous system becomes replete with blood from any cause like overexertion. It is commonly more superficial. It is presystolic in time, while the jugular pulse is systolic. Moreover, the false jugular pulse is obliterated by pressure on the vein above the clavicle, while the true jugular remains distinct below the point of pressure. The liver may pulsate with each systole.

On *palpation* the apex-beat is found more forcible than normal, at least while compensation is maintained, and there may be a pulsation near the ensiform cartilage, caused by the *systole of the enlarged right ventricle*. As compensation wanes the impulse becomes weaker and irregular. Sometimes an intermittent *systolic thrill* is felt in the *fourth interspace* in the left mammillary line. Very rarely is there a systolic thrill at the apex.

The *radial pulse* in the early stage is comparatively unaltered. Later, it becomes frequent and irregular in volume. Appended (Fig. 113) is a sphygmogram of the pulse in advanced mitral insufficiency. It is of the type of the *pulsus parvus irregularis*.

Percussion generally finds enlargement of both the relative and absolute areas of dullness, upward in the direction of the left auricle, downward to the left and also to the right, the right border of the heart extending at times beyond the right border of the sternum. The impaired resonance thus produced rather by the right auricle than by the ventricle which in its enlargement pushes the auricle to the right.

Auscultation recognizes a systolic murmur in the mitral area, conducted with various degrees of loudness into the left axilla and under the angle of the scapula. This direction of its conduction is the distinctive feature of this murmur. It is usually soft, but occasionally rough, more rarely musical. Richard C. Cabot says that musical murmurs are heard more frequently at the mitral valve in regurgitation than at any other valve. A fading mitral systolic murmur generally means further failing compensation, and when compensation is completely gone it is substituted by incomplete valvular sounds, great irregularity, gallop rhythm, labored breathing, and all the signs of pulmonary congestion. The mitral systolic murmur is also sometimes heard distinctly to the left of the pulmonic cartilage, and rarely over the entire precordium. Not always loud enough to be easily heard, it may be brought out by exertion on the part of the patient.



FIG. 113.—Tracing of Pulse of Mitral Insufficiency.

The second sound of the heart is heard sharply accentuated at the pulmonary area until the tricuspid valve fails, when the accentuation fades away. The aortic second sound is less strong, corresponding with the smaller degree of hypertrophy of the left ventricle.

Differential Diagnosis.—The murmur of mitral regurgitation is not usually difficult of recognition through the features which have been described. A functional murmur is rarely heard at the apex. Should it happen that it is, it will not be conducted as is the organic mitral systolic murmur, and it is not heard behind and below the angle of the scapula. Aortic roughening produces a murmur heard at the same time as the mitral systolic, and may also be propagated to the apex, but the position of greatest intensity is the second interspace to the right of the sternum and the murmur is transmitted loudly into the great vessels of the neck, which is never the case with the mitral systolic murmur. The tricuspid systolic murmur occurs at the same time, but its point of greatest intensity is at the ensiform cartilage.

MITRAL STENOSIS.

Occurrence and Mechanism.—This lesion occurs as an uncombined or simple form of valvular disease in young persons, especially women, but is usually combined with mitral insufficiency. Seventy-six per cent. of all cases are said to occur in the female sex. In the simple form the orifice is stenosed, and the blood is restrained from passing freely into the left ventricle. It is backed into the left auricle, the lungs, right ventricle, and general venous circulation, but the left ventricle is not hypertrophied in simple mitral obstruction because no extra muscular demand is made on it, while hypertrophy of the left auricle is one of its most characteristic signs. Theoretically, the left ventricle should even atrophy from diminished function. Practically this does not occur, but the absence of the enlarge-

ment is of great diagnostic value. Excellent compensation is often maintained in mitral stenosis for many years.

The enlargement and dilatation of the left auricle is in rare instances enormous, especially in the form known as horizontal dilatation which has been especially studied by Owen, Fenton and Ewart.[1] The enlargement with corresponding dullness to percussion extends both to the left and right of the sternum and the auricle has been tapped under the impression that it was a circumscribed collection of fluid in the pleural sac.

Pure stenosis without regurgitation is possible if the mitral valve leaflets are fused without retraction, so as to form the funnel-shaped opening already described. In these cases a postmortem demonstration of insufficiency by means of the hydrostatic test is scarcely possible. Less frequently the mitral orifice viewed from above is a mere slit—Corrigan's buttonhole contraction—straight or slightly crescentic, in a smooth septum formed by fusion and contraction of the valve leaflets and tendinous cords. In some cases calcareous infiltration is added, and in a few rare instances deposits of urates are found. The ratio of buttonhole mitral stenosis to the funnel-shaped orifice varies with different observers—1 to 10 by A. E. Sansom, 1 to 13 by Hayden, 1 to 46 by Hilton Fagge.

Etiology.—Most frequently mitral stenosis is the result of endocarditis, acute or chronic, but it may in rare cases be congenital. In these cases, of which a number have been collected by Bedford Fenwick, the stenosis is secondary to narrowing of the tricuspid orifice, thus explained:—a small quantity only of blood being allowed to pass into the right ventricle and lungs, a diminished supply is sent to the left heart, whence both its cavities and orifices are reduced in size. Attention has been called by Teissier to the possible origin of mitral stenosis in tuberculosis and Robert H. Babcock has reported some cases which tend to confirm this view.[2] *No functional disorder can cause mitral stenosis.*

Symptoms.—These, often delayed by compensation, as in mitral insufficiency, are the same as in that lesion though palpitation and dyspnoea seem to be more prominent. In consequence of this similarity of symptoms the diagnosis of mitral stenosis is based largely on the physical signs. As in mitral insufficiency, in long cases and especially in children, clubbing of the finger-ends may be present.

Physical Signs.—Mitral stenosis may exist for many years without giving rise to physical signs except the murmur. *Inspection* consistently with what would be expected in absence of hypertrophy of the left ventricle, recognizes little or no displacement of the apex in pure stenosis. If there is any, it is due to the hypertrophy of the right ventricle which pushes the apex toward the left rather than downward and to the left. Nor is the true apex-beat increased in force, though there may be strong epigastric pulsation because of hypertrophy of the right ventricle, and in persons with thin chest-walls there may be an impulse in the *third and fourth interspaces*, to the left of the sternum, due to right-auricle hypertrophy. A left auricular

[1] Owen and Fenton, "A Case of Extreme Dilatation of the Left Auricle of the Heart," "Clinical Society's Transactions," vol. xxxiv., 1901, p. 183.
Ewart and Owen, "A Case Illustrating some of the Clinical Features of Horizontal Dilatation of the Left Auricle," "Ibid.," vol. xxxiv., 1902, p. 142.
Owen, "Horizontal Dilatation of the Left Auricle," "Ibid.," p. 147.
[2] Diseases of Heart and Arterial System, 1903, p. 252.

impulse, presystolic, may be noted in the second interspace to the left of the sternum, for the same reason as in mitral regurgitation. A *jugular pulse* may also be present if there is tricuspid regurgitation. A bulging precordium is possible only from great enlargement of the right ventricle and is not often seen. In children the lower sternum and fifth and sixth left costal cartilages may be prominent from this cause. There may be prominence of the right upper quadrant of the abdomen from enlargement of the liver.

Palpation shows that the apex-beat is without undue force, but it may be diffuse, and an impulse may be felt in the epigastrium, the situation of the apex of the right ventricle. The most marked feature recognized by palpation is the *presystolic thrill* at the apex, differing in this respect from the rare systolic thrill of mitral insufficiency. It is usually best felt in the fourth or fifth interspace, within the nipple-line. It is similar in rhythm to the presystolic murmur, but may be present without it. It is often absent. It is pathognomonic of mitral stenosis. Palpation may recognize tenderness in the region of the liver.

In moderate degrees of stenosis the *pulse* is not altered; in high degrees it is small, from want of blood and left ventricular power. Irregularity, like that of mitral regurgitation, is characteristic of advanced stages. Two tracings from cases of mitral stenosis are introduced in the text.

Percussion recognizes cardiac enlargement in the direction of the left auricle and right ventricle, but not of the left ventricle in pure mitral stenosis, that is, the area of cardiac dullness extends to the right of the sternum, and upward often to the top of the second rib.

Auscultation.—The most characteristic auscultatory sign is the pistol-like first sound, short, sharp and loud. A murmur does not occur in every case of mitral stenosis because of the feebleness of the auricular contraction, especially toward the end of life, when compensation has failed and there is not the force of contraction sufficient to throw the blood stream into audible vibration. Most characteristic is the abruptly terminating *presystolic* murmur, confined for the most part to the mitral area to the inner side of the apex-beat, though it may be conveyed upward, and it is even heard posteriorly, though rarely. It is true that the presystolic murmur is heard in atypical situations, especially in the axilla and below the angle of the scapula, more frequently than has commonly been supposed.[1]

The *presystolic murmur* of mitral stenosis is a diastolic murmur occurring at the end of diastole of the ventricle, because it is at this time that the auricular systole takes place, giving the propulsive force necessary to produce the audible vibration. It is a loud, rough, vibratory murmur terminating suddenly with the first sound, sharp and ringing and coincident with the presystolic thrill. The murmur terminates with the impulse, and as the two are not always easily separable, the former is commonly more readily distinguished by its qualities than by its time. It is often followed by a "sharp" first sound, which, in consequence of this character, is sometimes mistaken for a second sound. As the disease advances the presystolic element disappears and a murmur develops which may occupy

[1] See an excellent well illustrated paper on this subject by J. P. C. Griffith in the "Transactions of the Association of American Physicians," 1895.

the entire peroid of diastole. In such cases there is sometimes a short pause between the beginning or diastolic part and the terminal or presystolic part of the murmur. In the last stage the murmur may disappear altogether, leaving only the snapping first sound.

Differential Diagnosis.—The murmur of mitral stenosis ought not to be confounded with the murmur of aortic regurgitation, for the latter is heard loudest in a different situation, but moreover there is enormous hypertrophy of the left ventricle, which is wanting in mitral stenosis. The time of tricuspid stenosis is identical with that of mitral stenosis, but it is heard in a different part of the precordium—in the epigastrium. Tricuspid stenosis is, however, a very rare lesion. Much more reasonably might the murmur of mitral stenosis be confounded with the so-called *Flint murmur.* This murmur is heard at the apex, at the same site as the presystolic, and may be similar in quality. It occurs in high degrees of dilatation of the

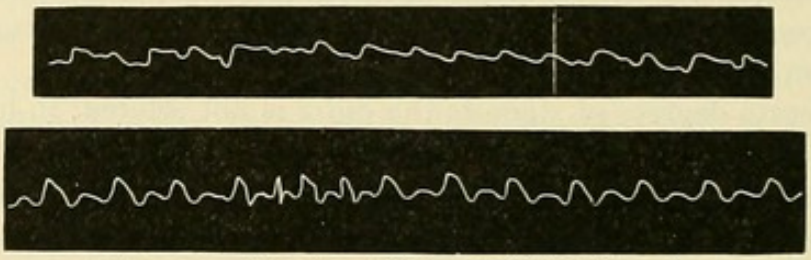


FIG. 115.—Tracing of Pulse in Mitral Stenosis.

ventricle, occurring in *aortic regurgitation,* and is due to the fact, according to the late Austin Flint, Sr., that in such dilatation the mitral leaflets cannot, during diastole, be kept back against the ventricular wall, but remain in the blood current, throwing the latter into audible vibration. It may be said of the Flint murmur that it is never as intense as mitral presystolic murmur and that it never occurs except in the presence of aortie regurgitation. Otherwise the acoustic qualities are similiar. The snapping first sound and systolic shock are also apt to be modified or absent. Accentuation of the pulmonic second sound is wanting in marked aortic insufficiency, and the other signs of aortic regurgitation are most hopeful to a diagnosis.

A rumbling sound succeeding a pericarditis in children, referred to especially by Broadbent and Rosenbach, has occasioned error, but this, too, is said to unaccompanied by accentuation of the first sound at the apex. It is a transient murmur often followed by recovery.

These sources of error are well illustrated by observations of Phear,[1] who investigated 46 cases of presystolic murmur in which no mitral lesion was found at autopsy. In 17 of these there was aortic regurgitation; in 20 there was adherent pericardium; in nine nothing more than dilatation of the left ventricle was found. In none was the snapping first sound, so common in mitral stenosis, recorded during life.

Very frequently the presystolic murmur is associated with a mitral systolic or regurgitant murmur, usually soft and not very loud, though sometimes it is distinct and is well transmitted into the axilla.

Accentuation of the second sound is marked, but confined to the pulmonary area, because there is no hypertrophy of the left ventricle. The

[1] "Lancet," September 21, 1895.

second sound may also be *duplicated*, because of the lack of synchrony in the closure of the aortic and the pulmonary valves. A. E. Sansom regards this reduplication as only a seeming one of the second sound. He regards it rather as the normal second sound followed by another sound due to a sudden tension of the mitral valve itself. He also says it occurs in at least one-third of all cases of mitral stenosis, and is rare in other cardiac conditions. The accentuation of the pulmonary second sound also disappears with the enfeebling of the contraction of the right ventricle.

The *pulse* is small, as would be expected from the small volume of blood ejected from the ventricle, but may be quite regular, as seen in the sphygmograms. More frequently it is irregular. Sometimes there is a rhythmical failure of an alternate heart-beat to reach the wrist, while the sphygmogram will show a small rise between two higher ones constituting the *pulsus bigeminus*.

On account of the difficulties mentioned, while the presystolic murmur is a valuable sign of mitral stenosis, it should not be alone relied upon for diagnosis, but should be taken in connection with other signs. Tricuspid stenosis may be associated with mitral stenosis or insufficiency, or both. With the loss of compensation the presystolic murmur disappears together with the thrill, and there remains only the sharp, ringing first sound.

In slight degrees of mitral stenosis the second sound is heard at the apex, but as the lesion becomes more serious it becomes fainter and eventually inaudible in this situation, though markedly accentuated in the pulmonic area. In advanced cases of mitral stenosis with much dilatation of the right cardiac cavities, the entire anterior surface of the heart may be made up of the right side of the heart. The right border of dullness being made up of the border of the right auricle, the left of the right ventricle, the apex of the heart being right ventricle. In the secases there is usually a loud systolic murmur at the xyphoid due to tricuspid regurgitation.

The physical signs of mitral stenosis are more changeable and fleeting than those of any other valvular disease of the heart.

Sansom lays great stress on the evidence of the cardiograph in the diagnosis of mitral stenosis, which enables one to judge of the relative length of systole and diastole. In stenosis the diastole may be greatly prolonged, or the diastolic intervals vary greatly in duration. In mitral regurgitation, on the other hand, a short interval only separates the systoles.

Complications.—Patients with mitral stenosis are subject to attacks of recurring valvulitis, with consequent embolism in different parts of the body. Embolism is a frequent complication of mitral stenosis. Pulmonary tuberculosis is found more often in association with mitral stenosis than any other form.

Mitral Insufficiency and Stenosis.

Occurrence.—More common than mitral stenosis as an uncombined lesion is stenosis associated with insufficiency, in which case we have the double mitral murmur of mitral insufficiency and mitral stenosis, sometimes with difficulty divisible into its two parts. Extreme irregularity of rhythm and pulse, with frequency and smallness of the latter, conspicuous thrill,

marked right-sided hypertrophy, and sharply accentuated pulmonic sound are characteristic of advanced stages. The presence of hypertrophy of the left ventricle points to associated mitral insufficiency and stenosis. When this combined lesion exists, mitral insufficiency is said to usually precede.

Aortic Insufficiency or Incompetency.

Occurrence and Mechanism.—By aortic insufficiency is meant an inability of the aortic valve to close the orifice of the aorta. This is the most serious of the valvular diseases of the heart commonly met. Next in frequency to mitral incompetency, *much more frequent than aortic stenosis*, with which it more often coexists, it is a disease of men rather than women, commonly adults at or before middle life. It includes 30 to 50 per cent. of all cases of chronic valvular disease. The width of the aortic orifice increases from birth to old age, while the valve cusps tend to shrivel, so that conditions favorable to incompetency coexist. It is more frequently associated with arterial sclerosis and less frequently the result of rheumatic endocarditis, though it may be thus caused. It is the lesion most frequently followed by sudden death.

When it exists, the aortic valves are incompetent to close the aortic orifice, either on account of the large size of the latter or of disease of the

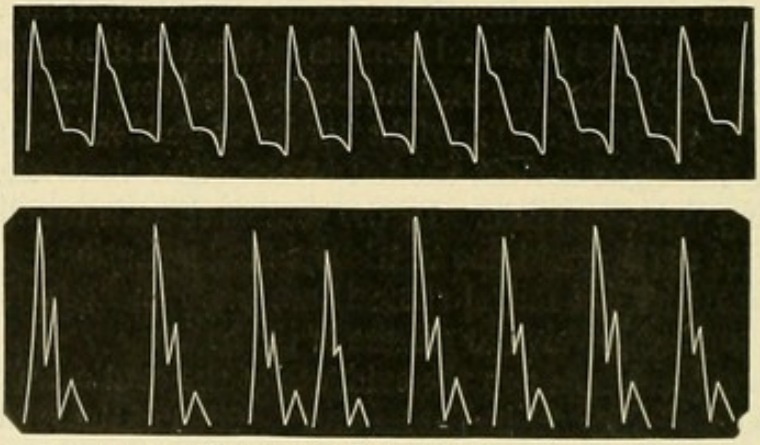


Fig. 115a.—Tracings of Pulse of Aortic Regurgitation.

valve segments, and the blood flows backward into the left ventricle during diastole. The ventricle, seeking to restore the balance, redoubles its energy and hypertrophies. The blood is thus driven into the aorta with great force, distending the arteries to an extreme fullness, which, however, falls promptly away, because of the backward flow into the ventricle at the same time with the forward movement into arteries and capillaries. This sudden falling away of the pulse, from extreme distention to collapse, is very characteristic of this form of valvular disease, and is called the "trip-hammer" or "water-hammer" pulse, also Corrigan pulse. To the careful observer it may even be visible in the exposed arteries, such as the corotid, temporal, and radial, while the aortic beat, ordinarily beyond reach in the suprasternal notch, may be felt in this situation.

The abrupt jerking impulse with sudden recoil is easily recognized by the finger on the pulse, which, however, fails to find the pulse as strong and

hard as would be expected from its appearance. On the other hand, it is soft and receding. It is commonly regular. A tracing of this pulse is seen in Fig. 115a. It is the typical *pulsus celer et altus*. A frequent and irregular pulse is much more serious in aortic valve disease than in mitral disease. *Sclerotic changes* in the *arterial walls* are not uncommonly associated with aortic incompetency. The systolic blood pressure is relatively high, while the third phase is frequently lost, and therefore the diastolic pressure cannot be estimated.

The product of this defect is the largest heart met in morbid anatomy, the left auricle and right ventricle often sharing in the enlargement. From its size the heart is called the *bovine heart*. It may weigh as much as 35 ounces (1050 gm.), and even 50 ounces (1500 gm.) or more. The cavities are enlarged and the walls are thickened, so that it furnishes an instance of eccentric hypertrophy. There may be ultimate dilatation of the arch of the aorta from the constant pounding of the blood against it in systole, while all the superficial arteries can be seen and pulsate forcibly.

The gradual enlargement of the ventricle may ultimately cause the mitral valve to yield. Compensation is still maintained for a time by hypertrophy of the left auricle, which also yields after a time, becoming dilated and allowing the blood to engorge the lung. Hypertrophy of the right ventricle then comes to the rescue for a time. Sooner or later it, too, yields, dilates, the tricuspid valve weakens, and finally gives way, allowing the blood to flow back into the venous side of the circulation, producing engorgement of the liver, stomach, kidneys, general dropsy—the train of symptoms described under mitral regurgitation.

Etiology.—Causes of insufficiency in addition to those considered under the general etiology of valvular disease are congenital malformations, including fusion of two leaflets, commonly those behind which the coronary arteries come off. Such fused leaflets are especially prone to valvulitis and its consequences. Aortic insufficiency is quite often caused by dilatation and aneurysm of the ascending aorta, giving rise to *relative* insufficiency.

Symptoms.—Like all other forms of valvular heart disease, aortic incompetency may be compensated for a long time, and elude detection for a corresponding time. Indeed, full compensation is said by some to be most usual in this form of valvular disease. Both *dropsy* and *dyspnea* are characteristically *absent* until compensation ceases, which is also the case until the mitral valve begins to yield. Then, however, both appear and may be very distressing. An especially frequent symptom is *dizziness* with *faintness*, particularly on rising quickly. Palpitation ensues on slight exertion, and this effect is in marked contrast to the comfort of the patient when quiet, when the pulse may be slow and breathing regular. In advanced cases, on the other hand, the patient complains of a constant "*beating*" or pulsation all over the body, especially in the head, which is exceedingly unpleasant. The patient is very apt to be *troubled in his sleep* and to dream, probably because of disturbed circulation in the brain. Even permanent mental symptoms may result from this cause, including *insanity* and *suicidal tendency*. Lesser degrees are *irritability* and *peevishness*, though these are not confined to this form of heart disease. *Precordial pain*, present also in stenosis, is frequent in this form of valvular disease. It may be a dull ache with a sense of con-

striction of the chest, or sharp and radiating down the arms, particularly the left, as in angina pectoris, which condition itself is also common. With the yielding of the mitral valve and loss of compensation come the symptoms of mitral disease already described.

As previously stated, this is the form of valvular disease in which *sudden death* is frequent. It has overtaken many a victim in the course of his daily vocation and without warning, though it is most apt to be induced by some slight overexertion or mental excitement. The cause of such sudden death is probably interruption of the circulation in the coronary arteries. This may be brought about in one of two ways. These arteries in common with others, are especially disposed to endarteritis with resulting sclerosis and atheroma, a condition which constantly invites thrombosis and obstruction to the circulation; or it may be due to defective circulation in these vessels, caused by the aortic regurgitation, for even if the blood enters the coronary arteries during systole, it must still receive in health some further supply in the recoil of the blood on the closed semilunar valves, which cannot take place when the valves are incompetent. On this variety of valvular disease, too, supervenes not infrequently acute infectious endocarditis of the grave type, with the train of symptoms and the sequelæ described. Embolism in various organs is also a complication.

Physical Signs.—Inspection often discerns the prominent left precordium, with the apex-beat lowered and to the left, and the visible pulsation far beyond the normal situation of the apex, all confirmed by *palpation*. The apex beat frequently being in the sixth interspace outside the midclavicular line. Palpation also recognizes at times a *diastolic thrill* over the base, in the carotids and subclavians, and sometimes in the aorta at the suprasternal notch. This is, however, much rarer in aortic regurgitation than the systolic thrill in stenosis. The Corrigan pulse may also be felt, either with the finger on the pulse or by grasping the wrist with the entire hand, the palmar surface of the wrist touching the pam of the hand, but is much more strikingly manifested in the sphygmogram. A *capillary pulse* is also sometimes demonstrable in the skin and mucous membrane. This may be observed by drawing a pencil lightly across the skin of the cheek or forehead; and on the mucous membrane of the everted lower lip by pressing a glass microscope-slide against it. It may often be well studied around the base of the finger-nail. Pulsation in the retinal arteries may be recognized by the ophthalmoscope. Pulsation may even be seen in the uvula as originally pointed out by F. Müller in 1889.

Percussion discloses increased dullness to the left and downward, and also, sometimes in advanced cases, upward to the left of the sternum, owing to hypertrophy of the left auricle, as well as to the enlargement of the ventricle upward.

Auscultation recognizes a diastolic murmur, long, loud, and blowing in quality, usually harsher than the aortic obstructive murmur, though it is also often soft and faintly heard. It may or may not replace the second sound of the heart. It is commonly well heard in the aortic area, but its seat of maximum intensity may be either in this area, in the third interspace to the left of the sternum, or at the midsternum between these two points. The murmur is naturally transmitted downward toward the ensiform car-

tilage along the left edge of the sternum. It is sometimes also well conducted toward the apex which is in the direction of the regurgitating column, it is conducted in the direction of the great vessels of the neck, at least. In aortic incompetency also occurs the Flint murmur, described under mitral stenosis. This murmur is additional to the distinctive diastolic murmur being produced at the mitral orifice (see p. 580). *The aortic regurgitant murmur is probably the most widely conducted of all cardiac murmurs.* Over the femoral vessels may be heard a short, sharp systolic sound, the pistol-shot sound.

Differential Diagnosis.—The aortic diastolic murmur is distinguished from the pulmonic diastolic murmur by its wide conduction, the hypertrophy of the left ventricle, the Corrigan pulse, and the capillary pulse. The tricuspid presystolic murmur is a diastolic murmur, heard in the same situations but it is a rare murmur and is unaccompanied by hypertrophy of the left ventricle. Diastolic murmurs without lesions of the aortic or pulmonary valves due to relative insufficiency, to hemic conditions or cardio-respiratory murmurs[1] must be distinguished as the murmur of aneurism.

Auscultation of the vessels furnishes interesting information in aortic insufficiency. It is well known that if the stethoscope be placed with slight pressure over the carotid artery of a healthy person, two sounds are usually audible, corresponding to the expansion and contraction of the artery. Of these the latter is simply the second aortic sound heard in the carotid. is probable also that the first arterial sound corresponding with the arterial expansion is produced by vibrations of the arterial wall induced by the blood driven into it from the ventricle. The second arterial sound is greatly diminished in intensity or even absent in aortic incompetency, since the valve remains open. The aortic diastolic murmur is sometimes faintly heard in the carotid. In aortic regurgitation there is *Traube's double sound,* in the distant arteries, especially the femoral and popliteal. The sounds are such that the two follow each other closely, so that the first seems preparatory to the second, or they are separated by a longer interval, like the two sounds of the heart. The first is a sharp systolic (pistol-shot) sound and is ascribed to a sudden filling of the unusually empty artery, and is probably an exaggeration of the sound heard in health, as above described. Traube explained the first of his sounds in this way, while he ascribed the second to a sudden relaxation of this tension. Friedreich pointed out that a similar double sound could be heard in the femoral *vein* in tricuspid insufficiency, which he ascribed to tension of the valves of the vein. It is claimed that the double sound is heard in other diseases of the heart, especially mitral stenosis, and even in aneurysm, but it is acknowledged to be most frequent in aortic incompetency.

Finally, there is *Duroziez's sign, a murmur* produced light by pressure with the stethoscope upon the femoral artery. Duroziez's sign will be more easily understood when it is remembered that a murmur may be produced by pressure with the stethoscope on any artery of the caliber of the carotid —a murmur during the expansion or diastole of the artery. During the collapse or systole of the artery, on the other hand, no murmur can be thus

[1] See a paper by Richard C. Cabot and Edwin A. Locke on "The Occurrence of Diastolic Murmurs without Lesions of the Aortic or Pulmonary Valves." "Johns Hopkins Hospital Bulletin," vol. xiv., May, 1903.

produced in health. In aortic regurgitation, however, it is different, a double murmur may be produced and it is the second murmur which is essential to Duroziez's sign. It is said that this sign dies out as compensation fails. T. Clifford Allbutt does not consider Duroziez's sign peculiar to aortic regurgitation, though Vierordt says it is. A right degree of pressure, to be determined by practice, is necessary, and the artery on which it is obtained is usually the femoral.

The gravity of aortic regurgitation is measured by the degree of hypertrophy of the left ventricle, by the irregularity of its action, a symptom which appears only in advanced stages of regurgitation, the extent of collapse of the artery in diastole, the degree in which the diastolic murmur replaces the second sound as heard at the aortic orifice. Irregularity of cardiac action is a much more serious symptom in aortic regurgitation than in mitral disease. Diminution in the loudness of the diastolic murmur is a serious sign.

Aortic Stenosis and Roughening.

Occurrence and Mechanism.—By aortic stenosis is meant a narrowing of the aortic orifice. Pure and uncomplicated aortic stenosis is probably the rarest of the valvular lesions. Writers have been led into error because the presence of an aortic systolic murmur has been interpreted as meaning stenosis, where it has been produced by simple roughening of the valves or of the vessel beyond them. Richard C. Cabot says that out of 250 autopsies made at the Massachusetts General Hospital, there was not one of uncomplicated aortic stenosis. Indeed it is difficult to conceive an aortic stenosis unaccompanied by insufficiency, although it is easy to conceive of insufficiency without stenosis. Stenosis is said to be relative when there is a normal orifice while the aorta is dilated beyond it. It occurs in older persons, and the older the person, the more likely are there to be calcareous deposits causing it. It may be congenital. When uncombined with insufficiency, it is the least dangerous of the various forms of valvular disease. The narrowed orifice prevents the free discharge of blood from the left ventricle into the aorta. The ventricle attempts to overcome this, and its walls hypertrophy in proportion to the degree of resistance, and often for a long time compensate for the obstruction—until dilatation occurs, when the danger really begins. The hypertrophy thus induced, usually of the simple form, is only second in degree to that produced by incompetency.

Symptoms.—The symptoms of aortic stenosis may be long deferred, so long as compensation is maintained, and when they do occur, they are usually those of a deficient supply of blood to the brain and heart itself—viz., *dizziness* and *fainting*. Succeeding exertion there is apt to be a *sense of constriction* or *oppression* and even *pain* in the precordium, which may develop into the severe pain of a true angina pectoris.

Physical Signs.—Inspection and *palpation* recognize usually a forcible impulse outside of its normal site, and at varying distances, in accordance with the degree of hypertrophy. Some describe the apex-beat as without force and indistinct. Broadbent says it is "a well-defined and deliberate push of no great violence." Palpation often recognizes a *thrill* of great intensity with each beat of the heart, more marked when dilated hypertrophy

is established. A bulging of the precordium may also be present, though less often than in incompetency.

The pulse is the *pulsus parvus et tardus*, slow in reaching its maximum volume, which is small. It is frequent, but regular, contrasting in the latter respect with the pulse of mitral disease. It is sometimes infrequent, *pulsus rarus*. Fig. 58 is a sphygmogram of the pulse in aortic stenosis.

Percussion elicits dullness downward and laterally toward the left, since, as a rule, the enlargement is confined to the left ventricle. There may, however, be slight enlargement upward to the left of the sternum if hypertrophy of the left auricle is added.



FIG. 116.—Pulse-tracing of Aortic Stenosis.

Auscultation discloses a systolic basic murmur, loudest at the aortic area—second interspace at the right of the sternum—conducted distinctly into the carotids, and even sometimes along the course of the aorta, behind and to the left of the vertebral column, into the popliteals and dorsal arteries of the feet. It is not, however, confined to the aortic area, but may be heard over the entire precordium. It is usually rough, at least until compensation fails, but may be soft and musical. It may be heard even at a distance from the chest. It is made louder by exercise. The *aortic factor* of the *second sound is very feeble*, or not at all heard, if the constriction be quite marked, because of the feeble recoil, the necessary result of the small amount of blood in the aorta.

Roughness of the *aorta*, and of the aortic ring, *dilatation* or *narrowing* of the vessel, however caused, may also produce a *systolic murmur;* so may roughness within the ventricle in the course of the outgoing column of blood. This is by far the commonest cause of an aortic systolic murmur. But these causes have a less positive effect upon the substance of the heart—that is, do not produce as marked hypertrophy of the left ventricle. Nor do these causes interfere with the production of a normal second sound, except, perhaps, dilatation, which in that event is accompanied by an aortic regurgitant murmur. From this it follows that the important point to remember in diagnosis is that an aortic systolic murmur rarely indicates aortic stenosis. So, also, anemic or hemic murmurs, which are always systolic and for the most part basic, may simulate aortic systolic murmurs, but these occur in young, delicate persons of both sexes, are often intermittent and without other effect on the muscular heart, while they are also unaccompanied by thrill. There may be roughness, too, in the pulmonary artery, which can be localized to the left of the sternum.

As already mentioned, stenosis of the aortic orifice is very apt to be associated with insufficiency, the same rigidity and adhesion which prevent complete patulousness of the orifice preventing also complete closure.

Differential Diagnosis.—Aortic stenosis is always accompanied by hypertrophy of the heart, a rough systolic murmur at the base a small pulse. Without the latter signs a systolic murmur at the base of the heart means simply one of the forms of roughening.

Aortic Obstruction and Insufficiency.

Occurrence.—This double lesion is a comparatively frequent one; indeed, it is commonly regarded as the next in frequency after mitral insufficiency, and therefore more frequent than either aortic insufficiency or aortic stenosis alone. It occasions a double basic murmur, systolic and diastolic, and is also a grave condition, giving rise to the same dangers as aortic regurgitation, and the same enormous hypertrophy of the left ventricle.

Diagnosis.—The diagnosis of this condition requires special mention, because it not infrequently happens that it is mistaken for *aneurysm of the arch of the aorta*, which is associated with a similar double murmur of which the systolic element is due to the roughness of the aorta and aneurysmal walls, and of which the diastolic is a sign of relative insufficiency due to dilatation of the aorta. The distinctive differences between the two conditions will be given in treating aneurysm of the arch of the aorta.

Prognosis.—What was said of aortic obstruction and insufficiency is true in general of these lesions when associated, but a study of blood pressure aids us in prognosis and treatment. Great differences in the extremes of systolic and diastolic pressure exists at times.

Tricuspid Insufficiency or Incompetency.

Occurrence and Mechanism.—Tricuspid regurgitation as a primary condition is extremely rare, and, when present, is probably the result of an endocarditis which during fetal life is more prone to attack the right than the left side. Endocarditis involving the tricuspid valve may, however, also occur in children—according to Byrom Bramwell,[1] more commonly than has been supposed. Infectious or ulcerative endocarditis also affects the tricuspid valve—according to Osler, in 19 out of 238 cases. More frequently tricuspid regurgitation is the result of a *relative insufficiency*, one of the terminal events of mitral disease, the tricuspid orifice yielding with the dilatation of the right ventricle, which takes place sooner or later, consequent upon the resistance to the movement of the blood through the engorged lungs. It is also one of the possible sequelæ of emphysema of the lungs and long-standing fibroid phthisis or chronic bronchitis, succeeding, too, a primary hypertrophy of the right ventricle, due to these causes.

Thus, out of 405 autopsies at Guy's Hospital in which evidence of tricuspid regurgitation was found, 271, or two-thirds, succeeded on mitral disease, 68 were due to myocardial degeneration, 55 to pulmonary disease, viz., bronchitis, emphysema, and cirrhosis of the lung. The effects of venous obstruction growing out of tricuspid insufficiency have been detailed.

Tricuspid insufficiency succeeding upon mitral insufficiency is not always accompanied by an audible murmur. It is evident that every case of mitral regurgitation associated with dropsy must be attended with tricuspid regurgitation.

Symptoms.—These are those described when treating of mitral disease after the stage of tricuspid regurgitation has been reached, dropsy more or

[1] "Amer. Jour. Med. Scio.," April, 1886, p. 419.

less general, engorgement of the stomach, liver, and kidneys, an enlarged, tender, pulsating liver, and a jugular pulse. The last two symptoms are regarded as pathognomonic.

Jugular pulse is often more forcible in the right than in the left jugular. There is also cyanosis, dyspnea, and pulmonary edema. The jugular pulse is systolic in time, and does not appear until the valves situated at the opening of the internal jugulars into the innominate veins yield. These give way first on the right side, because the course of the right innominate is straighter and communication is more direct. So long as the valve above the bulbus jugularis is closed, the pulse is confined to the bulb, but with the yielding of this valve the pulse becomes general throughout the vein. It is sometimes difficult to distinguish a true jugular pulse from the "physiological" or "false" jugular pulse, which may sometimes be seen in health and whenever the venous system is overfull. Pressure on the vein above the valves will cause the false pulse to disappear while the true pulse, coming from the right ventricle, will remain. The physiological or false jugular pulse alternates with the ventricular systole is presystolic—a negative pulse—while the true jugular coincides with the systole of the ventricles.

Physical Signs.—In primary tricuspid disease with regurgitation, *inspection* and *palpation* reveal an apex-beat diffused toward the ensiform cartilage and the epigastrium. *Percussion* detects enlargement toward the right edge of the sternum, due to hypertrophy of the right ventricle, which occurs for the same reason as hypertrophy of the left ventricle in mitral insufficiency. It is not, however, that the right ventricle protrudes to the right as much as that it pushes the right auricle over to the right.

To *auscultation* the systolic murmur thus engendered is almost invariably feeble, and is heard almost solely in the tricuspid area, just above and to the left of the ensiform cartilage. Occasionally only is the second pulmonic sound accentuated. There should be no confounding of this murmur with that of aortic regurgitation conducted toward the same situation, but different in time, nor with that of mitral regurgitation heard at no great distance, for the reasons already given. To these must be added a difference in quality and pitch between the tricuspid and the mitral murmur, not always, however, manifest.

Tricuspid Stenosis.

Occurrence.—Tricuspid stenosis is a rarer condition, but it may be an acquired one in association with left-sided heart disease as the result of rheumatic endocarditis, and of unknown causes. Ninety per cent. of cases are associated with mitral stenosis. It is much more frequent in women, fully 80 per cent. of all cases being in them. As in endocarditis of the left side, there are thickening, adhesion, narrowing.

A presystolic tricuspid murmur pointing to stenosis, in a case observed by Gardner, was found due to a growth from the endocardium of the right auricle, so placed as to fall over the tricuspid orifice in the manner of a ball valve. Fred. C. Shattuck has met one instance of tricuspid stenosis with mitral stenosis and regurgitation, along with adherent pericardium, hepatic cirrhosis, and slightly granular kidney, as determined by autopsy. In this

case there was a presystolic tricuspid murmur observed for three years before death.

Physical Signs.—Simple uncomplicated tricuspid stenosis is extremely rare, and cannot safely be diagnosed during life; it would be recognized by the presence of a presystolic murmur and thrill, best heard in the tricuspid area, unaccompanied by hypertrophy of the right ventricle. When associated with left-sided heart disease, the diagnosis is seldom made, because the murmur is masked by the coincident mitral *presystolic murmur*. In a very few cases only is it confined to this valve. Frequently there is no murmur. *Percussion* shows dullness to the right of the sternum, if there is dilatation of the auricle, which does not always occur.

Congenital stenosis of the tricuspid orifice occurs, but is usually associated with defects of other valves, which early cause death.

Other symptoms are cyanosis of the face and lips and, in the later stages, extreme and obstinate dropsy.

Pulmonary Insufficiency or Incompetency.

Occurrence.—Simple pulmonary regurgitation is rarely seen. It may, however, exist as a congenital defect (fusion of two segments), and the pulmonary valve has been found involved in ulcerative valvulitis.

Physical Signs.—It is easy from what has gone before to deduce the physical signs which are to be expected—a diastolic murmur heard in the pulmonic area, hypertrophy of the right ventricle, later jugular pulse, venous congestion, and cyanosis. The diastolic murmur may be confounded with that of aortic insufficiency, but the latter is accompanied with hypertrophy of the left ventricle, with Corrigan pulse and capillary pulse. A few cases are related in which a diastolic murmur has been found associated with defects in the pulmonary valves—in one, warty, which might have been the result of infectious endocarditis. All others are congenital. Among them is aneurysmal dilatation. Such was the case reported to the Pathological Society of Philadelphia by Edward T. Bruen (see "Transactions" for 1883).

Pulmonary Stenosis.

Occurrence.—The great majority of systolic murmur heard at the pulmonary orifice are functional. Pulmonary stenosis, though very rare, may, however, exist, in which case it is far more likely to be *congenital from arrested development*, although *intrauterine endocarditis* may also cause it. So, also, may infectious endocarditis, and in rare instances, atheroma. The valve leaflets are apt to be fused. When the lesion is congenital, it is commonly associated with patency of the foramen of Botal or foramen ovale, together with imperfect ventricular septum and tricuspid stenosis.

Physical Signs.—Pulmonary stenosis should furnish a systolic murmur in the pulmonary area, to the left of the sternum. The murmur may even be heard behind, between the shoulders, and it may be rough. It is accompanied by hypertrophy of the right ventricle. There may be a basic thrill,

as in aortic obstruction, but the pulse is uninfluenced. Compensation may be set up by means of a patulous foramen ovale, an open ductus arteriosus, or interventricular communication. The invariable presence of cyanosis due to venous obstruction and of attacks of dyspnea complete the picture and aid greatly in the diagnosis. Anemic murmurs at the same time and place are unaccompanied by cyanosis.

Walshe has described a case of death from thrombosis of the pulmonary artery in which he heard a pulmonary systolic murmur before the end came. Before a diagnosis of primary tricuspid or pulmonary valve disease is made the case must be carefully studied from all standpoints. The diagnosis is difficult.

Congenital Defects.

Congenital defects in the cardiac valves and orifices deserve a passing notice. They may be the result of endocarditis during fetal life or of arrest of development. Their most frequent seat is the right heart, and the most frequent form is *stenosis of the pulmonary orifice*, the effects and signs of which have already been considered. Another is a permanently *patulous foramen ovale;* or there may be a *defect of the septum of the ventricles*, or a communication between the aorta and pulmonary artery—a *persistent ductus arteriosus*—or *between the aorta and the vena cava or aorta and right auricle.* All of these intercommunications produce murmurs difficult to separate, and it is, after all, by attention to the general condition that the defect is recognized. The patient is a child of arrested development, more or less permanently cyanosed, with continued embarrassed breathing—all of these are conditions which point to the congenital defect. If there be added to these a persistent loud murmur at the base of the heart without other signs or symptoms of valvular disease, this may be due to congenital defect.

In addition to these, there are a large number of defects of development which are rather pathological curiosities than of clinical interest. Among these may be mentioned *acardia*, or absence of heart, met in the monstrosity thus named; *double heart*, sometimes present in high degrees of fetal defect; *dextrocardia*, in which the heart is on the right side, alone or with other viscera. In *ectopia cordis*, or dislocation, which is associated with fission of the chest wall and of the abdomen, the heart may be in the cervical, pectoral, or abdominal regions. Then there are anomalies of the cardiac septa, of which the patulous foramen ovale is the most frequent, various in degree. Next is a small defect in the upper part of the septum, between the ventricles, in what is known as the "undefended" space, or just anterior to it. A *bi*cuspid condition of the semilunar valves, from fusion of cusps, is often met—most frequently of the aorta. The combined valve is more liable to sclerotic change. Finally, there is fenestration of the semilunar cusps.

Relative Frequency of Valvular Defects.—The order of frequency of the various valvular defects is not entirely agreed upon. As to one, however, there seems to be universal concurrence, and that is that mitral regurgitation is the most frequent. After this, however, statistics differ.

This order, in the light of modern studies, must be corrected, except as to mitral incompetency. Frederick J. Smith, analyzing the registers and postmortem records of the London hospitals for eleven years—1877-87—and taking the fatal cases only, arrived at the following order:

1. Mitral incompetency.
2. Mitral stenosis.
3. Aortic incompetency.

} Of practically equal frequency.

4. Aortic stenosis.
5. Tricuspid stenosis.

To these we may add:

6. Tricuspid incompetency.
7. Pulmonary stenosis.
8. Pulmonary incompetency.

It is evident that the older observers mistook the aortic systolic murmur to mean aortic stenosis, when roughening only of some kind was present.

Prognosis of Chronic Valvular Disease.—Possible positive statements as to the prognosis in chronic valvular disease are few, so uncertain is it and so many circumstances influence it. Undoubtedly, valvular disease often exists where the subject is totally free from symptoms, and therefore quite unconscious of it. Yet such subject is not free from danger. On the other hand, 15, 20, 30, and even 40 years pass over such cases without inconvenience, compensation being easily maintained. Such cases are usually of mitral incompetency or stenosis, or both. Much depends upon the life led by the patient—whether one of ease and quiet, associated with proper food and clothing and without dissipation. Even when such disease occasions symptoms, the same measures may hold them in abeyance for a long time, and occasional judicious treatment may raise the patient from a serious condition to one of comfort. It is astonishing with what little disturbance women with these affections sometimes bear children. Of the lesions at the mitral orifice, incompetency is usually most easily compensated, then combined stenosis and incompetency, and finally stenosis only; but even the last exists at times without subjective symptoms in persons who have worked hard. After all, the prospect of life must be judged from the symptoms in each case. The compensation which is obtained by extreme hypertrophy and apex displacement is tottering. An additional danger in mitral disease, especially mitral stenosis, is that of embolism. Recurring attacks of rheumatism not only increase the latter danger, but augment the valvular defect. The supervention of dropsy and dyspnea indicate failing compensation, and though they may be overcome, it is with increasing difficulty at each recurrence.

Aortic incompetency is a much graver condition. Yet it, too, may be compensated for years. Much here depends upon the state of the arteries, the danger being increased when associated with sclerosis or atheroma, for these conditions are likely to effect the root of the aorta and the valves, and especially the coronary arteries. Any obstruction in these, as already stated, may be the cause of sudden death. Diseased condition of the coronary arteries, which may at any time be followed by complete obstruction and similar death. Overdistention, such as takes place during exertion

may be too much for a fatty heart already dilated, and becomes also a cause, of sudden death.

The most unfavorable of all forms of cardiac valvular disease is tricuspid regurgitation, which occasions obstinate dropsy and dyspnea.

Chronic valvular disease is regarded as much more serious in young children under ten years of age; this, in spite of the fact that many conditions favorable to compensation are present, such as integrity of heart muscle and vascular supply. Notwithstanding this, the valve lesion is apt to increase. Congenital defects in the heart are apt to destroy the lives of children in the first few years of their existence.

Finally, almost any serious illness, especially when involving the lung, increases the danger to the life of the subject of cardiac disease, while mitral disease, and especially mitral stenosis, invites pulmonary congestion and inflammations.

Treatment of Chronic Valvular Diseases of the Heart.—1. *Prophylaxis.* There can be no doubt that the number of cases of chronic valvular disease may be decreased by a careful treatment of the diseases which excite them or favor their occurrence, especially acute rheumatism. Rest and quiet should be prolonged long after the symptoms of pain and fever have subsided, and a second attack of rheumatism should especially be guarded against, as an unhealed endocarditis is sure to be followed by another attack. Temperance and the avoidance of excesses of all kinds which tend to load the blood with toxic substances constitute a prophylaxis of no small importance.

2. *Remedial Measures.*—In the first place, it is well known that there exist chronic valvular defects at either of the orifices which give rise to no symptoms whatever and are often accidentally discovered. Such patients need no drugs, and digitalis preparations are harmful. As a rule persons with a valvular lesion with complete compensation of the heart, should be told of their condition in order to avoid excesses of all kinds. On the other hand the physician must know his patient. Many nervous individuals are made miserable for life by the knowledge of a valvular lesion. To them heart disease spells sudden death. Such persons should avoid overexercise and excitement. Running or even walking rapidly, hurriedly ascending stairs, extremes of passion of all kinds, and especially of anger, should be avoided, as should also exposure and irregular living. In a higher grade of involvement of either orifice, the same treatment is demanded in a more imperative manner, since its omission results in a loss of compensation, manifested by dyspnea, palpitation, and precordial distress.

Treatment.—This is the same whichever orifice, or indeed in a large part where there is no valvular lesion. The cases may well be divided into two classes—those with slight symptoms of failing compensation; those with more severe symptoms, those where the muscular power of the heart is entirely inadequate.

General Principals.—The first essential is conservation of the energy of the heart, as Mackenzie puts it, safeguarding the reserve power of the heart. To this end mental and physical rest combined with proper protection of the muscular power of the heart are essential. Regulation of the habits of the individual of his food, his drink, his work, and his recreations are important.

Cases with Slight Symptoms.—When a patient notices dyspnea on exertion, some palpitation of the heart, vertigo, precordial distress, cough, with possibly a slight edema of the extremities, he must at once be told of the heart lesion if he is not already aware of it. He should then be put absolutely at rest until all symptoms disappear; usually no drugs are necessary. His diet should be milk, cereals, toast, green vegetables, eggs, a very small amount of meat. The bowels should be moved freely every day with salines or calomel. If, after a few days the symptoms persist, he should be given digitalis, either the tincture or the powdered drug. After a few days he should be allowed to sit up and walk daily a short measured distance, increasing the distance gradually. At this time the use of carbonated baths and regulated resistance movements, Nauheim or Schott treatment is of the greatest value. The patient then should be instructed to avoid all occupations, or exercise, or food, which causes the least return of symptoms. Digitalis should not be given in a routine way, but under strict control of the physician.

It appears this simple method is a necessity and better than attempting to discover the various causes for the rupture of compensation first. Get the heart compensated as the first essential, while this is going on study the patient's condition, his method of living, and regulate it in the future. It is a pernicious habit to simply give such individuals digitalis and allow them to go about their ordinary life. They will be symptomatically better, but will soon drift into a more serious failure of the heart muscle.

Cases with Severe Symptoms.—The more serious class of cases, is that in which the compensation is more completely broken, either because the patient has gone about his work so long that he has developed symptoms of severe dyspnea; much palpitation of the heart; edema of the legs; and venous congestion in the liver, kidney and other organs; or because this is one of a succession of attacks of failing compensation. The symptoms may be mild or so severe that they threaten life. In such cases absolute rest in bed is a necessity. After the first twenty-four hours and sometimes before, digitalis is demanded to steady the heart's action. The patient must be placed on a milk diet preferably. The bowels must be kept open. Sleep is imperatively demanded. For this purpose, bromide of potassium in fifteen grain doses, chloral hydrate in five to ten grain doses may be used; but morphine sulphate used hypodermically is of the greatest value, and may cause a case which threatens to terminate in death to take on a change for the better, which will finally end in comparative health.

After these patients begin to improve, even before they are out of bed, resistance movements, the so-called Schott treatment may be employed. Most of these cases need one of the digitalis group of drugs after they are about work again. As this class of cases presents so many distinct symptoms, it seems worth while to give the treatment best suited for the symptoms themselves.

Venesection has a distinct place in cardiac therapeutics, when the right heart is dilated the heart throbbing against much peripheral pressure, the second sound much accentuated, the patient cyanosed as with acute cardiac dilatation. Ten to twelve ounces of blood taken from the arm is often life saving.

Physical Methods

Nauheim and Schott Treatment.—It is in this condition that the Schott or Nauheim treatment is especially useful. It consists in the use of the carbonated saline baths at Bad Nauheim, associated with special exercises called "resistance" movements, originated by the brothers Schott. Fortunately, artificial baths may be substituted for the natural baths, or the treatment would have limited application. Ignoring for the present the rationale of the action of these baths, their therapeutic efficacy is undoubted. The waters of Nauheim have a temperature ranging from 82° F. (27° C.) to 95° F. (35° C.). Their important constituents are chlorid of sodium and chlorid of calcium.

The baths may be imitated at home by dissolving chlorid of sodium and chlorid of calcium in water, to which carbonic acid is added by decomposing bicarbonate of sodium by hydrochloric acid. K. N. B. Camac has calculated the required quantities of salt to each 40 gallons of water for six different strengths of the baths. The following are the proportions of sodium chlorid and calcium chlorid calculated by Camac. The proportions of carbonic-acid-forming constituents, HCl 25 per cent., 6 ounces; HCl 25 per cent. 7 ounces; HCl 25 per cent. 8 ounces, making three strengths of the latter, after the method recommended by Bezley Thorne, of London:

Bath No. 1: Sodium chlorid, 4 pounds; calcium chlorid, 6 ounces.
Bath No. 2: Sodium chlorid, 5 pounds; calcium chlorid, 8 ounces.
Bath No. 3: Sodium chlorid, 6 pounds; calcium chlorid, 10 ounces.
Bath No. 4: Sodium chlorid, 7 pounds; calcium chlorid, 10 ounces; sodium bicarbonate 1/2 pound; HCl (25 per cent.), 12 ounces.
Bath No. 5: Sodium chlorid, 9 pounds; calcium chlorid, 11 ounces; sodium bicarbonate, 1 pound; HCl, 1 1/2 pounds.
Bath No. 6: Sodium chlorid, 10 pounds; calcium chlorid, 12 ounces; sodium bicarbonate, 2 pounds; HCl, 3 pounds.

The alkali should always be slightly in excess, unless a porcelain or paper tub is used.

In preparing the baths, the salts, including the right proportion of bicarbonate of sodium, are dissolved in the water which should have a temperature of 95° F. (35° C.) gradually reduced at successive baths to 82° F. (27° C.). A course consists of from 15 to 20 baths. The bottle containing the hydrochloric acid is inverted and lowered until its mouth is below the surface, when the stopper is withdrawn and the bottle moved about so as to diffuse the acid as uniformly as possible through the water. In this way the bath is made ready in a few minutes. The carbonic acid is the most unsatisfactory feature of the artificial bath, since it is rapidly dissipated, and produces only feebly the effect of the acid in the natural baths. Hence the patient should be promptly put into the bath after the HCl is added, lest the CO_2 is lost before he can get the effect of it. A good plan is to give the baths on alternate days, using the weaker until its effects are exhausted, then passing on to Nos. 3 and 4 in the same manner. Nos. 5 and 6 are not often called for.

As already stated, the baths are most efficient in cardiac disease, but they are also useful in renal affections. Their immediate effect is a diminished pulse-rate, intensified heart-sounds, diminished breathing-rate, while the dilated heart is reduced in size—under favorable circumstances to

almost its natural limits. The effect is also to increase the action of the kidneys and that of the skin. These effects are apparent in a free flow of urine, which may continue for days and weeks. Metabolic changes are accelerated and improved; the deep-seated organs, especially the liver and pelvic viscera, are relieved of congestion; while the heart, relieved of its burden, and contracting strongly, derives from its improved coronary circulation material for the repair of weakened and damaged tissue.

Theodore Schott[1] has shown that an increase of hemoglobin in the blood succeeds upon the systematic use of Nauheim baths.

The exercises are not usually commenced until some very positive effect of the baths is secured, when they are associated with the baths or substituted after the latter are discontinued. The effects of these gymnastics are described as identical with those of the baths. The extremities become warm, the breathing is deepened, the sense of oppression is relieved, the pulse become slower, the dilated heart area reduced. Even the liver, which is so often enlarged in heart disease as a result of passive congestion, is said to be reduced in size.

The following condensed statement of the Schott movements is from Sir T. Lauder Brunton's "Lectures on the Action of Medicines," London, Macmillan and Co., 1898.

"I. The arms are to be raised slowly outward from the side until they are on a level with the shoulder. After a pause they should be slowly lowered.

"II. The body should be inclined sideways as much as possible toward the right, and then to the left.

"III. One leg should be extended as far as possible sideways from the body, the patient steadying himself by holding on to a chair. The leg is then dropped back. The same movements are repeated by the other leg.

"IV. The arms are raised in front of the body to a level with the shoulder, and then put down.

"V. The hands are rested on the hips, and the body is bent forward as far as possible, and then raised to the upright position.

"VI. One leg is raised with the knees straight, forward as far as possible, then brought back. The movement is repeated with the other leg.

"VII. With the hands on the hips, the body is twisted round as far as possible to the right, and then again to the left.

"VIII. With the hands resting on a chair, and the back stiff and straight, each leg is raised as far as possible backward, first one and then the other.

"IX. The arms are extended and the fists supinated. The arms are then extended outward, next inward at the height of the body.

"X. Each knee is first raised as far as possible to the body, and then the leg is extended.

"XI. This movement is the same as IX, but with the fists pronated.

"XII. Each leg is bent backward from the knee and then straightened.

"XIII. Each arm is bent and straightened from the elbow.

"XIV. The arms are brought from the sides forward and upward,

[1] "On some Hemoglobin Investigation." Reprint from the "British Medical Journal," 1904.

then downward and back as far as they will go, the elbows and the hands being straight.

"XV. The arms are put at a level with the shoulder, and then bent from the elbow inward and again extended.

"XVI. With the arms in front at the level of the shoulder and the hands stretched, the arms are opened out sideways and then brought together.

"XVII. The arms are bent from the elbow outward and extended.

"The movements should be slow and regular, each one being gently resisted by the nurse or attendant or by the patient himself, putting into action the muscles opposing the movement.

"There should be a pause of half a minute between each movement of the same class and a pause of one or two minutes between movements of an entirely different kind, as I and II."

Use of Remedies in Cardiac Decompensation.

Digitalis.—The preparations of this drug are the most useful and sometimes the least to be depended upon of any set of drugs in the Pharmacopeia. Digitalis when used with thoughtfulness, in active preparations is life saving. When used without proper considerations and in inactive preparations, not only may it fail to save life, but by the thoughtless use, or the use of a weak preparation, it may allow a fatal termination to a case which otherwise might have recovered.

In this age with commercial drug houses of known good reputations, fitted with laboratories and apparatus to efficiently test the value and strength of the preparations they make, whose word may be depended upon, there is not the slightest excuse for the general practitioner to use old or inefficient preparations. Tablets of the tincture and the hypodermic tablets of digitalin, are not to be depended upon.

Tincture of digitalis should be not more than one year old and should be tested physiologically. If this is used as a fat-free tincture it may be used hypodermically when occasion requires, without the slightest fear of abscess formations and without an extreme amount of pain, and certainly with good effect.

The infusion of digitalis must always be made fresh from leaves which are assayed and physiologically tested. When so used it unquestionably is the best form of digitalis to use when one desires a diuretic action plus the action on the heart muscle. The so-called infusions sometimes made up from an extract of uncertain strength are thoroughly unreliable.

The various forms of digitalis derivatives, which are upon the market, must be used with some degree of caution. Each manufacturer claims that his preparation of digitalone, digitoxin, or digitalin, or whatever it may be, acts better than any other form of digitalis. This when weighed in the balance of clinical experience, is found to be distinctly not true. There are very few of the various preparation which have not been tested by us. The ordinary preparation of digitalin is absolutely without any value, notwithstanding the fact that the manufacturer makes such great claims for its action. Digitoxin, or its solution digalen, is the preparation for which

the manufacturers claim all the merits of the digitalis preparation. It is of some value, but it is not the fact that it can be used hypodermically without untoward effects.

Digipuratum, like the other preparations, may be tried.

When the actual effect of digitalis is desired, either the tincture, the fluid extract, the powdered drug, or the infusion should be used.

The more important action of digitalis is that upon the heart muscle. The systole is strengthened and prolonged, and the diastole is shortened. It is because of this action upon the heart muscle, that digitalis is such an extremely valuable drug. It has, however, also an action upon the central nervous system, an inhibitory upon the medullary centers, and this action tends to lengthen the diastole, instead of shorten it, but this does not happen unless large doses are taken and signs of poisoning occur.

According to Cushny "the blood pressure does not seem to be augmented in man to any extent preceptibly by the methods in use for measuring it clinically, and in some instances it has been decidedly reduced."

One of the unfortunate effects of digitalis is local action upon the stomach. It is a well-known clinical fact, that when this drug is taken for a prolonged time or in large doses, vomiting occurs. This is probably the direct result of its local action, and not due to its general poisoning effect, although if the dose has been large enough and the drug has been taken during a long period of time, unquestionably some of the vomiting and other untoward symptoms will come about because of the central nervous condition.

Therapeutic Indications.—The great indication for the use of digitalis is cardiac insufficiency as indicated by the symptom complex of decompensation, dyspnea, cardiac palpitation and edema. Yet all attacks of cardiac insufficiency thus characterized are not helped by the administration of digitalis; indeed, some such cases distinctly contraindicate the use of the drug. The insufficiency which comes from long-continued fever, from septic infection, etc., is but little helped by its use, though certain cases appear to be better for its use.

Cardiac disease, valvular disease, when the heart is fully compensated and there is only the cardiac murmur to indicate any trouble, does not demand the use of digitalis, indeed, this drug is distinctly contraindicated under such conditions. If there is one error which we physicians are prone to commit, it is to administer digitalis on the mere presence of a heart murmur. Indeed the total examination of a heart to some seems to be summed up in the act of auscultation, to discover whether or not a murmur exists. Nothing can be further from the true cardiac examination.

Patients with cardiac dilatation, whether the failure of the left heart is due to valvular disease or to a primary myocarditis, should not be given digitalis when first seen. Instead the patient must be absolutely still in bed for at least twenty-four hours, so that important therapeutic measures may be tested, and to avoid the danger of overstimulating a heart muscle which is already working to the limit of its capacity. Then the digitalis can be given tentatively, in small doses at first, gradually increasing the dose and feeling one's way with it until the proper degree of stimulation may be obtained. Some patients with primary myocarditis are distinctly

harmed by the use of digitalis. There is no "hard and fast rule" by which one can determine the proper cases of this condition in which to use digitalis. As a broad rule, however, those patients with extreme irregularity, anginal pains, and very low pressure, contraindicate the use of the drug. The only safe procedure, however, is to follow the above rule, put the patient to bed and feel one's way with the drug.

A most improper use of digitalis is its use in cases of broken compensation of slight degree while the patient is on foot and about his business. It is improper, notwithstanding its good symptomatic results, because occasionally the drug stimulates the heart muscle and makes it work against the various local congestions, against the overexertion which is the cause of the symptoms, instead of removing the cause by rest, and then using the drug to tone up the muscle.

After a patient with cardiac symptoms who has gone through a siege of decompensation has recovered, so that it is proper to begin exercise, then digitalis may be continued for some weeks in order to continue the good effects upon the heart muscle. Here, however, one must be most careful to stop the drug when the heart can safely do without its aid; failure to observe this caution will most surely land one exactly in the position he would be, if the drug were used in the beginning of a case before symptoms of failure.

Tachycardia from nervous conditions, from an essential myocarditis, or from any cause save that of a failure due primarily to valvular insufficiency, is not favorably affected by digitalis. Patients with cardiac failure, with much edema, with scant urine, frequently respond better to full doses of the infusion than to any other form of digitalis.

The cumulative action, so much talked of especially in former years, is certainly rare. Usually too much digitalis is indicated by nausea, vomiting, vertigo, the heart becoming rapid and feeble.

To conclude, digitalis must be used in a physiologically tested and freshly prepared specimen. It must not be used in heart disease without symptoms. It is of little value and sometimes harmful in inorganic conditions and in chronic myocarditis.

It is life saving or useless, according to the efficiency of the preparation and the method of its use.

Strophanthus.—This may be used in the same manner and under the same circumstances as digitalis. Strophanthin given intravenously in 1/100 grain doses, is often of extreme value, especially in acute dilatation.

Caffein.—Caffein may be given in the form of the alkaloid. In the form of benzoate of caffein, it may be used hypodermically.

Camphor.—Camphor is an excellent stimulant of the heart, and may be used in the form of camphorated oil by the hypodermic method. When so used it is better to use the sterile ampoules put up by various firms, unless it can be made fresh by some pharmacist experienced in making sterile solutions.

Nitro-glycerine.—This is not a heart stimulant and has little place in the treatment of cardiac disease. It should be used when peripheral pressure is high and only in conjunction with digitalis.

Theobromin.—Theobromin, as stated above, is of great value in certain cases of cardiac decomposition accompanied by œdema.

Treatment of Dyspnea.—As the dyspnea is primarily the result of deficient blood aeration in the congested lungs, the same remedies which force the blood through these organs, and thus relieve the congestion, tend also to relieve the dyspnea, and often do so. When the dyspnea persists, it is frequently caused by effusions into the pleural cavity, which are most promptly and successfully removed by tapping. Repeated tapping may be necessary. Dyspnea not thus relieved demands an opiate, and of opiates under these circumstances, morphin is the best. One-fourth of a grain (0.0165 gm.) at bedtime, by the mouth or hypodermically, gives comfort. It should not, however, be given constantly, but at well selected intervals. Hoffmann's anodyne, given in fluidram doses (3.5 c.c.), will sometimes relieve the milder degrees, and should perhaps be tried first, as it is always desirable to put off the use of morphin as long as possible. Paraldehyd may be substituted for Hoffmann's anodyne in the same doses. Chloralamid is even a better remedy in 30 grain (2 gm.) doses. Sulphonal may be tried in full doses of 15 to 30 grains (1 to 2 gm.). Trional in the same doses is a similar drug. So is veronal in somewhat smaller doses, 7 to 10 grains (0.462 to 0.66 gm.). None of these is an anodyne. They are simple hypnotics, and cannot be expected to take the place of morphin, though they may be tried at first. All the coal-tar products are more soluble in hot liquids, of which milk is a typical form. Inhalations of oxygen should not be forgotton as sometimes giving signal relief in dyspnea.

Treatment of Dropsy.—In like manner the measures that relieve the congestion and dyspnea tend also to relieve the dropsy, but special means are also necessary. Here it is that full doses of digitalis are especially indicated, and at closer intervals. It is to be remembered, too, that the infusion is a better diuretic than the tincture.

But these measures are often insufficient. They may be materially aided by restricting the ingestion of liquids. With the tissues water-logged and secretion insufficient, it is plain that copious liquid ingestion only increases the difficulty. This refers to cases in which there is general dropsy which resists the ordinary treatment. It is sometimes better to omit solid food altogether and reduce the liquid to a minimum that will sustain life—not more than three ounces every two hours, and that only during the waking hours. To this may be added the use of purgatives. While diuretics sometimes fail us, we can always secure an effect from purgatives. A daily morning dose of Epsom salt or Rochelle salt or compound jalap powder is given. Elaterium or its active principle elaterin is valuable. The latter is less apt to produce nausea. Its dose is 1/40 to 1/10 grain (0.00165 to 0.0066 gm.), while that of elaterium is 1/10 to 1/2 gr. (0.0066 to 0.033 gm.). Then, when action of the bowels begins, full doses of digitalis, caffein, or spartein, associated with nitroglycerin, are almost sure to be followed by copious diuresis; and when diuresis starts up in these cases, it is astonishing what quantities of urine are passed. The association of nitroglycerin with digitalis at this stage may be helpful. The object of nitroglycerin is to dilate the arterioles and allow the blood to move freely through them; 1/100 to 1/50 grain (0.00065 to 0.0013 gm.) may be given as

often as the digitalis and simultaneously. One need not be afraid of this drug. To be of value it must be given in large doses frequently repeated. Elimination by the bowels and kidneys being simultaneously stimulated, the disappearance of the interstitial fluid is greatly favored and often rapidly brought about. If these measures be associated with paracentesis of the chest, which may be required, the diuresis set up is often enormous, while the swelling rapidly declines. As diuresis is established or hunger sets in the quantity of milk allowed may be increased, and when the dropsy has entirely disappeared, a cautious return to solid food may be permitted.

A time-honored remedy in the treatment of cardiac dropsy which should not be overlooked is the combination of calomel, squills, and digitalis, the so-called Niemeyer pill, in doses of 1/2 grain (0.03 gm.) of the first and 1 grain (0.065 gm.) of the second and third every three or four hours; this is sometimes most happy in its results. Still another remedy often very efficient in this form of dropsy is theobromin. It is obtained from cacao, and is chemically closely allied to caffein. Like caffein, theobromin is a renal diuretic as well as a heart tonic. The dose which is most satisfactory is 30 grains (2 gm.) in the 24 hours, conveniently divided into doses of 7 1/2 grains (0.5 gm.) every six hours. Larger doses may be given. A better diuretic of the same class is aceto-theocin (double salt of acetate of sodium and dimethyl-xanthin sodium). It may be given in doses of 4 to 8 grains (10.25 to 0.5 gm.) three to four times daily in capsule or solution.

Caffein is an excellent heart tonic and although not so reliable as a diuretic should not be forgotten. It is often given with good results alternating with the infusion of digitalis. It may be used hypodermically in shape of caffein sodio-benzoate in 3 grain (0.2 gm).

In many cases there comes a time when the measures above described become inoperative. Diureties will not act and purgatives are insufficient. Then it is that incisions in the anasarcous legs or Southey's tubes inserted in the legs for draining off the liquid effusion are sometimes very efficient. I generally prefer the incision, an inch long behind the inner malleolus of each leg. Enormous quantities of fluid are thus drained off after which diuretics again become active, and I have seen many a case rescued from the grave for a time at least. It is most important that antiseptic dressings should be employed to protect the incisions against infection.

Treatment of Irregularity of Heart Action and Palpitation.—For these symptoms, in addition to the cardiac tonics mentioned, belladonna is also a useful remedy. It may be combined with digitalis. A good belladonna plaster placed over the palpitating heart is one of the most efficient agents in subduing it. Nitroglycerin as already mentioned is often very useful to the same end—1/100 grain (0.00065 gm.), rapidly increased to 1/50 grain (0.0013 gm.), every four hours or oftener. It must be remembered that nitroglycerin is not a heart tonic but is of value in heart disease only when one desires to lower peripheral pressure in order to make the tonic action of digitalis more effective. It may also be combined with digitalis, as previously directed. Cardiac pain is also sometimes relieved by the same remedies. For this, however, hypodermic injections of morphin are sometimes necessary while palpitation may require the same drug.

Treatment of Sudden Acute Cardia Dilatation.—Immediate absolute rest

either in bed or on a chair is imperative. Here a hypodermic of 1/8 of a grain of morphine with 1/150 of a grain of atropin will frequently save life. Rest after the attack must be insisted upon, together with the use of digitalis and other remedies spoken of above.

DISEASES OF THE MYOCARDIUM.

The heart is subject to alterations in its muscular substance independent of valvular defect. Hypertrophy, dilatation, fatty infiltration, and fatty metamorphosis or true fatty degeneration, and atrophy are the most important. Acute myositis, abscess, and aneurysm of the walls of the heart are such rare conditions that they need only be mentioned in passing, especially as there is no way to recognize them before death.

Hypertrophy of the Heart.

Etiology.—Hypertrophy implies an overgrowth of muscular tissue, and is naturally the result of extra work, increased effort to overcome increased resistance, whatever its cause.

The resistance needed to excite increased action may be from within or from without, or due to nervous influence. Resistance from within is occasioned by obstruction to the outflow of blood from the heart at one of its orifices, or to increased intravascular pressure. Such obstruction is offered in the case of the *left ventricle* by aortic stenosis, congenital narrowing, aortic insufficiency, and mitral insufficiency. Increased intravascular pressure is caused by endarteritis and resulting sclerotic changes in the vessel-walls, by strong contraction stimulated by the irritation of toxic substances in the blood, such as accumulate in Bright's disease, or as the result of overeating or excessive drinking, especially of large quantities of beer; finally, by excessive physical exertion.

External obstruction to the contraction of the left ventricle is found in pericardial adhesions and *myocarditis*. Such hypertrophy is always eccentric. Hypertrophy of the left ventricle from nervous influence is seen in exophthalmic goiter and allied conditions, and in long-continued palpitation. Constant mental excitement is a possible cause.

In the case of the *right ventricle*, internal resistance is produced by pulmonary congestion due to mitral regurgitation or to mitral stenosis, to narrowing of the pulmonary vessels, such as occurs in pulmonary emphysema. Valvular lesions of the right side of the heart produce hypertrophy of the right ventricle, just as those of the left cause it.

Auricular hypertrophy is always eccentric—that is, while the walls are thickened, the cavities are also dilated. Hypertrophy of the left auricle is usually caused by stenosis of the mitral orifice, and to a less degree is a result also of regurgitation of the blood in incompetency of the mitral valve. Hypertrophy of the right auricle might also be expected as a consequence of regurgitation of blood from the right ventricle to the right auricle, but the resistance to the further backward flow into the veins is so much less than in the left side disease, hypertrophy is correspondingly less frequent. In like manner, even if stenosis of the tricuspid orifice is present, the same conditions prevent any marked degree of hypertrophy of the right auricle.

In all cases of hypertrophy due to disease of the valves it is likely that a certain amount of distention of the heart cavity by blood precedes the muscular overgrowth.

Symptoms.—Hypertrophy, being a process of compensation, is not at first attended by any symptoms. It is the result of a conservative effort of nature, by means of which symptoms are averted. But unlike the hypertrophy of the muscles of the blacksmith's arm, it tends ultimately to degeneration, and thus becomes the initial link in a chain of evil which is well stated by J. G. Adami:[1] "In the first place, it leads to an increased nutrition of the walls of the *arteries;* increased nutrition leads to increased connective-tissue growth of the walls; increased fibrous tissue of the walls leads to contraction and increased rigidity of those walls; the increased rigidity leads to increased resistance to the passage of the blood current. The increased resistance requires increased propulsive power on the part of the ventricular muscle—that is to say, increased work; the increased work of the heart leads to overgrowth and hypertrophy, and with this, heightened blood pressure and further increased nutrition of the walls, and now, at last, the stage is reached, this vicious circle continuing, in which either the vessel walls give way or the heart." From this standpoint increased blood pressure alone is sufficient to explain in come cases the anatomical changes—*i. e.*, the arterial sclerosis, atheroma, and fibroid thickening so constantly seen in valves and heart-walls without calling on chronic inflammation or specific agency. Certain it is that the two conditions react on each other, and it is more than likely that the former (increased blood pressure) may produce the latter (chronic inflammation) *de novo*, and many otherwise unexplained facts are rendered clear.

With degeneration we begin to have symptoms which are at first intermittent, brought about only by some temporary cause which excites the heart, such as exercise, mental emotion, fatigue, mental or physical, tobacco, or alcohol. There is a feeling of vague discomfort about the heart, seldom amounting to pain, sometimes increased when the patient lies on the left side. To this may be added palpitation, a consciousness of the beating of the arteries in the head, dizziness, headache, ringing in the ears, flushes or flashes of light, and a tendency to hemorrhage of the nose. So long as the integrity of the heart muscle is maintained the blood pressure in certain cases is increased and reaches 160 to 200 or more mm.; as the heart begins to degenerate the blood pressure rapidly falls.

Physical Signs.—While symptoms other than physical signs may be wanting, the latter are present from the beginning, increasing with the duration of the hypertrophy. These have been for the most part considered when treating of valvular diseases. *Accentuation of the aortic second* sound in hypertrophy of the left ventricle and of the *pulmonic* second in hypertrophy of the right ventricle should be spoken of.

Dilatation of the Heart.

Definition.—This has already been defined, so far as the state of the chambers is concerned. Dilated heart is of two kinds; first, acute

[1] "Notes upon Cardiac Hypertrophy," "Montreal Medical Journal," May, 1895.

dilatation; and, second, chronic dilatation, or dilatation accompanied by fatty degeneration. Of the latter, two varieties exist: (1) That succeeding valvular disease; (2) that succeeding hypertrophy due to muscular effort, especially when associated with alcoholic intemperance and other forms of dissipation. Acute dilatation may be unassociated with structural change, except as to mechanical arrangement of the muscular elements. Cloudy swelling may be present. The latter may be associated with fatty change.

Etiology.—Chronic dilatation is the last stage in a valvular disease the result of failing nutrition. The conditions under which this manifests itself have been described. Acute dilatation without valvular defect is the result of prolonged muscular effort, such as occurs in rowing, running, and mountain-climbing. Moderate degrees of distention occur with any decided muscular effort. The more marked degrees capable of mischievous consequences are the result of prolonged severe muscular exertion. The effect of moderate, well-regulated exercise on the heart, known as training, by which endurance is developed, is to produce eccentric hypertrophy, or hypertrophy with dilatation, which is not dilatation in the sense under consideration—enlargement of the cavity with thinning of the walls. The right heart is the seat of such dilatation. In overexertion the harmful effect of excessive acute strain is averted for a time by the safety-valve action of the tricuspid valve, permitting a regurgitation of blood into the right auricle. Dilatation has exceeded its physiological limit when the cavity is no longer able to empty itself of blood. While moderate degrees of acute dilatation may be recovered from, either rapidly or slowly, dilatation may be carried to degrees at which recovery is impossible and death results. Such results have followed rowing and mountain-climbing.

The so-called *irritable heart*, to which attention was first called by J. M. Da Costa in a graphic description based on a study of the cases of soldiers in the American Civil War, is an example of an abnormally dilated heart, a heart in which compensation has failed. Sudden dilatation may happen to hearts whose muscular substance is degenerated, though seemingly hypertrophied, as in chronic Bright's disease, where overexertion often brings on dilatation. In a few instances in malignant forms of the infectious diseases, such as scarlet fever and diphtheria, the nutrition of the heart may be so rapidly impaired by the toxic agency which causes the disease that dilatation occurs with very little or no undue intravascular pressure. All these belong to the second category, that of chronic or slow dilatation.

Symptoms.—The symptoms of "heart strain" are sudden *pain* in the region of the heart or epigastrium, *faintness*, *shortness of breath*, and *rapid, feeble action of the heart*. If it be not immediately fatal, the symptoms may pass off, but are renewed on the slightest exertion. In the acute cases described as due to the toxic causes of infectious disease, sudden death may be the only symptom. In some cases it may be preceded or not by very brief precordial distress. Less serious degrees may be associated with *faintness* or *palpitation* on exertion, *extreme feebleness* of the heart's action, and *dyspnea*. It is rather characteristic for these symptoms to pass away when the patient is at rest, to be renewed on the slightest exertion.

Symptoms growing out of dilatation of the heart, going also to make

up the sum of those constituting chronic valvular disease with failure of compensation, are *general venous congestion, dropsy, feeble, frequent, and irregular radial pulse*—rarely, on the other hand, a *slow pulse*. Frequent and irregular pulse may be due to impaired pneumogastric inhibition the result of anemia of the brain, slow pulse to scanty nutrition and a loss of the natural irritability of heart muscle. To anemia especially affecting the medulla oblongata may be ascribed Cheyne-Stokes breathing, also a symptom of the terminal stage of the disease. To it may be ascribed, too, symptoms simulating apoplexy, which characterize the slower dying in some of these cases. Palpitation, angina pectoris, and dyspnea—cardiac asthma, with syncopal attacks, coldness, and slow pulse (30 to 40)—are all symptoms more or less associated with dilatation of the heart. It is further characteristic of these symptoms of dilatation that they are often not transient or amenable to treatment by the usual heart tonics, of which digitalis is the type.

In some instances, especially in the dilated heart of pernicious anemia, there may be a full, strong, and regular pulse, but in the majority of cases the blood pressure is lowered.

High-colored, scanty urine of high specific gravity, sometimes containing hyaline casts and blood-disks, also result from cardiac dilatation.

Physical Signs.—When the termination is not immediate, physical signs may be recognized. To *inspection*, the impulse, if visible, may be diffused over a wide area, but is feeble and fluttering, a point of greatest intensity or an apex-beat being often wanting. At times it is found higher up and to the left of its normal position. If the right heart is chiefly involved, the beat as far as caused by the left apex, is completely wanting, while an impulse may be felt below or to the right of the ensiform cartilage, as well as a wavy impulse in the fourth, fifth, and sixth interspaces to the left of the sternum. A pulsation may be seen in the second left interspace, which, while sometimes presystolic, is commonly systolic. In the latter event it may be a further expansion of an already dilated auricle by blood regurgitating during systole of the left ventricle; or if presystolic, it may be the pulse of auricular systole. Such at least are possible explanations. The fact that at autopsies, even in extreme dilatation, the left auricle is found so far back from the thoracic wall as to be scarcely able to beat against the second interspace, does not preclude the possibility of this during life. In dilatation of the *right* auricle, on the other hand, there is sometimes seen an impulse in the third interspace on the right side which is clearly systolic and due to regurgitation from the right ventricle during its systole. The pulsating symptoms described in this paragraph are commonly seen in persons only with thin chest-walls. A vauable diagnostic sign is the sharp, short *first* sound resembling the second sound due to the loss of the muscular quality.

To *percussion* there is increased dullness to the right and downward toward the epigastrium or to the left beyond the normal line, though these boundaries may be obscured by an emphysematous lung. The results of *auscultation* are greatly influenced by complications. If cardiac murmurs are present, they may obscure all else. On the other hand, previous murmurs may disappear. The impulse is feebly heard as well as felt; the

first sound is feeble but pure— that is, shorter and more like the second, lacking, as it does, the muscular element. It may be scarcely audible, even in the absence of murmurs. It is sometimes reduplicated because of asynchrony in the action in the two halves of the heart. Sometimes there is a loud systolic murmur at the apex, due to relative insufficiency of the mitral valve, the true nature of which becomes apparent only in the event of its disappearance. The second pulmonic sound may remain sharp if there is dilatation only of the left ventricle and there is compensatory hypertrophy of the right; feeble if the right ventricle is involved. Finally, there is intermittent and irregular action; at times the characteristic *gallop rhythm*,[1] which is almost pathognomonic of dilatation, is present. The pulse is very rapid and feeble.

Diagnosis.—An acknowledged difficult matter at times is the distinction of *pericarditis with effusion* from the dilated heart. Whether inspection furnishes any information, depends mainly upon the stoutness or leanness of the patient. In the stout person nothing is recognizable in either condition. In the thin-chested the impulse is visible and wave-like in dilatation; it is not visible, or barely so, in pericardial effusion. The same is true of palpation, except that, if the patient leans forward, the impulse may be felt in pericarditis.

Percussion affords the most valuable information. If it brings out the well-known triangular shape of dullness, with the apex toward the inner end of the left clavicle, and the base in the fifth or sixth interspace, especially in the absence of a cardiac impulse, there must be *pericardial effusion*. Percussion of the dilated heart elicits a quadrangular shape or triangular with the apex downward. To *auscultation*, while the heart-sounds have lost their characteristic sharpness, they still contrast with the distant and muffled sounds in pericardial effusion. Especially if there is left any of the original hypertrophy, the second sound will retain some of its sharpness, while, if there happens to have been valvular disease, the murmurs remain to help us.

Bamberger's sign, described on p. 557, must be sought for in evidence of pericardial effusion. There may be encroachment on the lung in dilatation, but it is very much less in dilatation than in pericarditis with effusion. This encroachment in the case of dilatation does not give rise to Skodaic resonance in the axilla. While there is shortness of breath in both, it is less pronounced in dilatation and more influenced by exertion, being less while the patient is quiet.

Prognosis.—This is ultimately fatal in chronic dilatation; in fact, the stage of dilatation is the stage in which remedies become unavailing. At the same time, marvelous results sometimes follow treatment. We have seen general anasarca with effusions in the serous cavities disappear when least expected, so that one is never justified in giving an unqualifiedly unfavorable prognosis. In acute dilatation the prognosis depends upon the degree of the stretching and degenerating. If they are extreme, death follows instantly. If not followed by immediate death slow improvement and ultimate recovery are possible.

[1] For explanation of gallop rhythm see Barth and Roger, "Traité Pratique d'Auscultation." Thirteenth Edition, Paris, 1898, p. 352.

DEGENERATIONS OF THE CARDIAC MUSCLE.

The heart muscle is subject to parenchymatous degeneration, to fatty degeneration, to fatty infiltration to amyloid degeneration, to the hyaline transformation of Zenker, to calcareous degeneration, and to the changes known as brown atrophy.

PARENCYHMATOUS OR ALBUMINOID DEGENERATION (CLOUDY SWELLING).—This is a change in which the sarcous substance is converted into granular matter of albuminoid composition, which produces also more or less indistinctness in the striated appearance of the fasciculi. The albuminoid composition of the product is attested by its solubility in acetic acid, and its insolubility in either. The general effect is one of softening and flaccidity.

It is ascribed to some toxic agency, and occurs most frequently in the infectious fevers—typhoid fever, scarlet fever, diphtheria, and the like. It was at one time considered a consequence of high temperature, but this view is no longer held. It is believed also to be, at times, at least, the first stage of fatty degeneration, or to precede fatty degeneration. It is certainly at times associated with it. Cloudy swelling may disappear and the muscle resume its natural histology.

FATTY DEGENERATION OR FATTY METAMORPHOSIS.—In this change, the sarcous substance of the muscular fasciculi is directly converted into globular fat, as contrasted with the condition of fatty infiltration, in wnich the fat is deposited between the fasciculi. The little fat drops—and they are very minute, as a rule—are seen in rows parallel to the fibrillæ of the fasciculus, and all transverse striation has disappeared. As intimated, the cause of such degeneration is an interference with the proper nutrition of the heart muscle.

It may be *general*, when it has its most frequent expression in the dilated heart which succeeds upon hypertrophy, involving the walls of one or more cavities. It is also a result of the impaired nutrition of old age, of the grave infectious diseases, and of cachectic states generally—such, for example, as pernicious anemia. In the infective diseases and cachexias it may be associated with parenchymatous degeneration or succeed upon it. It is also a result of the action of certain poisons, as phosphorous and arsenic, the effects of which may extend to other muscular organs. Under these circumstances, the heart is generally enlarged (dilated), flabby, and relaxed, of a light yellow or yellowish-brown color, and very friable, permitting the finger to be easily poked through it. The papillary muscles and the trabeculæ in the left ventricle may be the seat of circumscribed fatty degeneration, and be dotted and streaked with yellow, fatty matter. Unlike parenchymatous degeneration, fatty degeneration, when once established, is considered irremediable.

Fatty degeneration of the heart may also be *circumscribed* in small foci variously distributed. Thus, it may be confined to the superficial or subpericardial layers, when it is especially the result of pericarditis. Or there may be numerous pinhead-sized foci in the subendocardial layer in cases of extreme dilatation.

Finally, there may be a single focus in the substance of the left ventricle of the septum, due to total obstruction of one of the branches of the coronary artery, usually the anterior, by a thrombus or embolus. The product is an area of fatty degeneration known also as *anemic necrosis*, or *white infarct*. In the early stage the infarction is brownish-yellow or hemorrhagic. Minutely examined, the muscular fasciculi are without nuclei, and later they break up into a cheesy detritus. The infarct is not always thus made of fatty débris, but may present a hyaline appearance. It may be the seat of rupture, and thus cause hemorrhage into pericardium, and immediate death.

Diagnosis.—The diagnosis of fatty degeneration, so far as recognizable, is that of dilatation, slight degrees and circumscribed fatty degeneration being unrecognizable, while considerable areas of partial degeneration may also exist without exhibiting symptoms. In fact, the presence of some dilatation of the cardiac cavities seems to be necessary to the production of symptoms—the feeble pulse, palpitation, and dyspnea being symptoms of the dilatation, rather than the fatty degeneration,

Prognosis.—This is grave. It is impossible to restore the degenerated muscular substance to its natural structure. With degeneration established death is liable to occur suddenly, and remedies which avail with an integral organ are useless here.

Treatment.—This embraces that of cardiac dilatation. Acute attacks should be met by stimulants, of which alcohol, aromatic spirit of ammonia. Strychnin is also indicated, and may be used hypodermically.

Fatty Infiltration or Fatty Overgrowth.—Strictly speaking, this condition is not a degeneration of the heart muscle, though it leads ultimately to fatty metamorphosis. It is the *cor adiposum* of the older authors, and differs from fatty metamorphosis in that the fat is infiltrated *between* the muscular fasciculi. In the true *cor adiposum*, the fat extends deep into the substance of the muscle, sometimes as far as the endocardium. It covers also the outside of the heart, at times so completely that the true muscular structure is invisible. This infiltration sooner or later interferes with the proper nutrition of the muscular substance, a true fatty degeneration results, with its symptoms, so far as any are manifested, and becomes ultimately also a cause of death.

The fatty infiltrated heart is commonly a part of general obesity, and occurs, therefore, at a time of life when this is usual—that is, between the ages of 40 and 70 years—and is more than twice as frequent in men as in women.

The condtion is inferred from the presence of extreme obesity associated with signs of cardiac weakness.

The **treatment** is that of obesity.

Amyloid infiltration invades the heart as it does other organs, attacking the blood-vessels and intermuscular connective tissue. *Zenker's hyaline transformation* attacks, on the other hand, the muscular fasciculi, causing them to appear swollen and transparent, and the striæ to be indistinct or absent.

Calcareous infiltration is a rare condition, in which the muscular fasciculi are infiltrated with lime salts.

MYOCARDITIS.

Chronic Myocarditis or Fibromyocarditis.

Synonyms.—*Fibroid Degeneration of the Myocardium; Fibroid Heart; Fibrous Myocarditis; Interstitial Myocarditis; Indurated Degeneration; Myodegeneration; Sclerosis of the Coronary Arteries.*

Definition.—A chronic disease of the cardiac muscle in which there is more or less substitution of the normal substance by fibroid or cicatricial tissue, either localized in patches or diffused throughout the organ.

Etiology and Pathology.—The condition is not, strictly speaking, inflammatory, the patches representing transformed areas of anemic necrosis, due to obstructive disease of the coronary arteries and branches. The disease in the coronary arteries is endarteritis, resulting in arterio-sclerosis. Through a diminished blood-supply, it causes degeneration of the muscular fasciculi, and their substitution by fibrous tissue. Only in the event of such diminished supply do the changes occur. Hence it is that arterio-sclerosis of the coronary arteries is not always followed by fibroid change. The causes of arterio-sclerosis of the coronary arteries are those of endarteritis elsewhere. The tendency to arterio-sclerosis is often hereditary. It is a disease also which seldom occurs prior to middle life, though sometimes seen surprisingly early. It might be said that it is natural to old age—one of its evolutional terminations. In pure, uncomplicated cases of myocarditis the valves are normal, while the muscle, on examination, is found dotted with *white, shining areas* present in varying numbers. Minutely examined, these are found made up of pure or partly fibroid tissue, the muscular substance being correspondingly destroyed. They are seated for the most part in *the left ventricle toward the apex* and in the *anterior wall*, though they may be found elsewhere. They may often be seen from the endocardial or pericardial surface as *cicatricial-like depressions*. Sometimes there is a single large patch known as a *fibroid patch*. The papillary muscles may exhibit the same fibroid change. Another cause of fibrosis is pericarditis which produces small and larger areas of degeneration in the shape of the milk-white fibroid patch on the surface of the heart and extending more or less into its substance. Mural endocarditis may produce similar patches on the inner surface of the heart.

The fibroid change may also be associated with valvular disease, the mechanical impediment to the movement of blood in this condition being the cause of a chronic venous congestion, which results in a fibroid infiltration; or the valvulitis may give rise to embolism of the coronary arteries or branches, thus cutting off nutrition. From the cardiac thrombosis which sometimes results there may arise cerebral, renal, and pulmonary embolism. Long-standing emphysema of the lungs results in similar congestion; so does obstruction of the pulmonary artery from any cause.

A further result of the fibroid change is dilatation of a part or of the whole of one of the heart cavities, producing in the former instance what is

known as *cardiac aneurysm*. Fibrosis may also be associated with hypertrophy without valvular disease, though the recognition of such combination before death must be a matter of inference, based on the presence of arterio-sclerosis elsewhere and of the causes of such hypertrophy.

Recently the term myocarditis has been extended to include fatty as well as fibroid change since the same symptoms may result from both.

Symptoms.—Slight degrees of fibroid change occasion no symptoms, while autopsies even disclose advanced stages of indurative myocarditis which were not suspected. In consequence of the frequent association, too, of endocarditis and pericarditis, the symptoms of these diseases are often combined and mask the distinctive symptoms of the fibroid change. Unmasked, the symptoms are, in a word, those of dilatation of the heart, including *dyspnea*, often so severe that the patient cannot lie down. With this may be associated *Cheyne-Stokes* breathing, commonly occurring during sleep. There may be *palpitation*, with *small, frequent*, and *irregular pulse*, or the pulse may be persistently *slow*. There is *precordial oppression*, with *attacks of faintness*, and, finally, *venous stasis* with *cyanosis, general edema, congestion of the liver, stomach*, and *kidneys, feeble digestion, scanty urine*, and *albuminuria*. These symptoms may set in gradually or suddenly. On such a heart, digitalis and other heart tonics are often without effect. *Angina pectoris* is also a symptom of indurative myocarditis, though it also occurs in other cardiac diseases, especially aortic stenosis. It will be described later.

Physical Signs.—Physical examination recognizes a *feeble impulse* which may be displaced to the left, oftcn scarcely appreciable, and, on percussion, enlargement of the cardiac area. The first sound lacks its muscular element and is morc like the second—more purely valvular, aud therefore short. Both sounds maintain for a time considerable distinctness, but ultimately grow feeble. Occasionally there may be a mitral murmur, which may be relative and transitory or permanent. Such murmur is explained by the experiments of Ludwig and Hesse, already alluded to, and more recently confirmed by Krehl. These go to show that a certain integrity of the muscles about the mitral orifice or of the papillary muscles is necessary to a complete closure of the latter. Such integrity is impaired by myocarditis, and the resulting murmur increases the difficulty of diagnosis. The murmur is systolic, soft, low pitched, heard at the fourth rib. There is, however, usually absence of accentuation of the pulmonic second sound characteristic of mitral regurgitation, though this may also be relatively present if the right ventricle happen to be less severely involved than the left. There may be systolic shock greater than would be expected from the feebleness of the cardiac impulse. The second sound is also sometimes reduplicated, or there may be gallop rhythm sometimes early. The mitral murmur in the fibroid heart is more variable and more subject to intermissions than that of mitral regurgitation due to valvular disease. The sudden addition of a mitral systolic murmur in a fibroid heart previously without murmur may also indicate a lacerated valve.

Diagnosis.—This is often difficult, requiring the opportunity of prolonged study of the case. For the most part, we are compelled to rely on the absence of the symptoms and signs of valvular disease, and the presence

of the symptoms of dilatation, the evidences of arterio-sclerosis elswhere, a persistently slow pulse, angina pectoris, the history of syphilis and of other causes, together with the age of the patient. When the fibroid condition is associated with murmurs, the diagnosis is still more difficult, and must, indeed, be a matter of probability, if even suggested, so much more likely are the signs to be interpreted as those of valvular disease, with which, however, the myocarditis may be associated. The presence of radial sclerosis is strongly confirmatory, but not essential.

Prognosis.—This is grave, or, to say the least, uncertain. Associated as it is with sclerosis and narrowing of the coronary arteries or branches, complete obstruction is liable to occur at any time, producing sudden death. On the other hand, the patient may live for many years with the heart the seat of considerable fibroid change.

Treatment.—This must mainly consist in treating the causes, and in a proper hygienic management. Habits of overeating and excessive drinking should be overcome. The avoidance of overexertion, associated with just sufficient exercise to develop the heart healthfully, should be observed. Outdoor life and a proper hygiene of the skin and body by bathing and massage are important.

Drugs which will remove the diseased condition of the coronary arteries and fibroid overgrowth do not exist. Still, the reputation of iodid of potassium as a remover of fibroid overgrowth and for the cure of syphilitic disease should be availed of. The iodid is also serviceable in producing vascular dilatation and facilitating the movement of the blood. For the symptoms of stasis and heart weakness, of dyspnea and of angina pectoris, the treatment is the same as that for these conditions under other circumstances. Digitalis is rarely indicated and may do harm.

The "Oertel cure," consisting in graduated hill climbing, proteid food and restricted intake of liquid, is recommended.

Acute Suppurative Myocarditis.

Synonym.—*Abscess of the Heart.*

This is a rare condition. It is always metastatic or pyemic in origin, in association with puerperal fever, malignant endocarditis, or other septic processes. It may occur in the septum, as well as the outer ventricular walls.

As such it is not recognizable before death, and is commonly discovered at autopsies. It may, however, rupture into the heart cavities, causing other metastatic abscesses, or into the pericardium, causing septic pericarditis and early fatal termination.

Aneurysm of the Heart.

This is a term given to two conditions:

1. A saccular projection from the ventricular surface of a sigmoid or cuspid leaflet, where the valve is weakened by ulceration through one of the lamellæ, the intravascular or intracardiac pressure furnishing the dis-

tending force. It is much more common in the aortic segments. The saccule may ultimately perforate, causing laceration of the valve.

2. Projection outward of a circumscribed portion of the muscular wall, which has been weakened by the fibroid patch or by an injury to the wall. Here, naturally, the left ventricle, too, suffers, and near the apex in more than half the cases. The resulting pullulation varies in size from 2/5 inch (1 cm.) or less to dimensions equal to those of the heart itself. The aneurysm may be sacculated or partitioned and even multiple.

There are no symptoms by which the condition may be recognized with any degree of probability. It may also terminate fatally by rupture into the pericardium.

Rupture of the Heart.

Rupture of the normally integral heart muscle does not occur. It is only when weakened by disease that such an event is possible. Fatty metamorphosis furnishes the most frequent predisposing condition, in 77 out of 100 cases collected by Quain. The softening due to obstruction of a branch of the coronary artery, as already described, and known as *massive softening*, is the most frequent cause of heart rupture, but the fibroid change, abscess, or ulceration, are all conditions which at times precede rupture. Morbid growths in the heart-wall, such as gummy tumor, cysts, and carcinoma, are also possible causes. Segmentation and fragmentation of the muscle has been observed but this condition is by some considered as of doubtful occurrence.

These preliminary conditions presupposed, any unusual strain, physical or mental, is sufficient to produce rupture, though this is not always necessary, especially in the case of the white infarct, where the degeneration is so great as to admit rupture with the ordinary pressure. It is naturally an event of the second and third half-centuries of life. It has occurred among the insane when perfectly quiet.

The anterior portion of the left ventricular-wall near the septum is the favorite seat. Rupture is rarely recognized before death, which usually follows in the course of a few hours.

The symptoms are precordial pain, a sense of oppression, dyspnea, pallor, pulselessness, and collapse. There may be enlargement of the cardiac area of dullness, owing to filling up of the pericardial sac, associated with feeble or absent apex-beat.

IRREGULAR ACTION OF THE HEART.

General Considerations.—According to Mackenzie the points proven by Gaskell in many papers and set forth in his article on the contraction of the cardiac muscle found in Shaefer's Physiology, edition of 1900, have completely revolutionized our interpretations of the symptoms found in certain diseases of the heart. Gaskell proved—*First* that the muscle fibers themselves have the inherent power of secreting a substance which will stimulate a fiber to contract.

Second.—They have the power of receiving a stimulus excitability.

Third.—The power of transmitting the stimulus to other cells, conductivity.

Fourth.—The power of contraction.

Fifth.—They have inherent tonicity.

Gaskell also calls attention to the fact that while all fibers of the heart are endowed with these properties, they do not develop to the same degree in all of them. Thus the venous end of the primitive heart has the power of stimulus production and excitability better developed than other parts, but if for any reason another portion of the heart is rendered more excitable, then the contraction will begin there. The primitive cardiac tube is represented in the mammalian heart by certain tissues. There is a very small mass of tissue at the mouth of the superior cava. This is believed to be the remains of the sinus venosus and has been called the sino-auricular node. It is believed that the heart's contraction probably begins at this point. Further remains of this tissue are found farther down, arising in the right auricle and passing across the auricular ventricular septum to be distributed in the ventricles.

This bundle of tissue now known as the bundle of His, "arises from a node of tissue, the A. V. node, situated in the right auricular wall near the mouth of the coronary sinus, passes over the auricular ventricular septum below the fibrous body and under the septal cusp of the tricuspid valve." It divides on the septum and is continued on the ventricles.

Gaskell, Erlanger and others have shown experimentally that the bundle conveys the stimulus from the auricles to the ventricles, and Mackenzie believes that if it chance that the bundle be rendered more excitable than the rest of the tissue, then it will start a contraction independently of the sinus rhythm.

While the functions of the heart muscle allow it to perform its movements independently, nevertheless nervous influence has a great power in affecting the action of the various fibers.

Mackenzie's groupings of the various types of irregularity are adopted and followed, retaining the division of nervous palpitation for clinical reasons only.

Increased Frequency of the Heart Beat.—The word tachycardia is loosely applied; some writers calling any sustained action of the heart above 120 a tachycardia. We will limit the term after Mackenzie to paroxysmal tachycardia, an organic condition. The normal number of heart beats varies with individuals in different ages and under certain circumstances. The average in infancy is from 130 to 140 per minute; in middle life from 70 to 80. At fifty it gradually increases. Normally the heart beat is increased by excitement, fright or exertion. As to the latter, an increase of ten beats when the individual rises from a prone position is perfectly normal; an increase of twenty or more would be decidedly abnormal, especially if the increase were sustained for some time. If a heart beat which is normally seventy is raised to ninety or over when the patient walks upstairs and especially if the acceleration continues some time, it usually means that the heart muscle is lacking in power and the heart is responding in number of beats instead of strength. The judgment as to whether an increase is abnormal or normal, however, must depend in a large measure upon the

usual performance of each individual heart. If the rate after a very moderate exercise is that which would naturally be expected after very violent exercise, certainly the condition must be abnormal.

The causes of this increased frequency on exertion are very numerous, myocarditis, dilatation as the result of valvular disease, any exhausting disease such as fevers and the anemias, the abuse of alcohol or tobacco and sexual excess, are among the most common.

Perhaps the most dangerous mistake is to consider an abnormally rapid heart where no evident cardiac lesion is found as nervous or neurasthenic. Doubtless some few cases are entirely extrinsic, but many such cases are due to a weakened heart muscle, the result of such a dyscrasia as tuberculosis. Certainly many cases of tuberculosis have among their first symptoms nervous disturbances accompanied by a rapid heart. Exophthalmic goiter has often a rapid heart action as its very first symptom, this rapidity of action being sometimes accompanied by a relatively high systolic blood pressure and low diastolic pressure.

Treatment.—Treatment of abnormal frequency of the heart beat will depend absolutely upon the cause; that of nervous palpitation will be discussed. If the case is one of organic valvular disease, the treatment is that detailed for that condition; if due to alcoholism, abstinence from alcohol will in great measure slow the heart; even the rapid heart of tuberculosis and exophthalmic goiter will respond to proper treatment for these conditions.

Diminished Frequency of the Heart's Action.—BRADYCARDIA, like its antithesis, tachycardia, is loosely applied. Before a case is considered to be a true slowing of the heart's action itself care must be taken to distinguish between a slow pulse and a slow heart. A radial pulse may be forty due to abortive systole or missed beat, and the heart itself be eighty or more.

Convalescents from acute fevers such as typhoid and diphtheria may have an extremely slow pulse. Certain poisons such as that affecting an individual with jaundice, chronic Bright's disease, cause a slow pulse. It occurs in certain diseases of the heart such as Stokes-Adams syndrome, where there is abnormal conduction of the stimulus from the auricle to the ventricle.

Finally, slow pulse may occur during labor and as the result of advanced age.

There seems to be no successful treatment except in those cases due to toxic agents, where the withdrawal of the poison will allow the normal rhythm to assert itself.

Irregular Action of the Heart. *Arrythmia.*—Gaskell has shown by experimentation, as before stated, that while the contractions of the heart muscle begin normally at the sinus, they may begin at any point in the heart which is more excitable than this usual point. This fact, proven experimentally by Gaskell and his followers, gives rise to the various forms of cardiac irregularity. It must be insisted at the beginning that the mere fact of irregular cardiac action does not carry with it any bad prognosis. Other characteristics of the heart beat are much more important. In certain young individuals, such for instance as those recovering from an infection, there is no danger in the mere irregularity. When the extra stimulus occurs at the origin of the cavæ, the heart is subjected to what

Mackenzie styles the *sinus irregularity*. It occurs frequently in the young after fevers and during slow respiration.

It occurs as a rule as the result of some irregularity of action of the vagus. Its symptoms are irregular pulse, frequently altered by respiration.

The heart sounds are clear and sharp. There is no murmur. The tracings show that the auricles are affected as are the ventricles, and therefore there is no trouble with the conducting bundle, there is no trouble with the heart itself.

Extra Systole.—Mackenzie would limit this term to those primitive contractions of auricle or ventricle in response to a stimulus from some abnormal point of the heart, but where otherwise the fundamental or sinus rhythm of the heart is maintained. He believes that this stimulus takes place somewhere in the course of the remains of the primitive tube, in the auriculo-venticular bundle beyond the auricular ventricular node (ventricular) in the past incorporated in the auricle (auricular), or in the auricular ventricular node itself (nodal). This symptom is often marked to the patient, who is conscious that his heart is dropping a beat. He is frequently much disturbed, mentally. On examination, the pulse often intermits, this intermission being due to an abortive attempt at systole of the heart which does not reach the radial pulse. This extra systole often being followed by a cessation of the heart's action through one cycle. Engleman explains this on the ground that the ventricle is so exhausted by the abortive attempt that it fails to respond to the next normal stimulus, to contraction. Mackenzie believes, however, that in man it is not the ventricle which is refractory and refuses to beat, but the primitive bundle is refractory and refuses to conduct the stimulus.

Examination of the heart itself will show a regular first sound followed by a short abortive systole. The first is followed by the radial pulse, the second is not. This condition is not in itself serious, it simply means that an abnormal stimulus is given to the heart, usually through some fault in the bundle or conducting apparatus. It is not always the result of cardiac degeneration. When there is no other symptom than an irregularity, no especial fear need be felt.

Treatment.—Assurance to the patient that the symptom is usually without danger. Avoidance of irregular living, undue exertion and other unhygienic acts which give rise to undue cardiac stimulation are important. Removal of all sources of worry and graduated exercise is advised.

Nodal Rhythm.—Paroxysmal tachycardia—continuous irregularity of the heart.

This condition embraces the greater number of cases of cardiac irregularities, and is present in practically all severe cardiac failures. Mackenzie believes most of the cases are the result of rheumatism where deposits are found in many parts of the cardiac muscle and often along the auriculo-ventricular bundle. It often occurs where there is disease of the coronary arteries, giving rise to cardio-sclerosis. In these cases the ventricle and auricle beat together or the ventricle slightly precedes the auricle. In certain cases the rapidity of the heart is not increased, but frequently the patient finds himself unable to do his usual work without distress. Sometimes, however, edema and dyspnea supervene and the patient becomes

incapacitated. The prognosis depends altogether upon the condition of the cardiac muscle. The treatment is that advised in decompensation from valvular disease.

In other cases there is great rapidity of the heart beat. There is fibrillation of the auricle. The patient at first is simply aware of a rapid pulse. Gradually, however, he becomes unable to do his usual work. Cyanosis, dyspnea, and edema supervene. Under proper treatment, rest followed by proper exercise, the heart may regain much of its strength, followed sooner or later by another breakdown. In the cases due to rheumatism digitalis is of value. In fibroid heart it is valueless.

True Paroxysmal Tachycardia.—(Delirium cordis.) This is the condition represented by the class of cases which for the greater part of the time seem to be in perfect health with no symptoms directed to the heart, but which are suddenly seized with cardiac action. If the heart is examined between the attacks, either no abnormality is noticed or there may be a condition represented by feeble first sound without any other apparent disturbance. Sometimes the attacks occur in individuals with well-marked valvular lesions.

The attack is characterized first of all by sudden severe rapid heart action. This may give the individual little or no distress. It may last for a few minutes or for days. Sometimes there is absolutely no dilatation, at others a dilatation rapidly or slowly develops with all the accompanying distress. Sometimes during the attacks a well-marked murmur develops, at others nothing is heard except the rapid heart beat. The exciting cause is not certain. The attack may come during the night, awakening the patient from sleep. It may come apparently as the result of indigestion. It rarely follows exertion, though in one of our cases a rather prolonged series of weeks of overwork have preceded practically all the attacks.

The prognosis depends upon the integrity of the heart muscle. The attack is not necessarily of serious import. One case on our record had many of these attacks, lasting from an hour or two to two or three days. He died at 76, his arteries and heart at the time of death showed no macroscopic change.

The **treatment** is unsatisfactory. One case on record could stop an attack by swallowing ice-cold water. Most attacks under our care have responded to a hypodermic of morphin. The latter remedy is somewhat dangerous, however, on account of the fear of causing a morphin habit. Digitalis is valueless in the attack.

Heart Block. *Stokes-Adams' disease, ventricular rhythm.*—In certain cases of arrythmia, the irregular action of the heart is due to faulty conducting power of the bundle, instead of irregular stimulation as in the classes described in the preceding paragraphs. This lack of power of conduction may be due to a simple delay in the conduction of the stimulus; to an interruption of the stimulus; or to a complete blocking of the stimulus.

These variations due to interference of the bundle have been frequently demonstrated experimentally by Gaskell, Erlanger and others, and have been described by Mackenzie (and many others). Clinically, the cases can be fairly well recognized by observing the radial pulse and the jugular pulse. The radial pulse, representing the ventricular systole, beats slowly,

varying from 20 to 40 per minute. The jugular pulse is seen to beat much more rapidly. This diagnosis can be absolutely confirmed by the use of the polygraph which shows the slow ventricular beat and the more rapid auricular beat.

The cases where the ventricular beat is in response to a delayed stimulus from the auricle can be distinguished by means of a polygraph tracing from those cases in which there is entire failure of the bundle to conduct any stimulus from the auricle to the ventricle. They can be also distinguished by clinical signs.

The cause of this condition in man is invariably due to some sclerotic change of the heart muscle, frequently accompanied by sclerosis of the coronary arteries, usually rheumatic or syphilitic. Stengel reports a typical case of complete heart block with autopsy where the bundle was destroyed by a salerotic area. There are cases of heart block, according to Mackenzie, in which the heart block is shown to be due to vagus stimulation or to digitalis, but these cases are temporary and not permanent.

Mackenzie sounds a warning against the persistent use of digitalis where the heart begins to drop a beat under its influence. The complete syndrome of Adams-Stokes' is characterized as follows in a case observed by one of us. The case during the quiescent stage was marked by a slow radial pulse corresponding to the action of the ventricle. During this time there was rapid pulsation in the jugulars. During the life of the patient she had many epileptic form seizures always preceded by an unusually slow pulse. It was never our privilege to observe the case during an attack, but her attendant always noted a disappearance of the radial during the unconscious stage.

Stengel, in the case quoted above, noted the absence of the ventricular systole for one minute at a time. The patient was thought to have died.

The prognosis in these cases is bad because of the certain myocardial change which the condition indicates.

The treatment is summed up in the advice so to watch the life of the patient that sudden strain upon the heart is avoided; anger; undue sexual excitement; overexertion, particularly walking in a high wind, should be avoided. Care should be taken to avoid indigestible food, as an attack of indigestion may bring on a complete failure of the heart to act.

Drugs are of little value. Digitalis is not only useless, but may do positive harm.

NERVOUS PALPITATION.

Definition.—By this is meant an unnaturally frequent, regular, or irregular beating of the heart, of which the patient is uncomfortably conscious, but which is unattended by any organic disease of the organ. This does not mean that there may not be functional or accidental murmurs. *Such murmurs are always, however, systolic, a diastolic murmur always indicating organic disease.*

Etiology.—There are numerous causes of palpitation. In the first place, it is much more frequent in women than in men. Again, it is prone to occur at the time of puberty in girls, and at the menstrual period and climacteric in women. Indigestion is a very frequent causal agent. Mental

emotion, including fright, anxiety, and grief, diseases of the uterus and stomach, sexual excesses, overwork, are all active etiological elements. The "irritable heart" described by Da Costa, based on observations made on soldiers in the late Civil War in America, has for its most striking symptoms palpitation. Overwork and excitement were its chief causes, abetted by exhaustion from illness.

Symptoms.—The "*beating*" referred to is, of course, the chief symptom. It varies greatly, however, in degree and duration. At times there is a mere fluttering, lasting for a few minutes. At other times the pulse-rate may reach 160 or more and be scarcely countable. The rapid heart-action is sometimes associated with a sense of *weakness* or "*goneness*" in the epigastrium, and sometimes with *nausea*. The face is usually pale, but is sometimes flushed. The physical signs usually add nothing to the undue beating noted on auscultation, though, as already mentioned, there may be functional murmurs systolic in time at the base of the heart, more rarely at the apex. The normal heart-sounds may be somewhat sharper and clearer, or they may be more blurred.

Diagnosis.—The conditions with which nervous palpitation may be confounded are *myocarditis* and *fatty degeneration of the heart* and *dilatation*, the symptoms of which, it will be remembered, are similar; or one of the arrhythmias before described, where there is organic disease in the conducting apparatus of the heart.

In all these conditions the history of the case and the physical examination must decide the diagnosis. Careful tracings of the jugular pulsations as compared with the radial pulse will throw much light upon the condition. A diagnosis of nervous palpitation must never be made unless organic changes in the heart can be absolutely excluded. Mackenzie includes these cases in his *x* disease and classes them as due to sinus stimulation. It has eeen thought worth while to retain the name nervous palpitation to mark these cases due purely to causes outside the heart itself. The nervous affection is, however, a less serious one, characterized by intermissions during which the heart is quiet. Its subjects are also of the anemic nervous type, whose history greatly aids the diagnosis, and they are commonly younger.

Treatment.—This is by rest, nerve sedatives, and a suitable moral treatment of encouraging words and a confident manner. A few more grains of sodium bromid, repeated every hour, may be useful. When the patient is weak and anemic, he should be built up and strengthened by iron, quinin, and strychnin.

ANGINA PECTORIS, OR STENOCARDIA.

Definition.—Angina pectoris is a symptom complex referred to the heart, characterized by pain sometimes mild, sometimes intensely severe, situated beneath the lower end of the sternum, over the heart, and sometimes conducted to the shoulder and to the arm, usually the left. Sometimes the attack is accompanied by a severe, painful contraction of the chest.

Etiology.—Mackenzie believes that these attacks of pain are always due to exhaustion of the contractile function of the heart muscle. The lesions of the heart which accompany some of these cases are so varied that it

seems correct to assume that this is the underlying cause. Obstructive disease of the coronary arteries, aortic stenosis or insufficiency, pressure by a tumor or other cause, dilatation or enlargement of the heart beyond the capacity of the coronary arteries to nourish are the lesions most frequently found. Of these endarteritis of the coronary is the most common. Excessive use of tobacco has been accredited with the direct effect of causing angina. The exciting cause of the attack is usually some overexertion or mental emotion calling for some additional effort from an already crippled ischemic heart. These events are more apt to produce this effect after a meal because a full stomach encroaches on the heart. The taking of food alone, even in moderate amount, may excite an attack. Still more, excessive eating and indigestion, however caused, become exciting causes. It is much more common in men than in women. Aortitis affecting the root of the vessel, general arteriosclerosis and high blood pressure are very common. The individuals who are most commonly affected are business men living under great strain, eating immoderately or improperly.

Diagnosis.—Sometimes many of the patients who apply have an oppression immediately under the xiphoid, usually following exertions and accompanied by expulsion of a large amount of gas from the stomach. They believe themselves to be suffering from indigestion. Other cases have a more severe distress amounting to an actual pain in the cardiac region, transmitted toward the left shoulder. Still other cases are suddenly seized with an excruciating pain in the region of the heart, a numbness in the left shoulder, arm and hand, and a feeling of impending death. Sometimes the face is pale, sometimes it is ashy in color; beads of perspiration often stand out on the face during these attacks. The duration of the attack is from a few seconds to many minutes. The lighter attacks disappear immediately upon the patient resting from the act that he was doing when the attack began. Often the attack ends by belching air, the air which is expelled is the result of the patient's having sucked in air during the attack.

Diagnosis.—The commonest condition with which the mildest forms of angina pectoris is confused is indigestion, pain in the upper epigastrium. Belching of gas at times with relief gives color in the eyes of the laity to the thought of indigestion, but to the physician, the age of the patient; the history of the usually strenuous life; high blood pressure; slight increase of heart dullness with a valve-like first sound together with the fact that the attack occurs almost always with exertion or on excitement, especially if the pain is conducted toward the shoulder and arm, mark the case as one of true angina. In certain nervous individuals, attacks simulating angina pectoris occur. These attacks, if they are simply hysteria, usually occur in females, last much longer than the attacks of true angina and do not give the feeling of impending death. The physical signs above given are wanting. Such cases have been called pseudo-angina.

Prognosis.—Prognosis in angina pectoris, even in its mild form, is grave, because of the extremely frequent grave cardiac lesions which are its cause. On the other hand, in cases which occur, as Mackenzie has pointed out, from the exhaustion of the heart muscle, brought about by overuse of tobacco, by worry, by work and frequent pregnancies, with the absence of arterial degeneration, the prognosis is not very grave.

Treatment. *Prevention.*—It should be pointed out to men with responsibilities that worry over their work; eating unusually and improperly; undue indulgence in alcoholics; in other words, overworking the machinery of the body, is fairly sure to bring about premature arterio-sclerosis, which, if it attacks the coronary arteries as it well may, is certain to bring about degeneration of heart muscle, which in turn may bring about angina pectoris.

Treatment of mild attacks may be summed up in hygienic regulation of the individual life, cessation of the acts which are bringing about the degeneration of the muscle; proper food; moderate eating and avoidance of alcoholics. The very mildest attacks usually pass away quickly by the individual's standing or sitting still. If the case is more severe, the inhalation of the contents of a Pearl of Amyl will often break up the very severe attacks, the administration of chloroform or the hypodermic injection of morphin is indicated, as well as the use of nitrite of amyl or nitroglycerin. If the cases are the result of conditions not bringing about cardiac degeneration, the correction of the habits of patient and method of living will stop them entirely. The treatment of the attack itself is the same as that due to organic heart disease.

Many cases, whether severe or mild, succumb during the attack.

DISEASES OF THE BLOOD-VESSELS.

ARTERIO-SCLEROSIS.

SYNONYMS.—*Angio-sclerosis; Endarteritis chronica deformans; Atheroma of the Blood-vessels; Arterio-capillary Fibrosis.*

Definition.—An inflammatory thickening of the walls of blood-vessels, chiefly of arteries, beginning in the intima, but extending also to the media and adventitia, associated also, more or less, with degenerative changes.

Endarteritis obliterans is an inflammation of the endarterium which, partly by its immediate product and partly by thrombosis and the organization of the resulting clot, produces complete obliteration of the artery with resulting gangrene.

Etiology.—There is a tendency to atheroma in the arteries of the old, as an evolution process quite independent of exciting causes. This tendency also varies greatly in different families, being very strong in some and absent in others. Men are more frequent subjects than women. There are many exciting causes, among which are especially overeating and drinking, with consequent accumulation of irritating matters in the blood, syphilis, the gouty poison, and lead. Chronic Bright's disease and diabetes mellitus are especially frequently succeeded by it; more rarely acute articular rheumatism. In the latter the rheumatic poison, whatever that may be, is probably the responsible agent, and in Bright's disease it may be retained excrementitious matter. In diabetes it is the sugar in the blood. Two classes of cases may, however, be associated with Bright's disease, in one of which the arterio-sclerosis is general and primary, causing interstitial nephritis, and in the other it is secondary, the result of Bright's disease. One set of observers regard all cases of interstitial nephritis as secondary.

Among these Sir William Gull and Henry D. Sutton, of England, and Arthur V. Meigs, of Philadelphia, have been conspicuous by their writings. Still another cause of arterio-sclerosis is increased arterial tension due to prolonged muscular exertion. The toxins in the blood of the various acute infectious diseases may also cause endarteritis and sclerosis.

Morbid Anatomy.—The aorta is the most frequent and conspicuous seat of the changes ascribed to chronic endarteritis, but the carotids, subclavians, brachials, radials, and ulnars, the iliacs, femorals, and especially the arteries of the brain and coronary arteries of the heart, are frequently involved. The arteries to viscera, like the stomach and liver, are rarely affected, while the pulmonary arteries take an intermediate place. On the other hand, the latter are sometimes invaded to the exclusion of the aorta. Whatever invites high tension in the lesser circulation tends to produce sclerosis in these vessels. The portal vein may also be invaded.

Appearances differ in arteries of different sizes. Those in arteries of moderate size are best studied in the superficial vessels. They are tortuous, stand out conspicuously, and feel hard to the finger, under which they may be made to roll. These features are often recognizable in the temporals and less plainly in the radials. The smaller arteries and veins with transparent walls, especially in the brain, exhibit to the naked eye white patches which are the seat of the atheroma. On slitting them open, the inner surface of these and other arteries will be found to have lost its natural smoothness, to be rough and uneven, while the lumen is more or less encroached upon.

Minutely examined, the appearances vary with the stage. The first stage is that of cellular infiltration, represented by the translucent yellowish areas of intima thickened to three or four times its natural thickness. Later these young cells are in part converted into connective tissue, causing the primary hardness of the vessel-walls. In the second stage the cells of the connective tissue and the surface cells of the intima undergo fatty degeneration, and the intercellular substance liquefies. In the third stage, which is not reached in the smaller arteries, or, indeed, usually in those below the aorta, there occurs a further liquefaction with the formation of the so-called atheromatous abscess, whose contents are not pus, but the well-known atherom-pulp, representing the débris of fatty degenerated cells, including fat drops and cholesterin crystals. Alongside of the atheromatous patches appear also plates or scales of calcareous infiltration of the intima, produced by a deposit of lime salts in the intercellular substance of the deeper layers. The atheromatous abscess sometimes undermines the intima, forming sinuous cavities, and after evacuation there results the atheromatous ulcer. Both the calcareous plates and ulcers furnish inequalities which favor thrombosis. In the later stages of the more diffuse form of arterio-sclerosis, especially studied by Councilman, the media or muscular coat and the adventitia are also invaded, the former mainly by atrophic changes, alongside of which, at times, is a homogeneous hyaline infiltration. In this form the capillary walls are also thickened, especially those of the glomeruli of the kidneys, in some of which the vessels become obliterated.

A *calcareous* infiltration of the *muscular* coat without previous inflammation may be found in old age in arteries like the radial, crural, and temporal. Still another primary degeneration is the *fatty erosin* of Virchow,

extending through the intima and media as a transverse fissure thought to be the starting-point at times of dissecting aneurysm.

Effect of Arterial Sclerosis.—The effect of these changes is to produce rigidity and narrowing of the vessel, a loss of the propulsive power residing in the elastic coat, a slowing of the current, and increased intravascular resistance. These events tax the compensating power of the left ventricle, which therefore hypertrophies. This hypertrophy keeps up as long as its nutrition is maintained.

But another effect of obstructed circulation is defective local nutrition, some of the consequences of which have already been considered in the study of the fibroid heart. Similar interstitial overgrowth and contraction may be met in the kidney and have been referred to. Localized softening of the brain also succeeds upon atheroma, though this event is usually preceded by thrombotic obstruction favored by the sclerosis. A more frequent accident to the brain is rupture of one of these atheromatous vessels, succeeded by the symptoms of apoplexy and hemiplegia. Such rupture may be preceded by an aneurysmal dilatation. Finally, aneurysm of the larger vessels has for its almost indispensable condition, except in traumatic cases, atheroma of the dilated vessel. Both events—the primary atheroma and the subsequent dilatation—are favored by the increased intravascular pressure.

Symptoms.—Superficial vessels in a state of atheroma are easily detected, as for example in the temples, by their *dilated, tortuous* outline in which pulsation is sometimes apparent; in other situations, as at the wrist, antebrachial and popliteal spaces, they may be recognized more or less by the touch. Distinction should be made between simple increase of tension and thickening of vessel-walls, though the two are constantly associated. The vessel in both instances is hard and requires some force to compress it, and between beats it is still full and can be rolled under the finger, but the artery with the thickened wall, if firmly enough compressed to obliterate the blood current, can still be felt beyond the seat of compression. In many instances, on the other hand, the changes escape detection until a fatal apoplexy gives notice of their presence. In most of these cases, however, if attention had been directed to the patient, the previously described condition of the arteries would probably have been recognized, while a certain degree of hypertrophy of the left ventricle would also, perhaps, have been detected. It does not follow, however, that the absence of atheroma in one place implies its absence in another, since fatal rupture of an artery in the brain has occurred when there has been no sign of sclerosis in the radials. It should not be forgotten that prolonged cardiac hypertrophy and the increased tension incident to it may produce atheroma, or the two may be the result of the same cause—as, for example, contracted kidney.

The clinical symptom of general arterio-sclerosis is, first of all, an anemia; which simulates in appearance very closely that present in malignant disease. Stengle has called attention to this peculiar anemia. The individual is breathless, cannot take any sustained action without distress. Frequently there is low blood pressure, sometimes high blood pressure.

Depending upon the organ most affected, the patient has in addition symptoms referable to that organ. If the arteries of the brain are most

affected, he has vertigo; fleeting palsies, headaches and often faintness. If the heart is most affected he has cardiac symptoms. Intermittent claudication is the result of faulty blood supply to the muscles of the legs due to arterio-sclerosis.

Certain cases where the kidney is most affected, the renal symptoms are in the foreground. Certain cases of arterio-sclerosis of the vessels of the cord give rise to symptoms not unlike those of tabes. The blood pressure is not as a rule very high, although it may be high, due simply to the general condition.

Abdominal Arterio-sclerosis.

Osler calls attention to the fact that there are certain cases of true angina pectoris with abdominal pain, which may be due to anginoid-spasm of the sclerotic abdominal vessels.

Arterial Tension as a Symptom.

Hypertension.—All individuals with arterio-sclerosis have a plus arterial tension, but not, as before stated, a very high pressure, which is a constant symptom of other conditions; for instance the blood pressure of a man of 30 is 125–130; but after this age when arterio-sclerosis is the rule, the pressure is always plus, being 140, 150 or even 160, being perfectly normal for persons of more advanced age. The higher blood pressure, 160 and over, is the result of many different conditions, the most common of which (as Janeway and others have so well pointed out) is chronic interstitial nephritis. The question which is primary, the nephritis or the tension, will not be discussed here. But Janeway has shown by a long series of autopsies that cases which during life had hypertension as one of the main symptoms usually had more or less sclerosis of the kidneys at autopsy.

When the heart begins to fail in these conditions, the blood pressure drops. Usually high blood pressure is a symptom of a failing compensation in damaged hearts; and under these conditions is compensatory and necessary to the patient's well being. Certain cases of anemia of the brain are accompanied by high pressure and here it is a danger signal. Toxemia of pregnancy has one of its initial symptoms in high blood pressure, and here too the high pressure is a danger signal. Lead poisoning is another condition in which there is a high blood pressure due to a circulating poison.

Low blood pressure is seen in all the asthenic states, in many of the acute conditions; in heart disease where the tonicity of the muscle is entirely wanting, in certain cases of cardio-sclerosis where the heart muscle is very weak.

Cardiac hypertrophy is not always demonstrable to percussion, as the enlarged heart may be covered by an emphysematous lung, also often present in the aged, in whom atheroma is most prone to occur. On the other hand, the usual sharp accentuation of the aortic second sound is present if the hypertrophy has not given way to dilatation of fibroid induration. Cardiac murmurs do not occur unless the atheroma invades the valves to produce insufficiency, stenosis, or roughening of the aortic orifice or aorta

near the orifice. This is not so very rare in old persons, apart from the relative insufficiency due to aortic dilatation.

Treatment.—Treatment is mainly the removal of conditions causing it, such as too free living, gout, lead-poisoning, and syphilis; together with rest and quiet, the avoidance of excitement, also aperients to lower the arterial tension, a slight increase of which is often the last straw required to produce an apoplexy. The iodid of potassium has received general endorsement, though from different points of view. Thus Vierordt gave the iodids on the assumption that they promote resolution of the sclerotic product; Huchard and Balfour on the ground that they dilate the arterioles and thus lower the blood pressure.

More recent studies by Boehm, Prévost, Corin, Stockman, James Burnet and Rolleston, go to show that the iodids do not reduce blood pressure, yet they admit that the drug is useful in arterio-sclerosis. Burnet claims that a further effect of the iodids is to increase elimination, thus removing certain irritant constituents of the blood.

As such, they ought to be useful in arterial sclerosis and probably are, Moderate doses should be continued a long time. In conjunction with this the usual cardiac tonics should be employed with a view to promoting a proper circulation of the blood.

ANEURYSM.

Definition.—An aneurysm is a more or less circumscribed dilatation of a blood-vessel. Aneurysm is known as true or false. A *true* aneurysm is one in which any or all of the coats of the blood-vessel share in the dilatation, though one or two may disappear later in the course of its growth. A *false* aneurysm, on the other hand, is one in which the coats of the artery have disappeared to a greater or less extent and in which the walls of the sack are composed of the surrounding tissues or newly formed fibrous tissue.

1. Aneurysm may be saccular, fusiform, cylindrical and cirsoid, serpentine, or there may be arteriectasis. The "cirsoid" aneurysm is one in which a blood-vessel of medium size and its branches are irregularly dilated and contorted like a varicose vein. In an arteriectasis there is a dilatation of the entire artery. Saccular and fusiform aneurysms are the more frequent. The "neck" of an aneurysm is a constricted portion by which a saccular aneurysm is attached to the main trunk.

(*a*) Traumatic aneurysm. In traumatic aneurysm the initial event is some injury from without to one or more of the coats of the vessels, as the result of which the resistance to intravascular pressure is diminished and a protrusion of the intima through the yielding media takes place, the latter being the most passive of all the coats. The simplest illustration of this form of aneurysm is the antebrachial aneurysm caused by accidental wounding of the brachial artery in venesection of the median vein. The blood pushes out the intima and antebrachial fascia and forms a sac communicating with the artery through the wound.

A second form of traumatic aneurysm is the aneurysmal varix or anastomotic aneurysm, in which the blood from the wounded artery passes

directly into the adjacent vein through the wound made at the same time, causing a dilatation of the vein. This is resisted by the valves, which, however, give way to the extent of two, three, and even more pairs before the current is successfully resisted.

(*b*) Dissecting aneurysm is one in which, in consequence of a perforation through the intima and media, the blood dissects between them and the adventitia. They are frequent in the aorta. The blood may dissect from this point around the arch of the aorta, even as low as the diaphragm, before it returns to the lumen of the vessel.

Etiology.—The aneurysm most frequently encountered by the physician is the saccular and fusiform form. Its most frequent essential cause is endarteritis and its consequences, including the more acute stage of cellular infiltration, as well as atheroma. The coats thus weakened yield to the intravascular pressure. The intima is capable of a considerable degree of expansion without rupture, while the media is entirely passive and yields very soon to the distending force. The adventitia alone seeks to guard the sac against rupture by reactive overgrowth. The causes of endarteritis, already discussed, such as syphilis, alcohol, and other toxic substances variously introduced into the blood, are responsible for the more usual forms of arterio-sclerosis which furnish the initial lesion of aneurysm. But weakening of the coats is caused also in the smaller vessels by emboli, after the lodgment of which the proximal part of the vessel often becomes dilated. Such embolus may excite an endarteritis, or may occasion direct violence to the vessel-walls if it be hard or sharp, as is often the case with a fragment of a calcified valve. Muscular compression exerted by muscles in certain stiuations may also produce it. Such may be the origin of popliteal aneurysms so frequent in footmen, who maintain a rigidly erect position.

Aneurysm is a disease of men rather than women. It is rarely seen until after the 40th year of life.

Aneurysm of the Thoracic Aorta.

Thoracic aneurysm occurs in the arch of the aorta, in its ascending transverse and descending portions, and in the thoracic aorta below the arch. Such aneurysm may but slightly exceed the normal caliber of the vessel, or it may be six inches (12 cm.) or more in diameter.

The greater frequency of aneurysm in the male sex and during early middle life is recognized. To the preexisting conditions of atheroma there may be added the effect of extreme exertion in lifting, or muscular strain of any kind, the effect of which is always to increase intravascular pressure. Partly because they are points of least resistance, and partly because they are in the line of successive impingement of the whirling blood stream, there are certain points of selection in the aorta which are quite constant seats for beginning aneurysm.

Symptoms of Thoracic Aneurysm.—Apart from the physical signs, the most important of the symptoms due to thoracic aneurysm are the results of pressure of the growing aneurysm, hence they are called *pressure symptoms*.

The first of these is *pain*, which may be sharp and acute when nerves

are directly involved, or dull and boring when the result of pressure on bone. In the latter case, too, it is localized; in the former it may extend all over the chest and down the arms, simulating angina pectoris. It may be unilateral. It may occur in aneurysm of any part of the arch, but is more frequent in that of the ascending limb.

Shortness of breath, especially on exertion, is a frequent symptom. It may be due to pressure of the aneurysm on the trachea, or on a bronchus, especially the left. Dyspnea may be increased on changing position *Dysphagia* from pressure of the tumor on the esophagus is a frequent symptom, especially in aneurysm of the descending aorta, anywhere in the thorax. Dysphagia is sometimes associated with *broncho-esophageal fistula*.

Cough and alterations in the voice are important symptoms. The latter include hoarseness, aphonia, and stridor. Some of these symptoms may be produced by direct pressure on the trachea itself, others by pressure upon the left recurrent laryngeal nerve. A stridulous voice, unaccompanied by dysphagia or aphonia, was early pointed out by Thomas Jolliffe Tufnell as indicating that the pressure in on the right side of the trachea and does not affect the esophagus or recurrent laryngeal nerve. Cough may be caused by tracheal pressure or by a resulting tracheo-bronchitis with copious thin or mucous expectoration, sometimes bloody. The cough is often brassy in character.

On the other hand, hoarseness, aphonia and various degrees of paralysis of the vocal cord are due to paralysis of the recurrent laryngeal nerve, commonly the left, which passes around the arch of the aorta and is, therefore, more likely to be involved than the right. The paralytic phenomena may be present without other laryngeal symptoms, hence any alteration of voice in a person exhibiting palpitation or dyspnea calls for a laryngoscopic examination. When paralysis is total on the left side, such examination may show little alteration in the position of the vocal cords in *ordinary* breathing, or the left may be a little nearer the median line. On *deep inspiration* the right vocal cord is well abducted, the left remaining quiescent in the so-called cadaveric position, midway between that of inspiration and phonation. The attempt at *phonation* is more or less abortive. During it, the right vocal cord may go to the median line, leaving a small opening between it and the motionless left cord, or it may even cross the line to its paralyzed neighbor.

Partial recurrent paralysis results if only the twigs distributed to the *abductor* muscle—*i. e.*, the posterior cricoarytenoid—are involved in the pressure. There ensues gradually a permanent shortening or "paralytic contracture" of the antagonistic adductors of the same side, and the affected cord is drawn by this into a position of constant phonation—that is, to the median line. The result is that the voice may be entirely natural, the paralyzed cord being in the position of adduction, while its tension is mainly regulated by the external branch of the superior laryngeal nerve, the sensory nerve of the larynx which is uninfluenced in aortic aneurysm.[1] In these cases quiet breathing is also unimpeded.

These phenomena imply, of course, a destructive lesion of the nerve, the result of pressure, which may be preceded by a primary neuritis.

[1] For the muscles involved see Diseases of the Larynx.

Such neuritis and resulting irritation of the entire pneumogastric may account for certain attacks of extreme *dyspnea* sometimes experienced by subjects of aortic aneurysm. Associated with the neural degeneration is also found *atrophy* of the left posterior cricoarytenoid or abductor muscle, while the adductors, the lateral cricoarytenoids and the arytenoid remain nearly intact. Constant dyspnea is more likely to be due to direct compression of the trachea.

Other *nerves* may also be *compressed*, especially the intercostal, vagus, and sympathetic. By compression of the intercostal nerves, pain may be caused; of the vagus, vomiting; and of the sympathetic, inequality of the pupils and unilateral sweating.

Pressure of the aneurysm on a bronchus may lead to retention of secretion and fetid bronchitis and bronchiectasis, and favor the inoculation of tuberculosis, thus accounting for the frequent association of tuberculosis of the lungs and aneurysm, or the whole lung may be collapsed.

Spitting of blood is an occasional symptom, which may be the forerunner of larger and more dangerous hemorrhage.

Still rarer is pressure on the thoracic duct, causing emaciation, though this symptom is more frequently due to mediastinal tumor.

Physical Signs.—Inspection does not always discover changes, but if the sac grows outwardly, sooner or later a swelling makes its appearance, to the right of the sternum if in the ascending limb, possibly raising a rib or the end of the clavicle; above and behind the sternum if in the transverse portion, raising the manubrium or boring its way through it; and to the left of the sternum if in the descending limb of the arch. As the tumor protrudes, the skin becomes smooth, shining, and tense over it, and may become gangrenous previous to rupture. Such a tumor may pulsate or not. Should elasticity of the sac be lost, either as the result of calcification or the lining of the sac with successive layers of coagulum, such dilatation becomes impossible, and pulsation does not occur. The pulsation is, however, of great importance to the diagnosis. When present, it is synchronous with the systole of the ventricles. The heart itself is sometimes displaced downward, as seen from the lowering of the apex sometimes as low as the sixth interspace and outside the mammillary line. Hypertrophy of the left ventricle rarely occurs, and when present, is not nearly so extreme as in aortic valve disease.

If the aneurysmal tumor press upon the great veins of the neck, there may be venous engorgement and edema on one side of the neck or both, according as the innominate vein of one side only is compressed or the descending cava itself. The aneurysm may rarely rupture into the descending cava, resulting in a form of varicose aneurysm, producing, in addition to the ordinary signs of aneurysm, sudden distention of the veins in the upper half of the body, edema of the face, hands, and arms, cyanosis, systolic venous pulse, and purring thrill.

Eye Symptoms.—The pupil may be contracted or dilated depending upon whether the sympathetic is irritated or destroyed.

There may be unilateral sweating or flushing depending upon the same cause.

Palpation also recognizes the impulse of the aneurysm if it is visible,

and sometimes when it is not visible. This beating is peculiar, being *expansile*, and differs thus from the rising of a tumor over a pulsating blood-vessel. A *thrill* is also often felt, a vibration in the walls of the sac caused by the whirl of the blood in it. It is by no means, however, invariable, and it may come and go. Very great *tenderness* is sometimes present over the seat of the protruding aneurysm. Palpation may also recognize the "diastolic shock," or recoil blow of the aneurysm on the closed aortic valve, if this be competent—a most valuable sign.

Then, there is the *tracheal tugging* of aneurysm first described by Surgeon-Major Oliver, and further studied by Ross and McDonnell, in Canada.[1] It is generally indorsed as a valuable sign. This is a dragging downward of the larynx with each systole of the heart. In Ewart's method the patient sits with his mouth closed, his head well bent backward, steadied against the chest of the examiner, standing behind him. The trachea is drawn up gently by inserting the ends of the fingers under the edge of the cricoid cartilage, when with each impulse the larynx is felt to be pulled downward. Oliver directs, with the patient in the upright position, the mouth closed, and chin elevated, grasping the cricoid cartilage between the fingers and the thumb pressing it steadily upward, when, if aneurysm exists, the pulsation of the aorta will be distinctly felt. There is much danger in this last method of mistaking pulsation of the carotids for a tracheal tug. It may be the sole sign of aneurysm. Cardarelli's sign of *lateral* movement of the larynx is similar, with an obvious difference. It is said never to be present in aneurysm of the innominate.

Alteration in the pulse in distal arteries is also a sign of considerable diagnostic value. The pulse-beat may be simply delayed as compared with the heart-beat. This is the natural result of the intervening sac which may receive temporarily a considerable amount of the blood required to produce the pulse wave. Or the pulse in one radial may be smaller than that in the other. It is chiefly when the aneurysm involves the origin of blood-vessels leading to the radials, as the innominate on the right and the carotid or subclavian on the left. If the right radial pulse is enfeebled or delayed, the aneurysm will be on the right, involving the origin of the innominate; if the left radial is influenced, the aneurysm is probably in the neighborhood of the left subclavian. Great care should be taken in the examination, and it should be made from the center to the periphery—that is, the carotids, the subclavians, the brachials, and the radials should be successively examined, as recommended by Sansom. These effects are variously produced. Thus, the aneurysm may narrow or distort the orifice of the blood-vessel by traction on it; or there may be atheromatous change in the branch vessel analogous to that in the aorta itself, which may cause narrowing of the orifice, while the possibility of this in the absence of aneurysm, is also to be remembered; or the aneurysmal sac may act as the elastic air-chamber in a pump, diminishing thus the pulsatile force in the vessel and branches beyond. It is particularly in the arteries of the lower extremities, by aneurysm of the descending thoracic and abdominal aorta, that this air-chamber effect is seen, and the pulse, even in the abdominal

[1] "London Lancet," 1891.

aorta and its branches, has been thus obliterated by a large thoracic aneurysm. There may be a difference in the blood pressure on both sides.

Percussion over the swelling of an aneurysm invariably elicits impaired resonance, varying greatly in degree and extent. On the other hand, the adjacent lung may be compressed, producing an area of dullness beyond the tumor itself. One case on our records had complete collapse of one lung resembling a tuberculous consolidation. The dullness is usually in the right upper intercostal spaces, especially if the aneurysm is in the ascending limb of the arch. Aneurysms in the transverse portion produce dullness in the middle line under the manubrium and toward the left of the sternum, while aneurysms of the descending part may produce dullness in the left interscapular and scapular regions posteriorly. Sometimes the impairment of resonance precedes the pulsation, though such dullness is of uncertain significance.

Auscultation is no exception, as compared with the other modes of physical investigation, as to the inconstancy of its information, sometimes furnishing the most distinctive signs, while at other times it is totally negative.

The murmur or bruit heard over an aneurysm varies. Sometimes but one murmur is produced—systolic, corresponding with the first sound over the ventricles, but more intense; more rarely it is diastolic only. Not infrequently there is a combined or double murmur, both systolic and diastolic, the first intense and prolonged, the second fainter and shorter. It varies greatly, being sometimes rough, sometimes soft, and sometimes musical. The murmur is not infrequently absent. The mechanism of these sounds is not settled. The systolic is the most easily explained. There can be little doubt that it is produced by the inequalities which meet the entrance of the blood into the sac. The diastolic murmur, when the aneurysm is at the beginning of the aorta, will probably be an aortic regurgitant murmur, due to relative insufficiency of the aortic valve. When the aneurysm is distant from the aortic orifice, the diastolic murmur may be due to the recoil of the distended sac, propelling the blood through the outlet with additional force, or the whirling of the blood through the sac. Rarely in these distant situations there is a diastolic murmur only, probably thus caused.

A much more constant symptom is an *accentuated aortic second sound*, which is bell-like in character. It is rarely absent in aneurysm of the arch where the aortic valves are intact. It is an exaggeration of the second sound, recognizable by the ear and due to the elastic recoil of the aneurysmal sac. "It is the shock of the second sound that is heard and the recoil is felt." It is not always present, and requires a sound aortic valve to produce it in its most marked degree. Sir Douglas Powell holds that it is best studied with the wooden stethoscope, and that the binaural may fail to observe it. Ernest Sansom considers it best investigated by the ear direct, with only a slight intervening chest covering. It may be accompanied by or replaced by the diastolic murmur referred to. It is rarely, if ever, present with mediastinal growths, even when they perforate the sternum and produce pulsation.

But any one or all of these signs may be wanting. Particularly is this the case where the aneurysm occurs just after the aorta has left the heart. The most valuable is the pulsation distinct and separate from that of the heart, or, as graphically put by Da Costa, "what is more essential is to find

two points of pulsation in the chest—two hearts, apparently each with its own distinct beat, its own distinct sounds."[1]

The X-ray has been brought to bear on the diagnosis of aneurysm, and commonly a distinct demonstration of the tumor can be made, both by the fluoroscope and skiagraphy. It should always be availed of when there are persistent signs of pressure in the chest. Often a diagnosis is first made by the use of this method. Sailer and Pfahler have shown that a tortuous aorta may simulate an aneurysm.

Aneurysm Pointing in the Back.—It must always be remembered that an aneurysm, especially of the descending arch or the descending aorta itself, may be nearer the back than the front of the chest and may give the first physical signs there. In all cases where aneurysm is suspected the back must be especially examined.

In aneurysm of the Ascending Aorta there is more apt to be pain like that of angina pectoris, dyspnea, dullness to the *right* of the manubrium sterni from the second intercostal space upward, pulsation in the same region, displacement of the heart downward and to the left, delayed pulse in the peripheral arteries as contrasted with the heart's impulse, compression symptoms involving the sympathetic and the area of the superior cava, pressure upon the pulmonary artery producing a pulmonic systolic murmur, with hypertrophy and dilatation of the right ventricle if the aneurysm compress the pulmonary artery.

Aneurysm of the Transverse Part of the Arch furnishes more particularly pulsation in the *fossa jugularis;* tracheal tug; dullness on percussion over the manubrium and to its *left* in the first intercostal space; narrowing of the orifices of the innominate, the left carotid, or left subclavian, and resulting inequality of the pulse in the head and arm; pressure on the *left* innominate vein, with resulting congestion and edema of the *left* half of the neck and head. It is when in this situation that aneurysm compresses the left recurrent laryngeal nerve and causes paralysis of the left vocal cord, presses on the trachea, with resulting stridor and cough, and on the left bronchus, producing inspiratory dyspnea.

In aneurysm of the Descending Limb of the Arch of the Aorta[2] we look for the pulsation behind to the left of the vertebral column opposite the angle of the scapula or below. The bruit is faint or absent. In the *thoracic aorta below the arch,* in consequence of the air-chamber effect, we may find smallness of the crural pulse as contrasted with the radial, symptoms of pressure upon the left lower azygos or hemiazygos vein—*i. e.*, edema of the upper part of the abdomen and pleuritic effusion; also symptoms of pressure on the esophagus and left bronchus. The intercostal nerves may be compressed, producing intense pain in the course of their distribution, the vertebral column may also be eroded, the spinal canal opened, and the cord compressed, with resulting paraplegia. If the aneurysm project forward, which is rarely the case, it may press upon and displace

[1] Da Costa, "Medical Diagnosis," eighth ed., 1895, p. 507.

[2] The *descending part of the arch* of the aorta is somewhat arbitrarily terminated by anatomists at the lower end of the fifth dorsal vertebra, below which it is called the *descending thoracic aorta,* which terminates at the opening of the diaphragm in front of the last dorsal vertebra, below which it is the abdominal aorta. The symptoms of aneurysm of the descending part of the arch and the descending thoracic aorta do not differ widely.

the heart, causing palpitation, or it may also compress the esophagus, causing painful deglutition. It sometimes ulcerates and breaks into the esophagus. Obscure symptoms of this variety of aneurysm may exist for a long time before a tumor shows itself posteriorly between the shoulders, which is unmistakable at this late stage.

Aneurysm of the ABDOMINAL AORTA furnishes a pulsating tumor to the left of the vertebral column, to the left and above the umbilicus. The bifuration of the aorta takes place on the fourth lumbar vertebra, which point corresponds to the umbilicus. Sometimes a thrill may be felt and a systolic murmur heard, rarely a double murmur. Here, too, the smallness of the crural pulses, as contrasted with the heart's impulse and the radial pulse, may be observed, while in some cases the crural pulses disappear altogether. The symptoms vary somewhat, according as the aneurysm grows backward or toward the front. In backward pressure pain is also a striking symptom, and may be of two kinds, a fixed and constant pain in the back, caused by the pressure of the tumor on the solar plexus and splanchnic nerves, or a sharp lancinating pain radiating along the branches of the compressed lumbar nerves, whence pain in the loins, testes, hypogastrium, and in the lower limbs, usually on the left side. If the sac grows anteriorly, gastrointestinal symptoms may be present, such as vomiting, gastralgia, diarrhea, and even symptoms of obstruction of the bowel. Pain is also present, but is more likely to be fixed in the loins, epigastrium, or some part of the abdomen.

Erosion of the spine is much rarer in abdominal aneurysm than in thoracic. In emaciated persons the abdominal aorta sometimes pulsates so plainly that one is strongly reminded of aneurysm, and is a constant source of error, but under these circumstances there is absence of the systolic murmur and of the alterations in the pulse of the arteries of the lower extremity, and none of the pain described. Indeed, evident abdominal pulsation occurs far more frequently without aneurysm than with it. Abdominal aneurysm should not be diagnosticated unless there is a palpable tumor, a thrill with expansile pulsation.

ANEURYSM OF THE BRANCHES OF THE ABDOMINAL AORTA.—Of these, aneurysm of the *celiac axis* is most often mentioned and diagnosed, though not always correctly.

Aneurysm of the SPLENIC ARTERY is sometimes met. Ten cases were collected by Lebert out of 39 involving various branches of the abdominal aorta.

Aneurysm of the HEPATIC ARTERY is a rare lesion, some ten or twelve cases having been recorded. These aneurysms are not usually large, while the liver has been found greatly enlarged.

Aneurysms of the SUPERIOR MESENTERIC ARTERY have been found at necropsies.

Aneurysms of the RENAL ARTERY are more numerous. They are generally small, but may terminate in rupture and retroperitoneal hemorrhage.

Aneurysm of the INNOMINATE is especially indicated by its murmur, thrill, and impulse in the vicinity of the inner end of the right clavicle, which is sometimes raised by the resulting tumor; also by the comparative

absence of signs of pressure on the larynx or esophagus. The differences in the right radial pulse alluded to are especially present here. Compression of the right subclavian and right carotid diminishes the force of the beat of the innominate aneurysm, but is without effect in aortic aneurysm. Nor are there percussion signs of enlargement of the aorta.

If the SUBCLAVIAN is involved, the signs are further outward, on the outer side of the sterno-cleido-mastoid, while in aneurysm of the innominate they are found on the inner or tracheal side. To those named may be added symptoms of pressure upon the subclavian vein, producing swelling of the arm and neck; upon the right recurrent laryngeal, producing defective speech and dyspnea; on the sympathetic, producing contraction of the pupil, and on the brachial plexus of nerves, pain. Especially would these signs point to aneurysm of the subclavian if the pulse of the carotids is uninfluenced while the right or left radial pulse is influenced.

The very rare condition of aneurysm of the PULMONARY ARTERY may produce a swelling, with the other local symptoms described, to the left of the sternum, in the second interspace. A murmur is less constant and is not conducted into the vessels of the neck, while the superficial pressure signs are more conspicuous. There is lividity of the face, with dropsy, and the dyspnea is naturally very great. There is no cough or voice alteration. It is to be remembered, however, that the swelling of an aneurysm of the arch of the aorta *may* extend to the left of the sternum. Such an aortic aneurysm may break into the pulmonary artery.

An aneurysm of the HEART is not recognizable by physical signs and may only be suggested by symptoms.

Differential Diagnosis of Aneurysm of the Arch.—Further diagnosis distinguishes aneurysm of the aorta mainly from *mediastinal tumor*. There may be the same percussion signs, though percussion dullness is usually more irregular in mediastinal tumor. There is often similar pain; there may also be pulsation, but instead of the expansile pulsation extending in all directions, it is more heaving. Murmurs are not usual in the mediastinal tumor. The ringing, or accentuated second sound—diastolic shock—which may be present in aneurysm when the aortic valves are intact, or substituted by the diastolic murmur when the valves are incompetent, is absent in mediastinal tumor. Tracheal tugging may occur in mediastinal tumor. Differences in the pulse or changes in the voice. The state of the blood-vessels usually associated with aneurysm must be ascertained. Fever is often present in mediastinal tumor; very rarely in aneurysm. A differential diagnosis is often impossible, and experts have held opposite opinions on the same case. Should the patient develop a cachectic state and secondary glandular enlargements appear, presumption is in favor of mediastinal disease.

The resemblance of some of the symptoms of aneurysm of the ascending aorta to some of those of *aortic incompetency* is very close. The same pulsating aorta, the same double basic murmur with impaired resonance at the right of the sternum, may be present. Cases have been diagnosed as aortic regurgitation with stenosis, in which the autopsy disclosed perfect semilunar valves with, however, aneurysm and relative insufficiency, which caused the diastolic murmur. In aneurysm there is more rarely hypertrophy

of the heart than in aortic valvular disease. The age of the patient, if under 40, especially the history of heart disease in early life, the history of rheumatism, and the absence of the causes of atheromatous vessels, point to valvular disease. Though there may be pulsation at the root of the neck in both, in aortic incompetency the same strong pulse-beat extends to the wrists. Traube's double sound in the femorals and popliteals, though possibly otherwise caused, is still more frequently associated with aortic incompetency than any other lesion. Simple dilatation may, indeed, be present in aortic incompetency, but the pressure signs are wanting. Capillary pulse is absent in aortic aneurysm.

A *pulsating empyema* on either side of the upper sternum sometimes closely resembles a pulsating aneurysm, and the illusion is more complete because the pulsation is expansile. Pulsating empyemas are generally further to the left of the sternum than aneurysmal pulsation. Other signs of aneurysm are also wanting, unless it be tenderness, which may be present. There is leukocytosis in empyema and not in aneurysm. A rare condition is a *narrowing of the aorta* below the remains of the ductus arteriosus at the junction of the arch with the thoracic aorta, which produces small delayed pulse in the femorals, a thrill and murmur over the upper part of the sternum, but the extraordinary enlargement of the collateral vessels, especially the mammary and epigastric arteries, should set the question at rest.

In acute laryngitis we have often the cause—exposure to cold—to help us, though in the chronic form we have not. In laryngitis there is usually more huskiness and less stridor in the voice, nor is the cough so brassy, or the voice so uniformly changed; it is more likely to alternate with normal voice. In aneurysm the voice grows progressively weak until aphonia results. The dyspnea in aneurysm is more often attended with wheezing, and is sometimes relieved for a time by coughing. Stokes called attention to the fact that in aneurysm the stridor of the voice seems to come from the notch of the sternum, rather than from the larynx itself. In aneurysm the breathing sounds are more likely to differ in the two lungs. Then we have the laryngoscopic picture. There is no swelling of the cords in aneurysm, while there may be the paralytic phenomena detailed. Finally, in laryngitis there may be fever. The x-ray will frequently make an otherwise impossible diagnosis.

Prognosis.—Aneurysm is not infrequently found at necropsy without having been suspected and the aneurysmal sac entirely healed. In other cases the fatal termination is the first notification of its presence. When an aneurysm of the aorta is so developed as to exhibit its usual signs plainly, it is generally sooner or later fatal in some one of the modes already described. To foretell in which of the directions pointed out perforation will occur depends upon the accuracy with which diagnosis of its position can be made, and such diagnosis is at best a matter of probability. Only in cases in which aneurysm slowly erodes the anterior wall of the chest is there a gradual termination. Then there are sometimes repeated small hemorrhages, which gradually reduce the strength of the patient, who finally dies of exhaustion or of an ultimately fatal large hemorrhage. Perforation into the vena cava, pulmonary artery, and right side of the heart is a rare termination. The course of the disease may, however, be prolonged

many months, and if treatment is instituted early, it may contribute to such prolongation. When death does not occur from sudden hemorrhage, the symptoms may assume the type of chronic heart disease, for which, indeed, the condition is sometimes mistaken by the untrained observer. With failing heart come dyspnea, palpitation, dropsy, and death.

Treatment.—We seek in the treatment of aneurysm to *diminish intravascular pressure* and *restore the integrity of the vessel.* The former may be accomplished in a degree by placing the patient under conditions which will avert the causes of such increased intravascular pressure, which is constantly cooperating with the disease of the artery to produce further dilatation and ultimate rupture of the blood-vessel. This is, of course, best accomplished by absolute rest. It is plain the less frequently the heart beats and throws the weight of its blood against the weak blood-vessel, the longer will that blood-vessel last, while it is known to every student that the heart beats less frequently in the sitting than in the standing posture, and less in the recumbent than in the sitting position. On the other hand, it is evident that absolute rest is an impossibility. Yet it may be approximated in various degrees. It is impossible also to restore the integrity of the vessel, but to this end also measures are suggested which have for their immediate purpose coagulation of the blood in the vessel and obliteration of the sac. That this sometimes occurs numerous autopsies also attest.

The method which has met most favor is that now known as Tufnell's treatment, by restricted diet and rest though Valsalva originally suggested a restricted diet and practiced frequent venesections. Bellingham advised starvation without bleeding. The treatment was, however, revived by the late T. Jolliffe Tufnell and modified by G. W. Balfour who added the iodid of potassium. Tufnell's treatment consists in absolute mental and physical rest in the recumbent position, together with a moderate dry diet. The object of this is to diminish the blood-pressure and volume of blood, to increase the proportion of fibrin in the latter, and to promote its coagulation. The diet is as follows:

For breakfast, two ounces of bread and butter and two ounces of milk; for dinner, two or three ounces of meat and three or four ounces of milk or claret; for supper, two ounces of bread and two ounces of milk. Thus it is hoped to diminish the blood volume and reduce the pressure within the sac, to render the blood more fibrinous and to favor coagulation. The proper dose of the iodid of potassium is 5 to 20 grains (0.33 to 1.3 gm.) three times a day. It is supposed to act by increasing secretions, thus thickening the blood. To its efficiency in this direction we may add our testimony. Balfour also claims that it lowers the blood pressure by promoting the flow of blood through the arterioles. Boehm, Prévost, Corin, Stockman, James Burnet and Rolleston deny that the iodids lower blood pressure, though they admit that the drug is useful. It may be expected, also, that cases of syphilitic origin will be those especially benefited, but it is said that experience does not confirm such expectation. Occasional small bleedings amounting to a few ounces contribute to a favorable result including relief of pain. Evidences of improvement are reduction in the size of the tumor, diminished force of pulsation, and relief of pain. The Tufnell treatment should be kept up for several months, or as long as the patient will sub-

mit to it. It is said to be useful more particularly in saccular aneurysm communicating by a small orifice with the aorta. It is doubtful whether it is worth while to subject a patient with large aneurysm communicating with the aorta by a large orifice to the inconvenience of such a treatment, and whether it may not be better to advise him to live a life as quiet as possible and to await the inevitable, while we relieve symptoms as they arise, and remember especially that iodid of potassium is often one of the best remedies for pain.

A remedy said to favor coagulation of blood in an aneurysm is chlorid of calcium of which 10 grains (0.66 gm.) may be given four times a day. Acupuncture as a means of securing coagulation and contraction of the clot was suggested by Velpeau. It consists in placing a needle into the aneurysm with the hope that the blood will coagulate on it. Filling the aneurysm with *fine wire* has been suggested for the same purpose. The wire is introduced through a hypodermic needle, and a measured electric current passed through the wire. This measure gives much relief in certain cases. In others it is valueless. Ligation of the carotid or subclavian, or both, has also been done for aneurysm of the aorta with satisfactory results. It is, however, a formidable operation.

No other internal treatment for aneurysm other than that suggested—by iodid of potassium—has ever been of any use. As a part of the medicinal treatment of thoracic aneurysm it should be added that where there is violent action of the heart, *cardiac sedatives* are sometimes indicated to allay this, in addition, of course, to the enjoined rest. Among these sedatives we include aconite and veratrum viride in extreme cases, also cold to the seat of the swelling and to the cardiac region. When signs of cardiac decompensation occur the case must be treated as for that condition. Digitalis may then be given without fear of harm.

The treatment of *peripheral aneurysm*, as of the popliteal and femoral, is usually relegated to the surgeon, who will treat it by ligation or compression. Tufnell's method is also applied to peripheral aneurysm, for which, indeed, it was originally recommended.

The treatment of peripheral aneurysm by *compression* has long been an acknowledged method for the purpose, and though looked upon as a surgical procedure, is as medical as it is surgical. The method adopted which has been most successful is digital compression, which is exerted by relays of students or others available for the purpose. The effect is that in the course of 48 hours coagulation has taken place and the aneurysm is cured. Failing in these measures, ligation is practiced in case of the smaller arteries, but all details of this operation belong to the province of surgery.

SECTION V.

DISEASES OF THE BLOOD AND BLOOD-MAKING ORGANS.

THE ANEMIAS.

By *anemia* is meant any state of the blood in which there is a diminution of its total bulk, its red corpuscles, its hemoglobin—any one or all of these. The first is the condition which ensues from a large hemorrhage of any kind, as from the rupturing of an aneurysm, erosion of a blood-vessel, such as sometimes happens in ulcer of the stomach or in tuberculosis of the lung, or from a blood-vessel wounded in any way. In all instances, however, where the hemorrhage is not fatal the original bulk of the blood is rapidly restored by the absorption of water and salts from the tissues, while the hemoglobin and albumin remain deficient until they can be restored by suitable nourishment. Practically, therefore, anemias resolve themselves for study into conditions in which there is a reduction in the amount of hemoglobin through a diminution in the total number of red corpuscles or in the proportion of coloring-matter in each corpuscle, or both.

Anemias are further divided into *primary* or *essential*, and *secondary* anemias. The former, strictly speaking, include only those which are the direct result of a defect in the blood-making apparatus, while secondary anemias are those due to loss of blood, or some one of its important constituents.

Among primary anemias are included chlorosis, pernicious anemia, leukemia, pseudo-leukemia or Hodgkin's disease, and splenic anemia.

The secondary anemias are the direct result of trauma, accidental hemorrhage, chronic disease, or toxic agents.

SECONDARY OR SYMPTOMATIC ANEMIA.

1. *Anemias Due to Hemorrhage, however Caused.*—Traumatic hemorrhage, postpartum and other uterine hemorrhage, pulmonary hemorrhage, and gastric and intestinal hemorrhages comprise most of these; ruptured aneurysms and purpura furnish others. Parasites invading the intestinal canal may be causes of hemorrhage and consequent anemia. So may parasites elsewhere, as the *distoma hæmatobium* in the kidney.

In non-fatal hemorrhages from these causes the immediate loss of blood in hemophilia is rapidly made up by the absorption of water from the gastro-intestinal tract, but a long time is required, even under favorable circumstances, before the corpuscles and hemoglobin are restored. At other times regeneration is quite rapid, restoration being complete in ten days. The hemoglobin is always rather more reduced than the corpuscles, but both increase for a time *pari passu*, as shown in the appended chart. The albuminous constituents are more rapidly restored.

2. *Anemias Due to the Drain of Chronic Disease.*—Such are chronic

Bright's disease, suppurative processes, cancer, or prolonged lactation, arterio-sclerosis, tuberculosis, syphilis and certain intestinal parasites and chronic diarrhea. In this group belong the anemias of malaria.

3. *Anemia from Inanition.*—This results from starvation, which may be the practical consequence of diseases which interfere with the successful ingestion and assimilation of food, such as obstruction of the esophagus.

4. *Toxic Anemias.*—Finally, there remain the toxic anemias. These are the result of the presence in the blood of such substances as lead, acquired by painters or workers in lead-paint factories, type-setters, and type-founders: also arsenic from dress fabrics, wallpaper, and furniture coverings; mercury.

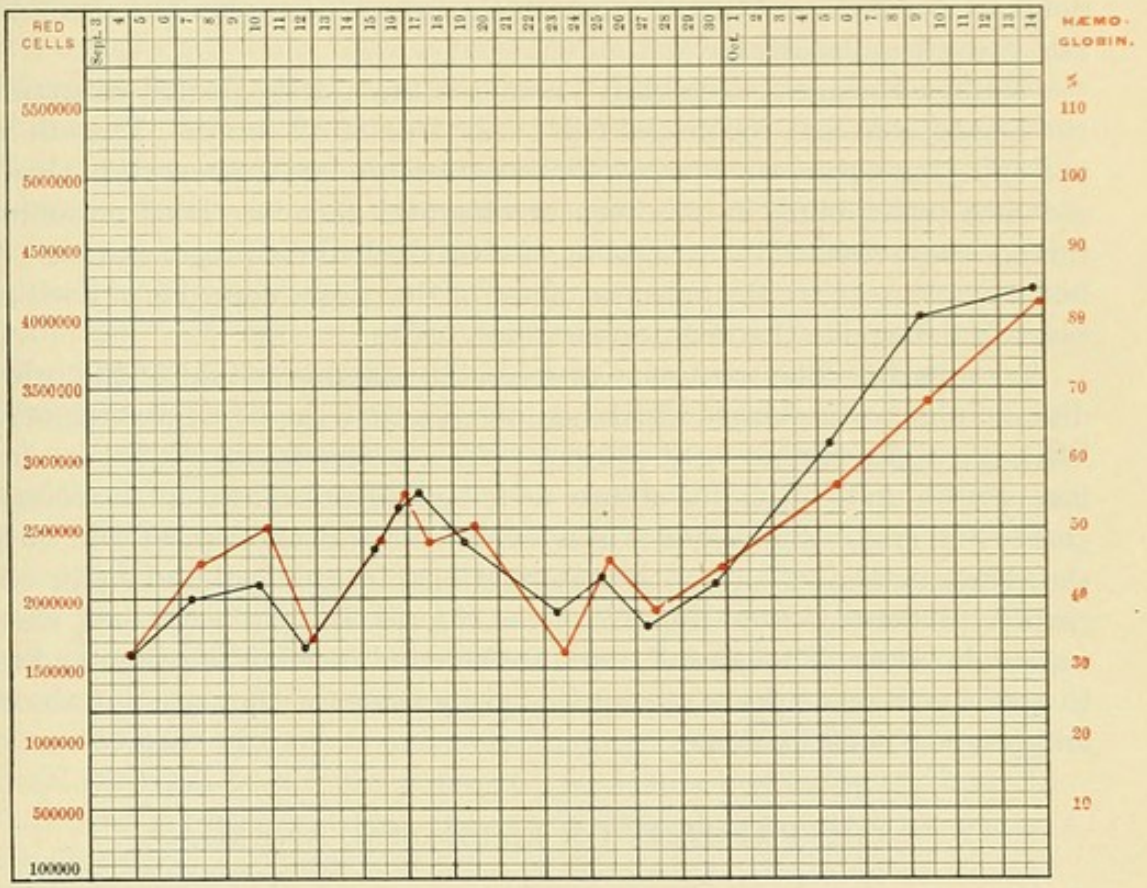


FIG. 117.—The Blood in Secondary Anemia. Hemoglobin Red, Corpuscles Black. From Case of Syphilis.

Symptoms.—The most commonly recognized symptom of anemia is a paleness of the skin, and this is undoubtedly present in the vast majority of cases. Yet a total reliance dare not be placed on it, for it sometimes happens that the skin and even the lips are pale, and yet no anemia is found when the blood is examined. On the other hand, the skin and lips may have a good color, and yet anemia be actually present. Weakness, faintness, vertigo, shortness of breath, and palpitation are also symptoms.

In addition to these are the *blood changes*, which vary with the degree of anemia. Both corpuscles and hemoglobin are reduced, not always *pari passu*, the hemoglobin commonly in somewhat larger proportion. The disproportionate lowering of the hemoglobin is explained by a more than natural paleness of the red corpuscles. Their average size is reduced, while in severe cases there is also a moderate poikilocytosis. Nucleated red cor-

puscles also make their appearance. The normoblasts and microblasts are the prevailing forms. They exhibit, after staining with Wright's stain, a deep-blue nucleus, while free nuclei are occasionally found. Microcytes, megalocytes, and poikilocytes are present in advanced cases.

The colorless corpuscles are moderately increased, such increase being represented by the multinuclear neutrophiles, while the small mononuclear lymphocytes are diminished. The leukocytosis gradually disappears with the return of the blood to its normal state. Myelocytes are exceptionally present.

Diagnosis.—In addition to the blood changes more or less common to all of these causes of anemia, the same general symptoms of pallor, lassitude, debility, dyspnea, and faintness which characterize the essential anemias are also present in less degree.

While a feature of secondary anemia is the nearly coequal reduction of the hemoglobin and corpuscles this is not true of all forms. Thus in lead-poisoning, as a rule, the hemoglobin is reduced in larger proportion than the red cells, resembling in this respect chlorotic anemia. Lead-poisoning is further characterized by a stippling of the red cells due to degeneration. The history of the case in the presence of one of the causes named is of itself sufficient to determine the diagnosis in many cases.

Treatment.—The treatment of secondary anemia is that of the primary disease, plus certain drugs which help to stimulate the blood-making organs. With the disappearance of the primary disease very rapid coequal rise in the hemoglobin and corpuscles occurs, as is beautifully shown in the foregoing chart (Fig. 117). Full doses of iron are well borne in these cases, and we have the choice of almost any of the preparations, including Blaud's pills of the carbonate, reduced iron, tincture of the chlorid, Basham's mixture, and the vegetable salts. Though full doses are here indicated, it is still unnecessary to give the massive doses recommended by some, as they are not absorbed and produce constipation.

THE PRIMARY OR ESSENTIAL ANEMIAS.

These include chlorosis, *for the present* pernicious anemia, leukemia, Hodgkin's disease or pseudoleukemia, and splenic anemia.

I. CHLOROSIS.

Synonyms.—*Morbus virgineus; Green Sickness; Chloremia; Chloranemia.*

Definition.—A primary anemia most frequently met in young girls, characterized by a very marked relative reduction in the hemoglobin of the blood.

Etiology.—As stated in the definition, it is a disease of females, and especially of young girls. Moreover, while it is especially a disease of young girls about the age of puberty, it is also possible in those who are older, as well as those who are younger. It occurs in children who have not reached the age of puberty. Niemeyer held that girls who menstruated at 13 or 14, in whom there was, as yet, no development of pubes or breasts, most invariably become chlorotic. The disease occurs the world over, and

is apt to be recurrent in the same individual. It is more common in blondes than in brunettes, in the weak and delicate, rather than the strong and vigorous. Yet this general truth is not without exception.

Among predisposing causes are overwork, especially in closely confined and ill-ventilated rooms, insufficient nourishment, and profuse menstruation. Menstrual derangement is, however, also a consequence as well as a cause. Sustained or reptated emotion, especially such as arise from sexual excitement and masturbation, is a cause. Homesickness and grief are included among causes, many of the cases occurring in recent immigrants.

The frequent association of constipation with chlorosis led Sir Andrew Clark to suggest that it might really be a copremia.

Morbid Anatomy.—Other than the changes in the blood, to be considered under symptoms, there is no essential morbid anatomy in chlorosis. Many years ago Virchow pointed out an imperfect development of the circulatory apparatus as more or less characteristic—that the heart was small, the right ventricle sometimes dilated, the aorta and its larger branches were poorly developed and thin walled. Such a condition, when present, is probably an accidental coincidence. There is no enlargement of the spleen or lymphatic glands. Imperfect development of the uterus and other genitalia has been noticed. The rarity of fatal termination in chlorosis may limit our knowledge of the morbid anatomy, uncertain at best.

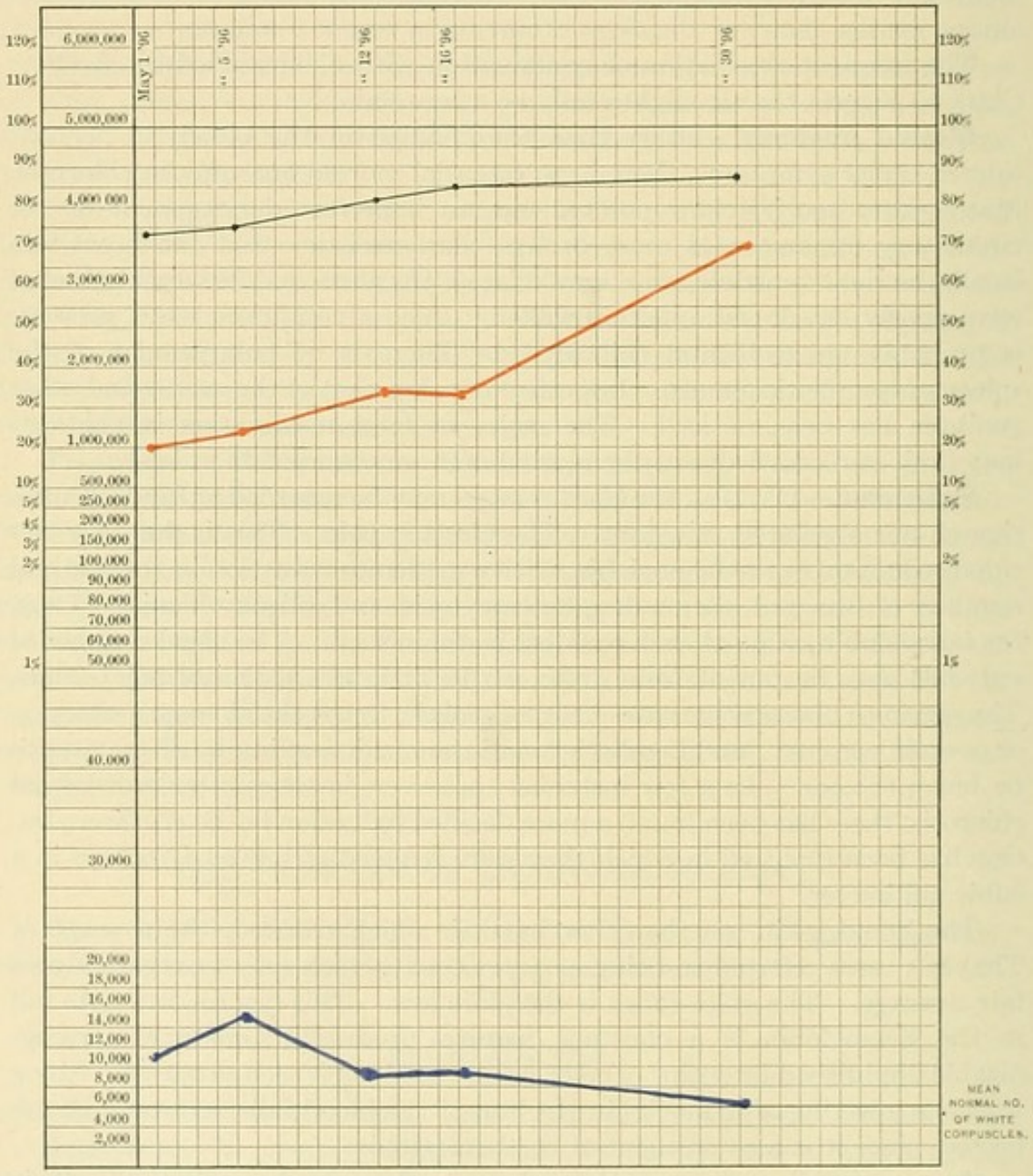
Symptoms.—Of these, the *blood changes* may be regarded as fundamental, though not absolutely constant. The blood is pale. This is due to a decided reduction in the hemoglobin, with a moderate reduction in the size and number of the red corpuscles—microcytosis and oligocythemia. Thus, the hemoglobin value of each red disk is diminished. The usual number of red cells may be put at from 3,500,000 to little less than normal. Thus, Thayer, in 63 consecutive cases in Osler's clinic, found the average 4,096,544, or over 80 per cent., and Lembeck found the maximum in one of 15 cases to be but 3,600,000. In a few instances, however, in cases of acknowledged chlorosis, there has been found a more decided reduction in the erythrocytes. One has been reported in which they were reduced as low as 1,190,000 in a cubic millimeter.

The hemoglobin, on the other hand, is much reduced, the average of Thayer's cases referred to being 42.3 per cent., which may be regarded as a fair average. The color index is therefore low. This disproportionate fall in the hemoglobin is a constant feature, producing sometimes a recognizable diminished intensity of color when the blood is seen *en masse*. Along with the lowering of hemoglobin the iron of the blood falls, as would be expected, since it is a constituent of the hemoglobin.

As to remaining changes, the red corpuscles may be *undersized*. This may be said to be a more or less characteristic change and with the diminished coloring-matter may be the only change. The red disks are sometimes appreciably paler than in health. They may be larger than in health (megalocytes). They may be altered in shape, constituting a small degree of poikilocytosis, a term suggested by Quincke. A very *slight* degree of *leukocytosis* may be rarely present, an average of 8467 in Thayer's counts, as contrasted with a mean normal of 6000 in the cubic millimeter, while the *blood plaques* in severe cases may also be increased. *Nucleated red cor-*

puscles are sometimes met, especially in the later stages, represented by the smaller forms (microblasts) which sometimes appear in crops. In this stage the corpuscles may assume irregular shapes. Myelocytes have rarely been met.

The blood alterations in chlorosis are distinctive enough to be considered diagnostic, when considered with the *other symptoms* and the absence of a



Red Corpuscles—Black. Hemoglobin—Red. Colorless Corpuscles—Blue.

FIG. 118.—Blood in Chlorosis.

causative disease. The patient is almost invariably a girl, generally between 16 and 20, who, although she may have been overworked, does not seem badly nourished; certainly she is not emaciated. There is often derangement of menstruation, and sometimes the girl is hysterical.

Most striking, though not invariable, is a peculiar pallor, very seldom exhibiting a yellowish-green tinge, extending to the lips, and especially the

mucous membranes, and which is responsible for one of the names of the affection—*green sickness*—this green color is certainly very rare in the cases now considered chlorosis. The patient is extremely weak, especially on exertion, and is short of breath. She is subject to vertigo, palpitation of the heart, and even irregularity of the heart's action. Physical examination will sometimes discover functional cardiac murmurs. Rarely, a compensatory hypertrophy of the left ventricle has been noticed, but never actual valvular disease. Sometimes a *bruit de diable* or murmur may be heard over the right jugular, disappearing when the patient lies down. Epigastric pain is also a symptom at times. It must not be forgotten that a chlorosis late in life, or *chlorosis tarda*, does sometimes occur. Fever is not rarely present. On the other hand, the hands and feet are often cold.

Diagnosis.—The diagnosis is based chiefly upon the age and sex of the patient, the peculiar paleness of the skin, the paleness of the lips, and the decidedly diminished hemoglobin, unaccompanied, with a proportionate reduction in the number of erythrocytes. The same lost normal ratio between the hemoglobin and the corpuscles is also a characteristic of *lead-poisoning*, which has, however, superadded its own characteristic symptoms, and is common in adult males and females.

The epigastric pain mentioned as occurring in chlorosis resembles that more common in *ulcer of the stomach*. The anemia which so constantly attends ulcer of the stomach, often in a high degree is, however, different from that of chlorosis, there being a corresponding decline in the number of the erythrocytes and their coloring-matter. The ordinary symptoms of gastric ulcer are wanting.

A not infrequent error of diagnosis in connection with chlorosis is the mistaking of it for a "decline," a *pulmonary consumption*, which it resembles in the pallor, the feebleness, and shortness of breath of the patient. The absence of emaciation, of cough, and of the physical signs of consumption exclude that disease. On the other hand, evidences of tuberculosis should always be sought where the symptoms of chlorosis prevail. It is a grave error to consider tuberculosis a chlorosis.

Most frequently chlorosis is confounded with *secondary anemia*, but the characteristic features of chlorotic blood are wanting, and there is always a presence of some other disease or poison. In advanced degrees of chlorosis the blood approaches that of *pernicious anemia*, but there is rarely the extreme reduction of red cells and pernicious anemia is rare in young girls while chlorosis rarely if ever occurs in adults.

Prognosis.—The prognosis is favorable when the disease is recognized and the proper treatment instituted. There are few results more satisfactory in therapeutics than those of a properly treated case of chlorosis. Time is, however, necessary, and too rapid a cure must not be promised, several months and even longer being sometimes required.

Treatment.—The treatment is preeminently by iron. The carbonate in the shape of Blaud's pill, made by a double decomposition between the carbonate of potassium and the sulphate of iron, is the best, 1 to 5 grains (0.06 to 0.2 gm.) being given at a dose three times a day. It should be made fresh. Much larger doses are sometimes given, as much as 45 grains (3 gms.) a day. We repeat that iron is given in too large doses in the majority of cases

for which is it prescribed. Most of it is unabsorbed, and therefore wasted. Nay, worse, that which is unabsorbed locks up the intestinal secretions by its astringency, produces headache, and makes the patient otherwise uncomfortable. But chlorosis is one of the few diseases in which large doses of iron are well borne. The reason is plain. It is the iron-holding constituent of the blood which is wanting, and the iron is needed to replace it. The blood is, as it were, hungry for it. Reduced iron or one of the vegetable salts of iron may be given. Next to iron comes arsenic. The efficiency of iron is greatly aided by union with arsenic, which should be given in increasing doses, but short of toxic effect.

Hydrochloric acid in full doses, originally suggested by Zander on the ground of supposed deficiency of this acid in the digestive fluid in chlorosis, is useful also in promoting the solubility of iron, as well as for its tonic and antiseptic properties.

But to give these drugs alone is not sufficient. In very severe cases *rest* in bed may be necessary to secure a rapid result, and this must be associated with an abundance of good food and fresh air. Daily massage, except during menstruation, is also a useful adjuvant. There is no condition in which the so-called "rest cure" is more efficient than in chlorosis. With a return of color to the lips, or, better, with the growing increase in the hemoglobin as measured by the hemoglobinometer, the patient should be permitted to be out of bed at first from a half-hour to an hour only, but this should be gradually increased until she is up most of the day. For a long time, however, fatigue should be avoided. To those who can afford it, a residence at the seaside materially aids convalescence. To the poor, a well-regulated hospital treatment is a boon for which there is scarcely a substitute. Many cases can be treated on foot from the beginning, care being taken to avoid fatigue. Symptoms rapidly disappear, but no case should be considered cured until both hemoglobin and red cells are normal.

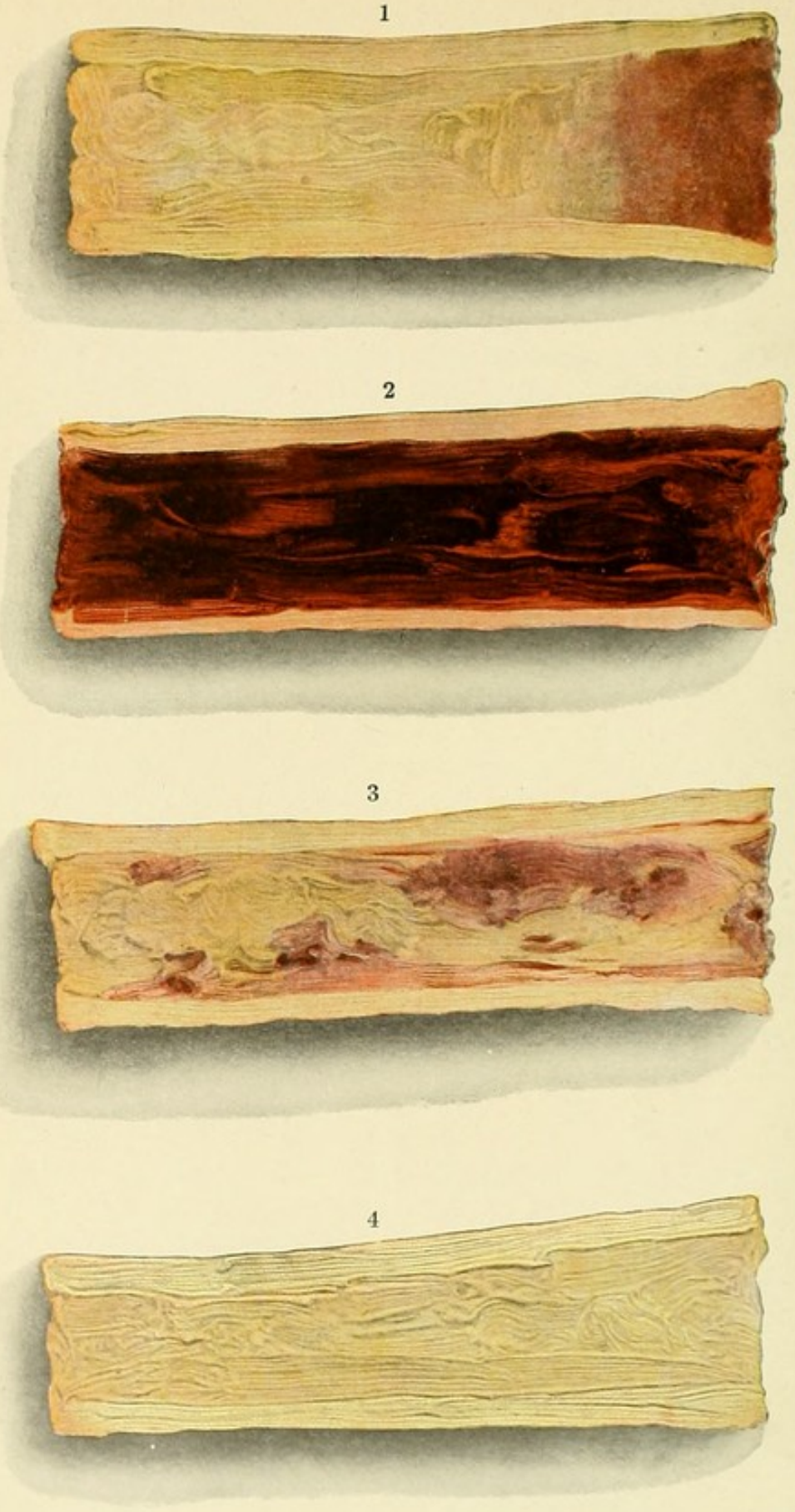
II. PROGRESSIVE PERNICIOUS ANEMIA.

SNYONYMS.—*Idiopathic Anemia; Pernicious Anemia.*

Pernicious or idiopathic anemia, originally described by Addison in 1855 in his celebrated paper on "Diseases of the Suprarenal Capsules." Interest in the subject was revived by Biermer in 1868, and since then it has been thoroughly studied anatomically and clinically. It is, however, still the least understood of all the anemias.

Definition.—Pernicious anemia is a progressive anemia of unknown cause with remissions. The red corpuscles and hemoglobin are both greatly reduced, the former in larger proportion, the color index being high.

Etiology.—The etiology of pernicious anemia is very obscure. Evidence is accumulating in favor of a toxic hemolytic origin, the toxins being the result of some one of the associated conditions. It is more common in males than females and in middle life. It rarely affects children. Pregnancy seems to be in some way responsible for a certain number of cases. It has also followed lactation. Cases with the blood picture of pernicious anemia as the result of atrophy of the stomach have been reported by Flint,

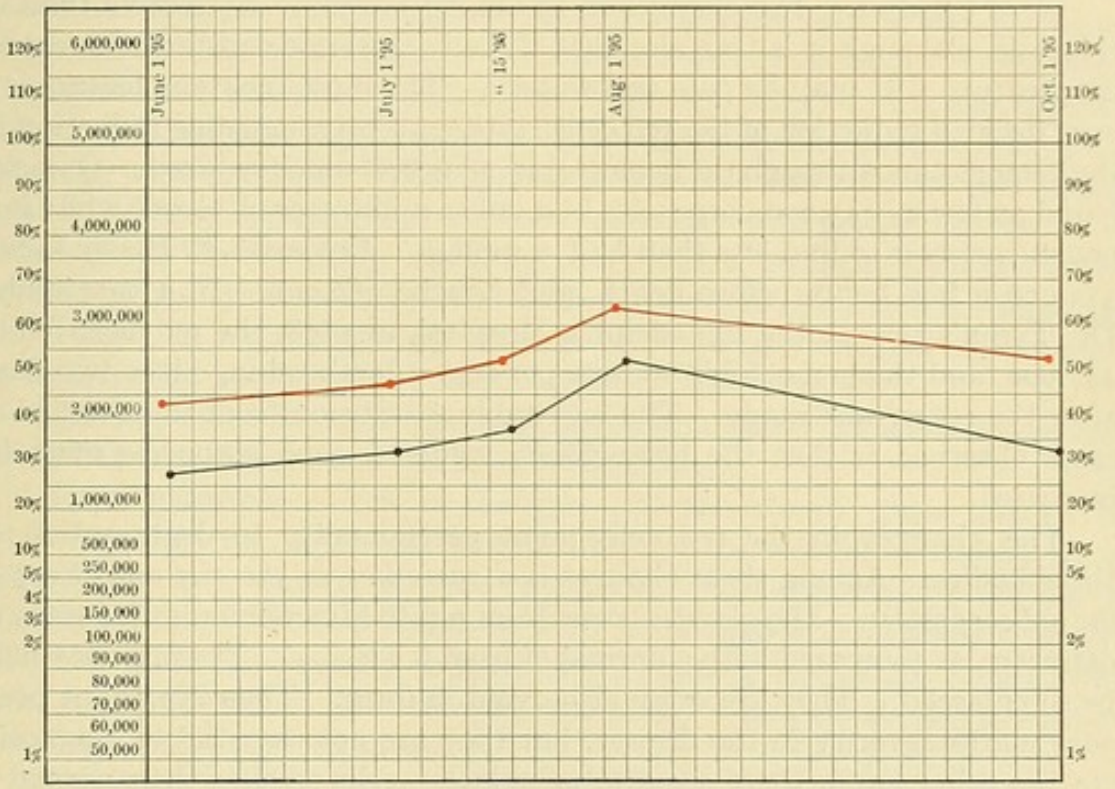


BONE MARROWS.—1. Normal. 2. Typical progressive pernicious anemia. 3. Scattered areas of megaloblastic degeneration in progressive pernicious anemia. 4. Aplastic anemia.—(*From Lavenson's paper on Aplastic Anemia. Transac. Assoc. American Physicians, vol. xxi*, 1906.)

(To face page 641)

Fenwick, Osler and Henry. *Intestinal parasites*, the *bothriocephalus latus*, may undoubtedly produce symptoms clinically indistinguishable from the general anemia which Addison characterized as "occurring without any discoverable cause whatever—cases in which there had been no previous loss of blood, no exhausting diarrhea, no chlorosis, no purpura, no renal, splenic, miasmatic, glandular, strumous, or malignant disease."

Symptoms.—The approach of pernicious anemia is most insidious, beginning with a gradual *progressive weakness*. What is first interpreted as a causeless weariness or languor grows slowly into an extreme debility, with faintness on the slightest exertion, and thence into a state of thorough muscular weakness, which ultimately prostrates the patient, and he is too weak to



Red Corpuscles—Black. Hemoglobin Red.

FIG. 119.—Blood in Pernicious Anemia.

rise from bed. To this succeeds a state of mental hebetude and bodily torpor. Rarely this extreme debility is substituted by a remarkable vigor. *Nor is there emaciation.* The body bulk is well preserved. The *skin* acquires gradually a *lemon-yellow* hue, whence the disease has been mistaken for diseases of the liver. The mucous membranes, on the other hand, are blanched, as may be seen in the lips, gums, and mouth. The sclera are pearly white.

Cardiovascular symptoms are especially conspicuous in progressive pernicious anemia. Hemic murmurs, visibly pulsating and throbbing arteries, even pulsating veins, have been noticed. The large, but soft, jerky pulse, resembling that of aortic regurgitation, was mentioned by Addison. The capillary pulse is also frequently seen, and hemorrhage, cutaneous and retinal, occur.

Digestive derangements form an important part of the symptomatology

of pernicious anemia. Indisposition to take food or, rather, a disgust for food, nausea, vomiting, and diarrhea are often troublesome symptoms. According to Hunter, sore mouth and sore tongue are often present. *Hydrochloric acid* is constantly *deficient* in gastric digestion (achylia gastrica). *Moderate* elevation of *temperature*, irregular and intermittent, is also noticed, while *nervous symptoms*, including numbness, languor, and even paralysis, are sometimes present. Paresthesia is especially emphasized as an early symptom, even the earliest. Symptoms with loss of patellar reflexes often cause the case to resemble locomotor ataxia.

The *urine* exhibits no constant changes, being sometimes pale and sometimes dark-hued. The dark color is ascribed by Mott and Hunter to an excess of urobilin.

Blood Changes.—The changes in the blood are more distinctive than in chlorosis, although it is true also that there is no single constant characteristic feature. It may be pale and watery. The most constant feature is a very decided reduction of the red cells, without a corresponding reduction in the hemoglobin, although the hemoglobin, *in toto*, is much reduced. Quincke found as few as 143,000 corpuscles in a cubic millimeter of blood, while it is not uncommon to find less than half a million. Frederick P. Henry found 315,000 a few hours before death, and Laache 360,000. In a case under Dr. Tyson's care at the Philadelphia Hospital, in 1898, the red disks fell to 437,000, and the hemoglobin to nine per cent., death taking place two days after the count was made. The inevitable conclusion from the average of cases observed is that the hemoglobin value of each corpuscle must be increased.

The changes in the red cells point preeminently to degeneration of these bodies. Among these changes is a *variation in the size and shape of the red corpuscles*. *Megalocytes* occur from ten to fifteen micromillimeters in diameter, as compared with a normal of from 6.5 to 9.4. The majority may be so enlarged. They are often also ovoid in form. Their abundant presence marks severity in the disease, but they are not essential to diagnosis. On the other hand, there are also *microcytes*—cells smaller than normal—and *poikilocytes*—corpuscles characterized by great irregularity in shape. While these irregular shapes were first demonstrated in connection with pernicious anemia, they occur in all severe anemias. *Polychromatophilia.*—A condition in which the erythrocytes stain irregularly and unevenly is always present and accompanies the enlargement of the cell. Basic granulation or granular degeneration of the red cell, a variety of polychromatophilia is very frequent. Degeneration of the red cells shown by a stippling when stained, is common.

Nucleated red corpuscles are a constant constituent of the blood of pernicious anemia, and have also been regarded by their discover, Ehrlich, as almost pathognomonic. Two kinds are found—first, the small, normal-sized corpuscle with its deeply stained nucleus (normoblasts), and certain large forms with pale nuclei (megaloblasts). They are not confined to this disease. Blood plaques are either absent or very scanty. Karyokinesis is rarely found in these larger cells, but when present in connection with the other changes it is regarded by some as almost pathognomonic. *Leukocytes* are usually diminished in number due to a decrease in the polymorphonu-

clear leukocytes, while there is a tendency to an increase of the mononuclear white cells, as compared with health.

Morbid Anatomy.—Various tissues have been studied in the effort to find a morbid anatomy for pernicious anemia. In the absence of lymphatic involvement or enlargement of the spleen, except sometimes in small degree, the *marrow of bones* has claimed close study. H. C. Wood described the red condition of the marrow of long bones in 1871. It was further studied in this country by William Pepper[1] and Tyson,[2] and abroad especially by Cohnheim.[3]

Although the appearances described by these observers are not identical, they are sufficiently constant to justify their association as more than accidental. *Summed up*, they amount to this: Marrow dark red; consistence less soft; fat vesicles absent; specific lymphoid cells increased, including marrow cells of various sizes, containing one or more nuclei; numerous nucleated red corpuscles present, especially the larger forms, the gigantoblasts of Ehrlich. These studies were made before the days of differential staining and counting. More recent studies add neutrophiles and eosinophiles. These appearances are now commonly interpreted as due to an effort of the blood-making apparatus to reproduce the disintegrated erythrocytes. They are not, however, constant, as the marrow is sometimes pale or yellow. It is a reproducing process, an irregular attempt at regeneration, a hyperplasia, as contrasted with "aplastic."

The appended illustrations from Lavenson's paper shows well the contrast between normal and diseased bone-marrow.

The deposition of *iron in the liver cells* has already been alluded to. It is found in the outer and middle zones of the lobules, and may be so distributed as to outline the bile capillaries. It is regarded by Hunter as characteristic. The liver itself is often fatty and is sometimes enlarged. The iron is, in like manner, sometimes increased in the kidney, and spleen, and these organs are not otherwise essentially changed, though the spleen has been found reduced in size.

The *heart muscle* is fatty, while the other muscles are unusually red. Other morbid changes are described, but they cannot be regarded as essential. Such are changes in the ganglion cells of the sympathetic, and *sclerosis of the posterior columns* of the cord, first studied by Lichtheim.[4] Softening of the upper part of the lumbar cord has also been reported by Sir Dyce Duckworth.[5] While the associated changes in the spinal cord are so constant—84 per cent. of cases collected by Cabot—that they cannot be regarded as accidental, experimental studies by Burr and Griffith intended to determine this relation to pernicious anemia resulted in nothing definite. Complete atrophy of the secreting tubules of the *stomach* has been described by Fenwick, and by William Osler and F. P. Henry in one case studied jointly by them.

Diagnosis.—The diagnosis of pernicious anemia may be uncertain at first, but the true nature of the disease soon declares itself. The intense

[1] "Progressive Pernicious Anemia," "American Journal of the Medical Sciences," October, 1895.
[2] "Die Betheiligung des Knochenmarkes bei perniciöser Anæmie," "Virchow's Archiv," 1877, lxxi, 118–126.
[3] "Virchow's Archiv," October, 1876
[4] "Congress für innere Medicin," 1887.
[5] "British Medical Journal," November 10, 1900.

anemia, extreme weakness, digestive derangements, and cardiovascular symptoms, in connection with blood-count of 1,000,000 or below, with a relative increase, or at least no proportionate diminution, in the hemoglobin, and an admixture of megalocytes, microcytes, and poikilocytes, point to a condition scarcely mistakable. It may be said, moreover, that almost never in the case of a pernicious anemia do the number of corpuscles fail to fall below 1,000,000. The large forms of nucleated red corpuscles have been regarded as characteristic, but are also found in leukemia (see also diagnosis of Cancer of the Stomach, p. 380). The marked remission of the symptoms is a marked feature of these cases.

Prognosis.—The prognosis is to-day regarded as less unfavorable than it was a few years ago, since recent experience has developed the fact that temporary improvement is not uncommon, and it is said that recovery sometimes takes place. Still, Addison's original prognosis, of a termination sooner or later fatal, is seldom astray.

Treatment.—Treatment of pernicious anemia is, moreover, not fruitless. The same measures which are almost a specific for chlorosis are not without effect in pernicious anemia. Accordingly, arsenic, to a less degree iron, good food, and favorable hygienic surroundings, are to be adopted. The arsenic treatment has been followed by results which justify the words "temporary cure," and it is said that permanent cure has followed. Such temporary cures have covered a period of three years. The best preparation appears to be Fowler's solution, in gradually increasing doses, until 20 and even 30 minims (1.3 to 2 c.c.) are reached, and this three times a day. It should be continued for a long time, for weeks or months, with intermissions of a few days if unpleasant results appear, to be again resumed. Arsenic is not a specific for pernicious anemia, but the results of its use are often surprisingly gratifying. Cacodylate of sodium has been recommended as less irritating than other preparations of arsenic, being suitable also for hypodermic use. The dose is 1/2 a grain (0.033 gm.), three times a day, hypodermically 1/3 to 1/2 grain (0.002 to 0.033 gm.) every other day. Atoxyl is still another preparation of arsenic given in doses of 1/3 to 1/2 grain (0.022 to 0.033 gm.) hypodermically every other day. Inhalation of oxygen has also been recommended, as advised in leukemia. The relation between chlorosis and pernicious anemia, already referred to, is sustained by therapeutic results. Certain cases of chlorosis very closely resemble pernicious anemia, especially when not arrested by treatment. The arsenic, administered as directed, is wonderfully well borne, nausea and vomiting being rare. Rest in bed is indispensable, but should be supplemented with massage, if possible. Food should be in easily assimilable shape, such as beef-juice, and peptonized milk. Salol has been suggested as an intestinal antiseptic, from the standpoint that the disease may be due to toxins absorbed from that canal. From the same point of view lavage of the large bowel through an opening into the cecum has been tried using peroxid of hydrogen and solutions of permanganate of potash. Recently, too, William Hunter has suggested the use of antistreptococcus serum to counteract possible general infection.

Transfusion of blood, which seemed at one time to give promise of

favorable results, discontinued, but has been revived and as much as 500 c.c. of human blood injected into a vein.

O. Huber[1] employs the intramuscular injection of blood, and reports four cases of which in three the results were favorable, but advises the coincident use of arsenic and other suitable remedies. He injects small quantities of defibrinated blood on the gluteal region—human blood from a person not suffering from fever or active disease. The blood is defibrinated by the use of glass pearls and filtered through linen, 10 to 20 c.c. being injected every five to eight days. It is thought to stimulate the body to restore the blood to its normal composition.

For intravenous injections care must be taken to secure a homologous serum. Most recently Ehrlich's 606 has been employed intravenously with advantage.

III. LEUKEMIA.

Definition.—A disease characterized by an enormous increase in the colorless corpuscles of the blood, by hyperplastic changes in the bone marrow, the spleen, and in the lymphatic glands, in a word in all the leukoblastic tissues.

The disease has been called *leukocythemia* as well as *leukemia*, the former of these words meaning white-cell blood, the latter simply white blood. From the etymological and histological standpoint, leukocythemia, suggested by Hughes-Bennett, is the more accurate term, but Virchow's term, leukemia, has become the one in common use.

Two forms are recognized, *myeloid leukemia* and *lymphoid leukemia*. In the former the marrow cells are present in large numbers associated with changes in the bone marrow, while in the latter the lymphatic tissues are also invaded and lymphocytes large and small predominate in the blood.

The extremely rapid course of certain cases of leukemia justifies its division into an *acute* and *chronic* form, acute leukemia being, however, relatively rare. Ebstein was the first to call attention to the acute form reporting a fatal case in which the whole duration of the disease, including a prodromal stage, was but six weeks. Similar cases are reported by others.[2] Fraenkel collected all the cases to 1895, including ten of his own. M. H. Fussell,[3] A. E. Taylor and Jopson collected 56 cases. Mina[4] in 1901, 69 cases. Since this time many cases have been reported. The duration of the chronic form may extend over years. Leukemic women have been repeatedly pregnant and have borne children at term.

Fraenkel concluded that all cases of acute leukemia are lymphoid or lymphatic in which the increase of white cells consists chiefly or almost firmed. It would seem, however, that both varieties may be acute or chronic. The lymphoid variety furnishing more acute cases, while the chronic is almost always myeloid.[5]

Etiology.—Nothing definite is known of the cause of leukemia. It

[1] Deutsche Med Wochenschrift, June, 1910.
[2] "Wiener klin. Wochenschrift," 1894.
[3] "American Medicine," March 5, 1904.
[4] "Wiener med. Rundschau," 1901, Nos. 37 and 38.
[5] See R. J. M. Buchanan's paper on leukemia in the Practitioner's Cyclopedia of Medicine and Surgery, Oxford, 1912.

occurs in all countries, in both sexes, and all ages, although it is more common in middle life and in males—both forms. Cases have occurred as early as the eighth week and as late as the 70th year. It is sometimes hereditary, but leukemic women have borne nonleukemic children. It has succeeded upon exhausting illness. Malaria has been assigned as a cause, and certainly its association with this disease has been seemingly more than accidental. To a less degree this is true of syphilis. Pregnancy is said to favor it, and to have been found in the lower animals.

The idea of the infectious origin of leukemia, advanced by Klebs and supported by observations of Osterwold, Roux, Byrom Bramwell, Pawlowsky, Kelsch, Vaillard, and others, seems well founded, but no single microorganism has been found associated. A case has, however, been reported where an attendant on a case of leukemia contracted the disease and died. The frequent association of leukemia with stomatitis and intestinal ulceration was pointed out by Hunterberger.

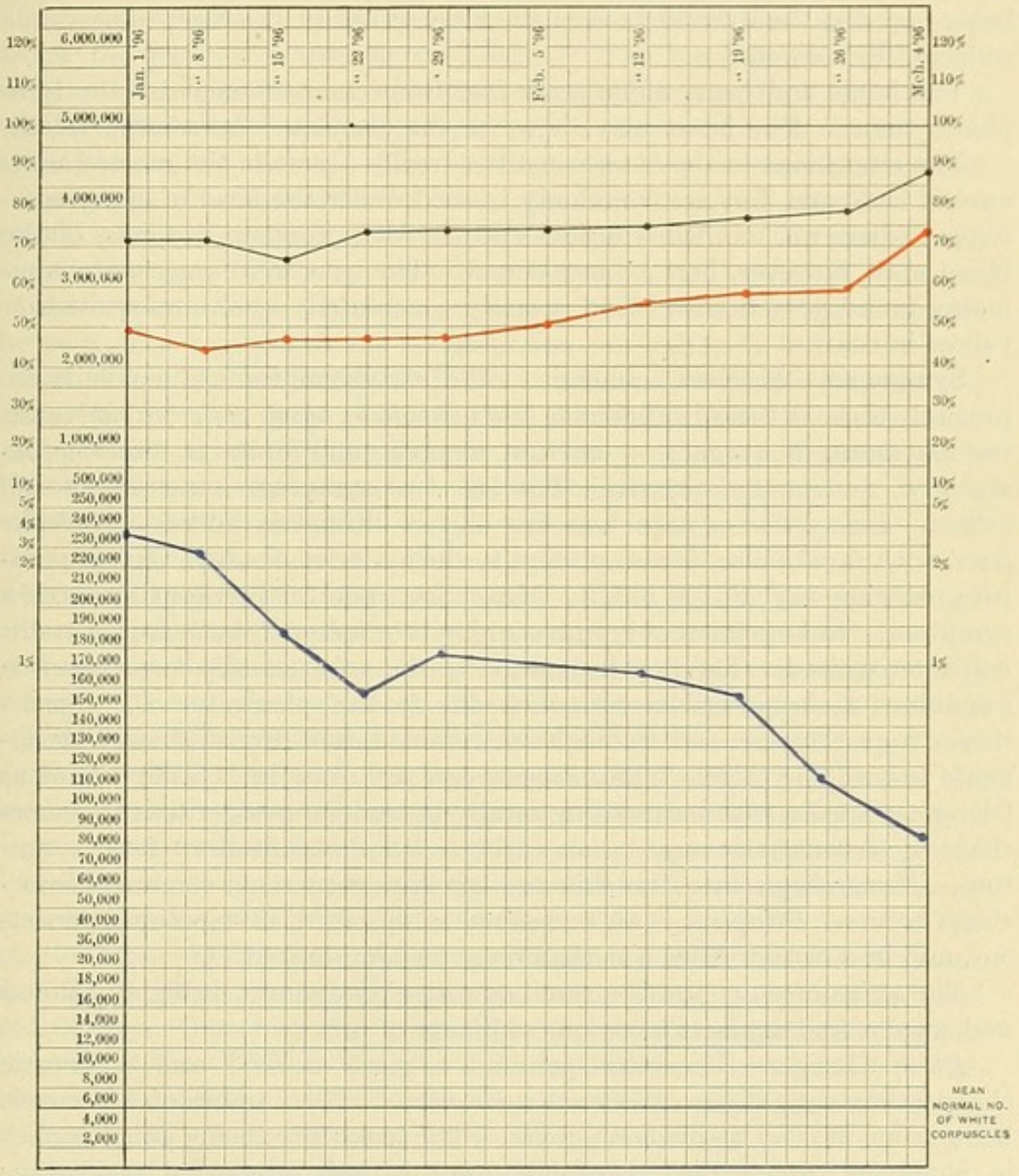
Morbid Anatomy.—Leukemia has a definite morbid anatomy, consisting in alterations in the blood and in the hemogenic apparatus, including the spleen, the lymphatic glands, and the marrow of bones.

The *spleen* is almost always enlarged. It may be adherent to the abdominal walls, the diaphragm, stomach, or other viscera. The splenic changes exhibit three stages in their development. In the first, the spleen is simply hyperemic, soft, and swollen, sometimes even ruptured. The Malpighian bodies share in the hyperemia, and may be slightly enlarged, but are overshadowed by the swollen pulp. In the second stage, hyperplastic changes make their appearance in the Malpighian bodies, and as these grow the pulp is intruded upon. They may reach such size as to be recognized by the naked eye as spherical gray nodules one to three lines in diameter, or they may be elongated or forked following the course of the blood-vessels. The third stage furnishes the granitic spleen, in which white dots are separated by dark streaks representing the destroyed pulp pigmented by the disintegrated blood. The spleen is now hard, and is cut with resistance. Its size may be enormous, and the organ may weigh from two to 18 pounds (1 to 9 kilos.). It does not attain as large size in acute leukemia as in chronic, being palpable in but 65 per cent. of cases. There is sometimes dropsy present. The mass of the blood may be measured (polyhemia) the heart and veins being distended with blood clots amounting, in a case of Osler's to 620 grams (20 ounces). Such blood may be whitish, from the admixture of a large proportion of white cells; sometimes the clots have a peculiar greenish color. It may weigh from 2 to 18 pounds.

The *lymphatic enlargement* is a true hyperplasia. Not only do the glands enlarge, but new foci of lymphatic tissue appear in various organs, as the liver and kidneys. All the more prominent groups may share in the enlargement—the cervical, axillary, inguinal, and perineal glands. The individual glands remain, however, soft. The lymphatic follicles in the tonsils and in the tongue, pharynx, and mouth may enlarge. This is also occasionally the case with the solitary glands of the intestine and the agminated glands of Peyer.

The *marrow changes* may be described, in a word, as reversion to the

embryonal type of medullary tissue. The fat of the adult marrow has disappeared, and a mass of lymph cells mingled with nucleated red corpuscles in all stages of development takes its place. The marrow is often *pyoid*, or it may be dark brown in color. The shell of bone is sometimes expanded. The lymph cells include numerous large mononuclear cells, many in the act of division, also multinuclear leukocytes. There are also nu-



Black—Red Corpuscles. Red—Hemoglobin. Blue—Colorless Corpuscles

FIG. 120.—The Blood in Leukemia.

merous marrow-cells or myelocytes and eosinophiles, like those found in the blood.

The *liver* is often enlarged, and, according to von Jaksch,[1] *pari passu* with the spleen, and it has this further peculiarity, that its edges are rounded, while in what he describes as *pseudoleukemia infantum* the *edges are sharp*, and the enlargement does not go hand in hand with that of the spleen. The

[1] See cases reported by J. Chalmers Camerson and Saenger, Sajous, "Annual" for 1891, E.

liver is also at times infiltrated with leukemic patches and nodules, not unlike miliary tubercles. The same is occasionally true of the kidney.

The *thymus* gland has been found enlarged in some cases of acute lymphatic leukemia, and even the *skin*, *stomach*, and *gastrosplenic omentum* have been the *seat* of growths, presumably lymphatic. In fact, there is no situation in which such growths may not make their appearance, although on the whole they are not common, only thirteen cases of nodules being found in the liver and ten in the kidneys out of 139 cases of leukemia collected by Gowers.

The lungs and heart alone seem free from encroachment by the lymphatic tissue. The heart may, however, be dislocated by a large spleen.

The alterations in the *blood* constitute really a part of the morbid anatomy of leukemia, but are commonly treated under the head of symptomatology, where we, too, will consider them. An increase in the mass of the blood may, however, here be mentioned. The heart and vessels are commonly found gorged with blood, usually coagulated, sometimes whitish or yellow in color.

Symptoms.—Myeloid Leukemia. The early symptoms of leukemia are precisely those of the other anemias, viz.: *Insidious onset, pallor, rapid breathing amounting to dyspnea* on exertion, *weakness* and *faintness, headache, indigestion*, and *loss of appetite*. The last two symptoms may precede all others. *Emaciation* is absent at first but is ultimately added. *Moderate fever*, with rapid pulse, is also present in the majority of cases, the temperature reaching 103° F. (39.4° C.). Headache, more or less continuous, is a symptom. Enlargement of lymphatic glands or spleen or both are generally easily recognized. The first intimation of the presence of a *large spleen* is sometimes a circumscribed *peritonitis* due to the splenic tumor. *Hemorrhages* from the nose and stomach are common, and dropsical swelling appears toward the close. *Nasal hemorrhages* are sometimes fatal. Thomas Oliver reported a case terminating fatally by sudden postperitoneal hemorrhage.[1] *Hematemesis* may be an early and almost initiatory fatal symptom. *Hemorrhages* into the skin are sometimes present; cerebral hemorrhage occurs. *Priapism* is an occasional symptom; it is sometimes persistent, and in a case of Edes was the first symptom noticed.

The *urine* often contains a small quantity of albumin, is highly colored and scanty, and deposits a copious sediment of uric acid.

Blood Changes.—The blood exhibits a most marked and diagnostic change consisting in an *enormous leukocytosis*. The number of white cells being from 100,000 to 500,000 per cm. The leukocytes being polymorphus in character, myelocytes, basophiles and entirely abnormal percentage relations between the polymorphonuclear and lymphoid cells. There is also a large number of nucleated red cells.

The maximum proportion of colorless corpuscles impresses decidedly the color of the blood *en masse*, making it pink, or even the color of chocolate and milk. There is an increase in all varieties of white cells. The polymorphonuclears (neutrophiles) include 30 to 50 per cent.; large and small lymphocytes 10.6; and while actually increased are relatively diminished; eosinophiles 4 per cent. and coarse granular basophilic or mast cells 1 to

[1] Sajous' "Annual" for 1890, vol. i., E, p. 12.

10 per cent.; the myelocytes foreign to the normal blood from 30 to 50 per cent.; nucleated red disks pockolocytes, and blood plaques are numerous.

Numerous *nucleated red disks* are present, especially in the myeloid variety. Cabot says "the striking point is the presence of very numerous nucleated red cells even in the absence of any signs of anemia. With over 4,000,000 well-formed and well-colored red cells we may have hundreds of erythroblasts in every cover-glass. They are as numerous in this form of leukemia (myeloid) as in the worst forms of pernicious anemia, even though the patient be feeling very well." The hemoglobin falls below the normal proportion, so that the hemoglobin content of each disk is lowered. There is occasional poikolocytosis. Reduction of the red cells comes late in the disease. The blood-plaques may be slightly increased.

Symptoms of Lymphoid Leukemia.—Lymphoid Leukemia may be acute or chronic. Acute Leukemia resembles an acute infection. The patient may give all the symptoms of typhoid fever and run a fatal course in from two to three weeks. Only a blood examination will make a certain diagnosis. The case may begin with an inflammation not unlike diphtheria and run a fatal course in two weeks. There is a leukocytosis the lymphocytes numbering from 80 to 90 per cent. of all the white cells. In a case examined by one of us, the white cell count was but 7000 just before death with 98 per cent. of large lymphocytes. Hemorrhages are common.

Recent observations[1] show certain cases of infection with from 70 per cent. to 90 per cent. of *lymphocytes* which recover.

Chronic Lymphatic Leukemia.—Is chronic in course; there is enlargement of all the lymph glands; a slight enlargement of the spleen. The blood contains a great excess of large and small lymphocytes often 90 to 95 per cent. The cases are arbitrarily divided intosmall and large cell varieties.

That the alkalinity of the blood is sometimes diminished is true; that it is ever replaced by acidity is not true, as was at one time held. Its specific gravity is lowered to 1030 to 1050. Leukemic blood coagulates slowly, a feature which has been ascribed to the presence of albumoses.

Leukanemia is a term applied by von Leube to a condition of combined leukemia and severe anemia. The disease lasts for from a few days to three months. It may begin suddenly with fever and severe tonsillitis to which are added weakness, hemorrhage, extreme pallor and rapid decline. There is often general glandular enlargement including the liver and spleen. The reduction of hemoglobin and erythrocytes with increase of lymphocytes usually the large form are conspicuous. The red cells may be as low as 1,500,000. The color index is high.

The blood picture is that of lymphatic leukemia.

Diagnosis.—The diagnosis of leukemia requires the aid of the microscope, but with it it becomes easy. (See Blood Changes.)

Chloroma is a tumor-like hyperplasia of the parent cells of the leukocytes, primarily in the red marrow of bones, and secondary in the periosteum. It is now classed among the leukemias. Chloroma differs from other forms of leukemia in its marked neoplastic type and in the green infiltrations and metastases.

Diagnosis.—This can be made only by the aid of the microscope.

[1] R. C. Cabot, Amer. Jour. Med. Sc., March, 1913.

Tubercular adenitis, malignant adenitis, general sarcomatous or carcinomatosis, chronic spinal enlargement might be mistaken for leukemia without a blood count.

Typhoid fever, diphtheria, and pneumonia have been the diagnosis in acute forms, but the blood picture described above will certainly make the diagnosis. A differential count is essential because frequently a great leukocytosis occurs in sepsis. We have notes of an appendicitis with 60000 white cells, 80 per cent. polymorphonuclear. In the terminal stages or under treatment the white cells may be greatly reduced, but the differential count will still make the diagnosis.

Prognosis.—The prognosis or leukemia is unfavorable, the best that can be expected from treatment being the deferring of the fatal end. Some rather remarkable fluctuations are noted, and cases of cure are even reported. Osler saw a case ten years after the original diagnosis was made by Wm. H. Draper. The lymphatic leukemias are the more acute and more intractable.

Treatment.—The treatment has heretofore been mainly with arsenic and iron, fresh air, and good food. Large doses of arsenic—as much as 30 drops (0.92 c.c.) of Fowler's solution, reached by gradual increment have been especially recommended and certainly should be tried.

Treatment by X-ray.—The Roentgen ray has been employed in the treatment of leukemia, and no treatment of leukemia and pseudoleukemia is of greater value. The results have not been uniform, although on the whole they may be said to be sufficiently encouraging to be used in every case. No cures have been reported, although frequent remissions, one in which there was no recurrences for three and a half years after the exposures. It must not be forgotten that marked remissions in the progress of leukemia occur under any treatment. Recent experience has shown that the rays must be applied not to the spleen but to the long bones. Roentgenologists think that when arsenic and X-ray are used together, a careful watch must be kept upon the patient. X-ray is dangerous in acute cases.

Benzol has lately been given in doses of from 7 to 15 minims—case reports show diminution of white cells and diminution in the size of the spleen with a general well being of the patients.

IV. PSEUDOLEUKEMIA—HODGKIN'S DISEASE.

Synonyms.—*Hodgkin's Disease; Lymphadenosis; Lymphadenoma; Malignant Lymphoma (Billroth); Adénie and Lymphadénie; Lymphatic Anemia.*

Definition.—The disease consists essentially in an anemia accompanied by an enlargement of the lymphatic glands and the formation of lymphatic foci in the spleen and occasionally in other glandular organs, but associated with only a slight increase in the colorless corpuscles of the blood. Hodgkin's paper, to which we are indebted for our first definite knowledge of the disease, appeared in 1832.

Etiology.—Its etiology is as undetermined as that of leukemia. Depressing influences of all kinds are believed to favor it. Tuberculosis has

been associated with it, but the conclusion reached after much discussion, is that it is not tubercular. The presence of an irritating (infection?) substance in the blood has been suggested, and the necessity of local irritation, associated with a lymphatic diathesis, has been insisted upon by Trousseau. It may occur at any age, but is more common in adult life, and in males.

Morbid Anatomy—Its morbid anatomy is, however, definite. There is both lymphatic and splenic involvement, the latter secondary to the former. The tonsils, intestinal lymphatic structures, and even the liver and kidneys may be invaded. There is, moreover, a deposition of new foci of lymphatic tissues decidedly more marked than in leukemia. The *lymphatic enlargement* usually begins first in the more superficial groups, as those of the anterior and posterior cervical triangles, the glands of the axilla, and the groin, but the entire lymphatic system may be involved, including the retroperitoneal glands, resulting sometimes in marked abdominal enlargement. Occasionally the overgrowth is limited to the deep-seated glands. Of the abdominal, the retroperitoneal are most frequently involved, producing tumors which have been mistaken for myomata of the uterus. The bronchial glands may also be involved, and by their pressure produce dyspnea and suffocation.

The lymphatic enlargement is a hyperplastic one, shared by the cellular and trabecular tissue in varying degrees. When the former predominates, the product is soft and exudes a milky juice on section; when the latter, it is firm and resisting. The individual glands are not disposed to fuse nor to become adherent to adjacent tissue, differing in this respect greatly from glands enlarged by the tubercular process. The enlargement also exceeds that in leukemia.

Histologically,[1] the earliest changes as found in the smallest glands consist in increased vascularity with hyperplasia of the lymphoid cells and active proliferation at the germinal center, together with proliferation of the reticular endothelium. Mitotic figures are not infrequent in the reticular cells. The lymphoid sinuses contain many small and large mononuclear lymphocytes, epithelioid cells and a few eosinophiles. As the reticulum increases it grows coarser and the glands harden. In the last stages almost all traces of the normal structure are lost and only here and there are seen fragmentary remains of the lymphoid follicles and sinuses. In the spaces of the reticulum lie many small and large lymphocytes, plasma cells in large numbers, polymorphonuclear lymphocytes, eosinophiles in enormous numbers, epithelioid cells and large uninuclear and multinuclear giant cells. The giant cells, also often found in large numbers, are apparently traceable through the epithelioid cells to the endothelium of the reticulum. The various cell types are not equally distributed, lymphoid cells predominating in some parts, epithelioid cells in others, and giant cells and eosinophiles in others still.

After the lymph glands the *spleen* is more frequently involved. It is enlarged in various degrees, sometimes enormously. The changes in

[1] For a complete and thorough report of the histological changes in the organs and tissues in Hodgkin's disease, to which we are indebted for much contained in the text, the reader is referred to the excellent paper of W. T. Longcope, "On the Pathological Histology of Hodgkin's Disease with a Report of a Series of Cases," published in the Bulletin of the Ayer Clinical Laboratory of the Penna. Hospital, October, 1903.

the enlarged spleen are quite different from those in Banti's disease or splenic anemia. On section, it is found filled with lymphomatous nodules composed of a tissue like that of the lymph glands. The earliest changes are seen in the Malpighian bodies. They consist in a hyperplasia of the lymphoid cells with early thickening of the reticular network and proliferation of the endothelial cells, forming grayish-white masses varying in size from a lentil to a walnut (1/4 inch to an inch—0.6 to 2.5 cm.), contrasting strongly with the dark red parenchyma. Typical uninuclear and multinuclear giant cells, eosinophiles and plasma cells are also present, sometimes abundantly. The connective tissue increases with the growth of the nodules in varying proportion. It is not reparative and represents an advanced stage of the disease.

The *bone-marrow* is soft, very largely composed of cells, the fat being more or less replaced by lymphoid marrow. Myelocytes and large lymphocytes are the predominating cells, but myelocytes and polymorphonuclear leukocytes containing eosinophilic granules may also be numerous, though few of the typical bone-marrow giant cells are present, while the small lymphocytes are not relatively increased. Nucleated red blood-corpuscles are sometimes present in small numbers, usually of the normoblastic type. According to Longcope the most remarkable deviation from the normal consists in the great excess of the eosinophilic leukocytes and myelocytes, though he also says they may be absent.

The *liver* becomes the seat of secondary growths and is enlarged. The initial change is a deposit of small foci of lymphoid cells, in which take place the same changes as in the spleen and lymph glands. To this is added perilobular cirrhosis and fatty degeneration.

Similar changes may take place in the *thymus gland* and the kidney. Indeed, there is no organ in the body in which they may not occur, not excepting the nervous system. Paraplegia has resulted from pressure on the cord by growths in the spinal canal. The posterior nares may be occluded by invasion of the tonsils and the numerous lymphoid follicles in the pharynx. In like manner the intestinal walls may be invaded, producing thickening, while even serous surfaces do not escape.

The latest studies by Fischer, Reed, Longcope, Simons and Yanasaki go to show that the process is inflammatory and infectious, distinct from tuberculosis and Banti's disease, although tuberculosis may coexist.[1]

Symptoms.—The symptoms of Hodgkin's disease are, again, the *pallor*, *weakness*, *dyspnea*, *palpitation*, *dizziness*, and other signs of anemia, concurrent with or even sometimes in advance of the glandular enlargement. There is quite often *fever*, very irregular and variable in degree, and cases have been observed by Murchison and De Renzi in which there was *paroxysmal glandular enlargement* coinciding with fever, the enlargement subsiding with the decline of fever, but not reaching the degree present prior to the enlargement. In a case of Laache's the glands diminished in size during the fever. In a case in the wards of the Hospital of the University of Pennsylvania in which the glandular enlargement was not conspicuous, there occurred an intermittent rise of temperature supposed

[1] Reed, Dorothy M., on the "Pathological Changes in Hodgkins' Disease with especial reference to its relations to Tuberculosis," Johns Hopkins Hospital Reports, vol. x., 1902.

to be due to peritonitis; an operation did not show any peritonitis but great involvement of the abdominal lymph glands.

The external glandular growths are variously conspicuous; occasionally, however, they are wholly absent. There is no fixed order of involvement, although the glands of the anterior and posterior cervical triangles commonly enlarged first, and with the acme of their growth produce a striking picture. The enlargement is not uniform, but at times remits and even ceases. It is said it may disappear altogether for a time. The glands are usually soft, sometimes there is even a sense of fluctuation.

The glandular enlargement themselves contribute further to the, symptoms by there effects. Thus, in the case of the bronchial glands *dyspnea from pressure* on bronchi or trachea may occur, and may also be intermittent. Pressure elsewhere may lead to *pleuritic or abdominal effusions*, while the enlargement of nerves in the growth may cause *pain*. Erosion of bone may result. *Bronzing of the skin* has been found associated with enlargement of the abdominal glands. A *purpuric rash* is sometimes present in Hodgkin's disease, due perhaps to the hydremic state of the blood.

Macroscopically, the *blood* appears thin and pale. Minutely examined, the red corpuscles are diminished in number, although not always. Minimum counts make 960,000[1] to the cubic millimeter, while in a case reported by Henry,[2] that of a boy of five, with enormous enlargement of the right cervical glands, there were 5,462,000 to the cubic millimeter. Thus, the diminution is less than in pernicious anemia. The hemoglobin is, however, reduced to at least 60 per cent., furnishing thus one of the conditions essential to anemia. There are few nucleated red corpuscles and poikilocytes, and especially microcytes. The leukocytes may be slightly increased, occasionally decidedly so; there is no approximation to the leukemic state of the blood, and the two states are distinct and separate.

Diagnosis.—The diagnosis requires some care, as more than one condition is attended by similar glandular outgrowths. Chronic and even acute *adenitis* has been mistaken for the early manifestation of Hodgkin's disease, while the converse has obtained perhaps more frequently. Time is the arbiter of such uncertainty.

A group of *tuberculous glands* resembles more closely the disease under consideration, but it is not usually difficult to distinguish between the two. Tuberculous glands are adherent to each other and to adjacent tissues, while the lymphadenoid growths are loose and easily movable. Tuberculosis rarely involves more than one group of glands, is characterized by caseation and suppuration, while the lymphadenoid growths almost never suppurate. Yet the tubercular process is the slower. Tuberculosis is commonly found in young persons under 20. Hodgkin's disease may occur at any age, but the average age is greater. It is more common in males. In Gowers' 100 cases, 75 were males and 25 females; 30 were under 20, 34 between 20 and 40, and 36 above 40.

Sarcoma also involves groups of glands, and in the beginning the consistence of the glands is similar to that in Hodgkin's disease. But this

[1] Case reported by Richard Geigel, quoted by F. P. Henry ("Anemia," Philadelphia, 1887), from "Deutsches Archiv für klinische Med.," 1885, Bd. xxxvii., p. 59.
[2] *Op. cit.* p. 67.

disease rapidly invades surrounding tissues, fusing with them, and destructive ulceration soon makes its appearance.

Carcinoma of lymphatic glands should also be mentioned as producing a somewhat similar growth, associated with cachexia, but it is for the most part secondary to cancer elsewhere than in lymphatic glands.

Finally, all the conditions named as possible to be mistaken for Hodgkin's disease are limited to single groups, while the latter always extends, and the fact of such limitation is of itself sufficient to preclude the disease.

From leukemia the disease is easily distinguished by the leukocytosis characteristic of the former.

Prognosis.—While the prognosis is ultimately fatal, the course of the disease varies greatly, and death seldom results in less than a year. F. P. Henry places the average duration of life at two years, but admits it is greatly modified by such circumstances as age and previous health of the patient.

Diagnosis can only be made certainly by postmortem. The appearance of the bone marrow is seen in the accompanying plate.

Aplastic anemia is a pernicious anemia differentiated from ordinary pernicious anemia by tendency to hemorrhage, with a lower color index, and with a smaller degree of erythroblasts. At postmortem, instead of the red hyperblastic bone marrow, the bone marrow is yellow, and there is an atrophy.

Treatment.—Treatment, too, may avert the fatal termination for a long time. Extraordinary results in this respect have followed the administration of arsenic, and even recoveries have been reported. Large doses, arrived at by gradual increment, should be attained and kept up until some physiological effects are observed. Such doses are from 15 to 20 minims (1 to 1.3 c.c.) of Fowler's solution. Particularly happy results are claimed for the hypodermic injection of arsenic. Especially is this recommended when arsenic is not well borne by the stomach. The cacodyllate of soda is now used hypodermically, the dose being 1/2 a grain to 3 grains. Supporting treatment of all kinds, including quinin, cod-liver oil, and the beso of food, is necessary.

Operative interference is sometimes necessary to avert danger to life, threatened by the encroachment of enlarged glands on vital organs and functions, such as respiration. It has even been claimed that the removal of a group of primarily enlarged glands has cut short the spread of the disease, but such an apparent result is rather an evidence of error in diagnosis. In view of the fact that at an early stage a diagnosis is impossible, the removal of a local group of glands should be recommended.

The X-ray treatment has been availed of in treating Hodgkin's disease it is claimed with results as flattering as in leukemia. The hope of permanent cure has even been held out. The rays are applied directly to the enlarged glands. At times success is very prompt, while at others long and patient treatment is necessary, as many as 500 applications extending over several years being necessary. When success occurs it is desirable to give an occasional dose at intervals to keep up the effect.

Status Lymphaticus.

Synonym.—*Lymphatism.*

Definition.—A hyperplastic state of the lymphoid tissue throughout the body, including the lymphatic glands, the spleen, the thymus, and the lymphoid marrow of bones, occurring chiefly in children and young persons.

This condition is rare in this country. It has been described by Poltau and other Vienna physicians, and by James Ewing, of New York.[1]

Morbid Anatomy.—The lymphatic glands most frequently affected are the pharyngeal, thoracic, and abdominal; those of the cervical, axillary, and inguinal regions less frequently and in less degree, while the lymphatic elements of the tonsils and the upper pharynx, and the solitary and agminated follicles of the small and large intestines, are often much involved. The spleen is moderately enlarged, while the Malpighian bodies stand out distinctly. The thymus gland is enlarged and soft, and on section may exude a milky fluid. The bone-marrow may be hyperplastic, and the yellow marrow replaced by red marrow. Along with these anatomical changes have sometimes been found rickets, and, again, hyperplasia of the heart and aorta.

Symptoms.—The symptoms in addition to the anatomical changes noted are a lowered power of resistance, sometimes evidenced by sudden death or death from insufficient cause. The subjects are said to be poorly developed and infantile in appearance. As might be expected, the recognition of the actual condition is not always easy, if it dare be assigned a separate place in the nosology.

V. SPLENIC ANEMIA.

Synomyms.—*L'epitheliome primitif de la rate; Splêmomegalie primitive.*

Primary Splenomegaly with Anemia.

Definition.—A chronic condition in which there is progressive enlargement of the spleen without marked swelling of the lymphatic glands, associated with more or less pronounced anemia. The term Banti's disease is applied to its termination in cirrhosis of the liver with ascites.

Etiology.—This is undetermined, except that there is reason to believe that the condition sometimes succeeds the infectious diseases. It is to be separated from the enlarged spleen, so often the result of chronic malaria, sometimes associated with a moderate anemia. It occurs alike in old and young, but is more common in adults, males rather than females four to one.

Morbid Anatomy.—The spleen is greatly enlarged, approaching the size of the leukemic spleen rather than the spleen of Hodgkin's disease. It is three or four times its normal size, but retains its normal shape. It is indurated, and its incisures are deep. Its capsule is thickened and opaque in spots and sometimes adherent to adjacent tissues, often true of any large spleen.

[1] "New York Medical Journal," July 10, 1897.

There is nothing distinctive in the histology of splenic anemia or of Banti's disease. The process in the spleen is commonly regarded as chronic inflammatory. There is a hyperplasia of all the splenic elements associated with diffuse increase in the connective tissue and overgrowth of the reticulum, destroying and replacing the Malpighian body, by a tuberculous product which may be as large as a pea and contain central giant cells. There may be endothelioid proliferation and multinuclear epithelioid cells scattered through sections, and especially about the Malpighian bodies, the centers of which may also be the seat of hyaline degeneration. Eosinophiles may be present; also plasma cells. In the stage known as Banti's Disease there is superadded interlobular cirrhosis of the liver with pigmentation, and sometimes chronic diffuse nephritis.

There are no lymphomatous masses in the spleen as in Hodgkin's disease or enlarged lymphatic glands.

The bone-marrow is cellular, made up chiefly of large mononuclear leukocytes and myelocytes some of which contain eosinophilic granules; also many normoblasts and a few megaloblasts.

Symptoms.—The symptoms are analogous to those of pernicious anemia, and include *pallor*, *weakness*, *dyspnea*, *palpitation*, associated with the signs of: First, *enlarged spleen*, evidence of which is sometimes shown by its weight and the pressure it exerts before other symptoms show themselves. There is also pain in the region of the spleen which increases on pressure and may radiate toward the back and loins. Rarely it may be due to perisplenitis, which may even invade the adjacent pleura. Finally, there results the cachectic state characterized by *emaciation*, *a deeper yellow color and pigmentation of the skin and mucous membranes*, a tendency to *hemorrhage* and *pyrexia*, with a possible temperature of 102° F., *edema*, *serous effusions*, *nausea*, *and diarrhea*, *extreme muscular prostration*, and *mental hebetude*. There is also said to be at times, as in lymphatic pseudoleukemia, an intermittent or course in the symptoms to the extent of apparent complete restoration to health in the intervals.

The *blood* exhibits the changes one would expect in cases in which there is destruction of the tissue devoted to its reproduction. It is anemic. The red disks are notably diminished, from 5,000,000 to as low as even 2,000,000. The hemoglobin is correspondingly diminished, with low color index. There is marked leucopenia.

Hemorrhage manifests itself especially as hematemesis, epistaxis, bleeding from the gums and even in the fundus of the eye. Hematuria and hemoptysis are infrequent; hematemesis is often excessive. Late in the disease there is ascites due to cirrhosis of the liver (Banti's disease).

An *increase in the urea* of the urine has been noted by Strümpell, and is regarded as evidence of increased proteid metabolism.

All clinical facts go to show that the spleen is responsible in some way for a destruction of erythrocytes and of their capacity for carrying oxygen.

The duration of the disease is from one to ten years.

A form of enlarged spleen described by Gaucher is known as the Gaucher type. The enlarged spleen is associated with enlarged liver. It attacks more than one member of a family. It presents a grayish red color on section with whitish spots and streaks. Histologically there are large

cells from 20 to 40 μ in diameter filling alveolar spaces. Gaucher himself described the condition as *epitheliome primitif* and it is commonly regarded an endothelioma and not a splenic anemia.

Diagnosis.—The diagnosis of splenic anemia depends upon the presence of splenic enlargement associated with the phenomena of anemia described, and the absence of glandular enlargement, so conspicuous in Hodgkin's disease, or of blood changes of leukemia. Anemic symptoms attend the chronic malaria so often associated with enlarged spleen, but the history of malaria in such cases is invariably present, while the degree of anemia in malaria is not so high.

Prognosis.—The disease, always prolonged, was formerly alternately fatal, but the prognosis has been modified by splenectomy.

Treatment.—The medical treatment is that for the other anemias, by iron and arsenic and nutritious food. Here as in other affections of the hemapoietic system the X-ray directed to the enlarged spleen has been followed by reduction of size and relief of pain. In one of Buchanan's cases complete restoration to health after extreme symptoms followed the administrations of pituitary extract.

It is in this disease that splenectomy has been so satisfactory. In Banti's collection it appears to have been successful in three out of four cases, and later statistics seem to confirm this statement. Banti recommended splenectomy in all cases except profound cachexia and in pseudo-leukemic hypertrophy of lymphatic glands. He considers ascites indicates checexia.

ERYTHEMIA.

Synonyms.—*Vaquezs Disease; Polycythemia vera. Polycythemia with splenic tumor. Osler's Disease.*

Definition.—A disease characterized by an increase in the number of red blood cells, a plethoric state, enlargement of the spleen and at times cyanosis. It has been studied by Vaquez in 1892 Rendu and Widal, Cabot, McKean, Saundly and Russell; in 1903; by Osler who stimulated interest in it; by Mackey and Anders in 1907, Stachelin in 1911 and Lucas in 1912. The last collected 149 unquestionable cases, in an exhaustive paper recently published.[1]

Pathology.—The disease is regarded as a lesion of the erythroblastic tissue of the bone marrow as leukemia is of the leucoblastic tissue. It is analogous to the hyperglobism of higher altitudes in which there is also increased activity of the erythroblastic bone marrow compensatory to lack of oxygen in the air. There is increased viscosity of the blood, which favors stasis and capillary engorgement. Rarely there is cheesy tuberculosis of the spleen.

Symptoms.—The most important symptoms are polycythemia, a congested state of the capillaries and veins, and enlargement of the spleen. The congestion may extend to the conjunctiva. In cold weather the congested appearance gives way to cyanosis, very striking in the face and hands,

[1] Lucas, Erythremia or Polycythemia with Chronic Cyanosis and Splenomegaly. Archives of Interna Medicine, 1902.

but subject to change of position. The enlargement of the spleen is moderate while the organ is also hard and firm and painless.

The change in the number of blood discs is remarkable, the polycythemia ranging from seven to twelve and even 13 millions per cubic millimeter, while the hemoglobin ranges from 139 to 160 per cent. The color index is low, the specific gravity high. The red cells are but little changed. In most cases there is a moderate or decided leucocytosis. In a few instances the white cells are reduced in number.

There is headache, flushing and vertigo, constipation and usually albuminuria. The blood pressure is high in the majority of the cases, but observes no law. There may be hemorrhage into the skin and mucous membranes. Recurring ascites was present in two of Osler's cases. Osler regards as a variety Geisböckss cases of *polycythemia hypertonica*, further characterized by increased tension, arteriosclerosis and nephritis. In this variety according to Anders the spleen is not enlarged.

Diagnosis.—This is easy when the three cardinal symptoms are present without emphysema or congenital heart disease; and the cyanosis is not traceable to coal tar products.

Prognosis.—This is bad for cure but cases last some time with fair health They die of cardiac failure, hemorrhage and recurring ascites.

Treatment.—The symptoms may be relieved by purging, bleeding and low diet and by inhalation of oxygen. Splenectomy is not advised. The X-ray proved useless in Osler's cases, but favorable cases have been reported.

HEMOGLOBANEMIC CYANOSIS

Definition.—A change in the composition of the hemoglobin of the blood, causing a permanent cyanosis.

Etiology.—By far the commonest cause of hemoglobanemic cyanosis is the use of headache powders sold freely by the druggist to the laity. The powders contain acetanilid or phenacetin or (acetphenetidin); almost any of the coal tar products including sulphonol, trional and other chemicals, such as potassium chlorate, carbon monoxide and sulphuretted hydrogen, will cause this hemoglobanemic disturbance.

Stokvis pointed out that some of the cases of chronic cyanosis with hemoglobanemia are associated with intestinal disturbances, to which disturbance he attributes the hemoglobanemia. He named these cases "Enterogenous Cyanoses." The Spectroscope will discover whether the blood change is due to methenglobin or sulphemiglobin.

Diagnosis.—This condition of chronic cyanosis may be easily mistaken for cyano is due to interference with circulation, either from heart or lung disease. But in the heart or lung cases, the symptoms and physical examination will show the presence of disease of these organs.

The cases which are due to disturbances of the intestinal tract have symptoms pointing to the intestines.

The drug cases usually come complaining only of the color of their skin, the lips, the finger tips; the whole surface of the skin is of a leaden hue, looking not unlike the poisoning in chronic Argyria. Sometimes there is rapid feeble heart action.

Examination of the blood shows a dark chocolate color which makes it impossible to estimate hemoglobin by any ordinary means.

Treatment.—Perhaps the most important point to remember is that the cases which are due to acetanilid soon acquire the habit of taking the drug and almost without exception attempt to deceive the physician. The drug causing the disease must be searched for carefully and removed. Many cases are much weakened by the continual use of the drug and need rest, tonics (such as strychnin) and good food.

In cases which are due to intestinal intoxications search for and removal of all parasites and food causing fermentation should be debarred and intestinal antiseptics such as salol and bismuth should be tried.

PURPURA.

SYNONYMS.—*Morbus maculosus; Peliosis.*

Definition.—A name given to several dyscrasic states, all attended by subcutaneous or submucous extravasations of blood. Such extravasations do not disappear on pressure, and vary in size from that of a pin-point to areas a centimeter or more in extent. When minute or punctiform, they are called petechiæ; when larger than this, ecchymoses. An indisposition on the part of the blood to coagulate is commensurate with the tendency to extravasation. Purpura is always a symptom rather than a disease, but in certain conditions it forms the most conspicuous symptom of a group which scarcely admits of any other classification. In this event an adjective term derived from some more conspicuous one of these symptoms, or from the name of some investigator who has described the condition, is added to give precision. In other instances it is so purely a symptom and plays so minor a rôle in the disease that it is called symptomatic. Under any circumstances it is not always easy to keep the varieties distinct.

SYMPTOMATIC PURPURA.

This includes the forms of purpura in which the petechiæ and ecchymoses are usually of minor importance. In a few instances in which the dyscrasia is very great they become by their number and extent indices of the degree of such dyscrasia. Such are: Old age.—Infectious diseases, toxic condition such as snake bite, cachexias, tuberculosis, leukemia, and so forth.

SCURVY.

SYNONYM.—*Scorbutic Purpura, Scorbutus.*

Definition.—A disease characterized by a dyscrasic state of the blood, associated with subcutaneous or submucous hemorrhages, by a peculiar spongy state of the gums, and extreme general weakness.

Etiology.—Less than half a century ago the idea of scurvy was always associated with the seafaring life, since sailors were its chief victims, though

almshouses and prisons also held their complement. In the food of these persons fresh vegetables and vegetable juices and organic salts were wanting. So it came to be acknowledged that such privation was responsible for scurvy, and proof of this belief was thought to exist in the fact that with the quicker voyages of ships and a supply of suitable food, scurvy had almost vanished from the nosology. According to Hutchinson[1] there are three theories as to the causation of the disease: First, that the disease is essentially an acid intoxication due to diminution of alkalinity of the blood, second, that it is caused by poisoning with ptomaines; third, that it is the result of a specific poison, the latter being based upon experience in the Boer War. All the theories however acknowledge that, privation, insanitary surroundings, exposure and overwork or deficiency of fresh vegetables form the predisposing causes which lead to the disease.

At the present day scurvy has become a rare disease, but is still met in camps, prisons, almshouses, and situations where the food causes named exist along with dampness, foul air, and depressing influences generally, among which nostalgia is supposed to be especially potent.

The disease attacks the old and young of either sex, though the old are more susceptible, and it happens, probably from accidental circumstances, that more males are affected than females.

Morbid Anatomy.—This consists in (1) alterations of the blood; (2) the extravasations of blood, which may be anywhere—subcutaneous, submucous, sub-serous, intermuscular, and interstitial. The blood changes are not distinctive. The blood is dark and thin, the blood-corpuscles and hemoglobin are concurrently reduced in number, and there is no leukocytosis. Rarely there is even sloughing of the skin and mucous membranes, leaving ulcerated patches in the skin and bowels. The spleen is soft and enlarged, and there may be degenerative changes as well as hemorrhages in the bladder, kidneys, liver, and muscles.

Symptoms.—The more evident symptoms are the *changes in the gums*, and the *deep-seated and superficial hemorrhages*.

The *gums* are swollen, soft, and spongy, with disposition to bleed easily. In the more severe cases there is ulceration, with loosening and falling out of the teeth, the tongue is swollen, and the breath excessively foul. The gums of young children and of the aged are more often uninvaded. In rare cases only is there *necrosis* of the jaw.

The *hemorrhages*, always petechial, appear usually first in the lower extremities, then on the arms and trunk, but they occur anywhere as roundish, dark-red spots which may assume larger size. They are rare in the face and scalp, and are less common under the mucous membranes and in deep-seated tissues. Subperiosteal hemorrhages may occur. Necrosis of bone may occur. Nasal hemorrhages may be frequent, melena and hematuria are rare, hematemesis and hemoptysis still rarer. The extravasations are slow to disappear, even when recovery takes place. The occasional sloughing has been referred to. A residual, slowly healing and sometimes foul ulcer results.

"Scurvy sclerosis" is a condition most frequent in the legs in which

[1] "The Relation of Scurvy to Recent Methods of Artificial Feeding," "N. Y. Med. Jour.," Feb. 23, 1901. "Scurvy, Not Rheumatism," "Phila. Med. Jour.," Feb. 2, 1901; "American Pediatric Society's Collective Investigation on Infantile Scurvy," "Arch of Ped." July, 1898.

infiltration takes place of the subcutaneous tissue and muscles producing a brawny discoloration, the skin covering which may be blood stained.

Other symptoms are *debility*, extreme in severe cases, and *anemia*. The *pulse* is small, feeble, and frequent, and corresponds to the heart's action, which is sometimes irregular; more rarely is it slower than in health. The *temperature* is normal, rarely somewhat elevated. *Sore throat* is mentioned as a premonitory symptom. In bad cases *nephritis* and *endocarditis* occur. Articular swelling is an occasional symptom; it is one of the results of the dyscrasia; so are *wheals* and *vesicles*. In some cases these conspicuous symptoms are much less pronounced and even absent.

Diagnosis.—This depends, as stated, on the etiology, the gingival changes, and the hemorrhages. It is these which chiefly distinguish it from the other forms of purpura.

Prognosis.—Sporadic cases always get well, and epidemic cases usually, unless too far advanced before coming under treatment.

Treatment.—This is usually most satisfactory when the necessary conditions are fulfilled—a restored wholesome hygiene and suitable food. Good ventilation and outdoor life in healthy localities, and plenty of fresh vegetables, fruits, and fresh meats, ordinarily suffice to accomplish a prompt cure. It is usual to give lemon and orange-juice as the types of the fruit-juices. Tonics and roborants, of which iron, quinin, and strychnin are the type, are the medicines needed. Calcium chlorid or calcium lactate may be used in doses of from 5 to 15 grains (0.3 to 1 gm.). Antiseptic and astringent mouth-washes should be used, and ulcers should be stimulated by local applications, of which nitrate of silver in solution is the best.

Infantile Scurvy.

Synonyms.—*Barlow's Disease; Periosteal Cachexia.*

Definition.—A cachectic condition of infants, associated with subperiosteal hemorrhagic extravasations. Due to improper food.

Etiology.—This might be said to be the dependence of physicians upon manufactures of patent foods. The papers read before the American Pediatric Society prove that the most common cause is the use of artificial foods, of these the proprietary foods take the lead. It, however, has been observed in children fed in apparently proper manner.

Symptoms.—Barlow's account is graphic. The condition exists essentially in a *hemorrhagic subperiosteal extravasation*, causing thickening and tenderness in the shafts of the bones beginning in the lower extremities, but invading also the forearm and arm to a less degree, more rarely the scapula, vault of the cranium, and face. Rarely there is intermuscular extravasation, later may appear bruise-like ecchymoses and rarely small purpuric spots. The resulting *tenderness* and *pain on motion* cause the child to keep quiet, with the legs drawn up; immovable (pseudo-paralytic) as long as undisturbed, and to cry out when handled. It is soon evident the pain is in the lower extremities. Then there follows obscure and ill-defined swelling around the shafts of the bones in rapid succession above the epiphyseal junction. The position of the limbs

is now somewhat different, the leg being everted and immobile for the cause above mentioned. The inability to move the limb depends on the pain. The lesions are symmetrical but not absolutely so. The *joints* remain *free*. The sternum and adjacent cartilages and a small portion of the contiguous ribs may be sunk bodily back as though subjected to violence. There may be a sudden *prolapse of an eyeball*. Along with these symptoms are profound *anemia* and *erratic temperature*, which may be subnormal, normal, or as high as 102° F. (38.9° C.).

The disease occurs at any period after four months, but it is most common from the ninth to the 18th month, and develops rapidly.

Diagnosis.—The disease with which infantile scurvy is most commonly confused is rheumatism. But in scurvy there is the history of improper food. There is swelling and tenderness along the shafts of the bones. In rheumatism there is swelling of the joints. The X-ray will show a swollen tibia or humerus. It may be confounded with Parrot's disease or syphilitic pseudo-paralysis or osteo-chondritis. Young children exhibiting symptoms of paralysis should be carefully examined for it. The lesion of scurvy does not involve the epiphyses and diaphyses except in rare cases while in sypilitic osteo-chondroses this is the characteristic lesion. In both there is pseudo-paralysis.

Treatment. The substitutions of the proprietary food or sterilized milk by modified cows milk, and the administration of whole-grain cereals. Orange-juice or lemon-juice will bring about a recovery which is marvelous in its rapidity and completeness. The child who gives all the above symptoms, in from 24 go 48 hours will be apparently in perfect health, when so treated.

Arthritic Purpura.

Synonym.—*Rheumatic Purpura.*

Definition.—The characteristic feature of arthritic purpura is a joint involvement. Hence it is also called rheumatic purpura.

Symptoms.—Three varieties are distinguished:

1. *Purpura Simplex, Simple Arithritic Purpura.*—This is a mild form, most frequent in children. The *articular pain* is very mild and attended with but slight fever. The *spots* are found for the most part on the *legs*, more rarely on the trunk and arms. There may be *digestive derangement*, manifested by loss of appetite and diarrhea. The condition terminates favorably in a week or ten days. It may be associated with a mild degree of anemia.

2. *Peliosis Rheumatica.*—Schönlein's disease. This is a much more serious affection from every standpoint, occurring usually in young persons from 14 to 30. The *joint* symptoms are pronounced and multiple, and there are decided *swelling*, *pain*, and *fever*, with a temperature of 101° to 103° F. (38.3° to 39.4° C.). The *eruption* first appears on the legs near the affected joint, but it has been present extensively on the arm, distant from the joint, followed by sloughing; in the same case were retinal hemorrhages. *Sloughing* and *necrosis* of the skin even have occurred. It may be

simply purpuric, or may be associated with urticarial wheals—exudative—or vesicles (pemphigoid purpura). When severe, it is often associated with hematuria and hemorrhagic nephritis with edema. Endocarditis is also a complication.

3. *Henoch's Purpura.*—This is a variety occurring most often in children, but also in adults, characterized by *severe gastro-intestinal* disturbance with colic *in addition to the previously named symptoms.* There are pain, vomiting, and diarrhea, rarely intestinal ulceration and perforation with fatal peritonitis. Acute enlargement of the spleen has been observed.

Here, also, recovery is the rule.

Diagnosis of Arthritic Purpura.—The diagnosis is easy by reason of the associated joint symptoms, but the same doubt exists as to a true rheumatic nature in all forms.

Prognosis.—This is regarded as favorable, but fatal terminations do occur, especially in peliosis rheumatica in which there is nephritis. Relapses in this form may occour at the samme time of year for several years in succession.

Purpura Hemorrhagica.

Synonym.—*Morbus maculosus Werlhofi.*

Symptoms.—This is a severe form of purpura characterized by *hemorrhage* from the *mucous membranes*, including nose, mouth, palate, stomach, and intestinal canal, in *addition to extensive subcutaneous ecchymosis.* It attacks all ages but particularly young and delicate girls. The brain and kidneys and serous membranes may also be seats of hemorrhage—apoplectic symptoms indicating the first. A *prodrome* of languor and weakness may precede for a couple of days, to be succeeded by a rapid succession of ecchymoses and hemorrhages. More decided constitutional disturbances follow, including typhoid symptoms and fever, though the latter is mild and may be altogether absent, even in severe cases. Death may take place from loss of blood or hemorrhage *into the brain*, or recovery in from ten days to two weeks.

In the *purpura fulminans* the hemorrhages are mainly confined to the skin, producing confluent ecchymoses and dense infiltrations covering large areas, with sanguineous blisters. The internal organs, on the other hand, remain free, while the urine and the bowel evacuations are natural. At times there is fever; at others, not. Hemorrhagic purpura has occurred after pneumonia and scarlet fever, and again in children apparently healthy. In these fulminating cases death may occur before purpura manifests itself.

Diagnosis.—As to diagnosis, *scurvy* is almost the only condition liable to be mistaken for purpura hemorrhagica. In the latter the gums are intact, and there is an absence of the conditions favoring scurvy.

Prognosis.—The termination is usually favorable in from ten days to two weeks, although there may be fulminating cases, usually in children, terminating fatally in 24 hours. Severe cases recover more slowly.

Treatment of Arthritic Purpura and Purpura Hemorrhagica.—Treatment is best directed to improving the quality of the blood and to build-

ing up the general tone rather than to the control of the hemorrhage, though the latter must not be entirely ignored. Almost all that has been said of the tretment of scurvy is applicable to these forms of purpura. Iron and arsenic are the typical blood-builders, to which nutritious food, including vegetable-juices, is to be added. Arsenic should be given in full doses, beginning with small ones and ascending rapidly.

In the articular forms the salicylates and salicin should be used in such doses as the stomach will tolerate.

Lactate or chlorid of calcium in doses of 15 grains, (one gram,) is a valuable remedy. The injection of a normal blood serum will give often immediate improvement.

Hemorrhagic Disease of the New-born.

Hemorrhagic Syphilis of the New-born.—Usually about from the *third* to the *fifth* day after birth hemorrhage is observed at the navel of the child, or it may occur earlier. Blood also flows from the mucous membranes of the mouth, the bowels, and the kidneys. The skn becomes jaundiced. The stomach rejects food, and though it may appear well nourished at birth, the child rapidly wastes, and dies at the end of a *week* or *ten* days. The autopsy discloses *syphilitic lesions* in the liver, lungs, nasal passages, and elsewhere.

Epidemic Hemoglobinuria of Infants, or Winckel's Disease.—As described by Winckel in 1879, in an epidemic at the Foundlings' Hospital, at Dresden, the first symptoms, noticed usually on the *fourth day after birth*, are a bluish tinge on the skin of the face, trunk, and limbs, with a more or less icteroid hue. There are *fever*, *rapid breathing*, and sometimes *cyanosis*. Occasionally there are *vomiting* and *diarrhea*. The *urine* is light brown, albuminous, contains methemoglobin, and deposits a sediment consisting of epithelium and tube-casts. The *blood* contains an excess of leukocytes and numerous granular bodies. The child *lives*, on an average, *two days*, though in one case death supervened in nine hours. The autopsy in this case disclosed yellow staining of the skin and internal organs; the spleen was large, hard, and darkened; the kidneys were dark brown in color, their tubules being filled with granular pigment; the liver and heart were fatty. There may be punctiform hemorrhages on the surface of the internal organs. There is no septic condition of the umbilical vessels. An infectious origin is not unlikely.

Acute Degeneration of the Internal Organs of the New-born, or Buhl's Disease.—How far this disease, described by Buhl in 1861, differs from Winckel's disease, or the latter from the former, remain to be settled, for, in the first place, fatty degeneration of the heart and liver is found in many cases of Winckel's disease, while in others there is found the general fatty degeneration of kidneys, liver, heart, etc., described by Buhl. In the second place, infants surviving the first few hours after birth in Bulh's disease have the same symptoms as those described under Winckel's disease, while the other symptoms, such as minute hemorrhages and bile staining of various internal organs, are not essentially different.

Morbus Maculosus Neonatorum.—Still another form of hemorrhage

from one or more of the surfaces, and especially of the alimentary canal, in the new-born is described under this title. The bleeding generally begins within the *first week*, but may be as late as the second or third week. *Hemorrhage from the bowels* (melæna neonatorum) is the most frequent form, but it may be from the stomach, mouth, nose and navel, or from the navel alone. It may be accompanied by hematogenous jaundice—indeed, by any or all the symptoms described under Winckel's disease—but differs in the occasional presence of *fever* and apparent *absence of postmortem* lesions, though ulcers of the esophagus, stomach, and duodenum have been found. It is generally fatal in from one to seven days. All these conditions can be appropriately considered as forms of purpura.

Treatment.—The treatment of hemorrhagic affections of the new-born often avails little, though recoveries take place, especially in the last-described form, in which C. W. Townsend reports 19 recoveries out of 50 cases collected. The injection subcutaneously or intravenously of fresh human or animal serum, however, suggested by Weil affords relief. Ten c.c. may be injected subcutaneously at one dose, using as much as 100 or even 200 c.c. in four or five days. Welch of N. Y. has lately brought this treatment before the profession, he claims a large percentage of recoveries.

The treatment demands *absolute rest* with the head low. Even the exertion necessary in nursing at the breast should be interdicted, and the infant should be fed, while recumbent, with a teaspoon, using also the mother's milk if this be not condemned as worthless. The utmost care in providing *uniform warmth* should be taken. This can be best accomplished by means of an incubator.

HEMOPHILIA.

Definition.—A hereditary disease, manifested by a tendency to uncontrollable hemorrhage, occurring in males but transmitted by females.

Etiology.—Instances of fatal hemorrhage from this disease were observed centuries ago, the first mention of it seems to have been by an Arabian physician who died in 1107. "Families of bleeders" were first described in this country by Dr. John C. Ottis of Philadelphia in 1803, who also first used the word bleeder (Osler). Of great importance because of its bearing on the marriage of these hemophilic subjects is the fact that the tendency is transmitted through the female line rather than through the male. Thus, if a man belonging to a bleeding family who is himself not a bleeder marries a woman who is healthy and not a bleeder, his offspring are exempt from the affliction. On the other hand if a woman a member of a bleeding family marry, she may have offspring who are bleeders. These facts were pointed out by Grandidier. The females of such families are never bleeders, and only some of the males. The families of bleeders are apt to be large, and their appearance is that of health, as a rule. It is said that blondes predominate, with delicate, soft skin and distinct, distended veins.

More cases are reported from Germany, Switzerland and the United States than any other countries while some hold that it is more common among Jews. In 1893 Sir Almroth Wright showed that the coagulations

of the blood in bleeders ranged between ten and sixty minutes instead of the normal five to six minutes. He also showed that bleeders and female descendents of bleeders had a leukopenia particularly of the polymorphonuclear leukocytes. Upon these facts it is supposed that the cause of the bleeding in hemophilia is some disturbance with the phenomenon of clotting. Howell diagrammatically states that in normal individuals the cellular elements of the blood and the wounded tissue provide thrombokinase, that thrombokinase+calcium+thrombogen form thrombin, that the union of thrombin and fibrinogen form fibrin. In which of these factors necessary to complete clotting the fault lies is not decided because of the still unsettled state of the whole question of clotting of blood.

Symptoms.—Attention is commonly called to a bleeder by the occurrence of a hemorrhage difficult to control, though induced by some trifling cause. Epistaxis leads in frequency in 169 out of 334 cases collected by Grandidier. The extraction of a tooth is one of the most frequent of these events. It may be the prick of a pin, or a scratch, or a slight cut, as in vaccination, or no cause may be discoverable. The tendency may manifest itself at the cutting of the umbilical cord at birth, or in Jewish children at the circumcision. On the other hand, the same accidents which are without result early in life may induce the hemorrhage later. It may be induced by simply blowing the nose. Other situations were the mouth and stomach. Hemorrhages also occur in the interstices of organs, and though interstitial hemorrhages do occur, they are usually the result of trifling blows, when the well-known "black-and-blue" appearance is produced. One of us has reported two brothers who had huge hematomata apparently without cause. Hemorrhages into the joints and periarticular tissues occur in and about the knee.

The external hemorrhages, including those of the mouth and nose, may be profuse and even fatal. They often last 24 hours or longer. When checked, reaction from them is rapid, and the victims quickly resume their natural appearance, though repeated hemorrhages may engender a permanent anemia.

Joint affections may be associated with this as with the acquired hemorrhagic tendency. They involve usually the larger joints, and may include swelling and pain, with fever, producing a close resemblance to rheumatism, or there may only be pain.

Diagnosis.—This is apparent if the family tendency is known, but repeated hemorrhages from both or infancy are necessary to the diagnosis. Alarming hemorrhage without sufficient cause should excite inquiry, but no single hemorrhage however large or inexplicable should be regarded as hemophilia without one can trace back a history to a family of bleeders.

Prognosis.—*The younger the subject, the more serious the outlook*, but even in young children the outlook is not so very serious. Measures are now at hand which fairly well control the condition.

Treatment.—This may be prophylactic. Wright administered thymus tablets and certain forms of yeast to bleeders with the effect of diminishing the tendency to bleed. The administration of the calcium salts also lowers the coagulation time of the blood in certain instances. The actual hemorrhage may be controlled here as in scurvy and purpura by the

subcutaneous injection of blood serum. Human blood serum is better than foreign serum because it will not cause anaphylaxis, but the latter may be used. As much as 200 to 300 c.c. may be used in 24 hours. The children of bleeding families should be carefully guarded against traumatic causes, however slight, while they should be carefully looked after from the hygienic and nutritive standpoints. Fresh air, daily bathing, outdoor exercise, and judicious measures intended to harden the threatened subject should be practised. Plain, wholesome, and nourishing food should be given, and due attention should be paid to digestion. As a part of the prophylactic treatment, too, is discouragement from marriage especially in the case of women.

During an attack absolute quiet must be enjoined. Styptics are to be employed locally. Of styptics, the solution of the perchlorid or persulphate of iron is the best, beginning at first with dilute solutions and increasing to the full strength of the official solution if necessary. Tannic acid is another good styptic, and if at hand, may be dusted well upon the part or applied on cotton to cavities. In epistaxis the nose must be plugged if the ordinary methods of applying these agents fail. Wright suggests a "physiological styptic" made from the thymus gland of calf or lamb by extracting it with a normal salt solution, 10 parts of the solution to 1 part of chopped gland.

SECTION VI.

DISEASES OF THE DUCTLESS GLANDS.

DISEASE OF THE THYROID GLAND.

GOITER.

Simple Goiter or Struma.

Synonyms.—*Bronchocele; Thyrocele; Thick Neck; Derbyshire Neck.*

Definition.—The name is derived from Latin, *guttur*, throat. Under this name are included all enlargements of the thyroid gland other than those due to inflammation, malignant disease, exophthalmic goiter, or parasites.

Distribution.—Simple goiter may occur endemically or sporadically, but in this country it is only sporadic. It is, however, quite prevalent about the eastern end of Lake Ontario and in the State of Michigan. It is still endemic in certain parts of Finland and of Switzerland (cantons of Freiburg and Berne), in Italy (in the Southern Alps and in Savoy), in England, the Himalayas, in South America and in Asia, in Siberia. In the cantons named as many as 80 and 90 per cent. of recruits are found goitrous. It has even occurred in *epidemic* form in Finland.

Etiology.—The exciting cause of goiter still remains unknown, although a belief has long prevailed as to the endemic form that some constituent of drinking-water is responsible for it. That locality is in some way responsible is shown by the fact that removal from a territory subject to it arrests its development, while, if a healthy family moves into a goitrous district, the disease develops in some one or more members. A change in the water-supply of a district where goiter has been prevalent has led to its disappearance, while the water in certain wells on the continent of Europe is known to produce it. In fact, certain water is said to be drunk by men who desire to develop in themselves a goiter in order that they may be exempt from military service. What the responsible constituent of the water is, is, however, unknown, but it is supposed to be an organic impurity.

It is much more common in women than in men, according to different authorities seven to 41 times as frequent. It has been suggested that this is because women drink more water. The disease generally develops after puberty, sometimes after 50. Congenital cases are known. It is sometimes hereditary, but heredity must be separated from the operation of one cause on different members of the same family.

Morbid Anatomy.—All simple goiters start in a true hypertrophy of the gland follicles, and the entire gland then resembles a "normal but enlarged thyroid," but ultimately assume special peculiarities, on which are based anatomical varieties. According to anatomical peculiarities assumed after the goiter sets in, there occur: (1) Vascular thyroid, in which

the vessels are enlarged and dilated; (2) Fibroid thyroid, in which there is an excessive development of fibroid tissue; (3) Colloid thyroid, in which the follicles are enlarged and filled with colloid matter; sometimes the contents of the follicles become calcified or undergo amyloid change.

Symptoms.—It may be said of the majority of goiters that they cause no inconvenience, and are mainly objectionable through the resulting deformity. The size attained varies: the enlargement may but slightly exceed that of the normal gland, or the organ may be very large and pendulous. It may be one-sided, or bilateral, or only affect the isthmus. It is characteristic of all goiters and enlargements of the thyroid of any kind that they rise up when the patient swallows, and tumors of doubtful locality may thus be located. The goiter is sometimes low down, behind the sternum, and can only be felt during deglutition. Occasionally goiters have a wholly aberrant position and give rise to symptoms dependent upon disturbance of the parts in which they are situated.

A goiter may press on the trachea, causing dyspnea, or upon the esophagus, causing difficulty in swallowing. When behind the sternum, it may press upon the veins in the neck, causing swelling of the face and head, and sometimes headache and drowsiness. There may be pressure on nerves, especially the pneumogastric, causing spasm of the glottis, paralysis of the abductor, and even complete paralysis of one or both vocal cords.

Treatment.—When goiter is endemic, it is important to have the water supply carefully examined for defects. When possible individuals with beginning goiter should remove from the goitrous locality. Water of the district should *be boiled*. The drug treatment of goiter consists principally in the use of iodin. It is undoubtedly efficient at times. The simple iodin ointment or ointment of the red iodid of mercury may be daily rubbed into the goiter. It is recommended that after applications of the latter the neck should be exposed to the rays of the sun. This treatment has been especially efficient in India. Injections of iodin into the cyst are also used—20 to 30 minims (1.3 to 2 c.c.) of a solution of one part in twelve parts of alcohol twice a week, a new point being selected each time, care being taken not to wound any vessels or nerves. This is a dangerous procedure and should never be used until other methods of applying iodin are tried.

Internal treatment is frequently efficient. Naturally, the iodid of potassium is conspicuous among remedies, in the usual doses—5 to 20 grains (0.3 to 1.3 gm.) three times a day. Lugol's solution of iodin can be used. Thyroid extract is also being used with disputed success. Bruns treated 12 cases with raw thyroid glands in doses of 75 to 150 grains (5 to 10) gm. twice a week at first and once a week afterward. Nine were benefited. Kocher, however, thinks that the results with thyroid extract are no better than with iodin, and this view is now generally held.

If the goiter is produced by local causes, a change of residence is, of course, desirable. X-ray application is often of benefit.

Surgical Treatment.—In view of the extremely successful surgical treatment of goiter in the hands of competent surgeons, proper advice to patients is that with a very small operative risk—about 1 per cent. by the Mayo's last 1200 cases—a goiter may safely be removed never to return.

This will save time and the possible appearance of severe or fatal hyperthyroidism in the course of a simple goiter.

Hyperthyroidism.

Synonyms.—*Exophthalmic goiter; Struma exophthalmica; Graves' Disease; Basedow's Disease; Parry's Disease; Cardiothyroid Exophthalmos; Tachycardia strumosa.*

Definition.—A disease characterized especially by enlargement of the thyroid gland, protruding eyeballs, and palpitation of the heart and fine muscular tremors, due to hyperactivity of function of the thyroid gland.

Etiology.—Exophthalmic goiter is much more common in women than in men. It is also more common in the young adult and in the middle-aged. The average age may be put down at from 30 to 31 years; Bryom Bramwell says 15 to 30 for women and 30 to 45 for men. It has been observed as early as two and a half years, and as late as 68. Heredity is a rare factor, but its influence cannot be denied. It sometimes happens that several members of a family are affected. Sometimes myxedema affects one member of a family, and exophthalmic goiter another. It occurs with especial frequency in neurotic families. Sudden mental shock, worry and grief, and physical fatigue are assigned as exciting causes. So are many acute diseases, of which rheumatism is especially cited, also typhoid fever. Some of these are more likely to be coincidences. Its association with diabetes mellitus, though infrequent, is a recognized one. Some perversion of function of the thyroid gland lies at the foundation of exophthalmic goiter.

Morbid Anatomy.—The anatomical changes are as a rule hyperplastic as to gland tissues and colloid content, though terminating also in atrophy. The iodin content increases directly as the colloid matter.

Pathology.—Louis B. Wilson comes to the following conclusions from the examination of 294 thyroid glands of Grave's disease for the Mayo clinic. The development of the disease occurs as follows.

1. Following a metabolic chemical, or extra-organismal irritant, thyroid parenchyma proliferates, over-functionates and degenerates.

2. This process primarily resembles simple adenomatous proliferation, or reminds one of adenopapilloma.

3. Either process may start in a gland not previously enlarged by retained secretion or in one which is already distended with non-absorbed secretion.

4. The severity of the symptoms depends upon (*a*) the amount of absorbable secretion; and (*b*) the patient's ability to neutralize the secretion.

From a clinical standpoint he comes to the following conclusions:

1. Very early acute cases show pathologically hyperemia and cellular hyperplasia.

2. Later acute, moderate, severe and very severe cases show greater parenchyma increase and in many instances evidence of increased absorbable secretion.

3. Cases which clinically are showing any remission of toxic symptoms show somewhere within the gland more or less evidence of decreased function

in the exfoliation or marked flattening of parenchyma cells, or of probably decreased absorption, by the presence of thick, gelatinous stainable secretion, the so-called "colloid."

4. Patients who have recovered from their toxic symptoms and are now suffering principally from long previously acquired heart or nerve lesions or from myxedema, show exfoliated or much flattened (probably non-secreting) epithelium and large quantities of well stained, thick, gelatinous, probably non-absorbable, colloid.

5. The recently developed, very mild, or probably mild cases of long standing show pathologically almost always some total parenchyma increase by the multiplication of alveoli, but apparently not greatly increased functionating power of the individual parenchyma cells of the adenoid type.

6. Simple goiters should be regarded as multiple retention cysts filled with non-absorbable secretion, cell detritus, etc.

Symptoms.—Of the cardinal symptoms mentioned in the definition, the cardiac and vascular usually appear first. The *palpitation* is extreme, delirious, as it were, the pulse-rate being commonly in the neighborhood of 120 to 140, and sometimes reaches 200. The slightest excitement augments the pulse-rate instantly. The *cardiac impulse* is *strong*, but the volume of the *pulse small*. The *heart-sounds* are loud, audible to the patient and even at a distance from the body, in one case described by Graves himself as far as four feet. A *systolic murmur* is often heard at the base, usually soft, but sometimes loud; more rarely at the apex, when it may be due to relative insufficiency of the mitral or tricuspid valve. The blood pressure may be high in the toxic cases, or low or normal in other phases.

Exophthalmos is commonly described as the second of the cardinal symptoms to make its appearance, but it is nearly as frequent as palpitation. The degree of this protrusion varies very decidedly. It may be so slight as to be scarcely noticeable, while again the peculiar staring effect arising from it is conspicuous, and attracts attention instantly. Exophthalmos may be present on one side only, although some do not admit such cases in the category of true exophthalmic goiter.[1] The eyes show a large amount of white, and the eye-lids when closed often cannot cover the eyes. It is in these extreme cases that *von Graefe's symptom* presents itself—a condition in which, when the eye is cast down, the lid fails to follow it as it does in health. *Stellwag's sign*, is met. In it, the palpebral fissure is increased in width, owing to the persistent retraction of the upper lid. It may occur with or without von Graefe's sign. Retraction of the lower lid is occasionally seen. Moebius considers Graefe's symptoms the result of *Stellwag's*. The patient *winks* less frequently than in health. *Pulsation of the retinal arteries* can be seen with the ophthalmoscope, but other changes in the retinæ are rare. There is little change in the pupils. A *lack in ability to converge* the two eyes was pointed out by Moebius. Exophthalmos is absent in 20 per cent. of cases. Riesman has described a loud to-and-fro murmur occasionally heard over the eyeball.

The *thyroid enlargement* commonly presents itself at about the same

[1] See paper on "Unilateral Exophthalmos in Exophthalmic Goiter" by Posey and Swindells, "Ophthalmic Record," May, 1904.

time as exophthalmos. The goiter is usually of moderate size, almost never reaching the dimensions sometimes attained by a simple goiter. The tumor is largely contributed to by its vascularity, though there is also an overgrowth of the proper glandular tissue of the thyroid. *Pulse* and *thrill* are both palpable, while a loud systolic *murmur* may be heard on auscultation. The enlargement may be on one side only. In other cases there may be no enlargement. This may also be apparent only, but we are informed by the Mayo brothers that they have operated on exophthalmic goiters hardly palpable which proved greatly enlarged on exposure. When such a condition is associated with absent exophthalmos, the disease may be completely masked.

Another set of symptoms already alluded to as nervousness is restlessness, a disposition to start at the slightest sound, and wakefulness at night. A part of this, or due to the same cause, at least, is "*tremor*," a highly important symptom, of such frequency as to be included by George R. Murray in his definition.[1] It may be best studied by holding out the hand with the palm downward; even better by laying the examiner's palm lightly upon the patient's fingers when the hand of the latter is held out. Occasionally the whole body is affected. It occurs *eight or nine times* in a *second*. Its extent is small, but not always the same. It may be seen, too, in the foot, and in some instances the whole body appears to tremble. It is generally equal on the two sides of the body, but has been unilateral when goiter and exophthalmos have been on one side. It resembles the tremor of fatigue or that seen in recovery from long illness. Another symptom included in the same category is a *sudden giving away of the legs*, so that the patient falls to the ground without previous feeling of faintness or giddiness. *Painful cramps* sometimes occur. *Localized muscular atrophy* is occasionally met.

Excessive *sweating* is a frequent symptom. It may be intermittent or irregular, or there may be a simple feeling of flushing without sweating. *Diminished electrical resistance* was pointed out by Vigouroux. This is a natural result of the constant moisture of the skin. *Polyuria* often occurs, caused, perhaps, as is the sweating, through nervous excitation. A dark *coloration of the skin* sometimes takes place, more decided in those situations in which the pigment is naturally more abundant, such as the face and arms. Yet the flexures of the joints, the axillæ, the genitals, and the inside of the thighs are also affected. The skin may be uniformly bronzed, or it may be darker in patches. Parts of the body which are subject to constant pressure are also disposed to take on pigmentation more deeply. *Erythema* is frequent. *Edematous swellings* of the skin in various parts of the body may occur, and are to be carefully separated from edema, the result of associated conditions, such as anemia, organic heart disease, etc. It manifests itself as swelling in the feet and ankles, and has been ascribed to vasomotor paralysis. The *nails* sometimes become thin, and occasionally have a corrugated appearance.

Gastro-intestinal symptoms are frequent, manifesting themselves by attacks of *diarrhea*, apparently of nervous origin, coming on suddenly without pain, with copious loose motions, of which there are two or three or more in a day. Uncontrollable *vomiting* may be associated with this.

[1] "Twentieth Century Practice of Medicine," vol. iv., 1895.

Acute forms are sometimes thus ushered in. The *tongue*, however, *remains clean*, and there is, *as a rule, no rise of temperature*. Sometimes there may be very slight fever. The skin discoloration and gastro-intestinal symptoms suggest those of Addison's disease, and it is not impossible it may have been associated. *Rapid breathing* is a frequent accompaniment, equaling 30 to 40 respirations a minute. It may be associated with *cyanosis* of the face and swelling of the vessels of the neck. *Intermittent albuminuria* is frequent, as pointed out by Begbie. *Derangements of menstruation* are less frequent than might be expected, this function being normally maintained in the majority of instances.

The *mental condition* has been alluded to. It may be added that fits of depression alternate with buoyancy, while the moral nature may also be changed to a degree amounting to melancholia and mania. Active cerebral symptoms are sometimes present. This mania is of bad augury. Dullness or stupor occasionally occurs and has been observed by us.

Among complications, *hysteria* and *chorea*, and even *epilepsy* are included.

Diagnosis.—When the case is carefully examined, the above symptoms can be found in sufficient degree to make a diagnosis, and a lack of diagnosis is usually due to faulty observation. Rapid heart, with either an enlarged thyroid which pulsates and over which a murmur can be heard or with muscular tremor, or with exophthalmos, is quite sufficient evidence upon which to base a diagnosis. It is true that no one of these symptoms is pathognomonic of Gravess disease, but a combination of two or more means practically a positive diagnosis.

Prognosis.—Exophthalmic goiter is frequently fatal, the patient dying of acute toxic symptoms. At times the condition remains permanent, with gradual change in the heart muscle, and in the gland itself, but in the course of time some cases improve greatly, and some get well. However, many cases as the result of overstrain, mental or physical, develop an acute thyroid intoxication and die of exhaustion. Unquestionably certain mild cases recover without any treatment.

In some rare instances a rapidly fatal course ensues, death taking place in a few days after the onset. The majority of cases run a chronic course, the symptoms persisting more or less for years. When death occurs, it is from failure of the heart or of thymic intoxication. It is generally preceded by an aggravation of all the symptoms. It may be sudden, as by syncope. Acute cases are reported, following one of the cited causes, in which the symptoms lasted a few days, and then disappeared completely.

Treatment. *Medical Treatment.*—Rest and protection from excitement are essential conditions to successful treatment. In cases of any severity rest, physical and mental, should be absolute. The patient should be in bed and protected from any disturbing friends. Milder cases may be about but under strict orders as to amount of work, rest and kind of food. The modern method of having an abundance of fresh air and sunlight in the room should be carefully attended to. The diet should be varied but easily assimilable—much such a diet as advised in typhoid fever. Ice bags to the heart and region of the thyroid are valuable. After this, the treatment has been mainly directed to the symptoms. The remedies heretofore used were mainly the bromids and digitalis: the bromids as nervous sedatives.

Drugs.—Among numerous remedies is one of the bromids for its sedative effect, ergot for its power of contracting the caliber of blood-vessels. By German writers, galvanism of the sympathetic is claimed to be of service. Theoretically, it should be. A constant current of from five to eight cells is used; the negative pole is placed on the fifth cervical vertebra, the positive pole along the sternum. Thyroid extract is contraindicated; it adds more of the active principle of the gland to a body already suffering from over-activity of the gland.

Belladonna is sometimes useful. In our hands digitalis has not been of value except when there is actual cardiac dilatation.

Opium in small doses has been recommended. This drug does quiet the individual, but its use should be restricted to cases which are in a severe condition. In such a condition as this where the case is likely to be chronic there is too great danger of the opium habit to allow the use of the drug. X-ray is recommended by the Mayos.

Surgical Treatment.—The results of *operative treatment* have been most satisfactory, especially at the hands of such surgeons as the Kochers, of Zurich, and the Mayos, of Rochester, Minn., U. S. A.

Charles Mayo advises a graduated operation in certain cases, and states that this method of selecting the time, the preparation, etc., has reduced the mortality to less than 2 per cent. In certain cases the toxic condition is so great that no operation can be undertaken until the patient is restored to a more normal condition.

As to the question of treatment of any given case. The proper steps seem to be the following:

Given a case of hyperthyroidism. If it is recent and of moderate severity, it should be subjected to medical treatment, of which rest is the essential element. If the case responds, no further steps need be taken unless it relapses. If it relapses, a surgeon should at once be consulted.

If the case is severe, it also must be put at once upon rest treatment, and everything done to reduce the toxic symptoms. After it is recovered, an operation should be undertaken by a competent surgeon. The mortality of surgical treatment in skilled hands is amazingly low. In 1913 the Mayos report 275 cases of removal of the gland with 1 death; 4 double ligations with no deaths; 363 single ligations with 5 deaths. These figures are probably lower than those of the country at large.

No condition needs better judgment as to the time of operation; operation in the midst of severe symptoms is followed by a high mortality. It should be done only by those skilled in this line of work.

The *serum treatment* for exophthalmic goiter proposed by Rogers and Beebe,[1] though it has passed the experimental stage, must be regarded as still on trial. For this they offer a specific antiserum which is made by injecting rabbits or sheep with the nucleo-proteid and thyro-globulin obtained from the human thyroid gland. The statistics are not as good as those of the operative treatment.

A second form of serum treatment is feeding with the milk of dethyroidized goats introduced by Lanz. It is obtainable in the shape of a substance called rodogen.

[1] Mutter lecture for 1907 before the College of Physicians, Philadelphia.

The X-ray has proven useful in a few cases. Faradism has failed of its purpose.

MYXEDEMA.

SYNONYMS.—*Cachexie pachydermique* (Charcot); *Cachexia thyroidea vel strumipriva vel thyreopriva* (Kocher); *Athyrea; A Cretinoid State Supervening in Adult Life in Women* (Gull); *Cretinism.*

Definition.—A myxomatous infiltration of the subcutaneous connective tissue of the body, characterized also by dryness of the skin, subnormal temperature, mental failure or lack of development and atrophy of the thyroid gland.

Etiology.—All forms of myxedema are the result of loss of function of the thyroid gland due to disease or removal of the gland.

Morbid Anatomy.—The morbid changes in the myxedema after death are those described as characteristic in life, but autopsy has disclosed the thyroid absent in nine out of ten cases of cretinism examined, confirming the theory of its origin. Enlargement of the *hypophysis cerebri* was found in six cases of cretinism by different observers, and Horsley says that the convolutions of the brain are ill-defined, and the blood-vessels small, even in proportion to the rudimentary condition of the nervous system.

Symptoms.—Three groups of cases are recognizable:

1. Pure myxedema.
2. Myxedema associated with endemic or sporadic cretinism.
3. Operative myxedema or *cachexia strumipriva.*

1. *Pure Myxedema.*—This is much more frequent in women than in men—at least as six to one—and occurs usually between the ages of 30 and 50, but is not confined to these ages, being found in those who are younger and older. Heredity is a recognized factor, acting usually through the mother. Several members of a family may be affected. The poor suffer most. It is said to have no relation to the catamenia, but has followed frequent pregnancies, injuries, severe hemorrhage, and mental disturbance. Most essential is some change in the thyroid gland. Formerly thought to be rare in this country, cases have multiplied since attention has been called to it.

The face is the chief seat of the myxedematous change, but the extremities, the trunk, the tongue, and even the internal organs may be involved. The *face is uniformly swollen, broadened, and flattened*, the *nose is broad*, the *mouth large*, all lines are obliterated, and expression is gone. The *skin* of the neck above and below the clavicle is thrown into folds of fatty and myxomatous tissue. It is yellow, translucent or waxy, dry and scaly. The cheeks and sometimes the nose are flushed. True edema may be associated, and there are rarely albuminuria and glycosuria. The *hands*

[1] The student is referred to five noteworthy papers on the subject of "Myxedema and Cretinism" in vol. viii., 1893. "Transactions of the Association of American Physicians," by Francis P. Kinnicutt, James J. Putman, M. Allen Starr, W. Gilman Thompson, and William Osler; to the "Atlas of Clinical Medicine," by Byrom Bramwell, for admirable illustrations; and to the exhaustive article on "Myxedema" by George R. Murray, of Newcastle, England, in the "Twentieth Century Practice of Medicine," vol. iv., 1895. Also to an address by Victor Horsley on the "Physiology and Pathology of the Thyroid Gland, published in the "British Medical Journal," December 5, 1896.

[1] Von Eiselberg's experiments were made chiefly on cats.

lose their natural shape, and were described by Gull as "spade-like"; the *feet* are also misshapen; the *gait* is slow and labored. The *mind is feeble*, slow in its action, memory is poor, while irritability and suspicion are added qualities, and sometimes there are delusions and hallucinations, ultimately often dementia. The organic functions are fairly well performed. *Atrophy of the optic nerve* is a rare but possible symptom, also *synovitis* from trifling causes. *Subnormal temperature* is characteristic, though in early stages the temperature may be normal or slightly above. In winter the patient always feels cold and hugs the stove. The course of the disease is slow, and the patient usually dies of some intercurrent affection unless proper treatment is instituted. Mild cases are not rare and are frequently overlooked. A paper by Pittfield of Philadelphia well describes the condition.

2. *Myxedema Associated with Cretinism, Congenital or Acquired.*—(Cretinoid idiocy: Idiotie avec cachexie pachydermique.) Cretinism is a form of idiocy associated with absence of the thyroid or with a functionless thyroid. It is myxedema in childhood. There is almost complete *arrest of mental and bodily development.* The cretin is a dwarf. In the *congenital* form, there is congenital absence of the thyroid, and the child is further characterized by its thick neck, short arms and legs, and prominent belly. The face is large, the lips are thick, and the tongue is large and often protruding, the hair is thin and stiff. All the bones of the skeleton are short and broad, the epiphyses swollen, but not ossified. The skull is short and broad, and the basosphenoid junction early ossified. The cretin resembles the rickety child, and may be confounded with it, but the bone changes are entirely different.

Acquired cretinism may start before birth and be barely appreciable at birth. More frequently the infant appears normal at birth, and the changes make their appearance between the *second* and fifth years. The arrest of development continues, or, rather, there is very slow development, so that at adult age the man or woman does not exceed in stature a child of from five to seven years. The myxedemic symptoms are similar to those described in pure myxedema.

From the fact, however, that the disease may start to develop later than infancy, there results a series of types intermediate between those represented by congenital and adult cretinism. This arrest of mental and physical development is, of course, greater the earlier the disease begins to develop; whence two cretins of the same age will differ materially if one has commenced to develop at or before birth, and the other not until seven or eight years of age.

True congenital cretinism—that is, cretinism which is *evident* at birth—is very rare. Some cases of anchodroplasia may have been diagnosed cretinism. In most cases, the child does not long survive its birth. In another form described by Horsley the disease s supposed to begin shortly before birth, but develops slowly, so that at birth it had not attained the degree incompatible with life, and the child can live. In this there is usually a goiter at birth.

Cretinism may be endemic, as in some parts of the continent of Europe; or sporadic, as in England, and America as well. The sporadic cases are, as a rule, without goiter, the thyroid glands being either undeveloped or

atrophied, while one-third also of the endemic cretins are without goiter. In either event, the gland is functionally dead, even though it may appear natural in size, the original true gland tissue having been replaced by an indifferent element. Endemic cretinism occurs in localities where goiter is also endemic—in the shut-up valleys of mountainous districts of Europe and Asia, to which it is confined. At one time—in 1847—a number of cases—some 24 out of a population of 350—prevailed in Cheselborough, Somerset, England, but the disease has died out. The endemic form is commonly ascribed to the use of certain drinking water, but no responsible constituents have been isolated. The child, being normal at birth, remains so until the change begins in the thyroid, when it becomes less lively, development is arrested, and the conditions described on page 676 slowly develop. The cretin may reach the age of 30 or 40 years, but ceases to change after the 20th year, whether the case be sporadic or endemic.

3. *Operative Myxedema or Cachexia Strumipriva.*—By this is meant a condition of myxedema the result of removal of the thyroid. It is more likely to follow total than partial removal of the thyroid, but does not follow every case, having been observed in 69 out of 408 cases. Cases of operative myxedema are very rare in this country.

Diagnosis.—This is easy. The *edema of Bright's disease or heart disease* may be confounded, especially as albuminuria and casts are sometimes present in myxedema; but the peculiar flat face, the absence of pitting on pressure, and of the signs of heart disease are distinctive features of myxedema, and will not be overlooked by those familiar with the condition. Certain forms of idiocy, however, may easily be mistaken, as may achondroplasia. Achondroplasia is, however, characterized by a bright intellect.

Prognosis.—This was regarded as unfavorable until the use of thyroid extract was suggested by George R. Murray, of Newcastle, England, in 1871, based upon the satisfactory effect obtained by Bettencourt and Verrano, in 1890, in ingrafting sheep's thyroid in human subjects having myxedema, the idea being in this manner to substitute the juice or secretion of the gland. Every expectation was realized. In 1892, Howitz, of Copenhagen, and soon after, E. L. Fox and H. Mackenzie, in England, substituted for the hypodermic use the administration of the gland itself or some preparation of it by the mouth. At the present day, the effects of the administration of thyroid preparations in myxedema are among the marvelous results of medicine.

Treatment.—The treatment at the present day is, therefore, solely by preparations of the thyroid gland. The gland is best administered in the shape of tablets of the dessicated gland. A small dose must be first given, one grain, three times a day and its effect carefully watched. If no effect follows, the dose should be doubled, and further increased if necessary. As improvement takes place, smaller doses should be given at longer intervals, until finally one grain of the gland is given once a day. During the first week the patient should be watche,d with a view to guarding against overaction of the remedy—great emaciation, diarrhea, vomiting and tremor, in a word hyperthyroidism may occur from overdoses.

The myxedema being removed, it is necessary, of course, to continue

the treatment in this second stage by such doses as will maintain the cure, for it is to be understood that, as the thyroid is still functionless, the omission of the treatment is followed, sooner or later, by its return. The quantity required varies in different cases, but it is found to range, the more precise dose being determined by trial. A single daily dose is preferred by Murray to a smaller dose more frequently repeated. A fall of temperature below normal, a slight return of swelling or of other symptoms, indicate that too small a dose is being given, while acceleration of the pulse indicates that the dose should be reduced. In a climate not subject to great variations the same dose may be given the year round. In hot weather a smaller dose suffices than in cold, and a dose that has been found sufficient during the summer may not be enough in the winter.

The results of treatment in cretinism are as marvelous as in myxedema. They include not only the removal of the hideous deformity and the restoration of intellect, but also an increase in height. A case under observation was corrected from a drivelling idiot at 3 years to a bright boy at 10 years of age. The earlier treatment is commenced, the more prompt and marked is its effect. It is useless if the child is many years old before the treatment is begun. The treatment must be continued through life.

DISEASES OF THE PARATHYROID GLANDS.

The discovery of the parathyroid glands has opened a new field of physiological and pathological study which has not yet been completely explored. There are two pairs of these little glands on each side of the lateral lobes of the thyroid, ovoid in shape and 6 to 8 mm. in length. They secrete an important internal secretion supplementing that of the thyroid gland and controlling calcium metabolism. The latter is established by their extirpation, which is followed by severe muscular spasm, paralysis, dyspnea and death, counteracted sometimes by intravenous injection of parathyroid extract or by the transplantation of parathyroid structures or even their ingestion by mouth. Their secretions may be harmonic to secretions of other duetless glands. Although various spasmodic derangements are ascribed to deranged functions of these glands, tetany is the only one whose symptoms are sufficiently traceable to justify its classification under the heading of disorders of the parathyroids.

TETANY.

SYNONYMS.—*Tetanilla; Intermittent Tetanus; Hypoparathyreosis; Status Parathyreoprivus; Contracture des nourrices.*

Definition.—A condition of deranged metabolism exhibiting continuous or intermittent tonic spasm of the extremities, usually symmetrical, but occasionally confined to one limb, rarely even becoming general. It is due to disease of the parathyroid gland or insufficiency.

Etiology.—Tetany must now be ascribed to deranged function of the parathyroid glands. It occurs in children and in adults.

Tetany in Adults.—Among possible predisposing causes may be men-

tioned digestive derangement, dilatation of the stomach, hyperchlorhydria, and diarrhea; rheumatism, whence this form is sometimes called rheumatic tetany; open wounds; laceration. Pregnancy, acute fevers, and diphtheria are also alleged causes. What relations these conditions just metioned have with parathyroid disease is unknown. It has been suggested that gastric tetany and that of the infectious fevers is connected with disturbance of calcium metabolism. Its presence in nursing women, as pointed out by Trousseau, may have to do with the drain of calcium in the formation of milk which is rich in calcium, and in pregnancy with the formation of the bones of the child. The analyses of MacCallum tend to confirm this explanation.

Postoperative Tetany.—The effect of removal of the parathyroid glandules in producing tetany is well known. A few days after the parathyroids have been removed the patient is seized with severe convulsions of spasmodic type—coma and death.

Infantile tetany occurs in rickets, in digestive diseases, and is characterized by the usual spasms—by laryngismus stridulus.

Symptoms.—The characteristic *spasm* is usually limited to the hands and feet, arms and legs. In the hands the thumbs are flexed into the palms, the fingers firmly bent at the metacarpophalangeal articulation, but straight elsewhere. The fingers are adducted, the ring and middle fingers sometimes overlapping. The wrists are flexed, the elbows bent, and the arms folded over the chest. The hand is described as the obstetrical hand, from the position caused by the cramp. In the lower limbs the knees and hips are stiff and extended, the feet extended, and the toes adducted. Sometimes there is dorsal flexion of the foot and flexion at the knee. Contractions may last from a few hours to several days. The term *continuous* may be applied to those cases in which the contractions have lasted uninterruptedly for over two days, and *intermittent* when they do not last longer than two days without permanent or temporary disappearance. Following this standard, Griffith found 38 cases intermittent and 25 continuous. The spasm is always associated with *tenderness* or *pain*, the latter being often extreme. At other times these symptoms are present only in the beginning of the attack or when the members are handled. Rarely the muscles of the back, neck, and face are involved; and there may be *trismus*, the angles of the mouth being drawn out.

Associated symptoms are *stridulous respiration*, laryngismus stridulus, regarded by some as an essential part of the disease.

Further interesting phenomena, especially studied and called cardinal symptoms, are *contraction caused by tapping over nerve trunk*, as, for example, the facial nerve, known as Chvostek's symptom. Another is Trousseau's symptom—the production of *spasm by pressure* upon *a large artery or nerve*, especially in the arm; and still another is Erb's symptom—*increased electrical excitability*. *Inability to urinate* may be present, and anesthesia has been recorded among symptoms. There may be *slight* elevation of *temperature* and *frequent* pulse.

Diagnosis.—The rarity of this disease sometimes causes it to be overlooked, while differences of view as to what constitute its essential symptoms also cause a different diagnosis. Thus, some would exclude the

carpopedal spasm of children; while Gowers, Dana, and Griffith include these cases under tetany. Many cases of *mild spasm succeeding gastro-intestinal irritation* and the like would be regarded by some as tetany and by others not. It possesses nothing in common with *tetanus*, whose name it so closely resembles, but whose symptoms are totally different.

Prognosis.—This varies with the cause. Operative cases are mostly fatal if not treated by parathyroids. Cases due to gastric dilatation are fatal unless the gastric condition be operated upon. The fatal cases are those associated with dilated stomach, gastric carcinoma, and thyroidectomy. The disease has a marked tendency to return, and is most common in late winter and early spring.

Treatment.—In postoperative tetany the immediate grafting of parathyroids and the feeding of parathyroid tissue should be begun at once. The *cause* of the condition should be sought and, if possible, eliminated. After this, *remedies calculated to diminish nervous excitability* should be administered; also wholesome hygienic measures availed of, including *massage*, *passive motion*, and *electricity*. *Warm baths* are especially recommended. The cases attended with severe pain may require the hypodermic use of morphin, and delayed response to the latter may even demand chloroform inhalation.

The rational treatment of tetany includes any measure which will restore the function of the parathyroids. Parathyroid may be administered by the mouth, but the effect is found to continue only so long as the adminstration is continued. The hypodermic injection of a serum from the same source promises more permanent results, but most promising of all is the transplantation of the parathyroid itself, as suggested by Pays and repeated by Halsted. Conforming to what has been said at the outset of the consideration of this subject, calcium should be effective. Calcium lactate is given in doses of 15 grains (one gram) every three or four hours. Lavage with saline solutions. The bromids may be serviceable and in extreme cases chloroform anesthesia may be necessary. When the condition is due to gastric dilatation the cases should at once be operated upon by gastro-jejunostomy.

NEOPLASMS OF THE THYROID.

The thyroid is subject to a variety of morbid growths, among which may be mentioned:

1. *Adenoma*, which occurs as an encapsulated growth, varying considerably in size. There may be nodules in both lobes. Metastases of growths resembling thyroid tissue are reported to have been found in the lungs and bones of the body.

2. *Primary medullary cancer*, as a rare growth with a tendency to invade the trachea and esophagus, developed from the epithelial cells of the follicles. *Secondary cancer* has also been reported.

3. *Tuberculosis*, always supposed to be a possible but rare disease, has been found by Chiari in seven out of 100 postmortems on persons who had had tuberculosis. Bruns refers to six cases of tuberculous goiter.

4. *Syphilis*, including gummas. 5. *Hydatid disease*. 6. *Actinomycosis*.

5. *Abscess* of the thyroid is an occasional event.

The *treatment* of these abnormalities is surgical, except in the case of tuberculosis and syphilis, which demand the usual antituberculous and antisyphilitic remedies.

DISEASES OF THE SUPRARENAL CAPSULES.

ADDISON'S DISEASE.

Definition.—A term applied to any degenerative disease of the suprarenal capsules, especially tuberculosis, atrophy and carcinoma attended with pigmentation of the skin. It is associated also with asthenia of the muscular and vascular system.

Etiology.—Its etiology is obscure, being that of the morbid states constituting its morbid anatomy. Blows on the abdomen or back have preceded it, so has Pott's disease, and numerous other events which may or may not have been causal. It occurs 119 times in men to 64 times in women.

Morbid Anatomy.—This includes (1) tuberculosis with fibrocaseous and calcareous degeneration—the most frequent lesion; (2) cystic degeneration; (3) fatty degeneration; (4) simple atrophy; (5) chronic interstitial inflammation which may lead to atrophy; (6) malignant disease, including carcinoma and sarcoma; (7) hemorrhagic extravasations; (8) embolism.

Pathology.—Its pathology is as obscure as its etiology. Experiment helps us only to this extent, that removal of the suprarenals in animals causes death by progressive weakness and toxemia. The cortex of the suprarenal capsule is composed of epithelial elements, the medulla of a mesh work containing nerve cells, nonmedullated nerve fibers and certain polymorphous cells known as "chromaffin" cells—cells which take a brownish pigmentation in solution of chromic acid. These cells are also found outside of the adrenals in the ganglia of the abdominal sympathetic, in the carotid glands, in the coccygeal gland, the parovarium, the epididymus, and along the course of the aorta. Collectively these elements are known as the "chromaffin system."

The special function of the medulla in which the "chromaffin" system shares is to distill an internal secretion, epinephrin, whose purpose is to stimulate the nervous mechanism regulating blood pressure and pigmentation—to play, in a word, the rôle of hormone. And thus is explained the muscular and vascular asthenia, and excessive pigmentation incident to disease of the suprarenals, and the arterial scleroses which follows upon the intravenous injection of epinephrin. The glycosuria resulting from such injections may be due to an inhibiting effect on the pancreatic function in glucose metabolism.

Less is known of the function of the cortex. Some influence on the sexual function is suggested pro and con, according as the activity of the secretion is increased or diminished. In pregnant rabbits the cortex became twice as thick as in the nonpregnant state. It has been suggested, too, that the cortex produces a product which influences nitrogenous metabolism favorably, preventing auto-intoxication or favoring it by absence.

Symptoms.—Some of these morbid states appear to be totally without

symptoms, the conditions having first come to light at autopsy. Other symptoms may be produced by any of them, and there are none distinctive for any one state. They include:

Pigmentation or *bronzing* of the skin. This was first noted by Addison as accompanying lesions of the suprarenal capsules, and such association thus constituted. Addison's disease since the publication of his paper in 1885. It is a disease of adults, being rare under 35 years. The lesion of the suprarenal most frequently thus associated is the fibrocaseous tuberculous one. But, as stated, the pigmentation may accompany any one of the above-named lesions, or it may be absent. As to the *color* itself, usually the first symptom to attract attention, it ranges from light yellow to deep brown, and even almost black. It is deeper on the more exposed parts of the body, where the normal pigmentation is greater, and therefore is commonly first seen on the face and hands. In rare instances only is it general. It is associated at times with unpigmented patches—leukoderma. It is noticeable also at times on the mucous membrane of the mouth, conjunctiva, and vagina, and very rarely even upon serous membranes in patches.

As stated, there are no other symptoms which are distinctive of any one of the lesions of the suprarenal capsule described, but among those which are more or less constantly present are *rapid cardiac action, anemia extreme debility* and *general languor, irritability of the stomach,* and quite often *diarrhea.* The irritability of the stomach is manifested by anorexia, nausea, and vomiting, and may be a very early symptom. The heart's action is feeble, the pulse correspondingly small and rapid, the blood pressure is low. A case in a man the pulse was 130 and feeble. There is also often a tendency to fainting. There is *dyspnea.* It will be remembered that this symptom attends the asthenia and death succeeding removal of the adrenals in animals. At other times there is *headache. Mental hebetude* goes *pari passu* with bodily weakness, while the other symptoms commonly associated with the latter condition are also present—namely, dizziness and ringing in the ears. Ultimately, the *asthenia* becomes so profound that the patient cannot rise, but keeps his bed, growing weaker and weaker, until he dies of sheer exhaustion. Sometimes there are *convulsions,* possibly due to brain anemia.

The *urine* is usually normal, although occasionally there is polyuria, and sometimes the urinary pigments have been found increased.

Diagnosis.—Pigmentation alone, at least unless it be very decided and general, is never sufficient to justify a diagnosis of suprarenal disease, since other abdominal affections are known to produce a similar condition. Among these are tuberculosis of the peritoneum, cancer and lymphoma; pregnancy, uterine and even hepatic disease. In the hardening of the liver sometimes associated with *diabetes,* pigmentation has been noticed. All these facts go to show that the nervous system must have some powerful influence, supporting the second theory. The same testimony is afforded by the pigmentation which attends *exophthalmic goiter. Protracted filthiness* and vagabondism also produce discoloration of the body which is not distinguishable *per se* from that of Addison's disease. Deep general pigmentation has been found associated with *melanotic cancer.* Finally, pigmentation is sometimes the result of the prolonged administration of *arsenic.* It is

well, therefore, to seek carefully for signs and symptoms other than pigmentation before a diagnosis is made. In the case of our own referred to, there were pulmonary tuberculosis and tuberculous disease of the spine, with pigmentation and asthenia, on which was based the diagnosis of Addison's disease, confirmed by autopsy. Certain cases of hypopituitarism reported by Cushing, closely resemble Addison's disease.

Prognosis.—In a well-determined case of Addison's disease, as might be inferred from the nature of the causes, recovery is impossible, though the course of the disease is commonly prolonged and improvement may take place. In a few cases only is the course rapid. From 18 months to several years usually cover the duration.

Treatment.—This is principally symptomatic. We aim to restore the condition of the blood, and, of course, above all, iron is indicated. It may be associated with that other tonic so constantly used with iron especially—arsenic. An excellent preparation of arsenic is the solution of the chlorid, which is as good as Fowler's solution and mixes well with the chlorid of iron. The doses are the same as those of Fowler's solution—from 3 to 5 minims (0.18 to 3 c.c.); or the iron and arsenic may be given in pill form as the carbonate of iron and arsenious acid, and to this strychnin may be conveniently added. If very asthenic, the patient should be kept in bed and fed with nutritious, easily assimilable food, of which peptonized milk and broths, beef-juice, cod-liver oil, and glycerin are the type. The diarrhea should be treated as other diarrheas, with bismuth and other remedies. For the nausea the usual gastric sedatives, including ice, carbonic acid water, champagne, milk and lime-water in small doses, koumiss, whey, and the like are suitable, massage may be helpful.

With the knowledge which has grown out of the treatment of myxedema with thyroid extract, no treatment of the combination of symptoms known as Addison's disease would be complete without the administration of some similar preparation of the adrenal gland. There have been prepared an extract in the shape of the tincture, a powder and a glycerin extract; and the glands are eaten fresh or dried. The equivalent of two a day is recommended. Of the powder, 3 to 5 grains (0.2 to 0.3 gm.) are given three or four times a day. At the present day, solution of adrenalin 1–1000 would be employed in doses of from 5 to 15 minims or more. In an analysis by E. W. Adams of 97 cases, treated by suprarenal extract, there was seeming permanent relief in 16. In two cases treated at the Johns Hopkins Hospital, one died of an acute infection after all severe symptoms had disappeared, and at the autopsy the suprarenal bodies were found sclerotic. The remedy is still *sub judice*, and as the disease is rare some time must elapse before its value can be determined.

OTHER AFFECTIONS OF THE SUPRARENAL GLANDS.

The suprarenals are subject to hyperplasia and hypoplasia. The former occurs in association with chronic nephritis and in arterio-sclerosis. No explanation is offered of the former, but the arteritis may be due to an excess of the internal secretions poured into the blood from excess of function. Precocious development of sexual organs has been found with

hypertrophy and tumor. A case has just been reported by Henry Jump of Philadelphia. Hypernephromata are frequent tumor formations. Hypoplasia, absence of the adrenals or their medulla is found associated with anomalies of the brain and low blood pressure. The latter would be a natural consequence of deficient internal secretion.

DISEASES OF THE SPLEEN.

Most of the morbid states of the spleen which possess clinical interest are considered in connection with diseases of the blood and with malaria.

Splenitis.—Splenitis occurs rarely as the result of extension of inflammation from a neighboring organ, such as the stomach, perinephric tissue, the diaphragm and lungs, or as the consequence of injury.

The symptoms are *tenderness* and *enlargement* in connection with the inflammatory conditions of adjacent organs referred to, and it is upon the association of such symptoms with those in the spleen itself that the diagnosis depends.

Perisplenitis.—This may occur as the result of the same causes as produce splenitis, and may be recognized by the presence of palpable friction fremitus. It may be suppurative in association with abscess of the spleen giving rise to one form of subphrenic abscess.

Abscess of the Spleen.—Abscess of the spleen occurs along with pyemic processes elsewhere, in the presence of the usual causes of pyemia. It is characterized by tenderness and enlargement. A remarkable case associated with suppurative splenitis occurred in the wards at the Pennsylvania Hospital, after metritis following criminal abortion. It was also associated with luxuriant mitral valvulitis. Such abscess may break into the stomach, bowel, or lungs, as well as into the peritoneal cavity.

Rupture of the Spleen.—This arises from severe injury, also from extreme and sudden acute hyperemia, due to malignant malaria, and from rapidly growing splenic tumors. The symptoms are sudden pain in the region of the spleen, collapse, pallor, and death, associated with the causes named. Conner has reported a case in typhoid fever.

The Amyloid Spleen.—This appears as a hard, smooth, and enlarged organ, associated with amyloid disease of other organs, such as the liver and kidneys, especially when there has been long-continued suppuration, as in hip disease, osteomyelitis, tuberculous consumption, or syphilis.

Atrophy of the Spleen.—On the other hand, the spleen may be reduced in size by fibroid overgrowth and contraction due to syphilis.

Hemorrhagic Infarct of the Spleen.—Infectious hemorrhagic infarct results in abscess of the spleen. The noninfectious is the result of embolism by a noninfectious embolus such as arises from the cardiac valves in acute or chronic endocarditis, from clots in the cavities of the left ven-

tricle, or from clots in aneurysm in the large arteries. After the kidney, the spleen is the most frequent seat of such lodgment.

Symptoms.—The infarction is sometimes ushered in by chills, vomiting, and painful enlargements, the true nature of which can only be inferred when the causes named are present or the symptoms of embolism elsewhere occur simultaneously.

Neoplysms of the Spleen.—These are represented most frequently by gummata, which are almost never recognized before death. *Carcinoma*, *sarcoma*, and *tuberculosis* occur, but are not recognizable by special characters. A nodular and uneven spleen may be regarded as due to cancer when associated with cancer elsewhere, sarcomatous when there is general sarcoma, tuberculous if there is tuberculosis elsewhere, and syphilitic if associated with the hostory of syphilis, especially the congenital form.

Echinococcus of the Spleen.—The spleen may present a fluctuating tumor the nature of which can only be determined by the certain knowledge that a tumor of the same kind exists elsewhere, or by the recognition of hooklets in the aspirated fluid. Should the fluctuating tumor be associated with chills and fever, it is more likely to be abscess, which, it is to be remembered, may also begin as echinococcus disease which later takes on suppuration.

Wandering Spleen.—This is a term applied to a condition of the spleen analogous to the movable kidney and liver. It is the direct result of an elongation of the gastrosplenic ligament and splenic artery and vein. Under these circumstances the usual splenic dullness in the midaxillary line, between the ninth and eleventh ribs has disappeared, and the spleen can usually be felt elsewhere in the abdominal cavity, usually, however, on the side below its normal site, whence it may be pushed into the natural situation to leave it immediately as the upright position is assumed. Rarely, it is found in more distant situations, even in the pelvis. At times it may form attachments by inflammatory adhesion in the new situations, making its restoration difficult or impossible.

Symptoms.—The symptoms are not unchanging. The most constant is a *dragging sensation*, while there may also be the *effects of pressure*, which vary with the situation. There may be pressure on the ureter or bladder, causing difficulty in micturition; upon the bowel, causing partial obstruction or pain from compression of sensitive parts. The same train of nervous symptoms which attends floating kidney may also be present.

Diagnosis.—Some difficulty of diagnosis may result in consequence of such vagueness of symptoms. There may be a question between the existence of wandering spleen and *fecal tumor*. With the former, the normal splenic dullness is wanting, though the well-known fact that the dullness is sometimes very small in health may give rise to error. A freely movable *cancer of the pylorus*, a tumor so movable that it may be felt in the left hypochondrium, may occasion similar difficulty, which must be settled in the same way. And so with other abdominal tumors of movable nature—the normal splenic dullness remains. The question as to whether a *movable* organ is the spleen or *kidney* is not likely to be a knotty one, even if the mov-

able kidney be the left, if the same guide be availed of. The difference in outline of the two organs may be recognized in persons with thin abdominal walls, and. in rare instances, by the splenic notch. The possible coexistence of a movable spleen and a movable kidney is to be remembered.

Treatment.—The treatment must consist of mechanical measures to keep the spleen in place. Surgery must frequently be employed. They are variously successful.

DISEASES OF THE PITUITARY BODY.

The pituitary body or *hypophosis cerebri* is a ductless gland seated in the cella turcica. According to Cushing "it arises from an epithelial pouch which buds off from the roof of the buccopharyngeal cavity. This pouch meets and partly envelops a corresponding prolongation (infundibular) from the adjoining base of the anterior cerebral vesicle, the tip of which becomes thickened into the infundibular body (neurohypophysis, or pars nervosa). By the subsequent formation of the sphenoidal bone the lumen of practically all of Rathke's diverticulum except its tip becomes obliterated. This unobliterated tip of the pouch comes to enfold the infundibular body as a ball is held in a catcher's mitten," and thus the pituitary body is formed. It is composed of two lobes, a larger anterior and a smaller posterior, the two lobes being united by an isthmus. The larger anterior lobe is made up of large, granular, epithelial cells arranged in columns and surrounded by venous spaces into which the secretion is discharged. The smaller posterior lobe is mainly nervous in composition (pars nervosa) and invested with epithelial cells. The secretion of this lobe is thought to pass into the cerebrospinal fluid. The isthmus also contains an epithelium which secretes a colloid substance like that of the thyroid. It is essential to life and is concerned with calcium metabolism. It controls the growth of the skeletal tissues, bone and cartilage. It has to do with the development of fat and holds some relationship with other ductless glands, especially the thyroid, adrenals and sexual glands. It is materially influenced during pregnancy and the changes induced by pregnancy are said to be permanent so that from them it can be averred the subject has been pregnant. Like the adrenal secretion it inhibits the pancreatic secretion and may, therefore, have some causal relation to glycosuria. It increases blood pressure and would seem to cooperate with the adrenals but antagonize the thyroid.

Cushing in his book, "The Pituitary Body and Its Disorders" 1913, reviews all the literature and has the most recent exposition of this most interesting condition. He classified all the disorders of the Pituitary body under the name Dyspituitarism. This author divides cases based upon experimental evidence and the observation of fifty odd clinical cases as follows:

GROUP I.—Cases of dyspituitarism in which not only the signs indicating distortion of neighboring structures but also the symptoms betraying the effects of altered glandular activity are outspoken.

GROUP II.—Cases in which the neighborhood manifestations are pronounced but the glandular symptoms are absent or inconspicuous.

GROUP III.—Cases in which neighborhood manifestations are absent or inconspicuous though glandular symptoms are pronounced and unmistakable.

GROUP IV.—Cases in which obvious distant cerebral lesions are accompanied by symptomatic indications of secondary pituitary involvement.

GROUP V.—Cases with a polyglandular syndrome in which the functional disturbances on the part of the hypophysis are merely one, and not a predominant feature of a general involvement of the ductless glands.

One of the marked results of Cushing's studies is the establishment of the fact that cases which are affected by the hyperactivity of the glands show first the result of this overactivity in either gigantism or acromegaly, and then later develop symptoms indicative of hypoactivity of the gland, these becoming mixed cases.

Inheritance is believed to have a certain effect which makes the hypophysis susceptible to alterations in its functional activity.

Traumatism is also believed to have a marked effect upon the development of certain cases.

Adolesence, *pregnancy*, and the *climacteric* are also believed to have effect in causing changes in the gland, it is certain that in pregnancy there is a functional hypertrophy of the gland.

He divides the symptomatology as follows: "(1) Neighborhood symptoms, (2) general pressure manifestations, (3) the secretory or glandular symptoms proper, and (4) the polyglandular manifestations."

Headaches are common, severe and persistent.

Deformity of the sellatcursica can be made out in certain cases by the X-ray. Atrophy of the optic nerves and disturbance of the fields of vision are among the common results of certain tumors.

Epistaxsis and marked discharge of mucus from the pharynx often supposed to be due to sinusitis.

Acromelagy and *gigantism* are the result of hyperplasia in the gland, the former after adolesence the latter when young.

Launois and Roy say "gigantism is acromegaly in individuals whose epiphyseal cartilages are not ossified, whatever may be their age." In other words, both acromegaly and gigantism are the same condition brought about by overactivity of the gland occurring at different ages. In Cushing's words, "The disease, in short, is the expression of a functional instability of the pars anterior, doubtless brought about by some underlying biochemical disturbance which leads to the elaboration of a perverted or exaggerated secretion containing a hormone that accelerates skeletal growth (the long bones if epiphyseal union is incomplete, of the acral parts if epiphyseal ossification has taken place). Since the functional disturbance is probably a fluctuating one, with periods of increase and remission, epiphyseal ossification may occur during a period of quiesence in the disorder. A subsequent recrudesence with resumption of the perverted functional activity will then serve to superimpose acromegalic manifestations on primary gigantism. Acromegaly, in other words, cannot precede gigantism, but always occurs, as gigantism which has become acromegalized."

Hypopituitarism causes *skeletal undergrowth* which occurs when gland-

ular insufficiency begins before the full stature is attained. When lack of activity begins before adolesence the skeleton has the feminine type.

Adiposity is common in cases where there is a lack of activity of the gland. He believes, "We have attributed this particular symptom-complex of *adiposity, high sugar tolerance, sub-normal temperature, slowed pulse, asthenia* and *drowsiness* to a secretory deficiency of the posterior lobe; and a further argument in favor of this view is the reverse condition—namely, the emaciation, spontaneous glycosuria with hyperglycæmia, and the slightly elevated temperature—which follows posterior lobe administration."

Polyuria and polydyspia have followed sella decompression. Blood pressure is low in cases of hypopituitarism.

Drowsiness and mental inactivity are also symptoms which occur. Certain symptoms "pigmentation of the skin, asthenia, low blood pressure, and to these may be added hypoglycæmia" often occur in hypopituitism which greatly resemble Addison's disease.

Treatment.—Some cases require medical treatment, others operative.

Surgical methods are used to correct pressure disturbances, to combat functional hyperplasia, to afford relief to neighborhood symptoms.

"The chief service of surgical therapy in hypophyseal maladies is to afford relief to neighborhood symptoms. A lesser service has been shown to be the palliation of the manifestations of increased intracranial tension, just as in the case of tumors originating elsewhere. Surgery may come to render a third service in the partial extirpation of the gland in states of hyperpituitarism and there remains a possible fourth service that may be rendered in states of hypopituitarism through glandular implantations.

Acromegaly and Gigantism.

Historical.—It was first described by Marie, of Paris, in "Revue de Medecine," 1886. It had, however, been previously described under other names, as "hyperostosis of the entire skeleton" by Friedreich, as general hypertrophy, or "makrosomie," by Lombroso, as "giant growth," by Fritsche and Klebs. Since then numerous cases have been reported, and the disease was exhaustively described by Arnold, of Heidelberg, in Ziegler's "Beitrage," in 1891.

Definition.—A disease characterized by enlargement of the bones, especially the bones of the hands, feet and face.

Etiology.—It is a disease of early adult life, usually occurring under 30 and is, perhaps, slightly more frequent in women. Heredity, syphilis, and the specific fevers have preceded the disease, but no necessary relation has been shown. Hyperactivtiy of the pituitary body is the cause.

Morbid Anatomy and Pathology.—This consists in a true hypertrophic enlargement of the bones, except the superior maxillary, which contributes to the enlargement of the face by a dilatation of the antrum, while the lower jaw is simply enlarged. As stated the enlargement is uniform and symmetrical instead of involving only the shaft as in osteitis deformans, or the ends as in arthritis deformans, and is quite independent of rheumatism. Hyperplasia of the pituitary body has been a striking feature in most cases which have come to necropsy, in every one of thirty-four collected by Furni-

val. Marie early sought to make these changes responsible, as disease of the thyroid is for myxedema. Persistence and enlargement of the thymus gland have been found, and atrophy as well as enlargement of the thyroid.

A further study of acromegaly in connection with "giantism," "dwarfism and "cretinism," go to show that it is at least probable that all of these are the result of some deranged function of the pituitary gland. It is well known that giantism may degenerate into acromegaly, while a comparison between the skeletons of a dwarf and a macrocephalic suggests that they are opposite extremes of one and the same process. Of further interest in this connection is the embryonic relation between the pharyngeal tonsil—adenoids in the vault of the pharynx, and the extraordinary influence they have on nutrition—and the pituitary body, which are at one period of development in connection and subsequently separated by ossification at the base of the skull; while not infrequently in early life they remain connected by a fibrous cord running through the body at the sphenoid.

Symptoms.—The most striking features are the enlarged bones, especially those of the hands and feet, the appearance of the former being well characterized as spade-like, while the fingers and nails are broad. The legs and arms, on the other hand, are not elongated early, but late in the disease: and the forearms and legs may increase in circumference; while the ends of long bones, like the femurs, are often prominent. The scapulæ, clavicles, sternum and the ends of the ribs are also sometimes involved. The proper use of the hands is not interfered with. The head and face are enlarged, the spaces between the teeth are increased, while the neck appears short and the inferior maxilla may project beyond the upper, and the lower lip protrude in consequence. The ears are unduly prominent, while the cartilages of the nose, eyelids, and larynx are enlarged and thickened, as is also sometimes the tongue. The spinal column may be involved, and there may be kyphosis. The muscles on the other hand are sometimes atrophied and the genitalia are unusually developed. The skin, though coarse and exhibiting a tendency to perspire, is not thickened as in myxedema.

Among other symptoms are mental dullness, a sense of fatigue, and quite severe pain in the head and extremities, alteration of voice due to changes in the tongue and larynx, and possibly to paresis of the vocal cords; impairment of special senses of taste, smell and hearing; blindness due to optic atrophy; thirst, shortness of breath, asthmatic attacks, palpitation, and even hypertrophy of the heart. In a number of cases bitemporal hemianopsia has been observed and was due to pressure on the chiasm by the enlarged pituitary body. There are menstrual derangement and early cessation of the menses in women. The alterations in the thyroid have been alluded to, and an area of dullness over the manubrium is ascribed by Erb to persistence of the thymus.

Diagnosis.—This is easy. The difference between acromegaly and osteitis deformans has been mentioned. In osteitis deformans, too, as pointed out by Marie, the face is triangular, with the base upward, while in acromegaly it is ovoid, with the large end downward. Acromegaly has been mistaken for congenital progressive hypertrophy or giant growth, but in the latter only one limb is usually involved and the shaft of the bone is affected.

Prognosis.—The duration of the disease is long and usually ultimately

fatal, although it is sometimes arrested. The fatal cases are probably those with tumor of the pituitary body.

Treatment.—None has been found to be of any value. Naturally one thinks of the possible utility of extract of the pituitary gland, though if the condition be the result of excessive pituitary secretion, but little can be expected from such use. In fact such has been the result in the few cases in which it has been tried.

Infantilism.

Infantilism or dwarfism and pseudo-obesity are consequences of depressed pituitary function, but not every case of dwarfism is thus caused. Thus cretanoid infantilism is the result of defective thyroid function and has been described on page 676. That variety of dwarfism known as idiopathic infantilism (so-called Lorain type) as well as the form of pseudo-obesity known as *lipomatosis universalis asexualis* (*dystrophia adiposo-genitalis*) are so regarded and the same may be true of Dercum's disease (adiposis dolorosa) and achondroplasia, but it cannot be confidently asserted.

Then there is cachectic infantilism which is produced by any prolonged diseased state as hook worm disease, syphilis, chronic malaria and congenital heart disease. There is also said to be a toxic infantilism due to the prolonged effect of tobacco and alcohol. Whenever it does occur sexual development ceases and physical development remains at the same stage.

I. Varieties of Infantilism (*So-called Lorain Type*).—In this variety, as described by John Thomson, the figure resembles that of a child. When the patient is stripped, however, the outlines are found to be those of an adult, simply reduced. The head is proportionately small, the trunk well formed; the shoulders proportioned to the hips, and the prominences of the bones and muscles are normally maintained. The subject is a miniature man (or woman), not one who has retained the characteristics of childhood beyond the proper time. There is no growth of facial, pubic or axillary hair, yet the genital organs though small are well shaped and proportioned to the size of the body. The intelligence in both sexes is generally normal.

The cause of this form is not positively known, but there is reason to believe it is due to deranged pituitary secretion. It has also been called "angioplastic infantilism" because associated with defective development of the vascular system.

II. The Hormonic Type.—This includes several varieties directly dependent upon change in the functions of the ductless glands. The most important are:

(*a*) *Thyroidal or Cretinoid Infantilism.*—Already described (p. 676).

(*b*) *The Frölich type, dystrophia adiposo-genitalis*, associated with a tumor. See diseases of Pituary gland.

(*c*) *Pancreatico-Intestinal Type.*—Cases of infantilism associated with intestinal changes have been reported by Bramwell, Herter, Freedman and others. Bramwell thought the pancreas was at fault, and his cases improved under treatment with pancreatic extract. In Herter's case there were looseness of the bowels, often fatty stools, and a change in the flora of the intestine with a rise in ethereal sulphates in the urine.

(*d*) *Ateliosis (Continuous Youth) and Progeria (Premature Old Age).*—Under these terms Hastings Gilford describes two types of dwarfs. Ateliosis also includes two sub-varieties, the asexual and sexual. The asexual is an infantilism unassociated with cretinism, syphilis, or congenital heart disease, often more a delay than an arrest of development. In the sexual form there is a like delay in development until puberty when the sexual organs mature and the body becomes set as a miniature man or woman. This is termed the "Tom Thumb" type of dwarf.

DISEASES OF THE THYMUS GLAND.

The thymus, a ductless gland primarily of epithelial structure, has assumed at birth a lymphoid type so pronounced that it is classed among the lymph glands. Supposed to produce an internal secretion, its function is unknown, while experiments made with extracts from the gland have as yet taught us nothing. Extirpation, followed at times by change of function in other organs and tissues, is at other times without effect. Again, the gland has been held to have some connection with the sexual function and the development of the bones and nerves. The former claim is attested by castration which is followed by permanence and enlargement of the gland. From birth up to two years when it reaches its acme of development it weighs seven to ten grams (105 to 150 grains). A weight of 20 to 30 grams (2/3 to 1 ounce) means abnormal size. The thymus occupies the space between the *manubrium sterni* and the spinal column, equal to 2.2 cm. or about an inch, scant space for enlargement without encroaching.

1. Simple hypertrophic enlargement appears to be the most frequent abnormality. It may attain a weight of 30 grams (an ounce) or more. Among its results either directly by pressure on the trachea or reflexly are

First, Thymic Stridor, Inspiratory and Expiratory.—The enlargement may be congenital or acquired after birth. The stridor varies in intensity, being especially aggravated by crying and coughing. It has been mistaken for diphtheria, and intubation has been practiced for this reason, but intubation furnishes no relief unless the tube be carried well below the upper thoracic strait. Moreover thymic stridor is never associated with hoarseness.

Second, Thymic Asthma or Kopp's Asthma.—A persistent and more severe form of asthma characterized by a progressive tendency to increase in severity. Attacks of this asthma may occur in a child without a previous symptom. Kopp ascribed it to thymic enlargement. In evidence that an enlarged thymus is the cause, its removal has been followed by complete recovery. On the other hand recovery has been spontaneous after the lapse of more or less time whence one may infer spasmodic contraction. Kopp's view, though widely accepted, was much disputed notably by Friedleben in 1858, but reaffirmed by Grawitz in 1888.

Third, finally *thymic death* (mors thymica), a condition in which death occurs suddenly with or without a history of previous respiratory difficulty. Between thymic asthma and thymic death, the distinction rests purely upon the degree of severity for there is no sharp line between them. The same reasoning as to etiology applies to both and the same difference of opinion exists as to it. Causes assigned for thymic death are first, enlargement

of the thymus mechanically encroaching on the trachea; second, a nervous influence which may be associated with lymphatic and thymic enlargement (status lymphaticus, with symptoms pointing to cardiac failure and sudden death. No one doubts that a certain number of deaths are due to thymic enlargement, these being, however, generally preceded by gradually increasing dyspnea so that death is never quite sudden. The precise mode of death is not settled. It may be purely mechanical or due to laryngeal spasm succeeding on a certain degree of tracheal stenosis. The truly sudden deaths are cardiac associated or not with status lymphaticus. Another possible cause of thymic death is by pressure of the enlarged thymus on the heart and great vessels, the vagi and recurrent laryngeal nerves, but this has not many adherents.

It must not be forgotten that there are other causes of sudden death in infants than those concerned with the thymus. Among these is "overlying." Careful postmortem examinations should be made in all cases of sudden death in infants.

Treatment.—Modern medicine has developed a treatment for enlargement of the thymus which includes measures prophylactic against pressure and excitement, to fortify surgical treatment by such measures as might be needed in emergency, *i.e.*, arrangements for intubation and tracheotomy. Curative treatment consists in intubation, Roentgen irradiation, treatment for rickets, syphilis and operation.

II. Atrophyof the thymus is found incidentally and appears to cause no special symptoms, but is rather the result at times of marasmus and the wasting diseases of children.

III. The thymus is the subject of other abnormalities often not recognized until after death. Such are hemorrhage, tuberculosis and syphilis, dermoid and other cysts. Mediastinal growths comparatively frequently start from the remnants of the gland. The thymus is the subject of postmortem softening at one time supposed to be a morbid state.

IV. A persistence of the gland after the age of puberty at which it has usually disappeared is not unusual, associated with conditions which are generally accidentally coincident, including exophthalmic goiter whose association has also been regarded as other than accidental.

SECTION VII

DISEASES OF THE URINARY ORGANS.

GENERAL SYMPTOMATOLOGY.

Four important symptoms more or less characteristic of diseases of the urinary organs, and especially of disease of the kidneys, may, for the sake of brevity, be considered at the outset of our studies of these affections. They include *albuminuria, renal dropsy, uremia* and *tube casts*.

ALBUMINURIA.

Definition.—By albuminuria is meant a condition of the urine in which it contains some of the forms of albumin of which at present we need consider only serum albumin and globulin. The sources of the albumins in urine are various, and may be conveniently divided into *extrarenal* and *renal*.

Extrarenal Albuminuria.

The pelvis of the kidney, the ureters, the bladder, the urethra, and in the female the vagina and uterus in addition, are the most important sources of extrarenal albuminuria. In all of them it is almost invariably the serum of *pus* formed during catarrhal inflammation which furnishes the albumin. The presence of pus-corpuscles, therefore, in sufficient number in the urine commonly explains the source of such albumin, which is, moreover, usually small in quantity—never more than about one-tenth the volume of urine tested, even with the most copious sediment of pus. It must not be overlooked, however, that the two sources, kidney disease itself and the mucous surfaces referred to, may coexist, in which event careful microscopic examination will sooner or later discover tube-casts, while the quantity of albumin will be larger than can be accounted for by the presence of pus alone.

Menstrual or lochial blood need only be referred to as sources of albumin in the urine hardly likely to be overlooked by any physician; while hemorrhage from any one of the mucous surfaces referred to, as well as from the kidney itself, would be a source of albuminuria. It is usually comparatively easy to determine whether a hemorrhage has its source in the kidney or in the mucous membranes previously mentioned. In the former coagula are rarely present, for the blood, entering the ureter slowly, becomes intimately mixed with the urine. It imparts to it, too, when acid in reaction, a *smoky hue* which is very characteristic. The coloring-matter of the corpuscles is commonly dissolved out by the urine which is thus tinged, and on standing, the stroma of the corpuscles sinks to the bottom as a brownish sediment. The microscope reveals these corpuscles shrunken, almost colorless, and often crenated. We have said that the smoky hue is present only in acid urine.

When the latter becomes alkaline, either by spontaneous or artificial change in reaction, it assumes a brighter red hue, the degree of which depends upon the quantity of blood. When blood comes from the pelvis of the kidney or the ureter in any quantity, coagula which are molds of the ureter are sometimes found, the descent of which is often attended with severe pain. Such coagula are frequently the result of bleeding from hypernephromata.

Another source of albuminous urine, though not likely to cause error, should be mentioned—viz., the so-called chylous urine, or chyluria, in which in consequence of some as yet imperfectly understood communication between the lymphatic system and the urinary tracts, chyle enters the urine and imparts its physical and chemical characters thereto. These are the presence of albumin, and a milk-white appearance due to the presence of fat in a molecular state.

The kidney itself may be the seat of suppuration, and contribute through the pus thus added to an albuminuria.

Renal Albuminuria.

The Immediate Cause of Renal Albuminuria.—Albumin in the urine is an abnormal condition, and while renal albuminuria suggests some change in the epithelium of the glomeruli, its presence must by no means be interpreted as a serious diseased condition of the kidneys. As above stated it may be extrarenal. These conditions being excluded we have left actual diseased conditions of the kidney itself or abnormal conditions of the blood or of other organs which so effect the kidney that it allows albumin to pass over into the urine. Suppuration of the kidney, actual inflammation or intestitial change in the kidney and congestion or hyperemia of the organ are the conditions in which changes in the kidney are the original source of the albumin.

Renal albuminuria also occurs as a secondary symptom in diseases other than renal. First may be mentioned the albuminuria of fever, such as that of typhoid fever, small-pox, etc. The febrile albuminuria alluded to is not usually large, and disappears with the decline of the disease. It is in great measure the result of irritation of the kidney by the infectious agent; possibly also, in part, the result of diminution of the cardiac force propelling the blood through the kidneys, and of the resulting turgor. Abnormal substances in the blood such as sugar, lead and bile pigment may produce renal albuminuria, especially if long maintained, probably because of their effect on the epithelium.

Other conditions in which albuminuria thus occurs are anemia, leukemia, diarrhea, cholera, lead colic, also certain conditions of the brain and spinal cord, including hemorrhages into the brain, meningitis, epilepsy. tetanus, and others.

The significance of renal albuminuria has altered greatly as our knowledge of this subject has increased. While large albuminurias of renal origin can scarcely be due to anything else but renal disease, and the degree of albuminuria is within limits a measure of the seriousness of the disease, yet the important fact remains that there may be true albuminuria, usually moderate, in which there is no disease of the kidney whatever; there may also

rarely be Bright's disease in which there is no albuminuria. The significance of albuminuria is always increased by its association with tube-casts, yet there may be both albumin and casts in urine where there is no Bright's disease, while again, there may be Bright's disease without albumin or casts. Such cases are infrequent, and yet this possibility must be acknowledged.

Physiological or Functional Albuminuria.—The possibility of a physiological or functional albuminuria at the present day is generally conceded. By it is meant an albuminuria unassociated with other symptoms. There are no tube-casts or feeling of ill-health. Such albuminurias are often discovered accidentally, especially by examiners for life-insurance. Much care should be exercised in concluding upon the nature of an albuminuria suspected to be functional. In the first place, it should be small, not exceeding one-tenth the bulk of urine tested, and though it is not necessary that it should be absent on rising, yet it is a strong point in favor of the functional nature if it is absent at this time and present only after some exertion has been made or on taking food. No tube-casts should be in the urine, the urea should be in sufficient quantity, there should be no retinal change, no hypertrophy of the left ventricle, no high tension to the pulse, nor even a suggestion of dropsy. Further, this condition should be watched over a considerable length of time before the conclusion is arrived at that we have to do with a harmless functional albuminuria.

It may be that these albuminurias are due to some changes in the epithelium or the blood pressure, but there is no way of proving it and presumably it passes away when the albumin disappears. The term *cyclic albuminuria* is given to such albuminurias as appear only at a certain time of day, and albuminuria of adolescence when it disappears after the establishment of maturity. The term orthostatic albuminuria is applied to these same conditions.

Other proteid matters are sometimes found in the urine, such as globulin, mucin, peptone, albumose and hemialbumose, but, except in the case of globulin, their clinical significance is not sufficiently determined to justify their further consideration in a text-book. A certain amount of *globulin* is always associated with albumin. In an ordinary serum albuminuria the ratio commonly maintained is 10 to 18 of serum albumin to one of globulin, the ratio in the blood being one of serum albumin to 1 1/2 to 3 of globulin. A like ratio holds for these constituents in pus. This ratio in albuminuria is at times exceeded, especially in the case of the amyloid kidney, where I regard the presence of globulin in large amount may be considered of diagnostic value.

Albumosuria is of greater significance when associated with *multiple myelomata* (*Kohler's disease*) and its long-continued presence suggests these tumors of bone, a well-recognized condition, of which many cases have been reported chiefly in males over 40, and terminating fatally. Albumose is occasionally found in association with other tumors of bone.

Tests for Globulin and Albumose.

The student is referred to works on Clinical Diagnosis for tests for albumin and globulin.

BACILLURIA OR BACTERIURIA.

Bacteruria is a condition of the urine charged with bacteria commonly pathogenic. The bacilli may arise from a focus in the bladder or urinary tract when it is known as *primary*. Or they may come directly from the blood when the affection is *secondary*. The bacteria commonly found are the typhoid bacillus, the colon bacillus, the streptococcus and gonococcus. The number of bacteria may be so great as to make the urine slightly turbid, or its appearance may not be altered.

Again the bacteria may be associated with cystitis or pyelitis and their other signs, or they may be unattended with any symptoms of inflammation. The urine is acid usually with the bacillus coli, and alkaline with the staphylococcus. It may be associated with phosphaturia. It is variously amenable to treatment and commonly easily curable by hexamethylenamine.

PHOSPHATURIA.

The term phosphaturia should be limited to a condition in which more than the normal quantity of phosphoric acid in combination is excreted in the 24-hour urine. This can only be determined by careful volumetrical analysis of the 24-hour urine. The amount of P_2O_3 thus excreted according to Hammarsten is from 1 to 5 grams or an average of 2.5 grains. Such a condition was described by Prof. J. Teissier of Lyons in 1876 under the name of essential *phosphaturia* or *phosphatic diabetes*. To the true phosphaturia are added some of the most distinctive symptoms of diabetes mellitus, viz., polyuria, thirst, emaciation, dry skin and pruritus. Charles Henry Rolfe, in England, has described thirteen cases with like symptoms. It is well named, therefore, "phosphatic diabetes." As much as 7 to 9 grams were found by Teissier in the 24-hour urine. To those who would make a difference between the two titles, phosphaturia would be a condition in which there is simply an excessive precipitation of phosphates due to a high alkalinity or deficient acidity of the urine, such phosphates, chiefly lime, being further precipitated on the application of heat. Such precipitate does not employ an excess of secretion of phosphoric acid but simply a deficient acidity.

The phosphoric acid in the urine is devised chiefly from the phosphates ingested with food. A portion results from the decomposition of nuclein, protagon and lecithin. The phosphoric acid thus resulting is combined with sodium, and potassium (fixed alkalies), forming the *alkaline phosphates* and with lime and magnesia forming earthy phosphates. The alkaline phosphates predominate, and of these the phosphate of sodium. Of the earthy phosphates the phosphate of lime preponderates. After the ammoniacal fermentation of urine within or without the bladder there is formed the ammonio-magnesian or *triple* phosphate in the shape of triangular prisms and feathery or stellar crystals also known as stellar phosphates.

It has long been an impression that there is a close relation between the metabolism of tissues and especially nervous metabolism and the excretions of phosphoric acid in the shape of phosphates. The German hold to this

view firmly and claim that on the part of the English Bence Jones erred in claiming that no increased elimination takes place in nervous diseases, and that an absence of an absolute increase in the excretions of P_2O_3 does not disprove an increase of phosphoric bodies since in many nervous diseases phosphorus appears in the urine not fully oxidized, that is as P_2O_3, lecithin and glycerophosphoric acid.

OXALURIA.

Oxaluria is another condition which in the past has been assigned an exaggerated importance. It is a condition of urine in which oxalate of lime in the characteristic crystalline shape is constantly present. Our own belief is that except as a condition favoring the formation of gravel and calculi it is of no importance. Instead of being a cause of a state of mental depression, often associated with it, both it and the melancholy and the deranged digestion and nervous function are the results of a deranged metabolism.

Most of the oxalate of lime in the urine is derived from vegetable food in which rhubarb is conspicuous while the intestinal fermentation of ingested carbohydrates with associated gastritis and the absence of hydrochloric acid is another source. Finally in a small way oxalate of lime is produced in the economy, either in the blood or at the moment of secretion in the kidney, from nuclein which is one of the antecedent states of oxalic acid as of uric acid. Connective tissue and gelatin, when ingested in large quantities, increase the output of oxalic acid probably because they stimulate the formation of nucleus. Abnormalities in the conversion may be responsible for unusual output.

INDICANURIA.

Indicanuria so-called requires a short notice only. It owes its name to the presence of indoxyl sulphate of potassium which is colorless, and is present in small quantities in all urines. When it is brought in contact with strong oxidizing agents, as the mineral acids, it is decomposed and indigo is set free. It is derived from indol, a product of proteid decomposition in the intestine. Indol is absorbed and converted into indoxyl which later unites with potassium sulphate. It is increased in gastric intestinal autointoxications sometimes abundantly, but later studies attach less importance to it. It may be a constituent of renal calculi. Rarely it decomposes spontaneously.

Cystinuria is a rare condition of the urine in which six-sided plates of cystin are found in it; sometimes aggregated to form small calculi. The sediment persists through life. One of us has seen one such case.

Alkapton is a substance sometimes found in urine which reduces Fehling's solution and thus misleads, but unlike glucose it does not ferment and is not removed by the polarizing saccharimeter.

Melanuria is a somewhat vague term applied to dark hued urines which are rendered thus by various causes, some of which have been considered. They include hemoglobinuria and hematuria, the urine of certain cases of

melanotic tumor, urine surcharged with indican, certain intensely jaundiced urine, the urine secreted after the ingestion of certain fruits and vegetables, certain medicines such as sulphanol (hemato or phogrinuria), carbolic acid, resorcin and others.

LACTOSURIA is the presence of lactose in the urine, rather common in women whose milk has just begun to be excreted. It reduces Fehling's solution, but cannot be fermented by yeast.

RENAL DROPSY.

Renal dropsy does not differ essentially from cardiac dropsy, though it is less directly traceable to venous obstruction and consequent transudation. It very frequently appears first in the face and upper extremities, and this fact alone goes to show that something else than obstruction to the circulation enters into its causation. On the other hand, it is well known that there may be advanced renal disease without dropsy when one would naturally expect obstruction. Venous obstruction must, however, be considered as one of the contributing factors, especially when in the lower extremities. Aside from this we must have recourse to theory to explain it. A hydremic state of the blood has much to do with the transudation in certain cases. At first thought this explanation would seem scarcely sufficient to account for the edema which appears to early in many cases of acute Bright's disease, but this objection is more apparent than real, for one need pause for a moment only to realize how quickly blood may become hydremic when there is almost total anuria, while the habitual quantity of water continues to be ingested daily. Moreover, diminished urinary secretion often exists for some time before attention is called to it. We believe that sufficient stress has not been laid on this factor. Increased permeability of the capillary walls produced by toxic substances in the blood also forms transudation.

As the disease advances, the conditions favoring the hydremic state continue and grow so that in advanced stages this element doubtless contributes largely. It is more than likely also that in this stage alterations of some kind in the vascular and lymphatic walls contribute tc facilitate the transudation, while the diminished elasticity of surrounding tissues may also constitute a factor. Where edema does not occur, its absence can only be explained by the presence of continued secretion of water by the kidneys or by supplemental secretion by some other organ, as the skin, the lungs and bowels.

Chlorid Retention.—Strauss, in Germany, and Widal and Javal, in France, in 1902 and 1903 called attention to the fact that there is an abnormal quantity of sodium chlorid in the tissues of persons affected with renal dropsy, reporting cases in which it was possible to cause edema to appear and disappear almost at will by varying the amount of chlorid in the diet. The rationale of these events is supposed to be as follows: In nephritis the ability of the kidney to excrete chlorid is decreased, whence their accumulation in the tissues and the consequent accumulation of water in the same tissues in order to keep the chlorids in solution. Although there

appear to be exceptions and the parallelism is not always demonstrable, the correctness of the statement is generally admitted. An opposite theory, that the primary trouble in nephritic edema is the retention of water to which the retention of salt is secondary, is not sustained by the weight of evidence. Chlorid retention has been observed in some cases of cardiac dropsy and with some cases of edema occurring in patients with inflammatory disorders, but there is no evidence to show that there is any causal relation between the two conditions or that the chlorid retention is more than can be explained by the retention of fluid in the edematous parts. The effect of chlorid retention is also to increase albuminuria.

On the other hand the recent experimental studies of Richardell,[1] P. F. Richter and Pearce show that vascular injury and renal injury the result of the effect of toxic elements of the blood as well as plethoric hydremia all enter into the causation of edema.

The degree of dropsy varies greatly in different cases and different forms of Bright's disease, being in some cases trifling and in others enormous, including, ultimately, invasion of serous cavities like those of the pleura, peritoneum, and even pericardium.

RENAL SUFFICIENCY AND INSUFFICIENCY.[2]

The Phthalein Test.—In 1910 Geraghty and Rowntree,[3] following the work of Abel and Rowntree[4] published their method of estimating renal function by means of phenolsulphonephthalein. Prior to the above publication numerous other methods such as phloridzin glycosuria, cryoscopy, electrical conductivity, and the estimation of eliminated urea, methylene blue, rosanilin, or indigo-carmine, had been brought forward to estimate this most important function. From cumbersome apparatus, painful injection, irregular secretion, or quantitively unknown proportions and the impossibility of accurate determinations, these procedures ar open to serious criticism or limited in their interpretation, and are being superceded by this new test which bears the mark of mathematical accuracy. The drug phenolsulphonephthalein, is absolutely non-irritating and non-toxic; practically it is excreted entirely by the kidneys with extraordinary rapidity; it is secreted chiefly by the renal tubule cells, which appear to have a selective power for it, with an ability to excrete it in high degrees of concentration. In the normal individual, when given intramuscularly, it makes its appearance in the urine in from five to eleven minutes; 38 to 60 per cent. being excreted in the first hour after its appearance in the urine, and 60 to 80 per cent. during the first two hours. Those diuretics that stimulate the renal cells to increased activity cause some increased secretion of phenolsulphonephthalein, while those that act mechanically produce no increased secretion.

The *technic* is as follows: A catheter is introduced and the bladder completely emptied. Exactly 1 c.c. of the prepared solution of phenol-

[1] The production of edema on experimental study, etc. Report from the "Archives of Internal Medicine," June, 1909.

[2] We owe much of this section to Dr. Alexander Randall.

[3] Geraghty and Rowntree, Jour. Pharm. and Exper. Therap., July, 1910, i, 579. Bibliography to date: "Jour. Amer. Med. Assoc.," 1912, vol. lix., p. 1813.

[4] Abel and Rowntree, "Jour. Pharm. and Exper. Therap.," Aug., 1909, vol. i., 231.

sulphonephthalein, which contains 6 milligrams of the drug, is injected deep into the lumbar muscle, the exact time of the injection being noted. As this is quantitative work an accurately graduated syringe (Record) is necessary. The urine is allowed to drop from the catheter into a test-tube in which has been placed a few drops of a 25 per cent. solution of sodium hydroxide. The phthalein, straw-colored in the acid urine, immediately indicates its presence in the alkaline test-tube contents, by a rose-pink coloration. In patients without urinary obstruction, or residual urine, the catheter may be withdrawn at the time of the appearance of the drug in the urine, and the patient requested to empty the bladder into a receptacle at the end of the first hour, from the time of its first appearance, and into a second receptable at the end of the second hour period. If there is any contraindication to the passage of the catheter, a rough estimate of the time of appearance may be obtained by having the patient void at frequent intervals, or one may allow ten minutes (as the outside limit of normal) and have him void at the end of the first hour and ten minutes, and again at the close of two hours and ten minutes. Such methods, however, are not advised, as they are prejudicial to accurate quantitative work and may allow faulty deductions. In case there is residual urine, due to urinary obstruction, the catheter should be fastened in place and corked at the time of the first appearance of the drug, the bladder being completely emptied by this means at the end of the allotted periods without any loss of the total amount of the drug eliminated. The first and second hour specimens as collected are measured and each alkalized by the addition of 25 per cent. sodium hydroxide solution until the brilliant purple-red color is obtained. Each one is then placed in a liter graduated flask and sufficient clear tap-water is added to make 1 liter. The solutions are stirred thoroughly and a small filtered specimen of each is taken for colorimetric determination. The quantity of the drug in each specimen can be estimated by comparing it with a standard solution in either the Duboscq or the modified Autenrieth-Königsberger colorimeter, or by comparison with a set of tubes of known percentage content. If the Duboscq instrument is used, a standard solution of 3 milligrams of phenolsulphonephthalein to the liter is advised, setting the Vernier scale at 10 in the standard solution cup for fine colorimetric reading. With the modified Autenreith-Königsberger apparatus a standard solution of 6 milligrams of phthalein to the liter is used. In cases the output of the drug is low, the urine specimen may be diluted to only 500 c.c., or to 250 c.c. and a mathematical correction made after colorimetric estimation.

This test is of distinct advantage in determining functional derangement;

a. In acute nephritis;

b. In chronic interstitial nephritis;

c. In chronic parenchymatous nephritis (to a less degree);

d. In surgical disease of one or both kidneys (aided by the ureteral catheter);

e. In the differential diagnosis of uremia from conditions simulating it;

f. In indicating impending uremia before the existence of clinical evidence;

g. In determining to what degree renal insufficiency is responsible for the clinical picture in cardio-renal disease;

h. And, when ureteral catheterization is performed, in showing the absolute amount of work performed by each kidney as well as its reserve force in health or disease.

It is to be borne in mind that such a test estimates renal function only, and that often a markedly altered kidney may have a remarkably good function; moreover, that renal function is a variable quantity allowing of a wide range of change, often in a comparatively short space of time. The curve of excretion, with an abrupt rise and a gradual fall, as the concentration of the drug in the body decreases, with a normal maximum in the first hour and a marked decrease during the succeeding periods should be taken as an indication that normal function is being performed, while it is to be remembered that diseased kidneys tend to work constantly at their maximum and each hour's elimination may be equalled, or excelled, by the succeeding ones and the total of a longer period of time, than one hour, may equal that of a normal pair of kidneys for the same space of time.

In cases of nephritis when the drug is excreted only in traces, or not at all, a grave prognosis should be given even though no signs of uremia exist.

A case just observed with double renal tumors. At the first observation was passing urine of normal specific gravity in about normal amounts with neither tube casts nor albumin. No phthalein was excreted in two hours. The patient three days after was given another test and passed no phthalein. In less than two weeks she became uremic and died comatose. The blood contained a high percentage of urea.

Other tests are used enabling the laboratory worker to tell with considerable accuracy whether the kidney is functioning or not. But they are not practicable for the clinic.

For further consideration and the details of the technic of these tests the reader is referred to works on Clinical Chemistry and especially to a paper by Rowntree and Fritz, "Studies of Renal Functions," "Archives of Internal Med.," Mar., 1913.

UREMIA.

Uremia is a symptom peculiar to renal disease. It may be defined in general terms as a condition due to retention within the blood of excrementitious substances which it is the function of the kidney to excrete. When we come to separate these substances, we are, however, completely at a loss, for no clinical or experimental studies have as yet given us the required information. Ligation of the renal veins has been equally futile in producing them. C. A. Herter has shown that the toxicity of the blood-serum is increased in uremic states, while extirpation of the kidney or a part of it increases the accumulation in the blood of urea and nitrogenous substances of the creatin class. It is probable also that the alloxuric bases, xanthin, hypoxanthin, which are virulently toxic, contribute to the sum of toxins responsible for uremia. Retained ammonia was held responsible by Frerichs and others.

A second view ascribes uremia to deranged kidney metabolism which Brown-Sequard long ago suggested might depend on an internal secretion.

Such disturbance, especially that of the muscles results in a large increase of urea and nitrogenous bodies of the creatin class, as is shown by extirpation of a part of the kidney. Rose Bradford, Hughes and Cortes conclude that the poison is albuminous and different from any of the normal ingredients of the urea.

A third theory ascribes urea to nephrotysms residing in the renal substance which is specifically destructive of renal cells when injected into an animal. This is apparently confirmed by the observations of Fillüller who ascertained that uremia occurs in those cases where there is most destruction of renal substance.

Finally some clinicians—and preeminently Rees and Traube—sought to explain certain of the more active nervous symptoms, such as the coma and convulsions, by supposing a localized edema of the brain or its membranes.

Symptoms.—It is not unlikely that *gastrointestinal symptoms* including the loss of appetite, nausea, vomiting, and headache which sometimes usher in an attack of nephritis may be due to retained excrementitious matter. At all events, these same symptoms may be the initial ones of a uremia, coming on suddenly when the cause is unsuspected. Vomiting thus caused may persist, and the patient perish in consequence. *Headache* is often occipital, extending down the neck. To it is superadded *dizziness*.

An early symptom is *drowsiness*, which may be sudden or gradual in its onset and may be slight or decided. From the latter degree the transition is easy to the next symptom, *coma*, from which the patient may or may not be temporarily aroused. Alternating with the latter may be epileptoid *convulsions*, which are the most alarming and dangerous symptom of Bright's disease. This is not always the order of succession of these symptoms. Convulsions may succeed drowsiness, but as often precede, and they may occur without warning. Indeed, there may be no suspicion of Bright's disease whatever until a convulsion suddenly occurs. Drowsiness, in like manner, may be the first symptom of the renal disease to attract attention, others being overlooked or possibly even absent. The convulsion exhibits every grade of movement, from the slightest twitching to the most violent epileptiform spasm.

Suppression of urine, an almost constant symptom, is frequently the initial one and should at once excite suspicion. Accompanying it is often a breath of urinous odor, and when vomiting accompanies scanty or suppressed urine, the vomited matters sometimes have the same odor, the constituents of urine being thus supplementarily eliminated. *Renal insufficiency* as determined by the phthalein test (p. 699) is an important evidence of uremia.

Defective vision or actual blindness—amaurosis—suddenly occurring is another symptom of acute uremia which sometimes supervenes upon others, or it may itself usher in the complication. This blindness, it must be remembered, is altogether different from that which is the result of organic retinal changes, which are rare in acute nephritis, but common in some of the chronic forms. It may be due to retinal hemorrhage but is often unassociated with demonstrable retinal change. Uremic amaurosis

often disappears as suddenly as it sets in. Uremic *deafness* is also possible, but is a rare event.

Itching of the skin is another symptom sometimes present in uremia. It is probably due to the irritant action of urea upon the nerves of the skin as it is being supplementarily eliminated by that organ. That such increased elimination takes place is attested by that rare but still unquestioned occurrence, in which the entire integument is covered with a frost-like coating, which has been found upon analysis to be made of crytals of pure urea. Cramps especially in the muscles of the calf is included by some among the symptoms of uremia.

Another symptom of uremia which belongs rather to the uremia of chronic renal disease is shortness of breath—*uremic asthma*. This is an asthma which differs from bronchial asthma in the absence of spasmodic contraction of the bronchi but that it is uremic in origin, we think, is very doubtful. We believe it is more frequently a cardiac asthma, due to dilatation of the heart succeeding the hypertrophy so often present in chronic Bright's disease. It may be that true spasmodic asthma is produced by uremia, but it must be exceedingly rare. The attacks are likely to occur suddenly at night. To it alone should the name uremic asthma be given. Paroxysms of dyspena are also caused by edema of the lung, which is not infrequent in acute nephritis. It is recognized by the presence of fine, moist râles. Of course, it is not impossible for nephritis to occur in an asthmatic, whose attacks would then occur as before, or they might be more frequent or rendered more unmanageable by the uremia.

Cheyne-Stokes breathing is also a symptom of uremia and may last for a long time. It may occur quite independently of coma. It is more than likely that other disturbances of breathing, even some which closely resemble Cheyne-Stokes variety, may be due to disturbances of the respiratory center.

The question of *temperature* in uremia has not been satisfactorily settled. Usually there is an absence of any elevation, with at times a tendency to subnormal temperature. Sometimes there is an abnormally high temperature. But in true uncomplicated uremia in the majority of cases the temperature will not much exceed the normal. It is true, too, that toward the very end there is commonly a rise, but this attends dissolution in so many diseases that it cannot be considered characteristic.

The *pulse* is often slow in uremia before the appearance of severe symptoms: sometimes as infrequent as from forty to fifty, but with severe symptoms it becomes more frequent.

Acute mania and *delusional insanity* (*Folie Brightique*) may also be symptoms. Such acute delirium is not very frequent and other conditions are sometimes mistaken for it, as for example the delirium of *mania a potu*. Rarely melancholia and paralysis including hemiplegia and even monoplegia are symptoms. These may occur independently of a convulsion or succeed it, in which event there may be may not be any coarse lesion found at necropsy. True uremic plasies are of undoubted occurrence.

The following table, although not distinctive gives a general idea of the differences between the effects of cerebral hemorrhage, alcoholic narcosis and uremia.

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.	May be stertorous breathing.	Sterterous breathing.
No odor.	Odor of alcohol.	No odor, unless urinous.
Paralysis; hemiplegia.	No paralysis usually.	Paralysis often present, but may be atypical.
Pulse slow and strong or irreg.; arteries often sclerotic.	Pulse frequent and feeble.	Pulse at first strong, later weak and rapid; high blood pressure; arterio-sclerosis.
Coma sudden and deep.	Coma gradual.	Coma gradual or sudden.
Convulsions late; may be unilatural.	No convulsions.	Preceded by general convulsions, headache, etc.
Urine generally negative.	Urine generally negative although patient may have Bright's disease.	Urine albuminous and contains tube casts.
Apoplectic habit; heart may show hypertrophy.	Red face and nose, heart often weak and dilated; myocarditis.	Edema and pallor; heart hypertrophied.
Moderate increase of blood pressure; Hypertension however ,caused by large intracranial hemorrhage.	No rise in blood pressure.	Blood pressure very high, 200 mm. or more.

Renal insufficiency as evidence by testing is conspicious in uremia. See especially previous section on Renal Sufficiency.

TUBE-CASTS.

Tube-casts or "cylinders," as they are sometimes called, are molds of the uriniferous tubules. Their origin is not always the same. They may be produced by the coagulation of a fibrinous constituent of the blood which having exuded into the tubule entangles whatever it may have surrounded in its liquid state; subsequently it may contract and slip out of the tubule into the pelvis of the kidney, whence it is carried to the bladder and voided with the urine. Casts rarely exceed 1/25 inch (1 mm.) in length.

Two other possible modes of formation of casts must be mentioned, according to one of which the cast represents disintegrated and fused cells which may be the epithelial lining of the tubules, red corpuscles, or leukocytes; and according to another, of a secretion from these same cells as originally suggested by Rovido. That casts are sometimes formed according to the first, at least, of these two methods is not unlikely, although it is more likely that even in these cases the basement substance which cements the cells and this débris is the same exuded substance above referred to; while there is reason also to believe that the so-called "cylindroids" or mucus-casts originate in the second way.

That casts are sometimes found in urine free from albumin in undoubtedly true. Persons in whom such casts occur are frequently healthy.

The mechanism of the formation of the different varieties of casts, on the supposition of an albuminoid basis substance exuded from the blood, is very simple. Thus, suppose a tubule to be filled with detached and loosely attached epithelium at the time the coagulable material is poured into it. These elements are entangled, and, as the casts contract, are car-

ried out in the shape of an "*epithelial*" cast (Fig. 121). If the tubule should happen to have contained blood, the cast entangling it is called a "*blood-cast*" (Fig. 123); if white corpuscles or leukocytes, a "*pus-cast*" (Fig. 122). Casts containing even a few blood-corpuscles are also called blood-casts.

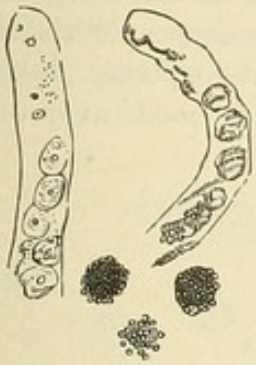


FIG. 121.—Epithelial Casts and Granular Fatty Renal Cells.



FIG. 122.—Pus Cast.

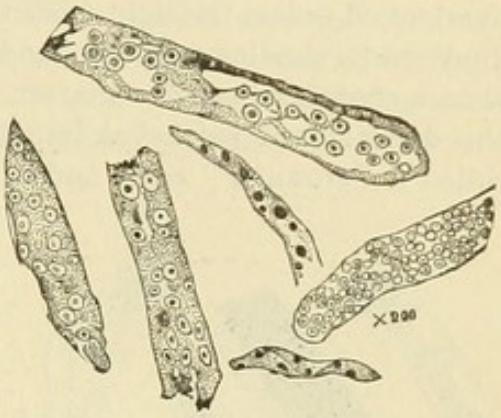


FIG. 123.—Blood Casts—(*after Whittaker*).

The basic substance of blood-casts is most probably the fibrin of the blood. If the epithelium be firmly attached to the basement membrane of the tube and remain behind when the cast passes out, or if the tube be entirely bereft of epithelium, then is the cast a "*hyaline*" (Fig. 124) or structureless

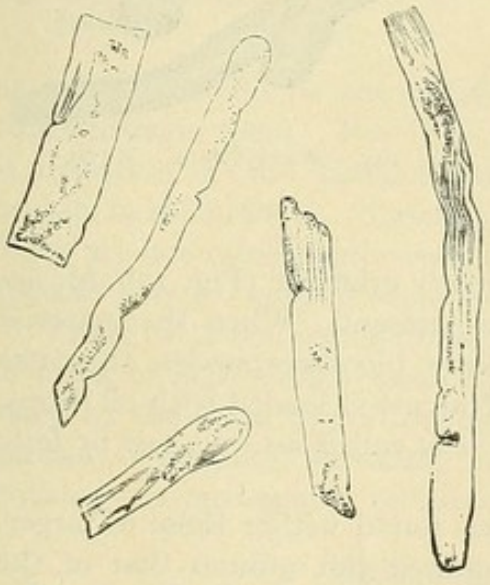


FIG. 124.—Hyaline Casts. × 210.

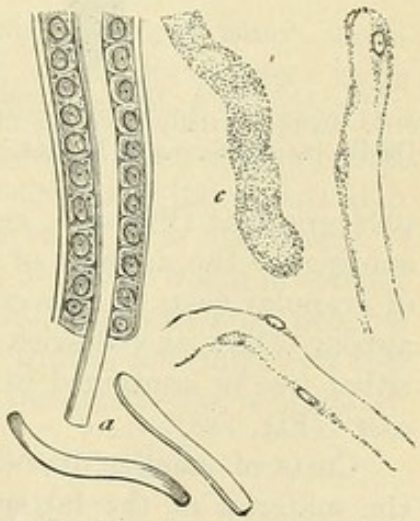


FIG. 125.—Hyaline and Granular Casts. Illustrating the Formation of the Former at *a*.—(*Rindfleisch*).

cast. In the former instance the cast is of *smaller* diameter, and in the latter of *larger*, the diameter in the latter being that of the former plus twice the thickness of an epithelial cell. Figure 125, *a*, from Rindfleisch, explains this sufficiently. From causes like these, as well as a subsequent contraction of the cast itself, the diameter of casts may vary considerably,

ranging commonly from 1/2500 to 1/500 inch (0.01 to 0.05 mm.). A cast is seldom completely hyaline, generally containing a few granules and sometimes one or two glistening oil drops, but it is still called *hyaline*. Completely hyaline casts are, however, not infrequent. Less intense irritation seems to form hyaline casts while more intense irritants produce granular casts which in the course of time may become hyaline. A variety of hyaline cast, more solid in appearance and resembling molten wax, is a "*waxy cast*" (Fig. 127). Some hyaline casts are so delicate as to be overlooked unless the light from the mirror illuminating the field of view be modified by shading with the hand or by manipulation of the mirror itself. If a cast contains granular matter, which is generally the granular débris of the degenerated epithelial lining of the tubule or blood-corpuscle, it is called a "*granular*" cast, and *highly* granular (Fig. 126, *a*), *moderately* or

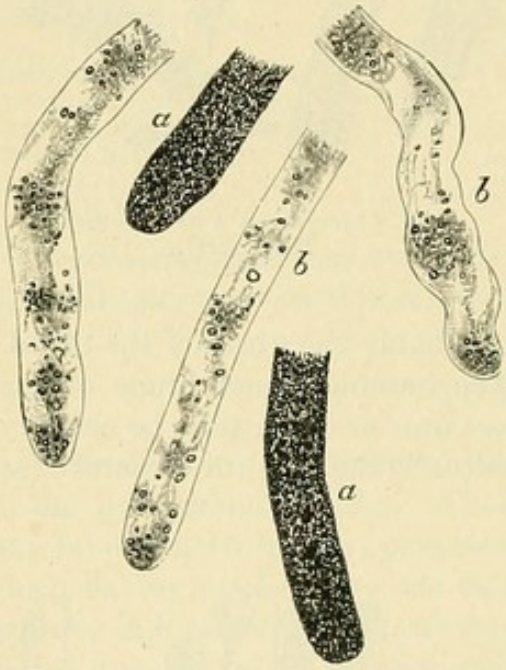


FIG. 126.—*a*. *a*. Dark Granular Casts. *b*. *b*. Casts Partially Hyaline, Containing Oil-Drops and Granular Matter. ×225.

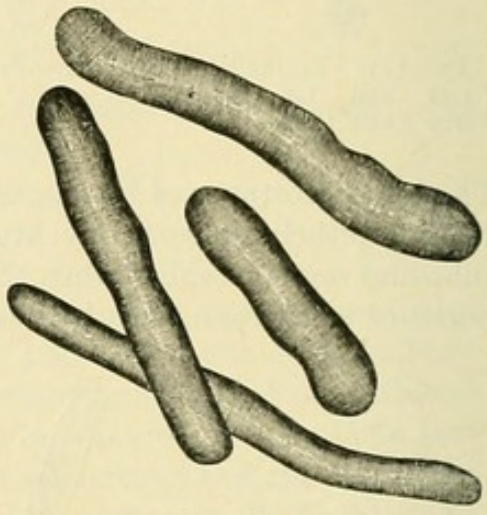


FIG. 127.—Waxy Casts. ×150.

pale granular (Fig. 125, *c*), *slightly* or *delicately* granular (Fig. 126, *b*), according to the amount of granular matter present. When the material of granular casts is derived from broken-down blood-corpuscles, the casts appear yellow or yellowish-red. Finally, if a cast is loaded with oil drops, either free or contained in epithelial cells, it is called an "*oil cast* or *fatty cast*" (Fig. 128).

Casts of smaller diameter are sometimes found within those of larger, the material of the latter having been poured out around that of the former after it has undergone some contraction. This occurs usually with waxy or hyaline casts. In consequence of the mode of formation previously referred to, hyaline and waxy casts vary considerably in diameter, some being as narrow as 1/1000 inch (0.025 mm.) and even narrower, while others are as much as 1/500 inch (0.05 mm.) wide. There is no doubt that some of these are formed in the straight or collecting tubes near their openings on the papillæ. To these a limited number of epithelial cells is sometimes attached.

Studies of the origin of casts experimentally produced by Wollenstein, Ribbert, Schleaht and R. M. Smith do not lead to results widely different from the above. For a fuller discussion of this subject, however, the reader is referred to a paper by Henry A. Christian, "Jour. Amer. Med. Assoc. vol. liii., 1909, "Clinical Value of Recent Studies in Experimental Nephritis."

Mucus-casts, Cylindroids.—Casts are occasionally found which are apparently pure *mucus-molds* of the uriniferous troubles (Fig. 129). Unless covered by accidental elements, as granular urates or phosphates of lime, they are smooth, hyaline, or gently fibrillated molds, especially characterized by their great length, in the course of which they may divide and subdivide, diminishing in diameter as the division proceeds, showing positively that they come from the kidney. Yet there is no albumin, or merely

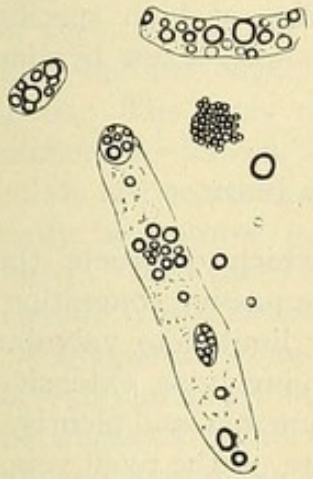


FIG. 128.—Oil-casts and Fatty Epithelium. × 200.

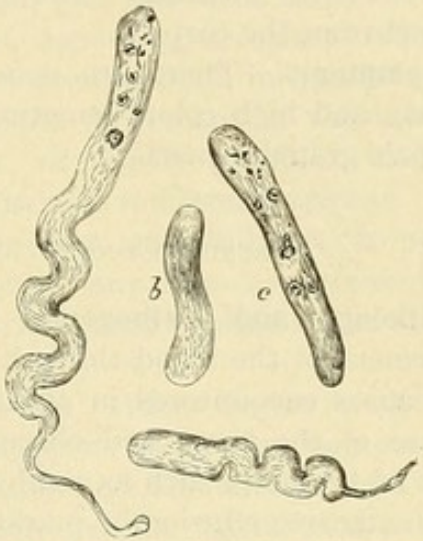


FIG. 129.—Cylindroid or Mucus-casts. × 200.

as much as could be accounted for by the presence of pus which sometimes attends them. For they are particularly apt to occur where there is irritation of the bladder, which is apparently extended through the ureters to the kidney. Under these circumstances they are frequently met. They are not infrequently voided in cases where the urine has a very high specific gravity, 1030 or higher, containing an excess of urea and urates.

These casts are not identical with the bands of mucin which are found in urine of highly acid reaction. The mucin bands are probably precipitated by the acids, are often beset with granular urates, and might on this account be mistaken for casts. At the same time the mucus-cast is probably nothing but pure mucus or mucin.

DISEASE OF THE KIDNEY.

DERANGMENTS OF CIRCULATION.

Active Congestion.

Etiology.—Active congestion occurs as the result of poisoning by cantharides or arsenic, overdose of turpentine, copaiba, cubebs, and carbolic acid and in acute fevers. It is identical with the first stage of acute nephritis, however caused. The kidney in acute fever has usually been considered as

actively congested, but recent experiments by Walter Mendelsohn go to show that it is really anemic during fever, *i. e.*, small, pale and bloodless, so that the organ is really comparable to the anemic kidney of cholera, in which, too, there are albuminuria and nutritional changes of a degenerative kind analogous to the cloudy swelling affecting the renal cells in acute fever, but more advanced in degree. Postmortem this state of the kidney is not found in fever, it is large, red and swollen as all can attest who have to do with either.

Morbid Anatomy.—The kidney of active congestion is slightly enlarged, swollen, and, after removal of the capsule, brown or mottled. On section, the cortex is wider and darker than in health, the blood-vessels are overfull, the Malpighian capsules are distended, and the cells are the seat of cloudy swelling. The medulla is less markedly red and is sharply defined from the cortex.

Symptoms.—There are none except a scanty urine of high specific gravity and high color, sometimes small albuminuria, with a few hyaline and pale granular casts.

Passive Congestion or Cyanotic Induration.

Etiology and Pathogeny.—While any agency which obstructs the movement of the blood through the kidney may cause passive congestion, the causes encountered in actual practice are mostly limited to valvular disease of the heart and chronic pulmonary disease involving extensive areas of the lung, such as emphysema, interstitial pneumonia, and pleurisy, with extensive effusion or marked adhesions. Pressure on the renal veins by tumors, the pregnant uterus, or ascitic fluid acts similarly.

In any event the mechanism of its production is the same. The blood is crowded into the venous side of the vascular system. In pulmonary or pleural disease the obstruction begins in the lungs instead of in the heart, but the mechanism is the same. Pressure by tumor on the renal vein is even more direct.

Morbid Anatomy.—The kidney of cyanotic induration or passive congestion is hard, firm, and bluish-red on its external surface. In the earlier stages it is enlarged simply from the presence of the large amount of blood detained in its vessels. The stellate veins are unusually distinct. The capsule strips off easily, and on section the enlargement is found to involve the cortex, but the veins of both cortex and medulla are engorged, that of the straight veins causing the medulla to appear darker in hue than the cortex. The Malpighian bodies, on the other hand, are not always engorged. The cut surface of the kidney is moist and succulent, but the microscope reveals no further changes either on the cortex or the medulla, the epithelium being unchanged.

After some duration the kidney is slightly if at all larger than the normal organ, though rarely smaller. The other superficial characters of hardness, smoothness, and bluish-red color, however, remain. Sometimes there appears a slight tendency to lobulation. At this stage the capsule does not strip off quite so easily, and may drag small portions of the parenchyma with it. There may then be seen some shallow depressions. On section, the

vessels are less turgid, and the relations of the cortex and medulla are not much altered. There may be a slight overgrowth of interstitial tissue and a small-celled infiltration between the tubules. The Malpighian bosdie are sometimes shriveled, and the epithelium of the tubules is granular and slightly fatty.

Symptoms.—These are primarily those of the diseases of which it is the consequence, of which *anasarca*, is of more importance. To such is superadded *scanty urine* of *high specific gravity*, containing usually a small amount of *albumin* and a few *small hyaline casts*.

The dropsy first involves the lower extremities, in the area drained by the inferior vena cava. There also occur, however, effusions into the pleural sac and peritoneum, and the hands and arms may be involved.

The *urine* is scanty and of high specific gravity, often 1030 to 1035, and even higher. It is turbid with urates, depositing a copious sediment of them and of uric acid. The albumin is usually small in quantity, but may become larger if the obstruction to the movement of the blood is great. The casts are small, transparent, or faintly granular, and not numerous—indeed, often absent. Blood discs are sometimes present. The solids are secreted in normal amount. In fact, such kidneys can apparently be restored to their normal function at any time by proper treatment. Very rarely does it happen that an intersital nephritis is produced. *Uremia is rare in this form.*

Diagnosis.—Passive congestion exists to a certian degree in all cases of valvular heart disease without compensation, but higher degrees may be suspected when the urine becomes scanty and albuminous, and when all the symptoms of the cardiac affection become aggravated. When the physician sees the patient after the symptoms of passive congestion have become marked, it is often a nice question to decide which of the two conditions is primary, the cardiac or the renal condition; but this subject will be further considered in treating of the relations between kidney disease and heart disease.

Prognosis.—With the addition of the renal complication, the inconveniences and annoyances of the cardiac disease become greatly aggravated, while the difficulties in the way of successful treatment are greater. Yet the results which sometimes follow appropriate and energetic treatment and the substitution of favorable for unafvorable hygienic surroundings, such as succeed the admission of a neglected outcast to the wards of a hospital, are often astonishing. Under these circumstances it is not unusual for the dropsy to decline, the albumin and casts to disappear, and the patient to be restored to comparative comfort. In the meantime the cardiac decompensation has been temporarily retarded.

Treatment.—As intimated under prognosis, the substitution of favorable for unfavorable hygienic surroundings, if the former exists, is the primary requisite. Shelter, warmth, test, and good food are indispensable. After this digitalis is the sheet-anchor, for evident reasons. We have here to deal with a dilated, weak, failing heart, unable to drive the blood forward. Its power must be increased, and we have a remedy capable of doing this in digitalis. Sufficient doses must, however, be given, whether of the tincture, powder, or infusion. A 1/2 ounce (15 c.c.) of the infusion

may be given every six hours to an adult; of the tincture, not less than 10 minims (0.65 c.c.), or twenty drops, to be reduced when diuresis set in. Under such doses, if the cardiac disease is not too advanced, the quantity of urine may increase, become clear, its albumin and casts diminish, and with these also the dropsy, dyspnea and restless, sleepless nights. All that has been said under the treatment of cardiac valvular disease of substitutes for digitalis is applicable here, and the reader is referred to that section.

Due attention must also be paid to the bowels, for the sake of securing prompt action of the diuretics, as well as the elimination which their free action accomplishes. The hydragogue cathartics, such as elaterium and the salines, are often excellent adjuvants.

ACUTE PARENCHYMATOUS NEPHRITIS.

Synonyms.—*Acute Nephritis; Acute Diffuse Nephritis; Acute Desquamative Nephritis; Acute Tubal Nephritis; Acute Bright's Disease; Acute Catarrhal Nephritis; Croupous Nephritis; Albuminous Nephritis; Hemorrhagic Nephritis; Acute Albuminuria; Acute Renal Dropsy.*

Definition.—Acute parenchymatous nephritis is an acute inflammation of the kidney, the tubular, vascular, and interstitial tissues being simultaneously involved in different degrees in different cases. In the majority of cases, the parenchyma, or secreting structure, is first and most invaded, whence the term parenchymatous nephritis, although diffuse nephritis is a more correct term.

Etiology.—Most cases of acute parenchymatous nephritis are caused by some poison of endogenous or exogenous origin which is carried by the circulation. Instances of the former are the toxins of scarlet fever or diphtheria, whence, therefore, it is frequent in children. A certain number originate from exposure to cold, especially cold and dampness, when the person is fatigued or exhausted and has been using alcohol to excess. The cold here probably acts as the exciting cause lowering the resistance of the kidney to the circulating poison. When acute nephritis supervenes on scarlet fever, it is usually not until the end of the second week, often when convalescence is well established. It may occur as early as the tenth day, seldom, if ever, later than the thirty-first.

Other grave infectious diseases, as small-pox, acute endocarditis, and acute articular rheumatism, typhus and typhoid fevers, pneumonia, malaria, tonsillitis, measles, erysipelas, pyemia, jaundice, and diabetes have been known to cause it. Skin diseases, as well as extensive burns of the skin, are acknowledged causes: the former rarely, but the latter almost always if the burns be sufficiently extensive. Toxæmia of pregnancy is a common cause. This is of extreme importance because if the toxæmia is discovered before the nephritis is severe the cases respond well to treatment. Probably the eclampsia which sometimes accompanies this condition is due rather to the toxæmia than to the nephritis.

In looking for the evidence of nephritis in acute infectious diseases, it must not be forgotten that intense febrile movement may cause albuminuria, independently of any structural change in the kidney due to the

toxic agent. When thus caused, the albuminuria is always small and may be disregarded.

Of exogenous causes certain specific poisons of vegetable and mineral origin are capable of producing acute nephritis. Among the best known of these substances are cantharides, turpentine, oil of mustard, wormseed oil, and phosphorus; in a less degree, the mineral acids, arsenic, nitrate of silver, lead, and mercury. Very large quantities of alcohol, when swallowed, have caused acute nephritis.

Uranium nitrate, potassium chromate, corrosive sublimate, cantharadin and arsenic are favorite poisons used in the production of experimental nephritis the first three producing the tubular or epithelial form while arsenic and cantharodin produce the vascular type. Uranium nephritis associated with an excess of water in rabbits, at least, is accompanied by a well-marked edema, pointing to an injurious effect on the blood-vessels as well. Moreover, the serum of an animal with uranium nephritis when introduced in an animal with chromate nephritis causes also a well-marked edema.

As may be inferred from the etiology, acute nephritis is often a disease of early age, although when due to cold or any one of the causes named except scarlatina, it is as much more likely to affect adults as these latter are more frequently subjected to such causes. It is rare after 40, almost inknown after 50. More males are attacked than females in adult life, evidently because they are more frequently exposed to the causes. But even in childhood there is a slight preponderance of cases in boys affected, which can hardly be thus accounted for.

Morbid Anatomy.—This varies with the stage of the disease, as well as its severity. In the first place, as ordinarily caused, the disease is symmetrical, both organs being alike involved. In the fully developed stage the kidneys are more or less enlarged, in the latter stages always so, sometimes to more than twice their normal volume, and they may weigh from eight to 12 ounces (240 to 360 gm.), those of children reaching the former, and those of adults the latter.

The capsule strips off easily, without dragging any of the parenchyma with it. Bereft of its capsule, the kidney itself is softer, inelastic, and doughy. Its surface is smooth and exhibits a peculiar mottled appearance, which is due to the fact that the little circlets of veins which form the boundary of the lobules are distinctly injected, while the area surrounded by each circlet is paler than in health, and in the more advanced stages even yellowish-white in color. This "irregular mixture of congestion and anemia," as Sir George Johnson early called it, is further contributed to by the injection of other veins indistinct in health. Spots of hemorrhagic extravasation may also be found scattered over the surface.

On section, it is evident that the enlargement is due to change in the cortex and the interpyramidal convoluted portion. The cut surface is smeared over with a dark red or chocolate-hued blood, but on scraping or washing it away the vessels are found injected like those of the surface, and between them the same paleness or yellowish-white hue is seen. The Malpighian bodies are enlarged and distinct, dark red, sometimes pale. Punctiform hemorrhages may also be present, as on the surface of the organ. The pyramids are dark red.

Minute Changes.—These are confined almost solely to the labyrinthine structure. They by no means always correspond in degree with what would be expected from the symptoms, being often entirely inadequate to explain them. The changes are *tubal*, *glomerular*, and *interstitial*.

1. *Tubal Changes.*—These vary a great deal with the stage of the disease. The earliest change assumed by the cells is cloudy swelling, a result of increased nutritive activity. In this state the cells are swollen and "cloudy" from a deposition of albuminous granules, which may obscure the nucleus. Although kidneys removed after death from cases of acute parenchymatous nephritis have, as a rule, advanced far beyond this stage, yet it is often possible to find points at which cloudy swelling exists alongside of more advanced stages, while alongside of these, again, may be tubes in which the epithelium is normal. The swollen cells are larger, and the tubes are therefore distended, increasingly so in a later stage, with granular cells, granular débris, and often red-blood disks and leukocytes. Under a low power, the tubules appear as black, more or less opaque lines. A closer examination of the *cells* at this stage, as obtained by scraping, shows them to be granular in various degrees. In some the nucleus is still visible, in others demonstrable by the aid of staining fluids only, and in others still entirely obscured. Occasionally a few fat drops may be present. In other situations the cells are so closely packed in the tubules that they cannot be differentiated, being apparently fused in one continuous, dark, granular mass. It is to these tubules, distended with granular cells and their débris, dark by transmitted light, but white by reflected, that the pale or white color seen between the injected blood-vessels is due. Casts of the uriniferous tubes are also found *in situ*, usually blood casts or small hyaline casts. Minute extravasations of blood, visible to the naked eye, have been referred to. They occupy the tubules, and in the "hemorrhagic" form the interstitial tissue.

2. *Glomerular Changes.*—The glomerule first is affected. The capillaries of the *tuft* are distended with blood, which bursts through into the Malpighian capsule, distending it with red blood-corpuscles and leukocytes. In a more advanced stage, the glomeruli may be paler, in consequence of the proliferation of the cells lining the capsule and covering the glomerule (glomerulonephritis).

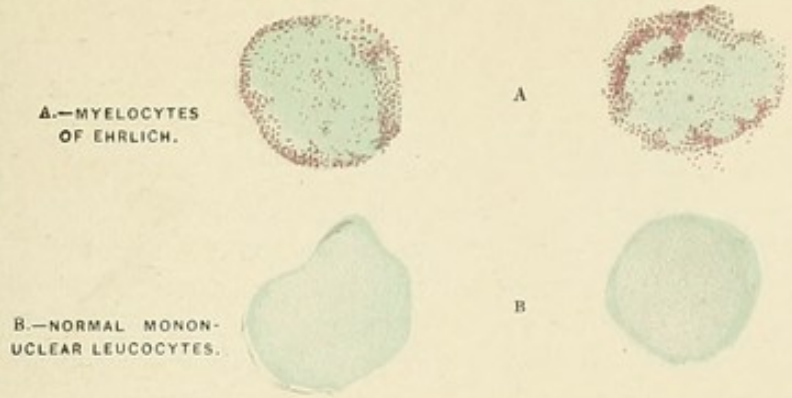
These glomerular changes are present in almost all cases. They include swelling and desquamation of the capsular epithelium, and an accumulation of cells in the interior of the capillaries (intracapillary), probably due to a proliferation of their endothelial lining or an accumulation of white blood-cells, or thickening and hyaline degeneration of the capillary walls. The last named vascular lesion is found in experimentally produced acute nephritis[1]. These are especially frequent in nephritis after scarlet fever or diphtheria.

3. *Interstitial Changes.*—In mild cases there is no interstitial change, no formation or deposit of new material between the tubes. In others there is a serous transudate, with few leukocytes in most cases, and red

[1] See the studies of Perace, Christian, R. M. Smith and Walker on "Experimental Renal and Cardio-renal Disease" published in the "Journal of the A. U. A.," the "Archives of Medicine" and "Journal of Experimental Medicine," 1909–1912.

PLATE VI.

FIG. 1.



1100/1

FIG. 1.—STAINED WITH METHYL-GREEN—ORANGE G—ACID-FUCHSIN. NUCLEI GREEN, NEUTROPHILIC GRANULATIONS PURPLE.

FIG. 2.



1500/1

FIG. 2.—STAINED WITH EOSIN-HAEMOTOXYLON. A.—MYELOCYTES OF EHRLICH. B.—POLYNUCLEAR LEUCOCYTES. C.—LYMPHOCYTE. D.—NUCLEATED RED CORPUSCLE. E.—DEGENERATED NUCLEUS. F.—RED CORPUSCLES.

PLATE EXHIBITING STAINED CORPUSCLES FROM THE BLOOD OF A CASE OF LEUKHAEMIA.

blood-disks. In severer cases there is a large outwandering of cells, and a small-celled infiltrate settles itself between the convoluted tubes and around the capsules. In cases of extreme severity, a diffuse nephritis involving both tubes and intertubular tissue may be present from the outset. In such event, the latter is uniformly pervaded more densely in certain places by leukocytes.

The epithelial lining of the straight tubes of the pyramids is unchanged, but the tubes themselves often contain cellular and granular material which has descended from the convoluted tubes.

Serous infiltration and effusion are present in various tissues when the patient is dropsical at the time of death. Among other tissues sometimes thus infiltrated are the membranes of the brain, constituting what is known as edema of the brain. The mucous membrane of the pelvis of the kidney may be injected, but otherwise unchanged.

Symptoms.—The mode of onset of acute nephritis is not always the same. Usually it is sudden. Less frequently the illness is ushered in by a chill or chilliness. More often the first observed *symptom* is slight *swelling* or *puffiness in the face*, below the eyes, associated with more or less *falling off in urinary secretion*. This *edema rapidly extends* to the upper extremities and trunk, and thence, if the disease does not abate, into the lower extremities and abdominal walls. In the male, the scrotum and prepuce are favorite seats of swelling. The great serous sacs are the last to fill with fluid in acute nephritis, although in bad cases ascites not infrequently occurs, while there may also be transudation into the pleural and pericardial cavities. The degree assumed by the general anasarca is sometimes enormous, resulting in extreme distortion. The eyes may be actually closed by the swelling, and movement of the lower limbs rendered almost impossible. Dropsy does not always follow the order here named. Much depends upon the position of the patient. Thus, if he be upon his feet, the latter may be the first to swell, or if he be lying in the recumbent position, the back may be the seat of the first swelling. While dropsy is a very frquent symptom in acute nephritis, it is not, however, always present. It is more particularly in the nephritis after scarlet fever and exposure to cold that it is a decided and almost invariable symptom. After the other infectious diseases it is frequently absent.

Modern studies have shown that changes of a very positive character in the glomerular capillaries occur in connection with experimentally induced nephritis.

In view of the fact that studies in experimental nephritis have discovered in the capillaries of the glomerule, a vascular lesion consisting in a type of hyaline degeneration favoring the transudation of serum, and since these studies tend to show that the edema of nephritis is dependent on some vascular lesion; and since, moreover, uranium nitrate which produces the glomerular lesion referred to is the one which experimentally most frequently leads to the accumulation of fluid in the cavities and subcutaneous tissue associated with renal lesion, Christian and his colleagues made a series of experiments to determine whether or not other demonstrable vascular lesions resulted from the subcutaneous injection of uranium nitrate in rabbits. These resulted in the conclusive that the degenerative condition is

limited to the glomerular capillaries and does not affect the other small vessels of the kidney or those of the heart, liver, spleen or mesentery. It is, however, likely that despite the absence of anatomical lesion some vascular lesion may exist as is shown in the case of arsenic which producing a paralysis of the capillary walls allows a leakage of serum tc take place, with no demonstrable inflammatory lesion.

Not infrequently the disease is ushered in by *nausea* and *vomiting* and very rarely by uremic symptoms (see p. 702).

Fever is not a marked symptom in acute nephritis; indeed, it is generally absent, unless as a part of the disease causing it. It is more apt to occur in children. To a less degree the same is true of *pain*. It is mostly absent, and when present amounts only to a dull ache, as a rule. *Nausea* and *vomiting* are not infrequent in the beginning. Sometimes these symptoms usher in the disease. The *pulse* is quite characteristically altered. While not materially changed in rate, it exhibits, especially in sphygmogram, a decided increase in tension, as shown by the broader apex and diminished dicrotic element. *Blood pressure* is increased, but much less than in chronic nephritis.

Uremia.—At almost any time in the course of an acute nephritis the patient is liable to *uremia* with the *train of nervous symptoms* usually known as *uremic*. Its causes and phenomena, so far as known, were considered under general symptomatology, page 702. When present, it adds a phase of extreme gravity.

Changes in the Urine.—Simultaneously with, and sometimes earlier than, the dropsical symptoms are *diminution in the quantity and alteration in the quality of the urine*. The former may amount to actual suppression. The urine is darker than natural, and often smoke-hued from the effect of the natural acid reaction on a small quantity of blood. Should the urine become alkaline, the color becomes a brighter red. The hue is more positively red if the quantity of blood is large, which is not often the case; but here again the peculiar tint returns if the blood is allowed to subside. The blood may disappear, to return again.

The *specific gravity* of the urine at first is high—1025 to 1030—mainly due to the diminished quantity, while the solids remain nearly normal. Later, if the symptoms abate, the specific gravity diminishes with the increase in the quantity; or, if the disease lasts for any length of time or passes over into the chronic form, a similar reduction in weight occurs; this may result in a specific gravity as low as 1010.

The chief alteration is the *presence of albumin*. This is generally large the urine often solidifying on the application of heat and acid, while it constantly contains more than half its bulks. This albumin is derived in part from the extravasated blood, and in part is a result of the inflammatory action. If estimated by weight, it will equal 0.5 to 1 per cent., and, in rare instances only, 1.5 per cent.

As to *sediment*, the urine af all cases of acute parenchymatous nephritis deposits a sediment which, in the early stages at least, is copious and brownish or reddish-brown in hue; later, it may diminish in amount and assume a lighter color. *Microscopical examination* reveals this deposit to be made up mainly of casts of the uriniferous tubules, free cells

from these same tubules, blood-corpuscles, red and colorless, and very constantly crystals of uric acid, together with granular urates. The casts include the varieties known as epithelial casts, blood casts, hyaline casts, waxy, and dark granular casts. Pus casts and numerous leukocytes are also sometimes present. The hyaline casts are probably pure fibrin. The epithelial casts consist of the same material, to which epithelial cells of the tubules are attached, and blood casts have blood-corpuscles caught in the coagulated exudate. The epithelium thus attached, as well as that which is found free in the urine, is variously altered. Some of the cells are merely the seat of cloudy swelling, others are decidedly granular, while others again are converted into compound granule cells or granular fatty cells by complete fatty degeneration. These arise as the disease advances. Casts containing a few oil drops may also be present, but much oil is not found until the case has continued for some time—in fact, become chronic.

Along with the diminished quantity of urine is often met a disposition to *frequent micturition*, the efforts at which are only partially successful, resulting in the emission of from a few drops to a tablespoonful. This frequent desire to pass water is a purely reflex symptom, the bladder being free from disease. It sometimes precedes, in point of time, all other symptoms. It is by no means constant.

The duration of acute nephritis is variable—from a few days to several months, while the acute form may become chronic. The former class of cases are fatal, for none which recover do so in a few days. The most rapid usually required a month. As to the cases of longer duration, the possibility of recovery at any time cannot be denied, but nothing is better determined than that the longer the duration the more difficult the cure. Of course, such cases are no longer acute.

Complications.—These are not numerous in acute, as contrasted with chronic Bright's disease, and some which are described as complications are not really such, but local symptoms. Thus, *edema of the lungs* occurs as a part of the general tendency to dropsy, due to cardiac failure, and may be a grave symptom, resulting in death by suffocation. It is not the result of an intercurrent bronchitis. *Pneumonia*, on the other hand, is an occasional true complication. *Inflammation of the serous membranes* is more truly a complication but not every case in which there is effusion into a serous cavity is inflammatory. Such effusions may be local dropsies. The exudate may become purulent, thus also increasing the gravity of the case. Pleurisy is the most frequent form, pericarditis next, and peritonitis next. The tubercular origin of the graver forms of pleurisy occurring in Bright's disease has been suggested.

Hypertrophy of the left ventricle is not a frequent complication of acute nephritis. It is a well-recognized one of chronic Bright's disease. Time is an essential condition to its production. It is not, therefore, until the nephritis has existed for some time that it commonly occurs. It does occasionally happen earlier. Thus, Dickinson reports a case recognized at eight weeks, and von Leube one at ten days succeeding the first symptoms. The infallible sign of hypertrophy is sharp accentuation of the aortic second sound, with or without demonstrable enlargement of the

normal area of dullness. *Arterial hypertension* present to a degree in all is less marked than in chronic interstitial nephritis.

Allusion has been made to *gastric symptoms* which very commonly attend acute nephritis, especially after scarlet fever. Samuel Fenwick[1] and Wilson Fox[2] have shown that these may be associated with organic changes in the stomach. Fenwick ascribes them to gastritis, as evidenced by increased vascularity of the mucous membrane, distention of the tubes by a confused mass of cells and granular matter, and occasional thickening of the basement membrane. To these, Fox has added thickening of the intertubular tissue.

Notwithstanding the frequency of convulsions in acute nephritis, structural *alterations* in the *brain* are almost unknown. Apoplectic effusions are rare, probably because of the integrity of the blood-vessels of the brain in the young, in whom the disease mainly occurs. Blindness which is not frequent. Albuminuric retinitis does not occur in acute parenchymatous nephritis, except with the extremest rarity.

Diagnosis.—The diagnosis of acute parenchymatous nephritis is ordinarily quite easy. The previous history of health the usually easily recognizable cause, the suddenness of the attack, the scanty and bloody urine with its high specific gravity, the copious albuminuria, the blood and epithelial and dark granular casts, the blood-corpuscles, free epithelium, and granular cells in the urine—these are a combination of symptoms which admit of only one interpretation. At a later stage, the absence of one or more of these symptoms may somewhat increase the difficulty, but it is scarcely possible to err if those which remain are duly considered. It must be remembered, also that an acute condition, such as this described, may supervene upon any one of the chronic forms of *Bright's disease* to be described, and this may give rise to some difficulty of diagnosis, but if there be hypertrophy of the left ventricle, it is likely that there was chronic disease before; in the latter case, too, there is apt to have been anemia existing for some time, previous edema, headache, and other symptoms of chronic Bright's disease.

Febrile albuminuria is quite often mistaken for acute nephritis by those who have had little experience, though the distinction is easy. In pure febrile albuminuria, the quantity of albumin is very small, and while there may rarely be a few hyaline casts, there are no blood-disks and no epithelial casts. The absence of dropsy is of no significance, for in the acute nephritis of the infectious diseases, except scarlet fever and diphtheria, there is seldom dropsy. There may also be febrile albuminuria in scarlet fever which is quite different from the nephritis occasioned by this disease. It occurs early, and in this stage the other features of febrile albuminuria are present, while the scarlatinal nephritis does not come on, as already stated, until after the end of the second week.

While the glomerular changes referred to are more usual in scarlatinal nephritis, there is no certain way of recognizing such condition, and the term glomerulo-nephritis, which is applied to the nephritis associated

[1] Samuel Fenwick, "The Morbid States of the Stomach and Doudenum," 1868, p. 177.
[2] Wilson Fox, "Medico-Chirurg. Transac.," vol. xli., p. 361.

with these changes, is scarcely justified from the clinical standpoint because there are no symptoms by which it can be recognized.

The diagnosis of *uremia* commonly easy, is sometimes difficult. This is especially the case when, instead of the usual complex list of symptoms detailed on page 702, there are but one or two. By no means every nervous manifestation conicident with Bright's disease is uremic. On the other hand, localized convulsions and hemiplegias, commonly ascribed to some anatomical lesions in the brain, are often uremic in origin. Given, however, a case of sudden convulsions or coma, or even muscular twitching, if it is associated with scanty urine and greatly diminished urea excretion, it may be ascribed to uremia, provided there is no cause which will explain it more satisfactorily.

We are paying less attention of late to the estimations of urea in the urine as an aid to the diagnosis of uremia because of other factors and especially food effects on the quantity. On the other hand the estimation of urea in the blood has acquired greater importance from these experiments of C. A. Herter and A. J. Wakeman[1] who showed that a positive increase in the urea of the blood succeeded upon extirpation of the kidneys from a normal average per cent. of 0.037 to an average of 0.315 per cent. in dogs. Similar increase in the urea of the blood was observed by the experimenters in several cases of uremia (See also p. 702 *et. seq.*).

Uremia has been mistaken for *opium* and *alcohol intoxication*, and it must be admitted that the coma in all three is very much alike. But one need only be forewarned to prevent such error. In opium-poisoning the pupils are contracted, in alcoholism they are dilated, in uremia they vary.

The phthalein test is of utmost value in deciding as to whether symptoms under observation are due to renal insufficiency or not.

Prognosis.—Grave as this disease is justly considered, recoveries from it are numerous and the prognosis is generally favorable. Even without treatment, cases may recover, and more recoveries follow a judicious treatment. The prognosis should, however, always be guarded, as insidious causes may produce death when it is least expected. Among the most important of these is uremia.

Bartels said that death from uremia in acute nephritis has never occurred in his experience, except when the disease has resulted from scarlatina or diphtheria; but Dickinson narrates a fatal case resulting from exposure, in which death was preceded by coma and other symptoms of evident uremic origin, and Tyson has observed similar cases.

Pulmonary edema is a cause of sudden death, the patient drowning, as it were, in his own secretions. Its onset is characterized by shortness of breath, frothy expectoration, and abundant small râles.

The symptoms of gravest import are, therefore, those of uremia, manifested in any one or all the various ways, the presence of any of the complications alluded to, and especially suppression of urine. Cases should not, however, be despaired of, even when there is complete suppression of urine. Always, however, this is the gravest of symptoms, and death generally ensues within a couple of days after it sets in. The possibility of sudden death should always be borne in mind, and mentioned to the

[1] "Blood Changes in Double Nephrectomy," "Journal of Experimental Medicine," vol v., 1899.

relatives of the patient, although the number of cases in which this occurs is not very great. Of course, the longer the duration of the case the less the likelihood of recovery.

Treatment.—Many cases of acute nephritis recover under the conditions of *rest*, *quietude*, and *warmth*, and it is further certain that, whatever other means of treatment are used, these three conditions are absolutely necessary to recovery. A patient with acute Bright's disease, therefore, whatever its mode of origin, should be put to bed, kept quiet, and covered warmly.

The *diet* of patients with acute Bright's disease should be of the simplest and easiest of digestion, and should contain a minimum of proteids. The irritability of the stomach in this disease has been alluded to, and it is important that food should be adapted to it. Milk may be considered the typical food, not merely because of its easy assimilation and nutri ious character, but because there is abundant testimony to prove that albuminuria diminishes under its use, while the amount of nitrogen contributed to the blood is less than by animal flesh. The combination of lime-water, and still better of carbonated water or Vichy, with milk, is an eminently suitable one. Koumys, Zoolak and buttermilk are also suitable. While solid animal food is not to be recommended, weak animal broths may be permitted, to break up the monotony of a pure milk diet. *Beef-teas and extracts should be prohibited as harmful.* Rice and farinaceous preparations generally are suitable adjuvants to the milk diet.

We should seldom, however, be satisfied with this treatment alone. The selection of other measures will depend somewhat upon the severity of the case. If the urine be suppressed, *dry cups*, or, in severe cases, wet cups to the loins may divert the blood and relieve the stagnation which always exists in the acutely inflamed kidney. Cups should be followed by a *warm poultice* to the same region, which, indeed, should be used under any circumstances, whether the cupping is necessary or not. Dry cups should not be allowed to remain on one spot longer than to secure a bright redness, after which they must be withdrawn or moved to another spot in the vicinity. By allowing them to remain too long, the blood is stagnated in the capillaries, its onward movement prevented, and there is, therefore, no derivation of blood from the involved organ.

The foregoing measures have for their object the direct relief of the congestion of the kidney. This is further accomplished by *purgation*, which supplements the action of the kidney. But a purgative is early employed not more for this purpose than to promote the action of other remedies. Absorption is slow when the blood-vessels are congested and there is a sluggish current. The cathartic relieves this turgor, and after its effect prompt absorption and action of other remedies may be looked for. The purgative most suitable is a saline. A simple dose of bitartrate of potassium, simple magnesia for children, citrate of magnesium, or Epsom salt for adults will be sufficient. The indication is to get a watery stool as soon as possible. In view of the fact that the stomach is often sensitive, it is desirable to use an aperient which is not nauseous or irritating, and to this end some one of the delicate effervescing preparations so common in modern pharmacy may be used.

Next, or simultaneously, the action of the skin should be promoted. This is done by maintaining warmth and avoiding cold, as already insisted upon. But we are not confined to these protecting measures. The skin may be made to do the work of the kidney itself, and thus one of the most alarming dangers of Bright's disease, uremic intoxication, averted, while at the same time the congestion of the kidney is also relieved. The class of remedies which produces this action are diaphoretics (warmth described is one of these), and of the simple remedies, none is better than the ordinary sweet spirit of niter, especially if it be combined with neutral mixture and small doses of ipecacuanha. If more active measures are required, pilocarpin may be given with caution in doses of from 1/24 to 1/12 grain (0.0027 to 0.0054 gm.). The further use of this important remedy will be again referred to in treating uremia.

Another method of accomplishing the same end is by *warm baths*, or, better still, by the warm pack, in which the patient is wrapped in a sheet wrung out in warm water and then enveloped in a sufficient number of blankets. Perspiration is thus copiously induced, and when thus caused is agreeable and never attended by the faintness which sometimes follows the use of the hot-air bath. In an ordinary severe case of acute Bright's disease, a single pack of this kind will often remove all urgent symptoms and happily inaugurate the convalescence. It may, however, be repeated daily, if necessary. Hot air or hot steam packs are more easily given with one of the various forms of cabinets on the market.

Diuretics are not indicated in the early stages of Bright's disease, they should be deferred until the measures just described have been employed. Digitalis is the diuretic most to be relied upon. It is necessary, however, to have a reliable preparation, and unless one is sure of the quality of the tincture, it is best to use a freshly prepared infusion.

Digitalis should, therefore, be given in sufficient doses—1/2 to 1 fluidram (2 to 4 c.c.) of the infusion to children, and 2 fluidrams to 1/2 fluidounce (8 to 16 c.c.) to adults—repeated every four hours, until an appreciable effect is produced on the rate of the pulse, when it should be diminished. Not until then can we look for a diuretic action. We prefer at first to give it alone. Later it may be combined with acetate, citrate, and bitartrate of potassium. The diuretic action of these salts probably depends upon the impetus they give to osmosis of fluids holding them in solution, thus filling the blood-vessels, which, in their turn, give out water to flush the kidney. To adults, 20 grains (1.3 gm.) of either may be given every two or three hours, freely diluted, because water itself is an excellent diuretic; from 5 to 10 grains (0.32 to 0.648 gm.) to children, as often. An important object, too, is to maintain an alkaline urine, which tends to dissolve exudates. For this purpose, the alkaline-mineral waters are also useful, or what is commonly known as cream-of-tartar tea may be drank instead of water. A teaspoonful of potassium bitartrate is put into a pint of boiling water, and taken cold as drink is wanted.

Fisher's Alkaline Treatment of Acute Nephritis.—Prof. Martin H. Fisher[1] premising that all the changes that characterize nephritis, viz., the albu-

[1] Nephritis and Experimental Study of Its Nature, Cause, and Principles of Relief. The 1911 Cartwright Prize Essay, N. Y., 1912.

minuria, the specific morphological changes noted in the kidneys, the associated production of casts, the qualitative variations in the dissolved solids are due to a common cause—the abnormal production or accumulation of acid in the cells of the kidney—concludes that the entire purpose of our therapy must be to get alkali into the patient to neutralize the acids present. To this end he recommends administration by the rectum of sodium bicarbonate crystallized, 20 grams, sodium chloride, 14 grams, and water enough to make 1000 c.c. This is administered high up in the bowel by the drop (Murphy) method at the body temperature. In a typical case with anuria, in an hour and ten minutes 30 c.c. of bloody urine were passed and in an hour 80 c.c. more.

From then on the "urine fairly streamed out" and by the fourth day albumin and casts had entirely disappeared.

Prof. Fisher's book contains the reports of numerous cases in which it was brilliantly successful. Dr. H. Lowenburg[1] of Philadelphia also reported at the recent meeting of the Medical Society of the State of Pennsylvania three cases with like success. It is evident that this treatment by introducing chloride of sodium in this blood involves views counter to those who advocate the salt-free diet in nephritis associated with dropsy.

Treatment of Acute Uremia.—The alarming and dangerous character of the symptoms of this condition demand a separate consideration of the measures required in their treatment. The treatment which has just been described is such as would be called for by an ordinary case of acute nephritis of a decided character. The tendency of it will be to prevent the retention of those toxic matters, whatever their precise nature, which constitute the cause of uremia. But all efforts in this direction sometimes fail, and we are called upon to contend with convulsions or coma, or, more frequently, both. How shall they be met? The indication has already been explained. Elimination is demanded. The kidneys are not acting, and the secretion of urine is suppressed. There remain, therefore, but the bowels and skin to operate upon. But the patient is unconscious and cannot swallow voluntarily. Such remedies, must, therefore, be used as do not require his cooperation. These are *croton oil* and *elaterium*. Of the former, 2 drops, slightly diluted with plain oil or glycerin, may be carried into the back part of the throat, or, in case of extreme necessity, undiluted, may be introduced into the mouth, whence it is quickly absorbed. Its operation may be facilitated by a rectal injection. Of elaterium, a quarter of a grain (0.0165 gm.) in solution may be administered by the mouth.

In like manner, the skin may be made to substitute the action of the kidney. The *vapor or hot-air bath or hot pack* should at once be availed of. The vapor may be conveyed under the bed clothes by a pipe from a vessel of water heated by a spirit-lamp, the patient, with the exception of his head, being well covered with a mackintosh and blanket. An ordinary rain-spout may be used. Hot air may be similarly conveyed, but does not act so quickly. Its action may be favored by moistening the skin. The hot pack is also very efficient and less uncomfortable. One of the various forms of cabinets is better.

[1] Proctoclysis as a Curative and Prophylactic Agent in Primary and Secondary Acute Nephritis in Children. Read before the Medical Society of Pennsylvania, Sept. 26, 1912.

Pilocarpin may be used hypodermically. One $\frac{1}{16}$ grain (0.006 gm.) of hydrochlorate may be thus administered, and if perspiration does not set in in a half-hour, it may be repeated. Its action is also greatly facilitated by warmth applied to the patient.

If the convulsions continue, *blood-letting* may be practised, to be followed by an intravenous injection of normal salt solution.

The *hydrate of chloral* is one of the most valuable remedies for the convulsion and should be one of the first measures tried. In the case of an adult, a dram (4 gm.) in solution may be injected into the rectum; 15 to 30 grains (1 to 2 gm.) for a child. Its use is sometimes followed by the promptest favorable results. *Chloroform* may also be used to control the convulsion while the eliminating measures are acting.

The *use of opium* requires mention. The caution which has always been suggested in its use is, in the main, a wholesome one, and I should prefer to produce hypnotic, sedative, and antispasmodic effects by chloral and the bromids whenever it is possible. At the same time there can be no doubt that it may be used cautiously with great benefit. It was in the convulsions of acute nephritis that the late Professor Loomis, of New York City, recommended it, although its wider use has grown out of this suggestion. His practice was to treat cases of uremic convulsions in acute nephritis with hypodermic injections of large doses of morphin—1/2 grain (0.033 gm.) or more.

The same measures which have been detailed, excepting the general blood-letting and chloral, may also be employed in the treatment of *suppression of urine* or of *obstinate dropsy* without uremic symptoms, with such modifications as circumstances may suggest, due regard being paid to the strength of the patient. They will be further referred to when discussing the *treatment of the chronic forms of Bright's disease*.

Sooner or later, also, in the treatment of acute parenchymatous nephritis supporting measures are rendered necessary to repair the losses which the blood suffers by the albuminuria, and to some extent also by the depleting measures of treatment. These effects should indeed be anticipated by proper diet, tonics, especially iron or strychnine, as indicated. These measures will also be more particularly alluded to in the treatment of chronic Bright's disease.

Treatment of Complications.—Complications should be treated by remedies called for by such conditions independent of the renal cause. Effusions into the plural cavities and abdomen are often best relieved by paracentesis or aspiration.

CHRONIC PARENCHYMATOUS NEPHRITIS.

SYNONYMS.—*Chronic Diffuse Nephritis; Chronic Tubal Nephritis; Chronic Catarrhal Nephritis; Large White Kidney.*

Definition.—A chronic diffuse hyperplastic process in the kidney, involving the epithelium, glomeruli, and interstitial tissue.

Etiology.—This cannot always be traced. While it is frequently a continuation of acute nephritis, more frequently it originates *de novo*.

To cases in the former category scarlatina and pregnancy contribute the greater number. To the second class belong insidious cases, the cause of which is often not treaceable. Habitual exposure to cold and dampness, such as residence in damp, cold houses, may cause some. Tubercular disease of the lungs is an undoubted cause. Great stress is laid by German writers upon malarial poisoning as a cause. Out of 1832 cases of malaria collected at Johns Hopkins Hospital there were 25 with nephritis. It may be the case in more southern parts of the United States, where malarial poisoning is more intense than in the Middle States. Alcohol is a cause, and the nephritis of confirmed drunkards and the employees of breweries may be thus accounted for, though it cannot be denied that the exposure to which some of the former class are subjected may be responsible. Males, and of these young adults, are the more frequent subjects. Sepsis in prolonged surgical affections may produce chronic nephritis.

Morbid Anatomy.—There are two distinct stages in the morbid anatomy of chronic parenchymatous nephritis if the disease is of sufficient duration—viz., the stage of enlargement, represented by the *large white kidney*, and that of contraction, or the *fatty and contracting kidney*. A special variety is *chronic hemorrhagic nephritis*.

1. *Stage of Enlargement.*—There are few more striking objects in morbid anatomy than a typical large *white kidney*. The kidney is large, smooth, white, or slightly tinged with yellow; weighs generally from seven to ten ounces (217 to 310 gm.), but is often much heavier. It is usually doughy, but sometimes elastic in consistence. The capsule, which may be thinner than in health, strips off easily, but occasionally drags a little of the parenchyma with it. When the smooth white surface thus uncovered, is examined, the little capillary circlets bounding the lobules in the normal organ are in some places indistinct, in others conspicuous; the same is true of the stellate veins of Verheyn. Numerous yellow specks are seen scattered over the surface. Hemorrhagic extravasations are also occasionally present, but very much more rarely than in the acute form. Alongside of these the greater translucency of more nearly normal areas results also in a characteristic mottled hue. *On section*, it is evident that the enlargement resides altogether in the *cortex*, which is also anemic, its intense white contrasting strongly with the pink hue of the cones, which, though paler than in health, are much less so than the cortex. Closer examination of the cut cortex reveals the same yellow specks as found on the external surface. They contribute, with similar less decided alterations, to form a series of dull white striæ which alternate with somewhat broader, translucent striæ radiating toward the surface; the former correspond to the area of the convoluted tubules and Malpighian bodies—the labyrinth—the latter to that of the medullary rays.

The pelvis of the kidney in chronic parenchymatous nephritis may be the seat of catarrhal swelling and a slight degree of hyperemia.

Minute Change.—Microscopic examination of thin sections shows the involvement of *tubes, blood-vessels and intertubular substance*. Of the former, many are found choked with granular cells and the granular débris of cells, causing them to appear, under the microscope, as black, opaque lines by transmitted light, very similar, indeed, to the tubes in acute nephritis.

In other situations the tubules are filled with fat globules and fatty cells. In places the lumen of the tubes is preserved, in others not. Other cells are the seat of hyaline change. Others still are nearly normal. The parts presenting a yellow tinge are those in which the fat has replaced the normal protoplasm, and this is the composition of the yellow specks already alluded to as visible to the naked eye. They represent a coil of tubules filled with oil drops or fatty cells.[1] Certain tubules contain casts, often of the waxy kind. Sometimes they are very numerous. Rarely, hemorrhagic extravasations are found in the tubules.

The *capillaries* of the cortex are completely or nearly empty of blood, which has been expressed from them by the distended tubules. To this and to the fatty cells is due the extreme whiteness of these kidneys, whence the name large white kidney. Many of the glomeruli are enlarged, their capsules thickened, their vessel-walls thickened and hyaline, their capillary and glomerular epithelium proliferated and degenerated.

The *pyramids* in chronic parenchymatous nephritis are more changed than in the acute form, but the changes in them are quite secondary. They are sometimes a little paler, owing partly to a granular and fatty alteration in the cellular lining of the straight tubules, and partly to the presence of cells pushed down from the cortex above them. On the other hand, they may be congested and darker in color. The straight tubes of the cones as well as the looped tubes of Henle often contain waxy casts.

In chronic parenchymatous nephritis the *interstitial tissue* is always increased, it may be said, as a rule in proportion to the duration of the disease. It is difficult to say when this overgrowth begins in any given case. Langhans reports a case in which death occurred five weeks after the appearance of the first symptoms, directly traceable to a thorough wetting, in which the stroma was *markedly thickened*. And in a case of Dickinson's already alluded to, intertubular cellular formation, "though approximating as much to pus as to fiber," was found within six weeks of the onset. Again, cases of much longer duration may be entirely without it. Interstitial *fibrosis* may, however, be considered as a superaddition of chronicity, and whenever a case is distinctly chronic, it may be inferred, with tolerable certainty, that it is present. In this overgrowth the quantity of the connective tissue between the tubules varies extremely, being sometimes so slight as to be discoverable only by the microscope in thin sections; at other times it is appreciable to the naked eye. Minute examination shows the thickened trabeculæ to consist of numerous round and oval nuclei, between which may be homogeneous or more or less distinctly fibrillated intercellular substance.

2. *The Stage of Atrophy.—The Fatty and Contracting Kidney or Small White Kidney.*—The interstitial new formation previously referred to possesses the properties usual to new connective tissue. Produced primarily to replace destroyed tubular structure, it shrinks and gradually contracts the previously enlarged organ, while obliterating in turn a corresponding amount of the same structure. The extent of contraction varies greatly,

[1] No satisfactory explanation has yet been offered of the great differences in the degree of fatty degeneration in the different kidneys of chronic parenchymatous nephritis or in different parts of the same kidney. Dickinson says the cells have a greater tendency to be fatty when cold is the cause.

increasing with the duration of the process. The kidney may continue as large and even larger than the normal organ, though smaller than the large white kidney, and its surface is uneven, lobulated, rough, and granular. Its capsule does not strip off easily, as from the large, smooth organ, but drags with it considerable of the tubular structure. The capsule removed, however, the surface of the kidney exhibits between the constrictions the same pallid, speckled appearance, distinct stellate veins, etc. already described; and on section the cortex exhibits the same anemic appearance, but may be narrowed. Microscopically, sections exhibit the same alternation of groups of normal and choked tubules alongside of other places in which the tubules, together with the Malpighian bodies at their extremities, are obliterated. Between them is found a large amount of interstitial tissue, and the Malpighian bodies are surrounded by concentric layers of the same. Even minute cysts, the result of obstruction of tubules by the constricting tissue, are found. The secondary origin of this form of kidney is not conceded by everyone, an independent primary origin being claimed for it.

3. A special form of this stage is *chronic hemorrhagic nephritis*. In this form brown hemorrhagic foci are scattered throughout the cortex between and in the tubes. The organ is still larger than normal, and presents in other respects the histology of this stage.

It not infrequently happens that along with the changes constituting chronic parenchymatous nephritis are found also those of *amyloid disease* Thus, in a large white kidney the Malpighian bodies will often strike the mahogany-red reaction with iodin characteristic of this condition, although the alteration may not be recognizable by the naked eye. Occasionally the change may even affect the afferent and efferent vessels.

Symptoms.—There are few distinctive symptoms of chronic parenchymatous nephritis. When not a sequel of acute nephritis it often begins insidiously, and, after a variable period of indescribable ill health, including often *digestive derangements*, an *anemic, waxy appearance* develops, with *puffiness of the face* and *swelling of the feet*. Ultimately, the *anasarca* may become general, involving the face, hands, feet, legs, thighs, and trunk. The serous sacs also frequently contain fluid, almost always in advanced cases. The swelling may be confined to the extremities or to the face, and may even be limited to more unusual situations, as the scrotum. On the other hand, dropsy is often entirely wanting, but as a rule it is manifest sooner or later, and no symptom gives the patient so much inconvenience. In advanced cases, the legs and thighs are twice their normal thickness. They are so heavy he can scarcely lift them, while they are often excoriated and moist with exuding serum, and smarting with irritation. Sometimes, as the result of spontaneous rupture of the skin, the flow of serum is profuse, saturating the bed-clothing and even dropping upon the floor; often with relief to the patient.

Another very frequent symptom is *anemia*, producing a peculiar translucent waxy appearance, quite characteristic and often alone sufficient to suggest the disease. But there may be very slight degrees which do not attract attention. Again, the *debility* of those suffering from advanced chronic Bright's disease is very striking. If able to walk, they soon get

out of breath—are soon exhausted. *Dyspnea*, especially on exertion, is therefore a frequent symptom, and sometimes is extreme.

The Urine.—The urine is diminished, although somewhat variable in quantity. It is often turbid, reddish-yellow, specific gravity changing as the quantity, highly albuminous, and deposits often bulky, cloudy sediment. At other times the sediment is scanty. The quantity of urine also increases as the patient improves or as the stage of contraction is entered upon, so that it may even exceed the normal. The *albumin*, while also large, varies as to its percentage amount with the quantity of urine passed—from five-tenths to two per cent., or from one-half to three-fourths of the volume of the urine tested. The amount of albumin lost in the urine is sometimes very large. It has even occurred that the percentage proportion of albumin in the urine has exceeded that in the serum of the blood from the same patient. The quantity of albumin has very little effect upon the specific gravity. Indeed, the lighter urines are generally those which have the larger amount of albumin, because highly albuminous urines often contain little urea.

The *sediment* is made up of variously granular casts, among which the dark granular are conspicuous by their numbers and size, and especially their width. There are also found oil-casts and casts containing entire and fragmentary epithelial cells, which are likewise granular and oily. Finally, yellow waxy casts are found. Casts vary in number, being sometimes scanty, but, as a rule, they increase with the development of the disease and grow less as it mends. Occasionally they are entirely absent for a time, even in this form of Bright's disease, sometimes as the result of treatment, when such absence may be considered a favorable sign. Sometimes, on the other hand, the tubules are choked with them, and they do not descend into the urine. Compound granule (granular fatty) cells and other forms of fatty renal cells are often numerous. Leukocytes are also often very numerous, while red corpuscles may be present and in the hemorrhagic form very numerous.

Uremia is infrequent in chronic parenchymatous as compared with acute nephritis and *contracted kidney*. It is more frequent after the stage of contraction is reached.

The Stage of Contraction.—Are there any symptoms by which we can recognize the stage of secondary contraction, which takes place sooner or later, provided the patient lives? The most reliable evidence that this has occurred is the presence of hypertrophy of the left ventricle and accentuation of the aortic second sound, although the possibility of an earlier hypertrophy cannot be denied. The increased vascular tension, mentioned as presenting itself even in acute nephritis, continues in the chronic variety to stimulate the heart to more forcible contraction, which must sooner or later result in hypertrophy. As already stated, time is required to reach this stage, and by the time hypertrophy is developed, contraction of the kidney is likely to have occurred. Long duration of the disease also affords presumptive evidence that contraction has taken place. If a case of undoubted parenchymatous nephritis continues under observation for a year or more, the process of contraction is likely to have commenced.

The *dropsy* diminishes and may disappear as the stage of contraction is entered upon. So, also, the *urine* changes in its properties. The quantity,

previously small, is increased, while the specific gravity falls below normal—1010 to 1015; the quantity of albumin is also much smaller than during the stage of inflammation. In these respects—absence of dropsy, larger amount of urine, and smaller amount of albumin—it resembles the true contracted kidney of interstitial nephritis, with which, indeed, it may be confounded in the absence of a previous history. But the casts continue to be quite numerous, and exhibit much the same character that they do in the stage of enlargement, although they too may be few, and if we have not a knowledge of previous history; the diagnosis between contraction secondary to previous enlargement and primary contraction the result of interstitial nephritis may be impossible. Uremia is more common in the stage of contraction than that of enlargement.

In the hemorrhagic form the urine almost constantly contains blood. The quantity varies somewhat and is diminished while the patient is in bed, but reappears the moment he arises.

The *duration* of chronic parenchymatous nephritis is variable, from a few months to years. Some mild forms last a long time, causing comparatively little inconvenience.

Complications.—The complications of chronic parenchymatous nephritis are the same as those of acute. Edema of the lungs, bronchitis, pneumonia, and inflammation of serous membranes are all liable to occur. Hypertrophy of the left ventricle is more common than in acute nephritis, but much less so than in interstitial nephritis. Derangements of digestion are very frequent, sometimes due to a more advanced stage of the structural changes described under acute nephritis. The acute blindness, unattended by retinal changes, described as occurring in the uremia of acute nephritis, rarely happens. Retinal changes while occurring are still uncommon as compared with interstitial nephritis, under which they will be described.

Diagnosis.—Many cases, especially, if advanced, are very easy of diagnosis. The anemia of the patient, the dropsy, the diminished urine of medium specific gravity, the usually large amounts of albumin, the numerous dark granular, and waxy casts of large diameter, free fatty cells, and fatty granular cells, especially if we are able to trace a history of long duration, all point to the disease; and if there is an antecedent history of scarlatina or exposure to cold, pregnancy, or long exposure, probability becomes certainty.

The symptoms of *amyloid* or *lardaceous kidney* very closely resemble those of the large white kidney, and it has been mentioned that the same causes are capable of developing both. It is often impossible to say which form of disease is present. It has usually been considered that if there is enlargement of the liver and spleen, or persistent diarrhea, and the cause is one which may produce lardaceous disease, it is certain that the latter condition exists; but observation has shown that the first two, at least, may be present, together with all the causes and other symptoms which are regarded as favoring lardaceous disease, and yet the disease be parenchymatous nephritis;[1] while the usual causes of lardaceous disease may operate to produce it in the liver, leaving the kidney intact. As a rule, there is not so much dropsy in lardaceous disease, casts are more scanty, and generally

[1] See an article by Paul Furbringer, "Zur Diagnose der amyloiden Entartung der Nieren," "Virchow's Archiv," Bd. lxxi., 1877, S. 400.

hyaline, granular, and waxy; hypertrophy of the heart and uremia and albuminuric retinitis do not occur. Often, too, the two forms of disease coexist, either as the result of the same cause, or the amyloid disease may be the result of long-continued parenchymatous nephritis.

Many mild cases are not easily distinguished from interstitial nephritis. The last is characterized by higher blood pressure, by hypertrophy of the left ventricle, often sclerotic arteries and polyuria, while remaining symptoms are often quite similar.

The stage of contraction is more difficult of recognition unless we have had the case for some time under observation and are able to trace its continuation with the stage of inflammation. The resemblance to the *contracted kidney* of interstitial nephritis is even closer in the third stage of parenchymatous nephritis. But here, again, the albuminuria is likely to be larger and the casts more numerous, including the numerous varieties mentioned instead of the scanty, small hyaline casts which attend interstitial nephritis. In the latter the quantity of urine exceeds the normal, while in the former, although the quantity is larger than in the stage of enlargement, it is still less copious than in true interstitial nephritis.

Prognosis.—This is unfavorable so far as recovery is concerned. Well-marked cases terminate usually within two years, and sometimes within a few months. Many cases, however, may be very much prolonged, and if they reach the stage of contraction, the patient may be tolerably comfortable for some time. But sooner or later the heart fails, the dropsy returns, and the patient dies of exhaustion, of intercurrent disease, or some one of the complications. Of the former, edema of the lungs or of the glottis and pneumonia are particularly dangerous.

Treatment.—While it occasionally happens that spontaneous recoveries from acute nephritis occur, this is not the case with the chronic form. Here the expectant plan of treatment does not suffice. As a rule the patient with chronic parenchymatous nephritis, if left alone, grows slowly worse, and although it may result in treatment may not often cure, marked improvement and may long avert the fatal end. There is always an intermediate stage between that of acute nephritis and the large white kidney, from which recovery may take place.

The chief indications in the treatment of chronic parenchymatous nephritis are two:

1. To improve the quality of the blood, which may have become anemic and further contaminated with urea, purin bodies and allied excrementitious matter.

2. To combat the symptoms and complications which form a source of great inconvenience and danger to the patient.

1. The first of these indications is chiefly fulfilled by the use of iron and strychnin, nourishing food of a suitable kind, a proper hygiene. Iron is regarded by many as almost a specific in chronic parenchymatous nephritis, and is prescribed constantly in the most reckless and thoughtless manner. *Large doses of iron should not be given.* They are useless, lock up the secretions, cause headache, and increase the danger of uremia. The well-known Basham's mixture is a great favorite. It is really a solution of acetate of iron, and, being made by adding to tincture of the chlorid of iron acetic

acid and solution of the acetate of ammonia, has the advantage of at least tending to diuresis. But the tincture of the chlorid of iron alone is an efficient preparation which is always accessible, and when combined with the sweet spirit of niter and freely diluted, is perhaps as efficient as Basham's mixture. Only a few drops should be given. To either one quinin and strychnin may be added, if desired.

With regard to *diet*, while it is true that a sufficient amount of food of good quality, is desired, those articles should be selected which contain a minimum of nitrogen. Experience has shown that when the appetite is good and large quantities of meat are eaten, uremia has been more frequent, whereas when the appetite has been bad and little food taken, uremia in chronic nephritis is uncommon. While, therefore, it is not necessary to omit all proteid food, it *is* desirable to limit it, and, while drawing upon the vegetable kingdom for food, to make up the deficiency in meats by the free use of milk. The good results of the milk treatment in cases of chronic nephritis are generally acknowledged and are evidenced by improvement in all the symptoms. From 2 to 3 quarts (2 to 3 liters) a day may be taken. The milk should not be skimmed, but, if rich may be diluted for by retaining the cream the casein is maintained in smaller proportion, while fat always suitable is in good shape to be assimilated. Rich milk is not desirable. It may be diluted with Vichy or carbonated water, or Apollinaris. It is not, of course, always necessary to confine the patient to a pure milk diet, but it should at all times constitute a large part of the food. In ordinary cases the milk may be supplemented by the softer juicy vegetables, especially by the cereals which with cream and sugar are admirably suited to such cases. Bread of all kinds not too fresh or hot is suitable and bread and milk are ideal food. An egg may be added once a day or even a small piece of meat of any kind. Fruits are eminently suitable while tea and coffee in moderation need not be excluded.

Next to diet, rest is a most useful measure in chronic nephritis, and an albuminuria reduced to a minimum while the patient is up may often be further reduced by putting him to bed. The beneficial effect of rest upon edema due to any cause is too well recognized to require other than an allusion. The advantages of rest in bed are, however, sometimes more than counterbalanced by the disadvantage to the patient of confinement and want of fresh air and outdoor life. These, of course, must be weighed, and that one adopted which serves the patient best.

Under hygienic measures is included suitable clothing. That next the body should be of wool or linen for it must be remembered, on the one hand that the skin is a powerful adjuvant to the kidney in its eliminating functions, and. on the other hand, that any interference with the action of the skin must throw more work on the kidney. Cold produces such interference, while warmth promotes the action of the skin, and no texture prevents the former or secures the latter more effectually than wool. For the same reason, while the maximum amount of fresh air is desirable, cold and dampness should be avoided or sufficiently guarded against.

[1] The more usual formula for Basham's mixture is as follows: ℞ Tinct. ferr. chlorid., f ℥ij (7.4 c.c.) Acid. acet. destillat., f ℥ij (7.4 c.c.); Liq. ammon. acetatis. f ℥iij (90 c.c.); Curacoæ vel syrupi simpl., Aquæ; aa q. s. ad j ℥vj (180 c.c.). M. et Sig. Teaspoonful or dessertspoonful twice a day, in half a tumbler of water. If the mixture becomes turbid, it is probably because some of the acetic acid has evaporated when a few more drops may be added to clear it up.

2. The second indication is to combat the symptoms and complications which cause inconvenience or jeopardize life. These symptoms are those of dropsy, effusions into the serous cavities, and congestions. The patients suffering from them are usually confined to the house, or go out of it at so great inconvenience as to make it intolerable to do so. Of dropsy, there is abundant evidence to the naked eye.

With regard to purgatives and diuretics, nothing need be added to what has already been said under acute nephritis (p. 718, 719). But as to measures which promote a decided action of the skin should be added the warm bath, the Turkish bath, warm-pack bath, and the hot-air or vapor bath already alluded to. Any of these may be used as convenience or the patient's choice may determine, while the frequency with which they should be used depends on the urgency of the case.

The "warm" is a very pleasant form of bath. The patient is wrapped up in a warm wet sheet, further enveloped in a sufficient number of blankets. A very comfortable sweat generally ensues, which is continued for an hour. In the use of the warm bath the patient is immersed at a temperature of about 104° F. (40° C.), and kept there for from half an hour to an hour. He is then removed and wrapped in blankets. Pilocarpin may be used tentatively. Some caution must be observed in the use of pilocarpin because of its tendency to produce edema of the lungs. They may be used about as often as the baths, usually on alternate days, occasionally daily, with advantage.

The judicious use of aperients is an efficient means of depleting the blood and reducing dropsy. The selection must depend on the urgency of the case, as sufficient has been said in treating of acute nephritis. But in many cases of chronic nephritis a stage is finally reached at which all treatment of the kind described fails to relieve the dropsy, which becomes eventually the sorest burden of the malady. The body becomes greatly increased in weight, the integument of the extremities is stretched almost to bursting, and sometimes it does rupture, followed by leakage, which, although in one way inconvenient, is in others a great relief to the patient. Acting upon this, physicians have long been in the habit of puncturing the swollen parts to drain away the fluid and diminish tension. It is a common practice to make a number of minute punctures with a needle or sharp-pointed bistoury, but free incisions may be made on the inner or outer side of the ankle of each leg. Free drainage is thus secured, often with great relief. Southey's tubes may be substituted at convenient places. They are introduced by means of a little trocar and after this is withdrawn fine india-rubber tubing is attached to the little cannula and carried to a suitable vessel outside the bed. Some remarkable recoveries have followed incisions. Great care should be taken to keep the tubes clean, as they are liable to become dirty and clogged.

Bearing in mind the effect of chlorid retention on renal dropsy successful treatment would naturally be favored by the elimination from the diet of articles rich in chlorids, seeking thus to reduce the chlorids ingested to two or three grams (30 to 45 grains) daily. This is accomplished by a diet including eggs, unseasoned meat, milk and unsalted butter, bread without salt, fresh-water fish, potatoes, rice, fresh vegetables, fruits and chocolate.

So too, recalling the work of Pearce alluded to, as to the effect of plethoric

hydremia on favoring edema, measures to diminish hydremia should be used. These would include iron, so popular in chronic Bright's disease in the shape of Basham's mixture; but in these cases the stomach has generally been tried to the utmost, so that the method of using iron hypodermically commends itself. The citrate of iron is commonly used. It is put up in ampules containing 3/4 grain (0.05 gram) and sodium glycerophosphat 1 1/2 grains (0.1 gram) and injected into the muscles in the arm or elsewhere every other day. It may be best at first to use only half the contents of one ampule. The treatment should be kept up some time.

The treatment of the complications is in no way different from that of the same conditions under other circumstances. The point to be impressed is the importance of being constantly on the lookout for them. Effusions into serous cavities are probably the most important. Edema of the glottis requires especial allusion, as a complication most alarming and threatening to life. Inhalations of steam may be tried, but prompt punctures or incisions are the only certain means of relieving the patient and saving life.

There are no measures directly curative in acute or chronic nephritis—that is, remedies which by their direct action remove the morbid state. All that can be done is to place the patient in a condition most favorable for nature's kidnly offices, which are always exerting themselves toward cure. This is accomplished by the measures recommended, which also eliminate the mechanical and poisonous products which interfere with recovery.

Operative Treatment.—Decapsulation of the kidney as a cure for chronic nephritis was proposed by the late George M. Edebohls, the suggestion growing out of some results of operation for floating kidney in persons who happened to have coincident chronic parenchymatous nephritis. His results were published in the "Medical News," April 22, 1899. His first thought was that the cure of Bright's disease was due to correction of the displacement of the kidney, and it was not until after three secondary operations upon kidneys which had been anchored some time previously, that he discovered the essential condition underlying the cure to be decapsulation, or decortication. The last paper of Edebohls read before the Section on Surgery and Anatomy of the American Medical Association, June 19, 1908,[1] was based on a total of 102 cases, of which 33 were claimed to be complete cures, 21 received no benefit, and 48 experienced amelioration short of cure. Of the 21 not benefited ten died soon after the operation, from the effects of the operation. In 29 cases he operated on both kidneys at one sitting. The operation consists in stripping off the capsule and cutting it away entirely, close to its junction with the pelvis of the kidney.

The success of the operation is seemingly due, as Edebohls suggests, to arterial hyperemization of the kidney, whereby an increased and adequate blood-supply is furnished which permits an absorption of interstitial and intertubular inflammatory products, thus relieving the tubules and glomeruli from the pressure previously interfering with their function. The operation should be done on certain cases. Many cases have been reported with almost immediate relief, but most of them have had fatal relapses and appar-

[1] Published in the Jour. American Medical Association, Jan. 16, 1909. The paper was read, on account of Edebohl' illness, by Samuel Lloyd. Edebohls died Aug. 8 1908.

ently all of them still have albumin and tube casts in the urine. Two of our cases died on the operation table.

CHRONIC INTERSTITIAL NEPHRITIS.

SYNONYMS.—*Contracted Kidney; Chronically Contracted Kidney; Renal Cirrhosis; Cirrhotic Kidney; Granular Degeneration; Granular Kidney; Red Granular Kidney; Gouty Kidney; Renal Sclerosis.*

Definition.—Chronic interstitial nephritis is a chronic process resulting ultimately in a shrunken kidney, in which there has been extensive destruction of the tubular substance and overgrowth of interstitial connective tissue.

Etiology.—Of the recognized forms of Bright's disease, interstitial nephritis shares with chronic parenchymatous nephritis a large number of instances in which the cause is undiscoverable. There are, however, some well-determined causes. There are certain cases of interstitial nephritis which are unquestionably the result of the exigency of our modern life. In all probability the kidney lesion is secondary to the arteriosclerosis which results from the manner of life. Anxiety; business care; worry particularly when accompanied by overwork, physical or mental; eating at irregular times of improper food or improper amounts of food, unquestionably gives rise to a large number of cases of arterio-sclerosis, accompanied by high blood-pressure. In a great many instances there is connected with this condition, a true interstitial inflammatory condition of the kidney, but the kidney condition is surely a result of the primary cause just as is the arterial condition in these cases. Nevertheless, the case finally resolves itself into one of insufficient kidney elimination and becomes literally chronic Bright's disease. *Gout* is associated with so many cases of contracted kidney that the term gouty kidney has become a well-recognized synonym for the product of interstitial nephritis. There are probably no cases of gout which have continued for any length of time which are not accompanied by interstitial nephritis. Uric acid and allied substances in the blood are probably the exciting cause. Another well-recognized cause is *lead* in lead-poisoning, the absorbed lead acting like the poison of gout. Hence painters, glaziers, workers in lead in any form, are frequent victims.

Long-continued *cystitis*, especially following gonorrhea, is a cause in a few instances, the inflammation traveling up the ureter to the pelvis of the kidney and thence to the intertubular tissue. The result of such extension may be either fibroid or suppurative nephritis.

Hereditary influence is occasionally a cause of contracted kidney. A remarkable case occurred in the practice of one of us. A man, aged 30, had granular kidneys. His father and mother both died of Bright's disease, aged 56 and 63 years. A brother aged 37. Two children of this brother when four and seven years of age, and a sister all had Bright's disease. A brother, aged 26, and a sister aged 34, have as yet exhibited no signs of Bright's disease. A maternal cousin died of undoubted Bright's disease, and other members of the family belonging to previous generations died with symptoms which suggest Bright's disease. There is no gout in the family.

Dickinson also relates the history of a family in which a hereditary albuminuria existed independent of gout.

Prolonged *passive congestion*, due to valvular heart disease, may become a cause of granular kidney. The same may be said of *stone in the kidney* causing numerous attacks of nephritic colic. Typical chronic interstitial nephritis occasionally ensued on such attacks of nephritic colic. Typical chronic interstitial nephritis occasionally ensues on such attacks of nephritic colic.

Interstitial nephritis is commonly a *disease of middle age*, the majority of persons in whom it is discovered being past 40. Under 20 it is uncommon, but studies by Heubner,[1] and Brill report 33 cases of interstitial nephritis the youngest was 18 and in Dickinson's 308 cases the youngest was 11. Cases as young as five years are reported.

It must be remembered that there is a tendency to overgrowth in the interstitial tissue of the kidney, as of other organs, in old age. Hence the term, *sensile atrophy of the kidney*. It is not safe, therefore, to call every instance of atrophied kidney met in the postmortem room a case of interstitial nephritis. The clinical history, or some one of the well-marked symptoms of the disease, as albuminuria or uremic symptoms, should have preceded to sustain the diagnosis.

As to *sex*, nearly twice as many men have the disease as women, because of the more frequent exposure of the former to the causes of the affection.

Morbid Anatomy.—In interstitial nephritis, both kidneys are involved, but there is often a marked difference in the extent of the disease in each.

Macroscopically, the organs are evidently smaller than in health, often less than half as large. Next to this reduction in size, the most striking feature of the contracted kidney is its uneven or granular surface, which is, however, not always recognizable until after the capsule is removed. Very characteristic also is the presence of cysts with more or less clear watery or gelatinous contents, often visible through the capsule. These are not invariably, but quite frequently, present. The capsule, itself thickened, strips off with difficulty, dragging portions of the secreting structure with it. Owing to the resistance which the blood meets in its passage through the kidney, a larger portion of it passes out of the organ by way of the capsule; hence the blood-vessels of the latter are dilated, as are also the lymph-spaces.

Bereft of its capsule, the kidney is hard, granular, tough, and usually darker than in health, whence one of its names, the "red granular kidney." This color is in strong contrast to the white or slightly yellow tinge of the fatty and contracting kidney, and although it is not always marked, and sometimes even substituted by a paleness, it is still easily distinguished from that of the contracting kidney of parenchymatous nephritis. The granules on the surface of the contracted kidney are distinct round and oval elevations of the surface, ranging in size from that of a pin's head to that of a pea, or from 1/25 to 1/5 inch (1 to 5 mm.). Those of smaller size are most numerous, and at first correspond with the lobules, the bases of

[1] *Brill and Libman.*—Contributions to the subjects of "Chronic Interstitial Nephritis in Children," Jour. of Experimental Medicine," vol. iv., 1899, p. 541.

which are visible on the surface of the normal organ. The larger ones result from the coalescence of two or more of the smaller. The granules themselves are of a lighter color than the depressed circlets between them, which are tinted with vascularity and have a purplish or faint red hue. The *cysts* already referred to are now more distinct (after removal of the capsule), and vary greatly in size. While equaling in minuteness the smallest of the granules, some of them are as large as a walnut. The larger are apt to be ruptures on stripping off the capsule.

On section, it is at once evident that the reduction in size of the kidney is largely due to a narrowing of the cortex, although the medulla is also contracted. The former may not be more than from 1/8 to 1/6 inch (3 or 4 mm.) in width, and exhibit every degree between this and the normal. The Malpighian bodies are smaller, less numerous, and can scarcely be detected by the naked eye, while the small arteries are more prominent from the thickening of their walls. Increased density and firmness of the organ are apparent. In a gouty subject, linear chalk-marks of sodium urate may be present, more particularly in the pyramids of straight tubules, and are contained within, as well as between, the latter. The little cysts referred to as seen on the surface may also be scattered throughout the section from cortex to papillæ, but they are more numerous in the former. They are not always present. The pelvis of the kidney may be unaltered. It is sometimes enlarged, and the calices are elongated from retraction of the pyramids. On the other hand, if the kidney is very much reduced in size, the capsule may be pursed up and proportionately smaller.

Minute Structure.—Minute examination of thin sections through the cortex clearly reveals the condition to be an excess of connective tissue, with destruction of the tubules and blood-vessels. The process is best studied if the sections include the capsular edge, as the disease progresses from without inward. In such sections may be seen extensive tracts of connective tissue separating the tubules, which, in healthy kidneys, are closely in contact without appreciable intertubular substance. The *tubules* themselves appear in places quite normal; in others they are represented by fragmentary portions in which the cells are still unchanged; in others, again, the cells exhibit a granular degeneration; some tubes are evidently dilated; others still are completely shriveled, while it is evident from the larger areas of connective tissue that many have completely disappeared. In a few tubules waxy casts are present. The Malpighian bodies are surrounded by concentric layers of nucleated connective tissue. Many of them are shriveled and atrophied, and an attempt to inject them with colored injecting fluids fails either partially or completely. Some thus altered lie detached from the tubules, with which they should be continuous. The granules on the surface of the kidneys are resolvable by the microscope into tubules, some of which are in a tolerably perfect state, some decidedly dilated.

The *cysts* originate partly in dilatations of obstructed segments of the uriniferous tubules and partly in dilated Malpighian capsules. Proof of the latter mode of origin is found in the fact that compressed capillary tufts are sometimes found lying up against one side of the wall of the cyst. The same overgrowth of connective tissue may be seen in the pyramids,

but it appears later, extends more slowly, and never reaches the degree found in the cortex.

The *blood-vessel* of the contracted kidney is the seat of important changes. In the first place, it shares with the tubules the compressing effect of the contracting new formation. As the result of this, a part of the capillary system is destroyed, and in the part thus destroyed are many capillary coils in the Malpighian bodies. Hence, as many afferent arterioles send their blood directly into the second capillary network, which is also cut down by the pressure. The vessels which remain are often sclerotic, dilated, and twisted, and in consequence of the destruction of numerous Malpighian bodies send much of their blood out through the capsule of the kidney. The intima is thickened, and the media and adventitia are invaded by hyperplastic connective tissue, but always to a less degree. Even arterioles whose walls have thus been thickened become involved in the atrophic processes affecting the glandular tissue of the organ, and ultimately disappear.

Associated with these changes are a *general arteriosclerosis* and *hypertrophy of the left ventricle* of the heart, sometimes also of the right. The final effect of these alterations is to produce a brittleness in the arteriole walls, which disposes them to rupture on very slight increase of intravascular pressure. Hence the frequent fatal termination of cases of interstitial nephritis by apoplexy, also the frequent nasal and retinal hemorrhages which characterize the disease.

The *retinal changes—retinitis albuminurica*—symptoms of which form so important a part of the symptomatology of chronic interstitial nephritis, are various and vary with the stage of each case. Many cases are first diagnosed by the ophthalmic surgeon. The changes include serous swelling of the disk and surrounding retina, hemorrhagic extravasations, dirty white splotches, representing fatty degeneration; also dilatation of the veins and capillaries, with fatty degeneration and sometimes hyaline thickening of their walls.

Symptoms.—The great obscurity as to the origin of a large majority of cases of contracted kidney is only equaled by the insidiousness of their approach. The beginning of the disease is certainly not characterized by any distinctive symptoms, and its progress is often unmarked by any, until those of uremia point to the beginning of the end. To the observing physician some obscure symptom may suggest an examination of the urine, or the peculiar tense and bounding pulse of hypertrophy of the left ventricle, or the more tangible symptoms of a slight swelling of the feet or ankles, recognizable only at night or through the unexpected tightness of a boot, may lead to the same examination. An accidental recognition of greatly *incresaed blood pressure* may suggest the disease.

Changes in the Urine.—Attention being called to the *urine*, it will be found to present characters which are more or less distinctive and lead easily to a diagnosis. When freshly passed, it is acid in reaction, copious, often exceeding the normal amount, and never scanty, except in the last stages of the disease. The quantity is often 60 (1800 c.c.), and may reach 90 ounces (2700 c.c.). The patient very commonly must rise at night, probably not more than once or twice, to void urine. There may be corresponding thirst. Consequently, the urine is light in color and of low spe-

cific gravity—1005 to 1015—and contains a trifling or moderate flocculent sediment. It is generally albuminous, but the albumin is small in amount and may be temporarily absent, or it may be absent before a meal and present after it. Later, however, the albumin becomes constant. It seldom exceeds one-tenth the bulk of fluid tested, and is very constantly a great deal less, showing a delicate line of white by Heller's nitric acid test. Tube-casts are present, but not usually numerous. They are almost solely hyaline and pale granular. Some of the hyaline casts are delicately so, requiring delicate illumination for their detection; others are distinct and sharply cut; others still contain two or three glistening oil drops. Casts may at times be absent and again reappear, as is the case with albumin. Toward the termination of cases of interstitial nephritis the urine diminishes in quantity, the specific gravity increases, and the casts become much more numerous, and include among them highly granular or dark granular and occasionally even blood-casts in addition to those mentioned, and there are sometimes a few blood-disks earlier.

As to the other symptoms, a feeling of *unaccountable weakness* or of being tired is very often present, but it is a symptom which occurs in many conditions, and should only be considered as suggestive. *Slight edema* about the feet and ankles is often present, being so slight as to escape detection, or it is discovered accidentally. When present it is significant, but it is often entirely wanting.

Hypertrophy of the left ventricle of the heart without valvular disease is so constant as to be alone suggestive of the disease. No case of interstitial nephritis has existed for any length of time without this condition supervening, and as few cases are discovered until they have existed for some time, few are found without hypertrophy. In more than one-half of cases, at least, hypertrophy is evident. It is recognized at first not so much by the resulting enlarged percussion area as by the sharp accentuation of the aortic second sound. Corresponding to this, the *pulse* is hard and resisting, indicating high tension and thickening. These two symptoms have, therefore, great diagnostic value. *Sclerosis* is distinguished from tension by obliterating the blood-current by pressure and feeling the artery beyond this point. The sclerosed vessel continues tangible; that of simple high tension disappears. A symptom of this stage is often an uncomfortable *pulsation* felt in the head and even in other parts of the body.

It is not easy to estimate the exact number of cases of pure interstitial nephritis associated with hypertrophy of the different cavities of the heart. The observations of Hasenfeld, in 1897, and von Hirsch, in 1900, found hypertrophy of all chambers in over 75 per cent. Von Buhl's results are essentially the same. Recently some attempts were made to settle this question by Nathaniel Bowditch Potter[1] and Horace Oertel, of the New York City Hospital, resulting as follows:

286 Autopsies, 113 Cases of Nephritis.

	Per cent. of Hyp. of L. V.	Per cent. of Hyp. of L. & R. V.	Per cent. of Normal or Atro. Heart.
Of 66 chronic interstitial	30.5	11.5	58
Of 22 arteriosclerotic interstitial,	27	9	64
Of 25 chronic parenchymatous,	12	4	84

[1] "Cardiac Hypertrophy as Observed in Chronic Nephritis," by Nathaniel Bowditch Potter. "Journal of the American Medical Association," October 27, 1906.

These observers did not follow Müller's accurate method or it is probable they would have found a large proportion of cases in which the left ventricle and both ventricles were enlarged. Of the cases of atrophic or normal hearts generally associated with deficient nutritive power they found a number in which there was reason to believe the heart was originally hypertrophied and became later atrophic.

An increase of blood-pressure as recognized by the sphygmomanometer is a very characteristic and diagnostic sign, the measure being anything above 160, and as high as 250 millimeters of mercury. Some observations by Dr. Robert I. Lee on Pathological Findings in Renal Hypertention published in the "Journal of the American Medical Association," vol. lvii., p. 1179, give an idea of increased blood-pressure as produced by renal disease. Dr. Lee followed to the autopsy table 53 cases in which the blood-pressure was over 160. In 38 cases or 71 per cent. renal lesions were found. In seven of the 38 cases or 13 per cent. of the whole series the lesions were essentially confined to the kidneys. Five of these seven cases showed pathologic changes classified as chronic glomerulonephritis, one as subacute glomerulonephritis and one as chronic interstitial nephritis. The blood-pressure ranged from 165 to 240 millimeters. Some kidney lesion was present at necropsy in 71 per cent. of the cases of hypertensions. Theo. C. Janeway found some kidney lesions present in 79 out of 100 cases of hypertension which came to autopsy.[1]

1. Hypertension may arise through purely quantitative reduction of kidney substance below the factor of safety.

2. Hypertension may arise in connection with the unknown intoxication which causes disturbances of the central nervous system and which we call uremia.

3. Hypertension may arise in primary irritability of the vasoconstricting mechanism from unknown, probably extrarenal causes, which lead eventually to arteriolar solerosis. In this type the disease in the kidney is the sequence, not the cause, of the generalized vascular lesion.

As the disease becomes more advanced there are added *cardiac symptoms*, including dyspnea, palpitation, and reduplication of the first sound. The last is probably due to a want of synchronism in the systole of the two ventricles. There is usually no murmur, because there is no valvular disease. The latter may be present. The patient may have had valvular disease prior to the renal malady, or the latter itself, by its long continuance, may have produced endocarditis and atheroma with an aortic systolic murmur. There may be a mitral murmur due to relative insufficiency. Valvular disease is, however, unusual. The hypertrophy of the heart is conservative, and all goes well as long as the power of the heart lasts. When the latter begins to fail and dilatation appears, the blood-pressure diminishes, and with it begins a train of symptoms, among which diminished secretion of urine and dropsy are the most conspicuous, along with gallop rhythm, dyspnea, palpitation, and dizziness. These symptoms may again be averted for a time by *hypertrophy* of the *right ventricle*, which is a further effort to correct disturbed compensation. Among derangements of breathing must

[1] While printing this edition Dr. Janeway's latest paper on Nephritic Hypertension has appeared in the "American Journal of the Medical Sciences" for May, 1913. He concludes that in the main the hypertrophied heart may be looked upon as the result of a persistent high blood-pressure.

be included Cheyne-Stokes breathing, commonly toward the end of the disease.

Dimness of vision due to retinitis albuminurica, already described on page 702 is a characteristic symptom. It is often the first recognized, and hence the diagnosis is frequently first made by the ophthalmologist. It is a sign of advanced disease. Some assign two years as the limit of life after its recognition, but this is too unfavorable a prognosis. The atheroma of the blood-vessels is the cause of another symptom which frequently determines the mode of death—rupture of a blood-vessel in the brain: in a word, *apoplexy*. This accident is more usual late in life, but Dickinson reports a case in which cerebral hemorrhage occurred in a girl of 12. The proportion of cases of recognized interstitial nephritis in which this happens is not large, but many cases of apoplexy are directly traceable at autopsy to unsuspected renal cirrhosis. Dickinson believes that of fatal cases of apoplexy, one-half are preceded by this form of disease. Hemorrhages in other situations are referable to this same altered state of the blood-vessels, as, for example, into the retina, from the nose, and even into the stomach. *Sudden blindness*, in addition to the dimness of vision due to retinitis albuminurica, is a symptom which occasionally presents itself. Amaurosis and amblyopia also occur, and may disappear, but dimness of vision due to retinitis albuminurica is a permanent symptom, though I have seen it improve under treatment. Auditory disturbances also occur, such as ringing in the ears, with dizziness and more or less deafness.

The termination by *uremia* occurs more frequently in this than in any other form of Bright's disease. Headache, drowsiness, convulsions, stupor, delirium, maniacal excitement, renal asthma, *restlessness*, nausea, vomiting—any one of these symptoms may usher in the dreadful train which is so likely to be fatal. E. C. Seguin (in 1880) especially called attention to occipital headache as a symptom of uremia.[1] Von Leube considers that even the intermittent headaches which occur in this disease, and which very closely resemble migraine, are probably due to uremia. Occipital headache occurs too frequently unassociated with any symptoms of nephritis to justify attaching much importance to it as a symptoms of uremia. Another form of headache characterized as uremic is a brow headache passing off at 10 or 11 in the morning. Temperature follows the same rule in uremia as in other forms of nephritis. The *convulsion* is commonly associated with a rise of temperature. *Dyspeptic symptoms*, with obstinate vomiting, particularly in the morning on rising, are apt to usher in a chronic uremia. *Diarrhea* is less common, but also sometimes occurs toward the close, when it may be very difficult to control.

The duration of this form of renal disease is indefinite. Always a chronic process, it may last for years undiscovered, and when discovered before it is too far advanced, the knowledge of its presence will suggest measures of precaution and treatment which may so prolong life that it need only be determined by its natural limit or some other disease. Yet complete recovery from well-established interstitial nephritis is unknown.

Complications.—These include bronchitis, pericarditis, pleurisy, pneumonia, and, more rarely, endocarditis, peritonitis, intertubular gastritis,

[1] "Archives of Medicine," vol. iv., No. 1, New York, August, 1880

and even inflammation and ulceration of the bowels. But all inflammatory complications, except bronchitis, pleurisy, and pericarditis, are less frequent than in acute nephritis. My own experience accords more nearly with the latter. Pericarditis is the most serious complication occurring. Pleurisy and pneumonia are also of tolerably frequent, Stewart found the former in 15 per cent. of his cases and pneumonia in 7 per cent. Acute endocarditis and peritonitis occur very seldom.

Diagnosis.—The diagnosis of an interstitial nephritis is usually easy, if in any way an examination of the urine is suggested. The increased quantity, the low specific gravity, small albuminuria, delicate hyaline, pale granular casts, and hypertrophy of the left ventricle, even in the absence of other symptoms, are sufficiently distinctive. The conditions which should suggest such an examination are a feeling of constant weariness, slight swelling of the feet, drowsiness, frequent headaches, confused intellect, dyspeptic symptoms, obstinate nausea, delirium, coma, and convulsions. High arterial tension should always suggest examination of the urine. It must be remembered, however, that albumin and casts may be present in the urine of individuals with high blood pressure without the renal lesion they suggest being the important one in the case.

The special condition from which it is most difficult to distinguish it is the milder form of *chronic diffuse* or *parenchymatous nephritis*, especially if the latter has reached the contracting stage. In fact, the symptoms are often identical, and unless the history helps us, it may be impossible to decide. The evidences of decided fatty change, such as the oil-cast or free fatty renal cell in the urine, settle the question in favor of chronic diffuse nephritis.

Assistance in the diagnosis between these two conditions may be had from the phenolphthalein test. The application of this has shown that in contracted kidney insufficiency of function is much more marked than in parenchymatous nephritis.

It is important, if possible, to distinguish between interstitial nephritis with secondary arterial sclerosis and *general arterial sclerosis with secondary contracted kidney*, but it is not always easy to draw a sharp line between these two conditions. Both are insidious, in both there is the absence of dropsy except in the very last stages of primary renal sclerosis, though here it is also rare, while it is wanting throughout in general arterio-sclerosis. In both there is a scanty albuminuria with very few hyaline casts, but if the opportunity presents to study urine for a time, it may be found that the albumin and casts appear earlier in the primary renal cases. Indeed, in general arterio-sclerosis there is often no albuminuria whatever, even though hyaline casts are present. In general arterio-sclerosis there are often brain symptoms (*simulated uremia*) due to anemia and imperfect circulation, namely, vertigo, tinnitus and amaurosis. In the latter condition retinal changes occur early, but they are not those of retinitis albuminurica and hemorrhage into the retina. As pointed out by de Schweinitz, there is thickening of the arteries which compress the veins where they cross them. These changes may occur early. Retinitis albuminurica, hemorrhages, and more serious derangements of vision occur only when the kidney lesion is primary. An important symptom characteristic of general arterio-sclerosis is a tendency to emaciation and loss of weight, a change of color, a pallor, and

loss of vigor which is characteristic. The condition of the arteries interferes with an adequate nutritive supply and the patient wastes as well as grows weaker and anemic. Perhaps the most characteristic difference is found in the blood-pressure. In general arterio-sclerosis blood-pressure may be increased, but not nearly as much as in renal sclerosis where the systolic pressure is often 170 to 220 mm. as contrasted with an average normal of 115 to 140 mm. An important etiologico-pathological difference is claimed by Gull and Sutton for the kidneys. According to them, the destruction of renal tubules is due to the pressure of new connective tissue itself resulting from the arterial changes, whereas in primary contracted kidney the renal parenchyma dies first and is replaced by connective tissue secondarily.

We have attempted to tabulate these differences between the two conditions as follows:

Primary Chronic Interstitial Nephritis.	*Primary General Arterio-sclerosis.*
1. Causes of chronic interstitial nephritis, such as overeating and drinking, gout, diabetes, syphilis, lead intoxication, etc.	1. Same causes.
2. Characteristic insidious onset, including digestive derangements, small albuminuria, few casts, with little or no evidence of arterial change at first.	2. Early appearance of arterial changes.
3. Edema, never at first, later rare.	3. Same.
4. Arterial pulsation often very annoying.	4. No pulsation in head or elsewhere.
5. Vertigo infrequent.	5. Vertigo common.
6. Albuminuric retinitis and hemorrhages into retina.	6. Retinal changes, but not hemorrhage, nor retinitis albuminurica.
7. Hypertrophy of one or both ventricles rather more frequent.	7. Rather less frequent.
8. High blood-pressure and high arterial tension before vascular change, is evident.	8. Moderate or lowered blood-pressure, moderate arterial tension.
9. True uremia.	9. Simulated uremia, due to changes in the circulation in the cortex.

Strictly speaking, all nephritis is primarily parenchymatous or tubular, but in what is known as chronic interstitial nephritis the changes are slower and the replacement by connective tissue more prompt and rapid, so that the interstitial element predominates and the cells are destroyed more rapidly.

Diabetes insipidus is comparable to contracted kidney in the increased quantity of urine of low specific gravity, but there is no albumin, casts are absent, and the urine is much more copious.

Prognosis.—The prognosis is unfavorable as to recovery, but favorable as to prolongation of life if the diagnosis be made sufficiently early. Cases with casts and small albuminuria may continue under observation for many years. If the diagnosis be delayed until the onset of uremic symptoms, little can be expected. But even at this stage, energetic treatment may still avert the immediate danger and prolong the patient's life. The possible sudden occurrence of convulsions and coma, and of death therefrom, should always be remembered and impressed upon the relatives of the patient. These constitute unfavorable symptoms, to which, toward the end, Cheyne-Stokes breathing may be added. It is important to remember that many of

the mild cases in which for years the symptoms are so little pronounced that it is hard to believe there is anything wrong, terminate in apoplexy.

Treatment.—From what has been said under prognosis, it is evident that the most hopeful result to be expected from treatment is the protection of the patient from the consequences of his malady, rather than the restoration of the kidney to its normal condition. Our power in the former respect depends largely upon the stage at which the disease is discovered. If detected at a period in which the urine is abundant, the albuminuria small, the casts few, and there is no edema, the indications are:

1. To maintain the integrity of the blood, by preventing the accumulation of toxins in the blood.

2. To treat, as they arise, the accidents and complications which are often so dangerous to the patient.

The first of these is best accomplished by dietetic and hygienic measures, aided by the use of a few remedies. First, as to *food*, all that was said under chronic parenchymatous nephritis is applicable to interstitial nephritis, because the appetite is still good, and a suitable selection can be exercised. As the toxic agents, whatever they are, have their chief source in the protein elements of food, it is plain that the larger the quantity of such food consumed, the larger is the accumulation of toxins. Now, while it is not possible nor, perhaps, desirable to exclude all nitrogenous food, it may be largely reduced. This is accomplished by the substitution of all or a part of animal flesh by milk and vegetables. On such a system the patient with contracted kidney may maintain apparently perfect health for many years.

Alcoholic beverages should be prohibited. In those, however, who have been habitual drinkers, the reduction must be slow and it may be necessary to continue small amounts of alcohol.

What has been said of clothing, fresh air, and exercise in connection with chronic parenchymatous nephritis is even more applicable to interstitial nephritis. Warmth of the body, maintained by woolen or linen garments next the skin to encourage its action, and the avoidance of damp and cold, which check it, are peremptory. The wetting of the body by rain, or of the feet alone, has frequently been the exciting cause of a fatal uremic attack. Heavy shoes should be worn in damp weather.

In this connection, sea-bathing requires mention. It is well known that sea-bathing sometimes induces albuminuria in normally constituted persons, or, at least, in individuals at other times free from albuminuria. This is probably due to a temporary congestion of the kidney, from introversion of the blood kept up by the duration of the bath. Still more mischievous, therefore, must be the effect of prolonged sea-bathing upon one whose kidneys are already damaged and incompetent to perform their office. Sea-bathing, therefore, or any form of cold bathing, should be interdicted to the patient with contracted kidney, or, indeed, with any form of chronic nephritis. Sea-bathing is especially mentioned because it is considered healthful, and persons remain in the water so long at a time. On the other hand, a daily warm bath at bedtime, and especially an occasional Turkish bath, is advantageous.

For the same reason residence in a warm, equable climate is often of sig-

nal service in interstitial nephritis; and cases are reported in which the albumin has disappeared and symptomatic recovery taken place during such residence.

Prolonged bodily or mental fatigue should also be avoided by these patients, as they have been known to be the exciting cause of uremia and death; especially are they so when associated with free eating and drinking. The patient should live a life as easy and as free from any of these influences as his circumstances will permit.

As to drugs, they are of limited utility. The moderate use of *tonics*, including strychinin, and iron, is useful to combat the tendency to anemia and weakness, which sooner or later follows. In this form of Bright's disease even more than in chronic parenchymatous nephritis, is the indiscriminate use of iron to be guarded against. Iron in contracted kidney, as often used, is a harmful drug. It locks up secretions, causes headache, and increases the danger of uremia. Only when there is evident anemia, as shown by blood examinations, should it be used, and then only in very small doses. Elimination is favored by stimulating the secretion of the skin, and this is best accomplished by an occasional warm bath, or, expecially, a Turkish bath, with thorough friction and protection from cold by woolen underclothing. The Turkish bath is an admirable remedial measure, especially before the disease is too far advanced.

Diuretics are not indicated in the earlier stages, because the secretion of urine is already free. The bowels should be kept regular by the use of the natural aperient waters, the Hunyadi, Friedrichshalle, Apenta, Veronica and Rakoczy, or an occasional blue pill, or a dose of magnesium sulphate. Of course, later in the disease, when the heart begins to fail and the urine is scanty, both diuretics and purgatives are indicated. The same principles are to govern us in using them as have already been laid down under acute nephritis. Very high arterial tension sometimes demands treatment. A certain amount is a result of the conservative train of symptoms, beginning with hypertrophy of the left ventricle, and is necessary; but when a resulting throbbing is unpleasantly, appreciable, especially if there is throbbing headache with flashes of light at each pulsation, tension should be lowered. However, this must be done with caution and with rest and hygiene rather than with drugs.

Nitroglycerine is of little value unless given in huge doses. Aconite or veratrum viride may be used in the cases where the blood pressure itself is the cause of symptoms and needs lowering.

The second indication mentioned, the *treatment of the complications* and *accidents* incident to the condition, resolves itself into the treatment of the bronchitis, the pericarditis, the pleurisy, pneumonia, endocarditis, gastric and intestinal disorders, which have been named as occurring, and especially of the most serious calamity of all, uremia. The treatment of the complications is that of the same conditions under other circumstances. Paracentesis is a measure which is often of signal service in effusions into the chest, and occasionally of the pericardium.

Dyspeptic symptoms are best treated by diet, regulation of exercise and the use of nux vomica and one of the mineral acids. Hypnotic sedative, and antispasmodic effects, when desired, should be produced by spulphonal,

trional, veronal, chloral, and bromids. Opium may be used tentatively. It is not as dangerous as is constantly thought to be the fact.

Finally, as to the treatment of uremia, the measures described in the treatment of uremia in acute nephritis are to be used. Apoplexy, which is not an infrequent termination of the disease, in consequence of the atheromatous state of the blood-vessel walls, is recognizable by the paralysis, general or partial—most frequently hemiplegia—which accompanies the unconsciousness. Remedies are here generally futile, but such may be used as are indicated for apoplexy. The upright position, bleeding, and, if the patient survives the immediate accident, nitro-glycerine, iodid of potassium, with a view to promoting absorption of the extravasated clot, may be used. Hemorrhages in other situations, as from the nose or alimentary canal, are treated by the same measures as when they occur under other circumstances. The close resemblance at times of the symptoms of uremia to those of apoplexy should be remembered.

As to special treatment, or treatment directed to the removal of the interstitial overgrowth in the kidney, there is none. Theoretically, the iodid of potassium ought to be of service. Unfortunately, the peculiar requirements of its administration—viz., the length of time during which the patient must take the remedy before any results may be expected, and the consequent difficulty in accumulating a sufficient number of cases—are such that it is almost impossible to determine whether it can be of any service or not. Owing to these difficulties, it is doubtful whether its exact possibilities have as yet been determined. There can be no disadvantage in administering it if the dose is so small as not to derange the stomach. Very rarely can more than a few grains daily be given. Bichlorid of mercury, in long-continued use in doses of, at first, 1/24 grain (0.0027 gm.), and later 1/50 grain (0.0013 gm.), kept up a long time, followed by improvement. Improvement in the imparied vision of alubuminuric retinitis follows its use. Certainly in the event of a clear syphilitic origin, the iodid of potassium should be used.

Operative treatment by decapsulation has been applied in contracted kidney as well as in chronic parenchymatous nephritis. Indeed, some believe it more indicated in chronic interstitial nephritis. Our own experience would indicate that it is more likely to be successful in chronic diffuse nephritis. See treatment of chronic parenchymatous nephritis, p. 730.

Serum Treatment.—So far the serum treatment of Bright's disease, some efforts made abroad by Dieulafoy, Renault, Vitzone, and others have not resulted in its extensive use. The sterilzied serum and defibrinated blood of the goat have been used and even extracts of the kidney of pigs. Fifteen c.c. of sterilized serum were injected into the subcutaneous tissue of the abdominal walls, it is said, with striking results in one or two cases.

AMYLOID DISEASE OF THE KIDNEY.

SYNONYMS.—*Lardaceous Disease; Albuminoid Disease; Waxy Kidney; Depurative Disease.*

Definition.—A morbid state of the kidney in which its structural elements are more or less infiltrated with a substance of albuminous composi-

tion and the luster of bacon, best recognized by the deep mahogany red color it strikes when treated with a solution of iodin. Osler rightly says, "it has no claim to be regarded as one of the varieties of Bright's disease, because it is generally a part of a wide-spread amyloid degeneration."

Etiology.—The most frequent cause of lardaceous disease is profuse and long-continued suppuration, such as occurs in chronic bone disease, whether tuberculous, syphilitic, or traumatic in origin, or such discharge as constitutes the expectoration in cases of chronic phthisis and chronic bronchitis with bronchiectasis. Syphilis itself, independently of the tertiary conditions which it produces, is a frequent cause of lardaceous disease. Cachectic states of any kind, chronic dysentery, ulceration of the bowels, and chronic albuminuria are possible causes.

Either sex is equally subject to lardaceous disease, but as men are more frequently exposed to its causes, it is in them rather more common. Very young children are rarely affected, for evident reasons, but in young persons from 11 to 30 it is most frequent. After 30 it grows gradually rarer. Tuberculous hip disease in children, especially, is a cause.

Morbid Anatomy.—The incipient stages seldom present alterations recognizable by the naked eye, unaided by reagents. But if, after section of the kidney, the cortex be treated by a solution of iodin and iodid of potassium,[1] numerous mahogany-red points make their appearance, or if by a solution of violet anilin, as many red or pink points these are the Malpighian bodies, whose capillary tufts are the first to be affected by the change. The kidney, in this early stage, is normal in size or very slightly enlarged. Its capsule strips off readily, revealing no changes, or a paleness or translucency which readily escapes notice, but may be recognized at the edges of a thin section. Very often, too, they are completely overshadowed by other changes, for amyloid kidney is most frequently a superadded event in the course of chronic diffuse nephritis while the same may happen in interstitial nephritis. The large white kidney of chronic parenchymatous nephritis is especially apt to exhibit a slight degree of lardaceous change, which may altogether escape notice unless iodin is used. Hence, *iodin should be tried upon all kidneys* removed at autopsy.

In a more advanced stage of uncomplicated lardaceous change the kidneys are both enlarged, usually symmetrically, but the extreme degrees of enlargement are commonly associated with fatty epithelium. Such organs were a pair weighing 23 ounces (715 gm.) which came under Dickinson's[2] notice. Johnson[3] refers to a case in which the two kidneys weighed 28 ounces (870 gm.). Rindfleisch[4] has seen a single instance of that very rare condition, complete lardaceous infiltration—including the basement mem brane of the uriniferous tubes, as well as the capillaries, the kidney being enlarged to nearly twice its normal size. In the simple forms of lardaceous disease the capsule is not adherent, but if interstitial changes coexist to any extent, it is adherent. The surface of the kidney is pale and anemic;

[1] *The Iodin Test Solutions.*—The best test solution for macroscopic purposes is one made by dissolving 2 1/2 grains (0.16 gm.) of iodin by the aid of five grains (0.32 gm.) of iodid of potassium in one fluidounce (30 c.c.) of water. The solution contains about one-half of one per cent. of iodin. For microscopic preparations a solution weaker than the foregoing, or a one-quarter of one per cent., of iodin dissolved by twice the quantity of iodin of potassium, is more suitable, and sometimes a solution containing as much iodin as water alone will take up answers best.

[2] Dickinson, *op. cit.*, p. 249.

[3] Johnson, *op. cit.*, p. 104.

[4] Rindfleisch, "Path. Histology," "New Syd. Soc. Trans.," 1873, vol. ii., p. 167.

occasionally, the stellate veins are conspicuous. The characteristic translucency may even be recognized in the organ in bulk, but it is more striking in sections. When the change is present in high degree, the edges of a thin section are almost as translucent as a similar section of bacon. On laying open the kidney the cortex is seen to be enlarged; it is pale, anemic, waxy, firm and resisting. The pyramids are normal in hue and area. The iodin solution added to such a kidney produces its peculiar coloration not merely in the Malpighian capillaries, but also in the afferent and efferent vessels and the vasa recta of the pyramids. In a still later stage, that of atrophy, the kidney becomes contracted, rough, and even distorted in shape. The capsule is adherent, and on section the cortex is found narrowed, sometimes as much so as in the contracted kidney of interstitial nephritis.

Minute Changes.—To microscopic examination in the first stage, the Malpighian bodies exhibit a lustrous or waxy appearance. They are enlarged and the capillary walls thickened. At this stage there is no visible alteration in the tubules or in their epithelium. In the second stage larger vessels are involved, the vasa afferentia and efferentia in the cortex; the vasa recta of the cones; also the second capillary network of the cortex, while an exudation occurs into the tubules of a glistening material which may form casts. Such casts sometimes strike the mahogany red reaction. At other times they have the composition of ordinary hyaline casts. It is to be remembered, however, that similar waxy casts are found in the tubules in other forms of chronic and even acute renal disease.

The arteriole walls are thickened by involvement of both interna and media. This thickening is attended by an extraordinary distinctness of the muscular fiber-cells of the circular coat. Later, the basement membrane and epithelial lining may be invaded, the cells swollen, translucent, and apparently fused. It is also quite usual for the epithelium of the cells to be fatty, and the capillary walls to contain aggregations of fat drops, while the urine in the later stages may contain oil casts and fatty cells.

In what has been called the third or contracting stage of lardaceous kidney, but which may be the ordinary contracted kidney on which the amyloid change has been grafted, minute examination reveals, in addition to the appearances described, hypernucleated intertubal overgrowth. Cysts are occasionally present for the same reason as in the granular contracted kidney; in like manner superficial granulations.

Symptoms.—One who has had syphilis, or who has phthisis, bone-necrosis, or other affection causing an exhaustive drain, may acquire this form of kidney disease without appreciable addition to his symptoms. A marked cachexia may be present. The albuminuria may be larger than that of a chronic nephritis. There may be numerous casts of every variety except epithelial and blood-casts, including waxy casts which are, however, not distinctive. Uremia is rare.

Senator announced some years ago that *serum globulin* is increased in the urine of amyloid kidney. Hypertrophy of the left ventricle or high arterial tension are not conspicuous.

But lardaceous disease of the kidney almost never occurs alone. It is always accompanied by similar changes in the liver, spleen, and often of the intestinal canal. Hence, evidences of alterations in these organs are more

or less marked. Thus, the percussion areas of the liver and spleen are almost always enlarged, and the blood-vessels of the stomach and intestines are often involved. In the former event obstinate vomiting, and the latter equally obstinate diarrhea, results. The latter is far more frequent than the former.

As to *duration*, the disease generally runs a very chronic course, which is limited only by the malady of which it is a complication. As such it is always of shorter duration than interstitial nephritis, and may be shorter than chronic parenchymatous nephritis, although the latter affection and lardaceous disease more closely resemble each other in respect to duration. When obstinate diarrhea and vomiting supervene, the end is usually not remote.

Diagnosis.—There are some instances in which lardaceous disease is easily recognized. If a patient has had syphilis with secondary and tertiary symptoms, or has long been a victim to phthisis, and he is discovered to be edematous and to have a large albuminuria, with an increased amount of serum globulin, with waxy hyaline and fatty casts and an enlarged liver and spleen and obstinate diarrhea, there can be little doubt but that there is lardaceous disease. But when neither of these two general diseases is present, or the phthisis has not existed a very long time, or there is not decided evidence of enlarged liver and spleen, we cannot be certain. While it is never safe to diagnose lardaceous disease without the presence of enlarged liver and spleen, such enlargement on the other hand, even when associated with large albuminuria does not necessarily imply amyloid kidney. The symptoms and course of the disease, particularly in its latter stages, are so like those of *chronic parenchymatous nephritis* that it is often impossible to separate the two. Further, there is every reason to believe that chronic nephritis is sometimes caused by the same dyscrasic conditions as produce the lardaceous disease. In such cases, too, therefore, a diagnosis is impossible. Finally, the two conditions may exist jointly.

Amyloid kidney is hardly likely to be confounded with chronic interstitial nephritis as each has distinctive signs, apart from those common to both.

Prognosis.—In prognosis much depends upon the presence or absence of the original disease causing the amyloid change. If the former cannot be cured, the latter is not likely to be. If the original disease is curable and the patient young, there are no limits to the possibilities, although it is scarcely likely that the kidney is ever restored to its normal state. Complete restoration of function is, however, possible. If the patient be past middle life, even if the original disease has disappeared, recovery is less likely and if the blood-vessels of the stomach and intestines, as attested by vomiting and diarrhea, are invaded, the disease is rapidly fatal.

Treatment.—Of the lardaceous disease it may be said with greater emphasis than of any other form of renal disease, "an ounce of prevention is worh a pound of cure." A due appreciation by surgeons and syphilographers of the causes of amyloid kidney would prevent the occurrence of many cases, the timely amputation of a limb long the seat of suppuration and the thorough treatment of syphilis being often all that is needed. To this end also frequent examinations of the urine should be made by those in charge of suppurating diseases, while albuminuria should be the signal for prompt

interference, if such be possible. Especially they who are watchful should be in charge of children with hip-disease.

In syphilis the faithful and persistent use of remedies for a sufficient time after all the symptoms of the primary and secondary stages have disappeared is essential. See treatment of syphilis.

If the cause continues to exist, the treatment of the amyloid disease is the treatment of the former—if it be syphilis, iodid of potassium, mercurials and salvarsan; if phthsis, cod-liver oil, iron, creasote and creasotal, quinin, an abundance of nourishing food, in which milk and cream should be conspicuous, alcohol, and restorative measures generally, together with fresh air and suitable exercise. Supposing the original disease to have disappeared, the treatment indicated is that of chronic parenchymatous nephritis, for the details of which the reader is referred to the section on that disease.

SUPPURATIVE INTERSTITIAL NEPHRITIS AND PYELONEPHRITIS.

SYNONYMS.—*Septic and Pyemic Nephritis; Interstitial Suppurative Nephritis; Surgical Kidney; Abscess of the Kidney.*

Definition.—Suppurative nephritis, due to invasion of the kidney or its pelvis by pathogenic bacteria, either by way of the circulation or the urinary tract. A milder nonsuppurative grade of this disease, characterized by cicatricial-like markings on the capsule of the kidney and sometimes by firm adhesions between the capsule and its fatty surroundings, may be named *capsulitis or perinephritis.*

Etiology.—Often this form of nephritis starts in the pelvis of the kidney as a pyelitis, and thence extends into the interstitial tissue of the organ. Such a condition is preeminently a *pyelo*nephritis. It may also start in the interstitial tissue of the substance of the organ as the result of infectious embolism or traumatism or obstruction of the tubules by concretions. Late studies have shown that infection from the systemic circulation by pathogenic organisms is more frequent than supposed. This is especially true of tuberculous abscess. The process may be limited to the kidney, its pelvis or may ultimately invade both. It is often impossible to separate the two conditions in diagnosis, and I do not, therefore, separate the two diseases, that is processes in the kidney from those of its pelvis.

A frequent contagium bearer is *retained* decomposed urine. Retention may be due to stricture of the urethra or even phimosis, to stone in the bladder or ureter or pelvis of the kidney. Perhaps there are always bacteria ready to avail themselves of favorable conditions, but a favorite route of introduction is by unclean catheters. In many of these cases inflammation of the bladder is an intermediate state. Calculous concretions in the substance of the kidney also furnish conditions favorable for the action of bacteria of suppuration.

Infectious emboli cause a small number of cases of suppurative nephritis. The emboli are usually derived from the valves of the heart in cases of ulcerative endocarditis, but they may also arise in putrid wounds, stumps, or other seats of putrid inflammation. The abscesses found in the kidney in

common with other organs in pyemia are thus produced. *Tubercle bacilli* are also causes, entering by either of the routes named, producing tuberculous pyelonephritis. Among the organisms found in the urine and held responsible are, besides the tubercle bacillus, the *bacterium coli commune*, the *proteus Hauser*, the streptococcus and staphylococcus. *Parturition* is not an infrequent medium of introduction of pathogenic bacteria, while the infectious fevers are recognized causes.

Traumatic agencies such as blows, kicks, or penetrating wounds in the neighborhood of the kidney, or falls from a distince and striking upon the sharp edge of a fence or similar object, may also cause suppurative nephritis.

Suppurative nephritis may occur at any age subject to the operation of the cause. The youngest patient I ever had was two years old.

Morbid Anatomy.—The appearances vary necessarily with the stage of the disease and also somewhat with the cause. In an earlier stage, if the inflammation pass from below upward, as is most frequently the case, the mucous membrane of the pelvis is first affected, being swollen and dirty gray in color, sometimes visibly congested. Later, the pelvis and calices may be dilated and the papillæ flattened. The distention may go on at the expense of the kidney until the whole organ is converted into a *pus-sac* bounded by a varying remnant of renal tissue. Such sac may be a constant source of pus, or if complete obstruction occurs, the pus may become inspissated and *cheesy*. The ureter is also often dilated, sometimes resembling, in consequence of such extreme dilatation, the intestine.

In tuberculosis extending *via* the urinary tract the *apices* of the cones are also invaded, it may be from the mucous membrane by continuity, or by direct lodgment of the bacillus. Successive portions of the kidney substance break down, and the ultimate product will be the same, a sac filled with liquid pus or *cheesy, putty-like substance*.

In other instances, especially when the kidney is invaded by way of the vascular or lymphatic system, as in *pyemic abscess*, foci of suppuration a millimeter and upward in diameter are scattered in the cortex and separated by sound renal tissue. They are surrounded by an intensely red border, are often visible through the cortex, and may be ruptured by dragging off the capsule. On section at an early stage, linear streaks of pus may be found in the medulla.

At a later stage these little collections of pus unite to form larger ones, these again to form others still larger, destroying the tubular structure of the kidney as they encroach upon it, and it is at this stage that cases of pyelonephritis not infrequently terminate unfavorably and the specimens come under observation. At first each of the abscesses thus formed is confined to the region of a single pyramid, and it not infrequently happens that a kidney is partitioned off into spaces corresponding with these. Before this occurs, however, the abscess bursts through the papilla and calyx into the pelvis of the kidney. Thus, in an opposite direction from that first described, the kidney may again be converted into a purulent sac.

When the *abscess is embolic* in origin, its seat is at first occupied by an area of intense hyperemia, resulting in *hemorrhagic extravasation*, which takes place also into the tubules, causing bloody urine. To this succeeds suppuration. The size and number of the abscesses depend upon that of

the plug obstructing the blood-vessels, which is usually one of the interlobular arteries or a vas afferens. The embolic abscesses may also be multiple, in consequence of the breaking of the embolus into a number of minute fragments. When the cause is *traumatic*, the process is not so easily defined. Circumscribed abscesses may occur, or the kidney may be converted into a soft, pulpy mass, a mixture of pus, blood, and broken-down renal substance.

In the variety described as *capsulitis*, cicatricial markings or adhesions to adjacent tissue constitute its morbid anatomy.

Symptoms.—The symptoms of this condition are not numerous and, apart from the characters of the urine, are not very distinctive, while the urine often fails to furnish any information. In milder degrees of pelvic inflammation before the kidney is invaded there may be no symptoms. *Pain* and *tenderness* are the constant, but considerable inroads may be made before pain results. On the other hand, it is often very severe, while the *tenderness* over the region of the kidney is pronounced. *This tenderness is the most distinctive and valuable symptom.* Usually the severest pain is in the renal region, whence it radiates toward the front of the abdomen and groin, and may be accompanied by retraction of the testicle. When the condition is the result of impacted calculus, the seat of the impaction is the primary seat of pain. It may be between the umbilicus and the pubis when the stone is low down in the ureter. The pain is always intermittent to a degree, sometimes totally so, but generally it is more or less constant, increased paroxysmally. Various positions are assumed by the patient with a view to easing the pain, among which lying on the face is not infrequent.

A distinct *tumor* may sometimes be discovered by palpation and percussion in the region of the kidney. This implies an enlargement of the organ, due either to its conversion into a purulent sac, or an augmentation of its size owing to the distention of its pelvis with pus or calculi or both. Very frequently it is due to perinephric invasion.

Fever is also a remittent symptom. Possibly in a very few latent cases it may be altogether absent, but except in these there is always elevation of temperature, with corresponding frequency of the pulse. These latter at times become decided, and in advanced stages the fever is septic, being followed by profuse *sweats*. In acute cases, especially pyemic, the beginning of suppuration is often marked by a *chill* and *high fever* or succession of chills, but in other instances it is quite impossible to recognize the beginning of the suppurative stage.

The *characters of the urine* may be *nil* or, as intimated, quite distinctive. Except in acute infectious cases, the urine almost invariably sooner or later contains pus, and unless it does contain pus, no certain diagnosis can be made. *Blood* is also a very constant constituent from cases of suppurative nephritis, but while such urine is scarcely ever examined by the microscope without discovering a few blood disks, the quantity is often not large enough to be recognizable to the naked eye. The quantity of *pus* varies greatly. While it may be so copious as to produce a heavy white opaque deposit one-sixth to one-fifth the bulk of urine, it may be represented by little more than a trace. This variation will occur at different times in the same case. Pus from the kidney and its pelvis is usually distinguished from that formed in the bladder by the absence of that glariness so characteristic of the latter, due to admix-

ture with mucus and decomposition products. Pus from the pelvis of the kidney is rarely fetid, as compared with pus from the bladder.

The urine is usually *diminished in quantity*. Complete suppression is not uncommon toward the close of extreme cases. Notwithstanding such diminution, the color may be pale and the specific gravity low, owing to the small proportion of solids; the range of specific gravity in a single case being from 1003 to 1016. In reaction the urine is faintly acid, neutral, or alkaline, and though often prone to rapid decomposition, is less so than the urine of cystitis. *It is always albuminous*, but the quantity of albumin is never very large, and varying generally *pari passu* with the quantity of pus and blood. When more albumin is present than is thus accounted for, it is likely that the parenchyma of the kidney in general has become involved. Such cases, are, therefore, more serious. In such cases, too, *tube-casts*, commonly rare in suppurative nephritis, may appear.

It sometimes happens that there is a sudden increase in the quantity of pus in the urine, followed by a gradual diminution, or the urine, previously clear, may suddenly become loaded with pus. Such occurrences indicate the removal of a temporary obstruction to the descent of the pus, or the rupture of an abscess from the kidney into its pelvis. It is barely possible that small portions of the substance of the kidney may be thus discharged, and identified by the microscope. Occasionally, also, the abscess, instead of rupturing into the pelvis of the kidney, perforates into the perinephric tissue, burrowing in different directions and producing fistulous openings. Perforations may thus take place posteriorly in the lumbar region, or anteriorly at the groin, into the colon, and more rarely into the lungs and liver, and even into the peritoneal sac.

The *course* and *duration* of suppurative nephritis vary greatly. Traumatic cases are comparatively rapid, either toward recovery or death. Pyemic cases may run their course in 48 hours, and are invariably fatal. But cases due to other causes—viz., impacted calculus, tuberculosis, stone in the bladder, or cystitis—may be prolonged indefinitely, while some terminate without being discovered. Sooner or later the patient generally succumbs to exhaustion, but life may be sustained for years with paroxysms of the severest suffering and a surprising degree of destruction of the kidneys. The *greatest danger to those thus affected is intercurrent illness* which is always more serious and more apt to terminate unfavorably. It is then that the kidneys, previously surprisingly sufficient in eliminating power, give way in this respect, the symptoms of uremia supervene, and the patient dies of this complication. It is well known that the operation for stone is much more likely to be followed by a fatal result when the patient happens to have a surgical kidney.

There are no *complications* peculiar to suppurative interstitial nephritis save those mentioned as causing it; or as resulting from unusual accidents, such as rupture and perforation into neighboring organs, or uremia.

In capsulitis the resemblance of the symptoms to those of stone in the kidney is often very striking, and has led to the diagnosis of nephrolithiasis with operation without discovery of a stone.

Diagnosis.—The diagnosis of suppurative nephritis may be easy or difficult. It is easy when there is the history of a traumatic cause followed

by hematuria, and later purulent urine, with tenderness and pain over the region of the kidney. If the urine contain pus constantly or intermittently, and in addition to this there be pain or tenderness in the renal region, suppurative nephritis may be averred with reasonable certainty. There are no distinctive cellular elements from whose presence in the urine it may be asserted that pus in a given case comes from the pelvis of the kidney or ureter, for though the little columnar or pear-shaped cells are referred to these sources, they also come from the urethra and bladder. *Catherization* of the ureters gives the only actually positive means of telling from which kidney the pus comes. Indeed, without ureteral catherization it is frequently impossible to tell even whether the pus comes from the kidney at all. In certain instances there is a localized abscess which does not discharge into the urethra. Under these circumstances a diagnosis is impossible except on general principles.

As to differential diagnosis, the only certain means of recognizing the *tuberculous form* is by finding the *bacillus* in the purulent urine, at the present day greatly facilitated by the use of the centrifugal apparatus. Should the symptoms described occur in a case of tuberculosis of the lungs, the tubercular nature of the nephro-pyelitis becomes quite probable.

Pyelonephritis is distinguished from *paranephritis* by the more circumscribed shape of the tumor, the absence of edematous infiltration of the lumbar region, and by the presence of purulent urine, unless it happens, as it rarely does, that the paranephric abscess breaks into the kidney and discharges by the ureter. Otherwise there is no pyuria in paranephritic abscess. Pain on flexing and rotating the thigh is characteristic because of the involvement of the psoas muscles. Pyemic abscesses of the kidney may be suspected if a pyemic process is present, and a chill supervene, followed by any or all of the renal symptoms described. By the injection of colargol into the kidney through the ureter and submitting the patient to X-ray examination Keene has been able to locate many cases of localized abscesses of the kidney.

Prognosis.—Operation has done much to improve the prognosis of late years. Traumatic cases may recover if the injury is not too extensive, while very grave injuries are usually rapidly fatal. Recovery, too, ensues on cases succeeding infectious fevers and pregnancy. Cases due to obstruction of the ureters cannot get well so long as the obstruction continues and as their removal is often impossible, such cases gradually grow worse. On the other hand, their fatal termination may be delayed indefinitely. It is often a matter of astonishment at necropsy, that the patient has lived so long, the barest remnant of secreting structure being sometimes found. Extensive repair may take place if the cause can be removed. Conditions of this kind occur when a stone has been removed after long presence either in the bladder, in the kidney or ureter. It is scarcely necessary to say that such persons are in imminent danger from the effect of exposure, acute disease, or other cause which tends to suppress the action of kidneys already crippled in function.

Treatment.—As *operation* offers the best chance of cure in many cases, a surgeon should be called early. There is no curative treatment except by operation for suppurative nephritis. Necessarily the operation can be per-

formed only if one kidney is affected. An operation on one kidney should not be performed unless *both* ureters have been catheterized and a phthalein test performed. Medicinal measures are mainly palliative. One of the most frequent indications is the *relief* of *pain*, which is often so severe as to call for powerful anodyne measures—opium and its alkaloids being absolutely essential. Hypodermic injections of morphin in doses of from 1/8 to 1/3 grain (0.008 to 0.022 gm.), repeated, if necessary, are favorite and effectual methods of relieving the intense pain, which is often due, not so much to the inflammation, as to the impacted calculus or other cause of obstruction. Suppositories of from 1/2 to 2 grains (0.03 to 0.13 gm.) of the extract of opium may be substituted. Hot fomentations and simple counterirritants, such as mustard, are also valuable adjuvants.

The *catarrhal process* in the kidney, its pelvis, and the ureter, and also in the bladder, may be treated with varying success. Diluents are indicated, and for this purpose any one of the numerous negative mineral waters may be used. The usual remedies are the balsams, benzoic acid, hexamethylenamine (urotropin) and phenylis salicylas (salol). Of the first, sandalwood oil is preferable because it is better borne by the stomach than copaiba. Given in gelatin capsules, each containing 10 minims (0.65 c.c.), of which one or two may be taken three times a day, a decidedly beneficial effect upon the catarrhal inflammation sometimes ensues, seen in the diminished amount of pus. Benzoic acid fulfills another indication, that of securing an acid reaction of the urine, which is very often either alkaline or so faintly acid that it rapidly becomes alkaline, and thus is predisposed to decomposition. The benzoic acid is best given in the form of capsules. For an adult 5 grains (0.33 gm.) four times daily are usually sufficient to keep the urine acid. Larger doses than these may be given, or benzoate of sodium may be used in 10 grain (0.6 gm.) doses, either alone or in conjunction with the sandalwood oil, the former before and the latter after a meal. To children smaller doses should be given, 1 grain (0.006 gm.) three times a day, increasing the dose. The various vegetable diuretics, as buchu, pareira brava, etc., are of little use in suppurative nephritis and pyelitis.

Urotropin or formin is the best of the three remedies named. It should be given in 5 grain doses (0.33 gm.) four times a day preferably on an empty stomach, in solution or in a capsule.

The constant and inevitable tendency in these cases to run down in general health, because of the drain and wear and tear, demands *tonics*, such as quinin, iron, and strychnin, while milk and other nutritious articles of diet are always indicated. The dangers to which the patient is subject from exposure, cold, and dampness should be averted by suitable care and woolen clothing.

Many of these cases are saved at the present day by a timely nephrotomy, by which the pus is discharged, the kidney drained, and calculi, if present, removed.

PARANEPHRITIS OR PERINEPHRIC ABSCESS.

Definition.—Parenephritis is an inflammation invading the capsule and connective tissue about the kidney, terminating almost always in suppuration.

Etiology.—A number of causes may be responsible for perinephritic abscess. Thus there may be rupture of a nephritic abscess through the capsule of the kidney; perforation of the bowel, most frequently seen in connection with appendicitis; extension of suppurative disease from the spine, as in caries of the vertebræ, or from a burrowing empyema; finally, blows and injuries may terminate in suppuration about the kidney.

Morbid Anatomy.—At autopsy the kidney is found surrounded by pus, which is usually posterior to it, rarely in front between the kidney and the peritoneum. The pus has often a fecal odor from contact with the large bowel. It may burrow in various directions, and even burst into the pleura and be discharged by the lungs; or it may work its way to the groin and appear at Poupart's ligament. In turn it may perforate the bowel or rupture into the peritoneum, bladder, or vagina. Occasionally the fatty bed of the kidney is found to be converted into a fibrous capsule fused more or less closely with that of the true kidney capsule. There is a milder degree of this condition which I have called capsulitis.

Symptoms.—Most cases are secondary to disease in the neighborhood. *Pain* and *tenderness* in the region of the kidney are the most constant symptoms. In addition there is a peculiar *edematous* or *boggy condition* in the same locality, giving rise to pitting on pressure. The position assumed is often distinctive, the thigh being flexed to relax the psoas muscle, tension on which, especially in adduction, increases suffering. The patient, if able to walk, relies as much as possible on the opposite leg, on which he leans, assuming also a stooping posture with the spine fixed. The whole attitude and behavior remind one of hip-joint disease, while the pain may even be referred to the knee, as in this disease. These symptoms do not, however, appear at once, and the approach is often insidious. At other times suppuration is ushered in by chills, fever, and sweats. Various directions of burrowing and seats of perforation were mentioned in treating of the morbid anatomy. The plastic form of fibroid paranephritis is without distinctive symptoms.

Diagnosis.—The diagnosis from *nephric abscess*, with which it is most likely to be confounded, has been considered. The attitude of the patient in lying or standing is like that in *hip-disease*, but the history elicits that in its incipiency the pain is much higher up in perinephric abscess, while examination shows that the swelling and tenderness are above the hip and not over the hip-joint itself. As most cases except those due to injury are secondary to disease in the neighborhood, it is not necessary to separate the two classes. Secondary forms are more sudden in their onset, though this is not always the case. Doubtful cases may be settled by the use of the X-ray.

Treatment.—This is by section and free drainage, for though spontaneous rupture sometimes takes place, it is apt to be preceded by destructive and dangerous burrowing, which should be anticipated by operation.

NEPHROLITHIASIS (STONE IN THE KIDNEY).

Definition.—Nephrolithiasis means "stone in the kidney," but the term is a general one, which covers the presence in the kidney, its pelvis,

or ureter of concretions large enough to justify the term "stone," of smaller masses appropriately known as "gravel," and fine particles known as "sand."

Morbid Anatomy.—Except in the case of "sand" which includes particles made up either of pure uric acid or oxalate of lime, gravel and stone, as found in the kidney and its pelvis, always have an organic basis through which are distributed the mineral matters which go to make up their bulk, and which remains as a framework after the mineral matters are dissolved out. The matters thus precipitated, in some one of the shapes named, in the order of frequency are:

(1) Uric acid and its compounds of sodium, ammonium, and potassium. (2) Oxalate of lime. (3) The phosphates of calcium and of ammonium and magnesium.

Only in the case of uric acid stones of small size, and of oxalate of lime stones likewise moderate in size, do we have the bulk of the stone made of a single constituent. More frequently it is the case that uric acid or oxalate of lime stone forms the nucleus, and about this aggregate in concentric layers, the phosphates, which make up the great bulk of all large stones as well as some stones in their entirety. More rarely a uric acid nucleus is surrounded by oxalate of lime or the reverse. Not only may the sediments become the nuclei of large stones, but foreign matters, such as a clot of blood or a fragment of any kind of matter accidentally reaching the urinary passages, may also play a like rôle.

The steps for determining the more precise composition of stones will be found in appropriate manuals on the examination of urine, but the three principal varieties present certain physical characters by which they can with considerable certainty be determined. Thus, *uric acid stones* are usually smooth or lobulated, dark red or reddish-brown in color, hard in consistency, and rarely acquire a size of a centimeter (0.4 inch) in diameter, while many of them are no larger than a lentil. They may be multiple. *Oxalate of lime stones* are very hard and uneven, so characteristically so that they have received the name mulberry calculi, from their resemblance to this fruit. Their hard-pointed projections produce exquisite pain in transit from the kidney through the ureter into the bladder. They attain about the same maximum size as uric acid stones, and are also often multiple. The *phosphatic stones* are white in color or grayish-white, quite soft, easily disintegrated, may often be crushed between the fingers, though at other times they are much harder. They attain the largest size, being often as large as a hen's egg. When stones lie in the ureter rather than in the pelvis of the kidney, they are apt to be more elongated, or sometimes, spindle-shaped, and present at times a spiral marking which is characteristic. Others are molded to the shape of the pelvis of the kidney with a prolongation for each calyx, which may be further branched—the dendritic or coral calculi. Rarer forms of calculi are made up of cystin, xanthin, carbonate of lime, and urostealith.

Etiology.—The rationale of the precipitation of sediments which aggregate to form concretions is not always the same, and is perhaps not thoroughly understood. In the case of uric acid the deposit takes place either because of the abnormal acidity of the urine, because it contains more than

the normal quantity of uric acid, or because, for some reason, the amount of water secreted is abnormally scanty. In either event the uric acid is precipitated in the excretory tubes of the medullary substance or in the pelvis of the kidney, forming minute concretions made up of from five to ten whetstone-shaped crystals, whence they descend in the form of sand or gravel to the bladder. At times the sediments grow by successive additions in the pelvis of the kidney, forming thus true *renal* concretions, whose descent into the bladder, if at all possible, is accomplished with the greatest difficulty and pain. The method in which such concretions form in the calices of the kidney around a papilla is well shown in Fig. 80.

Oxalate of lime calculi form similarly by the precipitation of crystals of this substance immediately after the urine is secreted.

Phosphatic concretions are rarely primary. In order that they may form, the proportion of phosphates must be largely increased, or the reaction of the urine must be permanently alkaline. More frequently phosphates precipitate around nuclei of uric acid or oxalate of lime, or foreigh bodies. The effect of these seems to be to cause an alkalinity in the urine immediately about them or, incases of more general cystitis, in all the urine in the bladder. This alkalinity causes the precipitation of phosphates about the primary nucleus and formation of stone of various sizes. Rarely layers of phosphates and uric acid alternate.

Symptoms.—It sometimes happens that a stone is found *postmortem* in the substance of the kidney or in the pelvis which was not suspected, but it is hardly likely that even in these cases symptoms were not present. They were simply overlooked or ascribed to some other cause. The most constant symptom of nephrolithiasis is *pain in the region of the kidney* associated with more or less *tenderness*. Like the pain of stone in the bladder, it is *aggravated by motion*, especially rough motion, and there are certain positions of the body in which the patient is made more or less uncomfortable. Sometimes the inflammation caused by the stone proceeds to suppuration, and the whole of the kidney, more or less, is substituted by a pus-sac. The pain is often suddenly aggravated when a large stone so lodges as to plug up the ureter and interfere with the descent of urine, or a small one descends through the ureter into the bladder. Under these circumstances comes the attacks of so-called nephritic colic, characterized by pain which may equal in severity any to which man is subject. It has other distinctive features. It radiates downward into the groin and the neighborhood of the bladder and down the inside of the thighs and into the testicle, which is often retracted. Sometimes it extends upward toward the diaphragm, and it is not always easy to separate the pain of nephritis colic from that of hepatic colic or appendicitis when the former is on the right side. It may happen that both kidneys are the seat of impacted stone, though this is a very rare event. There are often nausea and vomiting, a cold sweat appears, and the patient may collapse.

After the pain and colic the changes presented by the *urine* may be highly distinctive, aiding greatly the diagnosis; at other times the urine is absolutely negative. It very frequently contains blood, though the quantity is commonly small, and may be demonstrable only by the microscope. Especially is there blood in connection with fresh attacks of nephritic colic.

Pus is almost always present in small or large amounts. Cylindroids or mucus-casts may be found, true casts rarely. In some cases, too, uric acid crystals in the shape of red or brickdust-like particles either before or during an attack of nephritic colic point to the uric acid nature of the stone. The same is true of oxalate of lime crystals. In the case of the last two substances the urine is acid in reaction. Phosphatic stones may be suspected if the urine is alkaline in its reaction, as it is only possible for phosphatic sediments to form in the presence of an alkaline or neutral urine while uric acid crystals can only remain permanent in an acid urine. Oxalate of lime, the most insoluble of all crystals, occurs, however, in either acid or alkaline urine. In cases of gravel or sand, as contrasted with stone, there are no symptoms, as a rule, between attacks, as the stone must reach an appreciable size before it produces the constant or almost constant pain characteristic of it. In some cases there is complete suppression of urine ("obstructive anuria"), even when the kidney on the opposite side is normal, though more frequently when it is diseased, and death from uremia may occur in consequence. In this form of uremia there is often an interesting absence of symptoms of uremia until coma closes the scene preceded by unclouded mental condition. There may be minor degrees of convulsive symptoms, nausea and headache. The duration of this state may last from four hours to 14 days.

Diagnosis.—Nephritic colic may be confounded with *biliary colic*. Usually the symptoms of each are sufficiently distinctive. Jaundice in biliary colic when present comes on very soon after the obstruction begins. The stools are without bile and grayish-white in color. Usually the pain is more toward the epigastric regions as a center, and thence through the upper abdomen and perhaps through to the right shoulder-blade. The urine may be bile-stained, and responds to the tests for the coloring-matter of bile. *Appendicitis* frequently resembles renal colic, and renal colic may be mistaken for appendicitis. In appendicitis fever, leukocytosis, local tenderness and rigidity over the appendix are present, while in stone there is no fever, there are leukocytes and blood in the urine, there is frequent urination, and, as a rule, the pain radiates to the ureter. Thus, *clots of blood* may obstruct the ureter and give rise to all the pain occasioned by an impacted stone, and in the absence of a history of stone and of hemorrhage there are no symptoms by which the two causes of colic can be separated. In the case of *hydatid cysts* of the kidney, fragments of these, too, may be discharged, and in suppurating kidney *inspissated pus* may occlude the ureter. Renal colic is also produced when the ureter is compressed by any cause as a *twist in the ureter* of a floating kidney, or by compression of a tumor. The symptoms of stone in the kidney sometimes closely resemble those of *stone in the bladder*, but the pain in the latter, though it may be felt, to the back, radiates toward both sides; in stone in the bladder the urine contains more mucus and is alkaline or becomes readily so, while in nephrolithiasis the pus is purer and the urine acid. In all cases pointing to the presence of stone in the bladder the sound should be promptly used.

The most invaluable aid to the diagnosis of stone in the kidney is the X-ray examination, and in all cases of suspected stone this measure should be employed. Almost without exception a stone, if present, is disclosed, and many stones thus recognized have been found at operation which would

have otherwise been less searchingly sought. It still happens occasionally that a stone escapes recognition even by the aid of the X-ray. Rarely it has happened that the X-ray has apparently disclosed a stone when none has been found at operation. This is due to faulty technique.

Prognosis.—The prognosis of stone in the kidney is very much more favorable now than it was 20 years ago, in consequence of the safety with which operations can be performed. The kidney may be exposed, split open, and the parts reapposed and restored with perfect recovery, and whenever the diagnosis of stone in the kidney is made, an operation should be done. When many severe attacks occur without other conclusive evidence, an exploratory operation is sometimes justified for the sake of diagnosis. When the sand or gravel is so small as to pass the ureter, the suffering terminates with its passage into the bladder.

Treatment.—There are no medicines which, when administered, are capable of producing solution of a stone. In cases in which there is no such formation and we have simply to contend with gravel or sand, therapeutic measures to prevent its further formation, may be availed of.

Treatment of the Renal Colic.—The extreme pain of attacks of nephritic colic must be combated by anodynes, and for this purpose a hypodermic injection of one-fourth of a grain of morphin is indicated at once. Hot fomentations to the side help to keep the patient comfortable while the morphin is having its effect. A whiff of chloroform may be given for the same purpose. Frequently the dose of morphin must be repeated. It is rarely the case that less than 1/4 grain (0.016 gm.) of morphin thus administered is sufficient, and very frequently this dose must be repeated very soon. Hot poultices are useful to a certain extent in relieving pain when applied to the lumbar region and to the groins, while hot baths are of great sevice in relaxing spasm and allaying pain due to it.

The escape of the stone from the ureter into the bladder is followed by unspeakable relief, and its discharge from the urethra usually follows sooner or later with little or no pain, although a stone of considerable size may lodge in the urethra and require extraction. When stones are not discharged from the bladder they are likely to become the nuclei of larger stones in the bladder.

Repeated attacks of renal colic with pus and blood in the urine can only be interpreted as a stone remaining in the kidney or ureter. Such cases must have a diagnosis completed by X-ray examination and catheterization of the ureter. If the stone is found the case must be turned over at once to the surgeon for extraction of the stone. Persons who are subject to renal colic should lead a properly regulated life. Water in abundance, proper exercise, food regulated so the strength is maintained, but digestion preserved. Alcoholics must be avoided. If for any reason a stone cannot be removed, the case must be treated medically as an ordinary case of pyelitis.

TUMORS OF THE KIDNEY.

Definition and Application.—The term "tumor of the kidney" is applied to almost any enlargement of the organ due to morbid growth. Yet there are morbid growths of the kidney which are not sufficiently large to produce

appreciable change in its size. Thus, the *adenoma* does not usually exceed 4/10 inch (1 cm.) in diameter, though it may be two inches (5 cm.) or more. The same is true of the *angiomata* and *leukemic tumors*, of *fibroma* and *lipoma*, which sometimes form small white nodes in the fibrous tissue near the bases of the pyramids. *Lymph-adenoma* occurs in the kidney associated with similar disease of lymph glands, liver, and intestine. *Villous papilloma* sometimes grows in the pelvis of the kidney. *Syphilitic gummata* also belong to the group of moderate-sized tumors rarely producing symptoms.

Cysts, single and multilocular, acquire larger size, producing appreciable enlargement of the kidney. *Hypernephroma* is a form of tumor of the kidney ascribed to misplaced suprarenal tissue. There tumors grow often to enormous size. One has recently been examined by an exploratory operation which reached from above the edge of the ribs downward to the pelvis and anteriorly as far as the umbilicus. The malignant tumors, *hypernephromata*, *sarcoma* and *carcinoma*, and cysts, are, clinically speaking, perhaps the chief occasion of the term "tumor of the kidney." On the other hand, *renal abscess*, or pyonephrosis, does not usually earn for the kidney involved the name "tumor," while hydronephrosis does.

Symptoms.—Certain local symptoms are produced indifferently by any one of the tumors large enough to become clinically appreciable. In the first place, *renal tumors grow* for the most part *forward* rather than backward, because of the more yielding character of the parts in front of them than behind them. Rarely is there produced posteriorly more than a prominence with obliteration of the normal resonance commonly present between the kidney dullness and the vertebral spines. As a forward growth proceeds, a special effect results from the relation of the bowel to the kidney; as the ascending *colon* on the right side and the descending *colon* on the left lie *in front* of the corresponding kidney, the effect of enlargement of this organ is to push the bowel in front of it, the hollow viscus being recognized by the tympanitic percussion note. In this respect renal tumor differs from splenic tumor, and less invariably from that of the liver, since the bowel does not intervene between these organs and the abdominal wall, though, rarely, the small intestine may float between the liver and the parietes. Commonly, too, the hand may slide between the renal tumor and the liver on one side and the renal tumor and the spleen on the other, while it never loses the rounded border characteristic of the kidney.

By bimanual palpation with the palm of one hand placed in the lumbar region and the other in front below the ribs, pressure being made in both directions, the tumor may be recognized. In this examination, too, it will be noticed that the kidney permits a much more limited mobility during breathing than does the liver, although it is not totally immobile. The renal tumor, too, commonly resists lateral movement.

Again, *pain* in the region of the kidney is an inconstant symptom. It is often totally absent, at other times very severe, especially if the vertebræ and spinal cord are encroached upon. Because of pressure of the kidney upon the 12th dorsal nerve and branches of the lumbar plexus neuralgic pains in the abdominal walls may be present.

Diagnosis.—As to differential diagnosis, the presence of tumor being determined with either *carcinoma* and *sarcoma*, may produce hematuria,

though it is not frequent with them. The blood may be fluid or clotted, and is often molded in the pelvis of the kidney and ureter, which is rarely the case with blood poured into the pelvis under other circumstances. Hematuria is more frequent in carcinoma than in sarcoma. Both conditions are likely to produce pain. It is sometimes very severe and described as *boring* in character, when it is said to indicate destructive encroachment on the vertebræ. More frequently it is dull, radiating over the flank into the thighs. These tumors also produce *cachexia*. Very rarely they may be recognized by the presence of distinctive histological elements in the urine. This occurred in my experience in at least two instances. Frequently the urine is altogether negative.

Of sarcoma and carcinoma of the kidney between which no distinction was made until 40 years ago, the former is now regarded as the more common. Both may be primary or secondary. Sarcoma is a disease of early life, in fact, it is often congenital, when it is represented by that form known as rhabdomyoma, which contains striated muscular fibers. In more than half the cases it affects children under ten. On the other hand, renal cancer is not confined to later life as is cancer elsewhere, and it may even occur in children. More usually one kidney only is affected. Such organ is uneven, soft, even to a sense of fluctuation. Carcinoma also selects one kidney whence it may invade the pelvis and ureter. It affects the general health more rapidly, hematuria is more frequent and copious, but intermittent. If there be a superficial primary growth elsewhere and a renal tumor is present, the presumption is that it is of the same nature, but no certain diagnosis can be made between carcinoma and sarcoma unless the rare opportunity occur to examine fragments discharged with the urine. Unfortunately for diagnosis, the urine is too often quite free from any sediment, even of pus. Carcinoma of the kidney is apt to invade the renal veins and even the vena cava, and as such to cause metastasis in the lungs and in other organs as well.

From *ovarian tumors* renal tumors are distinguished by the fact that in the former the intestines lie in the flanks, giving resonance on percussion in that locality, while an enlarging kidney pushes the bowels in front of it. The ovarian tumor also grows from below upward and drags with it the uterus and appendages, as can be recognized by vaginal examination and by rectal touch.

Much more difficult is it to distinguish the renal tumor from *enlargement of the retroperitoneal glands* (retroperitoneal sarcoma, Lobenstein's cancer) as such enlargement also pushes up the intestines in front of it, giving rise to a tympanitic percussion note. Hematuria never occurs in retroperitoneal tumor, while it may or may not be present in renal tumor. The retroperitoneal tumor may press upon the ureter and the renal vessels and thus produce obstruction to the descent of the urine. The central situation of the enlargement in retroperitoneal tumors contrasts with the lateral growth in the renal tumors.

From *tumors of the liver* renal tumors differ in that the former sooner or later cause a bulging of the right hypochrondriac region, while the renal tumors rarely reach as high as to alter the configuration of the lower thorax. The sharper border of the liver tumor as contrasted with the rounded edge

of the kidney tumor is characteristic, while the freer movements of the liver with the breathing is also of value. *Splenic tumors* are not likely to be confounded with tumors of the left kidney. Splenic tumor protrudes from above downward and toward the umbilicus instead of from the lumbar region forward. It moves more with breathing, and its sharper edge and indentation may be recognized. It is always above or outside of the colon.

Treatment.—Renal tumor is beyond curative treatment by the physician. As soon as the diagnosis is made, a surgeon should be called and the question of operation considered.

Cysts of the Kidney.

Reference is here made only to such cysts as produce clinically appreciable enlargement of the organ. They include:

1. *Retention or obstruction cysts*, solitary cysts ranging in diameter from a centimeter (0.4 in.) to ten centimeters (4 in.) and larger. They may be present in one or both kidneys. These are probably primarily the result of stenosis of a uriniferous tubule behind which accumulates first urine, which is gradually substituted by an aqueous fluid in which may be found traces of urinary constituents. A trace of albumin may also be present. These cysts rarely give rise to symptoms.

2. *The congenital cystic kidney*, in which both organs are the seat of numerous round cysts varying in size from 1/5 inch (5 mm.) to one inch 2.5 cm.), may produce tumors of large size, so large in the foetus, that they have interfered with parturition. They contain a fluid which is at times clear, at others again turbid, colloidal in consistence, and containing albumin, cholesterin, triple phosphates, rarely urea and uric acid, and sometimes fat drops. Persons with these cysts may grow to adult life, and, indeed, such cystic kidneys have been found postmortem when not suspected. Commonly, the subjects die either before birth or shortly after. The exact mode of origin of these congenital cysts is not understood, but they are probably the consequence of a defect in development.

There may be no symptoms beyond that of an enlarged organ, or they may be those of interstitial nephritis with its secondary cardiovascular consequences. There may be a small albuminuria. Blood-disks may be found, but no casts.

3. *Dermoid cysts* are also occasionally met in the kidney, while a *general cystic condition* invading the liver and spleen as well as the kidney is described.

4. *Hydronephrosis* is a monocystic degeneration of the kidney starting in obstruction of the ureter, succeeded by dilatation of the pelvis and gradual wasting of the kidney substance, due to pressure of the accumulating fluid.

The obstruction causing this condition may also be congenital. As such it, too, may be large enough to impede labor. An oblique insertion of the ureter at such angle as to interfere with the easy discharge of the secretion may be the cause of its retention in the pelvis of the organ. Among recognized causes during life are, also, occlusion of the ureter by cicatricial adhesion, by lithiasis, by tuberculosis of the ureter, by pressure,

by tumors, by a retroflexed or prolapsed uterus, by bands of lymph in healed peritonitis and by twists in the ureter of a movable kidney. Finally, carcinoma of the bladder and even hypertrophy of the prostate and stricture of the urethra may be causes.

The contents of the tumor may be purely aqueous; more frequently they are slightly turbid; they contain a few pus-cells, more numerous if they are the seat of inflammation; also uric acid, urea, and albumin.

Its symptoms consist of those already described as common to benign renal tumors of sufficient size. An event which is almost pathognomonic is the occasionally sudden disappearance of the tumor simultaneously with the discharge of a large quantity of fluid from the bladder, followed by gradual refilling of the sac and return of the tumor. This intermittent discharge may be kept up for years. Such an event must be ascribed to a valvular obstruction in the ureter which at times yields to the pressure of the accumulated fluid; or it may be due to the undoing of a twist in the ureter of a floating kidney.

As to differential diagnosis of hydronephrosis *ovarian tumor* is the condition with which it is most frequently confounded. The relative immobility of the renal tumor, as contrasted with the mobility of the ovarian, may be mentioned; also the lumbar origin of the former, as contrasted with the pelvic of the latter, as determined by rectal and vaginal examination. Should the tumor disappear simultaneously with a copious discharge from the bladder, the evidence is, of course, conclusive. The history in the case of ovarian tumor will develop the events of menstrual and sexual derangement, which are absent in hydronephrosis. Hydronephrosis is not likely to be confounded with *ascites*. The changes in the position of the fluid with that of the patient, characteristic of ascites, and its bilateral situation distinguish it at once from hydronephrosis.

From a *circumscribed peritoneal exudate* hydronephrosis is distinguished by the different history, the greater tenderness of the former, and tympany of the subjacent intestine elicited by strong percussion. In renal abscess there is fluctuation, but there are also fever and sometimes chills. The *renal retention cyst* is at times indistinguishable from hydronephrosis. Both may be congenital or due to congenital defects, but should there be intermittent emptying of the sac, with refilling, hydronephrosis may be suspected. The diagnosis from *hydatid cyst*, so far as is possible, follows in the next paragraph.

5. *Echinococcus or Hydatid Cyst.*—Hydatid disease of the kidney is a rare affection, and when the enlargement caused by it is sufficient to produce physical signs, they do not differ essentially from those of hydronephrosis and cystic kidney. Only in the event that the microscope recognizes hooklets or scolices, or fragments of the cyst-wall in the urine or in the fluid obtained by tapping or in the discharge into other localities, such as the stomach, intestines, or bronchi, can a diagnosis be made with certainty. Such discharge into the pelvis of the kidney, if it produce obstruction, may also cause acute hydronephrosis. The presence of hydatids elsewhere, and of the hydatid fremitus, is presumptive evidence. The chemical and physical characters of the fluid from hydatid cysts are given under hydatid disease of the liver (p. 474). Like hydronephrosis and cystic tumor, the

hydatid kidney differs from ovarian tumor by its immobility, unless the disease should perchance invade a movable kidney.

Treatment of Renal Cysts.—The treatment of the whole list of affections included under cysts of the kidneys lies in the province of the surgeon, the chief office of the physician in these cases being one of diagnosis and relief of pain.

ANOMALIES OF FORM AND POSITION OF THE KIDNEY.

Normal Situation of the Kidney.—The normal situation of the kidney is on the quadratus lumborum and psoas muscles, the inferior end of the left kidney extending a variable distance below the edge of the 12th rib, while the right extends about 3/4 inch (20 mm.) lower down, the whole right organ being lowered by the position of the liver. The outer edge of the kidney is often in a line drawn vertically through the end of the 12th rib. Both kidneys descend about 1/2 inch (12.5 mm.) during deep inspiration. The kidney, if in its normal situation, is accessible to pressure just below the last rib at the outer edge of the erector spinæ muscle. Sometimes one or the other, more frequently the left, lies on the lumbar vertebræ on the sacrum, or in the inguinal canal.

Congenital Absence of the Kidney.—The total *absence* of *both kidneys* is possible in connection with extreme abnormalities and defect of development, but is incompatible with life.

Congenital absence of one kidney is not vary rare, the absent one being usually the left. Such absence may be suspected when over the normal situation of the organ a tympanitic note only can be elicited by percussion. In such event, the remaining kidney supplements the work of the absent one, and serious consequences only follow in the event of disease of the remaining organ. The ureter and pelvis of the absent kidney are absent also, but sometimes the remaining organ has two pelves and two ureters. Occasionally the rudiment of a ureter is present. Congenital atrophy of one kidney is even more common, but is discoverable only at autopsy.

Lobulated Kidney.—The lobulated kidney is the most frequent anomaly of form. It consists essentially in the persistence of the lobulation natural to the organ in the fetal state. This is acquired by the end of the eighth week of fetal life, after which it gradually disappears in normal development, but is still maintained with more or less distinctness throughout the first year after birth. The abnormal lobulation is variously distinct. Usually partial and superficial, the fissures are sometimes so deep as to divide the organ into separate reniculi, of which there may be from seven to 20. This lobulation, a rare event in man, is clearly seen in the kidneys of the lower animals, especially in the sheep and ox.

Horse-shoe Kidney.—The most striking of the anomalies of form is the horse-shoe kidney, in which usually the lower ends of the two organs are united either by true renal tissue or by a band of fibrous tissue. More rarely it is the middle segments which are united, and more rarely still the upper ends. In either event, this coalescence is usually associated with *displacement* of the organ, which is then lower down than in the normal condition, usually just above the promontory of the sacrum, more

rarely in the pelvis, and at times on one side or the other of the spinal column. In the fused kidney there are usually two pelves, with from two to four ureters. More rarely there is but one pelvis. The ureters pass over the front of the kidney. The renal arteries spring from the aorta at points corresponding to the situation in which the organ is found. Thus, when above the sacrum, the arteries spring from the back of the aorta near its bifurcation or from one of the common iliacs, while the veins enter the corresponding parts of the vena cava or iliac veins.

The horse-shoe kidney is generally first recognized at autopsy, or at operation, but rarely it may be recognized in its abnormal position above the sacrum, especially in thin persons.

The Movable or Floating Kidney.

Synonyms.—*Ren mobilis; Floating Kidney; Palpable Kidney; Nephroptosis.*

Description.—The normal kidney is commonly quite firmly retained in position by its capsule of fat and by a covering of peritoneum. The movable or floating organ exhibits a very different degree of mobility in different instances. The mobility may be so slight that it can be recognized only by the expert manipulator, or so great that the organ may be easily grasped by the hand through the abdominal walls. In the latter condition there is a mesonephron or peritoneal fold loosely attaching the kidney to the spine.

Etiology.—The movable kidney is more common in thin persons than in the obese, in women than in men. Indeed, it has been said that one woman out of every four has a movable kidney. It is six times as frequent in the working-classes. The right kidney is far more frequently movable than the left. Repeated pregnancies are assigned causes, as is also mechanical violence, as a fall or tight lacing. It is most likely, however, that the majority of floating kidneys are congenitally loose, and that this looseness may be increased by the conditions named. Frequently a floating kidney is only a part of a general enteroptosis.

Symptoms.—The floating kidney often occasions no symptoms. At other times it is responsible for a remarkable train of *nervous symptoms*, mainly reflex in character. These include *obstinate indigestion* of every grade, *flatulence, palpitation of the heart, cardialgia, neuralgic pain* almost anywhere in the body, but especially in the abdomen and cardiac region. Gastric crises identical with those characteristic of locomotor ataxia have been ascribed to floating kidney. *Irritable bladder* and *dysmenorrhea* are also consequences. It is an interesting fact that where the degree of displacement and the mobility are most marked, the reflex symptoms are least so. This is not without a parallel in other diseases, and in illustration may be cited the well-known fact that *prolapsus uteri* of moderate degree often causes decided reflex symptoms, while a complete procidentia produces often trifling local annoyance. The direct result of the displacement, so far as appreciable, is a sense of *dragging* or weight, which especially manifests itself while standing, walking, riding, or dancing, to which may be added a variable amount of *pain*. More serious symptoms sometimes manifest themselves as the result of *torsion* of the ureter, occasioned by

complete rotation of the kidney, in which the renal vessels and nerves are also involved. These are agonizing pain, associated with symptoms of collapse, such as nausea, and anxious expression, and scanty urination. They are caused in part by obstruction to the ureter and the backing of the urine on the kidney. These are known as Dietl's crises. Acute hydronephrosis may also be the result of such strangulation, which may be caused, too, by inflammatory bands. This condition ends sometimes as suddenly as it begins. Both hemorrhage and albuminuria are reported as results. Both are certainly rare. There may be other effects of displacement due to the location of the organ at times, of which irritation due to pressure upon the bladder may be mentioned as one. It is often very uncomfortable for the patient to lie on the one side opposite that on which the displaced organ belongs.

Diagnosis.—This is variously difficult. The kidney exhibits some mobility in health, descending also always 1/2 inch (1.3 cm.) with each deep inspiration. Movable kidneys are sometimes so loose and movable that they may be felt with ease through the abdominal walls. Between this ready recognition and that which requires the highest manipulative skill of the examiner there is every degree. At the present day, movable kidney is regarded as a much more frequent condition than was formerly believed. So frequently has the set of reflex nervous symptoms described been found associated with movable kidney that their presence should always suggest an examination for the presence of such an organ. The examination may be made with the patient in the standing posture, or when lying on the back. In the first he bends slightly forward, the hands being placed on a table, and the clothing thoroughly loosened. The right hand of the examiner is then placed in front immediately next the skin, below the hypochondrium, while the left is placed over the lumbar region. The patient is directed to respire deeply and regularly, and to relax during expiration. The region between the two hands is carefully palpated, when, if there is any marked degree of displacement, or rather of lowered position, the organ can be felt as a firm, smooth, oval body, somewhat sensitive to pressure, which produces a sickening pain quite characteristic. Most rarely the pulsation of the renal artery can be felt. The right kidney naturally moves with breathing more than the left, being pushed down by the liver. Sometimes the manipulation will be more successful in the knee-elbow position. When in this position, the movable kidney having fallen forward, a resonant note may be obtained by percussing over the normal situation of the organ; or the patient may be placed on the back with the side to be examined toward the edge of the bed, on which the physician may sit. The hands are applied as in the standing position, and manipulation is practised as described.

The displaced organ is hardly likely to be confounded with anything else. The *spleen*, which corresponds nearly in size, is also sometimes movable. Its shape is, however, different. Its anterior border is sharp and often notched. Sometimes both the left kidney and the spleen are floating. A *movable pyloric tumor* has been mistaken for a movable kidney. The passage of a stomach tube in case of doubt would clear it up.

Treatment.—As may be inferred, many cases of movable kidney re-

quire no treatment. In a few instances the symptoms are relieved by improving the general health; in others the patient is comfortable while lying on the back, and such comfort may continue for a time after rising. When decided symptoms attributable to the kidney are present, surgical treatment for fixing the kidney—nephrorrhaphy—is the most satisfactory treatment, and this sometimes fails. We have known an operation for removal of the kidney—nephrectomy—to be necessary after nephrorrhaphy had been attempted twice unsuccessfully. The use of pads and supports has been only partly successful. On the other hand, Charles D. Aaron had recently published a paper on the successful treatment of 442 cases without surgical intervention.[1] Progress has certainly been made in the success obtained by bandages made by skillful persons, and it is advisable to make an effort with a bandage before operation is tried.

IDIOPATHIC HEMATURIA.[2]

Definition.—So-called idiopathic hematuria is a hematuria the origin of which is unknown. In addition to the various causes of bloody urine already referred to in treating diseases of the urinary organs, and, in addition to malarial hematuria, there remains a form of renal hematuria of not very infrequent occurrence, for which none of the causes named will account. To this the term idiopathic hematuria is appropriate.

Symptoms.—The characteristics of the urine in this form of hematuria are in no way different from those of renal hematuria from other causes. The blood is intimately admixed with the urine, and is not, as a rule, found in the shape of coagula, as is so often the case when it comes from the bladder or pelvis of the kidney or in malignant disease of the organ. There is the usual smoky hue characteristic of acid urine containing a small amount of blood, becoming brighter red as the urine becomes alkaline, and darker red as the quantity of blood is increased. The microscope reveals numerous blood-disks recognizable by their usual characters, and often blood-casts and casts filled with the débris of red disks, or red disks so closely packed as to make it impossible to distinguish their outline. The urine is, of course, albuminous.

Next to the change in the urine, the most striking feature is the absence of other symptoms. The subject is not ill, is not weak, and complains of nothing. Occasionally a dull ache in the back is felt or supposed to be felt, perhaps because the patient thinks that since there is bloody urine, there ought to be pain in the back. The same may be said of weakness, but these symptoms are not usually complained of, though they may be present. Sir William Gull spoke of such hematuria as a "renal epistaxis." With the lapse of time, however, and the continuance of the symptom, positive weakness gradually supervenes.

Treatment.—Rest is an important and essential condition in the successful management of idiopathic hematuria. Calcium lactate is perhaps the most valuable remedy we have. Hexamethylenamine should be tried on general principles. The usual astringents, mineral and vegetable, known

[1] Jour. Am. Med. Assoc." Dec. 5, 1903.

[2] We realize that the subjects hematuria, hemoglobinuria, and chyluria, next to be considered, are not strictly renal affections, but it is difficult to classify them otherwise.

to be efficient in the treatment of hemorrhagic conditions elsewhere, are often without effect here, though these substances, including adrenalin, gallic acid, the persulphate of iron, and acetate of lead, alum, catechu, and kino, may be tried.

Recent studies by Weil, Moss[1] and others on the serum treatment of hemorrhagic diseases have shown the efficiency of daily intravenous or subcutaneous injections of fresh human or animal serum. It is reasonable to suppose the same treatment would be efficient in hematuria. Under ordinary circumstances 15 c.c. may be injected intravenously and 30 c.c. subcutaneously. *See Treatment of hemophelia.*

HEMOGLOBINURIA.

Definition.—In this interesting condition, the coloring-matter only of the blood is found in the urine; very rarely a few blood-disks or their fragments. In their absence, other criteria of the presence of blood coloring-matter must be sought. To do this, one may make Teichmann's hemin crystals, or if the spectroscope be available, the filtered and diluted urine produces the absorption bands of *oxyhemoglobin* between Fraunhofer's line D and E, or more frequently the three bands of *methemoglobin*, of which that in the red near C is distinctive. Sometimes both are present. The urine thus stained with hemoglobin or *methemoglobin* is dark brownish-red, and even black in color. It is also albuminous, and in lieu of the blood-disks are sometimes found yellowish-brown, irregular, and granular flakes, and sometimes cylindrical masses of hemoglobin.

Hemoglobinuria is always associated with hemoglobinemia, which is, however, less easy of demonstration. The hemoglobin is set free from the corpuscles and imparts a reddish hue to the blood-plasma. The disks themselves are paler, and yellowish-brown particles of hemoglobin may be demonstrated between the corpuscles. The number of corpuscles themselves may be reduced, falling to 4,000,000 and less.

Hemoglobinemia and hemoglobinuria may easily be separated into two divisions, toxic and simple paroxysmal.

Toxic Hemoglobinuria.—This is produced by toxic substances, which dissolve out the hemoglobin from the corpuscles. Such are sulphureted hydrogen, arseniureted hydrogen, carbon monoxid, carbolic acid, pyrogallic acid, naphthol, nitrobenzole; potassium chlorate in large doses, and the poison of certain mushrooms; also sometimes the poison of the infectious diseases, including scarlet fever, diphtheria, pyemia, yellow fever, typhoid fever, malaria, and even syphilis. The last has sometimes seemed to act as a predisposing cause, subsequently to which so trifling a thing as exposure to cold has caused it. I have seen it associated with pregnancy as a probable cause. Hemoglobinemia and hemoglobinuria sometimes succeed on extensive burns when the poison is probably the retained excretions of the skin. Both high and low temperature alone is said to have caused it. In malarial poisoning the hemoglobinemia may be the direct result of the action of the malarial plasmodium. The blood of one

[1] Moss, "Johns Hopkins Hospital Bulletin," July, 1911.

animal transfused into the vessels of another, must be added to this group, since it results in disintegration of red cells and hemoglobinuria. Then there is *Epidemic hemoglobinuria* of the new born with jaundice, cyanosis and nervous symptoms.

Prognosis.—This depends upon the dose of the toxin causing it and the other symptoms produced. Recovery is usual, but some cases are rapidly fatal.

Treatment.—This is that of the disease occasioning it. The same astringent measures may be tried as in hematuria, and restorative medicines may be given to rebuild the blood. Of these, iron is the most important.

Paroxysmal Hemoglobinuria.

In this, intermittent attacks occur. They come on suddenly, preceded by chills and fever, headache, and pain in the limbs, the temperature often reaching 104° F. (40° C.). The bloody urine follows in an hour or less, and may last four or five hours, or there may be two or three paroxysms in a day. At other times there is no fever or the temperature is even subnormal. Jaundice is associated with some cases, especially toward the end. At times, instead of the expected hemoglobinuria, there is only albuminuria. Ralfe explains this by supposing that the toxic agent has destroyed only a small number of corpuscles, the coloring matter from which is used up in the spleen and liver, while the globulin goes off in the urine. Von Leube especially calls attention to a *swelling* and *tenderness* of the *liver* and *spleen*, and says he has met these symptoms in lieu of the expected hemoglobinuria—in lieu even of albuminuria.

The occasional association of hemoglobinuria with Raynaud's disease is very interesting. The probability is that in most cases where the two conditions are associated, the preliminary hemoglobinemia is due to a separation of the hemoglobin from the red disks in the peripheral asphyxiated part of the nose, ears, fingers, or other parts.

As to other causes of paroxysmal form, *malaria* is undoubtedly one, though perhaps not so often as was once supposed. Another cause is excessive muscular exertion, especially when associated with cold, while cold itself is perhaps the most frequent of all causes. Mental emotion is sometimes a cause. It must be admitted that for the cases not explained by toxic agency no satisfactory solution has been presented.[1]

Prognosis.—The prognosis of the paroxysmal form is commonly favorable, though it may continue to recur for a long time.

Treatment.—This depends upon the cause. If it be malaria, the condition is easily curable by quinin. To seek the causes in many cases is to seek the unattainable, and the cases must be treated on general principles. Rest and warmth are essentials. After this, the same astringent remedies as those recommended under hematuria may be tried. Adrenalin is one of these. As cold seasons and cold weather favor it, a residence in a warm climate should be recommended when the condition persists. Nitrite of amyl is said to have cut short and to have prevented an attack.

[1] It is well known that horses are subject to hemoglobinuria, and that it occurs in them after exposure to cold, especially after having been stabled for several days.

NON-PARASITIC CHYLURIA.

Definition and Description.—A state of the urine in which the secretion is admixed with fat in a minute state of division, whereby the urine acquires a milky or chylous appearance.

The proportion of fat varies greatly, being at times only enough to impart a mere opalescence, while at other times the urine is scarcely distinguishable from milk, even the characteristic odor and taste of urine being wanting. The fat, on standing, often rises to the surface, like cream. By the microscope, in addition to this molecular fat and a few oil drops, numerous blood-disks are also found. These are sometimes so numerous as to impart a pinkish tinge to the fluid, and at times a spontaneous coagulation takes place, with the formation of a slight reddish clot, showing the presence also, of fibrin—*hematochyluria*.

Etiology.—To produce chyluria there must be brought about in some way a leakage from chyle vessels into the urinary passages somewhere between the kidney and the neck of the bladder. Yet no such communication has ever been found, so far as we know, though W. H. Mastin noticed the patulous mouths of several chyle vessels opening into the serous sac of a testicle which he laid open for the cure of a chylous hydrocele or lymph scrotum. Having ligated them, no recurrence of the hydrocele happened. Supposing such a communication to exist, how is it brought about?

Symptoms.—Few symptoms other than those of the chylous urine are present. It is usually intermittent, but may be persistent. There is sometimes a loss of strength from the draining off of fluid which is undoubtedly nutrient; at other times there is some pain in the back, and at others again painful urination due to obstruction of the urethra by coagula of fibrin, but in most cases the patient feels well and would not know there was anything the matter with him had not his attention been called to the urine.

Treatment.—No means have been discovered to check the leakage in the idiopathic form, which often persist for years and then subsides spontaneously.

See also article on Filariasis in section on Parasites.

Renal Infarct the Result of Heart Disease.—This form of kidney involvement, secondary to disease of the vascular apparatus, commonly heart disease, is more frequently seen on the postmortem table than recognized in the living subject. It is *embolic infarction*, produced by the lodgment in some branches of the renal artery of an embolus derived from the heart or a blood-vessel. Its most frequent source is a fragment of vegetation or clot from a diseased heart-valve, commonly the aortic. An embolus may also arise from a thrombus in a vein. If from the latter, it must be carried first to the right heart, and thence through the lungs into the left heart, and thence by the aorta to the kidney, and must, of course, be small.

The effect of the lodgment of an embolus in the kidney is a wedge-shaped hemorrhagic infarct which at first is dark red in color, standing out above the surface, but which in time whitens, contracts, and is ultimately absorbed, leaving a mere cicatricial mark.

Most frequently a renal infarct occurs without noticeable symptoms. Its occurrence, if looked for by reason of the presence of valvular heart disease, may be suspected if there is the sudden appearance of blood in the urine. A sudden pain in the region of the kidney occurring at the same time with hematuria would go to confirm the diagnosis. No treatment, except rest, is indicated, even if the event is recognized.

DISEASES OF THE BLADDER.

CYSTITIS.

SYNONYMS.—*Catarrh of the Bladder; Vesical Catarrh.*

Definition.—Cystitis is an infectious inflammation of the bladder excited usually by different varieties of pathogenic bacteria.

Etiology.—Among these are the *bacillus coli communis*, the *gonococcus*, *staphylococcus pyogenes*, *bacillus tuberculosus* and the typhoid bacillus.[1] The causes formerly assigned to such inflammation, though relegated by the above definition to favoring causes, are still very important. They include foreign bodies, such as stone, trauma, obstruction to the outflow of urine by enlarged prostate or stricture of the urethra. A frequent medium of introduction of bacteria was formerly catheters. The number of cases caused by catheterization has diminished because of the greater precaution taken of late in the care of instruments. Of acknowledged bacterial origin is also gonorrheal cystitis, which, succeeding an attack of gonorrheal urethritis, invades the bladder by extension.

Cystitis succeeds upon the introduction of substances in the blood, as cantharides and capsicum. Even the ingestion of certain articles of food has been followed by it. While traumatic agencies are often predisposing causes furnishing the conditions favorable to the operation of bacteria, they may also be exciting causes as may any mechanical irritants, independent of bacteria, if sufficiently irritating.

Morbid Anatomy.—The bladder of cystitis is a varied picture. There may be degrees so slight as to produce scarcely appreciable change in its appearance. At other times the mucous membrane is hyperemic and bathed with a mucoid or mucopyoid secretion of dirty-gray color. In many cases only the neck of the bladder and the part of the urethra passing through the prostate are involved. Again, the bladder is "ribbed," a result of straining. During this act the mucous membrane between the muscular trabeculæ yields, producing depressions bounded by the more unyielding muscular bands. On the other hand, in chronic cases permanent thickening of the bladder-walls may result. Finally, in the severest forms of inflammation due to pathogenic organisms, such as those associated with putrid urine, the mucous membrane may be covered with patches of false membrane, or the wall of the bladder may be infiltrated and undermined with pus, constituting the so-called phlegmonous or diphtheritic cystitis, from which there may result urethral and perineal infiltration. A further

[1] See an admirable paper by Thomas R. Brown on "The Bacteriology of Cystitis, Pyelitis," etc., "Johns Hopkins Hospital Reports," vol. x., 1901.

extension of the cystitis into the pelvic connective tissue about the bladder is known as paracystitis; this belongs to the province of the surgeon.

Symptoms.—While a division of cystitis into acute and chronic is justified by the suddenness and severity of symptoms in certain cases as contrasted with their slow development in others, yet the conditions so constantly verge into each other that a separate consideration of the two forms is not necessary. The first symptom is usually *a frequent desire to void urine*. Such frequency varies greatly in intensity. It may be every few minutes or almost incessant, several times an hour or once in two hours. After the primary frequency of disturbance it usually diminishes somewhat. Such frequency is often attended by painful straining. In severe cases there is always *tenderness* over the region of the bladder above the pubes, and in some cases there is constant *pain*. In these tenderness can also be elicited by pressure from the vagina and rectum, while catheterization is especially painful. In calculous cystitis pain is excited or aggravated by motion, especially such as is communicated to one riding in a wagon over a rough road.

As commonly met, there is rarely fever with cystitis, but the severe forms are attended with *moderate fever* and sometimes, in the diphtheritic variety, with *high fever*. Even when there is fever, the temperature does not exceed 100° to 102° F. (37.8° to 38.9° C.), though it may be higher. In certain acute diphtheritic cases of great virulence there are *chills, sweats,* and *high fever*. In advanced stages there may be *sepsis*, due to absorption of retained putrid matter from the bladder.

The *urine* presents striking changes, by which alone the diagnosis can be made. *First*, it contains pus in varying quantities, but it is especially characteristic of the pus of cystitis that it it associated with mucus, which imparts a glairy, stringy character to the urine, that increases the difficulty of its discharge from the bladder. The reaction of the urine when passed is commonly either alkaline or faintly acid, and if acid, it promptly becomes alkaline. This is due to the formation of ammonium carbonate out of the normal urea, the result of the operation of bacteria. The greater alkalinity thus resulting reacts upon the pus and converts it into a glairy matter resembling mucus, thus further increasing the difficulty of passing it. Under the circumstances the pus is loaded with amorphous phosphates of lime and glistening crystals of ammonio-magnesium phosphate. It is so viscid that it will not rise in the pipet, and must be cut with scissors to be manipulated for microscopic study. Blood is an almost constant constituent of the urine in calculous cystitis, and in the grave diphtheritic forms shreds of gangrenous bladder tissue may be discharged.

The question as to whether there is *pyelitis*, separate or associated with cystitis, is still more difficult. It is true that the pus in pyelitis is very much less glairy and viscid than that of cystitis pure and simple. However, there are no distinctive cellular elements which settle this question, though some assert there are. Even spasm of the bladder, commonly regarded as peculiar to cystitis, may be present in pyelitis. Rather must we rely upon tenderness in the neighborhood of the kidney on the one hand and in that of the bladder on the other. Marked intermission in the purulent discharge, especially if associated with attacks of nephritic

colic, which imply an obstructed ureter, point to *pelvic involvement.* Prolonged cases of cystitis should always be sent to a surgeon for cystoscopic examination to be followed by catheterization of the ureters or one of the elimination tests if circumstances make it necessary. Catheterization of the ureters may aid in the diagnosis, pus from the pelvis of the kidney being thus separated from the bladder contents.

Calculous cystitis may be suspected when pain in the region of the bladder is excited by motion, as in riding over a rough road, or at the end of the penis after micturition; also when there is blood in the urine or when the stream of urine is suddenly interrupted. These symptoms should immediately suggest the use of the sound, negative results with which must not, however, be accepted without qualification, as the stone may be concealed in a diverticulum. The X-ray is also an indispensable aid to diagnosis in calculous cystitis.

Prognosis.—The medical treatment of cystitis does not furnish a very satisfactory chapter in therapeutics. It includes such treatment as the physician is called upon to use, supposing the exciting cause, such as a stone in the bladder or obstruction in the urethra, to have been removed, whenever possible. Thanks to modern surgery; the enlarged prostate which is responsible for so many cases of cystitis is, in the vast majority of cases, removable without accident even in the old. Many cases due to other causes get well; others are only partially relieved.

Treatment.—*Acute Cystitis.*—Of this form the treatment is far more satisfactory, at least so far as the removal of the symptoms is concerned, than that of the chronic form. Rest in bed is a primary and essential condition. Leeches to the perineum should be applied more frequently than they are. A poultice to this same region and over the lower abdominal region is always useful, while a brisk saline cathartic should never be omitted.

As the feverish state which always accompanies cystitis is more or less constantly associated with scanty urine, concentrated and irritating to the inflamed mucous membrane, it is desirable at once to increase the secretion and thus dilute it. Copious libations of pure water, to which the citrate or acetate of potassium is added, in 15 to 20 grain (1 to 1.3 gm.) doses for an adult, should be allowed. The ordinary spirit of nitrous ether in dram (3.4 c.c.) doses every two hours is an admirable adjuvant, and may be combined with the official liquor potassii citratis, which contains about 20 grains (1.3 gm.) of citrate of potassium to 1/2 ounce (15 c.c.). Formerly, the mucilage of flaxseed or flaxseed tea was much used as a diluent menstruum for the diuretic alkalies indicated, but it is doubtful whether it is any more efficient than a like quantity of water. When there are much pain and straining, as is often the case, opium is indispensable, always in the form of a suppository, 1/2 grain (0.03 gm.) to 1 grain (0.065 gm.) of the extract, or a corresponding amount of morphin. Iced-water injections into the rectum, or pieces of ice similarly applied, are very efficient in allaying the pain and irritation when additional measures are needed. Injections of cocain into the bladder are useful in allaying the intense irritation. Not more than 2 grains (0.13 gm.) of cocain should be introduced into the bladder at one time.

Chronic Cystitis.—The successful treatment of chronic cystitis is a much more difficult task, for three evident reasons: Though the case is frequently a purely surgical condition and the usefulness of drugs by the mouth is decidedly questionable we will consider it here.

1. The constant presence in the bladder of the urine with its irritating qualities, especially so to an inflamed mucous membrane.

2. The difficulty in getting remedies to reach the inflamed surfaces.

3. The pent-up inflammatory products, which in their decomposition often make the urine still more irritating by exciting in it ammoniacal changes.

There is no doubt that, if the urine could be kept from entering the bladder during the existence of an inflammation, the latter would rapidly heal; that cure would be facilitated by obtaining ready escape for the pus and mucus; while happier results might also be reasonably expected if we could secure readier access for remedies to the inflamed areas. None of these indications can be met entirely. They remain, however, the conditions to be fulfilled, and while none can be thoroughly fulfilled, they may be variously approximated.

First, the irritating qualities of the urine may be diminished by the use of diluents, already recommended in the treatment of acute cystitis. Almost any of the negative mineral waters, so highly recommended by their owners, are useful for this purpose. Pure spring water is just as good and distilled water is even better. From one to two quarts should be taken daily. If the kidneys are equal to their office, a large quantity of light-hued urine of low specific gravity and relatively weak in solids will be secreted.

When it is purposed to go further and add to the efficiency of diluents, mistakes are often made. While one can scarcely go astray in adding alkalies to the fluid ingested in acute cystitis, it is very different with the chronic form. In this the urine is often alkaline, or ready to become so on the slightest addition of alkali to the blood. Such alkalinity of urine in turn favors decomposition, the effect of which is to convert the pus, if present, into a tenacious, glairy fluid which the bladder cannot evacuate. Notwithstanding this tendency, liquor potassii and other alkalies are sometimes administered under precisely these conditions—adding "fuel to the flame." The indication under these circumstances is to render the urine acid, if possible, although the means to this end are unsatisfactory. Benzoic acid has the reputation of doing this, and it is probably true of it when administered in sufficient doses. It may be given in the shape of a 5 grain (0.32 gm.) capsule, of which at least six must be given in a day to produce any effect. Benzoate of sodium may be given in 10 grain doses (0.6 gm.) every two hours. The same property has been assigned to citric acid, but this is a mistake, as all of the vegetable acids, when ingested, are eliminated as alkaline carbonates.

The second indication is to medicate the inflamed surface. Two ways suggest themselves:

1. By the internal administration of drugs.

2. By the injection of medicated liquids into the bladder.

To carry out the first method, an anormous number of infusions, decoc-

tions, and fluid extracts of vegetable substances have been suggested, the vast majority of which are absolutely useless, except as they serve by their quantity to act as diluents. Among the best known of these are buchu, pareira brava, uva ursi, and triticum repens.

The remedies heretofore most efficient in cystitis through their internal administration are the balsams. Sandalwood oil is easily borne, and is also an efficient remedy. It is best administered in capsules containing 10 minims (0.6 gm.). They can be given on an empty stomach before meals. It is desirable to impregnate the blood and to impart a balsamic odor to the urine. This is scarcely possible with less than eight capsules a day—two before each meal and two at bedtime. They should be followed by a little milk rather than water.

A valuable addition to drugs useful in the treatment of cystitis is hexamethylenamin, and it is most efficient. It is most indicated in subacute and chronic stages. The dose is 5 to 7 1/2 grains (0.33 to 0.5 gm.) in a capsule three to five times a day. It is not always superior to sandalwood. Occasionally this balsam is more efficient.

Both boric acid and benzoic acid are useful adjuvants to the treatment of chronic cystitis through their antiseptic effect on the urine, each in 5 grain (0.32 gm.) doses, rapidly increased to 10 grains (0.65 gm.). They may be given jointly, as in the following prescription:

℞ Sodii biborat., Ac. benzoic., } gr. x (0.65 gm.)
Infus. buchu, f ℥ ij (60 c.c.).
Three times a day.

Resorcin in 3 to 5 grain (0.198 to 0.33 gm.) doses and naphthalin in 2 grain (0.13 gm.) doses are recommended for the same purpose. Salol is a popular remedy, very large doses being advised—from 15 to 30 grains (1 to 2 gm.) every three hours.

The application of remedies to the bladder by injections is best considered in connection with the third indication—the getting rid of inflammatory products, the pus and mucus, and the matters resulting from their decomposition. The latter are not always present, but all who have had much experience with cystitis are familiar with the tenacious, glairy, mucoid matter, which will not drop or rise up in a pipet, glistening with large crystals of triple phosphate, and exhaling a stinking, ammoniacal odor which quickly contaminates an entire apartment. There is only one way to get rid of this, and that is to wash out the bladder, and often this is too long deferred. Tepid water should be used first, and the injection made through the soft, flexible catheter. Four ounces may be used with entire safety. After the capacity of the bladder has been determined, even more may be thrown in, because it is sometimes useful to distend the viscus a little, in order to reach the depressions and inequalities always present in advanced inflammations. These simple injections, practised once a day, or in severe cases twice a day, often result most happily. After a few injections with plain water some medications may be added. Salicylate of sodium, in the proportion of a dram (4 gm.) to the pint (1/2 liter), is one of the best. Its disinfecting qualities are undoubted. Boric acid, in the proportion of a dram (4 gm.) to the pint (1/2

liter), is also very satisfactory. Sir Henry Thompson's soothing lotion—of biborate of sodium an ounce (30 gm.), glycerin 2 ounces (60 c.c.), water 2 ounces (60 c.c.) and of this mixture 1/2 ounce (15 c.c.) to 4 ounces (120 c.c.) of tepid water—may also be used.

At the University Hospital to-day the emulsion of silver iodide has largely replaced the nitrate of silver. The bladder is first irrigated by a five per cent. boric acid solution. Two ounces (60 c.c.) of five per cent. emulsion of iodid of silver are then introduced into the bladder by a glass catheter and allowed to remain as long as the patient can hold it. The injection is made at first twice daily. If the patient is made more comfortable it is applied daily; later as improvement progresses every other day followed by increasing interval. Salt solutions may be used on intervening days. The bladder, if previously washed out by boric acid solution, should be irrigated with plain warm sterilized water in order to avoid chemical reaction between the iodide of silver and the boric acid.

Alum is an astringent which has been too often overlooked of late in suppurating processes in mucous membranes, and may be substituted for the salicylate with advantage when the pus does not disappear so rapidly as is desired. It should be more cautiously used than the salicylate of sodium. Sufficient of the powdered alum should be first added to a pint of water to give it a distinctly astringent taste, when the bladder should be washed out as described, while a small quantity may be allowed to remain after the last injection.

When there is a foul odor present, the bichlorid of mercury may be used in exceedingly dilute solution—not more than 1 to 25,000 at first—gradually increasing the strength if it is well borne. Carbolic acid may also be used in weak solution—1/4 to 1/2 per cent.—also peroxid of hydrogen, one part to four or five of water. Among other remedies recommended for use in the same way are acetate of lead, 1 grain (0.06 gm.) to 4 ounces (120 c.c.); dilute nitric acid, 1 or 2 minims (0.06 or 0.12 c.c.) to the ounce (30 c.c.).

Anodynes are indispensable in many cases of cystitis to relieve the patient of extreme pain and of the frequent desire to pass water, which are the result of the same cause. Opium and its alkaloids are the most efficient, and they are best introduced by the rectum. There appears to be no absorbing power in the bladder for opium; and there is no use in attempting to administer anodynes by that channel. Cocain, from which so much might reasonably be expected, is disappointing, its effects are fleeting though it should not be overlooked, that it may produce some of the symptoms of cocain poisoning. To avoid this not more than 2 grains (0.13 gm.) of cocain should be put into the bladder at one time.

The urethra can be cocainized. For catheterizing, a four per cent. solution may be injected into the urethra, allowing two or three minutes to elapse before the catheter is introduced. Then, through the soft catheter itself, a few drops of this solution may be injected in advance of the catheter, which is again pushed a little further; then a few more drops are instilled, the catheter is introduced a little further, and so on until the instrument enters the bladder. This is not always a safe procedure. Serious symptoms of cocaine poisoning can occur.

When there is greatly *enlarged prostate*, catheterization is indispensable,

and is often attended with the most happy results. It is often too long deferred because of the natural repugnance to the use of the instrument. The greatest precautions should be taken to cleanse the catheter after its use, in order to avoid sepsis. The catheter should be thoroughly boiled after and before its use.

A surgeon should be consulted in all cases of severity. Catheter life is distressing and short. In proper cases, prostatectomy is the only proper course to follow.

STONE IN THE BLADDER.

All that has been said in a general way of stone formation and the treatment of its tendency when treating of nephrolithiasis may be applied to stone in the bladder.

Symptoms.—The symptoms of stone in the bladder are practically those of cystitis, already described, aggravated by motion, especially riding over rough roads. As further distinctive of stone in the bladder may be mentioned pain at the end of the penis immediately after micturition. The only proof, however, of the presence of stone is its recognition by the sound or X-ray which sould be used in every case of cystitis.

Treatment.—For removal of stone in the bladder medicinal treatment is valueless, and operative treatment is imperative.

NEUROSES OF THE BLADDER.

Paralysis of Bladder.

If the spinal cord is cut above the lumbar enlargement, voluntary power to aid or suspend the action of the sphincter is lost, the bladder is given over to the lumbar cord as a pure reflex center. The urine accumulates as long as the action of the sphincter prevails, but as soon as a sufficient amount accumulates to stimulate the extrusor, the bladder is emptied more or less completely. Thus is produced one of the forms of incontinence, as when there is extensive lesion of the cord above the lumbar region.

If, on the other hand, there is paralysis of the *detrusor* muscle and the sphincter remain intact, there will be retention of urine. If, however, communication with the brain remains intact, by an act of the will the reflex contraction of the sphincter may be suspended and the bladder partially emptied by a straining effort, at least so far as pressure can be exerted by the abdominal muscles. Should the afferent or sensory nerves of the reflex arc be paralyzed either alone or in conjunction with the efferent to the detrusor, the bladder will become enormously distended; but if the distention continue, a point is reached when the sphincter is paralyzed by overstretching, when incontinence occurs and the urine dribbles away. There is the same effect if there be destruction of the cord at its lumbar enlargement. So long as the cord is intact the patient may partially empty the bladder by abdominal pressure. Again, if paralysis of the *sphincter vesicæ* occurs, incontinence succeeds as soon as urine has accumulated sufficiently to overcome the elastic closure of the bladder orifice. It may also be slightly delayed by voluntary innervation of the sphincter, but is

unrestrained during sleep. Hence at such time the patient wets the bed. Such incontinence is also manifested when the patient coughs or when in any way sudden pressure is brought to bear on the bladder. It is often seen in women who are said to have "weak" bladders. Combined detrusor and sphincter paralysis is followed by *dribbling* away of urine as soon as enough accumulates to overcome the elastic closure of the urethra, because there is no contraction of the bladder, and the outflow is a mere overflow.

Muscular Spasm of the Bladder—Cystospasm.

Symptoms.—In detrusor spasm sudden evacuation of the bladder takes place. This occurs in hyperirritability of the mucous membrane of the bladder or of the reflex center in the cord as soon as a small amount of urine accumulates in the bladder. It may be controlled to a degree by a voluntary impulse to the sphincter, but at other times it is irresistible, and is especially prone to occur during sleep. To this class of cases belong many of the instances of incontinence in children.

In spasm of the sphincter, on the other hand, the orifice is kept forcibly closed, though this closure, too, may be intermitted by action of the will, permitting thus a small quantity of urine to be discharged at a time. As the urine accumulates the discomfort increases still further, when an attempt is often made to empty the bladder by straining efforts. This sometimes reacts on the sphincter, producing further contraction, which may extend to the bulbo-urethral and sphincter ani muscles, causing painful spasm. Such spasm, too, forcibly resists the introduction of a catheter. It may be due to hyperexcitability of the sensory reflex center or to irritation directly in the neighborhood of the sphincter, such as intense inflammation.

A combination of spasm of the detrusor and sphincter muscles may exist, giving rise in high degrees to intense suffering. It may be caused by a simultaneous irritation of the two reflex centers in the cord or by intense irritation of the mucous membrane of the bladder reflected to both sets of muscles.

In addition to the nervous affections, chiefly of the cord, which may occasion these symptoms, modifications in the sensibility of the mucous membrane of the bladder, of the deeper urethra and prostate may also occasion them. These changes may be purely neurotic. R. Ulzmann has refined the subject of neuroses of the genito-urinary system to a high degree, referring many symptoms of the kind described to an "exalted reflex excitability" caused by "overstrained physical, but especially by exciting mental activity," long kept up. Among these he mentions fright, pain, grief, loss of property, and the like, as well as the "gonorrheal process," excess in venery, and masturbation, apart from the organic processes of hyperemia, and even inflammation, which may be due to gonorrhea. The pure neurotic representatives of this class are unattended with changes in the urine, which is normal in every particular. These are not very uncommon, and they are often extremely difficult to treat successfully.

A comparatively frequent representative of this class is due to a hyperesthesia of the vesical mucous membrane, as the result of which the presence

of the smallest quantity of urine gives rise to a pressing desire to empty the bladder, which is accomplished with spasm, pain, or other discomfort. As the result of this the patient must empty his bladder often—several times an hour, but much less frequently, if at all, at night. The urine is, as a rule, normal, and though sometimes concentrated, with a proportionate specific gravity, is still no more so than that which is commonly retained with perfect comfort. This occurs also sometimes in women.

Occasionally there is absolute loss of sensation in the vesical mucous membrane, apparently also functional, in consequence of which the urine accumulates without exciting the attention of the patient, and the bladder becomes thus overdistended.

Treatment.—*Of Incontinence* or *Enuresis.*—Previous to instituting treatment for these conditions the most careful inquiry must be made as to the cause, and its removal sought. This is often impossible, and treatment must then be empirical.

Incontinence most frequently calls for treatment. If due to disease of the cord, it is amenable to treatment so far as such disease is, and in the meantime the patient must be protected by catheterization from the overdistention which is so apt to precede incontinence. Incontinence due to weak sphincters demands that this weakness should be treated by full doses of strychnin, which may be advantageously given in gradually ascending doses. Tincture of nux vomica may be substituted in ascending doses until 15 minim (1 c.c.) doses or more are attained. Electricity has been highly commended for this form of incontinence, in the shape of faradization, one pole being applied to the lumbar part of the spine and the other in the urethra, in the vagina, or to the perineum, the sittings being continued for a few minutes each day or every other day. Cold douches to the perineum are also useful.

If incontinence is due to hyperesthesia of the mucous membrane or to irritability of the bladder, belladonna is the accepted remedy. It should be given in ascending doses, and toward evening if it be nocturnal incontinence, so common in children. The physiological effect of the belladonna should be produced. The bromids may be combined with it or used separately. If there is irritability of the lumbar cord, ergot commends itself through its effect of diminishing congestion of the cord. The urine should receive attention, since a high degree of acidity or the presence of sediments of uric acid and of oxalate of lime may become the exciting causes of incontinence.

Incontinence in children (which is the most frequent variety met in practice) is a source of great annoyance, but in the majority of cases it subsides spontaneously not later than the 12th year. In its treatment in addition to the measures suggested, close investigation should be made for causes which should be removed. Masturbation especially in asylums and institutions is apt to be a cause of incontinence and should be carefully inquired after.

Habit is sometimes a cause of incontinence in children, and encouragement of a cautious practice of holding the water may gradually correct the evil. Children should *not* be punished for incontinence, as the nervous apprehension excited only serves to make matters worse. General ill-

health and irregular habits are sometimes responsible, and when these are corrected the patient recovers. Phimosis is sometimes a cause, and should be corrected if present. Indeed circumcision is sometimes a cure even when phimosis is not present.

Of Retention.—An overfull bladder should always be relieved by the catheter, and catheterization should be repeated as often as necessary to prevent recurring distention while the cause is being treated. When the retention is due to weakness of the detrusor muscle, strychnin will be of service. Electricity may also be used—one pole being placed behind the pubes and the other applied to the lumbar region.

If retention is due to spasm, the cause should be carefully sought. The same irritations referred to as causes of incontinence may produce spasm, and some of the same remedies are useful to relieve it, as belladonna and the bromids. Warm sitz-baths and full baths and enemas of warm water may be used at a temperature of 95° F. (35° C.) two or three times a day. In the event of failure with these measures, more powerful anodynes may be used, including opium and morphin. These are best administered in the shape of a suppository containing from 1/2 grain to 1 grain (0.033 to 0.0066 gm.) of extract of opium, and 1/4 grain (0.0165 gm.) of morphin. Ultzmann recommends, in cases of frequent micturition due to hyperesthesia of the prostate, injections through the prostatic urethra by a catheter which just reaches the membranous portion of the urethra. The solutions used are a 1/4 to 1/2 per cent of carbolic acid and a 1/2 per cent. solution of sulphate of zinc, increasing the strength as it is borne to three, four, and five per cent. These should be used once a day with a syringe holding 4 ounces (100 gm.), and the whole quantity should be thrown into the bladder in the manner prescribed.

Other forms of spasm must also be treated by sedatives, and, strange as it may seem, the passage of a sound will sometimes relieve such spasms.

Unfortunately, the causes of either of these conditions cannot always be ascertained, and a cure must be secured by passing from one remedy to another until the correct one is arrived at.

HEMORRHOIDAL VEINS OF THE BLADDER.

Excluding all other causes of hemorrhage of the bladder heretofore considered, such as villous cancer, stone, and tuberculosis, there remains a cause of hemorrhage which, by exclusion, resolves itself into a hemorrhoidal state of the veins. Its subjects are only older persons, rarely under 60; it is rather copious and yet rarely fatal—in our experience never so—though fatal cases are reported.

Great care should be taken in the study of cases of this kind in order to make sure that the hemorrhage is not due to the more serious causes already considered, otherwise a grave mistake in prognosis, as well as in diagnosis. may occur. The bladder should be carefully explored by the sound, and if necessary, by the endoscope.

Treatment.—Hemorrhages from this source may occur and not be repeated, and it is this favorable termination, in the absence of stone or malignant disease, on which we are sometimes unfortunately compelled to

rely for the diagnosis. Should the hemorrhage continue, astringent solutions—1/2 per cent. and upward of alum and sulphate of zinc—may be injected into the bladder, always using the soft catheter. Absolute rest in bed should be insisted upon. At the same time, the astringent drugs and mineral waters recommended under the treatment of hematuria may be tried, but it is hardly to be expected that astringent effects can be produced in the bladder through the route of the circulation by medicines administered by the mouth. Turpentine in emulsion thus administered has appeared to me to be decidedly efficient in controlling these hemorrhages.

MORBID GROWTHS OF THE BLADDER.

The bladder is subject to myoma, myxoma, sarcoma, and carcinoma, especially the variety known as villous or papilloma; also to tuberculosis. Carcinoma may be primary, but is commonly secondary. The simplest histoid tumors are not clinically recognizable, one from the other.

Symptoms.—*Carcinoma of the bladder* may be suspected if, in addition to the usual symptoms of cystitis, hemorrhage is copious and persistent, if there is carcinoma elsewhere, and if there is rapidly-developed cachexia, and especially if there are other signs of secondary cancer in the vicinity. Occasionally villi of the papillomatous growth are passed in urine and easily recognized. The only certain means of diagnosis is in the cystoscope which, in the hands of a skillful manipulator, affords valuable assistance.

The symptoms of *tuberculosis of the bladder* are those of cystitis, and the recognition of the bacillus of tuberculosis, or better, a guinea-pig test, affords the only sure means of differential diagnosis between it and other forms of inflammation of the bladder.

Treatment.—If the diagnosis of villous cancer can be made early, the life of the patient may be prolonged by surgical procedures. The palliative treatment is that of cystitis. At the same time the counsel of a surgeon should be promptly sought.

The local treatment of tuberculosis of the bladder is that of cystitis. It demands the same general treatment as tuberculosis occurring elsewhere.

SECTION VIII.

DISEASES OF DERANGED METABOLISM (CONSTITUTIONAL DISEASES).

MYALGIA.

SYNONYMS.—*Myositis; Fibrositis.*

Definition.—A painful condition of voluntary muscles and their aponeurotic coverings, especially aggravated by motion and pressure. It affects especially large muscles, such as those of the neck, the shoulders, the arms, the back, the thighs, and the calves of the legs, and the intercostal muscles.

Etiology and Pathology.—Exposure to cold, damp and wet, and especially to drafts of cool air, as from an open door or window, is the most frequent cause. Any one of a number of poisons is capable of acting as an irritant to peripheral nerves whose irritation constitutes muscular rheumatism or myalgia. Such are the metallic poisons as lead and arsenic, alcohol, certain drugs, the poisons of the infectious diseases and of other diseases associated with deranged metabolism as *diabetes and gout.* Barometric changes with or without the approach of rain may increase the severity of the pain, more particularly in the chronic variety.

Its true nature is unknown, and whether it is an affection of muscular substance or of the intermuscular connective tissue is also unknown. From the supposed rôle played by the latter, the term "fibrositis" has been suggested.[1] Certain forms of myalgia, especially that of the back, are ascribed to gout. An infectious origin has been suggested. It is sometimes associated with articular rheumatism. Similar pain often succeeds muscular strain.

The division of myalgia into *acute* and *chronic* is not based on any essential difference but rather upon the duration of the pain and upon its disposition to recurrence. The term chronic is justified by those forms which recur with changes in the weather, and are either excited or relieved by them. It, too, is less localized than the acute. On the other hand, it is not inaptly at times called wandering. It is more common in men than in women, because of their more frequent exposure to its causes.

Symptoms.—The only invariable symptom is *pain*, aggravated by motion or pressure. Sometimes there is *swelling*. It is usually rather sudden in its onset, requiring at most but a few hours, and often less, to develop it. It is never accompanied by marked constitutional disturbance. The *pulse* may be somewhat accelerated, and the *temperature* may approach 100° F. (37.8° C.), but more often there is no fever at all.

[1] For an excellent paper by Arthur P. Luff, enlarging these views, see "Clinical Journal," Oct. 11, 1905. "Forms of The Diagnosis and Treatment of Some of the So-called Rheumatism."

Myalgia is especially named according as it involves special muscles. Thus, *lumbago* is a painful affection of the lumbar muscles and their tendinous attachments. The attacks come on under the conditions already named.

Rheumatic *stiff neck* or *torticollis* (as contrasted with congenital and spasmodic torticollis) is an affection of the side and back of the neck, forcing the patient to hold his neck to one side as the situation of least discomfort, and when he desires to turn his head he is forced to turn the whole body. If the spasm is in the trapezius the back of the head is drawn down toward the affected side. If the sterno-mastoid is affected, the chin is tilted and drawn away from the affected side. It is more frequently met in children and young adults.

Omalgia is a similar condition of the muscles of the shoulder and upper arm, making motion exquisitely painful. Ankylosis of the shoulder joint may be caused by delayed motion. *Pleurodynia* affects the intercostal muscles and makes breathing and coughing very painful, while a deep breath becomes impossible and sneezing an agony. The pectoral and serratus muscles may also be involved when the pain is higher up. It occurs more frequently on the left side.

Cephalodynia, or myalgia of the muscles of the scalp, *scapulodynia*, and *dorsodynia* are all forms of muscular rheumatism which explain themselves. It also affects the *abdominal muscles*, and a most intestesing instance of this form simulating peritonitis was published by myself in the "Philadelphia Medical Times," volume x., 1880.

The duration of the acute form is brief, seldom lasting for more than a few days, though there may be a tendency to relapse. The chronic forms are indefinite in duration.

Diagnosis.—This is easy for the coarser acute forms of omalgia, stiff neck, and lumbago. Myalgia may, however, be confounded with *neuritis*. In neuritis there is pain with tenderness more localized and along the course of large nerve trunks. Myalgia and neuritis are distinctly worse on motion; neuralgia less so. Myalgia is commonly relieved by the warmth of the bed; neuritis may be aggravated while neuralgia is uninfluenced by this cause and increased by cold winds. *Pleurodynia* is sometimes difficult to distinguish from intercostal neuralgia but attention to the points named will prevent mistakes. *Brachial neuritis* resembles omalgia, but the former is early followed by atrophy, while myalgia is not. From *pleurisy*, pleurodynia is easily distinguished by the absence of fever and of physical signs. The lancinating pains of *locomotor ataxia* and the pains of *incipient disease of the vertebra* resemble at first those of lumbago, but the special symptoms of these diseases are soon superadded. Attacks of iliosacral luxation are constantly mistaken for lumbago, and can be differentiated by the sudden onset of pain while performing some indifferent motion like stooping. The pain is freuqently so severe that the patient is unable to move about and almost immediate relief is given by strapping or bandaging the iliosacral joint.

Treatment.—The *acute* form of myalgia is occasionally amenable to treatment by the *salicylates* and *salicin*. Some phenomenally good results sometimes follow the use of these remedies. They are, however, inconstant, and if, after a fair trial, such results are not promptly attained, the

drugs should be omitted. If efficient, the same rules as to their continued use in reduced doses after relief has been obtained apply as in acute articular rheumatism. The group of muscles treated must be placed at *absolute rest*, and in the case of the thorax this is best accomplished by strapping the side with adhesive plaster. Rest may, however, be everdone, in case of muscles like those of the shoulder in which atrophy may result from too prolonged a rest. Another measure of great value is *dry heat*, applied by means of a hot-water bag covered with flannel, or by a warm flat-iron. To use a popular expression, a myalgia may thus be sometimes "ironed out." A flannel cloth should be interposed. With these measures may also be associated massage. Sometimes a single efficient treatment by massage is enough to "rub out" such a myalgia. Of less permanent utility are hot poultices, although they allay pain, at least. The same effect is accomplished by moist hot-air or vapor (steam) baths, which, by special appliances, can be localized.

The chronic form is also treated by massage, passive motion, and electricity, either the induced or direct current. Counterirritation by liniments, such as those made with chloroform, ammonia hydrate, or turpentine, have long enjoyed a reputation, but at the present day it is beginning to be questioned as to whether, after all, it is not the friction, rather than the liniment itself, which produces the good effect. Some efficiency in the liniment itself, must still be admitted, and I would advise its use as determined by circumstances. Hydrotherapy is more likely to be useful, and here the warm or cold pack is the better method of application. Dry-cupping is also often of service. Small blisters should not be forgotten.

General treatment should not be neglected: cod-liver oil, iron, strychnin, quinin, and good food are necessary measures when the patient is run down. Among diseases which need nutritious food, chronic muscular rheumatism is preeminent.

Joint Affections Simulating Rheumatism.

These include numerous joint inflammations of septic origin, such as occur in septicemia, scarlet fever, diphtheria, and the like. Excepting hypertrophic pulmonary arthropathy and arthritis deformans, they have all been appropriately referred to when treating of the infectious diseases.

HYPERTROPHIC PULMONARY ARTHROPATHY.

The club fingers and toes of chronic pulmonary and cardiac disease were known to Hippocrates from whom the fingers received the name Hippocratic fingers; but it was not until 1889 that this category of arthropathies was enlarged by Bamberger to include thickening of the long bones. It was further enlarged the next year by Marie whence the term Marie's syndrome- There are many subvarieties and modifications but usually there is symmet. trical involvement of the lower ends of the radius, ulna and metacarpals, more rarely the lower end of the humerus and of the tibia and fibula. The fingers are often involved and sometimes the fingers of one hand only.

There is no actual bone change fibroid thickening of the periosteum and surrounding capsular tissue in which also the blood-vessels are distended. The ultimate cause is unknown. It has been ascribed to the turgidity of the blood-vessels mentioned above; again to toxins causing periostitis. Marie favored the latter cause.

Symptoms.—Among the associated conditions are tuberculosis, bronchiectasis empyema, congenital heart disease, hepatic disease, chronic nephritis and diarrhea. The symptoms therefore include those of these diseases. There may be none except the enlargement which may be so gradual as to escape notice. Frequently there is pain and tenderness.

The fully developed condition is easily recognized, and typical club fingers once seen are never forgotten.

There is no efficient treatment and the condition slowly increases but probably does not shorten life.

ARTHRITIS DEFORMANS.

SYNONYMS.—*Rheumatoid Arthritis; Osteoarthritis.*

Definition.—A deforming disease of the joints, distinct from gout and rheumatism, and characterized by destructive changes in the synovial membranes, cartilages, and bone, periarticular inflammations, also at times by atrophic changes in the bones.

Etiology.—Although in the clinical features of its incipiency arthritis deformans sometimes closely resembles the mild form of acute rheumatism, they have no causal relation. *Heredity*, however, plays a likely, if not an important, rôle. Females are much more liable to the disease than males, especially sterile women and those who have had uterine or ovarian disease. A. E. Garrod collected 500 cases, of which 411 were females and only 89 males. It is a disease said to be as common in the rich as in the poor. It usually begins between the ages of 20 and 30, but it may occur in children under 12 and as late as 50. In Garrod's 500 cases there were only 25 under 20. It is comparatively rare in negroes. The ending of the menstrual period in women is a favorite time for its incipiency. The disease is probably infectious in origin.

Traumatism, often assigned, by the subjects of the disease as a cause, has commonly no well-sustained relation, but must be allowed as a factor in *monarthritic* cases. Exposure and cold are ruled out at the present day as exciting causes, yet sometimes it seems impossible to exclude them. On the other hand autogenetic toxic agencies such as arise not only from errors in diet but also from ptosis or other displacements of the abdominal viscera resulting in the undue detention of food and consequent fermentation and putrefaction, are allowed increasing importance. Gonorrheal and tuberculous microbes are among those causing the infection types. Certainly, insufficient food seems to favor the disease. Shock, worry, care, and grief are alleged causes. Most cases of "chronic rheumatism" are forms of this disease.

As the result of the various findings in etiology and morbid anatomy there has arisen a disposition to make two or three different types of the

affection. Hoffa, R. L. Jones and others make three types (1), rheumatoid or atrophic arthritis in which the synovial membranes and periarticular tissues are especially involved; (2), osteo-arthritis or hypertrophic arthritis in which the cartilages and bones are the primary seats of invasion. The third type is based on etiological distinctions rather than anatomic and is scarcely justified; (3) infective arthritis. Others omit the third variety or type.

Morbid Anatomy.—All three of the structures which enter into the formation of the joint share in the process, the changes beginning now in the synovial and periarticular tissues and now in the cartilages and bones probably. The active changes in the cartilages consist in a proliferation of the cartilage cells, succeeded by fibrillation of the intercellular substance, which subsequently undergoes mucous degeneration, liquefaction, and absorption. Thus, the bone ends are laid bare. These subsequently become atrophied, smooth, and eburnated. The bone ends and joint cavities are alike distorted; concavities may become convexities, and convexities concavities. The edges of the cartilages where overlapped by synovial membranes, thicken and form (hypertrophic) outgrowths, which subsequently ossify and become the osteophytes which contribute to the deformity of the bone, sometimes also forming rims or lips. The effect of the latter is to impair motion without producing actual ankylosis, except in very rare instances, which may include even vertebræ. The synovial membranes also become thickened and the fringes hypertrophied. Effusion is sometimes present in the joints and in the bursæ. Fragments of cartilage may be attached to the tufts, or, becoming detached, they may lie loose in the joint. Muscular atrophy also makes a conspicuous part of the morbid changes.

Symptoms.—If the joint lesions be made the criterion of the presence of arthritis deformans, any remaining difference in symptoms depends mainly upon the grouping and extent of these lesions. Hence it is more convenient to subdivide them into clinical varieties. Two such are easily made:

1. Multiple arthritis deformans corresponding to the rheumatoid or atrophic form of the newer classification including (*a*) Heberden's nodosities on the small joints and (*b*) the progressive form, in which large joints are successively invaded in an acute or a chronic manner.

2. The monarthritic or partial form, in which one or two joints are alone attacked, corresponding to the osteo-arthritic or hypertrophic type of the newer classification.

1. Multiple Arthritis Deformans. Rheumatic or Atrophic Arthritis.

(*a*) Heberden's Nodosities.—These are prominences or nodules which develop gradually on the sides and ends of the distal phalanges, especially of the fingers and sometimes also of the toes. Women are the most frequent subjects, and the development begins usually between the 30th and 40th years, and gradually increases with age, but varies also at times and seasons independently of this gradual increase. The pain and tenderness also vary, being usually worse when the hands become cold,

and especially when accidentally struck. At other times they are insensitive. These nodosities have no relation to gout and are especially contributed by the laity to gout, but this is an error. They are quite independent of the tophaceous deposits of gout, which are altogether absent in arthritis deformans. Persons in whom they are permanently present rarely have the large joints invaded, and, indeed, are said to have promise of long life. Subcutaneous nodules, are also found in arthritis deformans.

(*b*) The Progressive Form.—This may be acute or chronic. The *acute* form simulates in its beginning rheumatic fever. Among children boys are more frequently attacked than girls. There are swelling of the joints, tenderness, and fever. These may continue without material change for weeks, or may abate to recur with increased severity; on the whole, however, growing worse, until the permanently enlarged and distorted state to be described is established.

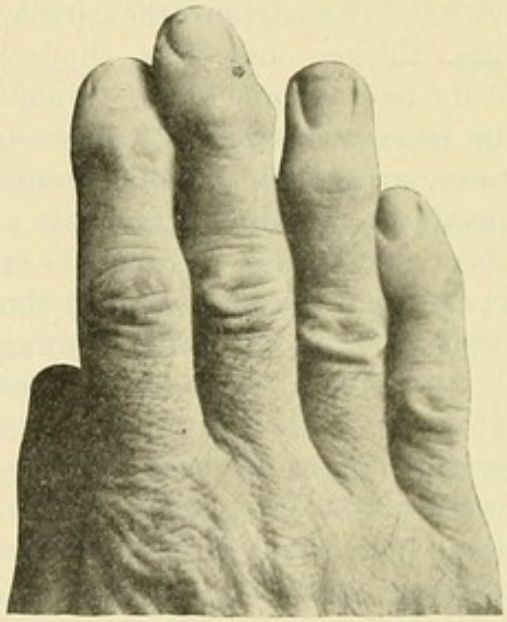


Fig. 130.—Heberden's Nodosities.
From a photograph of the hand of a patient of the author.

In the *chronic* form the same changes develop more slowly and without fever, maintaining with remarkable constancy a symmetrical order of development, the order of frequency being the hands, knees, feet, ankles, wrists, elbows, shoulders, jaws, cervical spine, hips, and dorsal spine. Striking changes are seen in the knees, which become enlarged and so fixed that the legs are constantly flexed on the thighs, and the thighs on the trunk. These *flexions* may be contributed to by *contractures*, which may, however, arise secondarily, subsequent to the flexion, or form *pari passu* with it. They are seen in the upper extremity as well as in the lower, producing the "seal-fin" deflection at the wrist and a rectangular bend at the elbow. The actual *enlargement* is exaggerated in appearance because of wasting of the adjacent muscles and thickening of the capsular ligament. Its surface becomes hard and shining. There may also be some *effusion* in the joint, though the condition has been called by the French *arthrite sèche*. Motion grows more and more difficult, until the joint is almost locked, and grating

and crackling attend attempt at motion. *Pain* varies greatly: at times it is very severe, at others it is quiescent, but it is always excited by attempt at motion. *Tingling*, *numbness* of the hands and feet, and *local sweating* and *skin pigmentations* are not uncommon among the early symptoms, and are regarded as trophic in origin. Day by day the patient becomes more helpless and, in the absence of fresh air, wan, weary, and anemic. Fortunately, in many cases the fingers are unencumbered, and the patient may be able to occupy himself or herself in some handiwork, such occupation serving to make more bearable a life of virtual imprisonment. *Weather* has its influences; diet rarely. The condition is singularly free from complications of all kinds.

Spondylitis.—The vertebral form is usually accompanied by changes in other joints. It is constantly mistaken for rheumatism, sometimes for tuberculosis, the entire spinal column is often fixed and inflexible, or sometimes only two or three if the intravertebral joints are involved.

Diagnosis.—This is rarely difficult. Arthritis deformans differs widely from *gout* in the total absence of tophaceous deposits, and from *acute rheumatism* in the absence of fever, though in the incipiency of the progressive multi-articular form there is a certain resemblance to *acute* rheumatism. But rheumatism does not leave behind a partly injured joint. Gonorrheal arthritis of polyarticular form may closely resemble the disease, but the causative factor is the one point to be depended upon.

X-ray examination of the joints showing rarefaction is valuable. Tuberculosis of the joints is another condition constantly mistaken for the condition. The atrophied shoulder of *omoneuritis* also somewhat resembles the monarticular form, but the greater tenderness and painfulness, as well as acuteness, of this affection distinguish it. The arthropathies attending *locomotor ataxia* and *syringomyelia* are distinguished by the symptoms peculiar to them and by the absence of osteophytes.

Treatment.—The most important part of the treatment is early diagnosis search for some focus of infection; in the sinus, bladder, urethra, intestine, tonsils, the removal of obvious foci will often clear up a case. The usual remedies for rheumatism are of little avail. Yet treatment is by no means unavailing, especially if instituted early, and we may always hold out to the patient the hope of arrest at some stage. The principle of treatment consists in efforts to *improve nutrition* by means of good food and tonics, of which cod-liver oil, iron, iodin, and arsenic are the most efficient. A systematic course of these remedies, continuous, except so far as judicious intermission may be necessary, will sometimes accomplish surprising results if instituted early and continued perseveringly. The iodid, either in the form of the pill or syrup, is the best preparation of iron. A grain (0.066 gm.) of the former and 15 minims (1 c.c.) of the latter are suitable doses three times a day. Massage is, perhaps, the single measure calculated to be of most use, in certain cases where the condition is confined to free contraction tissues. When there is an acute condition massage is often harmful. Disappointing as the treatment often is, in a few cases surprising results may be obtained. One of the most serious drawbacks in certain cases is the difficulty in securing outdoor life and the advantage of exercise. One of the objects of massage must be to substitute the latter,

while every possible effort should be made to have the patient in the open air as much as possible, and when his means will permit it, to take advantage of residence in warm but dry climates. It is very important to avoid the use of anodynes altogether, if possible. The relief afforded by them is but temporary, they militate against the effort at securing an improved nutrition, and, above all, there is danger of forming the morphin habit. Simple support by splints is sometimes a comfort to patients. The treatment by *hydrotherapy*, as carried out at Aix-les-Bains and Aix-la-Chapelle in Europe undoubtedly affords temporary relief. The same may be said of the treatment at the Hot Springs of Arkansas, Virginia, and North Carolina in this country, and at St. Catharine's in Canada. While general steam baths are contraindicated by reason of their debilitating effect, local vapor baths applied to separate limbs or portions of limbs by a specially constructed apparatus are sometimes useful.

Much was expected first from the hot dry-air or Tallerman-Sheffield treatment of this disease. Temporarily it does produce relief of pain, and certain writers claim permanent benefits have been secured.

The bowels should receive close attention, the body should be frequently bathed, preferably in warm water, and all measures desirable to secure the most perfect personal hygiene should be practised. This is another of the few diseases where an abundance of good, nourishing food is necessary. This is the more important when we remember that many cases originate among the poor and badly fed. These persons may take meat with great benefit. Much harm is occasionally done by putting the patients on a limited diet.

OSTEITIS DEFORMANS.

SYNONYM.—*Paget's Disease.*

Definition.—A chronic nonsymmetrical overgrowth of bones resulting in an enlarged head, dorso-cervical kyphosis, enlargement of the clavicles, flaring of the base of the chest and outward and forward bowing of the legs. The condition was first described by Sir James Paget in 1877, whence the name Paget's disease. Packard and Steele[1] collected 100 cases up to the date of their paper in 1891 but they cut out 34 as lacking *essential conditions, reducing the number of typical cases to* 67.[1] J. Chalmers Da Costa has added two not yet published of which one was for a time at the Hospital of the University of Pennsylvania. Higbee and Ellis estimate the total number up to the date *of their paper*, 1911, *to be* 158 *of which* 23 *were found in this country.*[2]

Its **etiology** is unknown. A mother and daughter are known to have had it. Also two brothers. It is more common in males. The average age of the cases coming under the observation of Packard and Steele was 61 but it may start at 21. Its subjects are generally past middle life. Some have ascribed the disease to syphilis, others to arterio-sclerosis or perverted internal secretion.

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[1] "Amer. Jour. Med. Sci.," vol. cxxii., p 552, 1901.
[2] Case of Osteitis Deformans, "The Journal of Medical Research," vol. xxiv., Jan., 1911.

Symptoms—The disease may begin with rheumatoid symptoms including extreme pain worse at night but this is not essential. The symptoms are chiefly objective. Attention may be attracted to the enlarged head by the fact that the hat has become too small, or the patient's friends remark that he is growing shorter and that his legs are becoming bowed. A patient of Osler's lost 13 inches in height. Headache, bronchitis and pigmentation of the skin may be present. The skull, spine and long bones are most affected, the face, hands and feet less. It may be confined to the femurs. In one variety the tibiæ and fibulæ were alone involved. The process is a thickening by new formation subperiosteal and myelogenous producing a fibro-osteoid tissue which does not calcify. The shafts of the bones are thickened and heavy, often twice as heavy as the normal bone. The skull may be 3/4 inch (19 mm.) thick and 71 cm. (28.4 inches) in circumference. In one variety the bones are deformed by multiple hyperostoses whence this variety is called "tumor forming." The dorsocervical kyphosis as well as bowing of the long bones contributes to reduce the stature.

Diagnosis.—This is easy when the disease is established. The coarser changes in acromegaly are similar, but in osteitis the face is triangular with the base upward; in acromegaly the face is egg shaped with the large end down. It is allied to osteomalacia and the two conditions merge but in osteitis deformans regeneration takes place synchronously with absorption. Fractures do not occur in osteitis.

Treatment is unavailing.

OSTEOGENESIS IMPERFECTA.

SYNONYM.—*Fragilitas Ossium.*

A condition of the fetus in which its bones fail to develop normally, reaching at birth a stage of great fragility wherein fractures are so easily produced that they may have occurred *in utero*. The defective development extends to the cranium and the fragility to all bones. At other times the extremities are bent and deformed. Though the disease is commonly fatal the bones sometimes repair and as the child grows older a natural firmness is acquired.

OSTEOPSATHYROSIS.

SYNONYMS.—*Fragilitas Ossium*; *Lobesteins Disease.*

Definition.—A condition characterized by great brittleness of bones and frequent fractures in consequence.

It is further characterized by the fact that the general health of the patient is otherwise good though the fractures may number as many as a hundred or more in a single case. They are generally painless and heal rapidly. They cannot be said to be spontaneous because they result from trifling causes such as turning over in bed, a slight blow, or even so trifling a cause as the act of chewing which may fracture the jaw. It

contrasts further with fractures of the more usual kind in that it occurs in the young rather than the old.

OXYCEPHALY.

SYNONYMS.—*Tower or steeple head.*

Definition.—A deformity of the cranial vault resulting in abnormal vertical dimension associated with exophthalmos and defective vision without mental derangement. It is further characterized by feebly marked supraorbital ridges. The forehead slopes to a pointed vertex and the scalp rising also abnormally gives the appearance of being set on the top of a comb. Usually present at birth it may appear as late as the sixth year.

The deformity is ascribed to premature synostosis of certain sutures especially the coronal. The brain thus restricted grows vertically instead of laterally and anteroposteriorly. The closure of the anterior fontanelle is delayed, its site displaced, but closure ultimately takes place, the original site being covered by thin and prominent bone.

The visual defect is due to optic neuritis and atrophy caused by the internal brain pressure as in tumor of the brain; whence too, the exophthalmos and headache and it may be the occasional loss of the sense of smell.

Decompressing operation has been done in the way of treatment.

LEONTIASIS OSSIA.—Is a condition which is characterized by bony outgrowths upon the cranium and face. A case at present under the care of one of us has great protuberances upon the temporal bones and upon the vault. She has at the same time a steadily increasing general arthritis. The entire head is distorted.

GOUT.

SYNONYM.—*Podagra.*

Definition.—An acute and chronic constitutional affection, due to an abnormal accumulation of uric acid and other purin bodies in the blood and tissues, causing various symptoms, of which arthritis is the most distinctive and significant..

Etiology.—The tendency to gout is more frequently inherited, than acquired. Between 50 and 60 per cent. of all cases of gout can be traced to ancestry, parents or grandparents. More men are gouty than women, and it is the male line through which the tendency is most frequently transmitted. It is not usually manifested until after 40 years of age, sometimes later, but the signs which are almost sure to eventuate in gout may show themselves before the 12th year. While overeating, especially of meats, and intemperate drinking, associated with the luxurious habits which grow out of the possession of wealth, are the most frequent causes of acquired gout, these last are by no means essential. Sir Dyce Duckworth's studies of gout in what is probably the richest field in the world, London, go to show that many of the peasantry of Ireland, among whom gout is unknown, became gouty after having lived for a time in London. This may be due to free indulgence in malt liquors. Such gout is often

spoken of as poor man's gout. Not every person who inherits a tendency to gout becomes gouty, since the fostering causes previously mentioned may be wanting. In others this tendency is so great as not even to require the favoring condition. Negroes are not exempt, and Osler reports three out of 59 cases admitted to Johns Hopkins Hospital, up to April 1, 1905.

While alcohol is an acknowledged cause of gout, it has been observed that something depends on the shape in which it is presented. Malt liquors, especially the "heavy" English ales and beers, strong in alcohol, are more active in the production of gout than the lighter beers consumed in Germany and this country where gout is less common. Strümpell believes, however, that gout is more common in Germany and especially in Bavaria than is commonly supposed. The strong and sweet wines, of which port and sherry are the type, are strongly predisposing, while pure whisky is less harmful. According to Futcher "It is quite probable that the increase of uric acid in the blood and urine, after the ingestion of alcohol and purin containing food, is due to the inhibitory action of alcohol on the "oxidase" which normally oxidizes uric acid into urea."

An interesting cause of gout is lead-poisoning. This is seen particularly in England and especially in London, as pointed out by Sir Alfred Garrod in 1854. It is, however, rare in other parts of Great Britian and Ireland, and is growing more infrequent in London; for in 1870, according to Garrod, 33 per cent. of people who suffered from gout had been poisoned by lead, while Sir Dyce Duckworth, up to 1890, found only 18 per cent. in hospital cases. It is a rare cause also in France and Germany. It may be, as suggested by Alexander Haig, that the effect of plumbism is to diminish the alkalinity of the blood and thus its solvent power for uric acid which is consequently precipitated. In this country the combination is comparatively rare.

Food and Exercise.—The relation of food taken to exercise indulged in is important. Improper amounts of food difficult to digest, especiably if much alcohol is used and little exercise taken, unquestionably predisposes to gout.

Injuries and blows on susceptible parts, and so slight a cause as pressure by a boot, are often predisposing causes. On the other hand, worry or shock may be exciting causes.

Pathogenesis.—Many theories have been proposed as to the metabolism in gout. For the exposition of those theories, the reader is referred to the various systems of medicine. The following statements seem to be well established. Gout is caused by faulty metabolism, which eventuates in the accumulation of an excess of uric acid in the blood and tissues. It has been established that there is a specific intracellular enzyme called nuclease, which has the power of liberating the purin bases, adenin, guanin, hypoxanthin and xanthin from the nucleo proteids of the tissue or of the food. These bases having been liberated are acted upon by the ferments, adinase, guanase, and xanthin oxidase which are present in the thymus, adrenals, pancreas, lungs and liver, and step by step the uric acid is built up. Schittenhelm has further shown that there is another specific tissue oxidase present in the kidneys, and muscle, which has the power of oxidizing the uric acid, and thus destroying it. The chief fault

in the metabolism would appear to be the lack of power to destroy the uric acid formed. It would therefore appear that persons who have difficulty in getting rid of the purin bases thus formed and destroyed, have gout in one form or another, whether it be in an acute attack or in the deposit of the biurate about the joints or in the tissues.

Morbid Anatomy.—As will be further evident in treating the symptomatology of gout, there is scarcely a tissue which may not be affected by it, but the morbid conditions which are more distinctive are, first, the characteristic inflamed great toe of acute gout—the true *podagra*. The angry, swollen, dark-red or mottled appearance of such a toe once seen is not forgotten. Similar though less striking changes are sometimes seen in the metacarpophalangeal articulation of the thumb.

The superficial changes in chronic gout are less distinctive, and are often not different from chronic arthritis due to other causes. But wherever uratic deposits are present in the tissues, there, by universal consent, is gout. They are found most often in joints and in the parts around them: first, the cartilages of the movable joints, then the ligaments, tendons, bursæ, and, finally, the connective tissue and skin, this being the order of feebleness in vascularity and nutritive activity. Frequent situations are the digital joints and cartilages of the ear; more rarely the cartilages of the nose, the vocal cords, the cornea, kidneys, marrow of bone, and expectoration. Cartilages impregnated with urates present the appearance of being smeared with whitewash or white paint, and when preserved in pure alcohol, maintain it for a long time. Minutely examined, cartilages are infiltrated on the peripheral surface, more rarely beneath, with acicular crystals of sodium urate. Rarely they are found in the bone under the cartilage. The cartilage cells are for the most part free, and after the urates are dissolved out, the tissue appears natural or slightly granular. The tophaceous deposits are the best known and most characteristic lesion of gout. About the digital joints, especially the knuckles of the hands, they sometimes ulcerate through the skin. The deposits are often associated with deflection of the fingers to the ulnar side—"seal-fin" type of hands—and of the toes outward, a late symptom not confined to gout—in fact, more common in rheumatoid arthritis. It is due to stronger action of the abductor muscles.

Deposits of biurate of soda called tophi appear on the helix and antihelix of the ear, in the subcutaneous tissue of various subcutaneous tissue of the forearms, tendo achillis, etc. The gouty tophi occurring about the phalangeal joints, must not be confounded with Heberden's nodosities, which are characteristic of rheumatoid arthritis. Their significance must be determined by other symptoms more essential to gout. They vary in prominence and tenderness at different times, being worse in gout, especially after errors of diet, but on the whole they slowly increase with the age of the patient and the persistence of other symptoms.

Of undoubted gouty nature are the little vesicles over the nodosities, called "crab's-eye" cysts. To these are to be added certain exostoses and ecchondroses, or "lippings," beneath the synovial membrane, at the edges of the cartilages, and round the heads of the phalanges, and even of larger bones like the femur, patella, and tibia.

Changes in the *internal organs* are most often confined to the kidneys and vascular system. Deposits of urates have been referred to. They are not constant, and are found usually in the interlobular tissue toward the apices, but also more rarely in the tubules. Ultimately the gouty subject acquires an interstitial nephritis, the well-known gouty kidney; though the term "gouty kidney" has also been applied to kidneys the straight tubules of which are found filled with uratic sediments, as is the case sometimes at necropsy. Arterio-sclerosis is almost always present, and must now be ascribed to the irritative effect of the purin bodies, which include xanthin bases on the one hand and uric acid on the other. The heart is hypertrophied in its left ventricle. There may be deposits of urate of sodium on its valves. Changes in the lungs are mainly confined to emphysema, which is found in many cases of long standing.

Symptoms.—*Of Typical Acute Gout.*—Persons subject to attacks of gout sometimes have *premonitory symptoms* suggesting the approach of an attack. These vary with the individual and are significant only in each case. They may be headache, neuralgia, any one of the numerous manifestations of deranged digestion, irregularity of the heart's action, palpitation, high tension of the pulse, depression of spirits, drowsiness, a disposition to yawn, a tired feeling—in fact, any symptom which the patient learns to associate with the attack. Attacks are apparently also invited or determined by anything which lowers the vitality of the patient. On the other hand, a supper with wine or a single glass of champagne will often produce an attack.

The first actual symptom of the typical attack is *articular pain, commonly in the great toe*, at the metatarsophalangeal joint, and with its appearance the premonitory symptoms usually pass away. The pain is extremely severe, sharp, shooting, and sudden, often arousing in the middle of the night a patient who has gone to bed apparently well and least expecting an attack. With this pain are the *swelling, heat,* and *discoloration* already described under morbid anatomy. Rarely, the attack begins with a *slight chill.* On the other hand, there may be pain without heat, redness, or swelling, and all the typical local anatomical features of an attack without pain. In some instances the attack develops more slowly. At times the first attack is so little distinctive that it is assumed to be something much more trifling, such as rheumatism or some slight injury, while the personal peculiarities, natural or acquired, always more or less influence the symptoms. After the outburst at night the extreme pain diminishes as morning advances, but it may recur the next night, and this goes on for four, five, or six days, when the attack terminates.

Some *fever* usually accompanies the onset of acute gout. The temperature promptly rises to 100° F. (37.8° C.) and even higher, but does not far exceed it, 102° F. (38.9° C.) being the usual maximum attained. As in other acute diseases, the temperature is higher in the evening. The local temperature, notwithstanding the sensation of heat, is five or six tenths of a degree below that of the axilla at the same time. The attack terminates with *desquamation* of the epidermis over the inflamed joint. During the acute attack a leucocytosis develops.

Changes in the Urine.—It is scanty, acid, highly colored, and of high

specific gravity. It deposits uric acid and urates on standing and cooling but this deposit is not an index as to the quantity of uric acid excreted. It often contains a small quantity of albumin.

Futcher has made some interesting studies as to the relations of uric acid and phosphoric acid excretion to the acute attacks. Both increased nearly *pari passu* after a low output before the attack and in its early part, but rose to normal limit shortly after the onset. Hematuria and oxaluria may occur.

A recognized symptom of acute gout, and sometimes the only one, is *pharyngitis*, and now the term "gouty sore throat" is one in common use, though it is doubtless also often used carelessly, many cases of infectious sore throat being maltreated on the ground that they are gouty in character. There seems no way of distinguishing it locally from other forms of sore throat in which there is no decided swelling.

Gout is said to be *retrocedent* or *metastatic* when it disappears suddenly from its external site and there are substituted for the outward symptoms derangements of some internal organ, especially the *heart* or *stomach* or *brain* or *urinary bladder*. In the first there appear cardiac symptoms of varying severity, including pain, shortness of breath, and irregularity in the heart's action; in the second, gastrointestinal pain, a sinking sensation, vomiting or diarrhea, often associated with intense mental excitement or depression; in the third, meningeal symptoms; and in the fourth, cystitis and prostatitis. More rare events are gouty orchitis, parotitis, and urticaria or other fugitive skin affections. Metastasis is more prone to occur in atonic cases. Sudden death has supervened in some instances, but postmortem lesions of a definite kind seem to be wanting, at least lesions which can be held responsible for the symptoms.

Of Irregular or Atypical Gout.—This includes a set of symptoms not so distinctive in themselves as peculiar in this, that they occur in persons who have had gout or who have a decided hereditary tendency thereto. These conditions being fulfilled, there is scarcely any superficial or visceral symptom which may not be of gouty origin, but among them may be named cutaneous eruptions, gastrointestinal disorders, various forms of headache and neuralgia, hot and itching palms and soles, especially at night, a similar condition of the eyeballs, lumbago and other muscular pains, cramps in the legs, arterio-capillary fibrosis and its consequences, iritis, bronchitis, pericarditis, cystitis with hemorrhage into the bladder, and others.

Some affections of the *teeth* occurring under the same conditions may be regarded as gouty. On the other hand, an easy method of satisfying a patient and hiding one's ignorance under a name is to make a diagnosis of "uric acid diathesis" or "uric acid in the blood" without the least foundation. Such terms should be banished from medical literature.

Among other organs the *eye* in its blood-vessels, retina and optic nerve falls heir to changes which are ascribable to gout, but the same law as to their necessary relation holds, by which we mean that identical conditions occur which are not due to gout, and the conclusion that they are thus related depends upon a definite knowledge of the previous existence of gout in the patient. An exception to this exists in the rare cases of actual

deposits of urates in certain situations, as the cornea, the crystalline lens, vitreous humor, and even the retina.

Of Chronic Gout.—As repeated attacks of gout occur and the patient grows older, there gradually accumulate the morbid changes described under morbid anatomy as more or less characteristic—the joints deformed by tophaceous and other deposits, the lipping, the seal-fin hand, the renal and arterio-vascular changes, interstitial nephritis, etc. The urine now is increased, lighter hued, and contains albumin and a few hyaline and granular casts.

Some further allusion should be made to the deformities thus resulting as symptoms of chronic gout. They appear especially in connection with

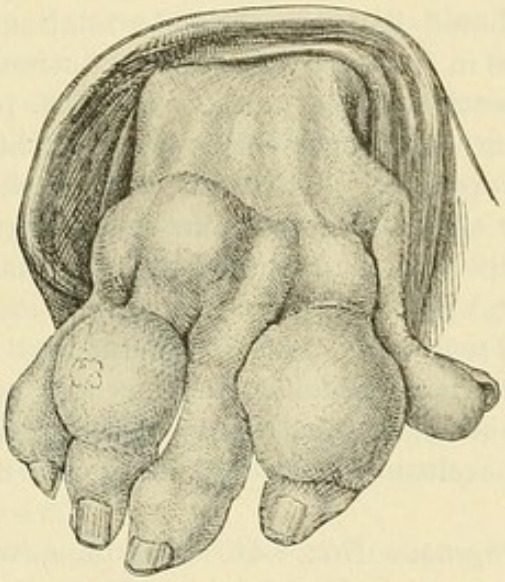


FIG. 131.—Tophaceous Gout.
Both hands were symmetrically affected, man aged sixty—(*after Duckworth*).

the toes and fingers, causing swellings, deflections, and torsions which produce the most fantastic shapes. Among these are deflected and abducted toes. The seal-fin hand, and the deformities caused by tophaceous deposits. It is important to remember that any of these except the tophaceous deposits may be due to rheumatoid arthritis as well as gout. The appended cut from Duckworth illustrates the appearance of enormous tophaceous deposits undoubtedly of gouty origin as contrasted with Heberden's nodosities.

Diagnosis.—Only two events point unmistakably to gout in an individual, viz., podagra or tophi in some portion of the body, ears, forearms or joints. Gout prefers the distal, smaller joints, and one of these rather than many. Frequent recurrence in the same joint is characteristic of gout rather than articular rheumatism. The gastrointestinal symptoms of gout are in no way distinctive. Nor are the skin affections or interstitial nephritis or cardiovascular changes, as the same may arise from other causes. Yet if they occur in a person who has had podagra or tophaceous deposits they are probably due to gout. Gout and cardiovascular changes may result from the same cause. The presence of lead-poisoning, on the other hand, lends support to a diagnosis of gout. The continued presence of uric acid sediments in the urine also lend a support to the diagnosis of

gout. On the other hand, after interstitial nephritis has set in the urine may be increased in quantity, of low specific gravity and light colored. Nodosities occur in gout or in rheumatoid arthritis alike, but the presence of crab's eye vesicles on them points to gout. A certain unexplained relation exists between gout, diabetes and obesity. All of these or any two of them occur rather frequently in the same individual; or they may alternate.

Treatment.—The treatment easily divides itself into two parts: *first*, that of the inherited tendency; *second*, treatment of the paroxysm or of the acute attack.

Treatment of the Inherited Tendency.—It is plain that those who inherit a tendency to gout should be taught from the earliest possible moment that, food and exercise should have an intimate relation. Food of moderate quantity and exercise in fresh air that keeps the strength up to par and the appetite good are essentials. The patient should be protected from sudden changes of temperature by proper clothing. Malt liquors and sweet wines should be especially excluded. Those who work hard and have insufficient food must be taught that malt liquors are to them a poison, that fresh air is essential, that milk and eggs should be the main stay of their diet rather than meats and pastry.

I. *The Dietetic Treatment.*—This is by far the most efficient of the treatment of gout, without which all else is only palliation. It consists in the proper combination of food taken, of the regulation of the *amount*, and of the practical exclusion of alcohol, especially malt liquors and light wines.

A Modified Nitrogenous Diet.—It is perhaps well proven, that the foods rich in nucleoprotein should be avoided, therefore, sweet-breads, liver, kidneys and brain should be debarred. Beef extracts are injurious. White meat on the whole is preferable to red meats because the latter contains more extractives though, boiled or stewed red meats may be allowed in moderate quantities, when broiled or roasted can be taken in small quantities. Meat soups and beef extracts should not be used. Fish may be taken in moderation. Eggs and milk are the safest forms of protein diet. Cheese is permissible. Vegetables and carbohydrates may be taken in any rational amount. Fruits may be eaten, although certain individuals have idiosyncrasies toward certain ones. Fats may be used but should be used in quantities which will not upset the digestion. Alcohol should not be used in any form, it certainly inhibits the function of the liver in destroying uric acid. If alcohols are insisted upon by the patients they should take whisky and brandy and not malt liquors. Tea and coffee can be used in moderation, never in excess, they are better than alcohol. The *amount* of the food is important, never excessive. Any amount which leaves a feeling of fullness or causes joint pains, afterward is too great.

An abundance of any good drinking water. So-called mineral waters, are still extensively used. They are probably not a whit better than any potable water.

Residence at Spas is desirable for those who can afford it, not so much became of the efficiency of the water, but because of the proper diet, the amount of water and amount of exercise taken as a part of the treatment.

If alkaline waters are demanded by the patients, Vichy, Baden Baden, Kissingen and Saratoga waters may be allowed.

The bowels should be kept moving daily. A habit can be established of daily evacuation. Salines such as magnesium sulphate, Rochelle Salts are best, this may be given in the form of Hunyadi water.

II. *The Medicinal Treatment and the Treatment of the Acute Attack.*—As a rule, the use of medicines is reserved for the acute attack. From the earliest history of the disease practice has recognized two classes of remedies in the treatment of gout—alkalies and purgatives—the object of both being to eliminate the offender, the first by producing soluble combinations which pass off readily by the kidneys, and the second to carry it off by the bowels. It is plain that a combination of the two principles might be expected to be more efficient than either one alone.

First as to alkalies and alkaline combinations. Salicylate of sodium is easily at the top, and while it is not so rapid in its effect in relieving the pain of an acute attack of gout as it is in rheumatism, it is nevertheless an invaluable remedy, excelling all others. During an attack it should be given in doses as large as can be borne. As a rule, adult men easily bear 15 grains (1 gm.) four times a day, or 10 grains (0.65 gm.) may be administered every two hours. Even larger doses may be given with advantage, if borne by the stomach. With relief to the acute symptoms the dose should be reduced; but, as in rheumatism, the remedy should not be discontinued, and between attacks smaller doses should be kept up for some time. These, however, may be substituted by the natural mineral waters to be presently alluded to.

After the salicylates, the alkaline carbonates have always held a high position in the treatment of gout. Half an ounce (15 gm.) of potassium bicarbonate a day in divided doses should be the initial treatment, continued, in smaller doses, when relief comes to the acute symptoms. It may be combined with a little lemon-juice to improve the flavor, or the citrate of potassium may be given in the same doses.

Among the eliminating remedies is the time-honored colchicum, a drug which is of undoubted value in gout, but which, in Tyson's experience, must yield the palm to salicylate of sodium. For a long time its action was inexplicable, and it came to be known as a specific in gout as quinin is in chills and mercury in syphilis. Modern studies have, apparently, solved this problem. Professor Rutherford has shown that it is one of the most powerful cholagogues known. This, taken in connection with what we now know of the office of the liver in urea formation, simplifies very much the solution of the problem. It explains, too, why colchicum produces its sedative and anesthetic effect without necessarily causing purgation. Indeed, some, as Sir Alfred Garrod, consider that its effects are best attained without purgation, and Garrod says that if cathartic action is required, it is better to combine some aperient with the colchicum, as when much purging and vomiting results from colchicum, nervous and vascular depression follows. It is not necessary to produce either violent purging ot vomiting. Whatever its mode of action, it sometimes operates in the most magical manner in relieving pain. The preparation commonly used is the wine. In this country the wine of the seeds is no

longer official, so that if the wine is ordered, that of the root is dispensed. This is more powerful than the wine of the seeds.

Colchicin, the active principle of colchicum, is also employed. Its dose is 1/50 grain (0.0013 gm.). The same dose may be employed hypodermically. A favorite modern remedy is the salicylate of colchicin in doses of 5 minims (0.31 c.c.), given in pearls or capsules. The other aperients commonly used in gout are the sulphates, of which magnesium sulphate is the favorite. Sodium sulphate is also used, and it is the constituent of the most actively purgative mineral waters already mentioned, viz., the Hunyadi János, Rakoczy, and Friedrichshalle, now largely used instead of the pure salt. It is also the largest constituent of the Carlsbad waters.

Colocynth is also employed as an aperient in gout, and advantage has been taken of this fact in the preparation of the secret remedy known as Laville's tincture, which is very largely used by the laity, and which undoubtedly has a very prompt effect in many cases of acute gout. The following has been published[1] as the composition of Laville's remedy, as determined by analysis:

Quinin	5 parts.
Cinchonin	5. parts.
Colocynthin	2.5 parts.
Lime salts	5. parts.
Water	82.5 parts.
Alcohol	100 parts.
Port wine	800 parts.

The lithium compounds—the carbonate and citrate—have not proved so useful as to cause us to prefer them to salicylic acid. Indeed, the early results of Garrod with them cannot be said to have been realized in modern therapeutics. Sir Dyce Duckworth says of lithia that it is a remedy better adapted to the chronic than to the acute phases of gout, and so we have been using it. Five grains (0.3 gm.) four times a day, freely diluted, is the dose usually administered, and with this the potassium salts are sometimes combined.

Local Applications.—For the relief of the acute attack of gout, leeches, blisters, and cold have all been discontinued, not only because they are useless, but also because their use has been followed by fatal attacks of the so-called internal gout. Warmth and moisture do. however, have a mollifying effect, which is increased if the liquid preparations of opium be associated.

It often happens that the pain in a paroxysm of gout is so severe that it is impossible to wait until the effect of the foregoing remedies is secured, and a hypodermic injection of morphin is absolutely necessary to relieve the sufferings of the patient.

All pressure by boots on joints disposed to gout should be carefully avoided, as well as injuries, as such influences undoubtedly act as predisposing causes. Muscular and mental fatigue are exciting causes of acute attacks, and should be avoided by the gouty.

Treatment of Retrocedent Gout.—The true nature of a metastatic attack having been determined, it must be relieved symptomatically, while

[1] "Druggist's Circular," October, 1889.

efforts to stimulate a true external attack may be made by the hot mustard foot-baths, sinapisms, and the like. It has even been suggested that a pint of champagne may be advised, this being the wine most frequently responsible for acute attacks.

DIABETES MELLITUS.

Definition.—A condition of deranged metabolism characterized by a constant copious secretion of a urine charged with glucose and due to some as yet imperfectly understood derangement of the glycogenic and glyco-destructive functions of the organism.

Geographical and Racial Distribution.—Diabetes is not a common disease anywhere, and it is variously frequent in different countries and races. The latest census report (1910) shows the death rate from diabetes in the United States to be 14.9 to 100,000 population, somewhat greater than the rate found in European countries. According to Dickinson, disease is more widely prevalent in the agricultural countries of England than in the cities. It is common in Sweden, on the one hand, and in southern Italy and India, especially in Ceylon, on the other, while especially rare statistically in Holland, Russia, and Brazil.

It is much more frequent among Hebrews than among Christians in the experience of almost everyone. It is rare in the negro race. It is a disease especially frequent among the rich and well-to-do, though the poor are not exempt. It is also a disease of adults, yet it has occurred in infants at the breast. The disease is most frequent between the ages of 30 and 60. It is more serious in the young, recovery in very young subjects being almost unknown. It is much more frequent in males than in females, in the proportion of nearly three to one, though Senator's[1] statistics show that under the age of 20 more females are affected than males. Little is known of the effect of occupation, though it is thought that occupations taxing the mind favor it. Heredity has in Tyson's experience been less conspicuous than European writers find it. From ten to 25 per cent. are thus traced by different Continental observers. On the other hand, it may occur in several members of a family. It is not unusual to find diabetes mellitus in some members of a family and gout in others.

Etiology.—Inseparably connected with the etiology of diabetes are the phenomena of sugar formation in the economy. During life there is constantly being produced and stored in the liver and muscles of man and the lower animals an amyloid substance, which was named by its discoverer, Claude Bernard, *glycogen*.[2] Its formula is C_6, H_{10}, O_{15}, that of starch, and the term *zoamylin*, or animal starch, was at one time suggested for it. The glycogen formation takes place whether animal or vegetable food be taken, but it is much larger upon a vegetable diet.

In health the amount of glucose in the blood is remarkably constant, ranging from 0.1 to 0.15 per cent. The source of this sugar in the blood is largely the carbohydrate food taken into the stomach. These starches are converted by intestinal and salivary digestion into simple sugars and

[1] See Senator's article on "Diabetes Mellitus" in "Ziemssen's Cyclopædia of Medicine," vol. xvi., p. 866, *ad fin.*

[2] Bernard, "Nov. Fonc. du Foie," Paris, 1853.

are absorbed largely in the form of dextrose, levulose and glucose. They are carried to the liver in the form of these simple sugars, and are converted into glycogen by that organ and stored there and in the muscles. It is evident that there are two other possible sources of sugar, the proteins and the fats.

It is now proven that the proteins are split by metabolic process into amino-acids, and from the fatty acids of these amino-acids glucose is formed, and is used in the economy.[1] The amount of sugar thus derived in normal metabolism does not exceed 100 grams per day according to Alonzo Taylor. This author believes also that the question as to whether the sugar of the blood is derived from the fats, is one of the future. Fat is resorbed from the intestines as such, and if there is any conversion of fat into sugar, this also must be a metabolic process.

In starvation sugar is certainly formed from the protein tissues and possibly from the fat of the body. The conversion of fat into sugar under these changed metabolic relations, is maintained by Von Noorden, "New Aspects of Diabetes," New York, 1912, who believes "the liver uses fats for the purpose of forming sugar, only when the poverty of other materials makes it necessary."

The glycogen formed from the food is stored in the liver and in the muscles in about equal amounts, about 150 grams being the maximum in each. C. von Noorden also considers that fat is converted into sugar in the liver.[2]

The presence of glucose in the urine, in appreciable quantities, is an abnormal condition, and its persistence, due to some change in vital organs, constitutes the disease, diabetes mellitus.

Glucosuria, or the presence of sugar in the urine, may be due to the overindulgence of glucose, to a temporary or permanent disturbance of the nervous organism, of the pituitary body, of the thyroid, or of the adrenals.

The term diabetes mellitus should be confined to that condition of glucosuria associated with thirst, polyuria, and permanent disturbance of sugar metabolism. As the basis for this permanent disturbance, there appears to be always, either an organic or a functional change of the pancreas.

Alimentary Glucosuria.—In perfect health glucosuria cannot occur from ingested starch. The normal glycogenetic function of the liver is superior to the digestive capacity of the intestine for starches (Taylor). Clinically, however, cases in which excessive intake of starches is at once followed by glucosuria are well known. There are individuals who are apparently in health but are usually fat. The sugar in the urine is readily controlled in these cases by limiting the starches, and one must consider that such individuals have some impairment of the glycogenetic function of the liver though apparently well. On the other hand, excessive ingestion of carbohydrates in the form of the sugars, especially glucose, may easily cause glucosuria in a normal individual. This fact is made use of in determiningthr carbohydrate tolerance of individuals. Normal individuals can take 100 grams of glucose on an empty stomach without developing

[1] Digestion and Metabolism, A. E. Taylor, 1912.
[2] New Aspects of Diabetes, New York, 1912.

glucosuria. Any case which develops glucosuria on taking a slight excess of starch, must be viewed with great suspicion as probably one of diabetes mellitus.

Glucosuria from Disturbance of the Pituitary Body.—It has long been known that acromegaly is often accompanied by glucosuria It has remained for Cushing to establish on a firm basis the relation btween disease of the pituitary gland and glucosuria. In this work published in 1912 Cushing makes the following statements based both upon clinical and experimental experience. "Normal posterior lobe, activity is essential, to effective carbohydrate metabolisms. An intravenous injection of posterior lobe extract produces glycogenolysis, and its continued administration in excessive amounts leads to emaciation. A diminution of posterior lobe secretions leads to an acquired high tolerance for sugars." In certain cases of excessive activity of the hypophysis there has been constant glucosuria with emaciation which dominated the whole picture for a long while.

Glucosuria Due to Disfurbance of Cromaffin System.—It is a fact established by Herter and others, that the injection of Adrenalin will cause rapid and severe glucosuria, but repeated injections fail to have as great an effect, and finally it looses its power. This action is antagonistic to that of the pancreas and is but temporary in effect. The clinical value of this observation is not well established.

Glucosuria Resulting from Nervous Disturbance.—Claude Bernard's puncture of the medulla between the levels of the origin of the vagus and auditory nerves (the well known "piqure") long since proved that certain disturbances of the nervous system can cause glucosuria. This same center can be excited by tumors and injuries and it can also be stimulated by fright, pain, and other nervous stimuli, thus causing glucosuria in the persons affected.

Von Noorden believes that this stimulation is carried through the sympathetic to the adrenals, and the latter are stimulated. Here glucosuria due to this type of stimulation is really adrenal glucosuria. He also believes that true diabetes can be greatly influenced by certain nervous disturbances. He believes there is no such thing as a true neurogenous diabetes. The adrenal and nervous glucosuria is only transitory. There is much danger in concluding that recurrent glucosuria is purely of nervous origin, for almost without exception these cases are neglected until one is face to face with well developed diabetes mellitus. There can be little doubt that shock to the nervous system may also be an exciting cause of glucosuria.

Pancreatic Glucosuria. True Diabetes Mellitus.—Opie in 1903 published in book form his classic work upon the effect of degeneration of the pancreas upon diabetes. Nineteen cases of diabetes with disease of the islands of Langerhans were cited. Since that time practically all experimenters and writers united in the opinion that all cases of really chronic diabetes are due to pancreatic insufficiency.

This insufficiency is due either to disease of the Islands of Langerhans or of a functional impairment of the pancreas. The action of the pancreas upon the sugar production is one of inhibition.

When the Islands of Langerhans are destroyed, the inhibitory power of the pancreas is removed, and more sugar enters the blood, a hyperglycemia results, and from this glusosuria, constituting a true diabetes mellitus accompanied with thirst, polyuria, and emaciation and sometimes with the tendency to acidosis.

The Liver.—Curiously enough diseases of the liver do not in any way interfere with the amount of sugar in the blood and do not so far as is known cause glucosuria. It is probable that cases of liver disease with diabetes are accompanied by disease of the pancreas.

The Kidneys.—Experimentally a severe glucosuria can be caused by the injection of *phloridzin* into animals. This glucosuria, according to Taylor, is not preceded nor accompanied by hyperglycemia, but is due to the reductions of the property of the kidney to restrain the elimination of glucose. It is doubtful if disease of the kidney is ever the cause of glucosuria, though cases of gouty nephritis are occasionally accompanied by glucosuria.

Morbid Anatomy.—The most important change in diabetes is in the *pancreas*. In a large series of cases 50 per cent. to 97 per cent. of the cases were accompanied by disease of the pancreas. The islands of Langerhans were always diseased where the pancreas was involved.

The *liver* is frequently enlarged and cirrhotic and in certain cases "bronzed diabetes, there is a cirrhosis of the organ.

The *kidneys*, primarily unaffected, are in many cases sooner or later influenced by the constant hyperemia to which they are subjected in eliminating the sugar. The appearances commonly met are those of hyperemia and overgrowth of epithelium—in a word, those of catarrhal nephritis. Occasionally the changes are more advanced, and the epithelium is fatty. More rarely granular contracted kidney is present, contributing a more serious significance to the albuminuria. These changes are not necessarily attended by albuminuria previous to death.

The *lungs* are often the seat of tubercular deposits and cavities resulting from their softening; also of bronchopneumonia and croupous pneumonia, which may terminate in gangrene. In many cases of diabetes the heart is found normal or corresponds to the general nutritive condition of the patient. Quite often it is enlarged.

Symptoms.—Almost invariably the earliest symptom noticed by the diabetic is *thirst*. Frequently diabetics drink quarts of water in twenty-four hours. *Polyuria* is one of the cardinal symptoms, three to four quarts of urine often being passed in twenty-four hours causing much loss of sleep because of frequent rising at night to urinate. The urine is of high specific gravity. It occasionally happens that a *dryness of the fauces* and a glutinous *viscid* character of the *saliva* attract attention before any other symptom. Sometimes it is observed that a drop of urine falling upon the boots or clothing and evaporating there, leaves a persistent white or yellowish spot due to sugar. *Dryness and harshness of the skin*, due to absence of perspiration, soon make their appearance and early attract the attention of those who ordinarily perspire freely, and occasion varying amounts of discomfort. *Itching* of the skin is frequently present, especially about the pubic bones. The *temperature* of the body is not increased, at this stage scarcely altered, although later in the disease it may be decidedly

lowered. If the further progress of the disease is not arrested, a *voracious appetite* becomes the next symptom, notwithstanding which the patient observes that he slowly *loses in weight* and grows daily *weaker*. Extreme *languor* and *weakness* are characteristic. The rapidity with which these symptoms succeed one another varies. Sometimes the course is very rapid, constituting an acute form; at other times the successive stages are exceedingly slow in developing chronic diabetes.

The early loss of *sexual desire* is common.

Blood-pressure in Diabetes.—The results of different observers as to blood-pressure in diabetes are not uniform. Our own experience would go to show that blood-pressure is certainly not increased in this disease *per se*. Eliminating complications, the tendency is to be normal or below the normal, say 115–130. Where complicated with arterio-sclerosis, a very frequent complication, of diabetes or by nephritis, blood-pressure is higher, reaching sometimes 160 or more. These results are in accord with those of Vaquez, Hensen, Theodore C. Janeway and Arthur R. Elliott,[1] of Chicago, as contrasted with Potain, Jaques Mayer, Tiessier Schott, Ebstein and others.

Alterations in the Blood.—It has already been mentioned that in diabetes the blood becomes highly charged with glucose ("hyperglucemia") which increases from a normal of 0.05 to 0.15 per cent. to 0.2 per cent., and in extreme cases to 0.57 per cent., and from this hyperglycemia comes glycosuria. From the presence of the first we should naturally expect a higher specific gravity of the blood-serum, which has been found as high as 1033, as contrasted with the normal 1028. On the other hand, the serum has been found thinner than normal, containing, according to different analyses, from 80.2 to 84.8 of water instead of the normal 78 to 79 per cent. The red blood-disks are often diminished. The alkalinity of the blood is also lowered, and as such diminution is at a maximum when oxybutyric acid is being excreted, it has been ascribed to the presence of this substance.

An abnormal amount of fat in the blood, producing the technical lipemia, was observed by the earliest students of diabetes, and is attested by many analyses, as well as by the milk appearance of the serum and the intraocular appearances described by Albert G. Heyl. The analyses of Simon shown from 2 to 2.4 per cent. instead of the normal 1 per cent.

Dyspeptic symptoms may appear at various stages, seldom very early. Acid eructations, flatulence, and epigastric pain, or an indescribable sensation described as "sinking" of the epigastrium, are among them. Excessive hunger amounting to boulimia is frequently though not constantly present. *Constipation* is sometimes a very troublesome symptom, and adds, in our experience, to the seriousness of the case; on the other hand, diarrhea is occasionally present.

The foregoing category includes all the symptoms which present themselves in the milder form of the disease. In severe cases all these symptoms become intensified. The patient complains of constant burning thirst, is continually urinating, and as constantly drinking water to quench

[1] "Clinical study of Blood-pressure Variations in Diabetes and their Bearing on the Cardiac Complications," "Jour. of Amer. Med. Assoc.," July 6, 1907.

his thirst, and, while often eating enormously, grows emaciated, although at the onset of the disease he may have been a robust, vigorous man.

As the disease advances there is a peculiar vinous or *acetous* odor of the breath, which has been compared to that of stale beer, and by Sir Thomas Watson to the odor of a place in which apples are kept. This is believed to be due to β-oxybutyric acid in the blood of severe cases of diabetes, and which shows in the urine as diacetic acid and acetone.

Complications.—*Boils* and *carbuncles* in the skin are also of frequent occurrence, favored by the malnutrition growing out of diabetes, and the former are occasionally the first symptoms recognized. The latter never occur early, but, when present, are frequently the immediate cause of death.

Gangrene of various parts of the body is another of this class of symptoms. It is sometimes spontaneous, but more frequently is immediately caused by some trifling injury which, under other circumstances, would be without result. It has been known to start from a blister and from the cutting of a corn. Beginning most frequently in those parts of the body most remote from the center of the circulation, as the toes, its progress and appearances are like those of senile gangrene. Sometimes, however, the gangrene is moist.

Eczema, with itching and burning of the labia and vicinity, is a frequent and troublesome symptom in women incident to the extremely frequent micturition. In the male the meatus urinarius is sometimes the seat of a similar irritation. Eczema elsewhere, as on the palms of the hands, is also a symptom. Tuberculosis occurs frequently. Roberts thinks it occurs in one-half the cases.

Acidosis.—The presence of diacetic acid and acetone in the blood is the result of the β-oxybutyric acid in the blood. It occurs in many of the very severe cases and in every case where a patient is suddenly deprived of his carbohydrates. The latter is not due to diabetes but is purely alimentary—the real index of acidosis is the daily output of nitrogen in the urine. Acidosis may be a danger signal but under proper treatment may last a long while without danger, it is characterized by a heavy vinous odor on the breath and the reaction of the urine to the chloride of iron test. When the toxic symptoms occur the patient passes into *diabetic coma*, first described by Kussmaul in 1874. This is a form of coma which often comes on in advanced stages of diabetes and terminates in death. The condition is one of suddenly or gradually supervening unconsciousness, with or without previous irritability or uneasiness, anxiety, vertigo, or symptoms resembling alcoholic intoxication. Sometimes it is preceded by obstinate constipation or intestinal catarrh or severe colicky and muscular pain. Convulsions do not occur, but the eyes are half open, the pupils dilated, and the eyeballs wandering. In addition to coma there are frequent and feeble pulse, *deep inspiration*, with short expiration, more or less frequent than in health, an actual air hunger and gradually invading cyanosis. The temperature, at first slightly elevated, is subsequently subnormal. The condition lasts for from 24 to 48 hours, when death supervenes.

Severe *neuritis* in the brachial and crural nerves is not infrequent.

In grave cases the *tendon reflexes* are diminished or absent. *Unilateral sweating* has been observed. Senator refers to three cases—two of the left half of the face and one of the right. *Edema* sometimes appears late in the disease, and is not necessarily the result of renal complication.

Among the rarer complications is *cataract*, the association of which with diabetes was long ago noticed by Prout. It develops rapidly and is nearly always symmetrical, involving both eyes simultaneously, but not to the same degree. It is sometimes a very nice point to determine whether cataract is due to diabetes or to the usual causes. The earlier the age at which it occurs, the more probably is it due to diabetes.

Other visual defects may occur. Among these are myopia, amblyopia, presbyopia, and loss of accommodating power from defect of the ciliary muscle. George E. de Schweinitz informs us that a sudden development of myopia between the 40th and 60th years without apparent lesion is characteristic of diabetes. It may be due to fine edema of the choroid, or a choroiditis which in turn determines an elongation of the axis of the eyeball and thus produces myopia.

The ophthalmoscope may reveal dilatation of the *retinal* vessels. Many years ago Albert G. Heyl[1] described a condition which he called *intra-ocular lipemia*, in which the light salmon color of the blood contained in the branches of the retinal vein and artery contrasted with the cinnabar-red of the vein and yellow-red of the artery, also by the greater width of these vessels and the lighter yellow of the fundus. Finally, *atrophy of the retina* and hemorrhagic and inflammatory affections of the eye have been described, and total blindness has been ascribed to the first named.[2] *Derangements* of other *special senses* said to attend diabetes are impairment of hearing, roaring in the ears, and derangement of smell and taste.

A *spongy* state of the *gums*, with recession and excavation, is an occasional complication, resulting in extreme cases in absorption of the alveolar processes and falling out of the teeth, called *pyorrhea alveolaris*.

Changes in the Urine.—The peculiarity of diabetic urine most noticeable to the patient is its enormous quantity, which has been known to exceed 70 pounds (31.78 kilos) in 24 hours, while apocryphal accounts of larger amounts are extant. From 70 to 100 ounces (2100 to 3000 c.c.) are frequent quantities. The quantity of urine passed is limited by the amount of fluid ingested, for while it is possible that the amount of the former secreted may exceed for a very short period the quantity of the latter ingested, it is evident that this cannot continue for any length of time, and, in point of fact, it is found to be almost invariably a little less, the remainder being removed by the lungs, skin, and bowels.

But the most important change is, of course, the presence of *glucose*. Of this, the quantity varies greatly in different cases and at different times in the same case. The sugar should be easily recognizable by the ordinary tests and should be constant. From what may be indicated as "evident traces" the proportion of sugar may reach, it is said, as much as 15 per cent. Reports of larger quantities are made which can scarcely be credited. The 24 hours' quantity varies similarly.

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[1] "Lipemia and Fat Embolism in Diabetes Mellitus," "N. Y. Med. Rec.," vol. xvii., p. 477, 1880.
[2] Dufresne, "De l' Amblyopie Diabétique," "Gaz. Heb.," November, 1861.

Concurrent with the increase in quantity of urine is an *absence of color*, which in extreme degrees is almost total, so that the urine may be as clear as spring water. More frequently it is perfectly clear but of a greenish hue. Almost all diabetic urine, sooner or later after exposure at a moderate temperature, becomes *cloudy* from the development of fungi coincident with fermentation. The *odor* of the urine is usually normal when first passed, but sooner or later, in consequence of fermentation setting up, it may acquire an acetous odor. The latter change also increases the normal acid reaction and maintains it much longer after exposure to the air than is the case with normal urine. This acetous odor is ascribed to acetone and diacetic acid. The urine may have a sweetish odor when passed, which has been compared to "sweet brier." Diabetic urine is sometimes quite free from *sediment*. At other times there is a copious sediment of uric acid. In the sediment may also be included the *penicilium* fungus, common to acid urine, as well as the more characteristic yeast or sugar fungus, or the *torula cerivisiæ*. This also sometimes appears as a *mold* on the surface of the urine.

Of *abnormal* constituents, albumin is often present—perhaps in one-third of all cases; some make it a larger proportion, some less. The albuminuria is not generally large and, in our experience, is not often a serious symptom.

Finally, *acetone, diacetic acid, and beta-oxybutyric acid* are all frequently met in diabetic urine. Source of these substances may be either protein or fat, the fatty acids probably constituting the chief source, diacetic or aceto-acetic acid being probably first formed and rapidly transformed into acetone. When but little diacetic acid is produced, it is all converted into acetone; when much is formed, both substances appear in the urine. The conversion takes place mainly in the urine, but doubtless also in the tissues or the blood, since acetone may be present in the expired air. To acetone is ascribed the vinous odor sometimes present in the urine. Acetone is produced in health in a slight amount in the normal decomposition of albumin, freely in certain diseases other than diabetes. According to von Noorden, these substances are formed in the disintegration of the albumin of the body and not of the food—in a word, when the patient is "consuming his own proteids."

Duration.—Though the course of a few cases of diabetes is so rapid as to justify the name acute, the number of these cases is not sufficient to justify a classification into acute and chronic. In such rapid cases death has taken place at periods ranging from two days to six weeks, yet in no instance can it be averred that the disease was of as short duration as it seemed, since it may have existed some time before it was discovered, while in several it was evidently of longer duration. It is true, therefore, that diabetes mellitus is a disease almost invariably of long duration. Cases of 15, 18, and 20 years' duration are reported. Tyson has had a number of cases under his care for more than ten years. The younger the subject, the shorter the duration and the more promptly fatal the result, while after middle age, by treatment, the duration may be indefinitely prolonged.

Diabetes mellitus is sometimes distinctly intermittent for a time, re-

gardless of treatment. Experience has taught us that a form of diabetes occurs, in which both polyuria and glycosuria may disappear without treatment, to recur again. Such cases are, however, easily controlled by treatment when discovered, while they are as certain to pass over into the permanent form if neglected.

Diagnosis.—The diagnosis of daibetes mellitus is very easy, yet it is often long overlooked by the practitioner. Unnatural thirst and copious diuresis should always suggest a chemical examination of the urine, and although there are sources of error in testing for small quantities of sugar, the quantities thus overlooked are not usually of clinical significance. In fact, glucose is more frequently declared present by inexpert examiners when absent than the reverse. Almost any one of the tests, therefore, which are found in the various manuals for the examination of urine, applied with ordinary care, will respond readily to quantities which are of clinical significiance. Lactose in the urine of women due either to the appearance of milk in the breast after birth of a child or its disappearance at the end of lactation will reduce Fehling's solution just as it is reduced by glucose. However it will not be fermented by yeast and can thus be distinguished.

In using Fehling's solution[1] a given quantity, one c.c., of Fehling's solution is placed in a test-tube, diluted with about four times its bulk of water, and boiled for a few seconds. If the solution remains clear, add immediately the suspected urine, drop by drop. If sugar is abundant, the first few drops will usually cause the red or yellow precipitate, but if the reaction does not occur, the dropping may be continued, followed each time by heating until an equal volume has been added. If no red or yellow precipitate occurs, sugar is absent. Now, Fehling's solution is so composed that if an equal volume is exactly reduced by an equal volume of urine, that urine contains one-half of one per cent. of glucose; if by half bulk, one per cent.; if twice the bulk, one-fourth per cent., and so on, whence one can easily estimate roughly the percentage. Should the urine contain more than one per cent. of sugar, it should be diluted one to ten and the result multiplied by ten.

If a reduction takes place on boiling the test fluid alone, a new supply may be obtained, or a little more soda or potash may be added, the fluid filtered, and it is again ready for use. Such spontaneous reduction of the cuprous oxid often occurs when Fehling's solution is kept for some time.

In judging the progress of a case of diabetes under treatment it is not sufficient to test the urine qualitatively, but a quantitative determination of sugar must be made. This may be done by the clinical method just described or by volumetric processes described in the manuals for the examination of urine, but the simplest process is the (3) *fermentation method* of Roberts. In this the specific gravity of the urine is taken be-

[1] *Fehling's Solution.*—Dissolve 34.652 gm. of pure crystallized sulphate of copper in 200 gm. of distilled water; 175 gm. of chemically pure crystallized neutral sodic tartrate in 480 gm. solution of caustic soda of specific gravity 1.14, and into this basic solution the copper solution is poured, a little at a time The clear mixed fluid is diluted to one liter, or 1000 c.c.

Ten c.c. of this solution will be reduced by 0.05 gm., or 50 milligrams, of diabetic sugar. If Fehling's solution is to be kept some time, it is absolutely essential that it should be placed in smaller bottles holding from 40 to 80 gm., sealed, and kept in a cellar.

Still greater security may be obtained by dissolving the cupric sulphate in 500 c.c. and the tartrate. salt and potash in 500 c.c., keeping the two solutions separate in rubber-stoppered bottles. Equal volumes of the two solutions are united when needed for use.

fore and after fermentation, and the difference in the two results indicates the number of grains of sugar in each fluidounce of urine. Suppose, then, the specific gravity before fermentation to be 1045, and after fermentation 1035: the quantity of sugar is ten grains to the fluidounce, or 0.65 gm. in 30 c.c. These figures can be reduced to percentage by multiplying by 0.23.

Tests for Acetone, Diacetic Acid, and Oxybutyric Acid.—In view of the important rôle in prognosis assigned to acidosis the regular examination of the urine for diacetic acid becomes almost as important as that for sugar. Of the numerous tests for acetone, most of which require the distillate for their successful application, Legal's nitroprussid of sodium test is the most satisfactory for the practitioner, because it does not require the distillate.

Legal's Test for Acetone.—A fresh, rather strong solution of sodium nitroprussid is made by dissolving a few fragments in a little water in a test-tube. To three or four c.c. of the suspected urine add enough liquor sodæ or potassæ to secure a distinct alkaline reaction. To the mixture then add a *few drops* of the nitroprussid solution, when the whole quickly assumes a red color, whether acetone is present or not, said to be produced by creatinin even more rapidly than by acetone. In any event the red color disappears; but if acetone is present, the addition, of a few drops of concentrated acetic acid causes a purple or violet-red color. If there is no acetone, this final change does not occur, while the purple color also fades in a little while, even if caused by acetone.

To test for *diacetic acid* add a few drops of a 15 per cent. solution of neutral or only slightly acid ferric chlorid to a small quantity of the urine, when a beautiful Burgundy-red reaction occurs. A precipitate of phosphates succeeds the adding of the first few drops, but this is redissolved by a further addition of the chlorid. The test is confirmed if, after heating the original fluid, there is no response on application of the chlorid of iron—the effect of heat being to dissipate the diacetic acid. A more brilliant reaction is obtained if the urine be first treated with a solution of acetate of lead, filtering out the white precipitate and testing the filtrate. Urine passed after the administration of salicylic acid, antipyrin, carbolic acid, salol, phenocol, kairin and other drugs furnishes a similar reaction.

Prognosis.—The prognosis of diabetes varies with the age at which the disease makes its appearance, the time which has been allowed to elaspe before treatment is instituted, and the treatment itself. Once thoroughly established early in life, or before 25 years of age, recovery is rarely possible, but even at this age, if treatment is instituted sufficiently early, much may often be done to avert the end. Diabetes is a disease in which the expectant plan of treatment is disastrous. It is a disease which never gets well of itself, and usually gets worse if not properly treated. At the same time the mild cases amenable to treatment are in a decided majority. When the disease appears after middle life in fat persons or those disposed to gout, and is early recognized and promptly treated, it is usually easily controlled; it is never safe to declare a case of diabetes absolutely cured. As a rule, however, even those who have apparently recovered must keep a watch upon their diet, and should at intervals have their urine examined with a view to sounding, as it were, their condition. We are entirely justified in saying to a diabetic patient, "As long as your urine remains free of

sugar you are practically as well as if you had no tendency to diabetes." On the other hand, for spare, nervous, and hard-worked persons, especially mentally overworked, under 40, there is a much more unfavorable outlook. Even here, if the co-operation of the patient can be secured, much may be done. Every intermediate degree of seriousness may occur. The cause of death is very frequently some intercurrent or consequent disease, as phthisis or diabetic coma. "If there is such a thing as complete recovery from the disease diabetes mellitus it must be by a resumption of normal carbohydrate metabolism. Now such return is favored by a rest of the glycogenic function by taking away its office. Hence the importance of completely, or as nearly as possible removing the sugar from the urine and in the earliest stages cutting out carbohydrate food. If the crabohydrate metabolism is eliminated by the removal of foods demanding this function, the rest thus secured permits a resumption of function more or less completely. Now in the early stages of the disease this is both possible and safe. Hence the treatment must be sufficiently vigorous from the outset to keep the sugar out of the blood as well as the urine. And when the blood is sugar free the greatest pains should be taken to keep it so."—von Noorden.

Treatment.—This resolves itself easily into the dietetic, the hygienic, and the medicinal.

1. *Dietetic Treatment.*—This is by far the most efficient, and no permanent results have ever been obtained without it. It consists essentially in the elimination from the diet of such articles as are readily convertible into glucose—viz., the carbohydrates. It is acknowledged that in the early stages of the disease only the saccharine and amylaceous foods fail to be consumed in the economy in the usual way and appear in the urine as glucose. Hence, if these be excluded from the diet and their place supplied by other assimilable articles, the symptom disappears, and the disappearance of this symptom seems to be, for the time being at least, the cure of the disease. In excluding the carbohydrates it must be carefully arranged that the patient receives enough and not too much food. The value of calories in an ordinary serving will be found in the list of foods allowable in diabetes.

The correct treatment of diabetes mellitus demands the full appreciation of three undoubted facts:

First, each case of diabetes is a problem in itself.

Second, the presence of sugar in the urine proves that the individual is taking into his stomach food which cannot be converted into energy, or the already energized food is being excreted in the form of sugar. These two facts mean that the person is on starvation diet.

Third, the presence of glucose in the urine means that at any time, perhaps at the time of examination, substances may be found which are poisonous to the economy. These circulating in the blood may cause death.

These postulates being accepted as facts, treatment becomes a question of dietetics and hygiene. Drug exhibition taking an entirely secondary place though a helpful one.

In treating diabetes mellitus we must realize that cases fall naturally into three classes, first, those individuals past middle life who without

treatment have constantly a fairly large amount of sugar and no acetone bodies in the urine, who are well nourished and apparently in perfect health; second, those patients who are on the border line, who from indiscretion or advancement of the condition can take but little carbohydrates and often have acetone bodies in the urine; third, those usually in the earlier years of life who suffer in many ways, who are distinctly ill, and who sooner or later succumb.

Treatment of the First Class.—It must be remembered that notwithstanding such individuals are apparently in perfect health, that they suffer frequently from neuritis, vertigo, furunculosis, optic neuritis; that they lend themselves as easy prey to any infection; that they are subject at any time, from any indiscretion, to a sudden acid intoxication which may end their lives, or that they may fall a victim very easily to suppuration or gangrene.

Therefore, such individuals should be under careful supervision.

General Treatment of Slight Cases.—It is very generally taught at the present time, that rather rapid and prolonged withdrawal of carbohydrate food in such individuals is likely to precipitate a fatal acidosis. Such has not been our personal experience. Surely diacetic acid and acetone appear in the urine under such circumstances, but it rarely does harm. Von Noorden is of this same opinion when he says (New Aspects of Diabetes, 1912): "The sugar in the urine diminishes and reaches zero after a few days of strict diet. But about two or three days after we have excluded the carbohydrates, we are horrified at finding a strongly positive reaction with chloride of iron; a quantitative estimation gives about 1 gram of acetone and 2 to 5 grams of oxybutyric acid, or perhaps considerably more. Unless the rationale of the formation of acetone is well understood, the "strict diet" will usually at once be abandoned. The doctor fearing the development of coma, will order carbohydrate food perhaps in lavish quantities and with precipitant haste. But he is wrong. By this he throws away all his chances of practically influencing the morbid processes peculiar to diabetes. This acetonuria is entirely physiological; healthy individuals would have behaved in exactly the same manner, if put upon the same diet. It is an alimentary and not a diabetic acetonuria. If, undeterred, we continue upon the strict diet, the acetonuria will again disappear in about eight to fourteen days, and the normal formation of acetone will again be established. In the many thousand cases which I have treated in the course of years, I have never seen one mishap due to the continuance of the strict diet under these circumstances."

Therefore, such cases should within a week begin an entirely carbohydrate free diet, great care being taken that a sufficient amount of food be given to keep the patient's general health in equilibrium. For this purpose the following list taken from Janeway may be used or one constructed from the list on page 813 of this book.

STANDARD STRICT DIET.

BREAKFAST.

2 Eggs.
Ham, 90 grams (3 ounces).
Coffee, with 45 grams (1 1/2 ounces) cream.
Butter, 15 grams (1/2 ounce) on the biscuit during the test period; cooked with the eggs if no biscuit or bread is taken.

LUNCHEON.

Meat, (steak or chop) 120 grams (1/4 pound).
Green vegetables (asparagus, beet greens, Brussels sprouts, cabbage, cauliflower, celery, chicory, cresses, cucumbers, egg-plant, endive, kohl-rabi, leeks, lettuce, okra, pumpkin, radishes, rhubarb, salsify, sauerkraut, spinach, string beans, tomatoes, vegetable marrow.
White wine, 2 claret glasses (6 ounces) or
Whiskey, or brandy, 2 tablespoonfuls (1 ounce).
Butter, 15 grams 1/3 ounce, with the green vegetables if no biscuit or bread is taken.
AFTERNOON TEA with 15 grams (1/2 ounce) of cream.

DINNER.

Any clear soup.
Fish, 90 grams (3 ounces).
Meat (beef, mutton, turkey or chicken) 120 grams (1/4 pound).
Green vegetables from this list, 2 tablespoonfuls.
Salad with 15 grams (1/2 ounce) of oil in the dressing.
Cream cheese, 30 grams (1 ounce).
White wine, 2 claret glasses (6 ounces) or
Whiskey or brandy, 2 tablespoonfuls (1 ounce).
Demitasse of coffee.
Butter 30 grams (1 ounce) on the fish, meat and green vegetables, if no bread or biscuit is taken.

BEDTIME.

Bouillion, with 1 raw egg.
Nitrogen—18 grams. Total calories 2550.
Omitting ham, nitrogen—15 grams. Total calories 2200.

In from two weeks to one month after the urine is sugar free, the patient may be given carbohydrates tentatively, beginning with 30 grams of white bread. This may be very slowly increased, daily examination of the urine being made to be assured that no sign of sugar appears. When sugar appears, then drop at once either to a strict diet which usually is necessary, or to one of the diets with which the patient did not show sugar in the urine.

Mild cases need this constant care, because they frequently can thus be kept in excellent health and never die of coma. Such cases under such care rarely need drugs. They, however, should avoid overwork, overexcitement, and must keep to their diet. These patients may be given salicylates daily, 10 grains of sodium salicylate, or 10 grains of acid acetyl salicylic. In this way the line of tolerance may be even increased. After these cases are well established in their routine, they may be given the table found on page 813 of this book for their guidance, but the urine should be examined every two or three months to make certain the case is progressing properly. In addition to these measures, it is highly important that the bowel movements be kept free. To this end phenolphthalein in doses of 5 grains, three times a day may be used, or Rochelle salts or Epsom salts. If desirable one of the purgative waters may be used, but they are in no way superior to the ordinary salines.

Treatment of Border-line Cases. (Moderately severe cases).—It must be remembered that any case of mild character, may at any time become a moderately severe case or a very severe one, either by the advance

of the lesion in the pancreas or by neglect. Therefore, the great necessity of keeping such a case without even a fraction of one per cent. of sugar. Attention to hygiene and to diatetics will usually do this in these mild cases, because of the very slow advance of the disease of the Islands of Langerhans.

A moderately severe case is one described as being unable to take 60 grams of white bread without the appearance of sugar in the urine, and which usually has acetone bodies in the urine. Until such a case is well started upon a diet which will keep them in the line of safety, it is much better to have the patient either in a hospital or sanatorium, or under a trained nurse. Daily examinations of the urine are a necessity. Careful diet is imperative. This is usually impossible in a private case left to his or her own resources.

Here the carbohydrates must be very slowly reduced. It is in these cases where a severe and dangerous acidosis may occur if these ingredients are rapidly reduced. At least two weeks should be occupied and the symptoms of acidosis carefully watched for. Von Noorden says that, "in accordance with our scheme of treatment we slowly reduce the input of carbohydrates to zero; the glucosuria diminishes, but does not disappear. At the same time, the amount of acetone bodies in the urine rises considerably, the reaction with chloride of iron becomes strongly positive, and we find 1 to 2 grams of acetone in twenty-four hours' urine. Nevertheless, we persevere with the restricted diet, and the amount of acetone continues to rise, or at least remains very high. No doubt by this we are exposing the patient to a certain amount of risk, but this can be reduced to some extent by administering about 10 to 15 grams of bicarbonate of soda. We also keep a sharp lookout on the patient, and as soon as we discover any symptoms pointing to impending diabetic auto-intoxication—such as headache, excitement, hyperesthesia of the stomach, etc.— we shall add carbohydrates to the diet. We have been advancing too rapidly, and must wait a little while, and then begin again to try and eliminate the carbohydrates." A diet which does well in these individuals is one poor in protein. This is also taken from Janeway.

TABLE III.—STANDARD DIET WITH RESTRICTED PROTEIN.

BREAKFAST:
2 eggs.
Bacon, 15 grams (1/2 ounces).
Coffee with 45 grams (1 1/2 ounces) of cream.
Butter 20 grams (2/3 ounces).

LUNCHEON:
1 egg.
Bacon, 15 grams (1/2 ounce).
Meat (lamb chops, ham or beef steak) 60 grams (2 ounces).
Salad with 15 grams (1/2 ounce) of oil in the dressing.
White wine, 2 claret glasses (6 ounces) or
Whisky or brandy, 2 tablespoonfuls (1 ounce).
Butter 40 grams (1 1/3 ounces).

AFTERNOON TEA with 15 grams (1 1/2 ounces) of cream.

DINNER:
Any clear soup.
Meat (roast pork, beef, mutton, turkey, or lamb chops), 90 grams (3 ounces).
Vegetables and salads—asparagus, beet greens, brussels sprouts, cabbage, cauliflower, celery, chicory, cresses, cucumbers, egg-plant, endive, kohl-rabi, leeks, lettuce,

orka, pumpkin, radishes, rhubarb, salsify, sauerkraut, spinach, string beans, tomatoes, vegetable marrow.
Salad with 15 grams (1/2 ounce) of oil in the dressing.
Cream cheese, 30 grams (1 ounce).
White wine, 2 claret glasses (6 ounces) or
Whiskey or brandy, 2 tablespoonfuls (1 ounce).
Demi-tasse or coffee.

BEDTIME:
Bouillion with 1 raw egg.
Nitrogen, 10 grams; total calories, 2500.
Omitting 30 grams of butter and 1/2 ounce of bacon, 2250 calories.

This diet list may be persisted in until the patient's urine is both free of sugar and acetone bodies. When this occurs, then the more free diet may be used, the one which is printed on page 813. If in spite of this strict diet, sugar and acetone both appear, then it is wise to put the patient on practically starvation diet, or what Janeway calls Green Days. This diet of Janeway's follows. It should be used for two days.

TABLE IV.—GREEN DAYS.

BREAKFAST:
One egg, boiled or poached.
One cup of black coffee.

DINNER:
Spinach, with a hard-boiled egg.
Bacon, 15 grams (1/2 ounce).
Salad with 15 grams (1/2 ounce) of oil.
White wine, 1/4 liter (4 ounces) or whisky or brandy 30 c.c. (1 ounce).

4.30 P. M.:
Cup of beef tea or chicken broth.

SUPPER:
1 egg scrambled, with tomato and a little butter.
Bacon, 15 grams (1/2 ounce).
Cabbage, cauliflower, sauerkraut, string beans, or asparagus.
White wine, 1/4 liter (4 ounces) or whisky or brandy, 30 c.c. (1 ounce).
Sodium carbonate, 15 to 30 grams (1/2 ounce to 1 ounce) in 24 hours.
Nitrogen, 5 grams, carbohydrates, about 5 grams—calories, 575.

The above limited diet should then be followed for two days by Von Noorden's oatmeal diet, and then again the patient is to be put upon the restricted diet, as is shown above in Table III.

TABLE V.—OATMEAL DAYS.

Porridge made from oatmeal, 250 grams (1/2 pound) with butter, 250 grams (1/2 pound) salt and pepper to taste.
Black coffee, light white wine, 1/2 liter, (8 ounces) or cognac, 60 c.c. (2 ounces).
The whites of six eggs may be added to the porridge if desired.

	N.		Calories.
Oatmeal	6.2	170.	1025
Butter	0.4		1975
	6.6		3000
Alcohol (40 grams)			210
6 eggs whites	3.6		90
	10.2		3300

In regard to this oatmeal diet, Von Noorden says he was severely criticised for suggesting its use at first. Now he is forced to criticise physicians because they use it too freely. It should not be used beyond two or three consecutive days. After careful watching and changes of diet, after weeks or months perhaps the patient will be both sugar free and acetone free, and then perhaps we can add a little carbohydrates to the diet

without the appearance of sugar in the urine. That is, if the patient does not have any hyperglycemia and hence no sugar in the urine.

Treatment, Severe Cases.—The severe cases of diabetes usually occur in the first three decades of life, but as before stated may occur at any age, They are those which cannot be made sugar free by the withdrawal of carbohydrates from the food, in other words, they manufacture sugar in the liver not only from carbohydrates but from proteins as well. Von Noorden is of the opinion that the albumen of meat, of casein and of the cereals are harmful in those conditions in the order named. It is important in these cases, to as much as possible limit the proteins as well as the carbohydrates. Here the list recommended with limited proteins may be used. Table III.

By severe cases one understands cases in which by careful nursing, honest obedience on the part of the patient, and by strict diet, glucose still appears in considerable amounts in the urine, usually with acetone and diacetic acid. Here the patient is best treated by a more liberal diet; 60 to 70 grams of the various starch foods are given daily. By interposing days of starvation (Green Days), days of strict diet, and oatmeal, the case can get along often well, again getting back to the diet containing starch. All the time these patients should be taking soda in large amounts.

Treatment of Acidosis.—In cases of slight glycosuria, as stated before, the acetonuria which occurs when carbohydrates are suddenly and completely withdrawn may be disregarded, or they may be given 15 to 20 grams of sodium bicarbonate daily.

In the more severe forms a careful watch must be kept and alkalies must be given constantly. The rôle that alkalies play is not that they prevent the formation of acid, they combine with them and cause them to be more readily excreted, and prevent the extracting of the alkali from the blood and tissues.

In the stage immediately preceding coma, where acetone bodies are present in the urine in large quantities, where there is the peculiar air hunger and tendency to somnolence, alkalies should be freely used, both by the mouth and by intravenous use of sterile solutions.

Diet as stated before, strict diet, oatmeal days, green days, fat days. Often one has to resort to carbohydrates. The bowels must be kept open.

Feeding is important. Levulose, 100 grams in lemonade, milk 250 c.c. every two hours, oatmeal diet.

Von Noorden suggests no food. Whisky given, 100–150 c.c. daily, will do them; then after three or four days, milk and oatmeal, soups.

Coma ends in death. Even Von Noorden with all his experience so states. This makes the treatment very unsatisfactory. Nevertheless, one should persist in giving such patients who are in deep coma, intravenous injections of four per cent. sterile solution of bicarbonate of soda.

Drug Treatment.—Salicylic acid perhaps is the best drug, but the opiates in the form of opium or codein are most valuable.

Health Resorts.—Residence at the various health resorts, is sometimes valuable for several reasons. In the first place, the patients learn that strict diet, attention to their general health, and obedience to orders will make them feel well, and perhaps will teach them to follow the instructions

learned at these institutions, when they get back to their ordinary conditions of life; but if a patient goes to a health resort with the idea that a few weeks or months' residence there will cure him, he goes with entirely a false idea. He must continue with the instructions he received there after he goes home.

The cases must, when able to be let alone, be given diet tables.

The following classified summary of articles of food admissible for diabetics will be found convenient for reference, with caloric value of a serving of each.

Shell-fish.—Oysters 84 cal., mussels and clams 50 cal., raw or cooked in any way, *without* the addition of flour.

Fish of all kinds, fresh or salted, including lobsters 84 cal., crabs 84 cal., sardines 269 cal., and other fish in oil; fish roe 128 cal., caviare. Fish average 150 cal. to a serving.

Meats of every variety except livers, including beef 144 to 238 cal., mutton 362 cal., chipped dried beef 150 cal., tripe, ham 227 cal., tongue, bacon 100 cal., and sausages. Also poultry 108 cal., and game of all kinds, with which, however, sweetened jellies and sauces should *not* be used.

Soups.—Clear bouillon and other soups, beef-tea and broth made without flour, rice, vermicelli, or other starchy substances; and without the vegetables named below as inadmissible. Soups are of very little caloric value for 16 to 20 cal. for each serving.

Vegetables.—Cabbage 35 cal., cauliflower, Brussels sprouts, broccoli, green string-beans, the green ends of asparagus, spinach, dandelion, mushrooms, tomatoes, lettuce, endive, coleslaw, olives, cucumber (fresh or pickled), radishes, sorrel, young onions, watercresses, mustard and cress, turnip tops, celery tops, artichokes, gherkins, okra, parsley, or any other green vegetables. Each serving amounts to from 8 to 30 cal.

Bread and cakes made of *pure* gluten, bran, aleuronat, roborat, soya, peanut- or almond-flour, inulin, with or without eggs and butter. Griddle-cakes, pancakes, biscuit, porridges, etc., made of these flours. Oatmeal porridge with cream. Where especial stringency is required, the last should be altogether omitted. The great difficulty in obtaining pure gluten flour is so great and practically all the gluten breads on the market contain much starch that one had much better use white bread in proportion as instructed above. The amount of white bread can be converted into proper amounts of potato and other starches.

Eggs in any quantity and prepared in all possible ways, without sugar or ordinary flours.

Butter and Cheese.

Nuts.—All except chestnuts, including almonds, walnuts, Brazil nuts, hazelnuts, filberts, pecan nuts, butternuts, cocoanuts.

Condiments.—Salt, vinegar, and pepper in moderate quantities.

Fruits.—Cranberries, plums, cherries, gooseberries, red currants, strawberries, acid apples, lemons, oranges sparingly—all without sugar. Acid fruits may be stewed, with the addition of bicarbonate of sodium instead of sugar.

Jellies.—None except those not sweetened with sugar. Saccharin may be used for sweetening instead of sugar. Jellies may be made of calf's foot or gelatin and flavored with wine.

Drinks.—Coffee, tea, and cocoa-nibs, with milk or cream, but without sugar. Also, milk, cream, soda- (carbonated) water, and all mineral waters freely; lemonade without sugar, acid wines, including clarets, Bordeaux, Rhine, and still Moselle wines, and very dry sherry. Unsweetened brandy, whisky, and gin. No malt liquors except those ales and beers which have been long bottled and in which the sugar has all been converted into carbonic acid and alcohol. Saccharin may be used for sweetening tea and coffee.

To be Especially Avoided.—Cantaloupes, watermelons, peaches, grapes, and all other sweet melons and fruits; potatoes (white and sweet), rice, beets, carrots, turnips, parsnips, peas, and beans; all vegetables containing starch or sugar in any quantity; sweet wines, including sherry, Madeira, port, and champagne.

2. *Hygienic Treatment.*—Next in importance to the dietetic is the hygienic treatment of diabetes. This consists in bathing, and attention to the skin, together with outdoor muscular excercise and perfect ventilation within doors.

The diabetic should breathe the freshest and purest air. While the cases are not numerous in which embarrassed respiration results in glucosuria, there are undoubted instances in which this has occurred, as in croup and whooping-cough; and it is well known that asphyxiated lower animals are likely to have glycosuria. Although the glycosuria thus resulting is probably reflex, it can hardly be expected that the diabetic should improve under unfavorable respiratory conditions. He should not, therefore, live, work, or sleep in a confined atmosphere, but secure the most perfect ventilation, spending much of his time out of doors, and sleeping in large, well-ventilated chambers, with windows open, etc. Especially should he avoid inhalation of irrespirable gases. Attention to the skin, is most important to the diabetic. He should bathe at least twice a week in tepid or hot water on going to bed in winter, and on rising take a cool sponge-bath daily. In summer he may take a cool bath on rising and on retiring. He should groom his skin thoroughly daily, either after the bath or independent of it on the days on which he does not bathe. Two tablespoonfuls of sodium carbonate to an ordinary bath is a suitable addition to the latter, softening the skin and facilitating its action by removing the effete epithelium.

Muscular exercise should be taken daily by the diabetic, both by walking and gymnastics. Glycogen is undoubtedly consumed in the muscles during their action, and it is quite certain that in diabetes there is an undue accumulation of sugar in the muscles. Exercise should be sustained regularly day by day, even in wet weather, care being taken to keep the feet dry, while it should never be carried to the point of fatigue.

Attention to other secretions, particularly to that of the bowels, is of the greatest importance. Diabetics who are constipated are always more difficult to relieve. It is probably partly on account of their action in this respect that the alkaline and alkaline-saline aperient waters, as those of Vichy, Vals, and Carlsbad, are so useful. To those who visit these springs, a part of the benefit is ascribable to the other favorable hygienic influences, such as rest, fresh air, and exercise, by which they are surrounded. Inde-

pendently of these influences, however, there is reason to believe that the alkaline waters are of service to diabetics, and when their cost is not a consideration, a quart of Vichy or Vals and half as much Carlsbad may be taken during the day, beginning before breakfast. The Vichy is a more alkaline water, containing 35 grains (2.3 gm.) of carbonates to a pint (0.5 liter), while Carlsbad contains but 11 grains (0.51 gm.), but twice the proportion of chlorids, eight grains (0.7 gm.) to a pint (0.5 liter), and nearly ten times as much sodium sulphate, or 19 grains (1.25 gm.); hence its more purgative quality to which some of the good effect may be due.

The waters of the celebrated Saratoga Springs in this country have an undoubted action on the liver, probably through the chlorids they contain, which are in very large proportion, reaching in the Geyser Spring 70 grains (4.6 gm.) to the pint (0.5 liter), and in the Empire and Hathorn, 63 grains (4.19 gm.) to the pint (0.5 liter). They contain no sulphates, but the carbonates are present in considerable proportion, though much less than in the Vichy waters.

3. *The Medicinal Treatment.*—Like all diseases in which treatment by drugs is relatively inefficient, diabetes has its full share of reputed remedies, most of which are useless. This dare not, however, be said of all.

The only drug that can be relied upon to produce an effect in diminishing glucosuria is opium. It seems that it was used for diabetes as early as the second century by Archigenes. It was also used by Ætius the physician, in the fourth century, and in the latter part of the 18th century and beginning of the 19th by Rollo, Frank, Tommasson, and especially the English physician, Pelham Warren, in 1812. It is certainly a useful agent in diabetes, but its use is united with disadvantages in the locking-up of the secretions. On aecount of its comparative freedom from these effects, codein has come to be the favorite alkaloid of opium in diabetes. It may be given in 1/4 grain (0.016 gm.) doses three times a day, or 1/2 grain (0.032 gm.) twice a day, increasing 1/4 grain (0.016 gm.) daily until the desired effect is produced or it proves useless. If the sugar disappears, the drugs should be gradually withdrawn. If constipation is caused by it, aperient remedies should be associated, and very suitable are the natural aperient waters, including the bitter waters, Friedrichshalle, Hunyadi János, Racokzy Püllna, etc., and in this country Pluto water from the French Lick Springs in Indiana and the water from the Vichy and Geyser Springs at Saratoga, N. Y. One of us has seen a patient entirely relieved under its use, and it alone, with no return of the sugar after its omission. Rarely as much as ten grains (0.65 gm.) a day are given, and its use should be deferred until other measures are found insufficient.

After opium, arsenic has longest maintained its reputation as a remedy in diabetes, and it should be used in all mild cases. Fowler's solution is the best preparation. Possibly it acts partly on the gastro-intestinal tract and partly on the red blood-disks, increasing their oxidizing power over glucose. The best plan is to give small doses long continued rather than to attempt to bring about its physiological action. Hence three drops twice or thrice a day, continued indefinitely, is now a favorite dose. Salicylates are quite strongly recommended by v. Noorden.

The bromid of potassium is sometimes efficient in diabetes accompanying

functional nervous disorders due to mental overwork or psychic disturbance. Bromin and arsenic are combined in the shape of Clemens' solution of bromid of arsenic, of which the dose is from 3 to 5 minims (0.184 to 0.3 c.c.). Veronal may be of use in like conditions.

Substances which possess the power of oxidizing sugar in the blood have long been sought. The alkalies, and especially the alkaline carbonates, acquired considerable reputation in the treatment of diabetes, after Mialhe claimed for them the power of destroying the sugar in the blood, and of neutralizing the volatile acids retained within the organism in consequence of the defective action of the skin. They prevent abstraction of alkalies from the tissues and continue to be used with results which justify the practice, and almost invariable practice is to place diabetic patients on sodium bicarbonate in doses of a dram (4 gm.) or more three times daily if there is acidosis. It neutralizes the acidosis which we have ascertained to be a dangerous condition. The efficiency of the alkaline mineral waters is thus explained.

Salicylate of sodium has warm advocates, and in gouty cases it may be useful. According to von Noorden, it is especially in neurogenous diabetes that the sodium salicylate is useful, quieting the irritability of the central nervous system.

Iodid of potassium has produced some striking results in the case of diabetes due to syphilitic lesions of the brain.

While we realize that drugs are not of much use in diabetes mellitus, it is a mistake to throw them aside. The patient often looks for them and when they are applied rationally and in safe and harmless doses they ought not be ruthlessly excluded from our treatment.

Treatment of Complications.

Eczema and Pruritus.—These sometimes intensely annoying symptoms commonly abate with the reduction of the glycosuria, but require also other measures. In the first place scrupulous cleanliness is necessary, accomplished by warm, tepid bathing. In addition, we may use solutions of boric acid 2 drams (8 gm.) to the quart (1 liter) or sodium hyposulphite, 1 ounce (30 gm.) to a quart (1 liter) of water; also zinc ointment, ointment of acetate of lead; solutions of corrosive sublimate, very weak—1 to 3000—and tumenol-sulphonic acid in 10 per cent. alcohol solutions. Carbolic acid 5 to 10 minims (0.3 to 0.6 gm.) glycerine ℥ij (8 c.c.) and water an ounce (30 c.c.) make a soothing preparation. As a last resort in pruritus nitrate of silver may be used in the strength of 20 grains (1.3 gm.) to the ounce (30 c.c.), making daily applications, which though sometimes painful, are ultimately effectual.

DIABETES INSIPIDUS.

Definition.—Any excessive secretion of nonsaccharine and nonalbuminous urine which has continued for a long time. Willis in 1674 separated it from diabetes mellitus.

Etiology.—The condition, unlike diabetes mellitus, affects more frequently younger persons, being rare in those over 50 years of age, relatively frequent in infancy, and most common between the ages of twenty and thirty. It may be congenital and not infrequently is hereditary.

As to sex, it is much more frequent in males than in females, affecting two or three times as many of the former as of the latter.

As to causes, Futcher reported nine cases of diabetes insipidus; in four of them was a history of cerebral syphilis, with improvement under iodid of potassium, the same uncertainty prevails as with diabetes mellitus. An examination of cases shows an association with a certain number of conditions, such as cerebral disease, including tumor of the brain, meningitis, paralysis of the sixth nerve, sunstroke, cerebrospinal fever, falls and blows on the head; exposure to cold and the drinking of cold fluids, drunkenness, pregnancy, hysteria, emotion, especially fright, hereditary influence, syphilis, and previous disease, etc., but this does not show causation. The proportion, however, of cases in which the condition is associated with brain disease and injuries to the head, taken in connection with the fact of Bernard's discovery that puncture of the floor of the fourth ventricle above the diabetic center produces polyuria without glycosuria, makes it very likely that central nervous irritation, however induced, is at the bottom of the symptom. It is reasonable to suppose, too, that diabetes insipidus may be the result of some irritation, direct or reflex, of this center in the medulla oblongata, or of the sympathetic ganglia in the abdomen. The latter explanation also applies to cases of polyuria attending the presence of abdominal diseases, such as tumor, aneurysm, or peritonitis, though it is doubtful whether these should be regarded as cases of true diabetes insipidus.

Cases associated with evident organic lesion as of the brain or syphilis are known as secondary or symptomatic while cases not thus associated are known as primary. From Futcher's reports there would seem to be a nearly equal proportion of each. Certain cases of tumor of the hypophysis are characterized by polyuria.

Morbid Anatomy.—The essential morbid anatomy of diabetes insipidus would be the lesions of the nerve centers or sympathetic ganglia which may underlie the symptoms. But as these are often undiscoverable, or at least indefinite, it is impossible to describe them. Notably is this the case with lesions of the third and sixth nerves. Associated central nervous lesions, when present, are found more frequently in the vicinity of the base of the brain.

Symptoms.—The *enormous secretion of urine* of almost spring-water-like clearness, and of specific gravity often as low as 1003, is the most conspicuous symptom, but more annoying, probably, is the *extreme thirst* which always attends it. These may be said to be the essential symptoms, others which may or may not be present being rather their consequence. Very constant among the latter are *dryness of the skin* and *absence of perspiration*. The health may be otherwise perfect, though *emaciation* and *weakness* are often present. The debility is sometimes extreme. Occasionally there are derangements of digestion, and sometimes also the appetite is ravenous, as in diabetes mellitus, though less frequently so. Severe lumbar pains are frequent, together with diarrhea and derangement of mental and sexual functions.

These symptoms may occur suddenly in the midst of apparent health, or they may supervene upon others or be substituted for them, chiefly those of a nervous character, which may be the result of the nervous lesion

causing the polyuria. Such symptoms are headache, restlessness, irritability, sleeplessness, what is commonly called *nervousness*, more rarely convulsions, delirium, paralyses—indeed, any one or more of the great variety of symptoms which result from organic or functional nervous disease. Sometimes these symptoms succeed upon the polyuria or are increased by it. It is certain that the milder nervous symptoms are sometimes the result simply of the inconvenience and annoyance caused by the two cardinal symptoms, polyuria and thirst. The patient is kept busy, as it were, night and day, in passing water. It is not surprising that such a patient should be fretful and irritable, and that sooner or later his health should be broken if the symptoms are not relieved.

Physical and Chemical Characters of the Urine.—As to the quantity of urine passed, it is enormous, exceeding often the amount passed in saccharine diabetes. As many as 43 liters (90 pints) are recorded by Trousseau, and one-fourth this quantity is common. It has been said, even, that the quantity secreted sometimes exceeds the amount of fluid ingested, but this is impossible for any length of time, unless water is absorbed from the atmosphere, which is not impossible. In point of fact, the water excreted is always a little less than that ingested, either as drink or in the solid food. As the quantity of urine excreted increases or its normal acidity diminishes, its color disappears and its specific gravity declines. In one case under our care the specific gravity was scarcely 1001, while the urine in moderate bulk was absolutely colorless. Again, a faint greenish tinge is exhibited by the urine in bulk.

The *duration* of the condition varies greatly. Sometimes it continues through life with no inconvenience except that from the constant diuresis and thirst. Willis records a case lasting 50 years. On the other hand, it is seldom of brief duration; indeed, there is needed a certain chronicity in order to admit it in the category of diseases. One case is reported as terminating fatally in seven weeks. Under prognosis will be found some further information as to duration, but it may be said, in general, that most cases which terminate unfavorably and most which recover completely do so within a year. Tyson had under his care for 11 years a lad of 17, who was able to work quite hard much of the time.

No complications arise except such as cause the disease or result from it. Among the latter is occasionally dilatation of the pelvis of the kidney, and atrophy of this organ is mentioned, due to pressure of the accumulated urine and resulting in a sacculated condition. The symptoms of the malady are almost always influenced, and sometimes even cut short, by intercurrent disease, especially of a febrile character, or even by a profound physical impression, as long-continued suppuration after a blister. The boy referred to was an aggravated choreic before he became diabetic.

Diagnosis.—The diagnosis of diabetes insipidus is very easy. The persistent thirst, polyuria, and absence of sugar from the urine are pathognomonic. The only possible error is mistaking the polyuria of chronically contracted kidney of *interstitial nephritis* for that of diabetes insipidus. In addition, however, to the fact that a careful examination for albumin will disclose it in the urine of contracted kidney, there are casts in the urine of nephritis and physical examination will show hypertrophy of the heart with

high blood-pressure, the quantity is never so large, nor is the thirst so extreme; so that it would seem only necessary to mention the possibility of such an error in order to avoid it.

Prognosis.—It is extremely unusual for a case of diabetes insipidus to terminate unfavorably unless there have been also present symptoms pointing to serious nervous lesion. Recovery is not infrequent. According to Roberts, of 67 cases collected, 16 are reported as complete recoveries and 14 ended fatally, nearly an equal proportion. The remaining 37 were still in progress. In cases of recovery or death the duration is comparatively short. Of the 16 recoveries, in nine the duration was less than a year; in one, four years; in two, 18 and 19 years, and in the remainder, some years. Of the 14 fatal cases, nine terminated in less than a year, one *in seven weeks*, and two in two months; the other two survived 18 months and 20 years, respectively. Of the 37 cases in progress, only five continued for a year or less. The remainder lasted for periods ranging from something over a year to 59 years.

These results seem to be tolerably independent of treatment. It may be said, therefore, that, as a rule, cases that last more than a year are likely to continue, but ordinarily only require to be furnished with an abundance of water to keep them tolerably comfortable. According to Dickinson, cases due to drunkenness are more likely to run a severe and rapid course, usually terminating fatally within a few months, and one terminated thus in two months.

Treatment.—A Wasserman test should be made in all cases. For syphilic cases are often amendable to treatment. The treatment of diabetes insipidus would naturally resolve itself into the treatment for the disease of which it is the symptom rather than of the symptom itself; but as the former is very frequently undiscoverable, it must consist mainly of efforts to diminish the secretion of urine, and with it the thirst.

First, it is generally conceded that there should be no restriction in the drinking of water or other harmless fluids, for the diuresis is not so much caused by the great ingestion of water as the thirst is caused by the diuresis. It should be mentioned, however, that one or two instances are reported wherein improvement seems to have resulted from such restriction; and if, as in some cases, a habit of drinking has been the initial event, moderate restriction may be reasonable. Caution should be used in the administration of drugs, though our experience is not that of Dickinson, who says that "remedies designed to restrain the urinary secretion seldom fail to do harm." The older remedies are ergot, opium, gallic acid, and valerian; of all, the doses ultimately used are usually large. In one patient the symptoms subsided under the use of gallic acid after full doses of ergot had failed. In another, probably due to syphilis, the effect of the iodid of potassium was shown in an aggravation of the symptoms whenever it was discontinued and an amelioration when it was resumed. Of all drugs, the iodid of potassium is most frequently followed by improvement.

Trousseau and Rayer claimed extraordinary results from the use of valerian, the former using the fluid extract in large doses—2 1/2 drams (6 c.c.) a day, which was increased to 1 ounce (30 c.c.) daily in one instance. Rayer used the powdered valerian and the valerianate of zinc, giving the

latter in pills in gradually increasing doses until 20 grains (1.25 gm.) a day were given. At the present day the more palatable elixir of valerianate of ammonia, combined with bromid of potassium, is to be preferred. A gradual reduction of the intake of salt and protein is said to be useful at times.

James B. Herrick reports a case[1] in which marked reduction in the amount of urine followed lumbar puncture. The constant galvanic current has been recommended, and in cases of spinal lesion may be expected to be of advantage.

Tonics and nervines, such as strychnin, iron, arsenic, salts of quinin, cod-liver oil, etc., are appropriately added to the treatment with a view to sustaining the strength of the patient, which is apt to fail. To these are to be added fresh air, sea air, exercise, and all possible favorable hygienic influences.

Hygiene is even more important than in diabetes mellitus, and should include a careful attention to the skin, warm clothing, warm baths, frictions, etc., in order to divert a portion of the circulation from the kidneys to the skin. The first should also be quenched when possible by bits of ice and acidulous fluids.

OBESITY.

SYNONYMS.—*Adipositas universalis; Polysarcia adiposa; Corpulence.*

Definition.—Obesity may be defined as an inconvenient accumulation of adipose tissue in the body.

Etiology.—The most usual cause of an excessive accumulation of fat doubtless is overeating associated with an inactive life; and though it may be true of some fat persons that they are really moderate eaters, careful examination will generally prove that they are not. Heredity exerts an undoubted influence, and we find corpulence running in families. Commonly it does not make its appearance until after 35 years of age, but in this country particularly it is often seen earlier, in boys and girls of ten years and upward. Gouty persons are often fat, probably from the causes which produce these diseases.

Of foods, each one of the representative varieties, albuminoids, carbohydrates, and fats, is capable of contributing fat, deposited in fat vesicles in the body, and it has even been said that albuminoids furnish more of the fatty tissues of the body than the carbohydrates. Certain it is that a person may become corpulent who eats very little fat. In most cases, however, corpulent persons are found to be liberal consumers of all three of the food elements. While the carbohydrates are direct sources of fat production, it is generally conceded that they act largely by sparing the fats derived from other sources. They decompose and oxidize so rapidly, and thus give themselves up so readily to force production, that the stored fats are not called upon. Thus it is that sugars and starches indirectly favor corpulence. To this class belong also alcohol, and especially beer, which contains over five per cent. of carbohydrates, in addition to from three to four per cent. of alcohol, and it is well known that liberal beer drinking fur-

[1] "Archives of Internal Medicine," July, 1912.

nishes a large quota of fat men. A second method in which large quantities of alcohol contribute to adiposis is by hastening albuminous metamorphosis, setting free the carbohydrate group of the fatty molecule readily converted into fat, which is deposited, among other situations, in the liver, giving rise to the fatty liver so constantly found in drunkards.

Another cause of corpulency is muscular inactivity. Fat is consumed by muscular contraction, and its absence must contribute to fat accumulation, and one need not go far to see its evidence in many who lead lives of idleness. Oertel has especially called attention to the fact that a simple diminished ingestion of fluids, without other changes in the diet, will reduce the amount of fat. The effect may be brought about in two ways; first, by diminishing the work of the heart and thereby favoring oxidation; second, by an effect which is not so much the diminution of fat as a withdrawal of water—a sort of "desiccation," as it is called by Strümpell.

The subjects of anemia and chlorosis often become fat, probably because of defective oxidation, growing out of a diminished supply of oxygen, which the crippled corpuscles are unable to carry in sufficient quantity.

Sexual continence probably contributes to corpulence, since eunuchs are well known to grow fat, and both women and men are disposed to grow fat when the sexual function begins to abate. Finally, corpulence itself favors the further accumulation of fat, first by interfering with the muscular activity of its subject, and therefore with the oxidation of fat, and, again, diminishing combustion by reason of a reduced demand for heat, the fat itself conserving heat by preventing its radiation.

In Cushing's book upon Diseases of the Pituitary Body attention is called to the marked accumulation of fat in diseases of the hypophysis together with the increase of sugar tolerance. The "symptom complex of adiposity, high sugar tolerance," etc., is indicative of secretary deficiency of the posterior lobe (of the pituitary body). In children of undergrowth of slow sexual development and very fat there is doubtless some disease of the hypophysis.

Morbid Anatomy.—There is no essential morbid anatomy of corpulence. Incidentally the liver may become infiltrated with fat and enlarged, as may also the heart; later there may be dilatation of the latter. The lungs, too, may become infiltrated with fat. Even the blood-vessels may contain fat drops in the media and intima.

Symptoms.—A description is scarcely needed of the anatomical condition which constitutes obesity. The round, plump face, the double chin and hanging cheeks, the enormous girth of the body, the pendulous belly and elephantine arms, legs, and thighs need no further description. The labored, waddling gait is often conspicuous. The first evident indication of harmfulness due to corpulence is an increased frequency in the breathing-rate, at first on slight exertion and later independently of it. This is in part a true cardiac asthma—due, first, to the fact that the heart cannot push the blood through the lungs rapidly enough to permit its aeration at the ordinary breathing-rate; and, second, to the fact that the motion of the lungs is also restricted. The latter is due to the accumulation of fat over the thorax and in the mediastinum, and to the accumulated intraabdominal fat and probably enlarged liver, which interfere with the proper descent of the

diaphragm. This leads at first to cardiac hypertrophy, further stimulated by the extra work demanded of the heart in propelling the increased bulk of the blood; further augmented by resulting arterio-sclerosis, and impeded venous circulation. Later the fatty infiltration of the muscular walls of the heart leads to further embarrassment in its action and to impairment of its nutrition, whence come cardiac weakness and ultimate failure, with edema, pericardial and pleuritic effusion, and sometimes sudden death.

The pulse, hard to find, is usually frequent, but may be slow and irregular. The heart can be examined only with difficulty, on account of the large accumulation of fat, and the normal sounds are feeble and distant. The situation of the apex can be found only by the aid of the stethoscope. Intertrigo is often an annoying symptom, and great care is required to avert it. Interstitial nephritis may be superadded. By no means all corpulent persons run this course. Many lead lives of considerable comfort.

Treatment.—This consists in acting upon two principles: first, furnishing less food to oxidize, and, second, increasing the oxidation of the fat in the body.

The first is accomplished by cutting down the quantity of all kinds of food, but especially carbohydrates. Sugar should be prohibited altogether, and saccharin substituted, if sweetening is desired. Bread may be taken in small amounts, say two ounces, well toasted and with it a thin layer of butter; or hard biscuit may be substituted. A cup of tea or coffee with a little milk may be allowed; also a single egg at breakfast or luncheon; meat once a day. The latter may be of any kind, and with it may be taken green vegetables, such as peas, string-beans, tomatoes, cabbage, spinach, Brussels sprouts, lettuce, celery and the like, omitting altogether rice, potatoes, and the farinacea, in general. A little cheese may be allowed.

Only small quantities of fluid should be permitted at meals—just enough to aid in the solution and digestion of food. This may be tea, coffee, water, or skimmed milk, the first two without sugar or cream. Beer, porter, and sweet wines should be prohibited, but a glass or two of hock or claret with an alkaline mineral water may be allowed.

A list may be made from the general list of diabetic diet. The amount necessary in an individual is about 2000 calories every 24 hours.

A diet of skimmed milk only is a sure way of reducing fat, and a start may be made with it, commencing with 2 ounces (60 c.c.) every two hours and increasing until from 6 to 8 ounces (180 to 240 c.c.) are attained. Unfortunately, very few persons will bear this treatment for any length of time, but, as stated, a beginning may be made with it, and when the patient tires, the other treatment just described may be instituted.

The second indication to promote oxidation is accomplished by exercise, gymnastics, walking, mountain-climbing, or cycling. The last has been effective in reducing the weight of the corpulent, and if combined with a proper diet, may be expected to do more. Massage is also useful, especially in co-operation with the Turkish bath and steam bath. These last help in the "desiccation" of the body, which in turn facilitates oxidation. Great difficulty is experienced in getting the patient to carry out the dietary and to exercise assiduously. Greatly in the way of exercise with a view to reducing adiposity is the unwieldy and cumbersome body and early fatigue which ensues on effort.

Certain health resorts have much reputation for their efficiency in reducing corpulence. Homburg, Marienbad, and Carlsbad are among the most celebrated of these, and I have seen many patients return thence after a cure of four to six weeks markedly improved in all the symptoms which come from obesity. The effect is probably altogether due to the strict diet, the systematic exercise, and the bathing, the massage, and the laxative effect of the waters, although the physicians at the various spas which have a reputation for reducing obesity claim also that the effect of the sulphate of soda, which is a constituent of most of these waters, is to stimulate oxidation in the direction of the fatty structures, while limiting the metamorphosis of the nitrogenous substances. This intermittent method of treating obesity, by recourse to baths and springs once a year while the intervening period is spent in free eating, is not to be recommended. It is much better to adopt a continuous method which may not be as rapid, but is persistent. It must be remembered that the average individual does well on a diet which contains 2300 to 2500 calories in the twenty-four hours. One must give a diet list to the patients consisting of mixed foods, the total calories of which for the twenty-four hours will not exceed the above amount.

Medicinal Treatment.—This is unsatisfactory. Dessicated thyroid has been administered with claimed advantage in doses of 1 grain (0.066 gm.) three times daily, and may be gradually increased to 3 and even 5 (0.35 gm.), with the same precautions as advised in the treatment of myxedema. Under this treatment the loss of weight is sometimes quite rapid, going to show that defective thyroid secretion may be a factor in causing obesity.

Systems of Diet.—Mention should perhaps be made of the so-called systems of diet for reducing corpulency, a number of which have been suggested. Those especially deserving of notice are the Banting system, Ebstein's method, the method of Dancel-Oertel, in addition to the mineral water cures. It is, of course, impossible in the limited space of a text-book to give these methods in full. The principles of their application will alone be considered and the student is referred to special sources for their description.[1]

(1) The Banting system consists in the administration of a large amount of albuminous food, especially lean meat to the exclusion of fats and carbohydrates; green vegetables being allowed *ad libitum;* (2) Ebstein's method demands moderation and restriction in the quantity of foods generally, and for the fat-forming carbohydrates substitutes fat itself, which does not increase stored fat and by diminishing appetite favors loss of weight. Sugar and potatoes are excluded; (3) the Dancel-Oertel method reduces especially the quantity of water and other liquids, only a maximum of 800 grams (about 1 1/2 pints) of water mixed with wine, and twice a day a cup of coffee or tea; of solid foods, nitrogenous alimentary substances and vegetables, especially such as contain little water, with fat only in such quantity as to render the dishes palatable. Oertel emphasizes the mechanical advantages of restriction in the use of water upon derangements of the vascular system; (4) von Noorden's dietary as follows: *Eight o'clock,* 80 grams of lean, cold meat, 25 grams of bread, one cup of tea with a spoonful

[1] "Dietetic and Therapeutic Hints to the Visitors of Bad Homburg." By Heinrich Will, 1893.

of milk, no sugar. *Ten o'clock* one egg. *Twelve o'clock*, a cup of strong meat broth *One o'clock* a small plate of meat soup flavored with vegetables, 150 grams of lean meat of one or two sorts, partly fish and partly flesh, 100 grams of potatoes with salad 100 grams of fresh fruit, or compote without sugar. *Three o'clock* a cup of black coffee. *Four o'clock*, 200 grams of fresh fruit. *Six o'clock* a quarter of a liter of milk, if desired, with tea. *Eight o'clock*, 125 grams of cold meat, or 180 grams of meat weighed raw and grilled, eaten with pickles or radishes and salad, 80 grams of Graham bread, and 2 or 3 spoonfuls of cooked fruit without sugar. Von Noorden advises also in addition to the three meals, smaller quantities of food at shorter intervals, so as to remove the tendency to weakness sometimes complained of by these patients. He permits also twice a day a glass of wine, and mineral waters, weak tea or lemonade *ad libitum* at the meal times or between. Occasionally he gives a "hunger day;" (5) the mineral water cures, as might be expected, are based chiefly upon the use of such waters, especially those containing sulphate of sodium and chlorid of sodium, the cold springs being preferred. The springs are thus classified: (*a*) cold waters containing sulphate of soda—Marienbad, Tarasp, Schulz-Tarasp, Frazensbad, Elster, Cudowa, and Rolitsch—carbonic acid waters; (*b*) hot springs containing sulphate of sodium—Carlsbad, Bertrich; (*c*) cold waters containing chlorid of sodium—Homburg, Kissengen, Nauheim, Neuhaus and Oeynhausen; in America the Saratoga springs; (*d*) springs containing iodin—Hall, Krankenheil, Salzschlirf, Kreuznach, Münster am Stein. The drinking of waters at these places is combined with the use of saline, carbonated, mud, and steam baths.

Prophylaxis should not be overlooked especially in the case of hereditary tendency on the principle of the old claim of an "ounce of prevention" as contrasted with a pound of cure, and much may be accomplished in families in which there is tendency to corpulence.

For *Adiposis Dolorosa* see Nervous Diseases, subheading Neuritis.

RICKETS.

SYNONYM.—*Rachitis.*

Definition.—"There is a disease of infants called the rickets, wherein the head waxeth too great, while the legs and lower parts wane too little" (Thomas Fuller, 1608–61). This quaint description of the celebrated English chaplain, written over 250 years ago, remains so nearly correct at the present day that we cannot forbear adopting it. It is further defined as a constitutional disease characterized by deformity in bones, due to cell overgrowth and deficiency in lime salts.

The term rickets is derived from the old English word *wrickken*, to twist. "The rickets" was evidently known for some time by the laity before it received its description by F. Glisson, in 1650, who suggested the change of name to rachitis from the Greek ῥαχις, the spine.

Etiology.—Rickets rarely begins before the child is six months old or later than the age of 18 months, though a form was described by Sir William Jenner coming on as late as the ninth or even the twelfth year. Again,

certain races tend to be rickety, especially the negro and the Italian. Foul air and bad food, absence of sunlight and exposure to dampness and cold, are more potent factors, and it is likely that a defective composition of the breast-milk, including a deficiency in the phosphates, is the strongest. Animal fat and proteids seem deficient in those foods which favor the development of rickets. There is also a lack of assimilation of lime salts in rickety children. Prolonged lactation may contribute to such deficiency.

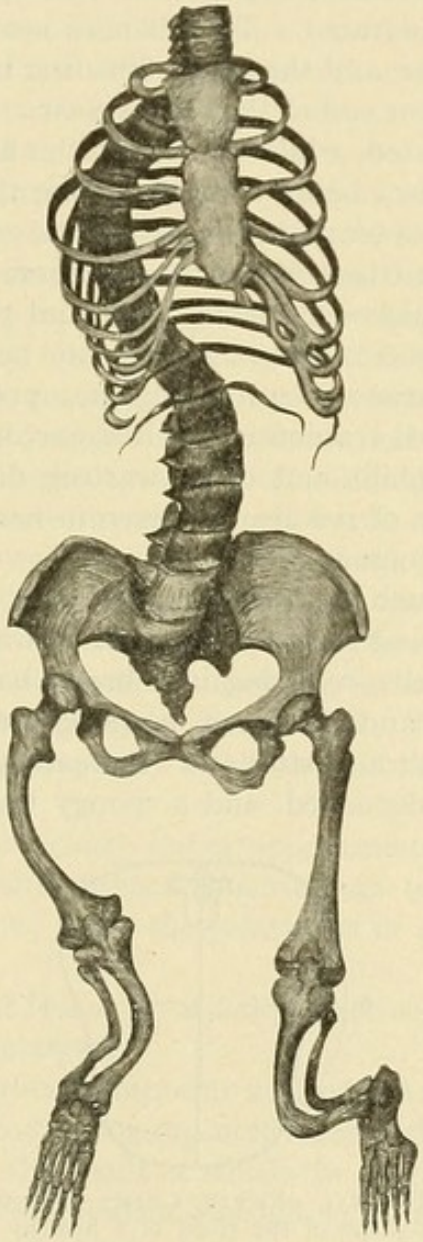


FIG. 132.—Deformed Skeleton from a Case of Rachitis—(*from Atlas du Musée Dupuytren*).

Poor feeding of infants with certain proprietary foods unquestionably causes rickets because of their deficiency in required constituents. Sterilized milk, and unhygienic surroundings are fertile sources of the disease. It is a disease of the city rather than of the country, and of the Continent of Europe rather than of America. Vienna, London, and Paris are prolific fields. In the first-named cities from 50 to 70 per cent. of all children brought to the clinics are said to be rickety. Parrot held that congenital rickets was a form of syphilis, basing this view on studies in the French capital. Probably achondroplasia. Boys and girls are equally liable to rickets.

Morbid Anatomy.—Minute examination recognizes numerous cells in the spongy spaces in the bone. The studies of Kassowitz lead him to believe that a hyperemia of the periosteum, the marrow, the cartilage, and the bone itself is the fundamental condition responsible for the abnormal development. His views may be regarded as a refinement and development of those originally suggested in 1650 by F. Glisson, who held that an excessive vascularity was at the bottom of the changes.

The morbid anatomy shades somewhat into symptomatology, and the two can scarcely be separated. The changes are mainly in the bones of the skull, the long bones, and the ribs. The first may escape if the disease sets in after the middle or end of the second year. The frontal and parietal eminences are exaggerated, while the top of the head and the occiput are flattened, the whole effect being toward making the head square or "box-shaped." The fontanels remain open some time—until the second or third year of life—while the edges of the bones where they come together to form the sutures are thickened, though soft and yielding. In addition to these changes, or instead of them, there may be large areas of delayed ossification in the parieto-occipital regions, producing yielding spots, constituting the so-called craniotabes of Elsässer; but as craniotabes occurs in connection with syphilis and other wasting diseases of young infants exhibiting no other sign of rickets, and even in new-born infants, it cannot be regarded as pathognomonic.

In the *long bones*, such as the radius and ulna, swelling of the cartilage between the epiphysis and shaft is apparent. Owing to the rapid proliferation of the cartilage cells, resulting in a broad band of jelly-like material between the cartilage and the bone, a spongy structure is rapidly built up, deficient in strength and stiffness. Beneath the periosteum the same gelatinous material is deposited, and a spongy tissue is formed instead of

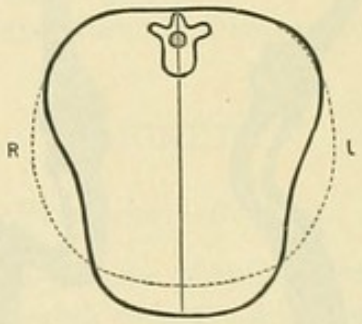


FIG. 133.—Rickety Chest—(*after Gee*).
Dotted line indicates the shape of the chest of a healthy infant about the same age.

normal bone. The process of bone formation does not proceed further. There is no deposit of lime salts. The periosteum is loosely attached. The long bones bend easily, especially the tibia, producing the characteristic bow leg, which may occur even before the child walks, when it is caused by sitting cross-legged. The thighs may also become bowed, the inner ends of the condyles prolonged downward and the tibia set outward, producing the "knock-knee." This does not, however, appear until the child begins to walk. In extreme cases the long bones may fracture. Sometimes both the femora and tibiæ are bowed forward.

Quite as characteristic are the changes in the chondral ends of the *ribs*

and in the shape of the chest. The former are enlarged and nodular at the junction with the bone, producing the well-known beaded appearance, which may often be recognized at a glance. The altered shape of the chest-walls, most marked in children who have had much cough, is due to the yielding of the soft costal ends of the cartilages and to a falling-in of the ribs at these points, while the sternum and cartilages are pushed forward, as seen in Fig. 85.

This is especially the case in the region between the fourth and eighth ribs, which may be so bent in as to form a vertical groove, increased during inspiration. Associated with this is sometimes a transverse groove, known as Harrison's groove, starting at the ensiform cartilage and passing transversely outward toward the axilla. At the same time the arch of the ribs below may be widened and the belly thrown forward by the arching inward of the vertebræ. Extreme degrees of this chest deformity produce the prominent sternum constituting the "chicken breast" or "keel-shaped" thorax. Other changes in the bones are an exaggeration of the normal double curve in the clavicle; a bending of the humerus, usually at the insertion of the deltoid, the radius and ulna may be curved and twisted, the articulations knotted and bulbous, loose and mobile, because of relaxed ligaments. The spine is also often altered, the change being for the most part an increase in the normal curve outward in the cervico-thoracic portion and inward at the lumbo-sacral. In other cases there is curvature. The scapula is often thickened, antero-posterior curvature. Lateral curvature is not so common. The pelvis is distorted and twisted, and the antero-posterior diameter is markedly lessened. The rickety pelvis is one of the well-recognized causes of dystocia. These changes are all the result of mechanical causes, such as the weight of the body or muscular traction.

Chemical analysis of rickety bones approximately reverses the normal proportion of organic and mineral constituents (calcium salts), reducing the latter to 35 per cent., while the gelatinous or organic matters amount to 65 per cent.

An *enlarged liver* and *spleen* are usually present, and sometimes also the mesenteric glands are enlarged.

Symptoms.—The earliest symptoms noticed are not invariably the same. There is usually *profuse sweating*, especially about the head and neck, and a mild degree of fever, as the result of which the child is inclined to throw off the bed-clothing. There is evident discomfort in being handled. The last symptom is apparently due to a *tenderness* of the skeleton, causing pain when the child is raised or danced up and down after the manner of amusing children. Along with these are the less distinctive symptoms of indigestion, indicated by nausea and vomiting, offensive stools containing partly digested milk, and flatulent distention, causing the belly to be prominent. Among other less essential symptoms may be mentioned *nervousness*, *restlessness*, *peevishness*, and *infantile convulsions*, the relationship of which to rickets is not accidental, and was pointed out by Jenner. *Tetany* and *laryngismus stridulus* are also often symptoms.

Concurrently it is noticed that teething is delayed, and we have the authority of Sir William Jenner that if there are no teeth at nine months

there is something rickety about the child. But dentition is often delayed after this time in children who were not rickety and who did not become so. In rickety children the teeth whch are cut soon decay. *Muscular weakness* is characteristic, so that the child cannot sit up and makes but a feeble or no effort to walk. Such muscular weakness has been mistaken for paralysis, whence it has been called the *pseudoparesis* of rickets. Close upon these symptoms, or at least within two or three weeks of the first symptom, follow the *skeletal changes* described under morbid anatomy, page 826. The head is large in comparison with the face, and the skin is pale and thin, and the child has often an old and a wise look quite beyond its years. The appearance of the beaded ribs, the bowed legs or "knock-knees," prominent belly, and curved spine often serve to make the diagnosis easy at a glance. The prominent belly requires some further description, as it varies somewhat at different periods. Before the child walks the normal cervical anterior curve may be increased and a posterior curve present from the first dorsal to the last lumbar vertebra, which may be recognized by holding the child up. After it begins to walk, however, the dorsal spine continues curved backward while the lumbar projects forward. The latter, therefore, contributes also to the prominent belly produced in part by the flatulent distention, and partly at times by an enlarged liver and spleen.

Complications.—These include especially bronchial catarrh and bronchopneumonia, the effects of which are aggravated by the conformity of the chest, the weakness of the ribs, and the feebleness of the respiratory muscles. Collapse of the lung is often a consequence of lung affections. Chronic hydrocephalus is a complication, while many of the conditions mentioned under symptomatology—viz., diarrhea, convulsions, laryngismus stridulus, and the like—may also be so regarded. The rickety child is weak and is vulnerable to all the illnesses of childhood.

Diagnosis.—This is usually easy, although, of course, all the symptoms detailed are not always present in their typical expression. The various spinal curvatures may be somewhat confusing. Thus, the question of *caries* may arise. But the rickety spine differs from that of caries by the wide curve, the absence of angularity, the flexibility of the spine, and the fact that by laying the child flat on its face the curve disappears. The other symptoms of rickets are also present. The lordosis of rickets produces a deformity resembling that of *congenital dislocation of the hip* and of *hip disease*, but here again other signs of rickets are present, while the distinctive signs of the disease in question are absent. Achondroplasia is frequently mistaken for rickets. For diagnosis see chapter on Achondroplasia.

Prognosis.—Rickets is never in itself fatal, and the course is toward recovery. But the child is always in danger from the complications. Such are bronchitis, bronchopneumonia, laryngeal spasm, and convulsions. Walking is always delayed, and the child may be still unable to walk at the end of the second or third year. Mention has been made of the fact that the rickety pelvis in women is one of the most frequent causes of difficult labor.

Treatment.—We should seek to avert rickets by a judicious prophylaxis which consists in keeping the health of the mother at the highest point at all times; this, not by organic food only, but by a judicious admixture of salts

such as are contained in the whole cereal grain, especially in wheat and barley. Frequent pregnancies and prolonged nursing, being acknowledged causes, should be interdicted.

The treatment of the child should be *dietetic, medicinal, hygienic,* and *operative or mechanical.* As the condition depends often upon the lack of ordinary good food, the simple addition of such food in lieu of the mother's milk, if this be found defective, may be all that is required, especially if it be possible to secure that rarely attainable article, a healthy wet-nurse. In the absence of this, beef-juice, the yolk of eggs, peptonized milk, and beef peptonoids may be substituted. Due consideration must, however, be paid to digestion in the selection of food, the stools should be examined daily, and if undigested residue is found, the food should be changed Cod-liver oil inunctions are invaluable, and though in some respects unpleasant. So many children seemingly wrested from death by their use that we value nothing more highly. Saccharine and starchy foods should not be allowed, except in very moderate quantities. The flours of the whole cereals, well baked and cooked as thin gruels and strained, make a suitable addition to the food, while the fruit-juices of orange and lemon may be given in small quantities. Medicines should be cautiously given. Among them are lime salts, as the hypophosphite of calcium or lactophosphate of calcium, 10 grains (0.65 gm.) of either three times a day, or lime-water, or the official syrups containing the salts mentioned. Doses should be carefully, regulated, as digestion is feeble. Minute doses of iron, preferably the citrate or malate, may be given. Phosphorus was recommended by Kassowitz, and is indorsed by Wegener, Jacobi, and Strümpell, in doses of from 1/200 to 1/100 grain (0.00033 to 0.00066 gm.) two or three times a day dissolved in olive oil or cod-liver oil. The principle of the administration of these two drugs is different. The salts previously mentioned are convenient modes of administering calcium, while phosphorus is supposed to stimulate *bone growth.*

The hygienic treatment is more important than the medicinal. Fresh air and outdoor life are indispenable. If the child is warmly clothed and well protected, it may be taken out even in cold weather. It should sleep in a room with the windows *up.* This can be done in the poorest surroundings, but is certainly neglected. There is no reason why the poor cannot get as good air as there is in the neighborhood, if they will keep windows and doors open. *It should not be allowed to walk or even to sit up unless properly supported*—in fact, should be handled as little as possible.

Mechanical appliances may be employed with advantage, especially in lateral bowing, before the bone is hardened. Forcible manual straightening may also be employed in moderate grades of deformity, but should be relegated to the experienced orthopedic surgeon. After ossification is complete, deformities may be corrected by the orthopedic surgeon, by osteotomy chiefly of the bones of the lower extremities, though the radius and ulna are sometimes operated on.

ACHONDROPLASIA.

SYNONYMS.—*Chondrodystrophia fetalis; Epiphyseal dystrophy.*

Definition.—A prenatal disease due to deficiency in the cartilage at the ends of the bones. The long bones cease to grow, while the flat bones, which are not formed from cartilage but from membrane, such as the cranial bones and scapulæ, have a normal growth. As a consequence the legs and arms are very much shortened, while the cranium and trunk are nearly normally developed. The bridge of the nose is depressed and the fingers are shortened and trident. The joints are enlarged from hyperplasia of the epiphyses. Hence the subject, whether child or an adult, shows the effects of the disease more especially when standing, the patient being always below normal height and often a dwarf. The shortening is increased by some tendency to bowing of the bones of the legs. The arrest of development begins in fetal life. Premature birth of achondroplastic fetuses is common.

Etiology.—The cause is unknown, but the joints are enlarged from hyperplasia of the epiphyses, while the arrest in the growth of the diaphysis seems to be due to fibrous outgrowth from the periosteum of the shaft and the epiphysis, restricting development of the former and causing failure of ossification of the cartilage cells.

Diagnosis.—The conditions with which achondroplasia can be confounded are rickets, cretinism, congenital syphilis and osteogenesis imperfecta. Achondroplasia is a congenital disease. The lesions are complete at birth. The deformities present are but exaggerated with the growth of the individual. Rickets is a postnatal disease. The lesions are entirely different in the two affections, and may at once be differentiated by the X-ray. In achondroplasia, the lesion is in the cartilage; the epiphyses are about normal; the enlargement at the ends of the bone is due to cup-like projections of the diaphyses. In rickets the enlargement at the ends of the bones is in the epiphysis itself. There are enlargements forming bosses at the muscular attachments in achondroplasia. These are absent in rickets. In achondroplasia the bones are hard. In rickets they are soft. The chest and trunk are normal in achondroplasia; they are affected in rickets. There is pug-nose in achondroplasia, which is absent in rickets. The vault is normal in achondroplasia and bossed in rickets. The bones affected in achondroplasia are those laid down in cartilage, while any of the bones may be affected in rickets. Achondroplasia is a permanent lesion. A patient with rickets may recover. Apert says, "An individual is born achondroplastic and remains achondroplastic, but an individual may become rachitic and recover."

The differential points from cretinism are the following: A cretin lacks intelligence. Achondroplastics are of normal or unusually bright intellect. The hair of cretins is scant and coarse; that of achondroplastics abundant and normal. The tongue of a cretin is protruded, and there is drooling; this is absent in achondroplastics.

The bone lesion in cretinism is simply an underdevelopment. This is well seen in X-ray plates. Cretins recover under thyroid extract when

treated early. This material has no effect on achondroplastics. Umbilical hernia is the rule in cretins, but absent in achondroplastics.

Achondroplasia may be mistaken for congenital syphilis. In syphilis the pug-nose is due to actual bone disease; in achondroplasia it is due to a premature union of the bones at the base of the skull.

The X-ray will always make the diagnosis.

To quote Schirmer in regard to achondroplasia and osteogenesis imperfecta: "In spite of the analogous clinical symptoms, the anatomic substratum of the two affections is entirely different. Achondroplasia is permanently a defect in the cartilage, while osteogenesis imperfecta is a functional disturbance of the periosteum and the bony tissue."

Treatment.—No known substance has yet been found to affect achondroplasia. Extract of thymus gland has been tried, but found wanting. Perhaps later experiments with some internal gland may be found of value, but, as the disease is practically complete at birth, there is little hope of this. Courtin has attempted a surgical procedure which so far has not been confirmed.

OSTEOMALACIA.

Definition.—A softening which takes place in the bones by a solution of lime salts subsequent to their complete development.

Etiology.—The precise cause is unknown. A geographical distribution, however, exists, in accordance with which it is common on the Rhine, in Westphalia, in eastern Belgium, and in northern Italy. In this respect it is similar to goiter, which prevails in special localities, and it has been suggested on this account that it may be due to some local cause. It is for the most part a disease of adults between 30 and 40 years old, and of women more than of men. It is favored by unhygienic surroundings, such as damp and badly ventilated dwellings. Frequent pregnancy is supposed to be an exciting cause.

Pathology.—There is, primarily, *increased vascularity*. To this succeed a solution and disappearance of the *lime salts* of the bone. These take place from within outward, from the marrow cavity, dissolving out first the lime salts, and then melting away the matrix, enlarging the central cavity until the cortical portion acquires a paper-like thinness. The whole bone has been compared to an "inflated and dried intestine." The product of the solution at first is a mucoid matter that mixes with the marrow. The latter soon loses its vascularity and gradually acquires a thinner but still viscid character and a yellow color. The periosteum is likewise hyperemic and at first thickened. The bones are soft, friable, and easily cut. They sometimes "feel like wet paper." The process is compared to the artificial solution of the earthy salts from bone by hydrochloric acid, and it is supposed that the solvent agent exerts its effect from the medullary spaces and Haversian canals. The process extends unevenly. *It differs from rickets in being a degeneration of fully formed bone, while the latter is a degeneration of developing bone.*

The favorite seats of the process are the vertebræ and the bones of the pelvis and thorax; also of the thighs. The result in the former is an S-

like curve of the spinal column, due to a kyphoscoliosis or backward curvature of the dorsal and a lordoscoliosis or forward curvature of the lumbar part, while the cervical portion in connection with the upper dorsal portion protrudes anteriorly. The thorax is distorted and compressed laterally, while the sternum is prominent and bent. The pelvis is also compressed laterally, the symphysis projects like the prow of a ship, and the sacrum projects forward producing a deformity of the pelvis often discoverable only by internal examination.

Symptoms.—The symptoms are slow in presenting themselves. The first recognizable symptom is usually *pain*, deep seated and severe, oftenest in the sacral region, nape of the neck, back, and thighs, and this pain is persistent and increased by motion, giving rise to a hobbling gait. There is also *tenderness*. *Walking*, therefore, becomes more and more *difficult* and finally impossible, and the patient takes to bed. But this affords no relief, the pain being kept up by the pressure of the bed-clothing and the weight of the body. In the meantime the deformities described under morbid anatomy take place, though those of the pelvis are less obvious externally. *Difficult labor* is an inevitable consequence should the patient conceive, just as it is in rickets. *Dyspnea* is a frequent consequence of compression of the lung by the distorted thorax. *Fractures*, complete and incomplete, are frequent events, even of the ribs as well as of the extremities. In this respect osteomalacia differs from rickets, in which the bones bend but do not break. Such fractures repair imperfectly. Sometimes, on the other hand, the limbs are soft and yielding, and may be bent like a lead pipe. The bones of the head and face are for the most part exempt, though the head is much bent toward the chest, making the stature lower.

The *general condition* of the patient often remains for a long time unaltered. There is little or no fever. The organic functions are normally maintained. The presence of lactic acid in the urine has been mentioned but is infrequent. It is said that phosphoric acid is diminished. Albumin is also sometimes present. Bence Jones's albumose was present in a case reported by Dock. Calcareous concretions have been found in the kidneys and bladder.

Diagnosis.—At first there may be doubt as to the nature of the disease, but as the characteristic symptoms present themselves, its real nature becomes evident. *Disease of the vertebræ and cord* has been confounded with it, but the hobbling gait peculiar to it does not usually resemble any of the gaits of spinal disease. Being a disease of adults, it is not likely to be mistaken for *rickets*. Moreover, it is a disease which affects the shafts of bones rather than the epiphyses.

Prognosis.—The disease is usually ultimately fatal, although death is often long deferred and the course is chronic—from two to ten years. Arrest sometimes occurs, but is only temporary. The disease again starts, and its course is generally irresistible. Death commonly takes place from exhaustion or from some complication like pneumonia. Recovery is not impossible. The so-called cystic degeneration of bone is said to be a consequence.

Treatment.—Theoretically, the indications are the same as for rickets

—viz., to supply the blood with lime salts. Practically, they have not proved of much value. They may, however, be prescribed in the shape of the syrup of the lactophosphate of lime in the dose of from 1 to 2 fluidrams (4 to 8 c.c.) or the syrup of the hypophosphates in the same dose or the latter in combination with iron or with cod-liver oil. Proper hygiene and good food are of the utmost importance. Phosphorus itself is a drug highly commended. (See Rickets.)

Women who are subjects to osteomalacia should be warned against marriage.

MULTIPLE MYELOMA.

SYNONYMS.—*Myelopathic Albumosuria; Kahler's Disease.*

Definition.—A disease characterized by bony deformities, especially those of the trunk, the presence of Bence Jones's albumose in the urine and a more or less rapidly fatal course. This is briefly treated under Albumosuria. Up to June, 1904, 35 cases were reliably reported.

Morbid Anatomy.—The cases which have come to autopsy have revealed a more or less diffuse neoplasm, sarcomatous in structure, invading simultaneously several bones of the trunk, without the occurrence of metastases. It consists of round cells resembling those of the normal cells of the bone-marrow. This neoplasm replaces the bony structure in the cavity of the bone sometimes causing swelling of the bone and spontaneous fracture.

Etiology.—The condition has as yet been traced to no cause.

Symptoms.—Along with albumose in the urine, severe intermittent pain in the affected bones is the most constant symptom. The pain may be in the thigh, in a part or all of it; in the bones of the arm, the sternum or the ribs, or the spinal column. The pain is described as dull and continuous. The pain disappears at times regardless of treatment There is at first no tenderness, but as the disease continues points of tenderness develop when moderate pressure or even the physician's percussion may cause intense pain. When the disease is established, motion of the body, even that of breathing, aggravates the pain. As it continues, extreme weakness develops with anemia. There may be also attacks of nausea and vomiting with intermittent diarrhea, although the viscera, including liver, spleen, and lymphatic glands are normal. There is no fever, the temperature not exceeding 99; the pulse is moderately frequent, rising to 120. The bones become friable and easily broken. There is no edema of the extremities, though death may be preceded by edema of the lungs. Pleurisy has been found antecedent as in Meltzer's case.[1]

Urine.—As stated, the urine contains large quantities of Bence Jones's albumose, one of the intermediate products of albumin digestion between albumin and the ultimate product peptone.

Albumose is thus recognized: It is precipitated from urine by nitric acid, more abundantly than albumin, as a rule, but is redissolved by heating

[1] Meltzer, S. J., "Myelopathic Albumosuria." Reprint from "New York Medical Record," June 18, 1904.

to the boiling temperature. It is precipitated when the urine is heated before it reaches the boiling point. When the temperature reaches the boiling-point the coagulum redissolves leaving only a slight turbidity. As the urine cools the albumose precipitates again, and again dissolves on boiling.

The course of the disease is usually rapid, although one case described by Kahler, that of a physician, lasted over eight years.

Diagnosis.—The presence of albumose in the urine and the above symptoms are pathognomonic of myeloma.

Prognosis.—The termination is invariably fatal usually after a rapid course, but sometimes the disease is more prolonged as in the case of Kahler's physician referred to.

Treatment.—No remedy has been found which can be regarded in any sense as curative. A local application of ice in Meltzer's case relieved the pain.

SECTION IX.

DISEASES OF THE NERVOUS SYSTEM.

GENERAL INTRODUCTION.

HISTOLOGY OF THE NERVOUS SYSTEM.

The difficulties in the diagnosis of diseases of the nervous system are gradually diminishing as the thread of its histology is being unraveled. The studies of Golgi, His, Forel, Waldeyer, Ramón y Cajal, Dejerine, Lenhossék, van Gehuchten, and others have considerably altered previously accepted views. A brief statement of the fundamental features of histology seems, therefore, necessary.

The studies of these and other observers resolve the nervous system into an immense number of units, to which Waldeyer has given the name *neurons*—whence the name neuron theory. Each *neuron* is made up of:

1. A nerve cell body.
2. Protoplasmic processes, or dendrites.
3. An axis-cylinder or axon continuous with the nerve-fiber.
4. Terminal ramifications of the axis-cylinder.

The *axis-cylinder* of a *motor* spinal cell gives off at different intervals lateral branches known as collaterals. These *collaterals* or *paraxons*, and finally the axis-cylinder itself, break up into many fine fibers, known as *terminal ramifications*, or *end brushes*, or *branch tufts*. Each neuron has been believed to be independent of every other—that is no protoplasmic process of one neuron is continuous with that of another, nervous communication being through simple contact or proximity. More recent investigations, however, throw some doubt on this. The protoplasmic processes dentritis conduct impulses to the cell, are *cellulipetal*, as named by Cajal; the axis-cylinders axons conduct impulses away from it and are *cellulifugal*. The nutrition of the neuron depends largely on the cell body. If the latter is intact, the processes are preserved. If it is injured they waste, or if they are cut off they degenerate; on the other hand, the cell body suffers when its processes become diseased.

The motor neurons having their cell bodies in the gray matter of the brain, are called central neurons; those neurons having their cell bodies in the spinal cord and in the ganglia on the posterior roots, are called peripheral neurons. The end brushes or terminal ramifications of a central motor neuron surround the body and protoplasmic processes of a peripheral motor neuron, while those of the peripheral neuron are in connection with a motor plate. The axis-cylinders of the central and peripheral neurons traverse chiefly the white tracts of the brain and spinal cord and the peripheral nerves. The cells of the anterior roots of the spinal nerves lie in the anterior cornua of the gray matter, and have the protoplasmic processes short and the axis-cylinders long. (See Fig. 135.)

The cells of the posterior roots are situated in the ganglia on those roots; the axis-cylinders of these cell bodies divide soon after leaving the cell body, one process passing to the periphery, the other to the spinal cord. Communication between different parts of the nervous system and with the rest of the body is thus rendered possible. The processes extending to the periphery receive impressions from the exterior and carry them cellulipetal to the ganglion cells on the posterior roots of the spinal nerves, whence they are conveyed by the axis-cylinders cellulifugal to the cord. This impression may result in a reflex act, or it may proceed to the brain and give rise to a volitional act through the motor tract.

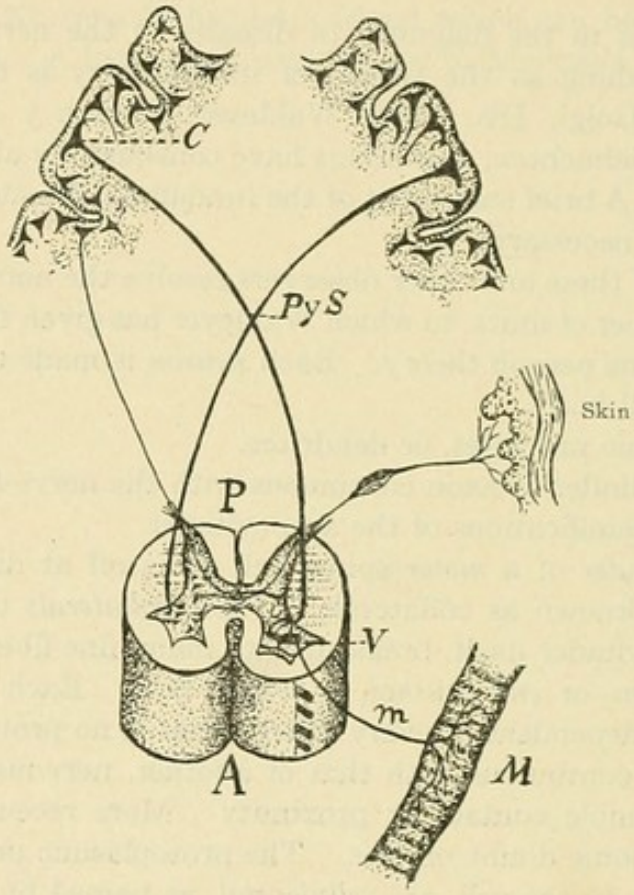


FIG. 134.—Diagram of an Element of the Motor Path—(*after Strumpell, modified*). *C*. Motor ganglion cell in the cerebral cortex. *Py S*. Lateral pyramidal tract, central or upper motor neuron. V. Ganglion cell of anterior horn. *m*. Motor nerve, peripheral neuron. *M*. Muscular fiber.

A motor impulse starting from the brain cortex must pass through at least two sets of neurons before it can reach the muscles. In this course it is cellulifugal from the cell in the cortex, cellulipetal to the cells in the gray matter at different levels in the anterior cornua, and thence cellulifugal from the latter cells to the various muscles of the body, ending in the end-plates. Hence we speak of the motor tract as being composed of two segments, an upper and a lower. The neurons of the upper motor segment have their cell bodies and protoplasmic processes in the cortex in front of the fissure of Rolando. The axis-cylinder processes run through the internal capsule and the cerebral peduncles, through the pons, medulla oblongata, and cord, ending in terminal ramifications around the protoplasmic processes and cell bodies of the lower segment. The neurons of

the lower segment are those having their cell bodies and protoplasmic processes in the anterior cornua of the gray matter, while their axis-cylinders leave the spinal cord by the anterior roots of the spinal nerves, to be distributed as described. The *upper segment*, in large extent at least, is a *crossed tract*—that is, the neurons composing it have their cell bodies and protoplasmic processes in the cortex, while their axis-cylinders cross the middle line to end about the cell bodies in the opposite half of the spinal cord; so that motor impulses starting in the left half of the brain produce contraction in the muscles of the right half of the body, and *vice versa*, although both sides of the brain probably innervate unequally each side of the body. (Fig. 135.) The *lower motor segment is a direct tract*—that is, its neurons, and the muscles to which they are distributed, are all on the sane side of the body.

The path for sensory conduction is also composed of segments, but the direct route of sensory conduction is more complicated and our knowledge is much less exact. The cell bodies of the lower neurons are in the ganglia on the posterior roots of the spinal nerves and in the ganglia of the sensory cranial nerves. These ganglion cells have a single process, which, after leaving the cell, divides in a T-shaped manner, one branch running into the central nervous system and the other toward the periphery. (Fig. 134.) The process which connects with the periphery is regarded by some as a protoplasmic process, while that which passes to the center is known as the axis-cylinder. The former runs in the sensory nerves, starting from the various specialized sensory apparatus of the periphery. The axis-cylinder enters the cord by the posterior roots. After entering the cord it divides into an ascending and a descending limb, which traverse the posterior columns. The descending branch runs a short distance and ends in the gray matter of the same side of the cord, giving off a number of collaterals, which also end in the gray matter. The ascending branch may end in the gray matter soon after entering the cord, or it may run in the posterior columns as high as the medulla oblongata, ending in the nuclei of the posterior columns. Thus the *lower segment* is also a *direct tract* terminating in the gray matter of the posterior cornua at different levels, and in the gray matter of the medulla oblongata. (See also section on Spinal Cord.) The *upper segment* starting from these is a *crossed tract*, crossing at different levels, so that sensory impressions are ultimately lodged in the brain on the side opposite that whence they start in the periphery. The so-called muscular sense, perhaps better called the sense of position, is probably conducted upward on the same side in the columns of Burdach and Goll on each side of the posterior median fissure. The exact termination of the sensory fibers in the cerebral hemisphere is not known, but they pass up in the tegmentum of the pons and possibly in the internal capsule. It is believed by many that these processes terminate in the optic thalamus, and that from here the impulses are conducted to the cortex by means of another set of neurons. Recent investigations seem to show that the sensory area of the brain is posterior to the Rolandic fissure.

Both motor and sensory spinal nerve roots are connected with definite segments of the spinal cord. They descend a variable distance within the spinal canal, unite within the intervertebral foramen, but external to the

point where the roots perforate the dura mater and pass through the foramina as spinal nerves. But in their distribution they do not retain the same definiteness, the same sensory and motor areas being supplied with nerve fibers from different segments of the cord, and there is an overlapping, as it were, of parts supplied by different nerve fibers. At the same time, by the combined aid of experiment and morbid physiology, we have learned that movements in certain muscles are accomplished by motor nerves which emanate from corresponding segments of the spinal cord, and that from certain sensitive areas are gathered up impressions which are carried to corresponding sections of the spinal cord. By the same means we have learned that there are areas in the cortex of the brain that preside over certain motions, and areas which have to do with sensation; though with respect to the latter our knowledge is much less definite. We know about as much of the cortical localization of the *special* senses as of sensibility. These facts are the foundation of what is known as *topical diagnosis*, in the case of the brain as *cerebral localization*, by which is meant the inference, from the study of local derangements of sensation, motion, and other functions of the more or less exact site of lesions in the nervous centers. These will be considered with appropriate detail in our study of the diseases of different parts of the nervous system.

GENERAL SYMPTOMATOLOGY.

(Investigation of a Case of Nervous Disease.)

The advantages of a careful method in the study of disease are perhaps more apparent in the case of the nervous system than that of any other of the anatomical divisions of the human body. This is partly because of the number and variety of the affections to which the nervous system is subject, and partly because of the association of certain identical symptoms with widely different lesions.

The primary steps of family and personal history are the same as for other diseases, including age, sex, occupation, and whether married or single. We may therefore pass at once to the study of such symptoms as are special.

I. Phenomena of Motion.—It is immaterial whether we examine first sensory or motor phenomena, but it appears somewhat easier to begin with derangements of motion, and of these (1) *voluntary motion* is naturally first investigated. To this end, the patient is asked to move his limbs, while the strength of whatever motion he is capable is easiest measured by resisting it, and by testing the power of his hand-grasp. For more accurate measurement of dynamometer is used, an instrument devised to measure both compression and traction, although it is more commonly restricted to the former. Advantage may be taken of the fact, too, that the same motion requires different degrees of strength in different positions of the body. Thus it is easier to draw up the thigh when lying on the back than when on the side, and it may be possible in the former position when it is not in the latter. Both extensor and flexor muscles must be thus tested. By such an investigation we discover the presence of a

complete *paralysis* or total loss of voluntary motion, and *paresis* or simple weakening of such power.

By a *monoplegia* is meant an isolated paralysis of one part of the body, as of an arm or a leg. By a *hemiplegia* is meant a paralysis of the entire lateral half of the body, including half of the face, one arm, and one leg, also known as unilateral paralysis. By a *paraplegia* is meant a simultaneous paralysis of the upper or lower halves of the body. Paralysis of the two arms is known as a *superior, or brachial, paraplegia*, of the two legs as an *inferior*, or *crural, paraplegia*, while the word paraplegia alone is often used for the latter condition. A *diplegia* is a paralysis in which upper and lower limbs are affected on both sides of the body, usually attended with spasm of all the extremities, although the term is also employed for bilateral facial paralysis. Though commonly congenital, diplegia may also be acquired.

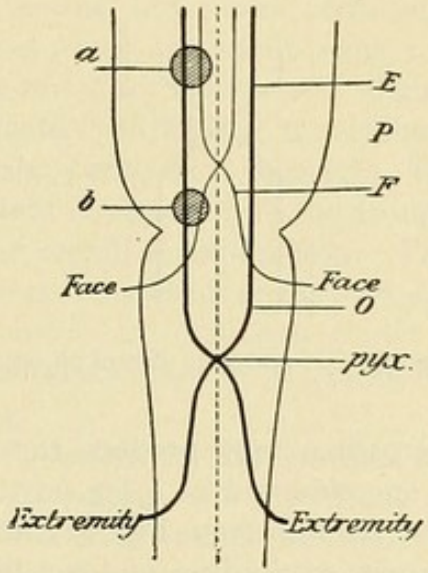


FIG. 135.—Illustrating Crossed Paralysis—(*after Hirt*).
O. Medulla oblongata. *pyx.* Decussation of anterior pyramids. *E.* Nerve fiber going to extremities. *F.* Nerve fiber to face.

Impairment of voluntary muscular power, as thus tested, must be the result of structural change in the motor area of the cortex, in the motor tract of the brain or cord, or impairment in the integrity of the efferent nerves, or it may be more rarely in the muscle itself, "myopathic palsy"; or the power of the will may be abrogated. In diseases of the peripheral nerves, when the paralysis is called peripheral, it is limited to the region of distribution of the affected nerves, whether one or many. It may be said in general that hemiplegia is the usual form of cerebral paralysis, while paraplegia is the expression of spinal paralysis. Monoplegias are due to lesions of the cortex, or of the anterior gray matter of one side of the spinal cord, as in poliomyelitis, or are peripheral palsies; cortical monoplegia is rare.

In all hemiplegias caused by lesions above the pons, the palsy, including that of the face and extremities, is on the side opposite the lesion, but in most lesions in the middle or lower part of the pons there is *crossed paralysis* —that is, there is paralysis of the extremities on one side, and of the face on the other side, provided the central fibers of the extremities and the

facial nerve are involved in the lesion. The reason of that is that the central fibers of the facial nerve cross much higher than do the fibers to the extremities, and in such a lesion the intra-medullary portion of the facial nerve, and not the central fibers connecting its nucleus with the brain cortex are injured. The result is a paralysis of the face on the same side as the lesion and of the extremities on the other. This would be the case with a lesion at *b*, Fig. 136. If, on the other hand, the lesion is higher up, above the decussation of both the facial and pyramidal tracts, as at *a*, the paralysis is on the side opposite the lesion in both face and extremities. Other nerves may substitute the facial in this crossed paralysis as the oculomotor (third nerve) or hypoglossal (twelfth nerve) or abducens (sixth nerve). In rarer instances it is possible that a lesion at the decussation of the pyramids, by cutting the motor fibers of one extremity before they cross, and

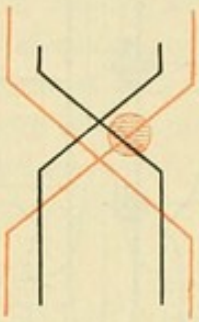


FIG. 136.—Illustrating the possibility of paralysis of arm on one side and of leg on the other.

those of another after crossing, may produce the very rare condition of paralysis of an arm on one side and of a leg on the other. That this is theoretically possible may be seen from Fig. 137, in which the black lines represent fibers to the upper extremities and red lines fibers to the lower, and the red circle the seat of a small hemorrhage.

(2) Having determined this question of muscular strength, and the corollaries which grow out of it, we have next to ascertain to what extent the power of *co-ordination* is influenced. Every muscular act requires the duly proportioned co-operation of a number of muscles; and as the complexity of the act increases, the number of muscles required to co-operate also increases. Such co-operation is termed co-ordination, and its absence is well recognized in the staggering gait of the drunkard, and the condition is known as *ataxia*. There are certain parts of the nervous system which preside over co-ordination—such as the cerebellum, the posterior columns, and probably the direct cerebellar tract, of the spinal cord. Disease of any of these may, therefore, produce ataxia. The ataxic or tabetic gait is described under Tabes Dorsalis, p. 911.

A corollary, growing out of the investigation of the co-ordinating power, is the study of *station*, or the steadiness with which one stands with the eyes closed or open, and it is measured by sway of the head and body, laterally and antero-posteriorly. In health a lateral sway of the head exists to the extent of half an inch (1.25 cm.), and an antero-posterior sway of an inch (2.5 cm.). A sway much beyond these limits is abnormal. Co-ordinating power is also tested by attempting to bring together the

ends of the index-fingers with the eyes closed, an effort which will be unsuccessful if co-ordinating power is lost.

(3) After ascertaining the condition of voluntary motion, co-ordination, and station, we must inquire into the question of possible *motor irritation* or *excessive muscular action* or *spasm*. Spasm may be continuous—*i. e.*, lasting for minutes, hours, or days—when it is known as *tonic* or tetanic; it may be intermittent or *clonic;* or it may be an admixture of both, when it is termed *tonic-clonic*. Tonic spasm is well illustrated by *trismus* or lock-jaw, while tetanic contraction of the muscles of the back produces *opisthotonos*, in which the vertebral column is arched and the body rests upon the back of the head and upon the heels. Tonic spasms are often attended with pain, probably due to pressure on intramuscular nerves, when they are called "cramps." Spasm occurs also in involuntary nonstriated muscular tissue. The presence of spasms implies irritation of motor centers, motor tract, or motor nerves, but motor irritation may also be excited secondarily by some reflex route, the result being a reflex spasm.

Spasm and paralysis are often associated. Thus, a limb may be paralyzed in a state of contraction, exhibiting a peculiar rigidity, and to such a condition the name *spastic paralysis* is applied. This condition may also exist as a state of persistent contraction of the antagonists of the paralyzed muscles, constituting the so-called *contractures*. Paralyses in which there is no such resistance to passive motion are known as *flaccid paralyses*.

Through the combination of tonic and clonic spasm result different varieties of morbid involuntary movements more or less complex. Some of these are the following:

1. *The Epileptiform Convulsion.*—This consists in a succession of clonic and tonic-clonic spasms extending over the whole or a part of the body, throwing the part involved into violent motion. The masseter and the temporal muscles share in the contraction, whence the tongue is often bitten. The convulsion of epilepsy is the type of this form, but the convulsions of uremia, or hysteria, and of organic disease of the brain may be epileptiform.

2. *Rhythmical Contractions.*—These occur in single groups of muscles, and are sometimes seen in apoplexy, and cerebral sclerosis. They may usher in the epileptiform convulsion, or the convulsion may terminate by a gradual substitution of the rhythmical contractions for the more violent spasms. Among rhythmical movements may be included *athetosis*, a peculiar slow, involuntary rhythmical movement, usually of the fingers and hands, but also of the head and trunk, or of the toes. The fingers make slow movements of the nature of extension and flexion, spreading and approximating each other in a striking way. They are a symptom of certain central nervous diseases, especially of the cerebral palsies of children.

3. *Tremors or Trembling Motions.*—These are limited movements—*i. e.*, movements of short excursion which rapidly succeed each other. Tremor is characteristic of paralysis agitans and of some other nervous affections. It occurs in old persons as senile tremor, and in abusers of alcohol and tobacco. When it occurs or increases during voluntary motion, it is known as *intention tremor*, and is characteristic of multiple sclerosis. The immediate anatomical changes on which tremors depend are not known.

4. *Single Contractions.*—These are either sudden twitchings or slow contractions of muscles, seen especially in diseases of the nerves—as, for example, in old facial palsy. They may be single or multiple and persistent. They may be the result of direct motor irritation or reflex in origin.

5. *Fibrillary Contractions.*—These are contractions of separate small bundles of muscular fibrillæ, comparable to the "quivering" of raw flesh. They are independent of voluntary or passive motion. They may be pronounced and wave-like over the muscular substance. They are seen especially when the motor nerve cells are degenerating, as in progressive spinal muscular atrophy or bulbar paralysis. The "quivering" of the eyelid and of the orbicularis muscle below the eye, the so-called "jumper," often an annoying symptom, is an instance of this condition.

6. *Choreic Movements.*—These are inco-ordinated movements, usually separated by short intervals of time, often first seen in the face, later in one limb or over the whole body. They may be very complex and general. They are characteristic of chorea, but also accompany other nervous affections, such as posthemiplegic chorea. Under the term posthemiplegic chorea, however, various movements are sometimes included.

7. *Constant or Co-ordinate Spasms.*—These consist in irresistible complicated movements, like moving forward or moving in a circle or rotating on the axis of the body; also complicated forms of spasm resembling jumping, laughing, screaming, all involuntary and forced. The first group of these is especially seen in disease of the cerebellum and cerebellar peduncles, the latter in severe forms of hysteria.

8. *Nystagmus* is a clonic rhythmical oscillatory and involuntary movement of the eyeball, usually horizontal, sometimes rotatory, more rarely vertical. It is noticed in congenital and acquired affections of the brain, including Friedreich's ataxia and insular sclerosis; also in albinism and in *miners* who work in dimly lighted mines, using the pick while reclining and directing the eyes laterally.

9. *Cataleptic Rigidity.*—In this there is also a tonic contraction of muscles whereby a limb remains for a considerable time in any position in which it may be passively placed, the will being abrogated. If the position of the limb be changed, the limb remains in the new position, and from a resemblance to the behavior of wax under like circumstances it has received the name of "waxy flexibility." It is characteristic of certain forms of hysteria, and may be produced at times in hypnotism. In hysteria it is commonly associated with anesthesia and loss of consciousness. It is also associated with psychoses, especially grave forms of melancholia known as *melancholia attonita* and with *katatonia.*

10. *Associated Movements.*—These are unintentional and uncontrollable movements which take place in muscles coincident with other motions actually intended—as, for instance, a motion in the arm when the patient wills to move only the leg.

(4) *Bladder control* and *rectum control.* Full control over the acts of these organs implies, first, an integrity of the sacral portion of the cord, in which reside the reflex centers regulating these acts; second, the integrity of volition, which, to a certain extent, fortifies such regulation; and, thirdly, integrity of the afferent and efferent nerve fibers con-

stituting the reflex arcs. Through the operation of the reflex center, bladder and rectum both empty themselves when a certain degree of distention is attained. Through the operation of the will such evacuation is put off to a convenient time. Through an undue irritability of the reflex center such evacuation is imperative, and does not bide the will, or it may take place while the will is in abeyance, as in sleep. Thus may be explained some of the cases of incontinence of urine in children. Again, if will-power is lost from disease of the cerebral cortex, evacuations of the bowels and bladder take place involuntarily so long as the sacral cord is intact, but not in a normal manner.

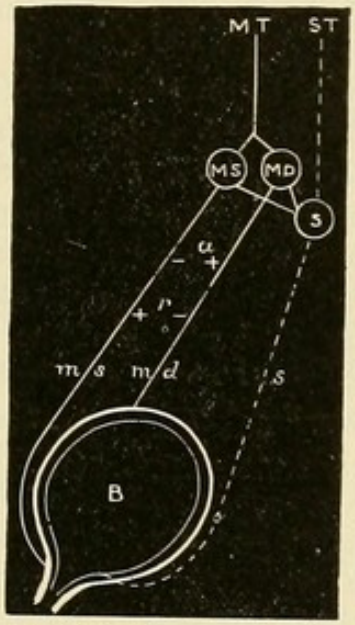


FIG. 137.—Diagram Showing Probable Plan of the Center for Micturition—(*Gowers*). MT. Motor tract. ST. Sensory tract in the spinal cord. MS. Sphincter center, and *ms* motor nerve for sphincter. MD. Detrusor center, and *md* motor nerve for detrusor. *s*. Afferent nerve from mucous membrane to S, sensory portion of center. B. Bladder. At *r* the position during rest is indicated, the sphincter center in action, the detrusor center not acting. At *a* the condition during action is indicated, the sphincter center inhibited, the detrusor center acting.

On the other hand, if the integrity of the sacral cord is lost, there will be no response to the sensory impressions conveyed from a full bladder or rectum, because the reflex arc is interrupted, and the organ remains unemptied; whence torpor or complete paralysis of the bowels and bladder are common symptoms of spinal disease; and while the repletion of the latter may finally overcome the resistance of its sphincter and lead to dribbling, the rectum may go on filling up until it is emptied by the finger or the handle of a spoon. A lesion situated higher in the central nervous system than the sacral portion of the cord may also cause similar disturbance of defecation and urination, probably because of a spastic condition of the sphincters, so that the latter do not relax until the bladder or rectum becomes distended, and finally they lose all function.

Again, if it should happen that the sphincter center is destroyed while the detrusor center is intact, there would be dribbling of urine from the outset, but this is not likely to occur.

(5) The state of the *reflexes*, as they are called, is next ascertained. As

here used, the term "reflex" is applied to a muscular contraction stimulated by a sensory impression, the simplest illustration of which is the retraction of the leg of the sleeper when the sole of the foot is tickled. For diagnostic purposes the reflexes are divided into the "cutaneous reflexes" and the "tendon reflexes."

The *cutaneous* or superficial reflexes are muscular contractions which take place in different parts of the body in response to irritation of sensory nerves of the skin, as by tapping the skin lightly or drawing the finger or a pointed instrument lightly over it. The sudden application of heat or cold or the prick of a pin or pinching are modes of excitation. The contractions are generally confined to the neighborhood of the locality irritated. The skin reflexes are much more easily excited in children than in adults, and in the lower extremities than in the upper; also with varying facility in different persons. They receive various names, according to the situations where they are readily excited. Thus we have the "plantar reflex," where contraction is excited by tickling the sole of the feet, resulting in a movement of the toes or foot, or even in a drawing up of the leg; the "cremaster reflex," contraction of the cremaster muscle and consequent drawing up of the scrotum on stroking or scratching the inside of the thigh. The retraction may take place on the one side of the scrotum only or on both. Then there is the "abdominal reflex," or a contraction of the abdominal muscles when the skin of the abdomen is stroked or scratched. A subdivision of the latter is the "epigastric reflex," produced by an irritation on the side of the thorax in the fourth, fifth, and sixth interspaces. The result is a dimpling of the epigastrium on the side stimulated. Cutaneous reflexes may be brought out in other portions of the body, as in the gluteal region by irritating the skin of the buttock. A contraction of the muscles about the scapula, the "scapular reflex," is produced by an irritation between the scapulæ. To test for the cutaneous reflexes is more important in the lower extremity than in the upper.

The *tendon* reflexes, or deep reflexes, are so called because they are generally elicited by striking upon tendons, while the corresponding muscles are placed slightly on the stretch, care being taken, however, to avoid all active tension in the muscle by the person examined. The blow is made either with the edge of the hand or with a hammer adapted to the purpose, commonly made of rubber. A sharp, sudden contraction of the muscle usually takes place. A similar, though less decided, contraction may be elicited by the mechanical irritation of parts analogous to tendons, as periosteum and fasciæ, and by striking the muscle itself. When the reflexes are in excess, sudden tension alone will excite them.

The most commonly tried of the tendon reflexes is the *knee-jerk*, or *patellar tendon reflex*, produced by striking the tendon of the quadriceps femoris between its insertion and the patella, while the leg is crossed upon its neighbor. The weight of the pendent leg gives a sufficient degree of tenseness. When the knee-jerk is normal, there is a decided rise of the foot with each blow of the hand hammer. This motion may become abnormally increased or diminished. A more limited movement may also be produced by striking the patella itself or the quadriceps tendon above the patella, and, when the reflex is exaggerated, by a very

light tap in these situations or even on the tibia. When thus exaggerated, the reflex may also be brought out in bed, as follows: the quadriceps tendon being put on the stretch by pressing the patella downward in the direction of the leg with the finger, the patella is percussed in the same direction. With each stroke there is a contraction, and the finger and patella are drawn upward. A "clonus," or repeated contraction, may even be produced in this way.

Similar is the *ankle reflex*, produced by tapping the *tendo Achillis* when the calf muscles are placed slightly on the stretch by a slight dorsal flexion of the foot. In health the ankle reflex is usually producible, but in disease in connection with this contraction is shown the most remarkable of the exaggerated reflexes, the "ankle clonus" or "foot clonus." It consists in contractions rapidly repeated so long as the tension of the calf muscle is kept up by pressing the foot toward dorsal flexion. From six to nine such contractions may occur in a second, and sometimes the whole leg is thrown into vigorous contractions. Occasionally a rotary or lateral ankle clonus is seen. One of the best ways to obtain the tendo Achillis jerk is to have the patient kneel on a chair with the feet projecting over the edge of the chair; the muscles are thus relaxed, and a tap over the tendo Achillis produces a movement of the foot.

The Babinski reflex or phenomenon is the extension or turning upward of the toes, and especially of the great toe, obtained by stroking the sole of the foot. In the normal individual, stroking the sole, if it produces any response, causes plantar flexion or turning downward of the toes, especially of the four outer toes. The Babinski reflex usually indicates a lesion or compression of the motor tract in the cord and brain, or probably also of the motor centers in the brain. The response is usually best brought out by stroking the inner surface of the sole from the heel toward the toe, although in marked cases it may be elicited by applying the stimulus to the sole in various positions and directions. Some observations have shown that in infancy the toes tend to turn upward normally when the sole is stimulated. The reflex is obtainable in about 70 per cent. of cases of hemiplegia and diplegia, and in about the same proportion of diseases involving the motor tract in the spinal cord.

Reflexes are also elicited in the upper extremities, but they are much less striking, and occasionally cannot be shown in health. The most important of these are the *arm-jerks*, produced by striking the biceps tendon at the elbow-joint in front, or by striking the triceps tendon above the olecranon. So-called *periosteal reflexes*—reflexes excited by striking the periosteum—may in exaggerated states be produced in the supinator longus and biceps of the upper extremity by striking the lower end of the radius and ulna; also in the adductors of the thigh by striking the internal condyle of the femur.

A *wrist clonus*, resembling the ankle clonus of the lower limbs, may sometimes be obtained when the tendon reflexes of the upper limbs are much exaggerated. It is produced by pushing the hand of the patient forcibly backward and holding it dorsally flexed; involuntary antero-posterior movements of the hand may then occur. The *jaw-jerk* is produced by tapping on the front of the jaw, while the closing muscles of the

jaw—viz., the pterygoids, masseters, and temporals—are placed on the stretch by partially opening the mouth.

The *ophthalmic* (supraorbital) reflex is a pure sensori-motor reflex, elicited by mechanical irritation (tapping lightly with the percussion hammer), or by the application of heat, cold, or pain-stimuli over the distribution of the ophthalmic branch of the fifth nerve, especially in the distribution of the supraorbital branch on the forehead. It is manifested by a fibrillary contraction of the individual fibers in the inferior half of the orbicularis palpebrarum. The sensory impulse travels through the supraorbital nerve (purely sensory) to the pons and thence through the facial fibers (purely motor) to the orbicularis palpebrarum.

The diagnostic value of this reflex lies in the loss of contraction resulting from a lesion cutting the arc in the ophthalmic branch of the trifacial, in the nucleus of the trifacial or of the facial in the pons, or in the fibers of the facial going to the orbicularis palpebrarum. It is therefore of value in localizing lesions of the pons, and differentiating a facial paralysis due to a lesion of the nucleus or its peripheral fibers from a supranuclear or cortical lesion, in which case the reflex is present and increased. It has the same significance as the reflex closure of the eyelids from irritation of the conjunctiva, as this also is a reflex in the distribution of the facial and trigeminal nerves.

Hence in a complete examination the "muscle jerk," or idiomuscular contraction, also known as mechanical muscular irritability, should be tested as well. It is done by a sharp, sudden tap on the muscle with the hammer. The response is of two kinds, first as a sudden contraction, and second as a hump-like rise which subsides slowly. The pectoral muscles are favorite sites for eliciting the pure muscle reflexes. It is, of course, impossible to deny that there is nerve as well as muscle irritation in such a blow.

Both the tendon jerk and muscle jerk are capable or re-enforcement by coincident muscular exertion, as in lifting weights or clinching fists, originally discovered by Jèndrássik[1] in 1883 in the case of the tendon jerk. Mitchell and Lewis[2] also discovered in the course of their study of ataxic cases that the pure *muscle* jerk or hump could be produced after the tendon reflex could no longer be elicited, and that both could be produced by the re-enforcement referred to after they had disappeared to ordinary conditions.

Significance of Abnormal Reflexes.—What are the conclusions to be drawn from modifications in the reflexes? In the first place, it is to be remembered that they vary somewhat within the limits of health. Especially is this true of the cutaneous reflexes, which are also less easily elicited than those of the tendons. In general terms, *diminution* or *absence of a reflex normally present in health* implies either, first, a breach of integrity somewhere in the reflex arc as formed by the centripetal nerve, the motor nerve cells in the spinal cord situated in the anterior cornua of the gray matter, and the motor nerve; or, second, an increase in the

[1] "Beiträge zur Lehre von den Lehnenfleehsen," "Deutsches Archiv f. klin. Medicin," vol. xxxiii., p. 175, 1886.

[2] Mitchell and Lewis, "Tendon and Muscle Jerk," "Trans. Assoc. of Amer. Physicians," vol. i., p. 13, 1886.

reflex cerebral inhibitory influence. The latter would be irritative. Thus, it is well known that disease of one cerebral hemisphere may lessen or abolish the superficial reflexes on the opposite or paralyzed side of the body soon after the onset of a hemiplegia. Breach of integrity may lie in the spinal cord or in the centrifugal or the centripetal nerve. If it is in the centripetal nerve, it may be accompanied by impaired sensation; if in the centrifugal, there will be defective motion. Disease of the centrifugal nerve and of the motor center in the cord may also cause degeneration and wasting of muscle with loss of its irritability.

Increase of the reflexes, on the other hand, implies increased irritability of the motor areas of the cord—when the reflexes are spinal (anterior cornua and possibly of the pyramidal fibers) or a withdrawal of cerebral inhibition, as in certain cases of destructive brain disease or disease of the cord high up. In the case of a cortical lesion the increase in the reflexes is greater on the side opposite to that of the brain lesion, but the reflexes on the same side as the lesion may also be somewhat increased. In certain diseases of the cord there is a *delay* in the manifestation of the *cutaneous reflexes* after the irritation has been applied to the skin, an interval of from ten to fifteen seconds being often recorde before the response ensues. *Increase of cutaneous reflexes* is manifested by an unusual readiness of response in the normal areas, or an extension of these areas beyond their normal boundaries.

In general it may be said that absence of the tendon reflexes is especially characteristic of poliomyelitis and tabes dorsalis, and of all peripheral paralyses and neuritis; also of advanced diabetes mellitus. Abnormal increase is present in spastic spinal paralysis and in cerebral paralyses, being due in the latter instance to withdrawal of the normal inhibitory influences.

Segments of the Cord Presiding over Certain Rexexes.—Further accuracy in the application of a knowledge of the reflexes and of their modifications is secured by a knowledge of the exact portion of the gray matter presiding over the most important of them. Premising that some of these centers are of considerable extent vertically, the following from Gowers may be regarded as approximate for each of the reflexes named:

Superficial Reflexes.—Plantar, opposite second sacral nerve; gluteal, fourth lumbar; cremaster, second lumbar; abdominal, sixth to seventh dorsal; epigastric, sixth dorsal; scapular, fifth cervical to first dorsal.

Tendon or Deep Reflexes.—Calf muscles (foot clonus), fifth lumbar and first sacral; knee-jerk, third and fourth lumbar; flexor digitorum and triceps, seventh cervical; biceps and supinator longus, sixth cervical.

(6) *Paradoxical contraction* is a symptom allied to the reflexes for which no satisfactory explanation has been afforded. It was first studied by Westphal, and is only occasionally observed. In the *tibialis anticus* muscle it is induced by forcibly flexing the foot on the leg. As a result, the foot remains thus flexed for a considerable time, after which it slowly relaxes. In one case the flexion continued for twenty-seven minutes. On repeating the flexion, the phenomenon recurs, but the response gradually diminishes in intensity. Contractions induced by faradism may similarly persist. It has been noticed in both spinal and cerebral disease,

including the early stage of tabes dorsalis, multiple sclerosis, and paralysis agitans. More rarely it may be induced in the flexors of the leg and forearms.

(7) *Electrical excitation of motion* is an important means of investigation in nervous diseases. In health nerves and muscles are excitable by electricity, and in diseased conditions these reactions are liable to change. Motion may be excited by electrical stimulus applied to the muscle through its nerve or directly to the muscle itself. The latter is called direct, the former indirect. This is equally true of the constant or galvanic current, and of faradism or the induced current.[1] Hence every complete investigation should include the use of both currents.

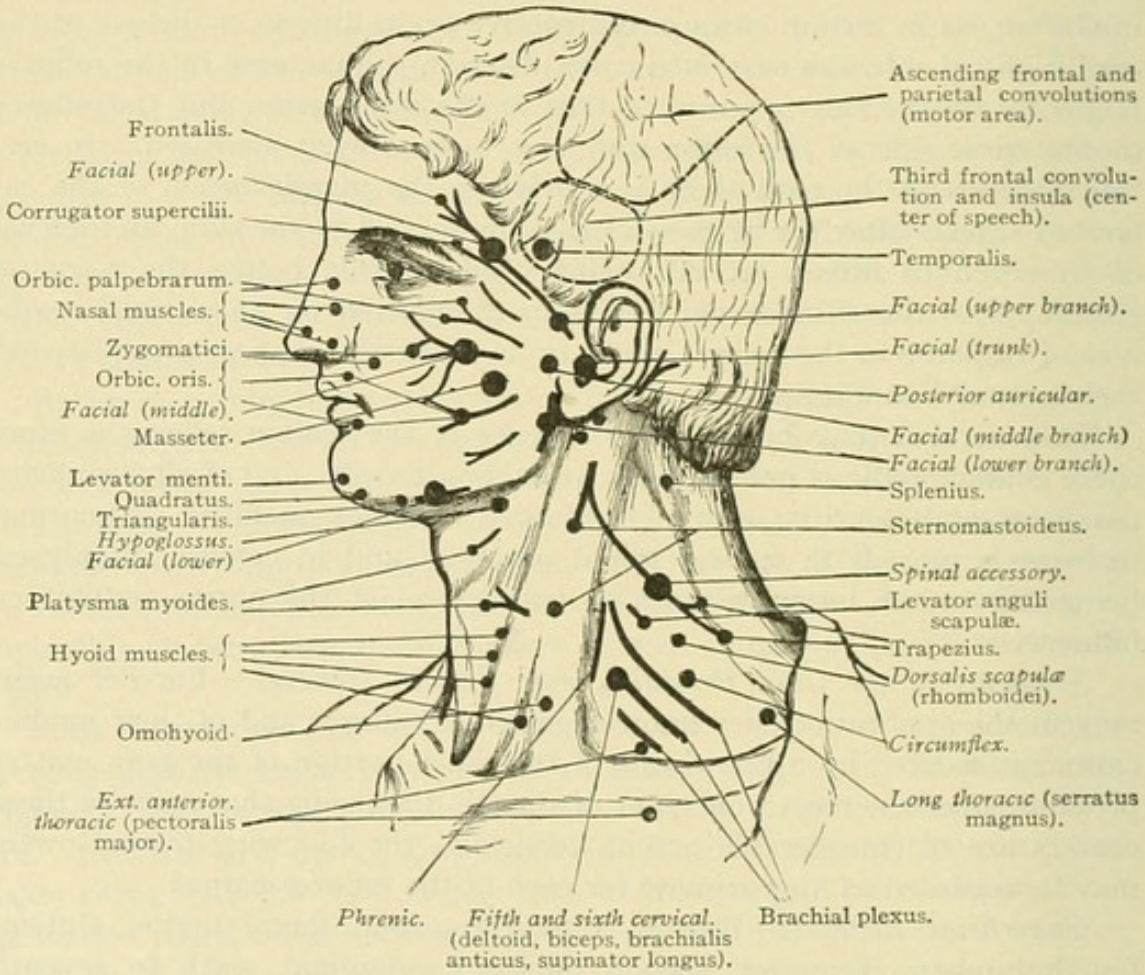


FIG. 138.—Motor Points on Face and Neck—(*after Erb and de Watteville*).

In order to test the electrical condition of muscles and nerves, one electrode, the indifferent pole, may be held in the hand of the patient or placed over the sternum or at the back of the neck, while the other or *testing* pole is applied to the nerve or muscles, selected in accordance with the well-known nerve points of Erb in Ziemssen's plates; or the indifferent pole may be placed on the nerve point of a given muscle or set of muscles, and the testing pole applied to the belly of the same muscle. The testing electrode should be small enough to permit the isolation of a single nerve or muscle.

[1] Under all ordinary circumstances electrical contraction produced in muscles is indirect—that is, through the nerve filaments distributed to the muscle. That the two are, however, distinct may be shown through the influence of curare, which destroys nerve irritability, but allows that of muscle protoplasm to remain.

With the faradic or galvanic battery contractions may generally be produced in health with great facility, either directly or indirectly, although stronger currents are required for direct stimulation. Contractions take place with the galvanic battery only at the making and breaking of the current by the "commutator" or "reverser." A definite law

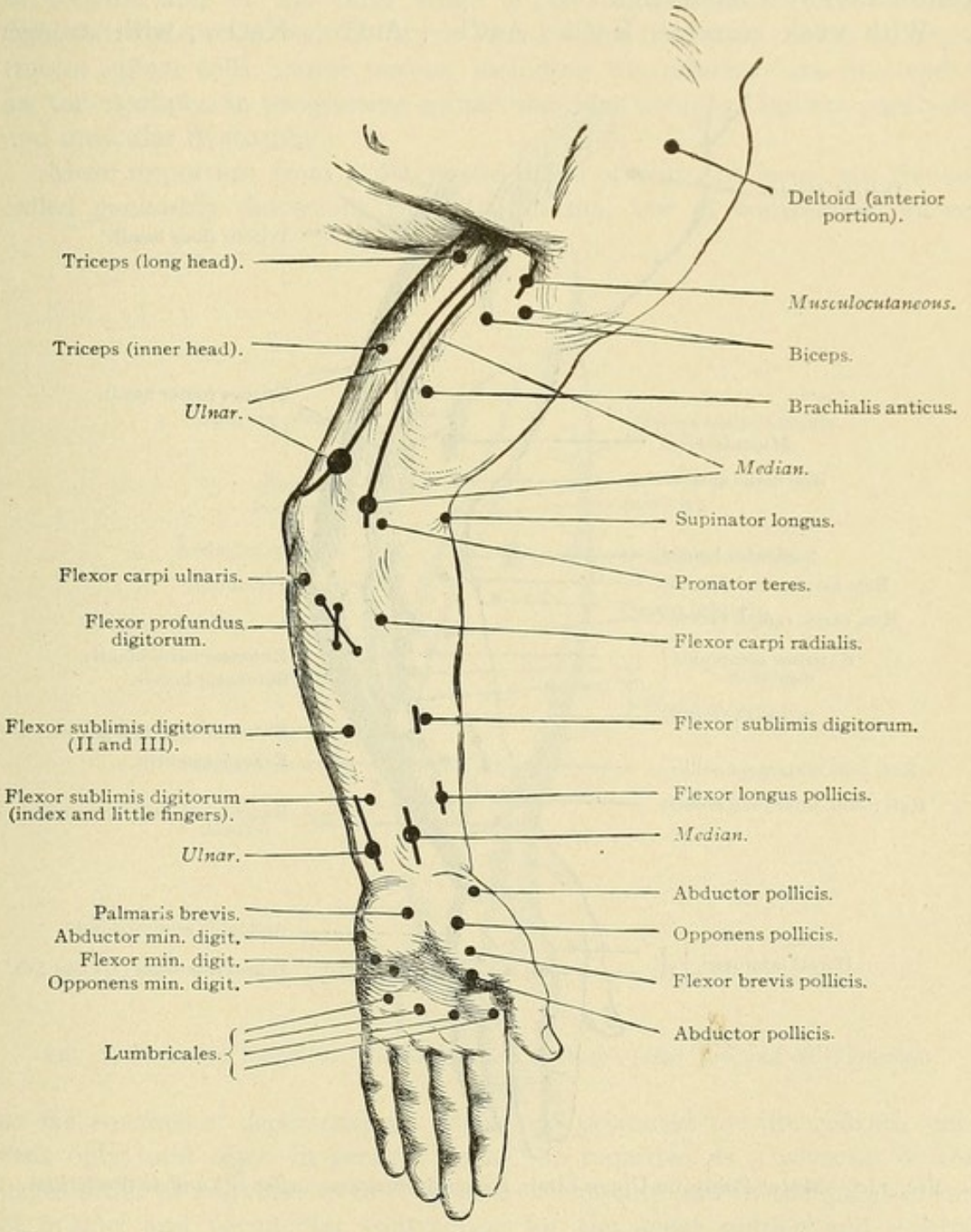


FIG. 139.—Motor Points on Upper Limb, Flexor Surface—(*after Erb and de Watteville*).

of response exists with galvanism. Thus, beginning with very weak currents, it is observed that contraction first takes place at the moment of that closure which makes the testing pole the kathode or negative pole—kathodal closure (KaCl). As the strength of the current is increased the kathodal closure contractions become stronger, and anodal closure (AnCl) contractions make their appearance. With still stronger currents

the anodal opening (AnO) contraction occurs, and, last of all, when the kathodal closure contractions become tetanic (Te), slight kathodal opening (KaO) contractions appear. *These facts are equally true of normal muscle and nerve and may be formulated.* Representing slight contraction by a small "c," decided contraction by a large "C," and the absence of contraction by a minus sign (−):

With weak currents, KaClc, AnCl—, AnO—, KaO—; with stronger

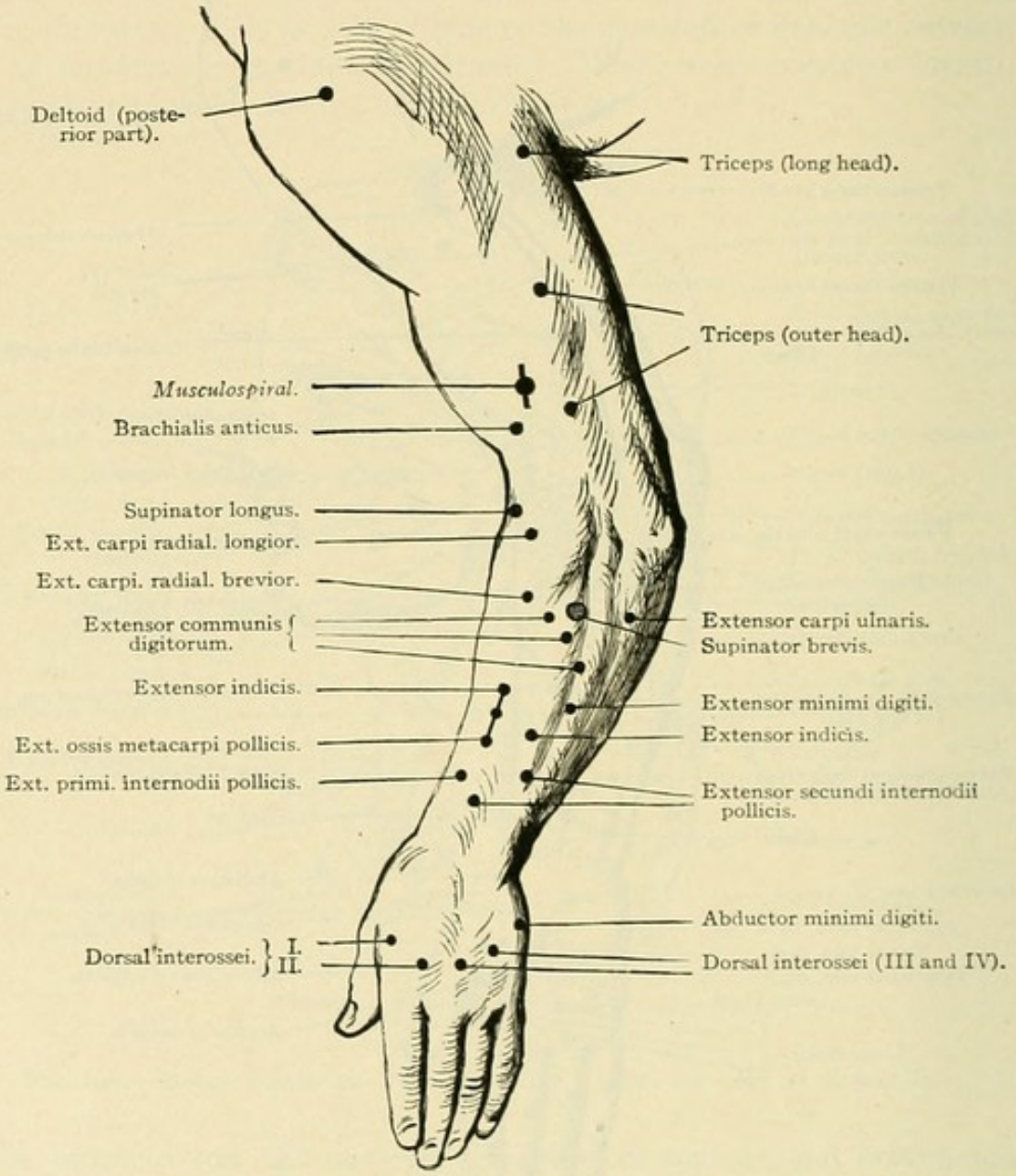


FIG. 140.—Motor Points on Upper Limb, Extensor Surface—(*after Erb and de Watterille*).

currents, KaClC, AnClc, AnO—, KaO—; with still stronger currents, KaClC, AnClC, AnOc, KaO—; with strongest currents, KaClTe, AnClC, AnOC, KaOc.

In *pathological states* two sets of deviations from the normal reaction to electrical stimulus are observed—viz., *quantitative* and *qualitative*.

In the *qualitative* deviations there is simply an increase or a diminution of the normal irritability of both nerve and muscle to either faradism or galvanism. These differences are, of course, most easily measured when

the alteration exists only on one side of the body, which may then be compared with the other. When both sides are affected, estimates can be made only by comparison with a healthy body or by the galvanometer. For this purpose superficial nerves, such as the facial, ulnar, and peroneal, are usually selected. *Instances:* Increased quantitative changes are found in tetanus and in the early stage of certain peripheral palsies, while diminished electrical excitability is found when the lower motor segments (motor spinal cells, motor nerves, including the muscles) are involved—as, for example, in progressive spinal muscular atrophy, bulbar paralysis, and muscular dystrophy.

More important from a diagnostic point of view, at least, are the so-called *qualitative* deviations from the normal law of contraction *known*

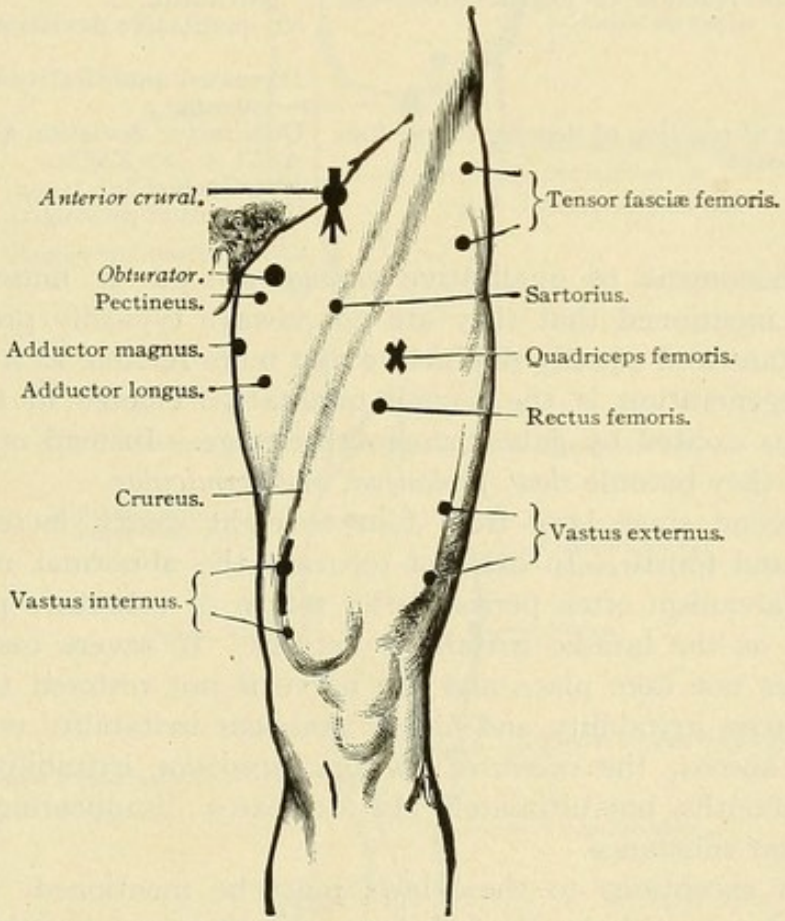


FIG. 141.—Motor Points on Thigh, Anterior Surface—(*after Erb and de Watteville*).

as the reaction of degeneration. These are produced by the *galvanic* current only, and may, in general terms, be regarded as a reversal of the usual order of response to interruption of currents and in the substitution of a slow and vermicular contraction for the usual sudden and jerking contraction. The entire group of events is best illustrated by describing the electrical phenomena which present themselves in an ordinary case of peripheral paralysis. In two or three days to a week after its appearance there begins a gradually diminishing response in the *nerve* to both faradic and galvanic currents. This goes on for one or two weeks, at the end of which time it disappears to both currents, even the strongest. During this same time the *muscle* is also losing its responsiveness to the faradic current, but not to the galvanic. There may be also at first a

slight diminution to the galvanic current, lasting, say, one week, and constituting the "first degree" or "first stage" of degeneration. But during the second week this is substituted by an increased excitability, so that there is now marked response to weak currents—incerased quantitative deviation. But there is also *qualitative* change. The anodic closure contractions become now as strong as or stronger than the kathodal closure contractions. Nay, more: the kathodal opening contractions, which in health were exceedingly weak and could be brought about only by the strongest currents, are now often stronger than the kathodal closure. This state of affairs for *muscle* may be represented thus:

MUSCLE CONTRACTION—REACTION OF DEGENERATION.

First stage of reaction of degeneration—one week:	Diminished quantitative response to galvanism. No qualitative deviation.
Second stage of reaction of degeneration—four to eight weeks:	Increased quantitative response to galvanism. Qualitative deviation as follows: AnCl = or > KaClc. KaOC > KaClc. Contraction prolonged and vermicular.

The phenomena of qualitative change are purely muscular, and it should be mentioned that they are not always typically present. Even more constant and equally distinctive and more reliable as a sign of reaction of degeneration is the second qualitative change in the muscular contractions excited by galvanism in this stage. Instead of being quick or sudden, they become *slow, prolonged, and vermicular.*

The second stage lasts from four to eight weeks, increasing during the third and fourth. In cases of recovery the abnormal muscle irritability to galvanism often persists after return of voluntary power, but it diminishes as the faradic irritability returns. In severe cases, when recovery does not take place and the nerve is not restored to its natural state, *all nerve* irritability and *faradic muscular* irritability remaining permanently absent, the *increased galvanic muscular* irritability may continue for months, but ultimately also decreases, disappearing finally with the muscular substance.

Certain exceptions to these laws must be mentioned. Thus, when the nerve lesion is slight, the fall in quantitative *nerve* irritability is sometimes preceded by a corresponding rise, or the rise may persist throughout and such rise may be considered as evidence of a slight lesion. Further, the change is not always the same to faradism and galvanism, and is often brought out much better by the slow interruptions in the faradic battery than by the rapid interruptions in the same or by the galvanic current. Gowers noticed in one instance moderate but prolonged diminution of faradic irritability when no change could be found with galvanism, and Bernhardt has noticed lessened irritability to faradism with distinct increase to galvanism in an ulnar nerve the seat of traumatic paralysis. Again, *faradic* irritability may not diminish to the same degree in the muscle as in the nerve in mild cases, and conduction of voluntary impulses from the brain may be possible when there is no response to electrical currents, and there may be response to electrical currents when there

is no conduction of voluntary impulses from the brain. In still milder peripheral paralyses there is no reaction of degeneration at all, whence a favorable prognosis may always be made. It is to be especially observed in recovery from nerve lesions that *voluntary* motion often returns decidedly earlier than the electrical excitability of peripheral nerves.

What do reactions of degeneration teach us? Simply that the disease is seated in the anterior cornua of the gray matter of the cord, or in the peripheral nerves. They teach us nothing as to the nature of the lesion. Upon the integrity of the cells in the anterior cornua and their "trophic

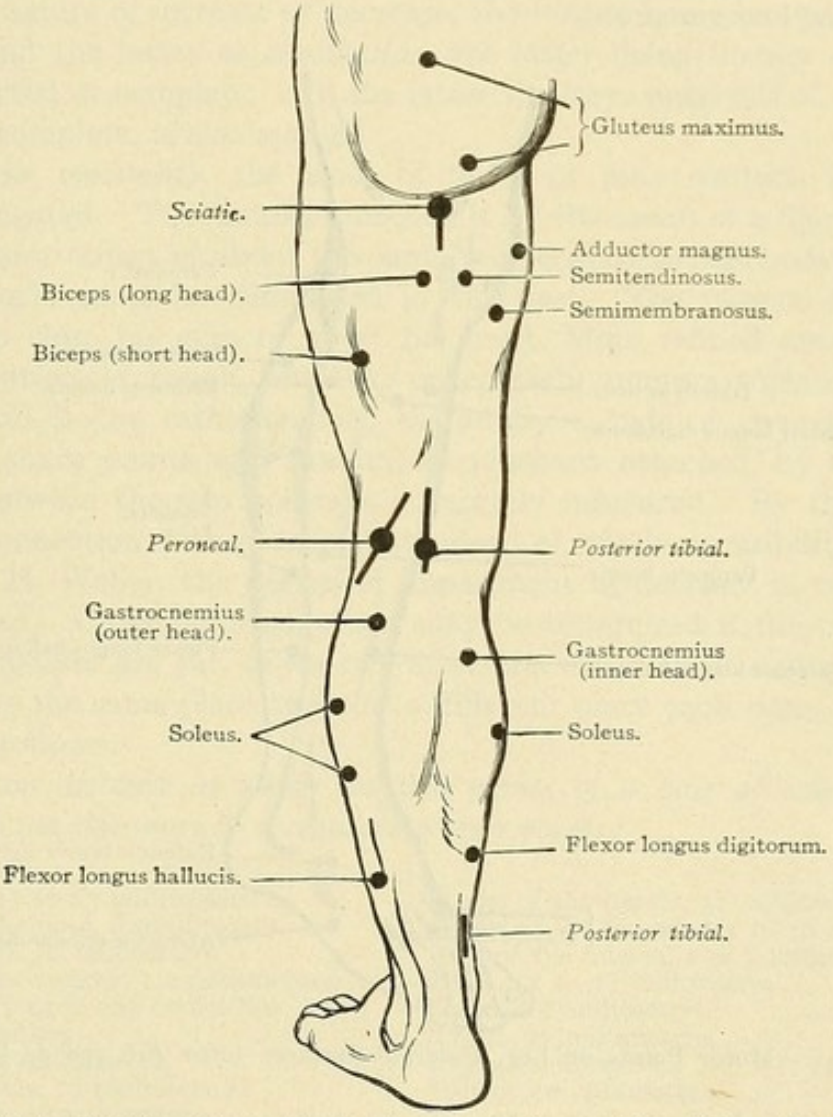


FIG. 142.—Motor Points on Lower Limb, Posterior Surface—(*after Erb and de Watteville*).

influence" depends the nutrition of the nerve and the muscle over which the cells preside. Hence with disease of the cornua result degeneration of the nerve and wasting of the muscle. The muscular fasciculi become reduced in size and ultimately totally disappear. This is associated with a certain amount of interstitial overgrowth. In the transition referred to, certain fasciculi assume the yellow, glassy appearance known as waxy degeneration. The *sensibility* of the muscle, if the sensory nerve is intact, becomes increased, and there may be pain, partly due to compression of the nerves by morbid contraction, and partly to a morbid sensitiveness of the nerve-endings and to the interstitial inflammation. The recovery

of the nerve is followed not only by gradual restoration of its power over the muscle, but also by restoration of the nutrition and development of the muscle. For this, however, much time is required, and it often remains permanently smaller than normal.

Lesions of motor nerves, whether inflammatory or traumatic, are followed by similar results—degenerative atrophy of nerve and muscle because of interference with the conduction of the trophic influence. Occasionally in cerebral palsies and in spinal paralyses in which the lesion is above these ganglion cells there is some wasting, but no reaction of degeneration is developed, because the nutrition is maintained by the intact cell body of the lower neuron.

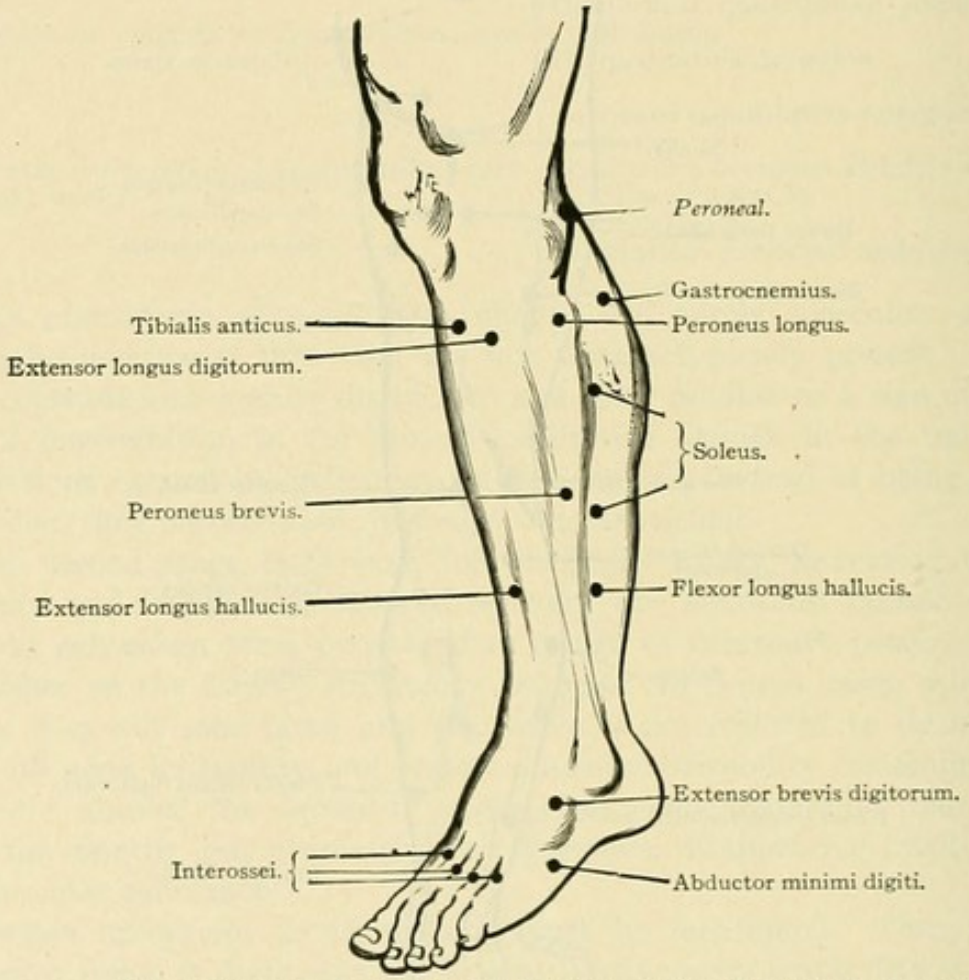


FIG. 143.—Motor Points on Leg, External Surface—(*after Erb and de Watteville*).

From the foregoing the diagnostic and prognostic value of the reaction of degeneration is at once apparent. The seat of the lesion, whatever its nature, is easily determined, in so far as it is within the cerebral or peripheral motor segments, but we may not be able to say whether the nerve-cells or their peripheral processes (the peripheral nerves) are diseased. We are also informed that recovery, though not impossible, must be delayed in proportion to the degree of degenerative reaction, because of the extensive repair necessitated in muscle and nerve. Much experience with the use of electricity should, however, be had before the physician permits himself to draw conclusions.

II. SENSORY PHENOMENA.—Under this head naturally fall first the *subjective sensations* of the patient. They include, strictly speaking, only

the various modifications of sensibility appreciable to him alone and independent to external impression—pre-eminently, *pain*. They also include those peculiar modifications due to internal irritation as contrasted with external impression, and known as *paresthesias*—viz., numbness, tingling, prickling, formication, or a feeling as of ants crawling over the skin; also a sensation like that of the contact of wool or fur—a furry feeling—vertigo, tinnitus aurium, or ringing in the ears, and a sense of unpleasant odors or tastes.

After these come modifications of the different varieties of cutaneous sensibility as excited by external impressions—*objective sensations*. They are of the nature of increase or decrease, the former being known as *hyperesthesias* and the latter as *anesthesias*, the latter being further characterized as partial or complete. To the latter the term paralysis of sensation, partial or complete, is also applied.

1. *Tactile sensibility*, the sense of touch or pure contact, is usually first investigated. The simplest method is by the touch of a finger, cotton wool, or blunt object of about the same temperature as the body, for both heat and cold must be eliminated in this test. The patient should be directed to close his eyes or avert his head. More refined measures are the application of rough, smooth, or coarsely uneven surfaces. More delicate still is the esthesiometer, essentially a pair of compasses with blunt and sharp points and graduated quadrant attached, by which the distance between the two points is accurately measured. By this instrument, in connection with a normal standard of relative sensibility worked out by E. H. Weber, the degree of impairment in delicacy of touch may be measured. Closer approximation may be recognized if the two points of the compasses are put down one after the other and varying the test by touching the same place twice or a different place each time. Weber's table is as follows:

Minimum distance at which the two points of a pair of compasses in contact with the skin may be recognized as two points:

Cheek, 11 to 15 millimeters.
Tip of the nose, 6 millimeters.
Forehead, 22 millimeters.
Tip of the tongue, 1.2 millimeters.
Back of tongue and on the lips, 4 or 5 millimeters.
Neck, 34 millimeters.
Upper arm, 77 millimeters.
Forearm, 40 millimeters.
Backs of the hands, 31 millimeters.
Backs of the fingers, 11 to 16 millimeters.
Tips of the fingers, 2 or 3 millimeters.
Back, 55 to 77 millimeters.
Chest, 45 millimeters.
Thigh, 77 millimeters.
Leg, 40 millimeters.
Instep, 40 millimeters.

These figures can, however, only be used within limits, as they are by no means constant for different individuals, or, indeed, for the same individual at different times. Marked deviations from them may, however, be accepted as indicating derangements of tactile sense.

2. *The sense of pain* is of equal importance to that of pure touch, because these two not infrequently fail to diminish or increase *pari passu* in morbid states. Parts insensible to touch may respond decidedly to painful impressions. Pain is most easily investigated by pricking with a pin or pinching a fold of skin, by painful electrical currents or painfully hot metals. The special term *analgesia* is applied to loss of sense of pain

while the tactile sense is preserved. Analgesia exists in peripheral and central nervous disease and may be observed especially in syringomyelia.

Tenderness or pain on pressure in the course of nerves should be studied in connection with the sense of pain. It is found in nerves which are the seat of inflammation, especially in sciatic neuritis and multiple neuritis.

3. *The sense of temperature* may be roughly tested by ascertaining the power of the patient to discriminate between the warm breath close to the skin and the cooler current produced by blowing from a distance. More precisely, the sense of temperature is studied by testing the ability to recognize differences in the temperature of flat-bottomed test-tubes filled with water of different temperatures and brought into contact with the skin. The therm-esthesiometer has been devised by Eulenburg for the same purpose, but the student is referred to works on nervous diseases for its description.

In health differences of 0.5° to 1° F. (0.27° to 0.55° C.) may be recognized on the fingers and face at temperatures from 80° to 100° F. (26° to 37° C.), while on the back differences to be recognized must amount to 2° F. (1° C.).

In disease we sometimes notice complete loss of sense of temperature, while the skin appreciates other forms of irritation, and, again, this state of affairs is precisely reversed; or the temperature and pain senses may be lost or impaired, while tactile sense is preserved, as in syringomyelia. This is known as dissociation of sensation. It occurs most commonly in syringomyelia, but has been seen in other diseases. Strümpell, has called attention to a peculiar reversal of the sense of temperature as the result of which cold objects appear warm. This has been noticed in various diseases.

4. *Sense of Locality.*—By this sense we know, without looking, what part of the body is being touched. While cutaneous sensibility may remain intact, the sense of locality may be seriously deranged. Thus, a patient may think he is being touched on the leg when the contact is with the foot.

5. *Delayed conduction of sensory impressions* represents a form of modified sensibility of which after-sensations are a further subdivision. In delayed conduction an irritation, more particularly a painful one, like the prick of a pin, is noticed by a patient after an appreciable interval, whereas in health the recognition is instantaneous so far as the unaided perception is able to judge. Touch and pain may even be thus separated, the immediate contact of the pin being promptly recognized, while the sense of pain presents itself a few seconds later. It is likely, also, that the sense of touch may be delayed.

6. *An after-sensation* is a prolonged sense of pain which succeeds a momentary impression. Such is the prolonged burning on the sole of the foot which sometimes succeeds the prick of a pin, or which may occur once or oftener after a short interval, as if additional pricks had been made. Occasionally an isolated prick of a pin is not perceived, and repeated pricks are necessary, the whole producing a painful sensation; this is known as summation of sensation.

These abnormal sensations occur particularly in diseases of the spinal cord or of the nerves, and especially in tabes dorsalis.

7. *The muscular sense*, it were better named the *sense of position* or of passive movement, is that sense by which we become aware of the position of any of our limbs without the aid of vision, as well as of any degree of motion by them. It is probable, however, that the sensibility of the articular surfaces, ligaments, tendons, and skin aids the sensibility of the muscles in furnishing this information, and it is better to call this sense the *sense of position* when we speak of it in reference to the position of the limbs, or *deep sensation*. This power is diminished in nervous diseases, and may be tested by having the patient first touch a certain object with his eyes open and asking him to repeat the act with the eyes closed; or by moving the fingers or toes of a patient and requesting him to give their positions when his eyes are closed and voluntary movements of the parts are restrained.

The "muscular sense" is not only thus estimated, but the strength required to lift a leg or an arm, more plainly evident when one is tired, is also measured through it. It is the muscular sense which causes the paretic to say that his leg feels heavy.

In tabes dorsalis, the muscular sense may be defective. It is also found defective in diseases of the peripheral nerves and in diseases of the lemniscus, or of the internal capsule, or of the nerve fibers passing to the cortex behind the fissure of Rolando.

The muscular sense is also estimated by the amount required to be added to an existing weight on the skin before the addition is appreciated. Thus it has been ascertained that in health an addition of 1/20 or 1/30 to an existing weight can be appreciated. Thus, if a weight of 95 gm. be placed on the skin, an addition of a single gram will not be recognized, but nearly five gm. must be added before the increase is appreciated, while if considerably more than this is necessary, it means that the sense of pressure is less delicate. Sufficiently accurate measures are coins of different weights. Temperature must be eliminated by placing non-conducting substances between the weight and the skin, while the part to be tested must also be supported.

It is not unusual to find, in paralysis of the muscular sense, failure to recognize a doubling and even tripling of weights. It is more especially in tabes dorsalis that such paralyses are found while the tactile sense proper is intact, a light touch of the skin being felt, while a considerable pressure is not appreciated.

Astereognosis is the inability to recognize objects, their nature and uses, by touch, as the result of cerebral disease, but not because of any affection of the peripheral nerves or spinal cord. Most frequently it occurs from lesions of the parietal lobe. The attempt has been made to limit the term, stereognosis to the recognition of the form and characteristics of an object, while symbolia is employed for the recognition of the object in regard to its uses. This distinction is likely to cause confusion.[1]

8. *Anesthesia* is said to be peripheral, spinal, or cerebral, in accordance with the seat of the broken conduction between the terminal ap-

[1] See Burr, "American Journal of the Medical Sciences," March 1901. "Therapeutic Gazette," Feb. 15, 1904.

paratus and the cerebral cortex. *Peripheral* anesthesia occurs after chilling of the skin through the action of ether, from cocain, aconite, veratrum, as well as corrosive agents like acids, alkalies, and carbolic acid. Spasm of the small vessels, forming the so-called spastic anemias, is also attended by anesthesia. The anesthesias of washer-women, who have their hands in water all day long, may belong to this class. Lesions of nerve trunks by pressure, inflammation, and degeneration may cause anesthesia. The *paresthesias* referred to—numbness, formication, and tingling—are among the effects of such lesions. *Spinal* anesthesias are found, especially in connection with disease of the posterior roots, posterior columns, and posterior cornua of the cord. Such a disease is tabes dorsalis especially. Anesthesia is found, however, also in myelitis, acute and chronic, and when there is pressure on the cord from hemorrhage into the spinal canal or pressure by diseased or broken vertebræ or from tumors. Such anesthesia is usually bilateral and is known as paraanesthesia. *Cerebral* anesthesia occurs as the result of hemorrhages, softening, or tumors, which impinge on the posterior limb of the internal capsule, through which the sensory fibers, probably after interruption in the thalamus, pass upward to the cerebral cortex. If the cerebral anesthesia affects half of the body, it is known as hemianesthesia, and the half of the body affected is opposite the hemisphere of the brain in which the lesion lies, since the sensory fibers also decussate, many of them, soon after their entrance into the cord through the posterior roots.

The hysterical anesthesias, and anesthesias due to the narcotics and anesthetics, are regarded as central in their origin. The hysterical hemianesthesia is much commoner than the organic cerebral hemianesthesia.

III. Vasomotor and Trophic Phenomena.—We pass next to the study of *vasomotor* and *trophic alterations*. Two sets of vasomotor nerves have been demonstrated by physiologists—the vasoconstrictors and vasodilators—the former contracting the arteries when stimulated and permitting their dilatation when paralyzed. The vasodilators are influenced in an opposite manner by the same agencies, but their number, so far as proved, is not great, as they include up to the present time only fibers in the chorda tympani, nervi erigentes, and sciatic nerve. Blushing may be the result of stimulation of vasodilators. Moreover, pathology has as yet failed to separate lesions of the two sets of nerves and their effects and vasomotor phenomena are generally regarded as results of a *paralysis* or of an *irritation* of vasoconstrictors. Instances of the former are redness, a feeling of warmth, and sometimes an actual elevation of temperature, sweating, all in circumscribed areas or half the body. They may persist or intermit. Instances of vasomotor irritation are pallor, coldness, accompanied by stiffness, formication, and even pain. These are phenomena of vasomotor spasm. A more or less permanent condition of the hands sometimes results, characterized by a blueness or mottled appearance accompanied by a lowered temperature further augmented by external cold. Still higher degrees are said to have produced circumscribed gangrene (Raynaud's disease).

Symptoms of vasomotor *paralysis* occur in connection with cerebral and spinal lesions and with injuries of the sympathetic system and nerve

trunks containing vasomotor fibers. The essential causes of vasomotor *spasm* are less easy to locate. It is found associated with prolonged convulsive seizures, and in angina pectoris at the beginning of the attack, as if caused by irritation of the sympathetic ganglia in the heart.

That *trophic* or nutritive phenomena are closely allied to vasomotor phenomena is commonly admitted. That they are under the control of the same nerves is doubtful, although the proof of the existence of separate trophic nerves is still wanting. Vesicular eruptions in the area of distribution of nerves, such as herpes zoster, certain atrophic skin diseases, pigmentations and depigmentations, such as morphea, Addison's disease and vitiligo, scleroderma, and the glossy skin which succeeds certain injuries to nerve trunks are illustrations of trophic influences. Similar are the changes in the skin, hair, and nails, as the result of which the first becomes dry, the second is lost or becomes rapidly gray, and the last grow brittle, thicken, or drop off. The latter events occur in connection with spinal and even cerebral lesions. The circumscribed edema known as acute angioneurotic edema and the more permanent condition of myxedema are also probably trophic. So, also, are the atrophies which result from disease of the cells of the anterior horns of the gray matter of the cord, or from injuries to nerves by which they are essentially cut off from the trophic cells; also unilateral facial atrophy including even atrophy of bone, and the still more remarkable spinal *arthropathies* of Charcot, as the result of which the joints enlarge or become the seat of effusions.

Finally, there is the acute bed-sore or eschar, so well described by Charcot,[1] beginning in an erythematous patch on which bullæ and blebs are rapidly developed, quickly succeeded by gangrene. While pressure or irritation may be necessary to the production of these sores as exciting causes, they are more easily invited in spinal paralyses than in non-paralytic conditions. Such results follow cerebral lesions and lesions in the medulla oblongata, spinal cord, and sympathetic nerves.

It is well known that the vasomotor nerves surrounding the various blood-vessels are derived from the sympathetic trunks, which, in turn, receive their vasomotor filaments from the roots of the spinal nerves.

IV. Mental Phenomena.—Under this head come the phenomena of consciousness or unconsciousness, coma, the state of the will, the various perversions of mental processes, including delirium, hallucinations, delusions, illusions, and insane acts. *Hallucinations* are deceptions of the special senses which appear to the individual as real. They have no external cause. The victim of delirium tremens who imagines that he is pursued by monsters of various sorts is the subject of hallucination. A *delusion* is a false belief which cannot be corrected by argument or experience. The deluded person imagines that he is the happy possessor of millions when he is actually a pauper, or complains of poverty although affluent. An *illusion* is based upon an actual perception, but an erroneous impression arises therefrom. In a hallucination no object is actually seen; there is no sensory impression. The idea of relief obtained on looking at a picture in the stereoscope is an illusion.

[1] "Lectures on Diseases of the Nervous System," Philadelphia, 1879.

Delirium is the more or less acute manifestation of one or all these perversions of mental process, associated with muttering or active speech suggested by them or with action growing out of them. Thus constituted, delirium may be the result of toxic states or acute disease other than of the brain.

The same preversions of mental process continued and unaccompanied by fever constitute *insanity*, which is probably always associated with structural change in the brain or its membranes, although such may not always be demonstrable. Other symptoms are added, however, in insanity, such as extreme depression of spirits, while hallucination, delusion, and illusion may be present in various degrees. Special insane acts should be specified and modifications of normal sleep noticed.

V. Alterations in Vision and Hearing.—In addition to the ordinary defects of vision, the *response of the iris to light* should be noticed; also its *accommodating power*. The former is absent in three-fourths of all cases of tabes dorsalis while the latter remains. The iris thus failing to respond to light, but retaining its accommodation to change of distance, is known as the Argyll Robertson pupil. Each eye should be tested separately, the other being covered. Finally, the eye-ground should be examined in every exhaustive study of a nervous case.

Modifications in *hearing* are the nature of increased and diminished intensity, and there is that very common symptom known as *tinnitus aurium*, or ringing in the ears, already alluded to as a good instance of a subjective symptom. Hyperacusis occurs in association with augmented acuteness of the other senses in acute affections of the brain or when there is hyperemia of the brain from any cause. It is also often complained of in hysteria. Deafness, on the other hand, is more frequently the consequence of disease of the ear itself. Ringing in the ears occurs in many conditions. Some more than usual impression on the acoustic nerve is the cause of tinnitus. In addition to the numerous forms of irritation due to ear disease, the blood in an adjacent vessel may be thrown into vibration and produce an audible murmur. On the other hand, tinnitus is sometimes due to intracranial irritation either of the nerve or of the auditory centers.

VI. Alterations in Breathing and Pulse.—*Alterations of breathing* are very common in nervous diseases. Respiration may be rapid or slow, and labored and sighing, or irrgeular, but especially peculiar is the *Cheyne-Stokes breathing*, in which, succeeding a long pause, so long sometimes that it seems as though the patient would never breathe again, follows gentle and shallow respiration, which gradually grows deeper and more frequent until an acme of dyspnea breathing is reached, when it again gradually diminishes in depth and frequency until the pause again occurs. It is an arrhythmical breathing of a periodic type. During the pause the pupil often contracts and the heart's action becomes less frequent. Cheyne-Stokes breathing may occur in various conditions of the brain, in which the respiratory center is influenced. The period of arrest varies from five to forty seconds, and the duration of each cycle may be from 15 to 75 seconds, and may vary.

A modification of Cheyne-Stokes breathing is a form in which there are periods of deep and energetic breathing which begin suddenly, and in which

the respirations gradually become shallower until they cease, and after a pause energetically recommence.

The *pulse* is influenced chiefly by diseases of the cranial contents, especially of the medulla oblongata, the cerebrum, and the meninges. It is at times very slow, as in meningitis and apoplexy, or when there is intracranial pressure from any cause or when there is pneumogastric irritation. It may be accelerated when there is inflammatory pyrexia or irritation of the cardiac center. Again, it may be irregular, acting through the nervous system, of which opium poisoning is among the familiar causes; uremia is another cause, rarely also is influenza.

VII. Focal Disease and Focal Symptoms.—The terms focal disease and focal symptoms will be often used in the following pages. By *focal disease* in the nervous system is meant a circumscribed lesion, no reference being made to the nature of the lesion as to whether it is softening, a tumor, or clot. By focal symptoms is meant symptoms caused by a lesion in one spot whether in the brain or spinal cord. Cerebral syphilis is a diffuse process, therefore usually not a focal disease. The *general* symptoms of a tumor in the motor cortex are headache, vertigo, choked disk, etc., the *focal* symptoms are paralysis of one or both limbs on the opposite side of the body, convulsions confined to these limbs or groups of muscles. Focal symptoms are localizing symptoms, or symptoms that indicate the focus of the disease or the region affected.

AFFECTIONS OF THE PERIPHERAL NERVES.

NEURITIS.

Definition.—Neuritis, or inflammation of a nerve, may be confined to a single trunk, whence it is called *localized;* or it may involve a large number of nerves, when it is known as *multiple* neuritis or *polyneuritis*.

Etiology.—Local neuritis. Exposure to cold is a frequent cause of neuritis, and the nerve most frequently affected is the facial. Trauma is another cause, including compression, contusions, or cuts, as with glass, or stretching and laceration such as occur when there are dislocation, fracture, and other violent injuries. Neuritis may also occur as the result of extension of inflammation from contiguous parts, as from caries in a bone through which the nerve passes, adjacent joint inflammation, pleurisy, and meningitis.

The causes of *multiple neuritis* are numerous, and by no means easy of classification. They include:

1. The commonly acknowledged poisons introduced from without: (*a*) Organic, including alcohol, by far the most frequent cause, ergot, morphin, ether, carbon monoxid, carbon bisulphid, benzine and its products, and anilin; (*b*) inorganic, including lead, arsenic, phosphorus, and mercury.

2. Endogenous toxins generated in the organism by chemical changes. Such is the cause of the neuritis of diabetes mellitus, whether glucose, oxybutyric acid, diacetic acid, or acetone, all of which are found in the blood in that disease.

3. *Toxins* inherent to the infectious diseases. Instances are malarial neuritis, leprous neuritis, beri-beri or so-called endemic neuritis, also,

probably, the neuritis of acute infectious jaundice (Weil's disease). Of neuritis also due to toxic products of pathogenic bacteria are diphtheritic neuritis, septicemic neuritis, the neuritis of smallpox, typhoid fever, tuberculosis, and possibly syphilis are instances.

4. Intrinsic states of the blood of undetermined nature, with which cold may or may not co-operate as an exciting cause—viz., rheumatism, gout; also the puerperal state, and chorea. Malnutrition, such as characterizes cachectic and senile states, cancer, tuberculosis, and wasting diseases generally are also causes. It is not impossible that cold alone may, by its operation, generate a poison capable of producing a polyneuritis, but more probably it acts by lowering the vitality of the nerves and rendering them liable to attacks from other agents.

Age and Sex.—Multiple neuritis is a disease of adults. Diphtheritic neuritis is the most common form observed in children.
The alcoholic form is more frequent than all others put together. More than one cause may co-operate, when one may be the predisposing and the other the exciting. Cold probably most frequently plays the latter rôle, but there may be others, such as anemia, and the like.

Morbid Anatomy.—An inflamed nerve is reddish, from hyperemia of the *vasa nervorum*, though the stage of demonstrable hyperemia may have passed away when the nerve comes under observation. In *perineuritis* and *interstitial neuritis* the primary change is in the connective tissue—in the former, an infiltration of the nerve sheath with leukocytes, and in the latter, of the interstitial tissue with the same cells. There may even be minute extravasations of blood. These changes are more likely to occur in places along the course of the nerve where it is exposed to special irritation, as in passing through foramina or over bone. The lymphoid cells gradually become fusiform cells, resulting in the formation of true connective tissue. The pressure of this new tissue gradually destroys the nerve itself, the medullary sheath being gradually broken up into drops, which subsequently disappear, while the nuclei of the sheath of Schwann increase; finally, the axis-cylinder also becomes granular and disappears—all this in varying degrees. The nerve fiber may be substituted by a fiber of connective tissue, in which there may be a deposit of fat, a condition seen in the lipomatous neuritis of Leyden.

In *parenchymatous neuritis* the primary change is in the nerve fiber itself. Here the medullary sheath and the axis-cylinder are the first involved, the former breaking up into drops, as described, and the latter into granules, both ultimately disappearing, while the interstitial connective tissue remains comparatively unchanged; but the nuclei of the sheath of Schwann proliferate and become a part of the interstitial connective tissue.

The muscles connected with the inflamed nerve also atrophy—in the case of the motor nerves, at least—being practically cut off from their center of nutrition. The change in the nerve is essentially the Wallerian change noticed in the nerve fiber of a cut nerve. In some instances the changes noticed in the sheath of Schwann extend over into the interstitial tissue of the muscle.

Symptoms.—Localized neuritis. There is not much constitutional disturbance in localized neuritis, though the thermometer may show some rise

of temperature. *Pain*, especially pain on motion, and tenderness, are the salient symptoms. The pain may be confined to the seat of the inflammation or may involve the distribution of the nerve, or the whole limb may be involved. It varies in degree and also in character, being sometimes burning and at other times aching, boring, or shooting. It is likely to be worse at night, and when in situations involving pressure on the nerve itself. The nerve may be swollen appreciably, and rarely the skin over it is reddened.

The pain in the trunk of an inflamed nerve is probably due to pressure on the *nervi nervorum*. Weir Mitchell has especially called attention to this. An interesting fact is that the nerves composed almost purely of motor fibers are less tender than sensory nerves. This would imply that fewer sensory nerves are distributed to the motor nerve trunks than to sensory nerves, or that some pain is felt by the sensory fibers which make up the inflamed trunk.

Mitchell also describes elevation of surface temperature and trophic disturbances, such as sweating, herpes, and effusion into neighboring joints. Other trophic derangements, including muscle wasting, associated with peculiar "glossy skin" or slight edema, may be present. Vesicles, bullæ, and herpetic eruptions may occur. The nails become brittle, rough, and marked with transverse ridges. The bones in the fingers may even become atrophied. There may be thickening of the skin and a condition resembling ichthyosis may be present. Ultimately the hyperesthesia and paresthesia may become anesthesia, though usually limited to small areas.

Motor disturbances, including twitchings and contractions, may be present.

The electrical condition of the nerves and muscles must be studied. It may be normal in slight cases. In more severe cases there may be the reaction of degeneration, with the slow, lazy contraction of the muscles, and the reversed reaction to opening and closing currents, described on page 850.

The course of the disease is variously prolonged. Many acute cases terminate favorably in a few weeks. More cases become chronic, extending over months and even years, after which they may gradually subside.

A rare variety is "ascending neuritis," in which the inflammation extends from smaller to larger branches, until finally most of the nerves of a limb may be involved, or possibly even the spinal cord, producing myelitis, with or without spinal meningitis. Paralysis may result from such a condition. This is possibly the rare form of paralysis that succeeds visceral disease, as that of the bladder. Even the corresponding nerves of the other side may be involved. It is the opinion of some of the best neuropathologists that this ascending neuritis occurs only from a suppurating wound. The theory of an ascending neuritis is not universally accepted.

The symptoms of multiple neuritis are easily divided into three classes: *Motor* weakness, *sensory* derangement, and inco-ordination. The first is the result of the involvement of motor nerves, and manifests itself usually first in the extensors of the wrist and fingers, flexors of the ankle, and extensors of the toes. The sensory disturbances are tingling, numbness, and pain, while the inco-ordination resembles that of tabes. According as one

or the other of these sets of symptoms predominates we have a motor form, a sensory form, or an ataxic form.

The onset may be rapid or slow. In the form, due to cold and exposure, it is usually sudden, with chill and fever and a temperature of 103° or 104° F. (39.5° to 40° C.), headache, and backache. The slow onset is characteristic of alcoholic neuritis, though it may be precipitated by some exciting cause, as cold, exposure, fatigue, or some other toxic state. Neuritis of slow onset is rarely febrile. In the initial stage *sensory symptoms* are numbness and tingling of the fingers and toes, palms of the hands and soles of the feet, and other parts of the lower arms and legs; then hyperesthesia, tenderness, and pain, more marked in the legs, sometimes associated with cramp in the calves. These symptoms may in mild degree precede the onset as premonitory for weeks and for months, especially in the alcoholic form.

Very characteristic is the *tenderness of the muscles* themselves, developed as they become weaker, and elicited by grasping them, the slightest pressure often causing the patient to cry out with pain. This is regarded as evidence that all the nerves of the muscles are involved, the sensory as well as the motor. The nerve trunks are also tender, although this tenderness is less marked than in simple neuritis, because the contrast with the hyperesthesia of the surrounding skin is less conspicuous.

The *motor symptoms*, seldom absent, soon follow the sensory phenomena just mentioned. They include palsy or inco-ordination or both in upper and lower limbs, but with this characteristic—that the involvement of the limbs is symmetrical and the distal extremities, as the feet and hands, are affected, the former more frequently. Motor symptoms may exist in the feet and sensory symptoms in the hands, the latter commonly preceding.

The muscles commonly involved are those supplied by the peroneal nerve in the lower, and by the posterior interosseous branch of the musculospiral in the upper extremity. With weakness in the legs comes *loss of knee-jerk* and *ankle-jerk*, quite frequently, but not invariably, depending, of course, on the involvement of the nerves forming these reflex arcs. The muscles above the knee are less frequently affected, and still less frequently those which move the hip-joint.

The paralysis of the muscles innervated by the peroneal nerve gives rise to a peculiar and distinctive walk known as the *steppage* gait, and occasionally it is unilateral, when only one peroneal nerve is affected. It is the gait of polyneuritis in which the foot drops, and in order to raise it from the ground and thereby to "shorten" the limb, the thigh is drawn up unnecessarily high and the knee is flexed excessively so that the gait resembles that of the "high-stepping" horse. The extremity of the foot strikes the ground first, followed by the heel, so that there is often a recognized interval of time between the two events. Closing of the eyes does not affect this gait. Occasionally the anterior tibial muscle may escape when the other muscles of the peroneal distribution are paralyzed.

As contrasted with the *diminished tendon reflexes*, the reflex action from the skin may be increased, especially when there is hyperesthesia, even when there is considerable motor paralysis, the movement being caused by the muscles which escape involvement. In severe cases, on the other hand, when there is much loss of sensation and motion, the skin reflex is absent;

exceptionally, it may be absent when sensation is perfect. Myotatic irritability is almost always lost, although in the early stages of the disease, or in those cases in which the anterior crural nerves escape, it may be preserved.

In the arms it is the extensors of the wrist and fingers which are first affected, and these symmetrically, illustrated by one of the best recognized toxic forms of neuritis, lead palsy. In the latter there is paralysis of the extensors while the extensor of the metacarpal bone of the thumb and the supinator longus usually escape, although in some cases of lead palsy these muscles are affected. After the extensors, the flexors of the wrist and fingers are involved, then the interosseous muscles, and, finally, the thenar and hypothenar muscles are attacked, always to a less degree than the extensors. The muscles above the elbow are less affected.

Occasionally the fibers of the *pneumogastric* are involved, causing frequent pulse-rate and paralysis of the vocal cords, cardiac failure, and death. Still more rarely the diaphragm and muscles of the thorax and abdomen are involved. The facial and motor oculi nerves are possible seats. Neuritis confined to the cranial nerves has been described. The sphincters are also rarely affected.

The muscles exhibit the *reaction of degeneration*, faradic irritability being lost, while galvanic irritability may be increased, but is not always altered in quality. In the nerves, irritability to both currents diminishes and ultimately disappears, although in the very first stage there may be increased galvanic irritability. In severe cases total loss of excitability may occur at once because of a corresponding destruction of muscular substance, instead of being preceded by an intermediate state of increased excitability.

Wasting of the muscles is sooner or later inevitable, unless the disease is of short duration, although it may be obscured by a temporary œdema or a condition of fatty infiltration, in which the fat accumulates between the wasting fasciculi, keeping up for a time the bulk of the muscle. The less affected muscles are likely to undergo shortening and contracture because of maintaining so long a fixed position, either from being given over to gravitation or as a result, of an effort to relieve pain. This alteration occurs most frequently in the lower extremity, contributing to intensify the "foot-drop" at the ankle, and more rarely to produce flexure at the knee-joint and to a less degree even at the hip, both of the latter being the result of posture. The foot-drop may be increased by the pressure of the bed-clothes upon the foot.

The sensory and motor phenomena are commonly associated *pari passu*, the latter extending from the hands and feet up the outside of the arm and leg. Very rarely either set of symptoms may occur alone.

Tremor is a marked symptom in some alcoholic cases and may precede loss of power.

Ataxic phenomena are usually associated with the sensory and motor symptoms. They are manifested by difficulty in balancing while standing, or by inability to execute finer movements with the fingers. Indeed, these may be the first symptoms, and may lead when studied to the knowledge of some defect in extending the wrist and fingers, or in raising the toes, or foot, from the ground while walking. The ataxia is more marked in the

lower extremities, and is believed to depend chiefly upon sensory nerve involvement, since these nerves are supposed to have most to do with co-ordination. Involvement of the motor nerves may possibly also cause ataxia. Because of the associated absence of the knee-jerk, the term peripheral pseudo-tabes has been applied to the ataxic variety. The symptoms may closely resemble those of tabes, but the phenomena always fall short of those of true tabes. It may be said, too, of the ataxic form that the sensory disturbances are sometimes less severe than in other typical cases. Absence of the Argyll-Robertson pupil and of vesical disturbance, rapid development of the disease, a history of the case suggesting a cause for neuritis, and, finally, recovery, are diagnostic points in favor of the ataxic form of neuritis as distinguished from tabes.

Trophic changes may occur in prolonged cases, including mainly glossy skin, arthritic adhesions, and thickening; also vasomotor derangement, shown by edema, especially about the ankles and the dorsum of the foot; also pallor of the fingers and changes in the nails and hair.

Mental symptoms are found more particularly in connection with the alcoholic form of neuritis. Besides irritability and general ill temper, more active symptoms are at times present. Hysteria and skilful duplicity in obtaining alcohol are characteristic. The phenomena may be those of delirium tremens or simple hallucination with extravagant ideas. Especially peculiar is the condition described by Wilks, and especially by Korssakow, in which there is a loss of appreciation of time and place, the patient describing with minute detail impossible journeys recently taken and persons whom he imagines he has seen. Convulsions and optic neuritis are rarely present; if present, they are probably due to meningeal inflammation. A simple mild delirium may occur in toxemic cases from the action of the poisons on the brain cells. Mental symptoms are not usually present in multiple neuritis from other causes.

The number and variety of the symptoms vary greatly in different forms, being most widespread in those cases due to alcoholism, to cold, or to combined causes, and limited in the cases due to metallic poisons, as lead. The more acute the case, the more widespread are the symptoms.

Diagnosis.—Localized neuritis. The disease is chiefly to be differentiated from *neuralgia*. This depends upon pain and tenderness in the course of the nerve and upon the limitation of the symptoms to its distribution. Neuralgia is more intermittent, and is relieved rather than aggravated by pressure. The presence of the paresthesia points to neuritis and the diagnosis is confirmed if there is ultimately lessened sensibility. In neuralgia, nerve and muscle reactions remain normal. It is possible, however, that neuralgia may result in neuritis. The distal pain of *central spinal disease* must be differentiated. In brachial neuritis the pain may radiate to the left side, suggesting *angina pectoris*, and there may even be a tendency to cardiac distress, but there is no tenderness in the course of the nerves in angina.

Special Variety of Localized Neuritis—Sciatica.

Definition.—This term is applied to all painful affections in the distribution of the sciatic nerve, some of which may be neuralgic, but the vast

majority are inflammatory and perineuritic, as it is the sheath of the nerve that is usually involved.

Etiology.—Sciatica is far more common in men than in women, in the ratio of about four to one, while brachial neuritis affects both sexes about equally. It is also a disease of adults, being unknown in children and very rare in the second decade. It is most frequent between forty and fifty, next between fifty and sixty, and next between thirty and forty.

Very rarely syphilis may be a predisposing cause. Exposure to cold is the most frequent exciting cause, especially after severe muscular exertion; while standing in water, sitting or lying on the cold ground, and the like are frequent causes. Pressure by mechanical agents and possibly muscular contraction may be a cause; also pressue by tumors and other new formations within the pelvis. In bilateral sciatica the possibility of intrapelvic tumor should be carefully considered. In addition to the intrapelvic causes referred to, secondary sciaticas may be caused by bone disease and other foci of suppuration external to the pelvis. A commonly overlooked cause is a movable sacroiliac joint.

Symptoms.—The leading symptom, is, of course, *pain in the course of the nerve*. Felt first in the back of the thigh, it also travels above the hip-joint, into the sciatic notch, behind the knee, below the head of the fibula, behind the *internal* malleolus, and on the dorsum of the foot. It may be more diffuse, but the course of the main trunk of the nerve is often indicated by it, and the points previously named, especially the back of the middle of the thigh and the sciatic notch, will often be pointed out by the patient as seats of special tenderness. It usually begins gradually, but it may start suddenly, especially in cases of rheumatic origin. Motion, particularly in walking, and positions in which the nerve is put in a state of tension or is compressed, aggravate it. A valuable sign of sciatica is pain produced by passive flexion of the thigh upon the pelvis with the knee extended (Lasègue's sign); by this means the sciatic nerve is stretched, and pain is readily produced if the nerve is inflamed. The characteristics of the pain are those already described under neuritis. The other more unusual symptoms of neuritis may also be present, as herpes, edema, and wasting, but the reaction of degeneration is seldom present. The loss of the *tendo Achillis* jerk is an important sign.

Diagnosis.—This is not difficult, although a careful study should be made of each case with a view to determining its primary or secondary origin. *Pelvic tumors*, especially in women, and *rectal accumulations* should be sought for. *Lumbago*, *hip-disease*, and *sacroiliac disease* are all to be recalled. Pain felt only in the outer side of the thigh is not sciatica. Some writers attribute all sciatica to joint diseases, but this view is not tenable. The rare cases of *sciatic neuralgia* are not characterized by tenderness. They occur in persons subject to neuralgia, and the pain is not influenced by position and motion, but is purely spontaneous. *Disease of the vertebræ*, of the *cauda equina*, and even of *the spinal cord* may produce sciatic pain; but here, again, tenderness is not so common in the course of the nerve, the pain is more likely to be bilateral, and changes in objective sensation may be distinct. Inflammation of the roots of the sciatic nerve, however, may extend downward. Bilateral pain may be indicative of disease of the nerve

roots, although bilateral sciatica from other causes does occasionally occur. The shooting pains of *tabes dorsalis* are like those of sciatica, but the other symptoms of the former disease are present.

Prognosis.—Cases of sciatica, however obstinate, usually sooner or later get well, although they may persist for months. A case came under Tyson's observation which lasted seven years, but recovery finally was complete.

Treatment.—Here, as elsewhere, if a cause is discoverable, it should be removed. Exposure to cold and dampness should be avoided, pressure by cicatricial tissue or dislocated bones should be relieved, and constitutional states favoring neuritis, such as gout and syphilis, should be corrected.

Of curative measures, rest is the most important. When a limb can be splinted, this should be done, pressure by muscular contraction being thus prevented. Fixation of the hip-joint by a plaster cast may be of great service and permit the patient to get about. Cold may be a useful application, as by an ice bag. In other instances heat, now dry and again moist, subserves a useful purpose. A blister or blisters may be applied over the tender nerve. Especially convenient is the Paquelin cautery, which should be used earlier than it commonly is; its application takes but a second, and may be rendered painless by previously applying, for a few minutes, a mixture of ice and salt to the spot to be burned, although this has been largely superseded of late by the more convenient ethyl chlorid. Morphin is sometimes indispensable, and the hypodermic method of application is best—1/6 to 1/4 grain (0.011 to 0.0165 gm.) for an adult. But the morphin habit is easily acquired, and the patient should not be allowed to use the syringe himself. Cocain may be similarly used—1/10 to 1/3 grain (0.0066 to 0.022 gm.)—and Gowers recommends it highly, more particularly for its power in arresting local transmission of the impulses that cause pain. Eucain is even better. Here, too, the injection should be made at the seat of the pain by the physician or a trusted attendant. Gowers, whose large experience always bespeaks respect, considers mercury also a most efficient agent, in the shape of a blue pill, 1 grain (0.066 gm.) once or twice a day, associated, if necessary, with morphin, the constipating effect of which it counteracts. Salicylate of sodium and aspirin are undoubtedly sometimes useful, as is also more rarely iodid of potassium. Strychnin is also of service. Injection of a considerable amount of normal salt solution into the buttock near the seat of pain has been recommended.

In the chronic form also Paquelin's cautery should be repeatedly used, or if not at hand, blistering may be substituted. Electricity here comes into play, and galvanism is the form to be used, the positive electrode being placed over the nerve or seat of pain, and the negative indifferently placed. A weak current is best, but its strength may be increased if such current be inefficient. The application should continue for about ten minutes. The wasted muscles recover as the inflammation subsides, but massage and galvanic electricity help them. Faradism is less favorably regarded, especially in the active stage.

Every case of sciatica should be at once ordered to rest, and the more complete the rest, the sooner the recovery. Splinting of the limb as recommended by Weir Mitchell.

For the relief of mild degrees of pain phenacetin and antifebrin, and

especially a combination of phenacetin and caffein citrate, say 10 grains (0.66 gm.) of the former and 3 grains (0.2 gm.) of the latter every two hours is often efficient. Acupuncture over the course of the nerve is of service for the same purpose—relief of pain rather than cure. Anodyne liniments may be used, and although not curative, do give some comfort and meet the wishes of the patient that something should be done.

In chronic cases change of scene is often of advantage, and if associated with thermal bath treatment may accomplish a cure in otherwise obstinate cases. The mud-bath is a measure of treatment applied in Europe with some success. In the chronic stage electricity also meets the demands of patients and friends and may do some good. The galvanic current should be employed. Massage is less efficient than in muscular rheumatism, though it should be tried. Nerve stretching is a very dubious expedient, it may cause serious consequences.

Multiple Neuritis.—The diagnosis of alcoholic cases is usually easy from the history, although sometimes skilful deception, especially in women, deprives the physician of this assistance. The distinctive features of the disease are the symmetrical localizations of the sensory and motor symptoms, first and mainly in the extremities, and the tenderness of the skin, nerve trunks and muscles. There are, however, great variations in different cases, even in those dependent on the same cause, some cases being very acute and general and even rapidly fatal, others slow with limitation to groups of muscles; some mainly motor, others sensory and ataxic (pseudo-tabes).

The possible sources of confusion are *rheumatism*, acute and chronic; *neuralgia*, *tabes dorsalis*, *poliomyelitis*, acute and subacute; *pachymeningitis* damaging the nerve-roots, *acute ascending paralysis*, and *hysterical palsy*.

In *rheumatism* the tingling characteristics of neuritis is not present, and although the tenderness of a nerve passing in the neighborhood of a joint, especially likely to be aggravated in motion, may be mistaken for joint pain, careful examination will elicit its true nature. Neuritis differs from *neuralgia* in the bilateral symmetry of the pain, and in the persistence of tenderness and hyperesthesia as contrasted with the spontaneous pain of neuralgia.

The ataxic form of the disease, especially the form called neuro-tabes (peripheral pseudo-tabes), sometimes resembles *tabes dorsalis* very closely. In neurotabes the lesion consists only of the nerve degeneration, while the spinal cord is free, its claim to the title being the fact that the loss of motor power may be slight in neuro-tabes, as in most cases of true tabes. The diagnosis from tabes may generally be easily made. The association of absolute paralysis or distinct weakness of extensors with inco-ordination would indicate neuritis. The "lightning pains" of tabes are seldom found in neuritis, nor are waist constriction nor pupillary symptoms, while the muscular tenderness is not found in tabes. Rapid onset of the disease and ultimate recovery occur in pseudo-tabes. The extreme hyperesthesia, so distinctive a symptom of neuritis, may be less valuable in diagnosis, because it is often absent in the ataxic form. Girdle pains, paralysis of the sphincters of bladder and rectum, are much more common in cord involvement, even in alcoholic cases, but may occur in multiple neuritis.

Poliomyelitis—inflammation, acute or subacute, of the gray matter of the cord—resembles the rheumatic and toxemic forms of neuritis, which have, like it, a febrile onset, *initial* rheumatic pains, and muscular wasting with the reaction of degeneration. But, again, we contrast the symmetrical distribution of the palsy of neuritis and its limitation to nerve distribution with the random distribution of poliomyelitis. Pain on pressure and subjective sensory disturbances are not prominent in poliomyelitis, but sometimes the symptoms are suggestive of the association of multiple neuritis with poliomyelitis.

In *pachymeningitis* which involves the nerve-roots, producing paralysis, wasting, and anesthesia, the legs do not suffer early in the disease, as a rule; and while the upper parts of the arms and trunk may be anesthetic, there is no tenderness of the nerve trunks, unless these also are inflamed.

Acute ascending paralysis (Landry's) resembles the most rapid form of multiple neuritis in some of its symptoms, but the paralysis usually ascends the trunk from the legs to the arms, and does not begin in the hands and feet *at the same time*, nor does it affect the trunk last, as in neuritis. There is, moreover, no anesthesia in typical ascending paralysis. There are, however, transitional cases between multiple neuritis and Landry's paralysis, and the term Landry's paralysis is somewhat in disfavor.

Some assistance in diagnosis may be had from the etiological standpoint; the history of metallic poisoning, of alcoholic excesses, or of exposure to infectious diseases, or the presence of diabetes being suggestive.

Prognosis.—*Localized neuritis.* The prognosis varies greatly, being favorable in mild and in most traumatic cases. Those consequent upon local suppuration are the gravest. In ordinary cases from cold or contusion recovery usually ensues sooner or later, although some last a long time and recurrences are not unusual, especially in neuropathic dispositions, in which, too, recovery is slower.

Multiple Neuritis.—A large number of patients with multiple neuritis get well, though slowly, especially if the cause be discovered and removed. Especially is this true of the alcoholic cases, although improvement does not always begin immediately on withdrawing the cause—indeed, the disease may even progress for a long time, and improvement may not be observed for several months. Hence the prognosis should be guarded. The acute and widespread cases are the most dangerous to life, and in such the prognosis should always be guarded. The involvement of the heart and of the muscles of respiration, including the diaphragm, is most to be feared. Pain in the trunk muscles is a grave symptom if the motor power of the limbs has diminished much. Paralysis of the diaphragm may be insidious and unnoticed until that of the intercostals is added, when there may be accumulation of mucus, bronchitis and death by suffocation. Involvement of the vagus nerves is manifested by frequency of pulse. Superadded involvement of the spinal cord increases the danger. At best, months are required for recovery, and even years may be necessary. Extensive involvement of the spinal cord precludes total recovery. The return of faradic irritability in nerve and muscle is favorable. To sum up with Gowers: "The prognosis is better in the sensory than in the motor form, better when the arm escapes than when all the limbs are involved, better in cases of chronic than acute

onset, and better if a case of apparently acute onset is really such, than if it succeeds slight symptoms of longer duration."

Treatment.—The removal of the cause, if possible, is a primary step in treatment. Along with this, rest is most important, and the rest should be complete—in bed, and this should be enforced in the earlier stages; later the pain and loss of motor power make rest obligatory. Care should be taken to avoid any pressure of the bed-clothes upon the feet, which might aid in the contracture of the muscles in the position of foot-drop. There should be no compromise with alcohol, although in some cases of great debility, when the cardiac action is feeble, gradual withdrawal may be justifiable. The patient should, on the other hand, be fed on the most nutritious food. Local anodyne applications may be resorted to to relieve the pain, and may be varied according to effect. Dry heat, moist heat, applications of lead-water and laudanum, and ointments of aconite and veratrum are some of those which may be employed. Wrapping in cotton or wool is sometimes beneficial. Warm baths are soothing; sometimes very hot ones give relief.

Postures assumed because of the relief they give to pain should not be too long permitted lest deformity result by contraction and adhesion, difficult or impossible to overcome. Dropping of the feet should be prevented by splints or by support with sand-bags. The same is true of flexion at the knee and hip.

As to drugs, they are of little use; the salicylates, phenacetin, antifebrin, and antipyrin may be useful in mild cases, and should be tried in doses of from 5 to 15 grains (0.3 to 1 gm.), but their action should be watched if the heart be affected. They are more particularly useful in cases due to cold. Extreme pain may demand the cautious use of morphin hypodermically in doses of from 1/6 to 1/3 grain (0.011 to 0.022 gm.) combined with 1/150 grain (0.00044 gm.) of atrophin, which modifies and improves the action of morphin most happily. For the mental symptoms the hydrobromate of hyoscin in doses of from 1/200 to 1/100 grain (0.00033 to 0.00066 gm.) hypodermically, or hyoscin in doses of from 1/400 to 1/150 grain (0.00016 to 0.00044 gm.) may be tried. Mercurials, so highly approved by Gowers in simple neuritis, are useless here. The iodids are sometimes beneficial in chronic cases and in cases due to lead absorption.

Roborant medicines, such as iron and cod-liver oil, are indicated to build up the patient, who is generally broken down. Electricity, massage and strychnin are very useful after convalescence has set in.

ADIPOSIS DOLOROSA.

SYNONYM.—*Dercum's Disease.*

Definition.—A condition first described by Dercum, in 1888, in which there are irregular deposits of fat in different parts of the body which are the seats of pain or tenderness or have been preceded by it.

Etiology and Pathology.—The subjects are almost always women. A neuropathic family predisposition, the alcoholic habit and syphilis, have been generally present. Sclerosis with diminution in size of the thyroid gland has been found and in a case of Burr there was a tumor of the pituitary

body. There is sometimes neuritis and later degeneration of smaller nerve branches, the main trunks being intact. The disease is probably a neuritis associated with peculiar fat formation and in relation with the ductless glands. Attention has already been called to the probable relation of this disease to disease of the hypophysis.

Symptoms.—Sometimes, after middle life, the patient, usually a woman, acquires irregular deposits or bunches of fat in various parts of the body. These gradually become the seat of burning, scalding, shooting pain and paresthesias. The masses of fat grow larger and become soft and pultaceous, but do not pit on pressure. There remain areas of the body quite uninvolved, especially the peripheral parts of the limbs. Hyperesthesias may alternate with anesthesia elsewhere than in the fatty masses. At times there is mental weakness, even dementia. As the accumulations grow there succeeds muscular weakness, at times extreme; the skin appears normal, the hands and feet remain normal. The skin is at times normal, at others it is pigmented or atrophied.

Diagnosis.—The disease differs from simple obesity in the lumpiness as contrasted with the uniform distribution of fat, and by the painfulness and tenderness. From myxedema it differs in the absence of the peculiar facies and other symptoms which attend myxedema.

Treatment is without effect. Local anodynes may palliate. Thyroid extract should be tried. Coal-tar derivatives may be employed, especially aspirin and phenacetin. Morphin should be put off as long as possible.

NEURALGIA.

Definition.—Strictly speaking, the term neuralgia should be restricted to such varieties of nerve pain as are unattended with structural changes in the nerve. Formerly, much that is now regarded as neuritis was called neuralgia, and it is probable that, as our knowledge grows, other so-called neuralgias will be eliminated. Finally, the border-line existing between neuralgia and neuritis cannot be drawn sharply, but as far as possible, the term neuralgia should be restricted to nerve pain without organic change.

Etiology.—Neuralgia is a disease of adults. It rarely occurs before puberty, and is relatively rare in old age. It is more common in women than in men, although not so very rare in old men. Heredity is responsible for a tendency to neuralgia. According to Anstie, fully one-fourth of all cases are the result of heredity. It is frequent in so-called neurotic families and in the so-called "nervous" person—*i. e.*, one who is excitable, anxious, and fretful in disposition. In this category, too, are the hysterical neuralgias. The debilitated, anemic, and poorly fed are liable to it. So are they who are overworked and worried.

The most frequent *exciting* cause is cold. Malaria is one of the most common causes, producing, especially, hemicrania, while the malarial cachexia also predisposes to neuralgia. The pain of carious teeth is not regarded as neuralgic, but when such pain causes irritation of the peripheral branches of the fifth nerve, a neuralgia may be produced in the distal distribution.

Symptoms.—*Pain* is the leading symptom. "Spontaneous pain," by

which is meant pain independent of neuritis or irritation of the nerve, and the modifications to which it is subject in severity and distribution, constitute, in fact, the disease. This pain is irregularly paroxysmal, shooting, darting, or burning in character, not usually increased by motion and if not relieved by pressure, may be by gentle friction. The more the pain is increased by motion and the more there is pain over the nerve trunks on pressure, the more is it a neuritis and the less a neuralgia. Yet we cannot literally adhere to this, as evidenced by the "tender points" of Valleix, which will be further referred to under the different varieties of neuralgia. Multiple dartings and shootings, separated by seconds or minutes of freedom from pain, are characteristic.

The absence of primary tenderness is also characteristic; but after the pain has continued for some time there often succeed tenderness of the skin and even a redness and swelling, the absence of any unnatural degree of which at the beginning is considered distinctive. These phenomena, including edematous swellings, are regarded as vasomotor in origin. Other vasomotor symptoms are hyperidrosis, increased secretion of saliva and tears, and elevation of temperature. Trophic effects are seen in shedding of the hair and its rapid blanching, and other symptoms to be referred to. Muscular twitchings are also not uncommon at the seat of the pain, and sometimes even muscular spasm.

The duration of an attack of neuralgia varies from an hour or even less to many hours. Sooner or later, if not relieved, it subsides spontaneously, though with a greater tendency to recur than when relieved by treatment.

Varieties Depending upon Nerves Involved.

Neuralgias are variously named in accordance with the nerves affected, whence we have the following varieties:

1. *Trifacial Neuralgia* (*Neuralgia of the Fifth Pair; Tic douloureux; Prosopalgia*).—This form involves one or more of the branches of the fifth pair, rarely all. It is more common than all other varieties of neuralgia combined. Here, doubtless, we have sometimes to do with a neuritis not always easily separable. One or more numerous tender points are usually demonstrable, of which those at the supra-orbital and infra-orbital foramina are the most conspicuous.

Of the branches of the fifth, the *ophthalmic*, or the first division through its supra-orbital branch, is that most frequently affected, giving rise to the well-known supra-orbital neuralgia. The pain radiates from the "tender point" at the supra-orbital notch over the anterior half of the head sometimes to the eye itself, the eyelid, and half of the nose. There may be injection of the eye and suffusion. There is sometimes pain in the occipital protuberance and cervical spines. This supra-orbital form must most frequently be distinguished from catarrh of the frontal sinuses, but the latter is more likely to be symmetrical, and while the pain is severe, it is duller, less shooting, and is accompanied by coryza; it terminates suddenly with a free discharge of purulent matter, sometimes offensive.

When the distribution of the *infra-orbital*, or second branch is involved,

the pain occupies the superior maxillary area between the orbit and the mouth, over the cheek to the ala of the nose. The "tender points" are at the emergence of the nerve below the orbit, at the side of the nose, over the most prominent part of the malar bone, and along the gingival line in the upper jaw, rarely in the upper lip.

When there is involvement of the third, or *inferior maxillary*, division, less common as an isolated form—except as to its inferior dental branch—there is a much more extensive area of pain, including the parietal eminence, the temple, the ear, the lower jaw, and the tongue. The "tender points" are in front of the ear where the auriculotemporal crosses the zygomatic arch, where there is often burning pain, and at the mental foramen on the chin. The movements of mastication and speaking may be painful, and there may be salivation. A herpetic eruption about the eyes or lips occasionally present points to neuritis. Atrophy and induration of the skin have been included in the symptoms, but these are ascribable also to a neuritis.

There is a pure *ocular neuralgia* involving the eyeball only. It may or may not be due to errors of refraction. Of these, hypermetropia, or far sightedness, is the most common cause. Either one or both eyes may be affected. It may be accompanied by dimness of vision.

A form of trigeminal neuralgia, called by Trousseau "epileptiform," consists in sudden, severe, and frequent attacks of pain, lasting from a few seconds to a few minutes, many times repeated during the day.

2. *Cervico-occipital Neuralgia.*—This affects the area of the neck supplied by the posterior branches of the first four cervical nerves, and the posterior part of the head supplied by the great occipital branch of the posterior division of the second cervical nerve, at the exit of which there is a tender point about half way between the mastoid process and the first cervical vertebra. Two other tender points are just above the parietal eminence, and between the sternomastoid and trapezius muscles. The pain may extend over the greater part of the neck and head, as far forward as the parietal eminence and the ear.

Exposure to cold or a draft of air is the most common cause of this form. Nephritis has been alleged to be a cause.

3. *Cervico-brachial and Brachial Neuralgia.*—This involves the area supplied by the four lower cervical and the first thoracic nerves, the area of sensory distribution of the brachial plexus.

The tender points are the axillary, the circumflex at the posterior part of the deltoid, the superior ulnar behind the elbow, and the inferior ulnar in front of the wrist. This form is often confounded with neuritis due to rheumatic affections of the joints or injury.

4. *Neuralgia of the Phrenic Nerve.*—This is rare, the pain in its area during pleurisy and pericarditis being rather a neuritis. The pain is at the lower part of the thorax, at the attachment of the diaphragm. Breathing is shallow, because pain is caused by the breathing movements. Coughing and even deglutition cause pain.

5. *Trunk Neuralgia.*—This naturally divides itself into two subvarieties: dorso-intercostal and lumbo-abdominal.

(*a*) *Dorso-intercostal neuralgia* covers the area supplied by the inter-

costal nerves from the third to the ninth, and is characterized by pain along the intercostal spaces or in parts of them. It is sometimes bilateral. There is usually a constant dull pain with or without acute stabbing exacerbations, or the latter may be excited by deep breathing or motion. There may be special tenderness at the points of emergence of the three branches of the intercostal nerve—viz., posteriorly near the vertebræ, anteriorly near the median line, and midway between these two points in the midaxillary line.

The term *pleurodynia* has been used with a good deal of vagueness. Strictly speaking, it should be limited, as it is by Gowers, to neuralgia of the pleural nerves. Consistently with this it should not be applied to pain localized in the course or point of exit of an intercostal nerve. It is very acute in character and excited by expansion of the thorax rather than by lateral movements of the trunk. The pain of herpes zoster is not a neuralgia, but a neuritis.

Another variety in this locality is the inframammary neuralgia of anemic women.

(*b*) *Lumbo-abdominal neuralgia* involves the posterior branches of the lumbar nerves, especially the ilioscrotal branch. The area of the pain is the region of the iliac crest, along the inguinal canal and the spermatic cord in the scrotum, or round ligament in the labium majus. The pain is often bilateral, sometimes resembling the constricting girdle pains of spinal cord disease, from which it differs, however, by its changing place. It is especially frequent in connection with the diseases of pelvic organs, particularly in women. The testes and penis are the seat of neuralgic pains.

6. *Neuralgia of the spinal column* is the more modern term for the "spinal tenderness" of the older authors. It is common in feeble and hysterical women, and a sequel of the modern railway accident under the name of "spinal congestion." The pain in most cases is felt along a considerable vertical extent of the spine, but is more intense in certain spots. The thoracic region is the most common seat, next the lower cervical, and least frequently the lumbar region. The pain in some cases is purely hysterical.

7. *Sacral neuralgia and coccygodynia* are defined by their names. These affections reside in the nerves between the bone and the skin, and are often exceedingly difficult to cure. The pain may really be due to organic lesions in the part.

8. *Neuralgia of the feet* includes painful heel, plantar neuralgia, and erythromelalgia. In the latter, first described by Weir Mitchell, vascular changes, including either acute hyperemia or cyanosis—probably of vasomotor origin—are associated with severe pain in the heel or sole of the foot. It is probably a neuritis in some cases. In some others it is caused by disease of the vessels. It is often mistaken for flat foot.

9. *Visceral neuralgia* means neuralgia affecting the gastro-intestinal tract, the kidneys, ovaries, and other pelvic organs. Idiopathic nephralgia, or neuralgia of the kidney, probably does not exist. It and testicular neuralgia are more frequently secondary to inflammation of adjacent urinary passages, but idiopathic testicular neuralgia is less rare then nephralgia.

Neuralgias are further classified according to character and cause. Thus, in addition to the epileptiform variety alluded to, there are reflex or

symptomatic neuralgias, traumatic neuralgias, herpetic neuralgias accompanying herpes, hysterical, rheumatic, gouty, diabetic, anemic, malarial, syphilitic, and degenerative neuralgias. Many of these terms are loosely applied. The term rheumatic neuralgia is often erroneously applied to myositis. It should not be used.

Very interesting and important is the subject of *reflex neuralgias* and *referred pains* which have been especially studied by the late Dr. Anstie and by Henry Head in England and Charles L. Dana in this country. Reflex neuralgias are due to disease in organs distant from the actual seat of the neuralgia. The fifth nerve is a favorite seat of such neuralgias. Thus, an irritation of the distribution of one branch of this nerve by a carious tooth may excite a neuralgia in another distribution of the same nerve. Illustrations of referred pain are the "pain in the back" or spinal cord pain in ulcer of the stomach, the left scapular pain in diseases of the liver, the sacral pain in uterine disease, and the testicular pain in renal colic.

Diagnosis.—Neuralgia is chiefly to be distinguished from *neuritis* and the effects of *pressure on nerves;* and also *rheumatism.* From *neuritis* it is separated by its unilateral distribution as contrasted with the more frequent symmetrical distribution of neuritis, although neuritis is not infrequently unilateral; also by its numerous remissions and intermissions, and the shifting of the pain from one spot to another. The fixed neuralgias are more difficult of separation from neuritis, especially mild cases. The severe forms of neuritis are soon recognized by the anesthesia which succeeds upon the hyperesthesia in the cases of sensory nerves, and muscular wasting with changes in the electrical irritability in mixed nerves. In the case of *compression of nerves* the pain is continuous, while the symptoms and consequences of neuritis will, sooner or later, show themselves. Nevertheless, doubt and error must not infrequently occur.

Myalgia differs in its localization in muscles or groups of muscles such as the lumbar or shoulder muscles, its continuousness and pain increased by motion.

Prognosis.—The prognosis in neuralgia is usually ultimately favorable, although some forms and cases are very stubborn. Especially true is this of neuralgia of the fifth pair. The more frequent the recurrence and the wider the distribution, the more difficult is the cure. On the other hand, the severity of the pain is not, in my experience, a measure of obstinacy to cure, some of the severest cases being easiest relieved. Hereditary cases are the more obstinate. The same is true of cases occurring in the decline of life. Epileptiform neuralgia is said to be incurable.

Treatment.—The treatment of neuralgia is divided into that of the *condition predisposing* to it and of the *paroxysm.* The anemias—especially chlorosis—malaria, and other predisposing causes should be corrected by quinin, iron, and arsenic. Good nourishing food is important. Change of scene and residence is often necessary. Reflex causes should be carefully sought for and removed. Until these predisposing causes are removed, the treatment of the paroxysm affords but temporary relief.

For the paroxysm quinin is by far the most efficient remedy, and will cure many cases. Two or 3 grains (0.12 to 0.194 gm.) should be given hourly until the paroxysm is relieved or decided cinchonism is produced.

The salicylate of cinchonidia is a valuable preparation. Some cases are relieved by phenacetin or antifebrin (acetanilid) in from 10 to 15 grain (0.66 to 1 gm.) doses. A combination of phenacetin and caffein, 3 grains (0.33 gm.) of the former and 1 (0.11 gm.) of the latter each, in hourly doses; is often efficient. Some cases can only be relieved by sulphate of morphin. The hypodermic injection is the promptest and surest remedy, in doses of from 1/8 to 1/4 grain (0.008 to 0.016 gm.), but morphin is a drug to be avoided in neuralgia, if possible, as the danger of acquiring the morphin habit is extremely great. The patient should never be allowed to use the hypodermic syringe himself. The use of anodynes is sometimes more than palliative, the repeated removal of the pain tending to prevent its recurrence. The combination of atrophin with morphin undoubtedly modifies the unpleasant effect of the latter drug and increases its efficiency.

Belladonna, and its active principle, atropin, are remedies which have long enjoyed reputation in the treatment of neuralgia, when uncombined with other drugs, but in our hands they have been feeble remedies. The doses recommended are from 1/6 to 1/2 grain (0.011 to 0.03 gm). of the extract and from 1/120 to 1/60 grain (0.0005 to 0.0011 gm.) of atropin. Aconite and gelsemium have also some reputation, especially in neuralgia of the fifth nerve. Gelsemium may be given in doses of 15 minims (0.92 c.c.) of the tincture, frequently repeated. Gelsemia may be given hypodermically in doses of from 1/60 to 1/30 grain (0.0011 to 0.0022 gm.), and aconitin in doses of from 1/250 to 1/100 grain (0.00027 to 0.00066 gm.), but the latter is a remedy so dangerous that it should be rarely employed. Cannabis indica is also sometimes useful in doses of 1/4 grain (0.016 gm.) three times a day or oftener, but the drug varies so much in strength that it cannot be relied upon.

Local applications are sometimes very useful. Pressure relieves many mild cases, especially when associated with gentle friction. Local anesthetics, such as menthol, the ointments of veratria and aconitia, are similarly useful; so is the tincture of aconite painted over the involved area. The local use of opiates, at least without first removing the epidermis, and of atropin (five per cent. strength), is, however, commended. The extract of belladonna, diluted with glycerin so as to admit its being smeared on, is sometimes useful. Frequent renewals of all these local applications should be made in the course of the day. Counterirritation by blisters or sinapisms, by chloroform either pure or variously diluted, and by camphor may be used. The last two may be applied on lint and covered with oiled silk. Both will blister if left on too long.

Acupuncture and *aquapuncture* are employed, the latter consisting of injecting water under the skin. For their local effect, also, *chloroform*, *carbolic acid*, and *osmic acid* have been injected *hypodermically*. From 15 to 20 minims (0.92 c.c. to 1.23 c.c.) of the first may be used, from 5 to 10 minims (0.31 c.c. to 0.62 c.c.) of the second, and 1 or 2 drops of a one per cent. solution of osmic acid in water and glycerin. Chloroform should be cautiously used in this manner, as it may occasion ugly sloughing. It is more especially in sciatica that these measures have been employed. *Local* applications of *heat* and *cold* have been found useful—cold by freezing or by the ethyl chloride spray; heat by the hot-water bag, or in the case of a

supraorbital neuralgia, by the nasal douche. Heat is usually more efficient than cold; indeed, the latter sometimes aggravates neuralgia.

Electricity is of uncertain value in neuralgia, but is sometimes very useful. The constant current is the form most frequently used, but faradism may also be employed. It is used in two ways: a strong current is applied at once with a view to removing the neuralgia promptly (this is scarcely to be recommended); in the second method a sedative effect is sought by a weak current, preferably of galvanism, just sufficient to produce a tingling or burning sensation. Experience goes to show that the direction of the current may be ignored, but it is commonly recommended to apply the positive pole to the painful part, the sponge being well wet with warm water, and if faradism is used, it should be with rapid interruptions.

The *surgical treatment* of neuralgia has been followed by brilliant results, and has met signal failures. The most common procedure is division of a nerve, or, better, the exsection of a portion of the nerve. It has been most frequently done in the case of the fifth nerve, and is almost always followed by temporary relief, but, sooner or later, an operation on the Gasserian ganglion or sensory root usually becomes necessary. Operation is to be recommended in intractable cases, and should be done at a point as near the origin of the nerve as possible, as second operations are not infrequently necessary on account of the recurrence of the pain. Injection of alcohol into the nerve has become a common method of treatment. The injection has even been made into the Gasserian ganglion.

Nerve stretching is also performed with a measure of relief less thorough than exsection, but in view of the fact that its disadvantages are less lasting, it is the better operation to do first in the case of certain nerves. It is important to remember that relief does not always immediately follow the operation. The sciatic is the nerve most frequently stretched, but the procedure is not to be recommended. The intercostals and branches of the fifth, including the lingual, have been similarly treated with satisfactory results.

TUMORS OF NERVES.

Definition and Morbid Anatomy.—Strictly speaking, the term neuroma should be restricted to tumors composed purely of nervous tissue, which are to be distinguished from *fibrous tumors* or *fibromata*, often seated on nerves and known as *false neuromata*. Some, however, dispute the existence of true neuromata, and they are certainly very uncommon. Another form of false neuroma is a variety of the small, subcutaneous, painful tumor —*tubercula dolorosa*—occurring in nerves of the skin in the neighborhood of the joints, on the face and on the breast. Myxomata, sarcomata, and even carcinomata are found in connection with nerves. The latter are commonly the result of extension by contiguity, infiltrating the connective tissue between the fibers. The nervous tissue represented in the true neuroma is usually fibrous, but very rarely ganglionic nerve cells are found, and in such event the tumor may be regarded either as dislocated nerve tissue or as a glioma the cells of which closely resemble true nerve cells. The nervous tissue may be of the medullated or nonmedullated variety—*i. e.*,

myelinic or nonmyelinic. Connective tissue varying in quantity is associated with both, producing various degrees of hardness, which is most striking in the multiple fibroneuroma.

An interesting variety is the *plexiform neuroma*, nodular and tortuous in appearance to the naked eye, the internal structure of which is composed also of interlacing nodular and tortuous nervous cords made up of connective tissue and nerve fibers. It is seen in connection with any of the spinal and even sympathetic nerves. The tumors may be extremely numerous. In a case lately presented by Weisenberg they were hundreds in number. It grows slowly, and probably begins in fetal life.

Fibromata of nerves are usually small, but may be three or four inches (7.5 to 10 c.m) in diameter and even larger. They are usually found seated in nerve trunks, or at their ends, are often multiple, and their number is sometimes large.

Etiology.—Nerve tumors which are not congenital may be traumatic. More than one member of a family has been found affected. Their growth seems stimulated by perversion in the healing process, since they are found on the ends of nerves in cicatrices after amputation. Growths of this character are truly neuro-fibromata, the others are usually fibromata.

Symptoms.—Neuromata may be totally without symptoms. At other times they are very painful, the *pain* being aggravated by pressure. There may be *numbness* and *formication* and even *loss of sensation* on the one hand, *muscular twitching and paralysis* on the other, the latter especially when the tumor is in the course of the nerve.

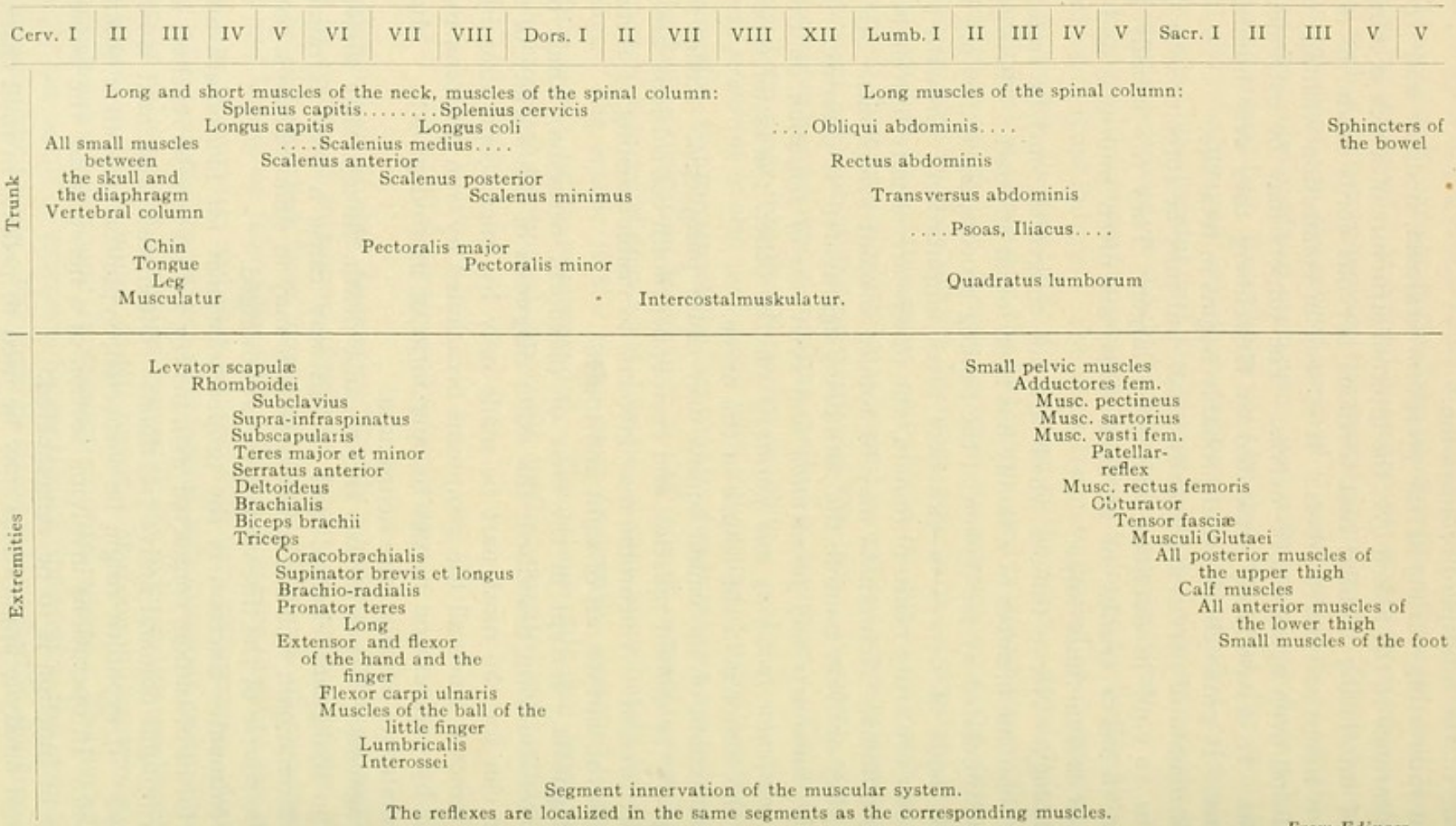
Neuromata of the cauda equina may cause paraplegia. Reflex spasm in adjacent or distant muscles, and even epileptiform convulsions, are occasionally present. A neuroma may give rise to visible swelling, or it may be beneath the surface out of sight and touch.

Diagnosis.—Except in the case of plexiform neuroma, which has a characteristic form described, the exact diagnosis of the variety of nerve tumor can for the most part be made only by microscopic examination after removal, since all the symptoms occasioned by true neuroma may be caused by pressure on nerves by any form of morbid growth. Multiple neuromata are usually false neuromata.

Prognosis.—Nerve tumors rarely cause death, though they sometimes undergo malignant change, and in this way cause a fatal termination. The extreme pain which is so characteristic may in time exhaust a patient, but the course of the disease is always prolonged.

Treatment.—Excision is the proper treatment for neuromata and all other forms of tumors connected with nerves, if they can be reached, and if the symptoms demand active treatment. Often such treatment is not demanded. If syphilitic origin be suspected, syphilitic treatment should be adopted. In operations involving section of a nerve trunk the possibility of loss of function is to be remembered.

Local anodyne applications may be used to palliate in mild cases, but they are useless in severe ones. Cocain in doses of from 1/6 to 1/2 grain (0.011 to 0.033 gm.) may be injected hypodermically, but morphin should not be used, as the conditions are especially favorable to the production of morphinism.



Segment innervation of the muscular system.
The reflexes are localized in the same segments as the corresponding muscles.

From Edinger.

The *muscular sense* "or sense of position" is probably conducted on the same side of the cord in the posterior columns, to cross in the medulla oblongata, and we have evidence that the tactile fibers ascend in the posterior and lateral columns. *Thermal* and *pain impulses* probably cross to the anterolateral columns of the other side very soon after entering the cord, and possibly ascend in or near Gowers' tract.

Spinal Cord Localization.—The areas of distribution of spinal nerves, sensory and motor, are not sharply defined for each nerve as it emanates from the spinal cord, while the regions supplied by these nerves overlap. At the same time physiologists and clinicians have been able to map out with approximate accuracy the motor and sensory areas corresponding to the distribution of each pair of nerves emanating from different segments of the cord. Among those who have especially devoted themselves to this subject are M. Allen Starr, Charles K. Mills, and Charles L. Dana in America, William Thorburn and Henry Head in England, and Sano, Bruns, Kocher, Strohmeyer and Seiffer on the Continent.

The results of various observers differ in detail, but agree in essentials. The appended table compiled by Edinger is from that originally compiled by Starr, further modified by C. L. Dana and C. K. Mills. When stood on end in front of the reader it will be found to correspond in outline to one-half of the body.

It must not be forgotten that these areas of distribution correspond to a nerve *constituted as it is when it emanates from a corresponding segment of the cord*, and not to a nerve as it is constituted immediately before it begins to spread.

The preceding table includes only the distribution of spinal nerves.

The following table includes the distribution of nerves starting from the nuclei in the pons and medulla oblongata, so far as these are concerned with motion:

Nuclei.	*Muscles.*
III Cranial.	Sphincter iris. Ciliary muscle. Levator palpebræ superioris. Rectus internus in convergence. Superior rectus. Inferior rectus. Obliquus inferioris.
IV Cranial.	Obliquus superioris.
VI Cranial.	(Upper facial group.) Rectus externus. Rectus internus of opposite side in lateral movements.
V Cranial.	Associated movement of levator palpebræ. Muscles of the lower jaw.
VII Cranial.	Facial muscles.
XII Cranial.	Lower facial group. Muscles of tongue.
IX Cranial.	Muscles of pharynx.
X Cranial.	Muscles of esophagus.
XI Cranial.	Muscles of larynx.

The study of the sensory areas is facilitated by the use of diagrams in which the areas are mapped out and indicated by color or a shading which will permit their easy separation, like those annexed, in which, too, the areas corresponding to each spinal segment are indicated by suitable lettering.

Interpreting by the data contained in tables and diagrams such motor or sensory derangements as may be present, one may deduce with more or

less accuracy the seat of the lesions in the cord producing them. It has been mentioned that motor localization, being more definite, its arrangement permits more exact inference than sensory derangements.

The union of both adds further facility. Results vary also according as a lesion involves only one-half or a complete section of the cord. It is evident that an injury involving the *entire transverse section* of the cord must produce, *first*, motor paralysis in all parts supplied with nerves emanating from segments below it. In less complete lesions correspondingly limited degree and extent of motor paralysis succeed. Such paralysis may

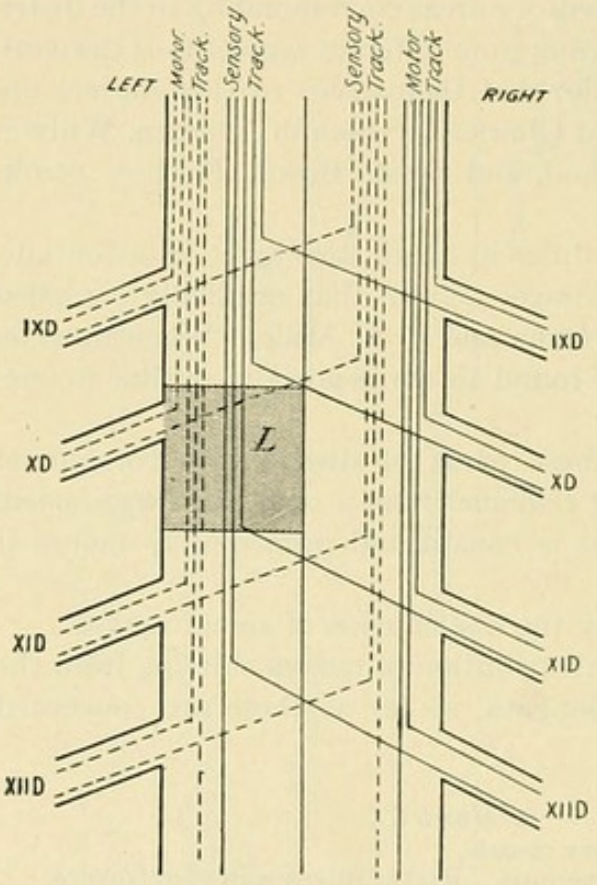


FIG. 144.—Diagram of Lesion Showing Brown-Sequard's Paralysis—(*after Starr*).

L. Lesion in left half of cord cuts off motor impulses to left leg, sensory impulses from right leg, and sensory impulses from eleventh dorsal nerve.

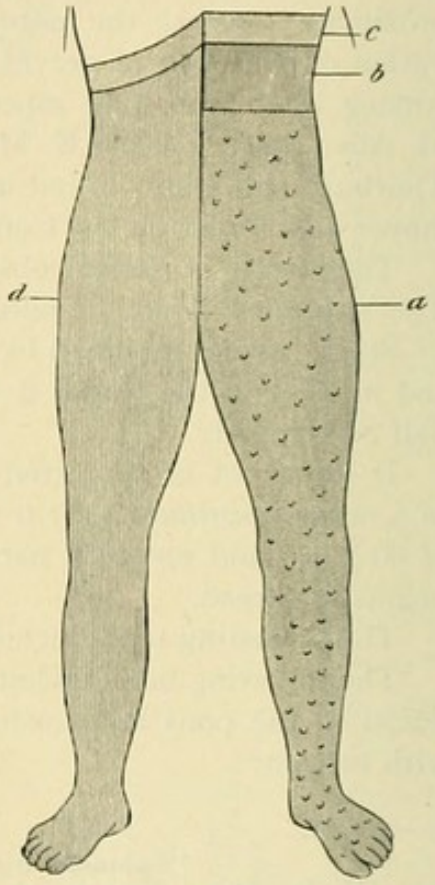


FIG. 145.—Schema Showing Chief Symptoms in Left Unilateral Lesion of the Dorsal Cord—(*after Erb*).

Oblique shading at *a* signifies motor and vasomotor paralysis; vertical shading cutaneous anesthesia at *b* and *d*; dots on a cutaneous hyperesthesia. *b*. Small anesthetic zone. *c*. Small hyperesthetic zone.

extend to the bladder and rectum. After complete or nearly complete section the muscles are usually flaccid and the deep reflexes absent. There is no rapidly developing atrophy, and the muscles respond normally to electricity. No satisfactory explanation has as yet been offered of the abolition of the deep reflexes in complete or nearly complete transverse lesion of the cord above the level of the reflex arcs; although neuritis is supposed by some to be the cause of this loss of the deep reflexes, it is probably not the cause in all cases. *Second*, there is impaired sensibility in the parts supplied by sensory nerves associated with corresponding segments below the lesion. Anesthesia does not, however, reach quite

to the level of the lesion, because of the overlapping of sensory areas by nerves which enter the cord above the section. Thus, if the section be in the segment of the sixth thoracic, the anesthesia may extend only as high as the area supplied by the seventh. Moreover, above the anesthetic area there is also at times an area of increased sensibility—the effect of the section possibly being to increase the sensitiveness of the cord above it by increasing its vascularity—due to section of vasomotor nerves by the lesion. By means of these facts we may be enabled to ascertain the level of the disease.

Muscular sense or sense of position is lost. *Reflex excitability*, at first slightly impaired, may subsequently be increased when the lesion is not complete and is above the reflex arcs, but may remain impaired in complete transverse lesions of the cord or in those portions of the body whose reflex arcs are situated in the damaged region of the cord.

The phenomena are modified if the lesion be a *hemi*-lesion of the cord. In such an event there is, *first*, motor paralysis in the portion of the body on the same side supplied by nerves whose cells of origin are below the lesion (Figs. 144 and 145), varying, however, with the seat of the lesion. If the lesion is in the cervical part of the cord, the motor paralysis is of the arm and leg on the same side, while if in the lumbar part of the cord there is loss of motion in the leg only of the same side. There is diminished sensibility in the arm and leg of the opposite side. The anesthesia may be to pain and to thermic sense only, the tactile sense being unimpaired. Such anesthesia exists on the *opposite* side, because of the fact, already mentioned, that one of the many routes of sensory impressions crosses the cord soon after it enters it from the periphery.

More than this: the sensibility on the *same* side, below the segment of the lesion, so far from being diminished as to touch, pain, and temperature, may even be slightly increased, possibly owing to the vasomotor paralysis caused by the lesion, in consequence of which, too, there may be a slight rise of temperature on the same side. Slight pricks may be painful, and the soles of the feet may be unusually sensitive. In the area corresponding exactly to the segment involved on the same side there is anesthesia, while just above it on the same side, again, there is a small zone of hyperesthesia. The anesthesia is due to the fact that the sensory nerves coming from the same side are cut just as they enter the cord. It begins somewhat lower down than the exact seat of the lesion, because of the overlapping of the upper sensory area. The hyperesthesia in the lower portions of the body on the side of the lesion has been said to be inexplicable, but may it not depend on hyperemia due to section of vasoconstrictor nerves? It may be for this reason also that the temperature is higher on the side of the lesion—from 1° to 2° F. (0.5 to 1° C.). The upper hyperesthetic zone above the anesthetic area on the side of the lesion may be explained as the result of irritation of sensory nerve fibers entering just above the lesion. The muscular sense or sense of position on the same side is impaired, a condition ascribed by Brown-Séquard to the fact that the fibers of this sense run on the same side uncrossed, and probably in the posterior columns, until the medulla oblongata is reached. Reflex excitability, at first diminished on the side of lesion, is subsequently increased and there is often a good ankle

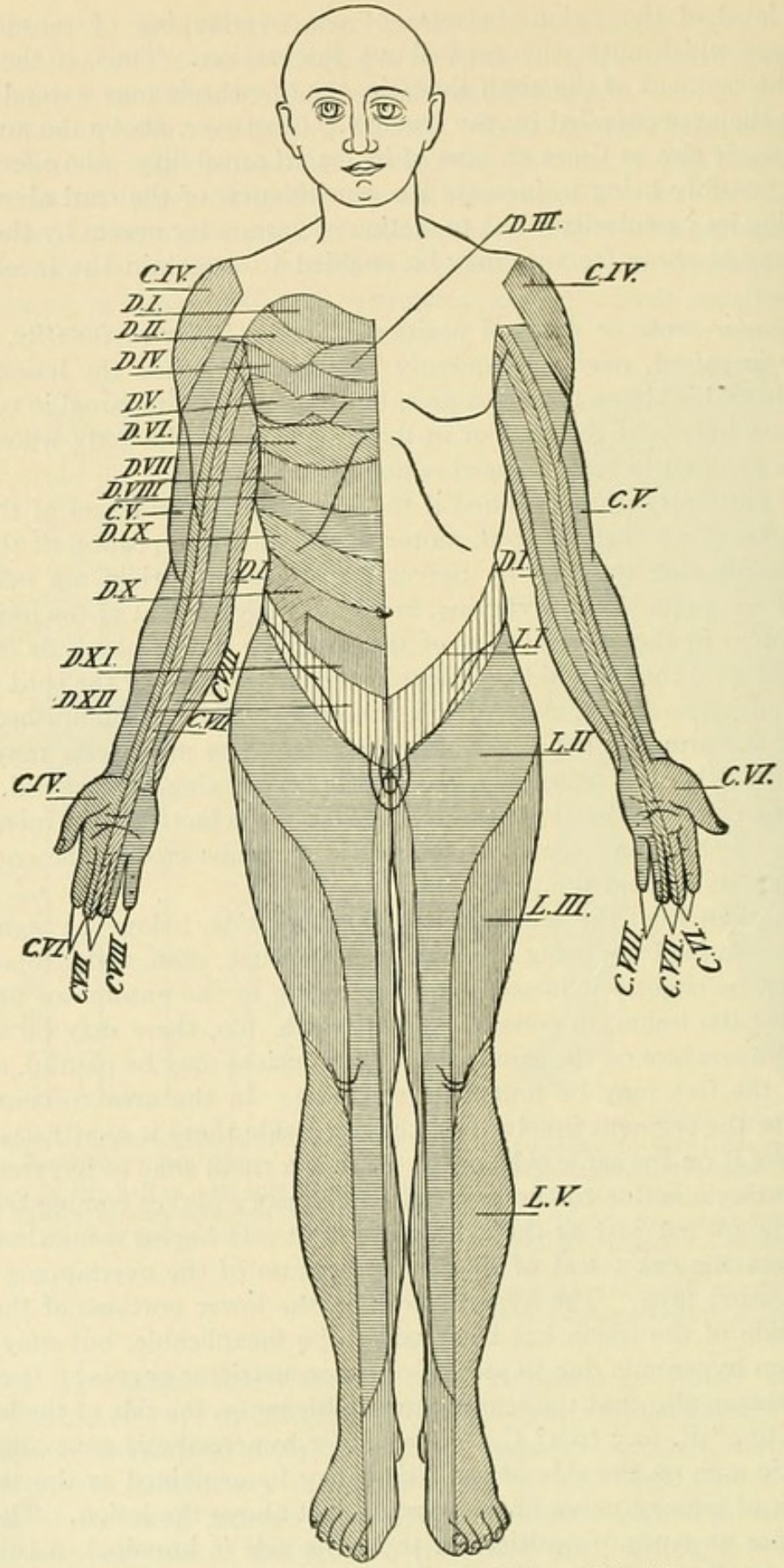


FIG. 146.—Diagram of Skin Areas Corresponding to Different Spinal Segments—(*chiefly after Starr. Trunk areas from Head*).

Roman numerals refer to nerves.

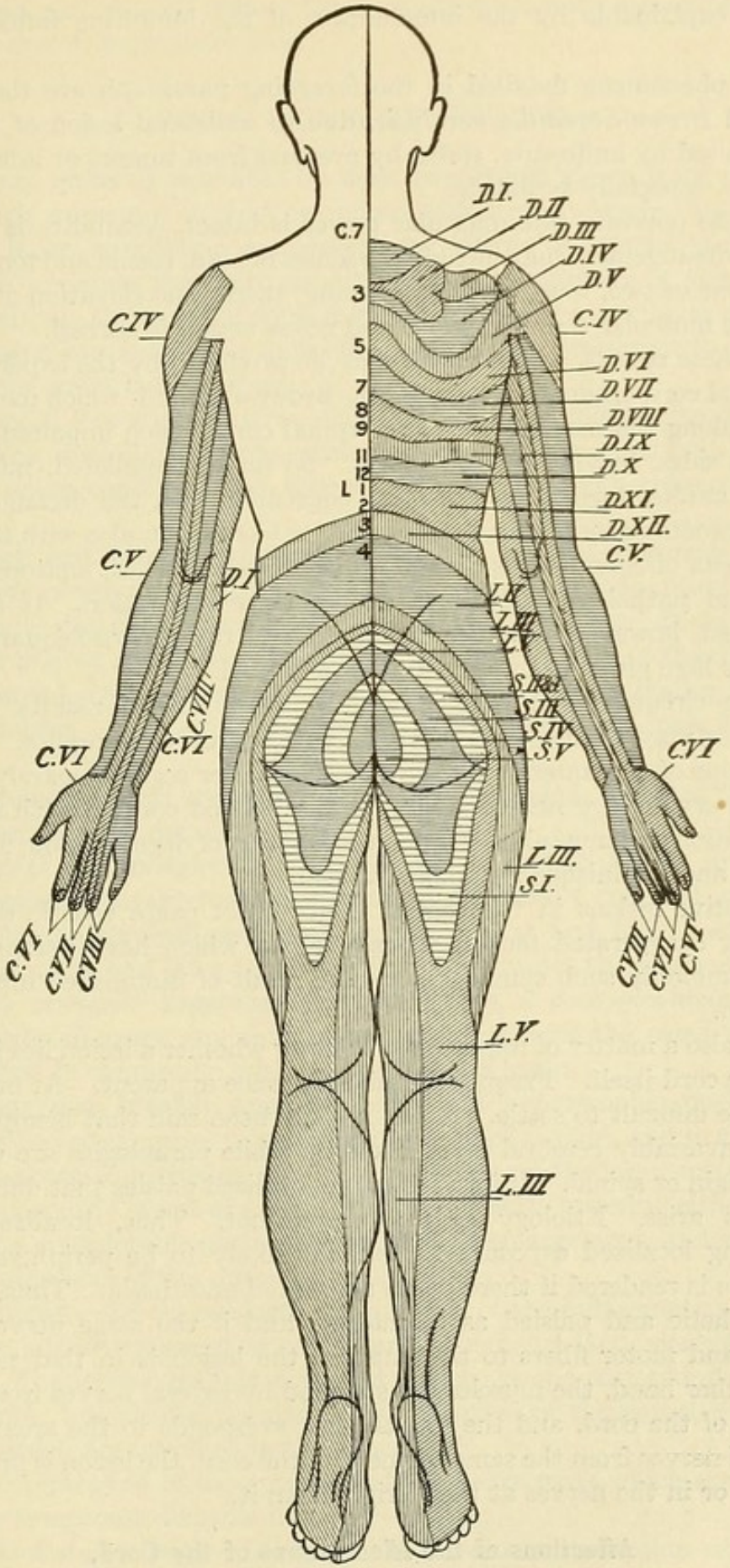


FIG. 147.—Diagram of Skin Areas Corresponding to Different Spinal Segments—(*chiefly after Starr. Trunk areas from Head*).

Arabic numerals refer to vertebra, Roman to nerves.

clonus, explainable by the interruption of the inhibiting influence from above.

The phenomena detailed in the foregoing paragraph are those of the so-called *Brown-Séquard's paralysis*, due to unilateral lesion of the spinal cord, caused by knife-cuts, stabs, by pressure from tumors or inflammatory products, especially syphilitic.

On the opposite side muscular power is intact, sensibility is impaired, and the derangement may include the senses of pain, touch, and temperature, or any one or two, touch usually escaping; there is no elevation of temperature, the muscular sense is intact, and reflex action is normal.

All these results, as described, may be produced by the experiments on the spinal cord originally suggested by Brown-Séquard, which included also section along the median line of the spinal cord, which impaired sensation on both sides, leaving motion intact. So far as completed, minute anatomical studies furnish results quite consistent with the derangements of motion produced by diseased states, and, to less extent, also with the morbid phenomena of sensation as illustrated by disease. Thus, anatomy, experiment and pathology contribute to the same conclusion. It should be mentioned, however, that the explanation of the Brown-Séquard type of paralysis here given has not been fully accepted.

More circumscribed lesions produce more limited results. Thus, a local lesion may produce paralysis in only a few groups of muscles. Destructive lesions of the anterior cornua produces lower segment paralysis in the parts innervated by nerves arising in the injured cornua, with secondary degeneration and muscular atrophy, the reaction of degeneration, diminished reflexes, and diminished muscular tension.

Irritative lesions in the central motor tract cause spastic conditions, including exaggerated tendon reflexes, all of which have been described. More commonly such symptoms are the result of diminished inhibition of the brain.

It is also a matter of importance to know whether a lesion lies in a nerve or in the cord itself. Frequently this is at once apparent. At other times it is more difficult to settle. It has already been said that hemiplegias are almost invariably cerebral in their origin, while paraplegias are usually of nerve origin or spinal. It is chiefly with localized palsies that difficulties in diagnosis arise. Etiology aids us somewhat. Thus, localized palsies succeeding localized exposure to cold are likely to be peripheral. Some assistance is rendered if there be an associated anesthesia. Thus, if a part be anesthetic and palsied as to motion, and if the same nerve supplies sensory and motor fibers to the muscles, the lesion is in that nerve. If, on the other hand, the muscles are supplied by several nerves from a given segment of the cord, and the anesthesia corresponds to the area of distribution of nerves from the same segment of the cord, the lesion is probably in the cord or in the nerves at their origin from it.

Affections of the Membranes of the Cord.

As in the case of the brain, the dura mater and pia arachnoid may be separate seats of disease, chiefly inflammatory, not quite so well understood nor quite so definitely separated in their clinical features. As in the case

of the brain, too, we call inflammation of the dura mater pachymeningitis; of the pia mater, leptomeningitis.

SPINAL PACHYMENINGITIS.

The dura mater is separated by loose connective tissue from the bony canal which surrounds it, and an inflammation may invade this outer or the inner layer, affording a *pachymeningitis externa* or *interna*, though it is not easy to separate these two conditions symptomatically or even always anatomically as the external form may extend to the inner layer and even the pia mater, but usually the dura offers an effectual resistance to processes which begin on its outer surface.

External Pachymeningitis.—This is usually secondary to disease of the vertebræ or similar morbid processes or to trauma or aneurysmal erosion. While an acute condition may thus supervene, it is much more commonly chronic.

Etiology and Morbid Anatomy.—Perhaps its most frequent cause is tuberculosis of the spine, with its pathological cheesy product and its traumatic result—the spinal curvature known as Pott's disease. It may be confined to a limited area, corresponding to the primary seat of the disease, or it may extend over a large area of the meninges, corresponding to six or eight vertebræ. Such inflammations seldom spread to the inner layer and pia.

Symptoms.—These are those of the vertebral lesion, together with those of the internal form detailed below.

Internal Pachymeningitis.—This occurs in two forms, first as an inflammation of the internal layer of the dura, usually confined, primarily, at least, to the cervical part of the cord. It was first fully described by Charcot in 1871, and later by his pupil Joffroy, under the name of "*pachymeningitis cervicalis hypertrophica*"; second, as a *pachymeningitis interna hemorrhagica*, in every way anatomically identical with the same disease to be described in connection with the dura of the brain.

Etiology and Morbid Anatomy.—Cervical hypertrophic pachymeningitis, ascribed to exposure to cold, to the abuse of alcohol, and to syphilis, is a chronic process, consisting in an accumulation on the inner surface of the dura of concentric layers of a firm, fibrinous growth, covering either a small extent or a considerable portion of the cervical enlargement, and sometimes causing adhesions of the dura to the pia.

Symptoms.—To the subjective symptoms of the inflammation itself are naturally added compression symptoms, which, in fact, overshadow the former. The former include *pain*, not merely at the seat of inflammation in the back, but also in the area of distribution of the spinal nerves, the roots of which are involved in the process.

The compression of the cord and of the nerve roots which are involved produces symptoms divisible into three stages:

1. *The Painful Stage.*—In this there is pain in the region supplied by the nerves whose roots are thus compressed—viz., that of the arms, cervical region, and occiput—pain at times of great severity. In addition are observed paresthesia, numbness, and tingling, rarely herpes.

2. *The Stage of Paralysis of the Upper Extremities.*—After two or three months the second period, or stage of paralysis, sets in—an atrophic paralysis in which there is weakness of the arms, resulting from pressure on the anterior nerve roots. The wasting affects certain muscular groups, as the flexors of the hands, supplied by the ulnar and median nerves, while the distribution of the posterior interosseous nerve to the antagonistic extensors may remain free. The result is the very striking claw-hand, or *main en griffe*. In extreme cases the atrophy of the arms and shoulders becomes very great. There may be anesthesia of the skin an this stage.

3. *The Stage of Spastic Paralysis in the Lower Extremities.*—If the compression of the cord continues, we reach the third stage of the disease. The motor fibers to the lower extremities which pass through the cervical cord become involved, and the result is a spastic paralysis of the lower extremities—a paresis with *increased reflexes*, and without wasting of the muscle, because the trophic centers for the muscles of the lower extremities in the anterior cornua of the lumbar cord remain intact. In cases of long duration, however, the compression of the cervical cord may lead to anesthesia of the lower extremities, paralysis of the bladder, and bed-sores.

The symptoms of the internal *hemorrhagic* pachymeningitis are not essentially different from those detailed, but are commonly superadded to those of hematoma of the dura mater of the brain, with which it is usually concurrent. It has generally been observed in the same class of persons, general paralytics and drunkards. It may occur at any part of the cord, or it may be limited to the cervical region, producing corresponding symptoms, but it is rarely recognized before death and is an extremely rare finding.

Diagnosis.—The superaddition to the symptoms of spinal caries of those detailed as characteristic of spinal pressure determines at once the condition. The forms arising in other ways are to be distinguished from *amyotrophic lateral sclerosis*, *syringomyelia*, and *tumors*. From the first it can be differentiated by the presence of the characteristic severe pain in the neck and arms, and by the absence of bulbar symptoms; from syringomyelia, by the absence of the sensory changes peculiar to that disease; but from tumors in the same locality it is often distinguished with difficulty because the pressure symptoms in both are the same.

Prognosis.—Cases are described in which decided improvement has taken place, but recovery or much improvement is improbable, when the process is internal as well as external.

Treatment.—The usual methods of treating spinal caries by extension or operation constitute the treatment of the external forms thus arising. The symptoms are to be relieved by appropriate measures. Baths, iodid of potassium, counterirritation, and electricity have been recommended.

The first three are reasonable; the last is of doubtful value. Iodid of potassium is indicated in cases of syphilitic origin. Joffroy recommends the application of the hot iron to the neck. Paquelin's cautery would answer the purpose as well.

SPINAL LEPTOMENINGITIS.

Acute Spinal Leptomeningitis.

Etiology.—As a disease separate and distinct from epidemic cerebrospinal meningitis, described under infectious diseases, acute spinal leptomeningitis may occur:

1. As the result of tuberculosis, its most common cause. When thus occurring, it is as a tuberculosis infection separate and independent of the tuberculosis extension in Pott's disease.

2. From localization of the poison of the infectious diseases, as syphilis and typhoid fever.

3. As the result of extension by contiguity.

Morbid Anatomy.—The pathological changes are similar to those of epidemic cerebrospinal meningitis. Injection, accumulation of fluid in the piaarachnoid space, either a serofibrinous or a purulent exudate, round-cell infiltration, and finally thickening of the membrane, all are more or less in evidence. As determined by the position of the body, the fluid exudate tends to gravitate downward or toward the posterior aspect. Not infrequently the morbid process more or less extensively invades the cord, especially at its peripheral portions, producing a meningomyelitis.

Symptoms.—The symptoms are those of the disease with which the meningitis is associated, in addition to fever and such other symptoms as are the result of vascular derangement and mechanical interference. These have already been detailed under cerebrospinal meningitis, including *pain in the back* of varying severity, *stiffness*, *sensitiveness of the spine*, symptoms of *irritation of nerve trunks*, and *disturbances of sensation*. The *reflexes* may be increased. Kernig's sign of "flexion contraction" at the knee-joint, described in connection with cerebrospinal fever, should be looked for. Examination of the fluid obtained by lumbar puncture is of diagnostic value.

Paralytic symptoms are a late and also a rare development. At such time the reflexes are sometimes diminished or abolished on account of the destructive involvement of nerve-roots or of the spinal cord. The urinary and bowel functions are sometimes deranged.

Diagnosis.—The diagnosis of simple acute meningitis in association with the infectious diseases should not be too hastily made, because of its simulation by these diseases. Such simulation is, however, less common with spinal meningitis than with cerebral. Here, as in cerebral meningitis, the etiological factor may help us out; while, on the other hand, given the disease, the special variety present cannot always be told. The tuberculous form is most easily recognized, because of possible pre-existing symptoms of the disease. Stiffness and pain in the back are not so distinctive as hyperesthesia and pain in distant parts supplied by nerves from the seat of special spinal involvement. Again, cases of spinal meningitis have been found on the autopsy table in which no symptoms were recognized during life.

Prognosis.—This is generally unfavorable in all forms.. Recovery may occur in the cerebrospinal form if Flexner's serum is used early, especially when the disease is secondary to the infectious diseases.

Treatment.—This is mainly symptomatic, and the details are those given under the head of cerebrospinal meningitis.

Chronic Spinal Leptomeningitis.

Etiology.—So rare is primary chronic meningitis that its existence as a separate disease may be doubted. It may, however, remain as a remnant of an acute inflammation, especially of epidemic meningitis. The possibility of its occurrence secondary to chronic disease of the cord, such as tabes dorsalis, is admitted, but it then almost never gives rise to symptoms. It is a possible consequence of syphilis and alcoholism.

Morbid Anatomy.—The distinctive morbid change would be a thickening and opacity of the membrane, and adhesions between the dura and arachnoid, localized or general. Certain white cartilaginous plates sometimes found on the posterior surface of the spinal arachnoid are not to be regarded as inflammatory.

Symptoms.—The symptoms would be those described in connection with the acute form, milder in degree and less definite. In fact, the diagnosis is rarely made. A long-continued, otherwise inexplicable stiffness in the trunk and extremities would justify suspicion.

Treatment.—This is symptomatic. Counterirritation would naturally be indicated if the diagnosis be made; Paquelin's cautery is the best instrument for the purpose. Antisyphilitic treatment should be used if syphilis be suspected.

HEMORRHAGE INTO THE SPINAL MEMBRANES.

Synonyms.—*Hematorrhachis; Meningeal Apoplexy.*

Hemorrhage may take place between the dura mater and its bony column, *extrameningeal*, or within the dura mater, intrameningeal. A third variety of spinal hemorrhage, *medullary*, into the substance of the cord, is described elsewhere.

Etiology.—Extrameningeal hemorrhage is almost invariably the result of trauma, such as concussion or fracture of the spinal column, puncture, or gunshot wound. The blood comes from the rich plexus of veins that surrounds the dura. A considerable amount of blood may be thus effused without compressing the cord. An aneurysm may burst into the spinal canal with fatal consequences.

Intrameningeal hemorrhage is rare, and is naturally more limited, as are the sources of the hemorrhage. Punctiform hemorrhages, such as occur in cerebrospinal meningitis, are possibly of little significance. Intrameningeal hemorrhages occur sometimes in connection with the infectious diseases, and William Osler observed two such cases in malignant smallpox, while they have been found after death from convulsive diseases, such as epilepsy, tetanus, and strychnin poisoning. So, also, in ventricular apoplexy blood in transit from the fourth ventricle into the meninges is not a very rare finding. Aneurysm of the basilar or vertebral arteries is, however, the most frequent cause of this form of hemorrhage.

Symptoms.—The symptoms in both varieties are those of pressure on

the cord, and may be slight and scarcely recognizable, or decided, with resulting paralysis and pain on the one hand, or anesthesia on the other. The symptoms are as sudden as is usually the event which causes them. Sometimes, however, the extravasation is slower and the symptoms are correspondingly gradual in their appearance. The absence of all cerebral symptoms from a complex including the above points to spinal rather than cerebral hemorrhage.

The extent of the paralysis and the other nervous symptoms depends on the seat of the hemorrhage. If in the lumbar region, the legs are alone involved, the lower deep reflexes may be absent, and the functions of bladder and rectum are impaired. If in the thoracic, there may be complete paraplegia, while the reflexes are retained, and there may be girdle pains. Herpes may be present. If in the cervical region, arms or legs may be paralyzed, and there may be pain or anesthesia in the upper extremities and neck. Embarrassed breathing, stiffness of the muscles of the neck, and even pupillary symptoms may be added when the hemorrhage is thus situated.

Diagnosis.—The diagnosis is based on the absence of brain symptoms in connection with the suddenness of the symptoms due to the disease and the history of possible cause.

Prognosis.—In certain cases in which the hemorrhage is small, contraction and absorption of the clot may take place, and the symptoms may pass away. In others the hemorrhage is fulminating and death follows early from involvement of the medulla oblongata in the pressure. In intermediate states there is corresponding improvement.

Treatment.—Conditions favoring the arrest of hemorrhage and the absorption of blood should be secured. Absolute rest is most important. If symptoms remain permanent, without aggravation, iodid of potassium may be used to promote absorption, and the usual measures intended to restore muscular and nervous power, such as massage, baths, and electricity, should be employed.

AFFECTIONS OF THE SUBSTANCE OF THE CORD.

General Considerations.

Two separate sets of pathological changes occur in the substance of the spinal cord. In one they are confined with marked constancy to certain *definite areas* which have precise functions residing in "systems of fibers," so that the clinical phenomena of the disease are exactly defined. These affections are called *systemic* diseases. They include such as tabes dorsalis, an affection of the posterior column; amyotrophic lateral sclerosis, a disease of the lateral columns and anterior horns. Why certain definite areas of the cord are especially involved, and why this peculiar selective systemic implication, we do not know any more than we know why certain poisons, such as curare, strychnin, and lead, select certain tissues for their operation.

In the second group there is no such limitation of area invaded, but the cord in its entire transverse section is involved in one large focus, or it is involved in several foci separated by areas of sound tissue. In this group

are included acute and chronic diffuse inflammations, the hemorrhages and traumatic lesions, multiple sclerosis, etc. These are the *non-systemic diseases*. Since, in the diffuse affections, all the parts involved in the systemic lesions are also affected, the symptoms of the latter are found associated with those growing out of the diffuse lesion. The diagnosis arrived at by a study of these symptoms is still, however, mainly a "topical" one, for it is an important fact growing out of the functions of the cord that all diseases involving certain areas produce the same symptoms, whence we infer the *seat* of the lesion rather than its nature or exact cause. This may, however, be determined with a varying degree of certainty from other symptoms.

A further peculiarity of all diseases of the substance of the cord is that its symptoms are commonly *bilateral*. This depends upon two causes: first, the fact that the two halves of the cord are in such close proximity that almost any cause of a violent kind, such as hemorrhage, affecting one half, must also extend its influence to the other; and, second, the cause of system diseases commonly select corresponding parts in two halves of the cord for their operation.

Again, symptoms vary according as the lesion affects the conducting path in the substance of the cord, to and from the brain, or a portion of the peripheral system of fibers within or without the spinal canal. The symptoms are accordingly known as "central," and as "root" symptoms.

SECONDARY SYSTEMIC DEGENERATIONS OF THE SPINAL CORD.

Very important in connection with nervous diseases is the subject of *secondary degenerations*. These succeed cerebral lesions and lesions in the spinal cord itself. They depend upon the fact, several times referred to, that a trophic influence is exerted by ganglion cells upon the fibers originating from them, so that the latter degenerate when the conduction of the trophic influence is interrupted or when the trophic ganglion cells are destroyed. For motor fibers such ganglion cells exist in two situations—in the motor areas of the cortex cerebri and in the anterior cornua of the spinal cord. The former exert on the motor fibers arising from them a trophic influence which extends down the cord as far as the latter. For sensory fibers in the cord the trophic influence resides in cells, probably on the posterior spinal root ganglia, and also ganglion cells in the posterior gray matter. The fibers of the lateral cerebellar column in the periphery of the cord arise in the cells of the column of Clarke, or posterior vesicular column—the group of cells in the inner part of the neck of the posterior horn.

Secondary Degeneration in the Spinal Cord after Cerebral Lesions.—If there be disease in the motor area of the cortex or in any part of the motor tract in the brain—that is, in the motor fibers of the corona radiata, the internal capsule, the crus, or the pons—interrupting conduction, a secondary degeneration of the motor fibers takes place below in the related pyramidal tracts, anterior on the same side of the cord, lateral on the opposite side, as far as the anterior cornua of the gray matter. In many cases there is slight degeneration in the lateral tract on the same side as far as the

lumbar region, showing that some fibers of each anterior pyramid find their way to the lateral tract on the same side. The relative proportion of the crossed lateral fibers and the anterior fibers that remain uncrossed varies within limits. In cases in which no anterior pyramidal tracts exist—that is, where all the fibers pass over to the lateral column of the opposite side—there is no descending degeneration of the anterior column.

Secondary Degeneration of the Spinal Cord after Transverse Lesion of the Cord Itself.—If a lesion be seated in any part of the cord affecting more or less its transverse section, the interruption of conduction in these fibers is also followed by secondary degeneration, which may be traced in two directions upward and downward, ascending and descending. Such lesions may be transverse myelitis, compression of the spinal cord, and tumors—any lesion, in fact, involving the whole of the cord.

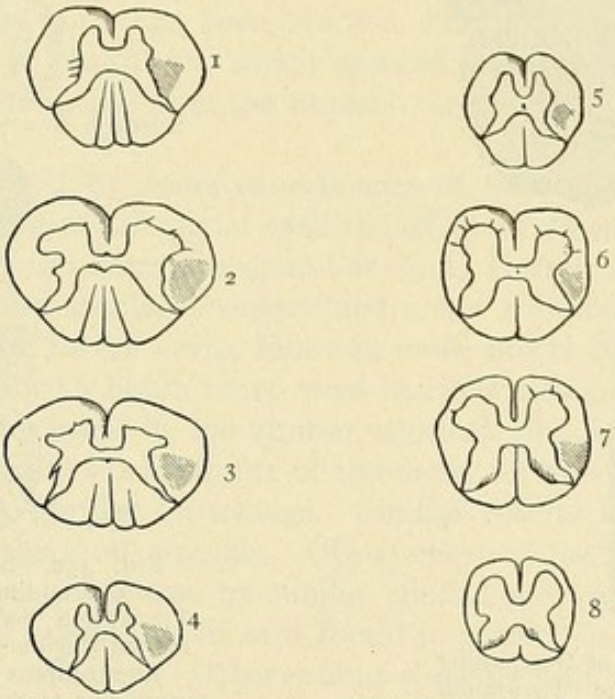


FIG. 148.—Secondary Descending Degeneration of the Pyramidal Tracts in a Primary Lesion of the Left Half of the Cerebrum—(*after Erb.*)

The lateral pyramid tract of *right* half is degenerated down to the lowest part of the lumbar region. 1–8. The anterior pyramid tract of left half is degenerated to beginning of lumbar enlargement.

The descending degeneration of the pyramidal tract is like the descending degeneration after cerebral lesions, except that after spinal lesions the degeneration of the pyramidal tract is usually more extensive; as the transverse disease affects the pyramidal tract on the two sides, the secondary descending degeneration will affect both lateral pyramidal tracts below the seat of lesion. The ascending secondary degeneration developing upward from the seat of lesion affects the columns of Goll—*i. e.*, the posterior median columns—and the columns of Burdach—*i. e.*, the posterior lateral columns and the lateral cerebellar tracts on the periphery of the lateral columns, because the conduction in those parts which receive their trophic influence from lower cells is interrupted. The ganglionic cells which act trophically on the fibers of Goll are probably in the ganglia on the posterior roots. The lateral cerebellar tracts share in the ascending degeneration,

because they are cut off from the cells of the column of Clarke, and when this occurs or these cells are destroyed, such degeneration may be traced upward into the restiform bodies. Fig. 149 shows secondary ascending and descending degeneration of the cord as occurring after transverse section in the upper thoracic region.

Clinical Effect of the Secondary Degenerations.—This is disputed, Charcot and some of the French clinicians ascribing to them the contractures and increase of the tendon reflexes in the paralyzed limbs of hemiplegia, while Strümpell and others think they have no clinical import. It is more prob-

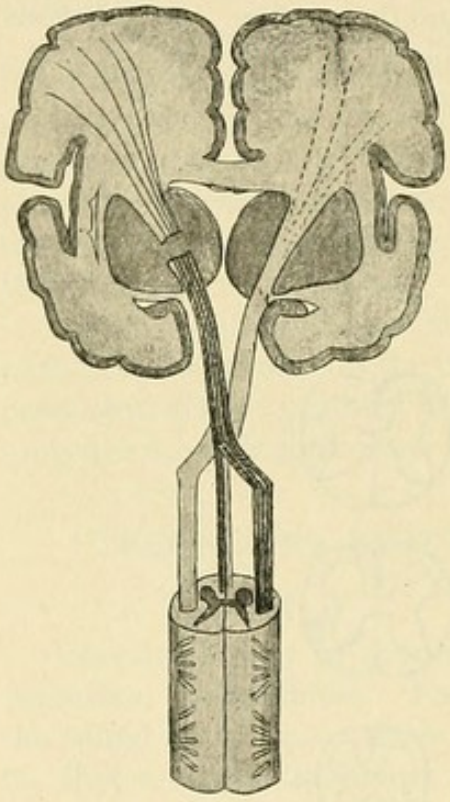


FIG. 149.—Diagram of Descending Degeneration of the Pyramidal Tracts Due to a Lesion in the left Internal Capsule—(*after Edinger*).

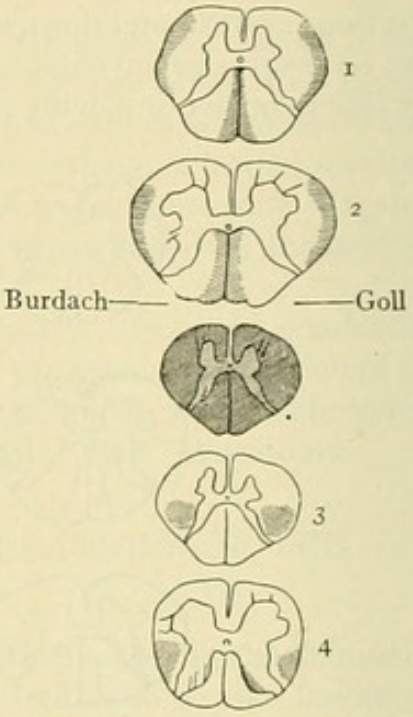


FIG. 150.—Secondary Ascending and Descending Degeneration in a Transverse Section of the Upper Dorsal Region—(*after Strümpell*).

The columns of Goll and the direct cerebellar tracts are degenerated upward, shown in 1 and 2, the anterior and lateral pyramidal tracts downward, as in 3 and 4.

able that the symptoms are caused by an interruption of the nerve-fibers and that the sclerotic tissue in the degenerated tracts produces no clinical signs of disease.

Secondary Degeneration in the Spinal Cord after Injuries of the Cauda Equina.—After fractures, caries, or other injuries to the lower lumbar vertebræ or sacrum producing injury to the cauda equina, or as the result of new-growths in this region, a secondary ascending degeneration takes place in the cord after the rupture of continuity. This is due to involvement of the posterior nerve-roots; whence the degeneration is confined to the posterior columns of the spinal cord, and in its distribution it resembles closely the state of the cord in tabes dorsalis. In the lumbar cord all the posterior columns are degenerated except a small median zone and the most anterior portion. The ascending degeneration grows smaller as we ascend, and finally is confined in the cervical cord to the regions of the columns of Goll,

which Include, in part at least, the prolongation of the fibers from the root zones of the lumbar and sacral cord.

Acute Affections of the Spinal Cord.

DISTURBANCES OF THE CIRCULATION OF THE SPINAL CORD.

CONGESTION.—From the standpoint either of clinical observation or postmortem examination but little is known of the phenomena of congestion of the cord as differentiated from inflammation. It is a well-known fact that active hyperemia may partly disappear after death. Congestion of the vessels is found under so many conditions that a diagnosis of inflammation based on this finding alone would not be justifiable.

ANEMIA of the cord has been studied clinically and experimentally. The phenomena of paraplegia which succeed profuse hemorrhages as of the uterus *postpartum*, and from the stomach, are fairly ascribable to anemia of the cord.

This is confirmed by some experiments of Stenson, who compressed the abdominal aorta of an animal with the effect of causing almost immediate paralysis of the extremities; and of C. A. Herter, at Johns Hopkins Hospital, in which paraplegia supervened a few minutes after the application of a ligature to the aorta, followed more slowly by paralysis of the sphincters.i Wthin 36 hours there were marked changes in the ganglion cells of the anterior horns in the lumbar segment, and, later, signs of softening. Within 14 days contracture of the limbs set in with atrophy of the muscles and with fibrillar twitchings. Similar results have followed the experiments of others on animals. Obstruction of the aorta by thrombi and emboli has been followed by similar clinical phenomena. In intense degrees of general anemia, such as is found in pernicious anemia, the cord is more frequently affected. Observations showing that the posterior and lateral columns are involved in pernicious anemia are numerous.

Embolism and *thrombosis* of the spinal arteries have been produced experimentally, with resulting *choreiform movements*. Embolism of the smaller vessels possibly occurs in connection with endocarditis. *Endarteritis* or its results are frequently found postmortem in syphilitic subjects as a nodular periarteritis or endarteritis, sometimes associated with gummatous tumors of the meninges; and as an *endarteritis obliterans* with thickening of the intima and consequent narrowing of the lumen, involving chiefly the arteries of medium and larger size. Sudden paralysis of spinal origin is likely to be from thrombosis of the vessels of the cord. *Miliary aneurysm and aneurysm* of the larger vessels of the spinal cord are very rare.

HEMORRHAGE INTO THE SUBSTANCE OF THE CORD.

Etiology.—Hematomyelia is a rare event. That it ever occurs primarily independent of disease is reasonably questioned. Its possibility must, however, be admitted, at least, as the result of traumatic casues, such as

falls. Great physical exertion is another possible primary cause; so are cold and exposure and tetanic and other convulsions. Repeated coitus is mentioned by Gowers as having been followed by hemorrhage in the gray substance at the top of the bulbar enlargement, and this cause may have operated in a patient of Tyson's who was suddenly seized with a paraplegia during one of a number of closely repeated acts of coition and while in vigorous health. Secondary hemorrhage is more frequent.

Morbid Anatomy.—The cord may be distended, infiltrated, or lacerated by the hemorrhage escaping into the meninges; if not too copious, the bleeding may be limited to the gray matter and may extend up and down the cord to a considerable extent. The blood undergoes the usual changes after effusion, *i. e.*, coagulates, becomes darker hued, then yellow, and, finally, at times, the seat of the hemorrhage is occupied by a cyst while numerous hematoidin crystals will be found in the residue. The blood may remain liquid for a long time.

Symptoms.—If the hemorrhage is in the lumbar enlargement or the thoracic region of the spinal cord, the effect is, as a rule, *sudden paraplegia*. If the cervical part of the cord is the seat of the hemorrhage, the arms as well as the legs are involved, and there may be embarrassed respiration, and possibly *sharp pain* in the extremities supplied by the nerves passing to the cord at the seat of the effusion. *Loss of sensation* follows later, while the reflexes also disappear. Softening often develops as a consequence of the irritative presence of the clot, and then follow its usual symptoms, including *trophic changes* and *fever*.

Diagnosis.—This is based upon the suddenness of the consequent events—acute pain and paraplegia—under the etiological conditions described, viz., trauma, and other causes. *Hemorrhage into the spinal meninges* is, of course, equally sudden or nearly so, but the symptoms of injury to the cord are less prominent, and there is little or no fever. In meningeal hemorrhage the pain is more severe and symptoms of irritation are more likely to precede the paralysis, while the paralytic symptoms are less persistent.

Prognosis.—The accident is rapidly fatal in the severest cases. In others paralysis may exist for a long time or may be permanent. In others there is slow but persistent improvement, and the patient may even recover. Hemorrhage into the cervical region of the cord is more serious because the center of the phrenic nerve which innervates the diaphragm is likely to be invaded. The presence of trophic changes renders the prognosis as to recovery more unfavorable.

Treatment.—This is identical with that for hemorrhage into the membranes. Absolute rest is the primary essential condition. Ice may be applied to the spine over the seat of the hemorrhage, and leeches or wet-cups in the same locality, although the benefit obtained by these measures is somewhat doubtful.

If the case is not immediately fatal, improvement is likely to follow the contraction of the clot, as in cerebral hemorrhage. Morphin to keep the patient quiet is indicated. Later muscular nutrition should be kept up by massage and electricity.

DIFFUSE MYELITIS (Acute and Chronic).

Synonyms.—*Myelitis; Transverse Myelitis.*

Definitions.—The line of demarcation symptomatically between acute, subacute, and chronic myelitis is not sharp, but the term *acute* is applied to that form of inflammation in which the symptoms come on suddenly. When requiring from two to six weeks for their development, it is called *subacute*. When a still longer time elapses before the symptoms reach a decided degree of intensity, it is *chronic*. At the same time it is plain that no very sharp line of demarcation can be drawn between these forms. When the whole thickness of the cord is involved to a small vertical extent—a common form—it is said to be *transverse;* if an extensive area, *diffuse;* when one small area, *focal;* when many foci, contiguous or distant, it is *disseminated*. Inflammation of the gray matter around the central canal, extending into the intermediate gray substance, is called *central* myelitis, which may be parenchymatous and interstitial.

Etiology.—The cause is often undiscoverable. There is an occasional hereditary tendency to it. It may occur at any age, but is more common in adult males. It may result:

1. From repeated exposure to wet and cold, or from overexertion, or from both combined.

2. Rarely from the acute infectious diseases, as smallpox, typhoid fever, or puerperal fever.

3. From syphilis, either as the direct result of primay infection, or secondarily from invasion of the cord by syphilitic tumors; the former appears within a few months or several years after the primary inoculation, the latter as a late manifestation, but a macroscopic gumma of the spinal cord is a very rare finding.

4. From tumors other than syphilitic.

5. From injuries to the spinal column, especially fractures, and from caries of the vertebræ. It is extremely difficult, nay, often impossible, to distinguish the inflammatory results of tumors and caries from those of compression; and, indeed, the symptoms due to tumors and caries are chiefly the result of pressure.

Morbid Anatomy.—The seat of invasion varies, the upper half of the thoracic cord being most frequently involved, but there may be cervical myelitis or lumbar myelitis. There may be a central focus and numerous small foci in the vicinity. The extent of the involvement varies at different levels. The softer reddish conditions indicate the more acute stage; the harder, grayer, and more contracted, the chronic stage—sclerosis.

Considerable experience is necessary to be able to recognize the changes in many cases of myelitis. The separation of the process into different stages is difficult or impossible. To the untrained naked eye the cord often appears quite normal. The expert examiner may recognize by touch that over a certain extent the cord may be either softer or harder and firmer. On section, the substance of the cord rises up more than in the normal state, the contour of the gray matter is less distinct, and sometimes has a hyperemic, reddish coloring, while the white matter is reddish-gray. There may be minute hemorrhages. The consistence may

be diffluent, constituting red softening. These foci of hemorrhage may give place to cavities. The gray matter may be involved throughout considerable extent. The meninges may be involved, producing myelomeningitis.

These changes become much more evident if the cord is allowed to remain in hardening fluid, and only after hardening can the lesions be satisfactorily studied.

Microscopic examination of the fresh cord reveals numerous *granular fatty cells*. *Blood-disks* and *leukocytes* may be present, the latter rarely in quantity to justify the name of pus or abscess. Thin sections stained by carmine give a very different picture even to the naked eye, the diseased tissues taking on the darker staining because of their greater richness in neuroglia. By the microscope it is found that in these portions the normal nerve tissue has partly or almost wholly disappeared. In some places axis-cylinders remain, possibly much swollen, and having lost only their medullary sheaths; in others the nerve tissue has disappeared. The *changes in the ganglion cells* are also definite; they have lost their processes and are rounder, or are entirely destroyed, while the *increase* of *neuroglia* goes on *pari passu* with the destruction of the proper nervous matter. The neuroglia occupies the enlarged meshes caused by the disappearance of the nervous elements. The cells of the neuroglia increase, and Deiters' spider cells may be numerous. The granular fatty cells may also be recognized, especially by osmic acid, provided no alcohol has been used in hardening. The blood-vessels are dilated and distended, and their walls are hyaline. The sum of these changes constitutes *sclerosis*.

In localized acute myelitis affecting the white and gray matter after injury the cord is swollen, the pia injected and soft, and on cutting the membrane an almost diffluent fluid may escape. In less degree the appearances first described are present. It is these cases which arise particularly by invasion from without or from compression in which the white matter is involved.

Localized areas of softening with blood accumulation constitute *red softening*. Abscess of the substance of the cord may occur, and at least nine or ten cases have been reported. Pus forms in the cord in considerable quantity only in purulent meningitis.

Symptoms.—The distinctive symptoms of myelitis may be preceded by constitutional disturbance, including *headache* and general *malaise*, and even *chill*, *fever*, and *delirium*. A temperature of 107° to 108° F. (41.7° to 42.2° C.) has been noticed. These symptoms are, however, unusual.

The characteristic symptoms vary greatly with the part of the cord involved, and no picture can be drawn to suit such differences of locality. Strümpell, therefore, will be followed to describe the symptoms more or less common to all localities, and, after this, such modifications or peculiarities of these as enable us to locate more precisely the process. The former include:

1. *Symptoms of Motor Paralysis.*—They are the most conspicuous and commonly the first recognized sign of developing transverse myelitis. Beginning with a tired feeling in one or both legs, followed by evident

weakness and then dragging, the paresis continues to grow until the patient is totally unable to make any active movement with his legs. This implies that the lateral columns of the cord, and especially the posterior part of the lateral columns, carrying the lateral part of the pyramidal tract, are involved, cutting off the motor impulses. In some instances this paralysis occurs very rapidly, and probably is from occlusion of the blood vessels of the cord. The motor paraplegia can occur in every form of myelitis—lumbar, thoracic, or cervical; but in the first two the upper extremities are intact. In the cervical, paralysis of the upper extremities also takes place. If one side of the body is involved more than another, it implies that one half of the cord is more intensely affected.

2. *Symptoms of Motor Irritation.*—These consist in spontaneous twitchings of the muscles of the paretic limbs, either rapid and short or slow and persistent. They occur at the beginning and during the course of the disease, and are variously severe. It is not always easy to distinguish them from increased reflexes, hence their diagnostic value is not great. Ataxia and intention tremor may occur in connection with involvement of the upper extremities and in the convalescence of acute cases, but they are very rare.

3. *Disturbances of Sensibility.*—These occur in marked degree much later in the disease than the motor phenomena. At the beginning there may be numbness, formication, tingling, and even girdle sensations, but severe pain is rarely present. When pain is present, it is an evidence of involvement of the vertebræ or meninges. In advanced stages, in addition to anesthesia, there may be paresthesia and striking hyperesthesia. The involvement of sensibility probably means that the whole transverse area of the spinal cord is intensely affected.

Disturbances of sensibility are useful in determining the segment of the cord involved because the lesion corresponds very nearly, or sufficiently so for practical purposes, to the level of the seat of the modified sensibility. Thus, in myelitis of the lumbar region the altered sensation extends nearly to the umbilicus, in the lower thoracic to the ensiform cartilage, in the upper thoracic to the level of the axillæ, while in the cervical the sensibility of the upper extremities is impaired. Total anesthesia is very rare, but is present when the transverse lesion is complete.

4. *The Reflexes.*—The effect of myelitis on the reflexes varies greatly with the seat of the disease and the degree and extent of the lesion. In the very incipiency of an inflammation of the cord we may expect all the reflexes, cutaneous and tendon, centering in the part involved to be increased, but with the progress of the disease the effect varies greatly and must be discussed in detail.

(*a*) *Skin Reflexes.*—The reflex arcs of the cutaneous reflexes are not definitely determined. Their connection with reflex inhibitory fibers from above is to be remembered, irritation of which fibers possibly diminishes, and interruption of which possibly increases, the sensitiveness of the reaction. In extensive lumbar myelitis the reflex path is broken and the cutaneous reflexes of the lower extremities are diminished, running about *pari passu* with altered sensibility. In thoracic and cervical myelitis the arc for the lumbar reflexes is intact, and if the reflex inhibitory influence is removed, these skin reflexes may even be increased. Experience shows,

however, that the skin reflexes in the leg may be diminished even in dorsal or cervical myelitis, in which event there must be loss of irritability in the fibers. The cremaster reflex has its arc at about the level of the first lumbar nerve, hence its loss means disease there. The lower abdominal reflex corresponds to the lower thoracic cord and the upper to the level of the fourth to the seventh thoracic vertebra. There is much need for careful study on the behavior of the cutaneous reflexes, as this subject is far from being fully understood.

(*b*) *Tendon Reflexes.*—The reflex arc of the patellar reflex lies at about the level of the second to the fourth lumbar segment inclusive. Hence the knee-jerk fails in disease of the lumbar cord involving the lateral part of the posterior columns and the anterior cornua of the gray matter. The ankle clonus probably has its reflex arc at the level of the first sacral segment. It is always absent in extensive disease of the posterior columns and gray matter of the sacral cord in this vicinity. *The absence of deep reflexes of the lower extremities is, therefore, one of the most valuable signs of myelitis of the lumbar cord.* In almost all inflammations *above* the lumbar cord—that is, of the thoracic and cervical portions—there is a decided *increase in the tendon reflexes of the lower extremity*, because these lesions destroy the reflex inhibitory influence. When, therefore, alongside of this it is remembered that the fibers which influence the condition of the tendon reflexes run chiefly in the *lateral* columns of the cord, we may conclude that the lateral columns are involved. In myelitis of the upper cervical cord the tendon reflexes of the upper extremities are often increased. It should be remembered that complete or nearly complete transverse lesion of the cord in the thoracic or cervical region may cause a loss of the deep reflexes of the lower limbs.

5. *Disturbances of the Bladder and Rectum.*—These are common in myelitis. There is, first, delay in micturition, finally accomplished by extra straining, but later all power to empty the bladder is lost—the *detrusor urinæ* is paralyzed. Still later the *sphincter vesicæ* is paralyzed, and then there is incontinence of urine. These symptoms occur in connection with paralysis in any part of the cord. The ultimate effect is almost invariably a cystitis, the result partly of decomposition, induced by germs introduced through repeated catheterization, even when most cautiously conducted, partly by the entrance of germs through the patulous sphincter. Such cystitis has also been ascribed to trophic influence. To the cystitis may succeed pyelitis and purulent pyelonephritis.

In myelitis there is at first obstinate constipation, followed by paralysis and incontinence of feces. This symptom does not give any information as to the seat of the myelitis. Defecation and micturition are sometimes reflexly aroused in abnormal degree when there is increased reflex irritability. Sexual functions, the centers of which probably reside in the sacral cord, are also often deranged in myelitis.

6. *Trophic Disturbances.*—These are most important symptoms, and valuable also in diagnosis. In cervical and thoracic myelitis the trophic centers in the lumbar cord are intact. The paralyzed muscles, therefore, do not atrophy excessively, though they may be somewhat softened from want of use. They retain their normal electrical excitability, or at least

the reactions are not qualitatively altered, although they may be quantitatively. On the other hand, genuine atrophy and the presence of the reaction of degeneration show that the anterior gray cornua or the fibers of the anterior roots of the lumbar cord are involved; in the upper extremities they show that the same portions of the cervical cord are involved. Bedsores are among the trophic phenomena, the possibility of the occurrence of which should always be vividly present. They are among the most unpleasant and most unmanageable symptoms, yet they may be guarded against; for, although favored by the deranged trophic influence, they require an exciting cause such as pressure, the irritation of secretions, or foreign substances to originate them. They occur over the sacral and gluteal regions, more rarely on the feet and inner sides of the knees. The total anesthesia often associated with advanced stages of the disease cooperates to permit the action of the exciting causes.

Other trophic effects often met are drying and hardening of the skin; glossy skin, also thick and brittle nails. Vasomotor disturbances also occur, producing congestion and mottling, and there may be slight edema of the paralyzed parts; also sweating, which may be localized. The temperature of the affected limbs may be lowered, and multiple arthritis may occur.

7. Disturbances in the area of distribution of the *cranial nerves* seldom occur, though bulbar symptoms are met in rare cases of cervical myelitis, when the process has extended to the medulla oblongata. Optic neuritis and pupillary changes, vomiting, hiccough, slow pulse-rate diminishing to 20 or 30, dysphagia, dyspnea, and syncope, have been observed in cervical myelitis.

By uniting the symptoms detailed and their mode of manifestation we may draw conclusions with a certain degree of positiveness as to the portion of the cord involved. The following table by Morton Prince in Dercum's "Text-book" will be helpful:

	Lumbar myelitis	Dorsal myelitis	Cervical myelitis
Paralysis	Paraplegia	1. Dorsal, abdominal, and intercostal muscles, according to height of lesion. 2. Leg.	Neck muscles, diaphragm, arms, trunk, and legs.
Sensation	Pains inlegs, or girdle pains around loins; hyperesthetic zone around loins; anesthesia of legs, complete or uneven distribution.	Girdle pain and hyperesthetic zone between ensiform cartilage and pubes.	Hyperesthesia and pains in certain nerve distributions of arms; below this, anesthesia of arms, body, and legs.
Atrophy	Of legs	Of dorsal and abdominal (and intercostal muscles, not subject to examination) corresponding to height of lesion; sometimes mild and slow of legs.	Atrophy of neck muscles (rare) or more commonly of arms.
Electrical reaction	Reaction of degeneration in atrophied muscles; or, in mild cases, quantitative diminution.	R. d. in dorsal and abdominal muscles; slight quantitative changes only in legs when wasted.	R. d. in atrophied muscles.
Bladder	Incontinence from paralysis of sphincter.	Retention, or intermittent incontinence from reflex action; later from overflow. Cystitis common.	Same as in dorsal myelitis.
Bowels	Incontinence from paralysis of sphincter, disguised by constipation.	Involuntary evacuation from reflex spasm, or constipation.	Same as in dorsal myelitis.
Reflexes, superficial	Lost	Temporary loss, then rapid increase.	Same as in dorsal myelitis.
Reflexes, deep	Lost	Temporary loss, then slow increase.	Same as in dorsal myelitis.
Priapism		Often present	Often present.

Diagnosis.—The difficulty of diagnosis is sometimes very great, because identical symptoms may be produced by other diseases, especially pressure diseases of the cord, such as are caused by tumors or hemorrhages, possible conditions which must be carefully sought.

Landry's acute ascending paralysis and *multiple neuritis* present some striking resemblances, and in some cases Landry's paralysis is due to myelitis. Both Landry's paralysis and multiple neuritis present rapidly progressive motor paralysis, but though sensory derangement may be a late development in myelitis, it is still a symptom belonging to it rather than to Landry's paralysis, which is a motor affection, while the trophic symptoms, the paralysis of the bladder and rectum, rapid wasting, electrical disturbances, and fever, pertain to myelitis. The resemblance to multiple neuritis is closer. In this, however, anesthesia is less complete, the wasting less rapid, and bladder and rectum involvement seldom present, and the parts affected are in nerve distributions.

Prognosis.—Almost all cases of myelitis are chronic after a more or less acute beginning, seldom lasting less than a year, often two or three years and even longer. The term acute is not, therefore, applied in its ordinary meaning, implying rapid course and early termination, but it is used to indicate cases which develop rapidly to their acme as contrasted with those that are slow. Even these cases become chronic. There are many who doubt the existence of a myelitis which begins in a chronic form. They hold that all so-called chronic cases of myelitis have an acute commencement. Rapidly developing cases passing to a fatal termination in from five to ten days may occur, but are rare. Certain cases, after reaching a given stage, remain as to symptoms *in statuo quo*, by which it is understood that the local lesion has healed, while function has not been regained because of the impossibility of restoring the normal structure of the cord. Remissions and improvements are less infrequent. Death is usually the result of exhaustion, although it may result from intercurrent disease.

Treatment.—This is for the most part to be directed to the relief of symptoms, no curative means existing beyond what nature herself provides. In cases with acute onset and pain, cups may be applied to the back. Even in syphilis it is thought by some useless to give the usual remedies, but it is safer in cases of suspected disease to give iodid of potassium in ascending doses to the extent permitted by the stomach, while the mercurial effect should be brought about by inunctions, 30 grains (2 gm.) to a dram (4 gm.) of mercurial ointment being rubbed in daily in different parts of the body, care being taken to select those parts having less hair. Salvarsan may be used. Tonics, such as iron, quinin, and strychnin, are useful in this as in other prolonged affections. Hexamethylenamine in five-grain doses three times a day must be given to keep the urine sterile.

The most painstaking attention must be given to the skin by bathing with alcohol and thoroughly drying after all washing, in order to prevent the excoriations which are often the beginnings of bed-sores, while the irritating effects of the excretions must be carefully watched, and if catheterization is necessary, it must be practised with the closest attention to

antisepsis. It may even be desirable to keep a soft catheter permanently in the bladder, to which a long tube is attached and the bladder thus kept drained. Should cystitis supervene, the bladder is to be washed out with aseptic precautions. When possible, the rectum should be emptied by enema rather than by purgatives, which should be cautiously used.

Electricity is elaborately directed by the German authors, although they admit that in the majority of instances it is principally a diversion to the patient. In the later stages, however, of the forms in which there is atrophy of the muscles, some advantage may be expected. The constant current is most highly commended, by large electrodes placed over the vertebral column, and a moderate stabile current or slowly labile current is passed for four or five minutes through the supposed seat of the disease. Peripheral galvanization or faradization of the paralyzed muscles and nerves should also be employed. Massage is useful, perhaps more so than electricity. The bladder may also be treated by electricity.

The bath treatment is carried out to various degrees of elaboration. The simple tub-bath with warm water furnishes the easiest form and may be quite useful, at a temperature of 85° or 90° F. (24° to 26° C.), in the cases with spastic symptoms. The baths should be at first limited to 10 or 15 minutes three or four times a week, and if well borne, may be increased to an hour daily. The water may be impregnated with sodium chlorid, using either the sea-salt or common salt, from 4 to 6 pounds of the former or from 5 to 10 pounds of the latter to the bath. When the patient is able to travel and avail himself of the actual sea-baths they may be expected to be beneficial. The Hot Springs of Arkansas and Virginia in this country may be resorted to. In Europe the thermal waters at Rehme and Nauheim in Hesse, Ragatz, in Switzerland, Teplitz in Austria, Wildbad in Wurtemberg, Gastein in Salzburg, Austria, and Wiesbaden in Baden are among those recommended; also the mud-baths of Carlsbad and Marienbad in Bohemia and Elster in southern Saxony.

ACUTE ASCENDING SPINAL PARALYSIS.

SYNONYM.—*Landry's Paralysis.*

Definition.—A symptom-complex first described by Landry, in 1859, characterized by an advancing paralysis beginning in the lower extremities, passing upward to the trunk and arms, and finally to muscles supplied from the medulla oblongata, including those of respiration; sensibility and bladder and rectum control remaining intact.

Etiology and Pathology.—It is most common in men between 20 and 30, and usually those who are strong and healthy. Cases have, however, been seen in children and old persons. *No anatomical lesions pathognomonic of the disease have been shown to be associated with it.* Hence attempts have been made to classify it elsewhere, and H. Oppenheim, James Ross, Neuwerk, Barth, and others regard it as a form of peripheral neuritis, Ross having found an interstitial form confined to nerve roots, while Neuwerk and Barth described a case confined to peripheral nerves. Other carefully studied cases failed to disclose such lesion. In some cases mye-

litis, especially poliomyelitis, is the cause. A toxic cause seems not unlikely. It is quite consistent with such cause that it should leave no local lesion, as well as that it should always seek the same spot. Gowers is especially disposed to ascribe the disease to such a cause. Some cases have, however, followed trauma.

Symptoms.—The characteristic symptoms are commonly preceded by a prodrome, in which *loss of appetite, general malaise, moderate fever, headache, backache,* and *tingling in the extremities* are conspicuous. These symptoms vary in severity and last from a few days to several weeks, when a *paresis* suddenly sets in, first of one leg and then of another, increasing rapidly, so that in a few days, sometimes in a few hours, an almost total motor *paraplegia* is developed. The paresis next extends to the trunk; in a few days or even less the arms are paralyzed. The muscles of the neck are next involved, and ultimately those of respiration, deglutition, and articulation, producing bulbar symptoms. Finally, facial paralysis and other disturbance of facial muscles may ensue. The paralysis is a flaccid one, and there is no tendency to spasm or resistance to passive motion. There is *not* usually a *change* in electrical reaction, although there is sometimes a rapid loss of faradic muscular excitability. The *reflexes are diminished* or absent, but the *muscles do not waste,* because death usually occurs before atrophy has had time to develop.

There is *no definite loss of sensation,* but in addition to the primary tingling referred to there is sometimes *hyperesthesia* and *muscular tenderness.* In other characteristic cases sensation is intact. More rarely there is a *blunted* and *delayed sensation.* The *special senses* are not affected, nor are the *bladder* and *rectum.* Sometimes there are *vasomotor edema* and *sweating.* The *spleen* has been found enlarged and slight albuminuria has been observed.

Diagnosis.—This is not always easy, the disease being simulated by *multiple neuritis, acute anterior poliomyelitis,* and ascending *myelitis.* All these may cause difficulty, and sometimes a distinction clinically and pathologically is impossible. The rapid motor paralysis, advancing from below, in the feet and hands, instead of from above, the absence of anesthesia, of wasting, and of electrical changes, are characteristic of *Landry's paralysis.*

Prognosis.—This is grave, and the possibility of a rapidly fatal termination, even in a few days, is to be remembered, the danger being from interference with the cardiac and respiratory functions of the medulla oblongata. Other cases terminate similarly in three or four weeks. If, on the other hand, the acute stage passes off, the symptoms of paralysis may cease to extend, and recovery is possible and has occurred in some cases.

Treatment.—The patient should be put to *bed* immediately, and *counter-irritation* should be applied to the back by dry cups, and maintained by gentler means, as by a mustard plaster.

Of internal remedies the apparent results from *ergotin* and *mercury* justify their further use. Gowers relates a remarkable case of recovery under the use of the former drug, 20 grains (1.32 gm.) having been given in the course of a night in divided hourly doses. Likewise cases of syphilitic origin have been reported, in which the *iodid of mercury* has seemingly

proved of service. The biniodid may be given in doses of from 1/50 to 1/30 grain (0.003 to 0.006 gm.). The *salicylates* have been advised. Both remedies are indicated if its toxic origin be admitted. *Perchlorid of iron* is recommended in traumatic cases, especially when there is evidence of septic poisoning. It is doubtful whether any of these remedies will be of much service.

If *swallowing is difficult* the patient must be nourished by the rectum or through the nasal tube, and if symptoms of respiratory failure come on, If the acute symptoms pass away and paralysis persists, the usual application of galvanism and faradization may be made for restoring muscular and nervous power.

Chronic Affections of the Spinal Cord.

SPASTIC SPINAL PARALYSIS.

SYNONYMS.—*Primary Lateral Sclerosis; Spasmodic Tabes Dorsalis.*

Definition.—A chronic disease of the spinal cord, characterized by stiffness and weakness of limbs with greatly exaggerated tendon reflexes, but without atrophy or sensory or vesical derangement.

Etiology.—The etiology is not always apparent, although the causes are probably many. The cases mostly begin between the 20th and 40th years. It may occur in children. It has been traced to syphilis, several times to trauma, to acute infectious fevers and the puerperium; the diagnosis in such cases usually has been without anatomical confirmation. Strümpell has called attention to a hereditary family type, found in male members, between the 20th and 30th years, and in some families the symptoms are first manifested in early childhood. A form closely related, but resulting from arrested development rather than from atrophy of the central motor tracts, is the spastic paraplegia occurring in children born prematurely, and sometimes classed as one type of Little's disease.

Morbid Anatomy.—The lesion which would be expected in spastic spinal paralysis is degeneration of the pyramidal tracts. In point of fact this condition is found, but it is likely to be part of a mixed lesion which may include that of myelitis, meningomyelitis, multiple sclerosis, or compression of the cord by tumors or by caries of the vertebræ. In a few cases, however, the lesion almost uncomplicated has been found by Strümpell, Dejerine and Sottas and others.

Symptoms.—The conditions may begin with a sense of *fatigue* and *weariness* in the legs, but the two essential and predominating symptoms are *increase of the tendon reflexes and motor paresis*. The first is the more unmistakable, constant, and characteristic. In decided degrees of this increase the contractions come on even with that amount of tension on the tendons which is produced by the weight of the limbs or any active or passive movements, while the reflex muscular tension or rigidity opposes any attempt at motion. The muscles feel rigid and firm, and the legs are found in almost permanent extension, while the feet are in plantar flexion. Any attempt, especially if sudden, to flex the leg at the knee or the foot dorsally meets with resistance. Yet if slow effort is made,

flexion may generally be accomplished, the leg, while undisturbed, remaining in the position assumed, whence the graphic term of Weir Mitchell "lead-pipe contraction." If the thigh be placed over the edge of the bed, the traction of the leg on the quadriceps extensor may be sufficient to excite vigorous extensor tetanus and a convulsive tremor of the whole leg, like that of ankle clonus. If the patient is examined in a bath, the spasms are less violent because the effect of the weight of the legs is diminished. The superficial reflexes are also increased.

Walking is interfered with in two ways, first by the *stiffness* in the legs, and second by the *paresis*. The legs are only partially, if at all, flexed at the knee, and the foot is not raised, but is pushed along the floor in short, difficult steps. Owing to the contraction of the calf muscles the toes are brought to the ground, and thus the patient walks on his toes; sometimes an ankle clonus is developed by contact of the toes with the ground. The legs are kept close together, the knees touch, and in certain cases adductor spasm may cause cross-legged progression. Stiffness is not always so marked. The effect is the so-called *spastic gait*. In some cases there is no paresis, and the peculiarity of the gait depends purely on the muscular spasm. The effect is what Strümpell calls *pseudoparesis*, or spastic pseudoparesis. The absence of actual paresis is shown by the fact that, notwithstanding the stiffness in the gait, the patient can still walk some distance, even miles.

With all this, the *patient is well nourished* and there is no wasting of muscles, which may even be hypertrophied, and outside of these symptoms he may enjoy excellent health. *Nor is there vesical disturbance.* There is no *sensory derangement*. Ocular symptoms are rare.

The tendency is for the symptoms to become gradually worse, but very gradually; finally the patient cannot walk at all, nor can he stand. Rarely the muscles of the trunk and arms become involved, presenting also a paresis with decided increase in the tendon reflexes without disturbance of sensibility or muscular atrophy. Such is the picture of spastic palsy, rarely, perhaps, seen in an uncomplicated form.

Diagnosis.—As stated at the outset, there is absence of sensory and trophic disturbance. The onset may be sudden, but is never so in typical cases, with progressive loss of strength, but no emaciation. Spastic symptoms, with increased knee-jerk, appear, followed by gradually developing paresis. The arms are often affected, but less so than the legs, and may escape. The course of the disease is slow, and mental symptoms similar to those of dementia paralytica may be present at the close. It resembles amyotrophic lateral sclerosis, but it differs in the absence of muscular atrophy.

Hysterical spastic paraplegia may furnish in the most striking manner the symptoms detailed. Every symptom to be mentioned may repeat itself more or less identically. It is, therefore, not necessary to name them. Moderate wasting is sometimes added. It occurs more commonly in women, and usually careful examination will reveal some distinct stigmata of hysteria.

Prognosis.—Spastic paraplegia of all forms except the hysterical is of long duration with little prospect of recovery. The upper extremi-

ties are tolerably free from derangement, and the mind is usually clear. Hysterical spastic paraplegia may end in recovery, if properly managed. When the cause is transient, also, recovery may be expected with removal of pressure, as in caries.

Treatment.—If *caries* is present, mechanical measures should be used to remove pressure. If *syphilis* is suspected, treatment by *iodids* and *mercurials* or salvarsan should be employed. Mercurial inunction is the most ready way of bringing about mercurialism. *Galvanism* and *faradization* are less useful in spastic conditions of the muscle than in those in which nutritional changes are more decided, but in hysterical spastic disease they are of signal use for their moral effect. The electrical brush is here the most useful instrument. It should be associated with *massage* and *passive motion*, and early attempt at locomotion should be encouraged and a positively favorable prognosis made. These, at least, tend to defer the immovable stage.

In any case friction, massage, and forcible flexion may be of benefit, but should be used cautiously, as the irritation produced in this way may possibly hasten premature contracture. Hydrotherapy is commended. The effect of the prolonged warm bath at 90° to 95° F. (30.2° to 35° C.) is often an amelioration of the spastic symptoms. The bath should be kept up for half an hour and manipulation practised during it.

TABES DORSALIS.

SYNONYMS.—*Posterior Spinal Sclerosis; Duchenne's Disease; Locomotor Ataxia.*

Definition.—A disease especially characterized clinically by loss of co-ordinating power, and by sensory and trophic symptoms; anatomically it is pre-eminently a disease of the posterior spinal roots and posterior columns of the cord, although the brain does not always escape, and the optic nerves are commonly affected.

Etiology.—The etiology of tabes dorsalis is not a satisfactory chapter The disease is more common in cities, affects more men than women, is rare in the negro, and is pre-eminently a disease of middle life, about one-half the cases beginning between 30 and 40, one-fourth between 40 and 50, and less than one-fourth between 20 and 30. It has begun as late as 66, and occasionally before the age of 20. Direct inheritance, independent of inherited syphilis, is unknown.

Of the direct causes, syphilis is believed to be the most frequent. From 50 to 90 per cent. of cases have been ascribed to it by different authors, Erb and Strümpell leading with the latter figure. Möbius even believed that all cases of tabes are due to syphilis, a view which Mott shares. Yet there are difficulties in tracing the relation growing out of the facts, first, that the pathological product is not anatomically a syphilitic one, and, second, that it does not respond to the treatment of syphilis. A reasonable explanation of these difficulties is one which ascribes tabes to a toxin analogous to that of the paralysis which follows diphtheria, acting especially

on the centripetal sensory fibers. Indeed it may well be questioned whether tabes ever occurs without previous syphilitic infection.

The Wassermann reaction has helped to clear up the cause in many cases. A large percentage of the cases have given a positive reaction. The reaction is best made by using the spinal fluid instead of the blood.

Prolonged exposure to cold and wet, over-exertion, physical and mental, especially sexual excesses and alcoholism are possible contributing causes. Tuczek, has shown that in chronic ergot poisoning symptoms like those of tabes develop, and with them a lesion appears in the posterior columns of the cord.

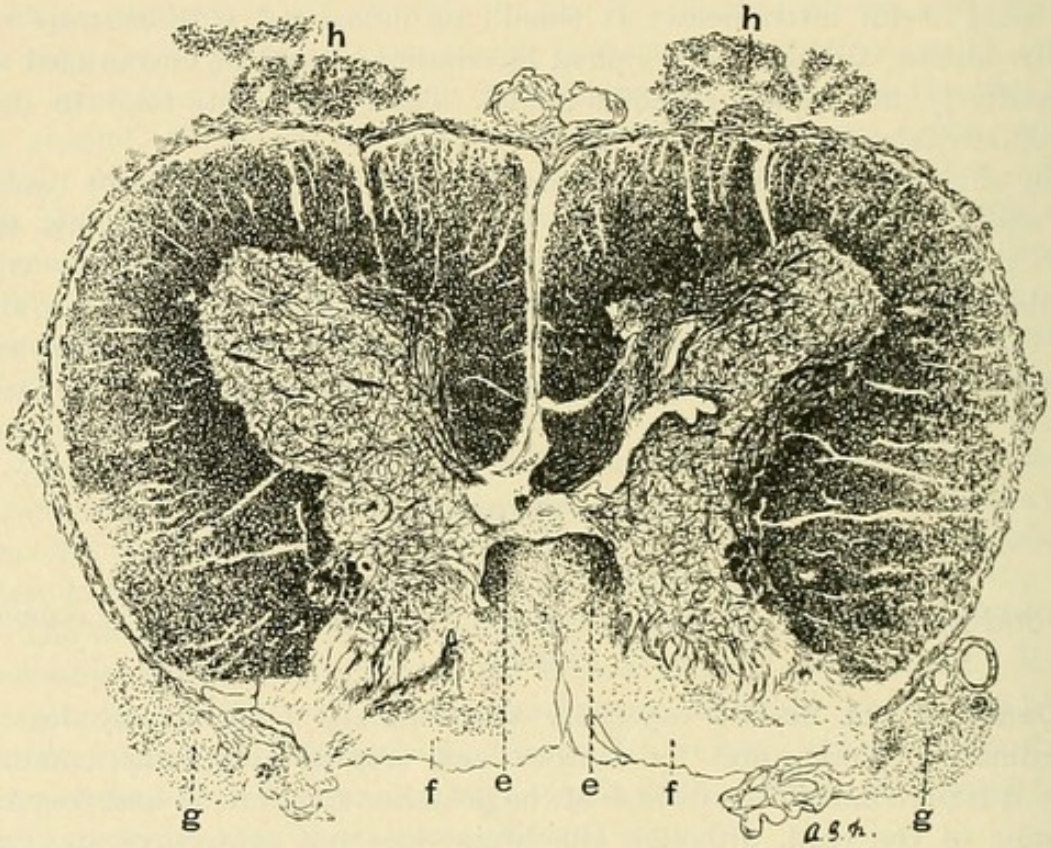


FIG. 151.—Lumbar Region. *g g*, degenerated posterior roots. *h h*, normal anterior roots. *f f*, degenerated posterior columns. *e e*, ventral fields of the posterior columns intact—(*after Spiller*).

Morbid Anatomy.—Tabes dorsalis is pre-eminently a disease of the posterior spinal roots and posterior columns.

Directing our attention to the spinal cord, in which are the most manifest changes, we find that, at times, even when inclosed in the membranes, it is noticeably small and thin, while through the pia we may see the *posterior columns* distinctly as a gray band throughout the length of the cord. The pia is, however, commonly thickened and opaque, especially on the posterior surface, sometimes more firmly adherent than is natural, while the blood-vessels also show signs of arterial sclerosis. The contraction of the posterior columns is more conspicuous on section. These columns are flattened instead of convex, while their gray translucent appearance is also evident, being due to the fact that the nerve fibers have been substituted by neuroglia tissue. Hence, also, the name "gray degeneration." In the cord hardened in Müller's

fluid the difference in hue is even more striking than in the fresh state. The posterior cornua and the posterior nerve-roots are small and gray.

On minute examination, in transverse sections stained by carmine or other staining fluid, the affected areas are more conspicuous, because of the deeper staining of the sclerosed tissue, while all parts of the posterior columns are not equally affected. In the *lumbar cord*, which, with the lower thoracic region, is usually the most frequently and seriously involved, the change affects chiefly the middle and posterior parts of the columns, while the extreme anterior portion, the so-called ventral fields, remains intact. The sclerosis is commonly most intense in the part adjacent to the posterior cornua, into which the posterior roots enter, also near

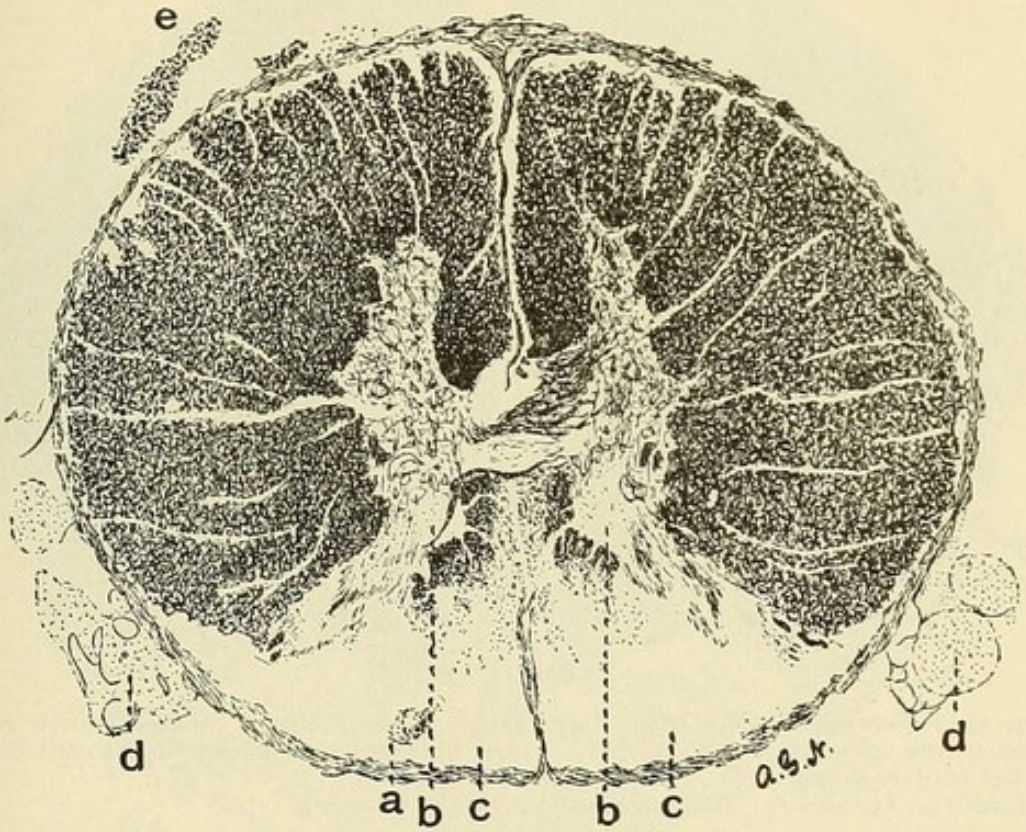


FIG. 152.—Thoracic Region. *d d*, degenerated posterior roots. *c c*, degenerated posterior columns. *b b*, degenerated columns of Clarke. *a*, small group of normal fibers from one or more posterior roots, lower in the cord, which were not entirely degenerated. *e*, normal anterior root—(*after Spiller*).

the surface of the cord. Ascending into the *thoracic cord*, the intensity of the disease in many cases gradually diminishes in the external parts of the posterior columns, and increases in their median portions. It presents in this way the distribution of an ascending degeneration, which in fact it is, receding from the commissure in the upper cervical region.

In the *cervical cord* the columns of Goll are chiefly affected, sometimes with the fibers in the root zones—that is, those portions of the columns of Burdach in which fibers enter directly from the posterior nerve roots, and from which fibers may be traced further into the gray matter of the posterior cornua; but two anterolateral areas in the columns of Burdach remain free from disease, at least for a long time.

Fig. 151 shows how the beginnings of the disease are localized in the

posterior columns. It is in consequence of involvement of the *posterior roots* that the corresponding posterior cornua into which they enter are also affected. The same is true of the medullated *fibers of Clarke's columns* (Fig. 152), which are also direct processes of the posterior roots, while the cells of the columns remain normal. Lissauer's tract, a narrow strip at the periphery of the posterior cornu, is early involved.

In advanced cases, in the *larger peripheral nerve trunks*, such as the sciatic, and in the finer branches of the sensory nerves, many degenerated fibers can be recognized. Some of these atrophies may be secondary, but modern clinicians are disposed to regard the peripheral degenerations of tabes as independent and primary, especially since, in addition to these, decided degenerative processes sometimes occur in the trunks of certain

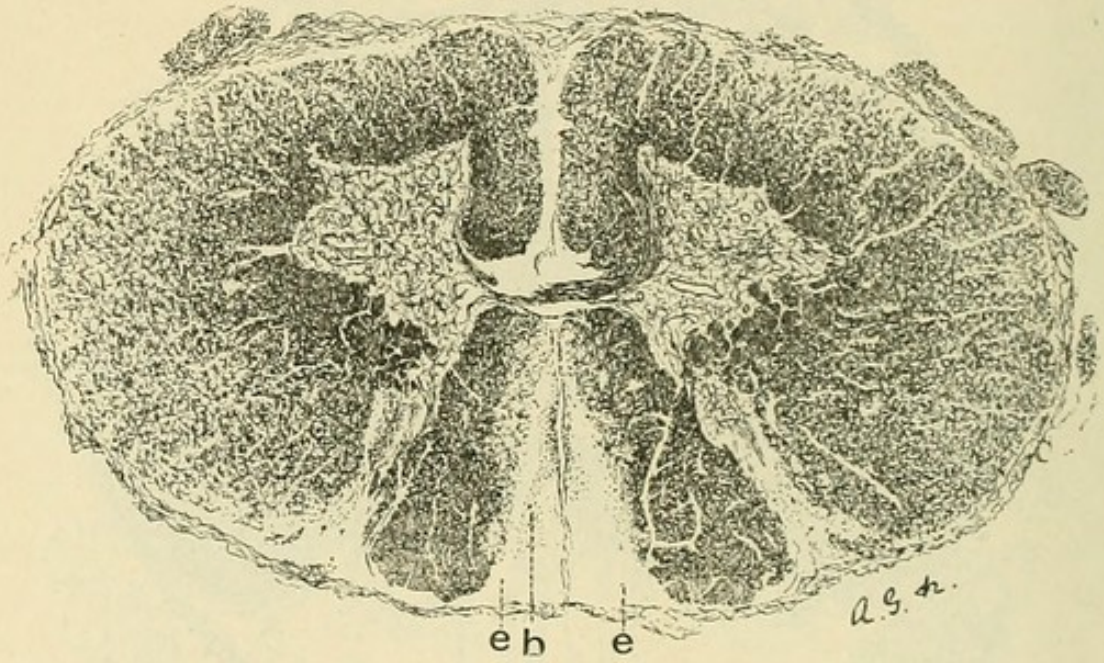


FIG. 153.—Cervical Region. The degeneration of the posterior columns is now nearly limited to the columns of Goll, *e e*. *b*, normal fibers from roots lower in the cord Figures were not entirely degenerated—(*after Spiller*).

Figures 151, 152 and 153, from an advanced case of tabes.

cranial nerves, such as the optic and oculomotor, and more rarely the vagus and auditory. They will be referred to in treating the diseases of special nerves.

Finally, there are even changes in the *brain* of various kinds. While the spinal ganglia on the posterior roots have been found invaded in a few cases only, it is the disposition of some observers to place the initial changes of the morbid process constituting tabes dorsalis in these ganglia, and thence the fibers ascending into the posterior columns. Thus considered, tabes dorsalis would be a general disorder of the central and peripheral nervous system, but limited mainly to sensory tracts, though motor ganglia and nerves do not altogether escape.

Other investigators place the primary lesion at the point where the roots penetrate the dura; and still others at the place where the posterior roots pass through the pia to enter the posterior columns of the cord.

Symptoms.—The characteristic symptoms of tabes are easily divisible into three sets: *motor*, *sensory*, and *reflex*. In addition to these there

are others not essential, but striking, including modifications of *special sense* and certain *visceral symptoms* characterized by pain and known as "crises." The special sense modifications include especially that of vision, while of "crises" the gastric are most striking.

The *motor phenomena* are usually the most prominent, whence the disease takes the name of locomotor ataxia, but this symptom may be absent for years, and hence the inappropriateness of the term. The distinctive symptom is a *loss of co-ordinating power* in the legs, having its simplest illustration in the unsteady gait of a drunken man. It is in-

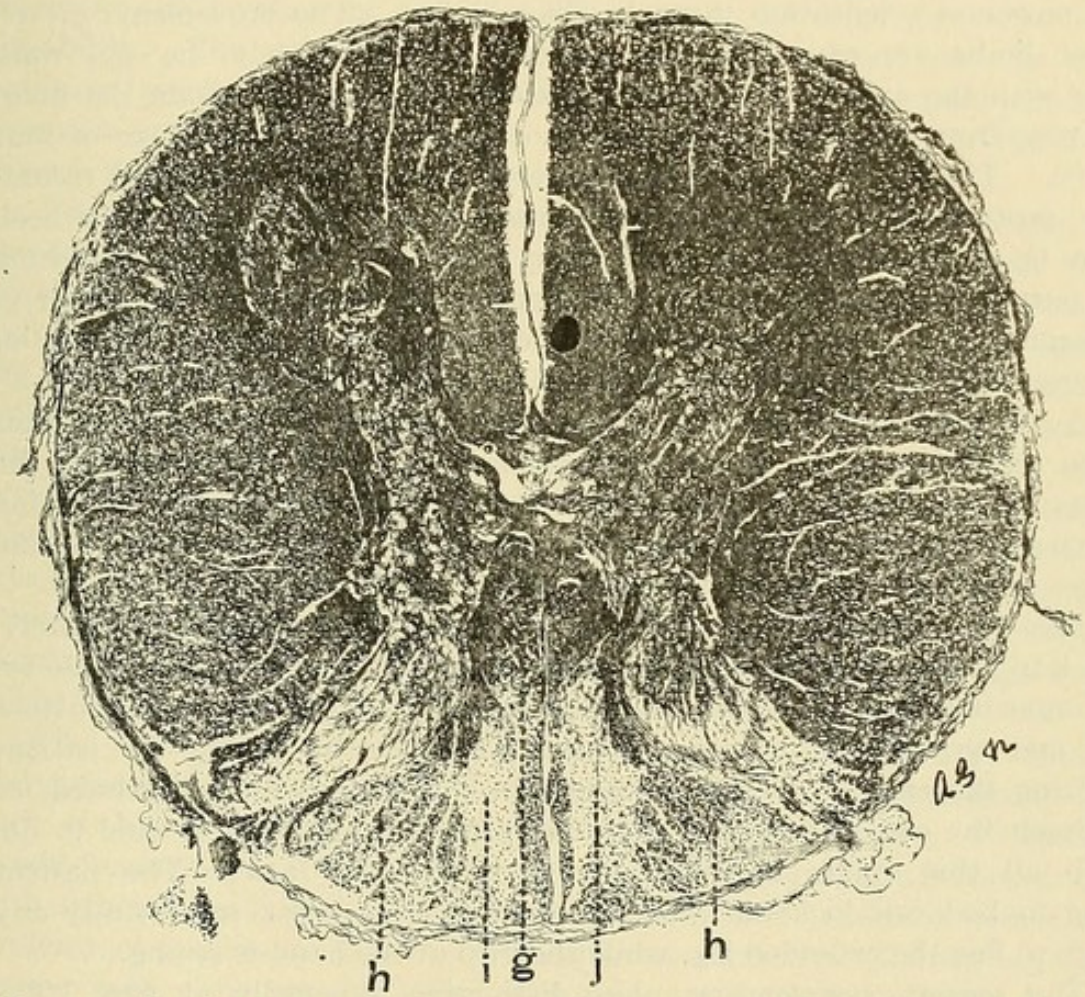


FIG. 154.—Lumbar Region. *h h*, posteromedian root zones (Flechsig) only slightly degenerated. *i*, middle root zones (Flechsig) degenerated. *g*, normal ventral fields. This section represents the earlier lesions of tabes.

Figure 154 should be compared with Figure 153—(*after Spiller*).

tensified when the patient attempts to walk with his eyes closed, and, indeed, in its early development does not appear except when the eyes are closed. It is usually *unaccompanied by a loss of power or muscular wasting*, but the latter may be extreme. On the other hand, *inco-ordination is by no means always the earliest symptom* and it may, indeed, never be developed, while there is usually a preataxic stage in most cases of tabes. The inco-ordination may be shown sometimes, before otherwise evident, by directing the patient to place the heels and toes together and then to close the eyes, when a swaying appears, as though the patient were going to fall—*Romberg's sign* or "tabetic swaying." This symptom is classed

by Strümpell among those of impaired sensibility in the soles of the feet and the muscles, whence follows defective control of muscular movements necessary to equilibrium. In health a slight unsteadiness under these circumstances is present which varies in different persons. In tabes the "sway" may be present even when the eyes are open. Certain symptoms often exist for a long time before being recognized by the patient. Soon the peculiar gait is noticeable. The foot is thrust forward too far, and brought down suddenly, with the heel first on the ground, with a stamp. This is the typical tabetic or "heel" *gait*. The patient cannot walk in a straight line, and the staggering becomes worse when the eyes are closed, because the power of orientation through vision is lost. The movements of the lower limbs are excessive and unnecessary. Ultimately, he can walk only with the aid of a cane or by keeping the eyes fixed upon the floor. He rises from the sitting posture with difficulty, often after three or four efforts. The loss of co-ordinating power may also be shown in the recumbent posture when the patient attempts to touch the knee with his heel, when he will carry it around and in front and behind without accomplishing his purpose. The ataxic gait is not confined to tabes, but may occur in disseminated sclerosis and cerebellar disease. In the latter closure of the eyes may not increase the ataxia of the gait, but frequently does.

A peculiar symptom called by Fränkel *hypotonia* is noted in this stage or earlier, viz., a peculiar muscular relaxation which permits the joints to be placed in a condition of hyperextension and hyperflexion which may give an appearance of backward curve to the lower limb. Sexual power diminishes or disappears early.

Inco-ordination also develops in *the hands*, but much more rarely, and late in the disease, though it may appear in them first. It is shown in connection with more delicate acts, such as picking up a pin, buttoning, and writing. It may be demonstrated also by asking the patient to bring the ends of two of his fingers together with his eyes closed, or to touch the end of his nose with one, which he may not be able to do. With all this ataxia the *muscular power remains intact*. The patient lying in bed can kick out with great force, and resist successfully any effort to flex the extended leg, while the grip of the hand is strong.

The *sensory symptoms* are less distinctive, especially at first. The most frequent of these—indeed, among the most frequent of all symptoms—are *pains* of a darting, shooting, or stabbing character, whence they are called *lightning-pains*. They are said to occur in nine-tenths of all cases. They resemble closely those of neuralgia, lasting but a second or two. They are most common in the legs, and are often accompanied by burning or tingling, especially in the feet. They may be felt in the trunk, arms, and even in the head. Commonly they do not correspond with nerves or affect joints. They are often considered by the patient as rheumatic pains. *A sensation of cold* is felt, also a feeling as though the limb were immersed in cold water. The pains are induced by fatigue or excesses or by temporary ill health from other causes, and are likely to come on at night. They may last hours or a day or two. There may be areas of *hyperesthesia and anesthesia*. A very curious sensation is felt in the soles of the feet when walking, a feeling as though *soft carpet or cotton*

were interposed between them and the floor. A painful sense of constriction about the limb or waist or around the entire trunk—*girdle pain*—is regarded as characteristic.

There are other disturbances of sensation, such as retardation of tactile, and more especially of pain sensation, wherein the prick of a pin, instead of being instantaneously felt, is *delayed* for several seconds. Another sensory symptom is difficulty in localization, manifested, for example, in referring a pin-prick to the right foot when it is made in the left—allochiria—or it may be felt in both feet—polyesthesia. In advanced stages the *muscular sense* also is impaired, and the patient is unable to indicate correctly the position of a limb. There may be other perversions of sensibility. The sense of pain may be lost or perverted; also the temperature sense—that, too, without derangement of the pain-sense or common sensibility. All varieties of sensation may be lost in the most diverse parts of the body and most irregularly.

Viscera pains, known as tabetic crises, among which the gastric are the most common, are also among the sensory phenomena. They may be laryngeal, rectal, nephritic, urethral, etc., and are sometimes exceedingly severe. The gastric crises are sometimes accompanied by vomiting of strongly acid gastric secretion. On the other hand, the vomited matters may be alkaline, the result of a reflux of the intestinal contents into the stomach. Nor are gastric crises limited to tabes. They may occur in other cerebrospinal disease, including general paralysis, multiple sclerosis, and subacute or chronic central myelitis. The laryngeal crises may be associated with spasm and dyspnea, with noisy breathing. Death is a possible termination from this cause. Rectal crises consist in paroxysmal pain and tenesmus, with a sensation as of a foreign body in the rectum.

The *reflex symptoms* consist in *impairment in reflexes* both tendon and cutaneous. The loss of the knee-jerk is one of the most frequent and early of these, occurring sometimes years before ataxia appears. Of itself it is not diagnostic, as it may be absent in healthy persons, but in association with lighting pains and certain ocular symptoms it is almost conclusive evidence of the disease. In by far the greater number of tabetics—at least 70 per cent.—the patellar reflex is wanting, with or without the Argyll-Robertson pupil. The skin reflexes fail *pari passu* with the loss of tactile sensibility, and it is doubtful whether they are ever present without this. The plantar skin reflex is that most frequently impaired, and after this are successively involved the gluteal, cremasteric, and abdominal. It happens rarely that in the early stages of the disease the skin reflex is increased, sometimes considerably, but even then the knee-jerk is absent or diminished.

Of the remaining symptoms the *ocular* are the most important. They include *ptosis* of one or both eyelids, producing a very striking appearance. It may be unaccompanied or associated with *external strabismus* and *double vision*. Rarely there may be paralysis of all the external muscles of the eye, producing *ophthalmoplegia externa*. The most remarkable eye symptom is, however, the *Ayrgll-Robertson pupil*, in which there is loss of reflex contraction of the iris in response to light, while the contraction in accom-

modation and convergence remains. According to Gowers, the loss of this reflex occurs in five-sixths of all cases. The contraction in accommodation is, however, not always maintained. Very rarely the reverse of the Ayrgll-Robertson pupil exists. Often the dilatation of the pupil which takes place in health when the skin of the neck is pinched cannot be produced and coincident with this is often unnatural smallness of the pupil—spinal myosis.

Finally, there is sometimes *atrophy of the optic nerve, producing the amaurotic form of blindness*. When it occurs, it is often an early symptom, usually commencing before inco-ordination; and, what is more singular, the ataxia often does not supervene—that is, there seems to be a tendency for the spinal malady to become stationary when the optic nerve is affected early, but in some cases the disease progresses notwithstanding early optic atrophy. The failure of vision usually begins with peripheral limitation and progresses slowly to total blindness, sometimes to a considerable extent before the patient notices it. Occasionally it ceases, and there may even be slight impairment.

Deafness may be present from disease of the auditory nerve; also, more rarely, *anosmia*, from atrophy of the olfactory nerve. Attacks of *vertigo* occur in some cases. Abnormalities in function of other cranial nerves may be due to similar involvement. Among these may be mentioned pain at one time and anesthesia at another in the area of the fifth nerve; also unilateral atrophy of the tongue.

There may be *delayed micturition* from weakness of the detrusor muscle of the bladder, or *incontinence* from paralysis of its sphincter, with partial evacuation of the bladder, and resulting cystitis. The anal sphincter is less frequently affected.

Vasomotor and trophic phenomena also occur, and may be predominating symptoms. They include local sweating of the palms and soles, or of half the head, edema, skin ecchymoses, herpes, and modified hair growth, loss of pigment from hair and skin, thickening of the epidermis of the sole, succeeded by blisters under it. Alteration in the nails, and onychia with ulceration, may be present; also decay of the teeth and the so-called *perforating ulcer of the foot*, which is almost peculiar to this disease. Only late in the disease may atrophy of muscles, sometimes associated with neuritis or involvement of the anterior cornua, occur. *Paroxysmal diarrhea* occurs, and has been regarded as vasomotor in origin.

The so-called *arthropathies* are an interesting trophic symptom and are directly the result of the disease. The most common is that known as Charcot's joint, anatomically similar to chronic affections in which the disease begins in the bone as contrasted with the synovial membrane, resulting in atrophy and in the destruction of bone and cartilage, while brittleness of bones, attended with spontaneous fracture or luxation, may occur. If union takes place, there is a superabundance of callus, with ossification or calcification of adjacent structures and of any newly formed inflammatory tissue. The large joints are those commonly affected and are painless when the seat of arthropathy. There may be effusion and even pus in the joints, but pus will not form unless the joint has been injured. The arthropathies may occur in the preataxic stage. They

may be excited by injury. The joints may also become greatly relaxed, while changes in the tarsal bones and articulations may cause the foot to become flat, with projection backward or inward of the tarsometatarsal articulations and of the tarsal bones, producing the "tabetic club-foot."

Cerebral symptoms also occur, but are rare, and may resemble those of *dementia paralytica*. It is not always easy to decide whether the dementia or the tabes is primary. The final stage of the disease, in which the patient is bed-ridden, is known as the *paralytic stage*.

Diagnosis.—The diagnosis, commonly easy when the characteristic symptoms are developed, may demand critical judgment in the early stage. The combination of lightning pains, absence of knee-jerk, early ocular palsies, including the Argyll-Robertson pupil, ptosis or squint, and ataxia are conclusive. Lightning pains and ocular palsies should always stimulate to thorough examination. The same is true of severe attacks of gastric crises in middle-aged men.

Differential Diagnosis.—Disease of the vertebral column with resulting compression of the spinal nerves is also associated with lancinating pain and absence of the patellar reflex, but the later symptoms are widely different. The same is true of deep-seated tumors impinging on the spinal cord.

Peripheral *alcoholic neuritis* and *arsenical neuritis* also may be associated with diminished knee-jerk, a pseudotabetic gait, and sharp pains, but the gait differs from the true tabetic gait, the leg being lifted high in order that the *toes* may clear the floor. The pain also follows the course of the nerves, which are tender on pressure and there is none of the shooting character. Nor is there reflex immobility of the pupils, and seldom bladder disturbance. The cases, too, are much more acute in character. *Multiple sclerosis* in rare instances presents similar symptoms, but defective speech, nystagmus, mental weakness, and ultimate apoplectiform seizures serve to distinguish it, and the tendon reflexes are usually exaggerated. In *diphtheritic palsy* there may be absence of knee-jerk, but the history of the case, the throat palsy, and all absence of pain are distinctive. *Ataxic paraplegia* also displays ataxia, but here symptoms of implication of the pyramidal tracts are present. In *cerebellar disease* there is also loss of co-ordination, and the knee-jerk may be absent, there may be headache, optic neuritis, and vomiting, but no lightning pains or sensory disturbance. Occasionally neuritis may present a clinical picture closely resembling tabes, known as peripheral pseudotabes. The rapidity of development, the absence of the Argyll-Robertson sign, and of implication of the bladder, and in some cases recovery, are the most important differential features.

General paresis and *tabes* sometimes merge, the latter developing on the former, or the former on the latter toward the end. Rapidly developed ataxia with mental symptoms often resolves itself into general paresis. Yet acute involvement of the posterior columns may be possible, producing ataxia.

Finally, there is the *nicotin* tabes of Strümpell, who has twice met, in men long working in tobacco factories, a set of symptoms consisting in painful sensation, absence of patellar reflex, contracted pupil, with reflex

immobility and uncertain gait, differing, however, from tabes in the presence of tremor and marked increase in the skin reflexes, especially in the lower extremities.

Course and Prognosis.—It is generally conceded that in no case of thoroughly developed tabes has recovery occurred. The disease may, however, be arrested. This happens especially if optic nerve atrophy has set in early, after which ataxia may not develop further, while the other symptoms may subside. In most cases of the disease, however, the advance is slow but irresistible. The duration of the first stage, characterized by absence of knee-jerk, and by the presence of the Argyll-Robertson pupil and of lancinating pains, lasts from a few months to 20 years, or longer. The second stage—that of ataxia—from which, indeed, the patient often dates the disease if the initial symptoms were slight, may then supervene gradually or suddenly. Finally, the paralytic stage supervenes, to be soon followed by death. Tabes is believed by many to assume a milder type more commonly now than was the case 20 or 25 years ago.

Treatment.—While recovery from tabes dorsalis probably never occurs, much may be accomplished by treatment in arresting progress and relieving symptoms. There is no specific treatment, although this effect has been claimed for more than one remedy.

The supposed frequent causal relation between syphilis and tabes renders the antisyphilitic treatment appropriate in all cases in which such relation can be traced or where it is suspected. To this end *mercurials* are to be administered until the specific effect is produced. This is best accomplished by inunction, a dram to a dram and a half being rubbed into different parts of the body daily, to be discontinued when the gums are affected. After this the *bichlorid* may be given in doses of 1/24 grain (0.0027 gm.) three times a day, in association with the *iodid of potassium* in ascending doses if well borne, or the *biniodid of mercury* may be given in doses of 1/24 grain (0.027 gm.) three times a day. If this treatment is found effectual, the iodid should be continued in the minimum doses, which will keep up the effect. The antisyphilitic treatment is more successful in those cases in which cerebrospinal syphilis simulates tabes. It may be harmful in true tabes. The value of salvarsan in tabes is a subject of more or less dispute, in some cases much benefit has been reported from its use. At the meeting of the Association of American Physicians, May, 1913, Dr. Swift reported the use of Salvarsan in tabes was reported in the following manner. The spinal fluid was examined for the Wassermann reaction, the character and number of the leucocytes and the globulin reaction. The patient was given salvarsan intravenously. After a certain time he was bled. His own blood serum was then injected into his spinal canal. This was repeated many times. Many of his cases showed marked improvement. Iodid of potassium may be tried apart from the indications of syphilis. The rest *treatment*, originally suggested by Weir Mitchell, has been found useful in arresting the disease, but it has been followed by permanent results. *Extension of the spinal column* and presumably of the cord by suspension of the body for from one to three minutes daily was used for a time, among others by Mitchell, but it has been discontinued.

In Germany *electricity* is still a popular remedy, and failure with it in this country may be due to imperfect and too brief trial. Erb's directions for galvanism are to place a moderate-sized anode in the vicinity of the sympathetic in the neck, and a large kathode on the side of the vertebral column for four or five minutes, moving it at intervals from above downward. Severe pain and vesical weakness are treated by galvanization and the faradic brush. The latter, as recommended by Rumpf, should be brushed over the skin of the back and extremities for five or ten minutes, using a strong current. This, however, does good only for the symptoms.

Hydrotherapy likewise maintains its popularity in Germany, although claimed by some authorities to be sometimes harmful, especially in the shape of hot baths and vapor baths and wet packs. The *tepid bath* is entirely safe and often symptomatically useful. Its temperature should be from 80° to 90° F. (26.6° to 32.1° C.), accompanied by gentle rubbing. *Wet compresses* upon the abdomen or legs at night sometimes relieve the pains. In Germany, too, there are numerous water-cure establishments in the hands of experienced directors, to which patients may be advantageously sent, but, unfortunately, there is nothing of the kind in this country which can be recommended. Oeynhausen-Rehme in Minden has the best reputation for its carbonic acid thermal salt baths, but the baths at Nauheim in Hess are similar. Mud and iron baths are found at Pyrmont, near Brunswick; Driburg, in Westphalia, Prussia; Elster, pleasantly situated in Saxony; Karlsbad, Marienbad, and Frazenbad, in Bohemia.

The *painful attacks* are often not relieved by the measures thus far suggested, and require more powerful treatment. The first to be used should be *phenacetin*, *acetanilid*, *salophen*, *aspirin*, and *antipyrin*, while *morphin* should be deferred as long as possible. It may, however, be necessary, when it should be used hypodermically. *Cocain* used in the same manner in doses of from 1/6 to 1/4 grain (0.011 to 0.165 gm.) is also sometimes efficient, while *cannabis indica* in doses of 1/4 to 1/2 grain (0.0165 to 0.033 gm.) of the extract may also be tried. Bandaging with a broad flannel bandage from toes to thighs has been recommended for the sciatic pain and pressure for the relief of painful spots. Massive doses of strychnin have been suggested for the same purpose.

Fatigue of all kinds as well as anxiety of mind should be *avoided*, while moderate exercise may be encouraged. The bladder should be emptied at regular intervals even though there may be no desire for micturition. Vesical anesthesia may lead to retention. Excessive use of the eyes should be avoided, as reading by a poor light or in a railroad train. Excesses in smoking, and especially in the use of alcohol, are harmful, as is also too frequent sexual indulgence. Overeating and the use of indigestible articles of food should be avoided, as gastric crises are invited by them. Excessive use of iodid may produce pseudocrises.

Great benefit has been obtained by the Fränkel movements. They are "based upon the education of the central nervous system by means of repeated exercises, whereby it is enabled to receive sufficiently distant stimuli from the limbs as to their position, although the available quantity of sensation is rather small. It is necessary, of course, that the movements be attempted and carried out repeatedly and with great attention." They

are too complex to be repeated here, and the student is referred to Frānkel's book.[1]

HEREDITARY ATAXIA.

SYNONYMS.—*Hereditary Ataxic Paraplegia; Friedreich's Disease.*

Definition.—A disease whose clinical features are especially ataxia and paraplegia, occurring in families and at an age much earlier than ordinary tabes, from which it differs also in the addition of peculiar symptoms associated anatomically with lesions in the posterior and lateral columns.

Etiology.—Its etiology is unknown. It is more common in males than in females, affecting 86 males and 57 females out of 143 cases collected by J. P. C. Griffith. Strümpell makes the opposite statement as to sexes, but other observers agree with Griffith. Of Griffith's cases, 15 occurred before the age of two, 39 before the age of six, 45 between six and ten, 20 between 11 and 15, 18 between 16 and 20, and six between 20 and 24. Cases have followed the acute infectious diseases.

Morbid Anatomy.—The whole cord is smaller than in health. There is a decided degeneration of the posterior and lateral columns, and the degeneration in the posterior columns may extend throughout the cord, involving the whole of Goll's column and nearly all of Burdach's, leaving a narrow band of normal tissue near the posterior cornua. Different opinions are held in regard to the condition of the posterior roots. The degeneration of the lateral columns involves the area of the crossed pyramidal tracts, the direct cerebellar and Gowers' tracts as well as the column of Clarke with degeneration of its ganglion cells. The pia mater over the posterior columns is sometimes thickened.

The disease seems to consist of a double morbid process, consisting in early degeneration of nerve elements, associated with a tendency to overgrowth of interstitial or neuroglia tissue. According to Dejerine and Letulle, it is a gliosis of the posterior and lateral columns, due possibly to defect in development.

Symptoms.—The essential symptoms are *ataxia with p raplegia.* Initial pains are rare. The ataxia is, however, peculiar. As in tabes, it begins in the legs, but it is swaying and irregular, more like that of drunkenness, more like cerebellar inco-ordination. The feet are not often raised too high, and while there is stamping, as in true tabes, it is less marked. Tabetic swaying may or may not be present. If present it is not increased when the eyes are closed. Ataxia is present in the reclining position.

The ataxia of the arms occurs early and is striking, the movements being choreiform, jerky, irregular, and swaying. The hand first moves an object in its efforts to secure it and then pounces upon it. There seems to be a superabundance of effort in voluntary movements, action is overdone, and prehension is claw-like. Again, the fingers may be spread out or overextended. The first manifestation of the disease in children is often a tendency to fall.

[1] "The Treatment of Tabetic Ataxia by means of Systematic Exercise," Freyberger's Translation, Philadelphia, 1902.

As the disease advances, irregular, *jerky movements* affect the *head* and shoulders, sometimes tremor-like. In most cases there is *nystagmus* when the eyes are moved laterally or upward, usually a late, sometimes an early, symptom. *Atrophy of the optic nerve* is rare, and the pupils are normal. *Speech* is sometimes impaired, generally as a late symptom—three, five, or ten years after the initial symptoms. Syllables are elided—the speech is *scanning*—with occasional movements of the tongue, but no twitching of the lips.

The *paresis* is at first slight—indeed, the power of the muscles is at first unimpaired—while there is rarely ever total paralysis. Some patients, however, never walk. The nutrition of the muscles is good. The *knee-jerk generally disappears early*, or is at least absent when the cases come under observation. In a few this symptom appears late, while in some atypical cases this reflex has been reported increased. *Sensory* symptoms are *not* usually *conspicuous*. There may be none, even in bad cases. At times there is delayed sensation or impaired sensibility to pain and temperature. Increased sensitiveness may be present. *Visceral crises seldom occur.*

While trophic lesions of the usual kind are rare, there occur peculiar *deformities*, especially of the *feet*. There is talipes equinus or equinovarus, and the patient walks on the outer edge of the foot. The *great toe* is over-extended or dorsally flexed, and occasionally this is the first sign of the disease. There may be *lateral curvature of the spine*. The disease may last from 20 to 40 years.

Diagnosis.—This is not difficult, although sometimes the disease is confounded with *chorea*, with the hereditary form of which it has certain points in common. The ataxia in early life, the club-foot, overextended great toe, spinal curvature, lost knee-jerks, nystagmus and scanning speech form a complex of symptoms not found in any other disease.

It resembles *ataxic paraplegia* or combined lateral and posterior sclerosis in more than its symptomatology, but the increased knee-jerk, foot clonus, and spasms of the latter disease are wanting. In cases of combined sclerosis in which the knee-jerk is absent, the family history and youth of the subject can alone settle the question. The loss of iris reflex in children points to tabes, the result of inherited syphilis. *Disseminated sclerosis* presents incoordination, nystagmus, and defective articulation, but the knee-jerks are almost always exaggerated, and intention tremor is characteristic.

Prognosis.—This is invariably bad, so far as recovery is concerned, although the disease lasts many years.

Treatment.—There is no treatment except such as will overcome tendency to deformity. The remedies used in locomotor ataxia may be tried.

Cerebellar Hereditary Ataxia has been described by Marie, Sanger-Brown, Klippel, and Durante. It starts after 20 years of age. There are ataxia, disordered speech, nystagmus, and heredity, but the knee-jerks are normal or exaggerated, there is Argyll-Robertson pupil, optic nerve atrophy with limitation of the field of vision, while there is no scoliosis or club-foot. The opposite is true of hereditary ataxia. Many do not recognize the cerebellar hereditary ataxia as a distinct symptom-complex.

Progressive Interstitial Hypertrophic Neuritis of Childhood is also a family disease. The symptoms are a combination of those of tabes

dorsalis with those of neurotic muscular atrophy (peroneal type of progressive atrophy). There are hypertrophy and hardening of peripheral nerves. It was first described by Dejerine and Sottas.

Toxic Sclerosis, especially of the posterior and lateral columns, results from such diseases as pellagra, ergotism, and pernicious anemia.

ATAXIC SPASTIC PARAPLEGIA OR COMBINED SCLEROSIS.

Synonyms.—*Progressive Spastic Paraplegia; Combined Lateral and Posterior Sclerosis.*

Definition.—A chronic disease of the spinal cord, characterized by symptoms which point to lesions of both lateral and posterior sclerosis, including, therefore, both spastic and ataxic features, the symptoms of one lesion being more or less modified by the other.

Etiology.—This is obscure. It is more common in males, is a disease of adult life in which overexertion, exposure, spinal traumatism, and sexual excess each has been an antecedent event. Less frequently than tabes does it follow in the wake of the syphilitic taint. It may be associated with general paralysis of the insane. Heredity has been observed in one-tenth of the cases, and the neurotic constitution seems to favor it. It is most frequently associated with syphilis or anemia.

Morbid Anatomy.—As the name suggests, lesions are found in both posterior and lateral columns. In the posterior columns they resemble those of uncomplicated tabes dorsalis, and are most intense in the cervical and thoracic portions of the cord, variously distributed, sometimes equally, at others preponderating in one or the other. The changes in the psoterior root zones are less pronounced than in true tabes. In the lateral columns the crossed pyramidal tracts and in the anterior columns the direct pyramidal tracts are chiefly involved, though the mixed zones of the lateral columns, the lateral limiting layers, and the direct cerebellar tracts may also be invaded. The gray matter and membranes remain intact. Most cases are not truly systemic but are examples of diffuse lesions with secondary degeneration. This is especially true of the cases due to anemia and syphilis.

Symptoms.—The symptoms are slow in their development, though occasionally the course is more rapid, the only modification in this being that months instead of years are sufficient to develop the distinctive features. Those of either lesion may predominate at first. More usually those of ataxia are the first to appear, including fatigue and even pain after comparatively slight exertion, unsteadiness of gait, increased with the eyes closed, though an associated stiffness, may prevent the typical gait of tabes. There is also more or less paresis.

Sensibility is also diminished in combined sclerosis, but less so than in pure tabes. There may be dull pain or numbness in the lower extremities and in the back or sacral region, but the lightning pains of tabes are rarely present; nor is the girdle sensation, while visceral crises very rarely occur. The Argyll-Robertson pupil is, also, commonly absent, but nystagmus is not infrequent.

The most striking difference in the symptomatology of ataxic paraplegia, as contrasted with true tabes, is the presence of *exaggerated reflexes in the former*, including knee-jerk and ankle clonus. Simple tapping of the patella or the belly of the quadriceps extensor brings out the former. The upper extremities are also often involved, and the chief symptoms here are weakness, inco-ordination with exaggerated wrist- and elbow-jerks.

Electrical reactions are unaltered, at least in the early stages of the disease. With advance of the disease the features of a purely lateral sclerosis become very pronounced; those of tabes less so. Muscular paresis and rigidity become marked, and the patient is unable to leave his bed. There is no localized *atrophy* of the muscles, although general wasting is not uncommon in the late stages of the disease. Very rarely there may be atrophy of the optic nerve, the ocular muscles remaining intact. The sphincters of the bladder and rectum are sometimes involved; at others not, that of the bladder more frequently, producing difficult micturition. On the other hand, by rest and tonic treatment the spastic symptoms may be diminished, while ataxia remains unchanged. The mind remains normal.

Diagnosis.—This is usually easy, enough of the symptoms of each lesion being present to show the existence of a combined disorder. The *absence of co-ordination on the one hand and increase of knee-jerk on the other* are the two antipodal symptoms around which others of each lesion cluster. The presence of the Babinski reflex is regarded as distinctive of organic as distinguished from functional paraplegia. So it is in association with other signs indicative of organic disease. It is not, however, pathognomonic and may be found in pure functional conditions, as, for example, uremia affecting the brain. Its presence, when of the typical type—*i. e.*, when the upward extension of the big toe, especially, is slow—strongly suggests organic disease of the central motor tracts.

Then, as to differential diagnosis, *myelitis* may present similar symptoms. On the other hand, myelitis is usually a disease of sudden development, characterized by a rapid increase of symptoms as contrasted with the slower course of the disease under consideration. *Friedreich's ataxia* resembles ataxic paraplegia closely in its pathology, but the exaggeration of the tendon reflexes is rare and the spasticity is absent in the former. *Cerebellar tumor* may be mentioned with better reason as a disease which may be confounded, but in this headache, optic neuritis, and vomiting are peculiar, and while there is ataxic gait, it is the reel of a drunken man, and not the inco-ordination of tabes. So, too, there may be spastic symptoms in cerebellar disease, but they are less decided than in combined sclerosis. *Disseminated sclerosis* is a disease with which combined sclerosis may be confounded, and although it is the less pronounced forms of each which give rise to doubt, it is important to remember that the disseminated sclerosis has been found postmortem in cases which presented the clinical symptoms of spastic paraplegia during life. Whence it is not impossible that it may also present in its earlier stages symptoms of ataxic paraplegia.

Prognosis.—This is unfavorable as to recovery, but the disease is so slow in its development that death commonly results from intercurrent disease or from complications favored by the disease itself, such as disorders

of the urinary organs, bed-sores, and septic complications. The disease may be arrested for a time.

Treatment.—The treatment is mainly symptomatic: warm baths and a warm climate for the spastic symptoms; massage and exercise for the ataxic symptoms. Electricity and spinal stimulants like strychnin are contra-indicated as calculated to increase the spastic symptoms, while bromids and belladonna may be of service in controlling these. If a specific history can be traced, the disease should be appropriately treated by iodids or mercurials, and when anemia is present the treatment should be directed to the improvement of this condition.

SYRINGOMYELIA.

Definition.—A term applied to all cavities in the spinal cord, most of which are surrounded by an overgrowth of neuroglia.

Etiology and Morbid Anatomy.—The cavities are formed by defective closure of the central spinal canal or by the breaking down of residual embryonal tissue or of gliomatous tissue. The cavity of a syringomyelia is usually in the posterior part of the cord, extending toward the posterior cornua. It may prevail throughout the entire extent of the cord, but in most cases involves only the cervical or thoracic regions or more limited areas. The transverse section is oval or circular, but it may be fissure-like or quadrilateral, even irregular. On the other hand, a primary hemorrhage of traumatic origin, or even without trauma, may be the starting-point of a syringomyelia, and it has been supposed that such a hemorrhage into the spinal cord, occurring at birth from difficult labor, may later in life cause the symptoms of syringomyelia. So, also, compression of the cord due to fracture or dislocation may furnish the condition which will result in cavity formation. The cavities may be multiple. The term hydromyelia, applied to the forms in which the cavity is merely the dilated central canal, is falling into disuse, and there is no real difference between this and the other varieties. It is probable that hydromyelia may change into syringomyelia.

Symptoms.—The milder degrees are without symptoms and are often overlooked. Symptoms usually make their appearance about the period of adolescence. They are mostly gradual in development, and are partly the result of the secondary processes of distention which derange natural function. The symptoms are influenced also by the situation of the cavity, which is found most frequently in the cervico-thoracic region, whence the arms and neck are correspondingly affected. They depend also on the greater involvement of the gray matter of the cord.

The essential symptoms are modified *sensibility;* chiefly to *pain*, *temperature*, and to a *less degree* simple touch; also muscular *atrophy*, the latter progressive in development; and *trophic disturbances*. The sensory symptoms are the earlier and more constant. The sense of tactile impression may be lost by involvement of its path, which, as has been said, is not precisely known after it enters the posterior roots, though it is probably in the posterior and lateral columns. The comparative rarity of this involvement may be said to be due to the difficulty in destroying this path completely. Derangement of the senses of pain and temperature is

probably due to implication of the central gray matter, since it is through it that these impressions probably radiate to the white conducting tracts of the opposite side. The extension of the process to the lateral columns probably explains the derangement of pain and thermal sensations, in portions of the body below the level of the cavity in the spinal cord. There may not only be a loss of thermal sense, but it may be reversed in that heat is felt as cold, and *vice versa*. So, also, subjective sensations are felt, including heat and cold, or, in their absence, pain, which may be neuralgic in character and irregular.

The muscular atrophy is the result of injury to the motor cells of the anterior cornua from compression or destruction of these cells. This causes degeneration of the nerves and wasting of the muscles, and along with it is a lowered electrical irritability. There is also *muscular weakness*, involving the trunk muscles, and possibly to this is due the lateral curvature. If the legs are affected, it is generally from simple spastic paralysis from pressure on the pyramidal tracts, but sensory changes in the lower limbs occur. Great wasting of the legs indicates lumbar involvement, and the presence of ataxic symptoms points to involvement of the posterior columns. The remaining symptoms are not essential, but may be incidentally present from the action of the causes which usually produce them.

The *reflexes* may or may not be increased, and may in rare cases be lost, while *tremor* of the limbs has been noticed in some cases.

Trophic symptoms are not rare in the parts affected by sensory loss. The skin may be glossy and thin, or thick and horny, while there may be eczema, herpes, bullæ, and even ulceration and gangrene. The nails may become fissured and drop off. There may be deformity and absence of the end phalanges and lingual hemiatrophy. *Vasomotor* disturbances are more common, including coldness, lividity, or redness with swelling and heat. There may be sweating, brittleness of bone, and joint changes like those of tabes.

The area of the *cranial nerves* may be invaded when there is involvement of the medulla oblongata. The phenomena may include paralysis of one vocal cord, the tongue and face, difficulty in swallowing, of breathing, and embarrassed heart's action. The eyes may be disordered, and the pupils unequal, but the other special senses escape.

Diagnosis.—This is based upon the sensory symptoms, and of these thermo-anesthesia and analgesia rather than tactile insensibility, together with muscular atrophy succeeding after some interval. *Cervical pachymeningitis* causes like symptoms similarly distributed. J. Hendrie Lloyd, in an important paper,[1] has also called attention to certain traumatic affections of the cervical region of the cord simulating syringomyelia. Cervical pachymeningitis runs a more rapid course; the anesthesia includes all varieties of sensation and corresponds more nearly in its distribution to that of the muscular atrophy, pain is more conspicuous, and the reaction of degeneration is commonly present in the wasting muscles, and later, signs of compression of the cord are observed.

The symptoms of syringomyelia are sometimes simulated by the anesthesia and wasting of *anesthetic leprosy*, but in the latter disease the trophic

[1] Read before the Philadelphia Neurological Society, March 26, 1894.

changes are more marked, the phalanges often drop off, while the sensory symptoms include all varieties of sensation. The nerves may be enlarged and the leprosy bacillus may be found.

Progressive muscular atrophy differs in the absence of altered sensation. An intramedullary *spinal tumor* in the same situation as a syringomyelia furnishes almost identical symptoms, and may have an identical origin if it starts from the neuroglia, but the symptoms may be more rapid in their development.

The diagnosis of syringomyelia is sometimes exceedingly difficult to make, as the characteristic disturbances of sensation may be absent.

Prognosis.—This is ultimately fatal, although the course is slow, extending over a long period. Toward the end the course is more rapid, death resulting from exhaustion or interference with the functions of the medulla oblongata.

Treatment.—This can only consist in measures to combat symptons, and tendencies to them, such as cystitis, bed-sores, and the like.

Morvan's Disease.

Synonyms.—*Analgic Panaritium; Analgesic Paresis with Panaritium; Painless Whitlows.*

Definition.—This term is applied to a chronic affection described in 1883 by a Breton physician named Morvan, which is characterized by neuralgic pains, tactile and thermal anesthesia, analgesia, and painless destructive felons (paronychia). The disease is probably in most cases the same as syringomyelia; in some instances it is leprosy. Twenty cases were recognized in a population of 50,000 in Brittany. One or two cases have been reported in America.

Zambuco, of Constantinople, found in the broken-down matter of the syringomyelic cavity of what seemed a typical case, Hansen's lepra bacillus. In two well-studied cases reported by Marinesco and Jeanselme to the Société Médicale des Hôpitaux de Paris, February 12, 1897, the typical lesions were found, but no bacilli.

COMPRESSION OF THE SPINAL CORD.

Synonyms.—*Compression Myelitis; Pressure Paralysis of the Spinal Cord.*

Definition.—Under this head are included all forms of paralysis due to gradual compression of the cord from whatever cause.

Etiology.—A large number of causes may operate in the way indicated, among which are tumors or inflammatory new formations, including syphilitic products either in the membranes or outside of them, caries of the vertebræ, especially the form known as Pott's disease or tuberculosis of the vertebræ, cancer of the vertebræ, echinococci and cysticerci in the vertebral canal. Extraspinal causes may also produce erosion of the vertebræ and compression of the cord; among these are aneurysm of the aorta, retroperitoneal sarcoma, lymphadenoid growths, and sup-

purating kidney; also retropharyngeal abscess. Pott's disease is by far the most frequent cause.

Morbid Anatomy.—The changes in the cord as the result of compression are best studied in the compressions due to dislocation of the vertebræ in the breaking down of the bodies of one or more from tuberculous infiltration, or as the result of intrusion into the spinal canal of foci of cheesy pus from the posterior surface of the bodies of the vertebræ. Macroscopically, the cord is often smaller, softer, and sometimes bent. In old cases it may be harder. The term myelitis has been applied to the changes thus produced in the cord, but careful examination fails in most cases to find any of the usual histological products of inflammation, and the condition is one of softening. In the early stages the axis-cylinders are swollen, and fatty granular cells may be present. The nerve-cells undergo more or less alteration depending on the degree of pressure. At a later stage may be seen a secondary overgrowth of neuroglia, replacing the destroyed nervous tissue, first loose, later firm and fibrillated. After a certain duration there may be ascending and descending secondary degeneration of certain systems of fibers in the spinal cord.

Symptoms.—When tuberculous disease of the spine is the cause, the resulting deformity—kyphosis—is usually seen long before the symptoms of compression of the cord are present. On the other hand, when the erosion is due to aneurysm or growths within the thorax or abdomen, the subjective symptoms appear before the deformity, or more frequently without external deformity. The first of these symptoms is usually *pain* at the seat of the compression, which often does not amount to more than a dull ache, while at another time it is extremely severe. It is also aggravated by bending or straightening the body. Again, the pain is distributed along the course of the nerves, when the compression is exerted on the nerve roots. Previous to such pain and associated with it are *paresthesias* of various kinds, such as numbness, tingling, and formication. More rarely there is *impaired sensibility*, the same degree of pressure which deranges the function of motor fibers having often no effect on the sensory. Marked anesthesia is rare, and then only in the last stages. When the lesion is confined to the thoracic region, there may be *girdle sensation* and *pain* in the course of the intercostal nerves.

With the foregoing soon become associated *motor symptoms*, which may consist in *stiffness*, giving rise to difficulty in moving arms or legs, with peculiarity of gait, or there may be simple weakness or *paresis*, increasing to complete motor *paralysis*. These symptoms rarely affect both arms or legs at once, but rather first the upper and then the lower limbs if the lesion is in the cervical region.

The seat of the more pronounced sensory and motor symptoms varies with the segment compressed. Thus, when the caries is in the *upper cervical region*, between the axis and the atlas, or between the latter and the occipital bone, there may be *spasm of the cervical muscles*, the head may be fixed, and movements may either be impossible or extremely painful. Retropharyngeal abscess may be the cause of such a symptom, as in a case in the Montreal General Hospital mentioned by Osler, where movement was liable to be followed by transient instantaneous paralysis

of all four extremities from the compression of the cord. The patient died in one of the attacks.

If in the *lower cervical* region, there may be *dilatation of the pupils* from interference with the ciliospinal center or nerve-fibers arising in this center. There may be *flushing* of the face and ear on one side or *unilateral sweating*, *rigidity of the muscles* of the neck, while the sensory and motor symptoms described, if present, will be found more pronounced in the arms. The deformity of tuberculous caries is not always marked in this locality, but after recovery evidence of its presence may be found in a conspicuous *callus*, which may cause permanent rigidity of the neck. The cortical inhibitory influence being suspended, both tendon and cutaneous *reflexes* are increased, sometimes so markedly as to produce in the lower extremities a pronounced type of the spastic paralysis, with increased patellar reflex and ankle clonus.

When the *thoracic* and *lumbar* segments are involved, only the *lower extremities* suffer from the effect of compression; commonly the paresis is late, though rarely it may appear before the deformity of Pott's disease. Girdle sensation and pain in the course of intercostal nerves were named above as sensory symptoms of compression of the dorsal cord. Here, as elsewhere, motion is affected before sensation. As to the reflexes, since the reflex arc for the lower tendon reflexes is in the lumbar region, compression of the *thoracic* cord should produce an increase in them, and this is usually the case. On the other hand, they are diminished when the *lumbar* cord is compressed. If the lower thoracic and lumbosacral region is affected, the sphincters are apt to be involved, and there is, first, difficulty in micturition, then retention, and finally incontinence with cystitis, but the sphincters may also be involved from lesions higher in the cord. Yet all these symptoms may disappear, and recovery take place after many months' duration of the disease.

Trophic symptoms may be present in the paralyzed parts. These may include herpetic eruptions in the course of the nerves, at other times derangement of nutrition, manifested by bed-sores forming on slight irritative provocation, rapid shedding of the epidermis and brittleness of the nails. With the involvement of their trophic center the muscles may waste.

Diagnosis.—This is easy when there are evident signs of caries of the spine, manifested by prominence of spinous processes of the vertebræ and by tenderness on pressure. Repeated examination of the spine should be made. Nerve-root symptoms, or symptoms resulting from pressure of nerve roots, as they pass out between the vertebræ, are always significant. They include radiating pains, girdle sensation, and hyperesthesia or anesthesia, spasm and wasting. Stiffness on motion in separate parts of the spinal column is also significant. Root symptoms are said to be more common in cancer than in caries, but any of the symptoms named have increased diagnostic value if there has been cancer elsewhere, especially of the breast, and if the age exceeds 40. There is much more pain attending the paraplegia of cancer—whence the term *paraplegia dolorosa*, when the pain is referred to areas anesthetic to tactile and painful impressions. Such is the case whenever erosion is wrought from the abdomen outward, as by retroperitoneal growths or aneurysm.

Prognosis.—This is unfavorable in all cases except tuberculous spondylitis, which often terminates in cure, for, sooner or later, especially with suitable treatment, the tuberculous process may cease and the symptoms of paralysis disappear, although, of course, the kyphosis remains. However, even here, if the paralysis has continued for some time, it is likely to be permanent. In some cases death is from miliary tuberculosis, in others from the exhaustion incident to bed-sores, cystitis, and pyelonephritis.

Treatment.—Only when tuberculous spondylitis is responsible is there hope of cure. The treatment is general, by the usual measures found useful in tuberculosis, such as cod-liver oil and creasote or creasotol, with such tonics as iron and iodin, good food, fresh air, and mechanical appliances suggested by the orthopedic surgeon. These should be so adjusted as not to produce pain. Their object is to produce extension and thus relieve compression, and if this is not accomplished, they are useless.

Along with the extension, rest in bed is a most important measure, and in many cases arrest is obtained by such rest. Local measures, like counter-irritation and the hot iron, are of no use—rather harmful than otherwise. The same may be said of electrical treatment and massage, except so far as they are useful to keep up the nutrition in the paralyzed muscles. On the other hand, warm bathing is useful in relieving pain and allaying discomfort.

Operative treatment—laminectomy—has lately been practised with a good showing of result, and it should be considered, at least, after other measures have failed. Treatment should be persevered in, as recovery takes place sometimes after paralysis has long persisted, and in no form of tuberculosis has the general treatment previously recommended been so useful.

In the incurable forms anodynes must be employed to relieve pain, including even the hypodermic use of morphin, which should never be used without bearing in mind the possibility of the patient acquiring the morphin habit.

TUMORS OF THE SPINAL CORD AND MEMBRANES.

Both the membranes and the substance of the cord may be seats of tumors, while the cord may also be invaded from the spinal column by enchondroma or sarcoma, but the dura usually offers a successful resistance to tumors on its outer surface.

Varieties.—From the spinal column enchondroma, sarcoma, endothelioma and cancer may intrude into the canal. External to the dura mater in the extradural space occur fatty malignant and tuberculous tumors, while parasites are also found in this region. The extradural tumors may spring from the dura or from the tissue between it and the bone, or may arise outside and pass through the intervertebral foramina. Subdural tumors may arise from the inner surface of the dura, the arachnoid, or from the pia, and may include sarcomata, syphilitic, tuberculous, and parasitic growths. The last two are rare, but both echinococci and cysticerci have been met, developing in the meshes of the arachnoid. Schlesinger collected 44 cases

of ecchinococcus disease. When the parasite is intradural it is round or oval and compresses the cord. The dura is not usually implicated, merely distended. Of the 44 cases only five were intradural, so that the extradural location is seven times more frequent than the intradural. The cysts are usually on the posterior surface of the cord and in the thoracic portion of the vertebral canal, at least in the extradural variety. They may be the size of a pea, of a walnut, or even larger. Their contents are clear and they often contain daughter cysts. Their growth is usually slow. It is said that the hydatids are sometimes found in the substance of the bone.

Fatty tumors are also rare, but have been found and are probably congenital, because when found they usually are associated with spina bifida. In the cord itself occur tuberculous, syphilitic, sarcomatous and gliomatous tumors. Glioma and sarcoma are the most common. Some of these tumors spring from the pia mater, but tuberculous growths also develop in the gray matter. Some tumors are compound, as myxosarcoma, etc. Sarcomatous or carcinomatous meningitis occurs infrequently.

The size attained by tumors of the spinal cord and membranes is necessarily limited by

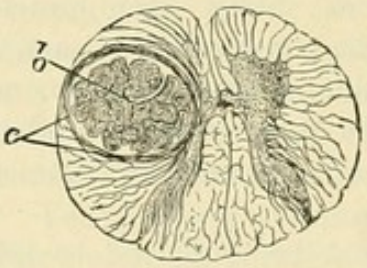


FIG. 155.—Sarcoma of the Lower Cervical Cord—(*Adamkiewics*).

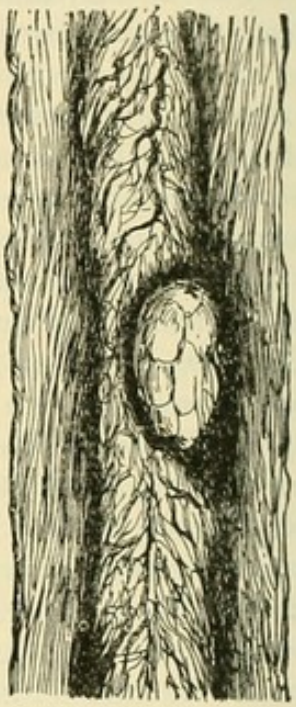


FIG. 156.—Sarcoma Compressing the Cervical Cord—(*E. Long Fox*).

the surrounding space. The largest do not exceed two inches (5 cm.) in diameter, and many are very small, not larger than a pea. They are usually single, rarely multiple. The so-called neuromata are usually fibro-neuromata. Tumors developing within the cord may lead to syringomyelia.

Symptoms.—These vary with the seat of the tumor and the degree of pressure exerted. When the latter increases slowly, the growth may reach quite a large size before serious symptoms occur. *Pain* is a frequent and conspicuous symptom, and is likely to be maintained by pressure on nerve roots which are in the way of the growth. The seat of pain varies with the course of the nerves impinged upon, and may be of every variety, such as "burning," "tearing," "stabbing," "aching," "girdle sensations," and the like. It may be unilateral or bilateral, and is worse, according to Horsley, when the tumor presses forward. Sometimes the pain is in the spine itself, which may also in rare instances be tender to pressure. When the growth is in the lower lumbar region, the pain may be referred to the soles of the feet, and may ascend from this seat. In other cases there is hyperesthesia of the skin, which may be associated

with pain felt at the level of the tumor, or pain may be felt in anesthetic areas. Very rarely pain is absent, chiefly in extradural lipoma.

Muscular spasm is also frequent, especially when the tumor springs from the membranes, when it may be very decided. There may be rigidity at the seat of the growth, most marked when the disease is at the more mobile parts of the spine, as the cervical region. Then there is apt to be pain in the vicinity, increased by motion. Spasm in the abdominal muscles may also be associated with *girdle pains*. *Contractures* may arise in the limbs, both those supplied by nerves directly irritated by the tumor and by those given off lower down. It is important to note the seat of the rigidity and its character, which may aid us in diagnosing the seat of the tumor, whether it is on the nerve roots or conducting tract of the cord. Thus, a tumor in one-half of the cord, in the cervical region, may cause persistent contraction of the arm and leg on the side of the growth, and in the early stage of thoracic tumors one leg only may be rigid at a time or one may be more so than the other. In the thoracic region the level of the pain is likely to be a little below the level of the growth, and the reflexes centering at this level may be lost, but retained in the legs.

Paralysis occurs sooner or later as constantly as pain, increasing gradually with the pressure. Paraplegia is more common, but all four limbs may be paralyzed by a tumor in the cervical region, one limb being usually affected before the other, though when the tumor is exactly central, both sides are affected simultaneously. Loss of sensation follows paralysis sooner or later. It corresponds in distribution to the motor palsy when the tumor is in the lumbar region of the cord, but if higher and on one side, the sensory loss may be greater on the opposite side; and the symptoms may be those already described under the head of Brown-Séquard's paralysis.

Atrophy follows involvement of the anterior cornua, and *vasomotor* disturbances may be marked. In cases of prolonged interruption *ascending and descending degenerations* may occur. Tumors not infrequently cause subacute or acute softening, whose symptoms may mask the clinical picture.

Diagnosis.—The characteristic symptoms are slow development of severe and constant unilateral root symptoms, later bilateral, at the level of the growth, and a progressive paralysis, motor and sensory. The radiating pain is usually at the level of the tumor or below. Pain in the spine itself is an important sign. Rigidity of the muscles of the spine, muscular contractions in the limbs, early and marked exaggeration of reflex action when the cord itself is involved, are also important signs, especially when associated with the history of syphilis or tuberculous disease. *Caries of the spine* may produce the same symptoms, but the radiating pains are less severe and the effects of compression of the cord are more likely to be bilateral, either from the first or soon after their commencement. Tenderness of the spine may generally be elicited by careful examination, while irregularity of surface, from the breaking down of the bone, sooner or later makes its appearance. When the *tumor* is *in the bone itself*, the symptoms at first scarcely differ from those of caries, though the pain on motion is usually worse in the former.

The symptoms of *cervical meningitis* also closely resemble those of tumor. They are, however, usually bilateral from the first and have considerable vertical extent. Central tumors covering a like area may produce identical symptoms, except that pain is usually unimportant. Pain and muscular atrophy in the arms without wasting occur in both extramedullary tumor and meningitis, but wasting is likely to develop later, and the diagnosis between the two conditions may be extremely difficult.

Chronic transverse myelitis also closely simulates tumor in its radiating pain, sense of constriction, progressive paralysis, and a differential diagnosis is sometimes impossible. The symptoms here, too, are from the first bilateral, while the radiating pain is commonly not severe in myelitis, which invades also larger areas of the cord.

Circumscribed serous spinal meningitis may closely simulate clinically tumor of the spinal cord. It has been found in association with necrotic ostitis of the vertebræ pachymeningitis, caries of the vertebræ, adhesions between the dura and pia, bony projection on the inner surface of a vertebra, and meningo-myelitis. A few cases are on record in which no complication seemed to be present. At operation or at necropsy a collection of clear fluid, resembling cerebrospinal fluid, is found in a cyst, the wall of which is made by the delicate pia. Nothing is really known as to the cause of this apparently idiopathic collection of fluid. Inasmuch as it is strictly circumscribed, it produces the symptoms of pressure upon the spinal cord, and cannot be distinguished clinically from spinal tumor. Removal of the fluid by operation may give complete relief from the symptoms, and there may be no tendency to recurrence. The recognition of the condition by surgeons is therefore of much practical importance, especially as the disorder is probably more common than the paucity of reports indicates. Sometimes the meningitis is circumscribed without cyst formation.

As to the exact seat of the tumor, in general terms it may be said that *when within the cord*, the symptoms are those of a gradually increasing paraplegia or of a Brown-Séquard's paralysis, while vasomotor disturbances are marked, and reflexes are bilaterally influenced, according to the law explained. Atrophy means involvement of the ventral cornua. Acute or subacute myelitis may be associated and complicate the clinical picture. Tumors in the *membranes* are characterized by early "root symptoms," including radiating pains, girdle sensation, and hyperesthesia or anesthesia. Irritation of motor nerves may cause spasm or wasting, with paralysis late in the disease.

The nature of the tumor may be inferred only from the history of the case, syphilis and tuberculosis giving the most valuable assistance. Its seat is suggested by the level of the transverse symptoms. It is never below these, while it may be a distance of three or four vertebræ above the nerves corresponding to the highest level of anesthesia or pain. The diagnosis of tumor from other transverse lesions of the cord may be at times impossible.

Prognosis.—In all forms the symptoms gradually increase until paralysis results, unless operative interference produces a more favorable termination—a practice which modern methods are rendering more frequent and justifiable.

Treatment.—When there is reason to believe syphilis is present, the antisyphilitic treatment may be used with reasonable expectation of success, as some cases of syphilitic meningitis simulate tumors. Beyond this, symptoms must be met as they arise. Attempts made of late years to formulate the laws governing surgical operations in these cases have been more or less successful, but wider experience is necessary before they can be thoroughly relied upon. We may, however, close this subject with the advice of Victor Horsley, whose studies on surgery of the nervous system entitles his opinion to the highest respect: "If it is clear that the growth is not syphilitic, and that no good can be done by other treatment, delay in an operation can only cause harm—can only result in a less favorable state for the proceeding, less chance of recovery, longer and greater suffering, and should, on every ground, be avoided."

LESIONS OF THE CAUDA EQUINA AND CONUS MEDULLARIS.

The *cauda equina* is the bundle of nerves coming off from the lower cord and occupying the spinal canal from the second lumbar vertebra downward. At this vertebra the cord itself terminates in the *conus medullaris*, prolonged into the thread-like *filum terminale*. Fractures and dislocations in the lumbosacral region may impinge on these parts, while the filaments of the nerves of the cauda equina may be invaded by tumors or compressed by cicatrices.

Symptoms.—*Compression* of the *conus* and of the *last sacral nerves* given off from it, such as may be caused by a dislocation of the first lumbar vertebra, produces paralysis of the bladder and rectum and loss of sexual power, whence it has been inferred that the anovesical center and the center for the sexual functions are seated in this part of the cord. This paralysis may be the only symptom or it may be associated with disturbance of sensation about the anus and in the perineum and external genital organs except the testicle, the latter being supplied with sensation from a higher segment of the cord.

When the *lumbar nerve-roots*[1] are involved, from the *second to the fourth* inclusive, there is paralysis embracing all the muscles of the thigh and leg except the outer rotators of the thigh, the flexors of the knee and of the ankles, the peroneal muscles, the long flexors of the toes, and the small foot muscles. There is also loss of sensation in the front, inner, and outer parts of the thighs and the inner side of the leg and foot.

Involvement of the fifth lumbar and first and second sacral nerves produces paralysis of the muscles just excepted, and loss of sensation in the outer and posterior part of the leg, foot, and sole of the foot. *Lesion of the third, fourth, and fifth* sacral and *coccygeal* nerves causes paralysis of the perineal muscles, the bladder, rectum, and of the external genitals, the coccygeus, with loss of sensation in the back of the thigh, anus, perineum, genital organs, and skin about the anus and coccyx.

[1] Of the lumbar nerves, the first root appears between the first and second lumbar vertebræ, the fifth between the last lumbar and the base of the sacrum. The four upper sacral nerves pass from the spinal canal through the sacral foramina, the fifth between the sacrum and coccyx.

SPINA BIFIDA.

SYNONYMS.—*Split Spine; Hydrorrachis; Myelocele; Meningocele.*

Definition.—A name applied to a congenital defect in the closure of the spinal canal, through which protrudes a sac-like portion of the dura containing cerebrospinal fluid, at times a part of the cord, either normal or altered, and forming also, as a rule, an external prominence of tumor covered by skin.

Description.—The tumor is found commonly in the lumbar and sacral portions of the spine, rarely in more than one place, very rarely throughout the whole column. Its size ranges from that of a small nut to that of an orange, and occasionally it is so large as to interfere with the birth of a child afflicted with it. On section of the skin the protruding sac of the dura is seen and beneath this the arachnoid. Rarely is the dura cleft so that the sac is formed by the arachnoid only. There may be a dilatation of the central canal—hydromyelia—when the substance of the cord is found more or less atrophied, while the central canal communicates directly with the cavity of the spina bifida. At other times the cord is normal, while its lower end may be adherent to the sac. A tumor of similar character is occasionally seen protruding through the skull.

Symptoms.—At first there are usually no clinical symptoms. By pressure the contents of the tumor can often be forced into the spinal canal, causing expansion of the fontanels and increase of cerebral pressure with its consequences—viz., somnolence, with changes in the pulse and breathing, which may be fatal if the pressure is continued. The absence of such symptoms goes to show that communication of the tumor with the spinal cord is cut off.

With the lapse of time the tumor usually grows slowly, and the effects of pressure on the spinal cord or cauda equina appear. These are paralysis, atrophy, anesthesia, bed-sores, vesical derangements, talipes varus, and trophic phenomena, of which perforating ulcer of the foot is one. The sac may burst, or the walls become inflamed, converting the contents into pus.

Prognosis and Treatment.—Unless removed by surgical interference, the child dies sooner or later of exhaustion. The tumor has been rarely obliterated by gradually increasing pressure or by injecting the cavity, after evacuation of the fluid, with iodin, producing obliteration through an inflammatory process. Other surgical measures may be found in text-books on surgery.

PROGRESSIVE BULBAR PALSY.

SYNONYMS.—*Polioencephalitis inferior chronica; Glossolabiopharyngeal Paralysis; Paralysis of the Tongue, the Soft Palate, and the Lips; Duchenne's Disease; Atrophic Bulbar Paralysis.*

Definition.—Bulbar palsy is a progressive paralysis invading the lips, the tongue, the palate, the pharynx and larynx, and in more advanced cases the lower face muscles due to lesion of the motor nuclei in the medulla oblongata (or bulb), whence arise the nerves distributed to those parts.

Etiology.—Primary progressive bulbar palsy is difficult to account for. It is more frequent in men, and sometimes heredity or family tendency is

noted. It has been ascribed to the overuse of the muscles of the mouth, as in the blowing of wind-instruments; to a tumor in the medulla oblongata or vicinity; while syphilis, to which so many of the unaccountable lesions of the nervous system are ascribed, is less commonly held responsible for this affection than for some others. Cold, emotional excitement, and extreme fatigue have all been named as causes. Most frequently, however, no cause is traceable.

Morbid Anatomy.—Most writers concede that the lesion starts in the motor nuclei of the medulla oblongata. It may be that the entire motor apparatus from the muscular fiber to the ganglionic cell is invaded simultaneously. Certain it is that bulbar paralysis is often associated both with progressive spinal muscular atrophy and amyotrophic lateral sclerosis, the symptoms now of one and now of the other preceding. There can be

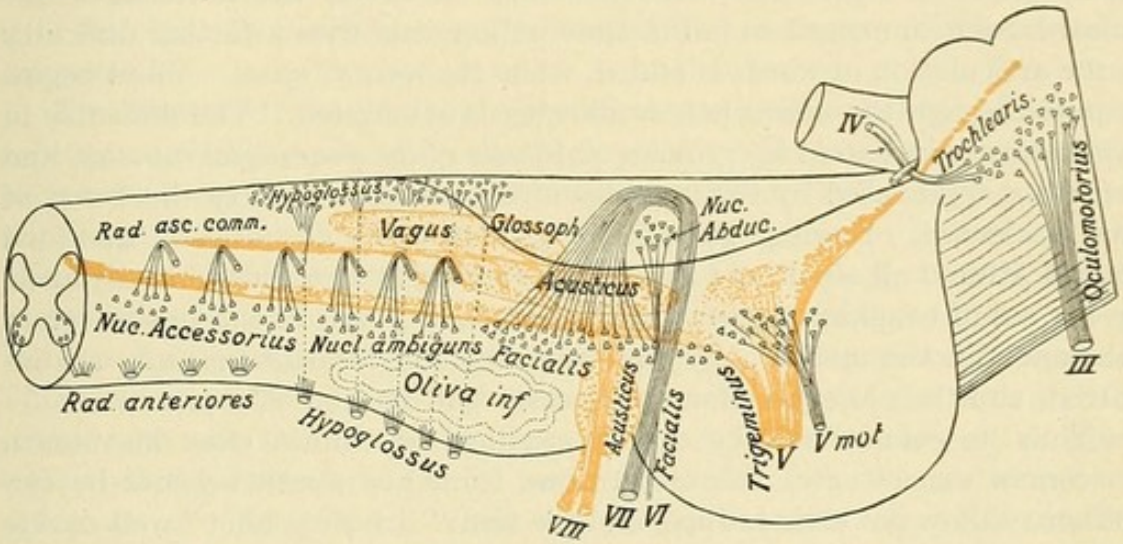


Fig. 157.—Situation of the Cranial Nerves—(*after Edinger*).
Cranial nerve nuclei, oblongata, and pons represented as transparent. Motor nuclei, black; sensitive nuclei, red.

no doubt that these three conditions are closely allied. The nature of the lesion is the same in each, the motor cells in each are involved, the muscles are wasted in each, though the particular ones involved vary as the situation of the motor cells is different.

The anatomical lesion is an *atrophy of the motor cells of the medulla oblongata*. The nucleus of the hypoglossus, the nucleus of the pneumogastric, to a less degree that of the facial and that of the glossopharyngeal are all involved, while the *sensory nuclei are intact*. Very rarely the nuclei of the ocular nerves, third, fourth, and sixth, are involved. From these nuclei the degeneration extends to the *nerves* which have their origin in them, and thence to the *muscles* to which they are distributed.

The nature of the degeneration is a more or less complete destruction of the motor cells. In addition, there is an overgrowth of neuroglia tissue and a thickening of the walls of the blood-vessels. The nerve fibers of the pyramidal tract may undergo degeneration.

Symptoms.—The symptoms of progressive bulbar paralysis are exceedingly gradual in their development. The first symptom noticeable is usually a *difficulty in the pronunciation of words* containing letters which require the use of the tongue in their formation, such as E, R, L, S, G (hard),

K, D, T, and N. Still later there is difficulty in pronunciation of words requiring the aid of the lips, as P, B, F, V, O, A (long), and the sound of O in tool, while whispering becomes impossible.

Symptoms.—Concurrently with these symptoms the *tongue and lips* are observed to *waste*, the tongue becomes thinner and narrower, the lips thin and compressed in appearance, the loss of power being commensurate with the degree of wasting. *Fibrillary tremors* are usually seen in the tongue, and the mucous membrane may be thrown into transverse folds. Finally, the *tongue cannot be protruded*, or can be brought only to the edge of the teeth, while the *mouth cannot be closed* because of complete paralysis of the orbicularis oris muscle. In more advanced stages other muscles of the face become involved, the labionasal fold is less distinct, and the face becomes expressionless.

Before this degree has been attained, however, the muscles of the *palate* have commenced to fail in their action, and thus a further difficulty in the articulation of words is added, while the *voice is nasal*. Fluid begins to pass through the nose when swallowing is attempted. The difficulty in swallowing is increased by growing *paralysis of the pharyngeal muscles*, and is further aggravated by the inability of the tongue to carry the bolus of food backward. Feeding the patient is a troublesome process, the food being scattered all about and sometimes thrown to a considerable distance, by the act of coughing facilitated by absence of power in the lips to retain substances in the mouth. By this time, too, the *laryngeal muscles* are involved, and the patient's efforts to speak result in mere grunts.

Thus he cannot talk, he cannot swallow, he cannot close his mouth he cannot expectorate, the saliva flows from his mouth because he can neither swallow nor close his lips, and the term "driveling idiot" well covers the impression caused by his appearance. Yet his mental powers are unimpaired, and may remain so until the last. The motor electrical phenomena in the muscles involved may be altered, and the *reaction of degeneration* may be present.

To these symptoms are to be added complications due to the paralysis. From the difficulty in swallowing, particles of food may enter the larynx, be insufflated to the deeper parts of the lungs, and there cause a pneumonia which may be fatal, or the fragment which enters the larynx may be so large as to cause death by suffocation.

In rare cases the lower distribution of both facial nerves is involved, producing *diplegia facialis;* but the upper distribution usually escapes. Or there may be paralysis of the ocular nerves to which it may be confined (anterior bulbar paralysis or progressive *ophthalmoplegia* of von Graefe). Even the muscles supplied by the spinal accessory and the motor branch of the trifacial may be invaded. In all these instances the nuclei of the corresponding nerves are affected.

Diagnosis.—The diagnosis is generally easy, the symptoms are so characteristic and so evident. For a typical case they must be purely motor; they must be disassociated from other muscular involvements which would go to make them a part of *progressive spinal muscular paralysis* or *amyotrophic lateral sclerosis*. If there are disturbances of sensation, paralysis of the upper division of the facial, of nerves of special sense, the

disease is not true bulbar paralysis. There must be some general involvement of the medulla oblongata, thrombosis, or embolism, a tumor developing near it or diffuse sclerosis through it.

There is a *glossolabiopharyngeal paralysis* of cerebral origin known as "pseudobulbar paralysis," in which there is partial or complete paralysis of the tongue and lips, due to bilateral and possibly even unilateral cerebral lesions. Close examination will, however, detect, sooner or later, deviations from the typical course, which include absence of fibrillary tremor, and of atrophy, and of reaction of degeneration. The symptoms tend, too, to occur first with the involvement of the limbs of one side and later of those of the other side. Mentality is much affected, and the reflexes may be exaggerated. Bulbar tumors run a like chronic course, but almost always present unilateral symptoms.

Prognosis.—The disease is invariably sooner or later fatal, although it is said that its progress may be delayed by treatment; this, however, is questionable.

Treatment.—If there be any suspicion that syphilis is the cause, *mercury*, *salvarsan* or *iodid of potassium* should be used. *Galvanism* is recommended, electrodes being applied to the two mastoid processes daily for two or three minutes, the current often reversed. The sympathetic nerve and the affected muscles of the lips and the tongue may be similarly treated, faradization being also substituted for galvanism in the case of the muscles. Deglutition may even be excited by galvanism when it begins to be impaired. This is accomplished by placing the anode on the nape of the neck and the cathode on one side of the larynx. At every cathodal closure, or every time that the cathode is carried across the side of the larynx, there is a reflex act of deglutition. When deglutition becomes very difficult, the stomach-tube should be used and nutrient substances thus introduced. Great care should be exercised in feeding the patient without the tube, lest the food pass into the trachea and cause suffocation. Hence, too, the use of the tube should not be too long deferred.

Iodid of potassium should be given in such doses as the stomach will tolerate, while salivation may be controlled by atropin—1/100 to 1/60 grain (0.00066 to 0.0011 gm.).

Acute Bulbar Palsy.

Etiology.—Besides the chronic or progressive form of bulbar palsy, there is an acute variety which is caused by hemorrhage into the pons and medulla, or possibly by thrombosis or embolism of the vessels supplying these centers—viz., tne anterior spinal, vertebral, and basilar. Inflammation of the medulla oblongata is also a cause, as in polioencephalomyelitis. Thrombosis may occur in any of the vessels, and is commonly due to atheromatous or syphilitic endarteritis.

Hemorrhage, thrombosis, and embolism are subject to the same causes here as elsewhere in the brain, but the cause of the inflammatory form of acute bulbar palsy is unknown. It is probably infection or intoxication and allied to poliomyelitis.

Symptoms.—In any event the symptoms are sudden. They are those already detailed in connection with progressive bulbar paralysis, but others

are added. There is usually no loss of consciousness, though there may be. There may also be deranged cardiac action and respiration, including irregular and frequent pulse, vasomotor derangements, and Cheyne-Stokes breathing. The temperature, normal at first, may rise to 105° to 107° F. (40.5 to 40.71° C.) and higher as a fatal termination is approached. Sensation is rarely affected. Most characteristic of all is the so-called *crossed paralysis*, described on page 932, which attends most hemorrhages into the pons, in which there is paralysis of the face on one side and of the extremities on the other; but the motor tract may not be involved, and in that case paralysis is not observed.

Diagnosis.—Suddenness of occurrence of the symptoms named indicates one of the accidents previously mentioned, while a crossed hemiplegia, provided it is of the limbs on one side and of the face on the other, is conclusive. When inflammation of the medulla oblongata is present, the phenomena of bulbar paralysis do not occur quite so suddenly. They may be several days or even a few weeks in developing, and may be preceded by prodromal symptoms, such as vertigo and painful sensations in the back of the neck. It must not be forgotten that certain cases of acute poliomeyelitis begin with bulbar symptoms.

Treatment.—The treatment is the same as for similar lesions elsewhere in the brain.

MYASTHENIA GRAVIS.

SYNONYMS.—*Pseudoparalytic Myasthenia; Bulbar Palsy without Discernible Anatomical Changes; Asthenic Bulbar Paralysis; General Profound Myasthenia; Erb's Disease; Hoppe-Goldflam Symptom Complex.*

Definition.—A disease beginning usually with weakness of the muscles of the tongue, lips, larynx, and eyes, followed by rapid exhaustion and temporary paralysis of the muscles of the extremities; by temporary recovery of power after rest; occasionally terminating in persistent paralysis.

Etiology and Pathology.—The disease occurs usually in those from twenty to forty years of age, and in both sexes alike. It is believed to be due to an autogenetic toxin. Congenital defect or abnormality either in the construction or mode of functionating of the neuro-motor apparatus, rather than in the muscles, has been suggested by E. Bramwell, and especially the lower motor-neuron. It has followed the infectious diseases, and in about one-fourth of the cases neuropathetic heredity has been noted. At necropsy no lesion was found for a long time, which would account for the symptoms, but more recently several cases have been reported in which a cellular infiltration of muscles was observed.

Symptoms.—These include ptosis, paresis of the facial muscles, difficult mastication, and difficulty in swallowing and talking. They are due to fatigue of the muscles involved, and the patient can talk a few sentences quite glibly, but his speech soon grows indistinct and ultimately incomprehensible. So with chewing and swallowing so far as the first mouthfuls are concerned, but these acts soon become impossible. The muscles of the extremities and trunk, as well as those innervated by the cranial nerves, are

involved, the same rapid fatigue supervening on effort. Thus one of Strümpell's patients could ascend a flight of stairs very well once, but in making a second effort had to invoke the aid of a bannister, while the third and fourth efforts were ineffectual. Such a condition is known as the *myasthenic* state. At times the abnormal fatigue and consequent symptoms are limited to the lower extremities. A similar effect succeeds on continued faradization of the muscles, first detected by Jolly, and is called the *myasthenic reaction*. Almost equally characteristic is the disappearance of fatigue after the muscles have been put at rest for a time.

Diagnosis.—In well-marked cases this is easy, but when the symptoms are less pronounced, there may be difficulty. Cases are often met, especially in hysterical women, who complain of inability to hold up the head, which clearly do not belong to this class. It is to be remembered that true myasthenia gravis is very different from hysteria. An ability to use the muscles at first, followed rapidly by loss of power must always be looked for, and these conditions may be applicable to the muscles of the lower extremities, as well as to those of the bulbar nerves. The absence of muscular atrophy is important in myasthenia gravis, though it has been reported in rare cases; the ocular and upper face muscles are more likely to be paralyzed in than bulbar palsy. The myasthenic reaction should be sought. The muscles respond normally to galvanism.

Prognosis.—This is not always unfavorable, but one must not be misled by the apparent improvement succeeding rest, which is often temporary.

Treatment.—It is evident from what has been said that rest is most important. Prolonged rest and the avoidance of mental excitement, and the use of massage and mild galvanization of muscles are recommended, and even central galvanization of the spinal cord and medulla oblongata. Galvanization of the respiratory muscles may produce unfavorable results. Since faradization excites the myasthenic state, it should not be used.

The nourishment, or mode of nourishment, is most important, in view of the fact that the muscles of mastication and deglutition are at fault. The food, therefore, should either be liquid or very finely minced, and unless deglutition is natural and easy, the stomach tube should be used, but great care should be exercised in its use for fear of producing exhaustion. Dark glasses may relieve the fatigue of the ocular muscles.

The drugs recommended are the usual ones: strychnin, arsenic, phosphorus, and other tonics, but no direct results have been traced to them. Calcium lactate seems to be of benefit and suprarenal gland may be tried.

AMYOTROPHIC LATERAL SCLEROSIS.

SYNONYM.—*Charcot's Disease.*

Notwithstanding the similarity of the clinical phenomena, and, to a certain extent, of the morbid anatomy of amyotrophic lateral sclerosis to those of the so-called progressive spinal muscular atrophy, to be next considered, there appears to me sufficient difference to justify a separate consideration.

Definition.—Amyotrophic lateral sclerosis is a systemic degeneration of the pyramidal tracts of the spinal cord, with atrophy of motor cells

in the anterior cornua and medulla oblongata, and consequent wasting of muscles, depending upon these cells for their trophic influence.

Etiology.—The causes of this condition are still essentially unknown. *Severe muscular exertion* has been assigned as a cause, as it has also of the allied affection, progressive spinal muscular atrophy. As in it, too, the *male* sex suffers most. It is a disease of *middle age*. It is probably due to the degeneration of an imperfectly formed central motor system. Some cases may be the result of syphilis.

Morbid Anatomy.—*A sclerosis of the crossed pyramidal tracts* in the two lateral columns and the *direct pyramidal tracts* in the anterior columns is essential to the morbid anatomy in a typical case. As important is *atrophy* of the corresponding *large ganglion* cells in the anterior cornua and medulla oblongata. The degeneration has been traced in the pyramidal tracts from the sacral cord upward to the pyramids in the medulla oblongata, sometimes even through the pons and crura into the internal capsule and central convolutions, in which, too, the large ganglion cells have been found atrophied. The nerve nuclei which are affected in the medulla oblongata are especially those of the vagus and hypoglossal nerves. The motor cranial nerves are sometimes degenerated.

The changes in the motor ganglion cells of the cord and the nerve nuclei in the medulla oblongata are analogous and produce corresponding results in the *muscles* supplied by the motor nerves originating from them. These results are an atrophy present in various degrees, some fibers disappearing almost entirely, others partially. The process is by fatty metamorphosis and absorption of resulting fat, leaving a residue of connective tissue.

Symptoms.—The clinical phenomena are in strict accord with what would be expected from the lesions, consisting in *muscular wasting* and *corresponding paresis*. Before the muscular wasting appears, a *sense of fatigue* succeeding slight effort may be manifested, followed by a positive weakness, primarily almost always in the *upper extremity*, first one and finally both. This is followed by *wasting* of the muscles of the *same* extremity, usually first seen in the thenar and hypothenar eminences, the interossei and the muscles of the extensor side of the forearm, while the flexors of the hand and fingers remain longer uninvaded. The atrophy is particularly well seen in the deltoid, and to a less degree in the triceps, still less in the biceps and shoulder muscles. Usually symptoms do not appear in the lower extremities, with the exception of exaggeration of the tendon reflexes, until some time after they have appeared in the upper, but occasionally the disease begins in the lower limbs.

When the lower limbs are affected, the patient tires easily in walking, the gait becomes unsteady and stiff, and rising from the chair becomes difficult. *Tremor* may appear in the legs. The paresis in both extremities is proportionate to the destruction of muscle, though first, at least, it is independent of the atrophy. Associated with muscular atrophy, sooner or later, is a *diminished electrical excitability*. Some excitability, however, remains as long as the muscles are intact, diminishing as their destruction spreads. A reaction of degeneration may also develop in the muscular fibers still intact. The excitability remains for the most part *intact* in the

nerve trunk because in any event a large number of fibers are preserved in their normal state.

A distinctive feature of amyotrophic lateral sclerosis is found in the *reflexes*, which, in strong contrast to progressive muscular atrophy, are *markedly increased*. Even in the early stages of the disease vigorous contractions are obtained by gently tapping the tendons of almost any of the muscles in the extremities. Always most conspicuous is the patellar reflex, while more rarely ankle clonus may be obtained. The same is true of the masseter reflex. In the arms the biceps and triceps and the flexors of the hands may be excited to strong contraction.

Contractures may take place in the later stages of the disease in the *arms and hands*, but not always. In the lower extremities, where the atrophic symptoms develop some months later and are less marked, *spastic* symptoms are a more prominent feature. The legs become rigid and some strength is required to flex them, though the muscles themselves are paretic. A typical spastic paraplegia may be produced, which is due mainly to the increase of the tendon reflexes, and a spastic paretic gait is common—that is, at first.

Later on in the disease *bulbar symptoms* may present themselves, manifested first by defects of speech, difficulty in retaining the saliva and in swallowing; and later still the lips and tongue may be seen to be atrophied, and ultimately there is difficulty in taking food, whence nutrition is impaired, and the patient gradually sinks. In some cases the disease may begin without bulbar symptoms.

Throughout, *sensibility* remains *normal* in the upper and lower extremities, and the superficial reflexes are not much altered. The *sphincters* are, as a rule, *unaffected*, although micturition may be disturbed. There may be constipation, but no actual paralysis of the bowel. Sexual power may be lost.

The successive involvement of the upper extremities, the lower extremities, and the bulbar centers marks quite well-defined stages of the disease.

Death comes ultimately from exhaustion, or more frequently through an inspiration pneumonia, caused by entrance of foreign matter into the air-passages as a result of defective deglutition, or through bulbar palsy.

Diagnosis.—The disease is distinguished from *progressive spinal muscular atrophy* by the invariable increase in the tendon reflexes, even in the early stages, as contrasted with their absence in the latter disease.

Prognosis and Treatment.—The prognosis is very unfavorable and the disease cannot be arrested. By rest in bed, massage, electricity, and hot bathing we may be able to defer the end somewhat. (See, also, Treatment of Progressive Spinal Muscular Atrophy.)

PROGRESSIVE SPINAL MUSCULAR ATROPHY.

SYNONYMS.—*Wasting Palsy; Progressive Muscular Atrophy, Type Duchenne-Aran; Duchenne-Aran's Disease; Cruveilhier's Atrophy; Chronic Anterior Poliomyelitis; Chronic Degeneration of the Motor Nuclei.*

Definition.—Progressive spinal muscular atrophy is a *progressive wasting* of more or less limited *groups of voluntary muscles*, associated

with degenerative *atrophy of the corresponding portion of* the motor *nerve tract*, including the ganglion cells of the anterior cornua, but *unaccompanied by disease of the pyramidal tracts*. The existence of this condition has been disputed, but degeneration of the cells of the anterior horns without degeneration of the pyramidal tracts has been seen by most reliable investigators. It is well to include the word spinal in the description of this disease, as thereby the disease is distinguished from progressive muscular atrophy from other causes.

Etiology.—In the majority of instances we fail to find a sufficient cause. Heredity has been regarded as playing an important rôle in its causation, but Strümpell considers the cases thus originating as instances of the juvenile myopathic variety of atrophy—that in which no nervous lesion is traceable. On the other hand, *excessive muscular exertion* seems to be more than an accidental coincidence. Exposure to cold, especially to very cold water, and the *infectious diseases*—typhoid fever, influenza, diphtheria, and syphilis—have all been held accountable, but it is likely that some of the atrophies thus resulting include other forms than the true progressive spinal muscular atrophy.

It is a disease commonly of adult males, most supposed cases among those who are younger being probably, as held by Erb, instances of the juvenile form of muscular dystrophy, although a rare family form of progressive spinal muscular atrophy has been observed in children.

Morbid Anatomy.—The *anterior horns of the gray matter* are wasted and reduced in size; their ganglion cells wholly or partially destroyed; the neuroglia is proliferated and is intercalated in places with spider cells. The *anterior nerve-roots* passing from the horns are *atrophied*, as are also the motor nerve filaments in the peripheral nerves. But the crossed pyramidal tracts in the lateral columns containing the crossed motor fibers descending from the brain to the cells in the anterior cornua are intact in typical cases. A slight degeneration may be seen in some cases in the anterolateral columns about the anterior horns. The *muscles* seen to be *wasted* before death are found converted into fat and connective tissue, a remnant of true muscular tissue remaining. At times also they are the seat of waxy change, at others still, narrowed but retaining their transverse striation.

Symptoms.—One of the most striking features of the disease is its *slow development*. Like its congener, amyotrophic lateral sclerosis, it begins most frequently in the *upper* extremities, seven out of nine times in Aran's cases. Of the upper extremities, the right was first invaded in 37 out of 62 of Sandahl's cases, the left 14 times, while the involvement was simultaneous in 11. The disease may begin in the *lower* extremities, as shown by Friedreich's statistics, according to which these were first invaded 27 times out of 146, the upper extremities 111, the lumbar muscles in eight.

The atrophy usually begins with the short muscles of the thumb, the abductor pollicis brevis first, then the opponens and the abductor. The consequent flattening of the ball of the thumb and its persistent approximation to the second metacarpal bone produces the so-called "ape-hand." Simultaneously, or almost simultaneously, the interossei begin

to waste, producing conspicuous depressions between the metacarpal bones, associated with loss of power to extend completely the terminal phalanges of the fingers. Atrophy of the lumbricales follows, producing a flattening of the hollow of the hand. The ultimate result is the characteristic *main en griffe* of Duchenne, in which the extensor tendons on the dorsum of the hand, and the flexors in the palm, may become as distinct as if dissected out.

From the hand the wasting creeps up the forearm and thence to the arm, or it may skip the forearm and pass into the arm, sparing usually the triceps extensor. In the forearm the muscles on the extensor (external) side are usually first affected, then the abductor pollicis and extensor longus pollicis, and later the supinators and flexors. It may come

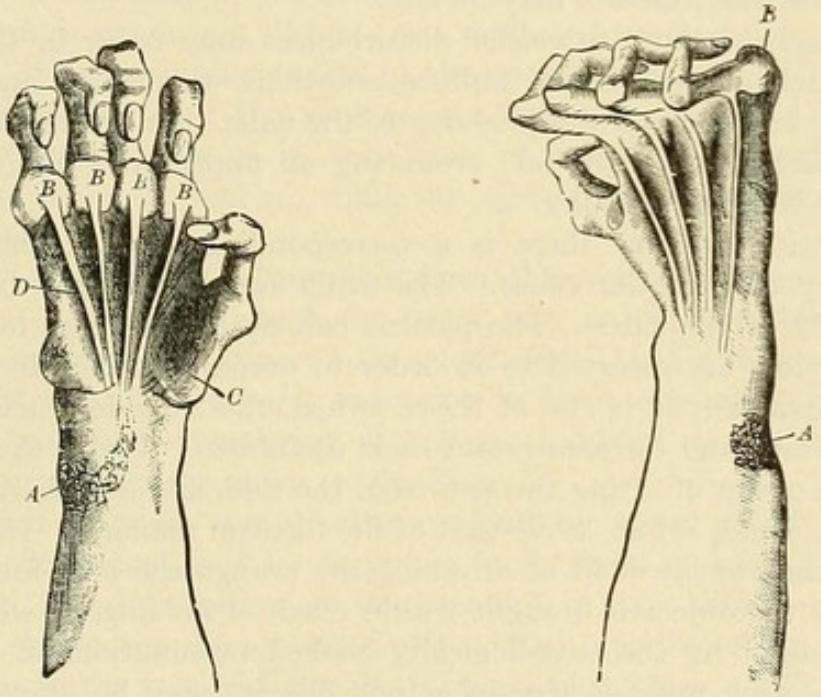


FIG. 158.—Position of Hands and Fingers in Ulnar Paralysis of Long Standing; Bird-claw Hand, "Main en Griffe"—(*after Duchenne*).

A, A. Wound of the ulnar nerve. *B, B, B, B, B.* Ends of the metacarpal bones. *D.* Tendons of the flexor sublimis digitorum. *C.* Muscles of the ball of the thumb.

to a standstill at either of these stages, or may involve the muscles of the shoulder, especially the deltoid, in which, indeed, it may begin, preferably in the right, passing thence to the scapular and trapezius muscles, the pectorals, the rhomboidei and latissimus dorsi, while a grotequeness of effect is often produced by reason of certain adjacent muscles retaining their natural size or being even seemingly hypertrophied. This is particularly the case with the inferior part of the trapezius and platysma myoides which are almost never involved. The disease may be arrested at almost any of these stages.

The lower extremities may escape altogether and the atrophy here usually develops late. The small muscles of the foot would naturally be the first affected. Very rarely there may be exceptions to this rule. The muscles of the face are invaded late or not at all, but ultimately even the intercostal and abdominal muscles may be involved. The result, then, is a veritable living skeleton, instances of which are sometimes exhibited. Deformities, including lordosis or anterior curvature of the spine may result.

With all this, *sensibility* is unaffected in the vast majority of cases, but the patient may complain of a numbness and coldness of the affected limbs. Very rarely *pains* precede the wasting in the muscles, when they are sometimes regarded as rheumatic. The *galvanic and faradic irritability* of the muscles progressively diminishes and disappears with the complete destruction of the muscle, the galvanic persisting longer. The *reaction of degeneration* may, however, be elicited late in the disease in certain muscles, more especially in the modified form known as "partial reaction" of degeneration. If the disease runs a rapid course, it may occur earlier and be more typical. *Fibrillary* muscular contractions may be present, and idiopathic muscular contractions, or myoid tumors brought out by a blow, may be thus produced. The bladder and rectum remain intact, but sexual function may be lost.

Sweating and other *vasomotor* disturbances may occur in the affected muscles, such as pemphigoid bullous eruptions, thickening and fissuring of the skin, and curving and grooving of the nails. In certain places there is an overaccumulation of fat, producing an appearance of hypertrophy when there is actual atrophy.

Along with wasting there is a corresponding *paresis*, the result of the atrophy and not its cause. The arms are flaccid and toneless and hang loosely at the sides. The patient can no longer dress himself, and various devices are resorted to in order to accomplish certain acts. Especially characteristic is one of these—when, the shoulders being first affected, the arm and forearm retain their usefulness. Under these circumstances the power of lifting the arm from the side, and especially of raising it above the head, is lost, while that of the forearm remains. Hence, if the patient wishes to lay hold of anything, he swings the arm forward with a jerk until the object is brought within reach of his fingers, when it may often be caught by the pathologically hooked terminations of these. So long as the neck muscles remain active, objects may be grasped by the mouth.

In true progressive spinal muscular atrophy the *reflexes* are entirely absent, at least in the wasted extremities, a natural result of the atrophy of the ganglion cells in the anterior cornua and of the centrifugal motor fibers of the reflex arc. The *special senses* and the *sphincters* remain normal.

Toward the close of the disease sometimes, and then only after it has existed for a long time, the phenomena of *bulbar* paralysis may present themselves after invasion of the ganglia of the medulla oblongata. These have been detailed in the section on that subject. They are by no means always present, even in advanced cases.

Diagnosis.—Muscular atrophy is not confined to the disease under consideration. It occurs in diffuse myelitis, in tumors of the cord and when cavities are formed in its interior, in multiple neuritis, and especially in amyotrophic lateral sclerosis. From all these named, except the last, it is easily distinguished by strict attention to the conditions and order of development of the symptoms—viz., insidious and progressive atrophy of groups of muscles to the exclusion of others, beginning usually in the hand or more rarely in the shoulder and upper arm; accompanied by a corresponding loss of power in the affected muscles and partial or com-

plete reaction of degeneration in the same, by diminished reflexes and fibrillar twitchings.

Differential Diagnosis.—From *amyotrophic lateral sclerosis* it is to be distinguished by its greatly *slower course* and *absence of the reflexes* and of *spastic symptoms*. It is also to be distinguished from *muscular dystrophy* in its various forms—the myopathic juvenile muscular atrophy of Erb, pseudohypertrophic muscular paralysis, and Duchenne's infantile type. In the *juvenile progressive muscular atrophy of Erb* there is also slow, symmetrical, and intermittent wasting, with weakness in certain groups of muscles, especially those of the shoulder girdle and upper arm, and later possibly the pelvis, upper thigh, and back, associated at times with true or false *muscular hypertrophy*, but usually unassociated with *fibrillar* contraction or reaction of degeneration. The average age, also, in the juvenile form is much less, Erb's cases ranging from seven to 46 1/2 or an average of 26 1/2 years, while in the spinal form or true progressive spinal muscular atrophy the average age is much greater. Of Roberts' cases, all of which seem to be true cases of progressive spinal muscular atrophy, the youngest was 20, while the ages of the remaining four were 38, 39, 47, and 67.

While in the *pseudo-hypertrophic* form there are also great weakness and wasting of muscles, though the latter may be obscured by the fatty deposit, there are *no alterations in the spinal cord*. It is a disease of childhood, and strikingly *hereditary*, beginning in the lower extremities, while progressive muscular atrophy is a disease of adults, is not hereditary, and begins usually in the upper extremities.

Duchenne's infantile type is characterized by onset at an *early age*, infancy or adolescence, and by *beginning in the facial muscles*. It is often hereditary. The distribution of the atrophy is very similar to that of Erb's form, when the disease has involved the muscles of the shoulders, but it begins in the face and may be confined to the face. The muscles of the hands and fingers are spared in Duchenne's form; *fibrillar tremors* are *not present*, and there *is no reaction of degeneration*.

Prognosis.—Many years are required to develop these symptoms in their entirety, and there may be spontaneous arrest, during which the patient may die of other causes. Sooner or later, if the patient lives, they recur, and their march is irresistible.

Treatment.—It has already been said that cure is impossible, although well-authenticated cases of arrest are reported. *Mercurials* and *iodide of potassium* or *salvarsan* should be used in cases of suspected syphilitic origin. Cooke reports a case of arrest under a course of mercury, after the disease had progressed for five years, during which many remedies were tried. In the main the treatment must consist of measures intended to *maintain the health and strength* of the patient and to counteract the muscular wasting. To the former end an abundance of nutritious food, fresh air, and outdoor life should be supplied, while tonics, including, especially, cod-liver oil, iron, arsenic, and strychnin, are indicated. The muscular wasting may be combated by *electricity* and *judicious massage*. *Both kinds of electricity* may be used, the faradic with rapid interruption to stimulate the circulation, or with slow interruption to excite individual muscles

to contraction. The current should be of moderate strength, not too frequently interrupted, and continued for a few minutes only. Duchenne recommended, particularly, treatment of important muscles, like the diaphragm through the phrenic nerve, or the intercostal muscles and the deltoids before they are actually invaded by the disease. In evidence of its usefulness he relates the case of a man who had lost many of his trunk muscles, and who was beginning to suffer from dyspnea, on whom faradization of the phrenic nerves, repeated three or four times a week, was of great service, enabling him to walk considerable distances and to go upstairs without fatigue. Another patient, whose arms were much wasted, became again able to support his family. The direct current—galvanism—is useful in advanced stages of the disease, when the strongest faradic currents fail to produce response. When galvanic currents fail to excite contractions, the treatment ought to be persevered in for a long time, using very strong currents at the onset, gradually reducing them as contractility returns. Remak, who especially advocated the use of the continuous current, advised placing the positive pole in the front of one mastoid process and the negative pole on the opposite side of the neck, near the spinous process of the vertebræ, not higher than the fifth cervical, by which he produced the contractions already described as diplegic in the fingers and other paralyzed parts. Galvanization of the sympathetic has been apparently useful in the hands of some, Erb reporting a case of complete cure.

Massage is especially important, and should be used in connection with electricity, but at a different time of day. Eulenberg refers to a case said to have been brought to a standstill by it.

Hypodermic injections of strychnin, from 1/100 to 1/40 grain (0.0005 to 0.002 gm.), are said, on the authority of Gowers, to have arrested the disease.

In families in which a hereditary tendency exists prophylactic treatment should be used. It should include hygienic measures of the kind already referred to and the avoidance of undue fatigue and exposure, and in the selection of an occupation these matters should be kept in view. On the supposition that the disease is a purely local one, gymnastics involving the exercise of the groups of muscles prone to attack are indicated, but assume less importance from the standpoint that it is a spinal cord disease. At the same time the patient should have the benefit of any doubt in the pathogeny, and as gymnastics are eminently calculated to improve the general health and thus indirectly to avert disease, their use is indicated on these grounds.

DISEASES OF THE BRAIN.

LOCALIZATION OF CEREBRAL DISEASE.

SYNONYMS.—*Cerebral Localization; Relation of Locality to Symptoms; Topical Diagnosis of Cerebral Lesions.*

Physiology.—The brain is the organ of consciousness and of perception of impressions and sensations—of memory, of thought, of origination

of voluntary motion, and of speech. It is also the seat of the instinctive acts. It has been learned from clinical observation in connection with studies at the autopsy table and from experiment that certain parts of the cortex are concerned with corresponding offices, especially motion, speech, vision, and hearing, so that from the presence of given symptoms the involvement of corresponding localities may be inferred. Allusion has already been made to the subject of topical diagnosis. Such diagnosis, it is important to remember, gives no information as to the nature of the lesion, the result being the same whether it be abscess, hemorrhage, or softening. We are simply informed that such and such area is involved.

I. The Motor Areas of the Cortex.

An examination of the following illustrations (Figs. 116 and 117) will convey an idea of the gyri and sulci of the surface of the brain.

Functional Assignments.—The motor region was formerly regarded as made up of the two central convolutions, anterior central and posterior central, also known as ascending frontal and ascending parietal; the posterior part of the three frontal convolutions; the upper part of the parietal

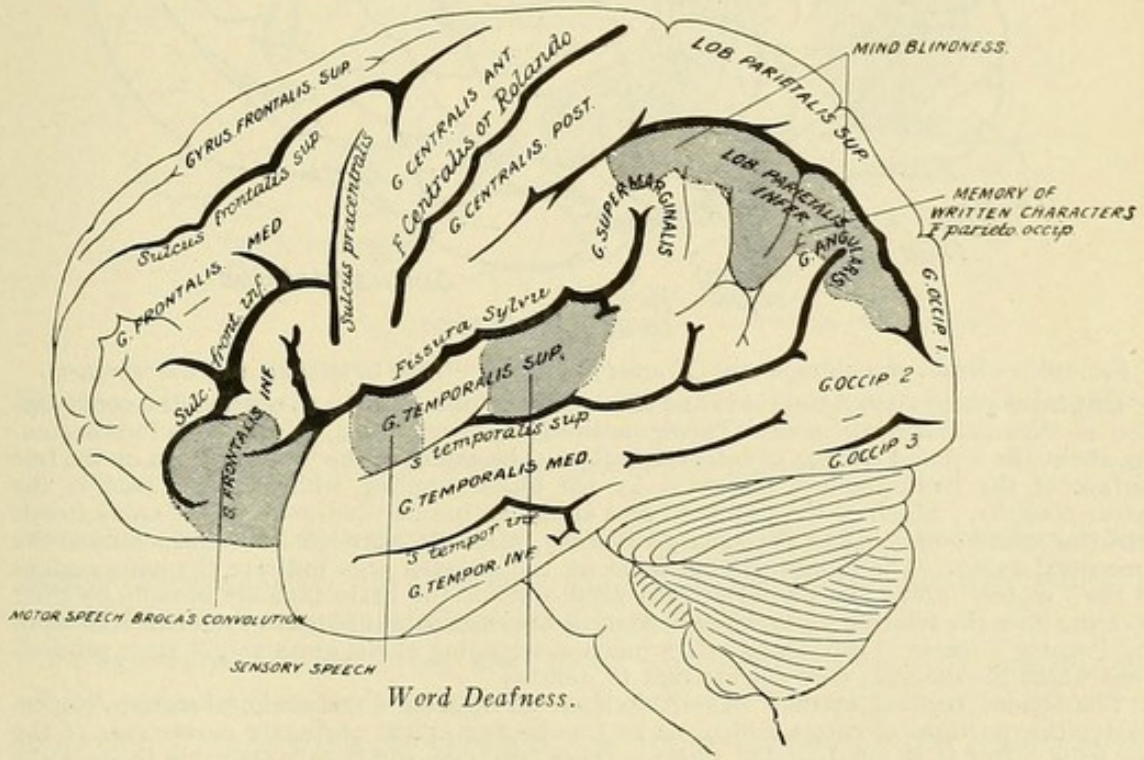


Fig. 159.—Lateral Aspect of the Brain—(*after Ecker, modified*).

lobe adjoining the ascending parietal convolution, and the paracentral lobule (Fig. 159) on the median surface of the hemisphere. The investigations of Grünbaum and Sherrington[1] on the gorilla seem to show that the motor cortex is entirely in front of the Rolandic fissure, and this view has been widely accepted (Fig. 160). All diseases which destroy any considerable portion of this cortical area invariably produce paralysis of the opposite half of the body, while no matter how extensive the destructive process

[1] Sherrington. The Integration Action of the Nervous System, London, 1906.

elsewhere in the cortex, motion remains intact if this is not touched. An acute cortical lesion sufficient to involve all the motor centers of one side and cause total hemiplegia would be likely to be fatal, while a smaller lesion, extending into the white matter, involving fibers coming from uninjured portions of the cortex, might produce a more extensive palsy than a more superficial cortical lesion.

We can even point out separate regions which act as separate centers for various groups of muscles. The center for the movements of the facial

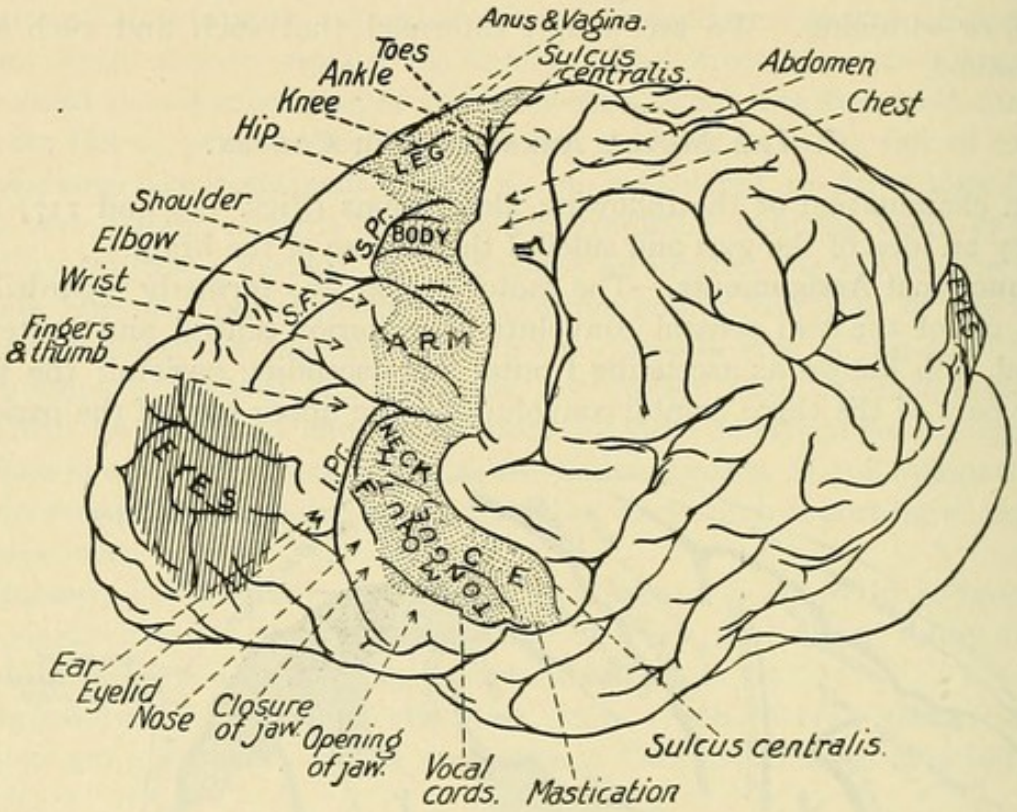


FIG. 160.—Brain of a chimpanzee (*Troglodytes niger*)—(*from Grünbaum and Sherrington*).

Left hemisphere viewed from side and above so as to obtain as far as possible the configuration of the *sulcus centralis* area. The figure involves, nevertheless, considerable foreshortening about the top and bottom of *sulcus centralis*. The extent of the "motor" area on the free surface of the hemisphere is indicated by the black stippling, which extends back to the *sulcus centralis*. Much of the "motor" area is hidden in sulci; for instance, the area extends into the *sulcus centralis* and the *sulci precentrales*, also into occasional sulci which cross the precentral gyrus. The names printed large on the stippled area indicate the main regions of the "motor" area; the names printed small outside the brain indicate broadly by their pointing lines the relative topography of some of the chief subdivisions of the main regions of the "motor" cortex. But there exists much overlapping of the areas and of their subdivisions which the diagram does not attempt to indicate.

The shaded regions, marked "eyes," indicate in the frontal and occipital regions, respectively, the portions of cortex which, under faradization, yield conjugate movements of the eye balls. But it is questionable whether these reactions sufficiently resemble those of the "motor" area to be included with them. They are therefore marked in vertical shading instead of stippling, as is the "motor" area. S. F., superior frontal sulcus. S. Pr., superior precentral sulcus. I. Pr., inferior precentral sulcus.

muscles lies at the lower end of the precentral convolution (Figs. 159 and 160). Near by and lower down is the center for movements of the tongue and vocal cords, while the center for the movements of the arm lies somewhat higher than that for the face—that is, about the middle of the anterior central convolution. From above downward the various segments are represented as follows: Shoulder, elbow, wrist, fingers—the index finger and, lowest of all, the thumb. The center for the leg lies in the uppermost part

of the central convolutions, but mostly in the paracentral lobule. Most anterior is the hip, next the knee and ankle, next the great toe, the center for the movement of which surrounds the upper end of the fissure of Rolando; still further back are the centers for the small toes. The center for the trunk is situated in the precentral convolution between those for the upper and lower limbs. The different regions are not sharply defined, but merge into one another.

As to the so-called *muscular sense*, it has been believed that it resides also in the motor area, while there have been those who have claimed for it also a separate and different localization. M. Allen Starr and A. J. McCosh[1] have reported a case of injury with symptoms which go to prove the latter view and to show that the seat of the muscular sense is "a spot in the brain about at the junction of the superior and inferior parietal

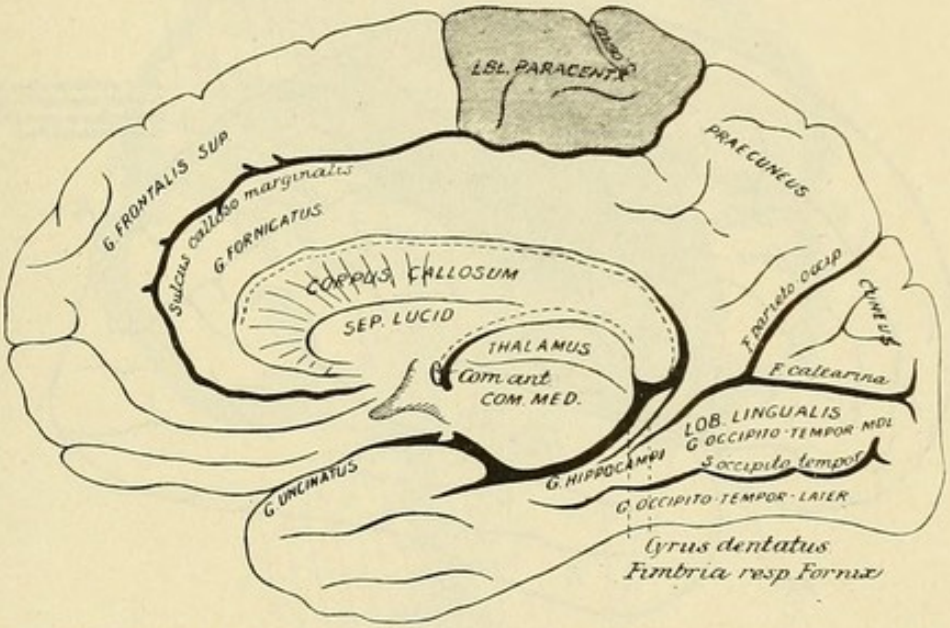


FIG. 161.—Aspect of the Median Surface of the Cerebrum as it appears when the Two Hemispheres are Separated—(*after Ecker*).
The gyri and fissures are indicated by the lettering.

convolutions, clearly posterior to the posterior central convolution," and many investigations have shown that the so-called muscular sense is probably largely represented in the parietal lobe.

These *cortical motor areas are united with spinal centers* by nerve-fibers which proceed from cell to cell in each, without connection with intervening cells. Their route is through the white matter of the hemispheres, where they form the corona radiata, the fibers of which converge to the internal capsule which lies between the optic thalamus and the caudate nucleus on the inside, and the lenticular nucleus on the outside. (See Fig. 161.) The anterior portion of the posterior limb of the capsule—the knee—is occupied by the fibers from the face, tongue, eye, and speech centers; behind these lie the fibers from the upper extremities, while those from the lower extremities occupy the middle of the posterior part. Thence the fibers of the motor path pass into the crus cerebri through its middle third, then through the pons, covered by the superficial transverse fibers of

[1] "Amer. Jour. of the Med. Sciences," November, 1894, p. 520.

this body, into the medulla oblongata, of which they form the anterior pyramids. At the lower portion of the medulla oblongata a large portion of these pyramidal fibers cross over into the opposite half of the spinal cord, constituting the crossed pyramidal tract of the lateral column, while a small bundle of fibers descends into the anterior column of the same side, forming the direct pyramidal tract, or Türck's column; some fibers probably pass to the lateral column of the same side. Both pyramidal tracts diminish in bulk as they descend, because they give off fibers which pass into the gray matter, dividing and subdividing, to come into contact with the protoplasmic processes which are continuous with the large nerve cells of the anterior cornua.

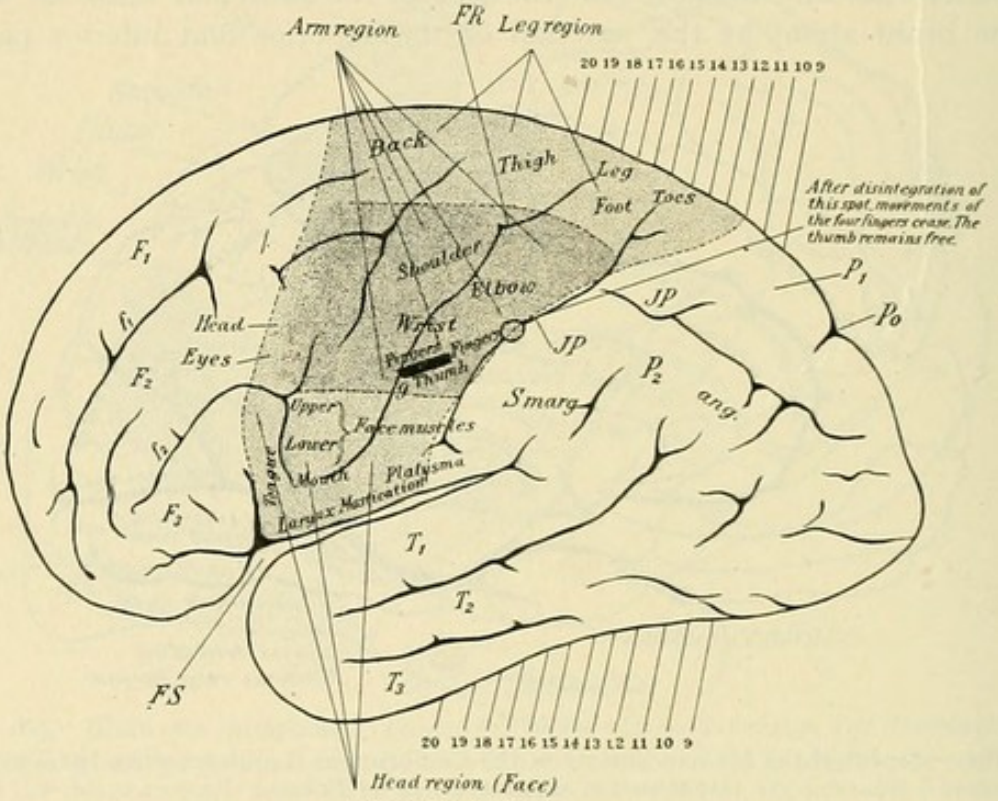


FIG. 162.—Lateral Aspect of the Brain—(*after v. Monakow*).

Lateral aspect of the human cerebral hemisphere. Motor fields (principal foci) after Allen Starr, W. W. Keen, Charles K. Mills, Victor Horsley, and Monakow's observations. *y*, Spot on the posterior central convolutions whose isolated irritation causes thumb movements, and whose destruction in a case of cranial injury caused continued defect of motion of the thumb and finger; also derangement of the stereognostic sense. The lines 9–9, 10–10, etc., 20–20 indicate certain planes of section in other figures in Monakow's monograph in Nothnagel's system.

These motor fibers form the *upper or cerebrospinal segment* of the motor system. Between the motor nerve cells in the anterior cornua and the muscles to which the motor nerve-fibers are distributed is the *lower* or *spino-muscular segment.* In response to the law already mentioned as to the nutritive independence of each neuron, each of these segments has a certain nutritional independence, depending for its integrity upon the integrity of its neuron, the upper or cerebral depending upon the cortical cells and the lower upon the large cells in the anterior cornua.

Lesions of the Upper Motor or Cerebrospinal Segment.—If, therefore, the cortical cells of the motor area degenerate, the fibers attached to them will waste as far as the beginning of the lower segment, and if the cells in

the latter degenerate or are cut off, not only do the nerve-fibers below them waste, but the muscles to which they are distributed as well. Accordingly, all the cases of paralysis due to *destructive* disease in the motor cortical region have been found associated with descending degeneration of the motor tract previously outlined, into the *direct* pyramidal tract in the anterior column of the cord on the same side, and the *crossed* pyramidal tract in the lateral column of the other side. At the same time the paralysis is accompanied by a *spastic condition*, manifested by an exaggeration of the tendon reflexes and an increase in the tension of the muscles, ascribed to a loss of the inhibitory control exerted by the cells of the cortex in the

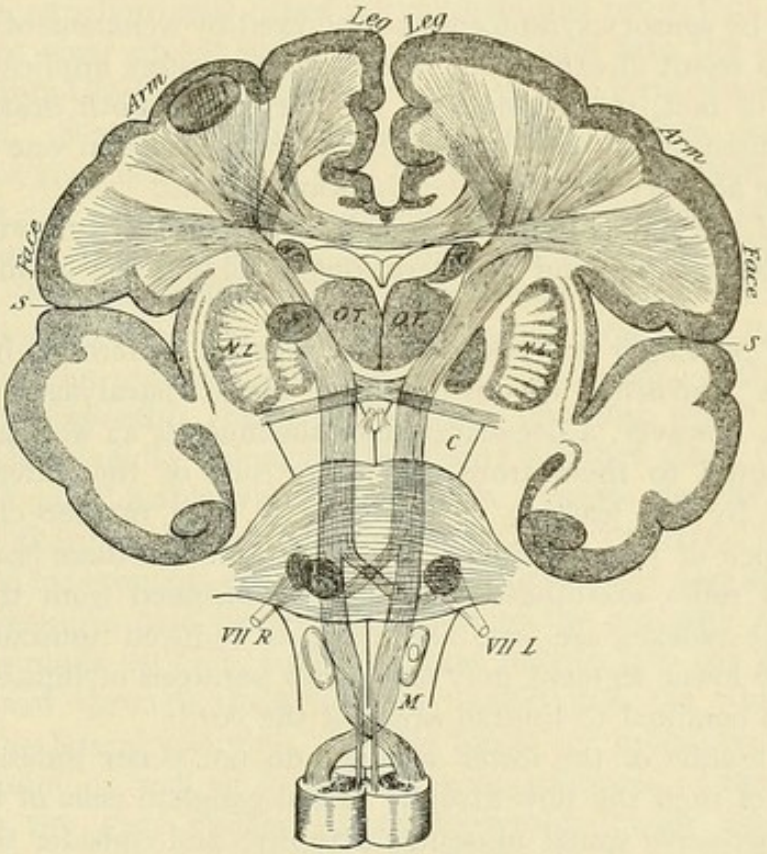


FIG. 163.—The Motor Tract—(*after Starr*).

S, *S*, Fissure of Sylvius. *NL*, *NL*, Lenticular nucleus. *OT*, *OT*, Optic thalamus. NC, NC, Caudate nucleus. *C*, Crus. *P*, Pons. *M*, Medulla. *O*, Olivary body. The tracts for the arm, leg, and face gather in the capsule and pass together to the lower pons, where the face fibers cross to the opposite seventh nerve nucleus, while the others pass to the lower medulla, where they partially decussate, to enter the lateral columns of the cord. The non-decussating fibers pass into the anterior median columns.

normal state. This explanation, however, is not entirely satisfactory. In other respects the paralysis due to cortical lesion does not differ from that due to focal disease lower down in the upper tract, except that the latter is apt to involve more muscles because of the compactness of the tract at this point. Atrophy is usually not intense in muscles paralyzed by lesion of the upper segment, but occasionally is observed in muscles thus paralyzed, but the reaction of degeneration does not occur. Lesions of the motor cortex are sometimes limited, causing correspondingly limited paralysis and even monoplegias, never, however, affecting less than a whole limb or a segment of a limb. A lesion may involve two centers lying close to each other, producing paralysis of the face and arm or of the arm and leg, but

rarely of the face and leg without involvement of the arm. It happens not infrequently that the whole motor cortex is involved, producing paralysis of one side—cortical hemiplegia. The lesion then is usually thrombosis of the middle cerebral artery.

Such is the effect of destructive lesion of the cortex. Quite different is that of *irritative* lesions. These produce convulsive seizures known as Jacksonian or cortical epilepsy, characterized by convulsions beginning in a single muscle or group of muscles and proceeding in a definite order to the involvement of other muscles corresponding to portions of the cortex affected. Thus, the convulsions may begin in the face, and extend thence to the arm and thence to the leg. The convulsions may also be accompanied by sensory symptoms and followed by weakness of the muscles involved, as a result of exhaustion of the motor centers implicated.

In point of fact, most lesions of the cortex are both destructive and irritative, consisting in the destruction of nerve cells in one center and increasing the activity of cells of neighboring centers.

Lesions of the upper segment include hemorrhages, tumors, abscesses, injuries, inflammations, and degenerations involving the brain and spinal cord.

Lesions of the Lower or Spinomuscular Segment.—Here, as in the upper segment, the *destructive* lesions produce motor paralysis. The added peculiarity is, however, a degeneration of the muscles as well as the nerve-fibers distributed to them from the motor cells of the anterior cornua, as evidenced by the wasting of the muscles, and further characterized by the presence of the reaction of degeneration. In these lesions there is also a loss of reflex excitability in the areas supplied from the segments destroyed, the reflexes are lost, and there is reduced muscular tension. Lesions of the lower segment may also cause paralysis of limited groups of muscles when confined to limited areas of the cord.

Irritative lesions of the lower segment do not occur unless we regard as the result of such the slow atrophy of the ganglion cells of the anterior cornua in progressive spinal muscular atrophy, and consider the fibrillary contractions found in this affection as a result of the stimulation of these cells in their slow degeneration.

II. Sensory Areas of the Cortex and Sensory Paths.

Our knowledge of the sensory areas is much less definite than that of the motor. Beginning at the periphery, we learn that sensory fibers emanating from tactile surfaces, like the skin, promptly and for the most part become associated with motor nerve filaments in the lower motor segment, the union of both constituting a mixed nerve. The two sets of fibers, however, separate again within the spinal canal, the motor filaments are continuous with the anterior roots, and the sensory enter the cord by the posterior, on which is a ganglion. The areas whence the posterior roots gather their nerves will be found in Starr's table on page 961. The precise routes of sensory impressions to the brain are not determined, but experiment and clinical pathology show that probably a considerable number of sensory fibers cross at once and become associated with other

fibers which ascend to the brain in the opposite half of the cord. The following seems to be the results of the latest histological studies:

The sensory nerve-fibers, entering the spinal cord from the spinal ganglion on the posterior root, pass to the posterior columns and divide dichotomously, one branch passing upward, the other downward. From these longitudinal branches arise short transverse branches which penetrate the gray matter and end in the tufts or arborizations which characterize the *distributary* ends of nerve filaments. These tufts or arborizations in which the sensory fiber ends in the gray matter are apparently in close contact, but possibly not in direct anatomical relation with the ganglion cells in the anterior *and* posterior horns and in Clarke's column. From these ganglion cells other nerve-fibers are projected, the course of which is not clear except as to those which pass into the anterior roots, and those from Clarke's column which pass over to form the ascending cerebellar tract. Some pass up the anterolateral columns, some decussate through the gray commissure with fibers from the opposite side. Many fibers from the posterior roots ascend in the posterior columns of the same side and decussate in the medulla oblongata to form the fillet or lemniscus. Further confirmation of this course is found in the fact that if a posterior nerve root is cut, the ascending Wallerian degeneration is seen only in the posterior columns of the same side, and ceases in the nuclei of the *funiculus gracilis* or *funiculus cuneatus*, which are ganglionic bodies in the medulla oblongata beginning another stage of the sensory path. It is questionable whether there are separate strands of conduction for tactile, thermal, or painful impressions, but probably there are such fibers. The experiments of Gotch, Horsley, and Mott also go to show that tactile impressions pass up the same side in the posterior columns, while impressions made by pain, cold, and heat radiate into the gray matter of the cord, and through these probably again into the white conducting tracts of the lateral column. Testicle fibers are probably in the anterolateral column as well as in the posterior columns. Diseases involving extensively the gray matter, as syringomyelia, cause alteration in the temperature sense, but also diseases of peripheral parts, as pachymeningitis and neuritis. Many hold very different views from those just expressed and believe that different fibers exist for the conduction of the different forms of sensation.

Many investigators believe that all the sensory fibers of the opposite side of the body are collected in the posterior third of the posterior limb of the internal capsule, just behind the motor fibers of the upper segment. Dejerine utterly rejects this teaching, and holds that the sensory fibers are mingled with the motor in the posterior limbs of the internal capsule.

Sensory Areas in the Cortex.—Much doubt exists as to the seat of the sensory areas in the cortex. Horsley suggested that the muscular and tactile senses are localized in the motor cortex, and that two of the three principal layers of cells in this region subserve these functions. The experimental studies of Munk lead to the same conclusions—that the so-called "sphere of sensation" lies in the same region as the motor centers of the cortex. Dana also has shown that many lesions of the motor area, especially in the hinder part, are associated with anesthesia, while Ferrier

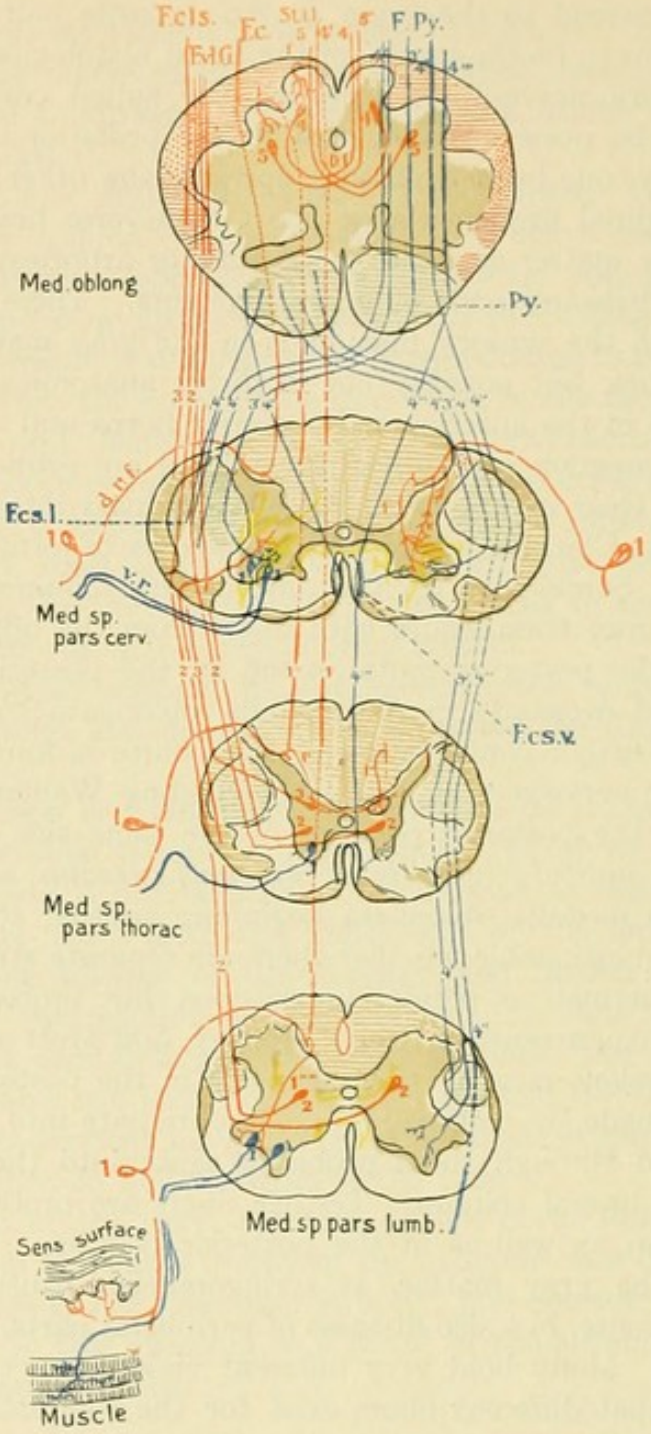


FIG. 164.—Sensory and Motor Paths in the Spinal Cord—(*after Barker*).

DESCRIPTION OF FIG. 121.

Black.—*Med. oblong.*, transverse section through the medulla oblongata at the level of the decussatio lemniscorum; *Med. sp. pars cerv.*, transverse section through the medulla spinalis pars cervicalis; *Med. sp. pars lumb.*, transverse section through the medulla spinalis pars lumbalis; *Med. sp. pars thorac.*, transverse section through the medulla spinalis pars thoracalis; *sens. surface*, sensory surface; *muscle*, muscle.

Red.—The areas of white matter in the spinal cord and medulla oblongata occupied by sensory axons are indicated by red lines or dots. The cell bodies and axons of sensory neurons are also colored red. *D. l.*, decussatio lemniscorum; *D. r. f.*, dorsal root fiber (central axon of peripheral centripetal neuron); *F.c.*, axon of fasciculus cuneatus; *F. cls.*, axon of fasciculus cerebellospinalis (direct cerebellar tract); *F. vl. G.*, axon of fasciculus ventrolateralis [Gowersi]; *St. i. l.*, axons of stratum interolivare lemnisci; 1, cell bodies of peripheral centripetal neurons (situated in the spinal ganglia); 1′, ascending limb of bifurcated central axon of peripheral sensory neuron extending from the pars lumbalis of the spinal cord to the medulla oblongata, being situated first in the fasciculus cuneatus, in higher levels of the cord in the fasciculus gracilis, and finally terminating in the nucleus funiculi gracilis. 1″, ascending limb of bifurcated central axon of peripheral sensory neuron pertaining to the thoracic portion of the spinal cord. It enters the fasciculus cuneatus, and passing upward, approaches the medial border of this fasciculus without, however, entering the fasciculus

considers the *hippocampal convolution*, and Schäfer the *gyrus fornicatus*, as the sensory center in the cortex. Clinical evidence on this point is not uniform. In some cases of motor paralysis there is undoubted simultaneous disturbance of sensation, in others not. By some the parietal lobe is considered the important sensory area, and the weight of opinion is in favor of this view. The muscular sense is also sometimes impaired in paralyzed limbs, in consequence of which the patient cannot tell with his eyes closed the position of the affected limb.

Among the cortical areas representing sensation must be included those for sight, hearing, smell, and taste, which will be considered in connection with affections of the peripheral nerve. Suffice it to say, briefly, that the auditory center is located in the first temporal gyrus, the visual in the occipital lobe, the cortical visual center being on the mesial surface in the *cuneus*, especially about the calcarine ("calcar," a spur) fissure, where are represented the opposite half visual fields. Some authorities include more of the occipital lobe than this in the visual area.

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ragcilis. It is seen to terminate ultimately in the nucleus funiculi cuneati. 1‴, ascending limb of bifurcated central axon of peripheral sensory neuron pertaining to the pars cervicalis of the spinal cord. It passes upward in the fasciculus cuneatus to terminate at a level higher than that indicated in the diagram. 1⁗, reflex collaterals extending from the central axons (or their subdivisions) of the peripheral sensory neurons to the ventral horns of the spinal cord, there coming into conduction relation with the cell bodies and dendrites of the lower motor neurons. 1, collaterals from the axons of the fasciculus cuneatus to the nucleus dorsalis [Clarkii]; 2, cell bodies in substantia grisea giving rise to axons of the fasciculus ventrolateralis [Gowersi]; 2′, axons of fasciculus ventrolateralis [Gowersi]; 3, cell body in nucleus dorsalis [Clarkii] giving rise to axon of fasciculus cerebellospinalis; 3′, axon of fasciculus cerebellospinalis (direct cerebellar tract); 4, cells of nucleus funiculi gracilis giving rise to axons of fibræ arcuatæ internæ which undergo decussation (decusatio lemniscorum) in the raphe; 4′, continuation of axons of fibræ arcuatæ internæ after decussation. They run cerebralward in the stratum interolivare leminisci. 5, cells of nucleus funiculi cuneati which give rise to axons of fibræ arcuatæ internæ which undergo decussation (decussatio lemniscorum) in the raphe. 5′, continuation of axons of fibræ arcuatæ internæ after decussation. Having had their origin in the nucleus funiculi cuneati of the opposite side, they now run forward in the stratum interolivare lemnisci.

Blue.—The areas of white matter in the spinal cord and medulla oblongata indicated by parallel blue lines correspond to the position of the fasciculi cerebrospinales (pyramidales). The cell bodies and axons of the lower motor neurons are also printed in blue. *F. cs. l.*, fasciculus cerebrospinalis lateralis or lateral pyramidal tract; *F. cs. v.*, fasciculus cerebrospinalic ventralis or ventral pyramidal tract; *F. Py.*, fasciculi pyramidales in the medulla oblongata; *Py.*, pyramis medullæ oblongatæ, *v. r.*, radix ventralis, nervi spinalis; 1, cell bodies of lower motor neurons situated in the ventral horns of the gray matter of the spinal cord giving off axons which go to form the ventral roots of the spinal nerves; 3′, axons of fasciculi pyramidales which undergo decussation in the decussatio pyramidum and pass down in the fasciculus cerebrospinalis lateralis of the opposite side of the spinal cord to terminate in the ventral horns of the cervical region. They throw the lower motor neurons which innervate the musculature of the upper extremity of one side under the influence of the pallium of the opposite side. 4′, axons of fasciculi pyramidales which undergo decussation in the decussatio pyramidum and pass down in the fasciculus cerebrospinalis lateralis of the opposite side of the spinal cord to terminate in the ventral horns of the lumbosacral region. They throw the lower motor neurons which innervate the musculature of the lower extremity of one side of the body under the influence of the pallium of the opposite side. 4″, axon of fasciculi pyramidales which does not undergo decussation in the decussatio pyramidum, but passes down in the fasciculus cerebrospinalis lateralis of the same side (homolateral fiber). 4‴, axon of fasciculi pyramidales which does not undergo decussation in the decussatio pyramidum, but passes down in the fasciculus cerebrospinalis ventralis to terminate in the ventral horn of the same side. It would throw the lower motor neurons governing a portion of the musculature of one side under the influence of the pallium of the same side. It is probable that in addition to these fibers of the fasciculus cerebrospinalis ventralis, which terminate in the ventral horn of the same side, there are other fibers (not shown in the diagram) which, passing through the ventral commissure, terminate in the ventral horn of the opposite side. (See text.)

Yellow.—Cell bodies, axons, collaterals, and terminals belonging to the fasciculi proprii of the ventral and lateral funiculi—(*Barker*).

Lesions of the Sensory Tract.—These may also be *destructive* or *irritative*. *Destructive lesions* would, of course, destroy sensation in the part whence the nervous supply comes to the point of lesion. Most frequently it is an injury to a peripheral nerve, though loss of sensation is rarely complete in the part to which such nerve is distributed, because that area may receive sensory nerves from another segment of the spinal cord. Complete transverse section of the spinal cord itself causes complete anesthesia in the parts supplied from the segment below the injury. The effects of a lesion invading one-half of the cord are detailed on page 882.

Irritative lesions of the sensory path cause paresthesias, including formication, tingling, numbness, and finally pain corresponding to the degree of irritation. The last is commonly due to irritation in the course of a peripheral nerve, though it may also be caused by irritation to the sensory path within the central nervous system.

CORTICAL AREAS COVERING SPEECH.

THE VARIOUS FORMS OF APHASIA AND THEIR ANATOMICAL LESIONS.

It has already been stated that almost our first accurate knowledge of cerebral localization was the discovery by Broca, in 1861, that derange-

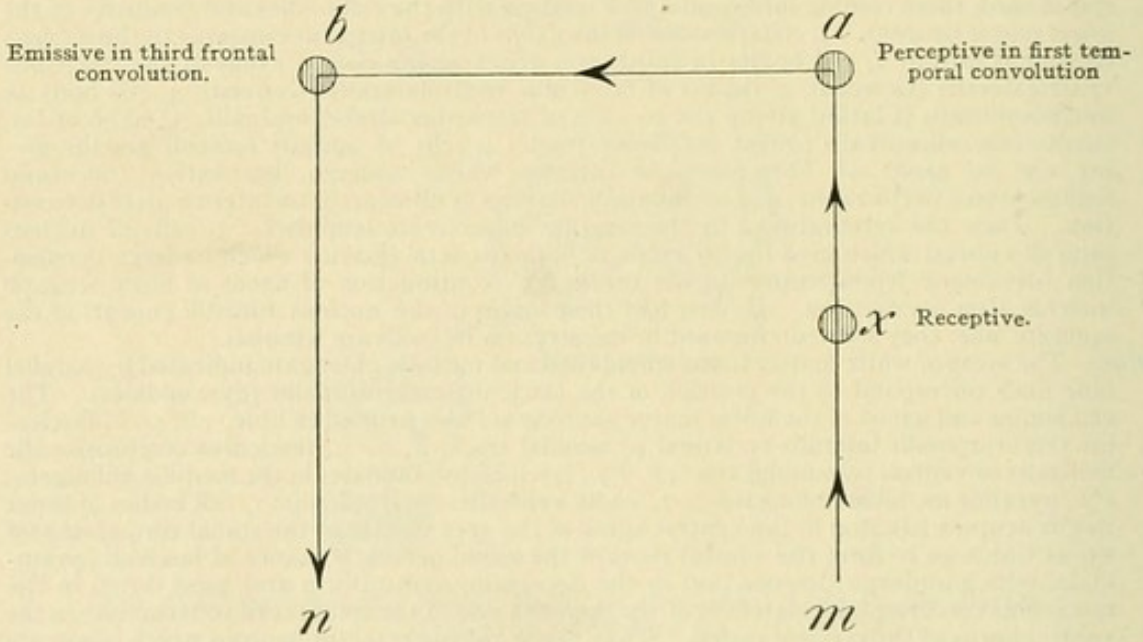


Fig. 165.—Primitive Speech Apparatus of the Child in Mechanical Repetition of Words, according to Wernicke and Lichtheim.

a, Sensory speech center. *b*, Motor speech center. *x*, Acoustic center of pure sense of hearing. *m-x*, Route to acoustic center. *b-n*, Motor speech tract.

ments of speech result from lesions of the third or inferior left frontal convolution. The loss of power to comprehend words correctly and to use them properly is covered by the general term *aphasia*. Further derangement consists in inability to articulate words, and is due to lesions of nuclei situated for the most part in the pons and medulla oblongata, regulating the action of the vocal cords, the tongue, and the lips, and is known as *anarthria* or *dysarthria*.

The study of the phenomena of aphasia will be facilitated by a brief

review of the conditions of acquired language. Language is acquired by the child gradually through imitation. Thus, when the mother teaches it to say "cat" or "bell" or "papa," she names the word, and its sound impresses the distribution of the auditory nerve, *m* (see diagram, p. 954) whence it passes to the *acoustic center*, *x*, and thence to the sensory *speech* center, *a*, in the first or *upper temporal* convolution, where it is stored as a sound memory. From this it passes from behind forward along the association fibers to *b*, the motor speech center in the left inferior *frontal* convolution (Broca's center, propositionizing center of Broadbent),[1] whence the muscles of articulation are put into operation and the word

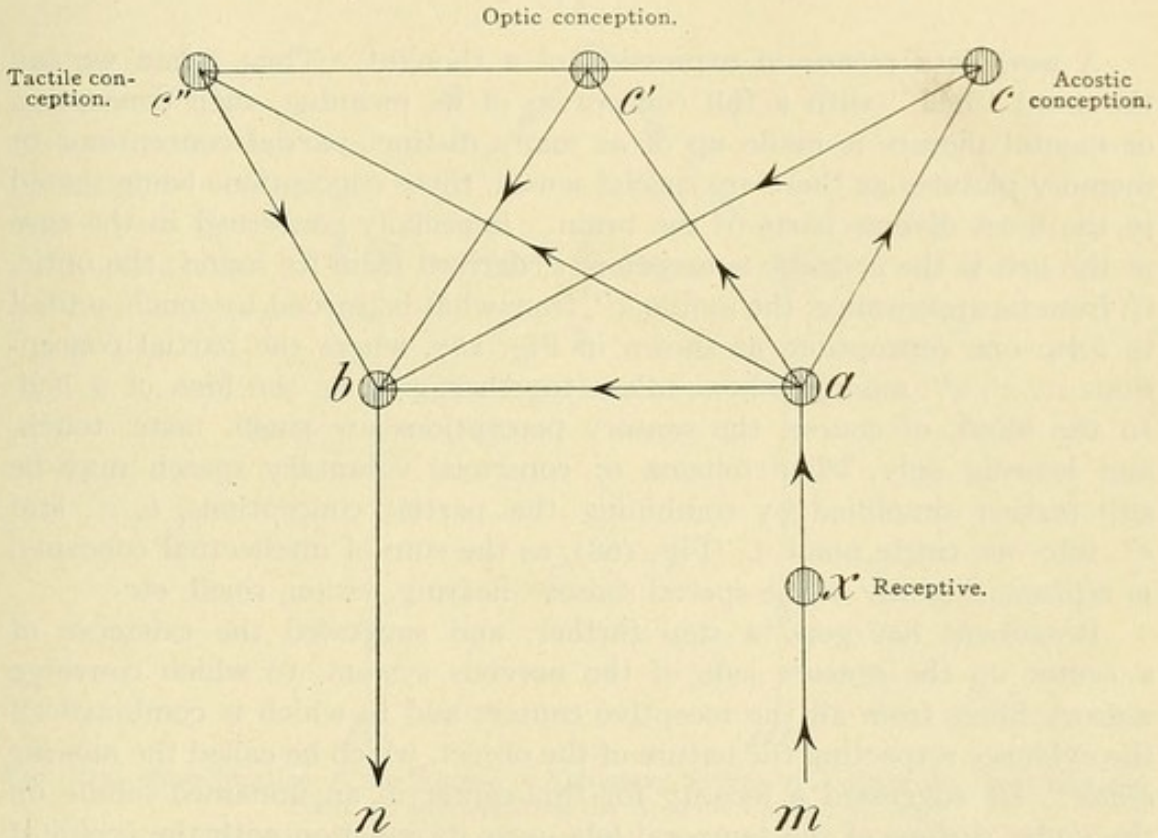


FIG. 166.—Wernicke's Schema, Showing the Association of the Various Partial Conceptions to Form the Whole Conception or Word Image of an Object.

For the sake of simplicity only three partial conceptions and three sensory areas are shown, instead of the many which go to make up our notions of complex objects. The letters *a*, *b*, *m*, and *n* have the same application as in the previous figure, but *x* may indicate the seat of any of the special senses—hearing, vision, smell, touch.

is spoken. Thus, the speech mechanism consists of *receptive*, *perceptive*, and *emissive* centers.

The development of *voluntary* speech in the child continues through the accumulation of associated ideas in the perceptive and emissive centers, *a* and *b*. The *word image* or picture which is the foundation of every word is made up of the sum of a number of partial conceptions of memory pictures acquired by experience and stored for further use in the different sensory areas of the cerebral cortex. Thus, the memory of the *sound* of a word as spoken, the memory of the *appearance* of a word as written

[1] That is, the center where thoughts are set in a framework of words, but through which utterance is not consummated; whence other cortical centers are necessary to motor speech, and these are found caudal of Broca's convolution at the foot of the anterior central convolution. This region Broadbent calls the *uttering center*.

or printed, as well as the *muscular movements* needed to speak the word or write it, are distinct from one another and yet associated. Loss of one of these memory pictures or derangement in their association impairs the integrity of the word image and produces such defects in the use of the word as are covered by the different varieties of aphasia. These derangements have been arranged in two divisions, according as the defect is in (1) the receptive and perceptive and (2) emissive function of the brain, the former constituting the *sensory* aphasias, the latter the *motor*.

The Physical Basis of Thought—Apraxia.

A *word* is a means of expression of a thought. Thus, when we say the word "bell," with a full conception of its meaning, such conception or mental picture is made up of as many distinct partial conceptions or memory pictures as there are special senses, these conceptions being seated in the most diverse parts of the brain. Especially concerned in the case of the bell is the acoustic conception, *c*, derived from its sound; the optic, c′, from its appearance; the tactile, *c″*, from what is learned by touch, united to form one conception, as shown in Fig. 167, where the partial conceptions, *c*, *c′*, *c″*, among others, taken together, give us the idea of a bell. In the blind, of course, the sensory perceptions are smell, taste, touch, and hearing only. The schema of conscious voluntary speech may be still further simplified by combining the partial conceptions, c, c′, and *c″*, into one single point, *C* (Fig. 168), as the sum of intellectual concepts, *m* representing any of the special senses—hearing, vision, smell, etc.

Broadbent has gone a step further, and suggested the existence of a center on the sensory side of the nervous system, to which converge sensory fibers from all the receptive centers and in which is combined all the evidence respecting the nature of the object, which he called the *naming center*. He suggested a locality for this center in an unnamed lobule on the under surface of the temporal lobe near its junction with the occipital lobe. Charles K. Mills[1] has reported a case of glioma with autopsy which goes to confirm Broadbent's speculation and to locate the exact position of this center in the *third temporal convolution*. Its correlative center is the *propositionizing* center in which names or nouns are set in a framework for outward expression or utterance.

The loss of these memory pictures is known as *apraxia*, which may be defined as a state in which there is impairment or loss of the power to recognize the nature and purpose of objects, and which is something apart from aphasia. In one form of it any object, such as a watch, a knife, or a spoon, may be taken up and handled by the patient, but all knowledge of its use or purpose is gone. Such a condition, when dependent on loss of the visual memories, was well named by Munk *mind-blindness*. A person formerly familiar with the tick of a watch or the sound of a bell no longer interprets such sounds aright; or is unable to follow melodies or appreciate music as he once did. Thus we have *mind-deafness*, or

[1] Dercum, "Diseases of the Nervous System" by American authors, p. 427, 1895.

auditory amnesia, or in the case of music, *amusia*. Again, the odor of the rose and violet no longer suggests these flowers, giving *mind anosmia;* or the taste of an orange, *mind ageusia;* or the soft feel of fur or velvet gives no notion of these substances, *mind atactilia*. For the sum of these defects the term *apraxia* is now used, but mind-blindness and mind-deafness are the most important subvarieties. Apraxia may occur alone, but it is usually associated with sensory or motor aphasia. In simple apraxia the patient may be able to read, but the words arouse no intelli-

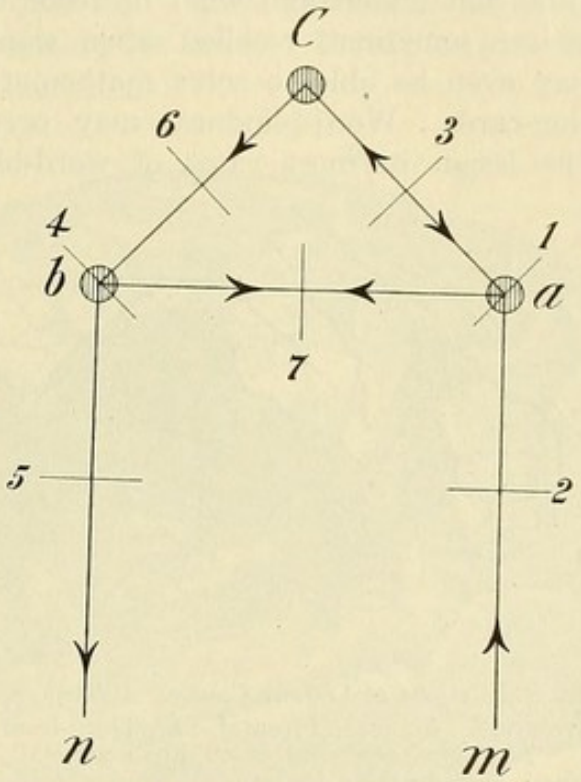


FIG. 167.—Simplification of the Schema of Voluntary Speech by Uniting the Ideas into the Point, *C*, and Omitting the Acoustic Center, *x*.
The letters have the same meaning as in Figures 122 and 123.

gent impression in his mind. Some observations go to show that the lesion in mind-blindness is in the *supramarginal and angular gyri*, or in the tracts interior to these in the white matter beneath them; and possibly mine-blindness only occurs when this area is injured, in the left hemisphere in right-handed persons, and in the right hemisphere in those left-handed. Mind-blindness is, however, at times functional and transitory, and as such is associated with many forms of mental disturbance.

There are as many varieties of apraxia as there are organs of sense, but the most common appears to be psychical or mind-blindness, generally associated with the form of aphasia known as word-blindness. The seat of the lesion in mind-atactilia has been placed in the gyrus fornicatus, hippocampal gyrus, precuneus and parietal lobe, but it is doubtful whether all these parts are concerned in this function. The parietal lobe probably is the most important. Mind anosmia in the uncinate and hippocampal gyri, and mind ageusia probably as in anosmia.

Aphasia, or Loss of the Faculty of Speech.

Aphasia is sensory or motor according as it is caused by a loss of memory of words, or by an inability to enunciate—according as it is the receptive or the emissive center which is at fault.

Sensory Aphasia. Including Word-blindness, Word-deafness, Amnesic Aphasia.—By *word-blindness* is meant loss of the memory of the appearance of a word. In this condition the patient does not recognize words which he sees on the written or printed page, and although he may be able to pronounce them after hearing them or write them at dictation or copy them, he does not understand what he reads or writes. On the other hand, *figures* are sometimes recalled when words are forgottten, and the patient may even be able to solve mathematical problems and to recognize playing-cards. Word-blindness may occur alone or with *motor* aphasia. The lesion in most cases of word-blindness has been

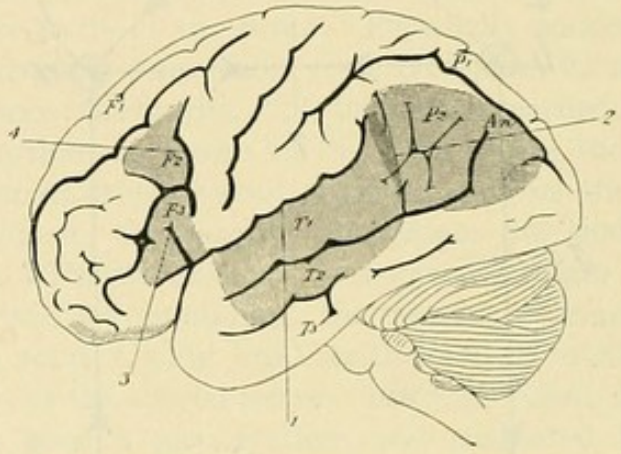


Fig. 168.—Situations of Lesions Causing Aphasia—(*after Starr*).

F^1. First frontal convolution. F^2. Second frontal. F^3. Third frontal. T^1. First temporal T^2. Second temporal. T^3. Third temporal. P^1. First parietal. P^2. Second parietal. 1. Lesion of word-*deafness* and deafness for musical sounds, or mind-deafness, according to M. Allen Starr. 2. Lesion of mind-blindness and word-*blindness*, according to Ferrier. 3. Lesion of motor aphasia. 4. Supposed lesion of agraphia.

in the *angular* and *supramarginal* gyri on the left side, as located by Ferrier, but this area is not believed by all to be the center for word-seeing. *Alexia*, or inability to read, is a corollary growing out of this, as is also *agraphia*, or inability to write, so far as it depends on sight. It is often associated, as already stated, with *mind-blindness*, but may occur independently of it.

Word-deafness is a condition in which the patient has forgotten the significance of spoken words, although he hears them as sounds. The words of his own language are as a foreign tongue which he does not understand, while there is deafness also to musical sounds—*amusia*, the "*Tontaubheit*" of the Germans. Word-deafness is commonly associated with other forms of sensory aphasia in various degrees, but cases of pure word-deafness occur in which the patient has been able to read and to speak, but is unable to recognize the meaning of a word when spoken. It is a rare variety of deafness whose *lesion* is placed by most students of the subject in the first *temporal convolution* or its posterior part, but Starr, basing his conclusion on 50 cases which he has collected with au-

topsies, places it, with Seppilli, in the *posterior half* of both the *first and second temporal* convolutions of the left side in right-handed persons, and of the right side in left-handed persons, as shown in the drawing. Recent investigations indicate that the posterior convolutions in the left island of Reil in right-handed persons is important in word hearing.

A simple variety of sensory aphasia is *amnesic aphasia*, in which the patient simply forgets words—just as we are all, at times, at loss for a word. Such a person sees a dog or another animal, knows perfectly well what it is, but cannot recall its name. The moment, however, the word "dog" is suggested, he knows all about it. In disease usually a number of words are thus lost. Such aphasia is called *amnesic*, because it is really a loss of memory for words. It may be partial, as when a patient

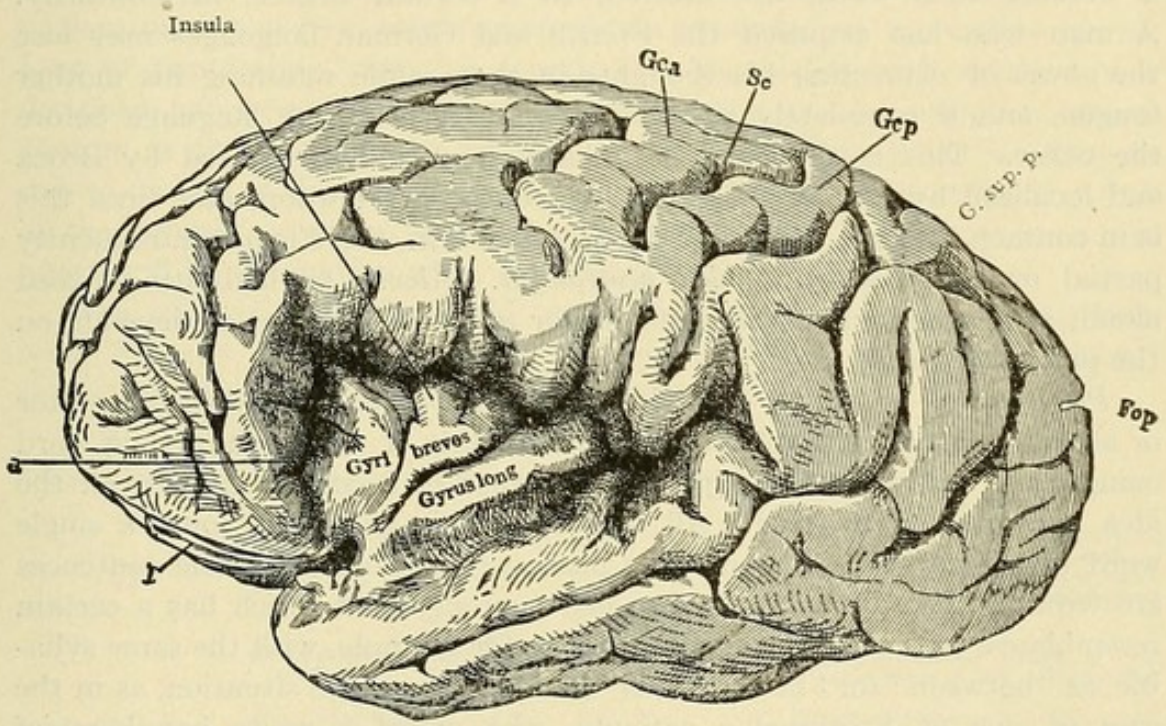


FIG. 169.—The Left Hemisphere, with the Fissure of Sylvius Drawn Apart in Order to Show the Convolutions in the Island of Reil or 5th Lobe. The Island of Reil is covered by the *pars opercularis* or posterior part of Broca's convolution, which is here drawn aside—(*after Henle*).

Sc. Sulcus centralis. Gca, Gcp. Gyrus centralis, anterior and posterior. Fop. Fissura parieto-occipitalis.

forgets nothing but his own name and remembers all other words, or when he is able to express himself in another tongue. If permanent, it is probably due to a *break in the association tract*, to be later considered, and should be so limited. Word-deafness may be distinguished from amnesic aphasia by asking the patient to do some act, such as to touch an object, when he will respond correctly if he has simple amnesic aphasia, but will not if he is the subject of word-deafness.

Allied to amnesic aphasia is sensory or *amnesic agraphia*, in which a word cannot be written because it cannot be called to mind. A person thus affected may be unable to write voluntarily, but may be able to write at dictation if he is one who writes much. As already mentioned, agraphia also occurs as a part of word-blindness so far as it depends on sight.

Motor or Ataxic Aphasia or Aphemia—Alalia.—In this condition the memory of the muscular action necessary to transfer the word image into speech is lost. There is disturbance of the emissive center, *b*, in which this transfer takes place. The patient knows perfectly well what he wishes to say, but cannot say it, though he may make the greatest effort to do so. Nor can he repeat a word after hearing it. The degree varies greatly. In complete cases he may be able to read, though not aloud, and understand what is said, but cannot say a word himself. More commonly, he can say one or two words, such as "no," "yes," while in mild cases he may simply misplace or omit letters, say "widow" instead of "window," or "wrelsters" instead of wrestles." Singularly, too, when in a passion he may be able to say the right word or to swear. This is because such words are uttered, to a certain degree, involuntarily. A man who has acquired the French and German languages may lose the power of expressing his thoughts in them while retaining his mother tongue, and if completely aphasic, he may recover one language before the other. This is the form of aphasia long ago recognized by Broca and localized by him in the third left frontal convolution, and since this is in contact with the center for the face and arm, there is not infrequently partial or complete *right-sided hemiplegia*. *Alexia*, or inability to read aloud, is a necessary corollary to motor aphasia so far as it depends on the power to speak.

Paraphasia, or *mixed aphasia*, and *monophasia* are allied to motor or ataxic aphasia. Paraphasia is a confounding of words, the wrong word being used instead of the right one, because of a confusion between the idea and the proper word. All degrees of this also occur, only a single word being sometimes erroneously used, while in others whole sentences are wrong. The patient may also use a wrong word which has a certain resemblance to the correct one, beginning, for example, with the same syllable, as "between" for "bewitch"; or the idea usurps the situation, as in the case of one of Strümpell's patients, who called a white handkerchief "snow." In these cases the *association* or *conduction tract* between the *perceptive* center and the *emissive* center is broken, whence it was called by Wernicke *aphasia of conduction*. The lesion in paraphasia is usually in the island of Reil and in the convolutions which unite the frontal and temporal lobes. But any disturbance in the association processes of language, no matter where the break lies, may cause it. In *monophasia* the patient can command but one syllable or one word or a short phrase, which he repeats over and over again.

Motor agraphia must also be distinguished from sensory. Sensory agraphia is sometimes amnesic—that is, the patient cannot write the word because he cannot call it to mind; at others it is a part of word-blindness. Motor agraphia is quite independent of ability to read aloud—that is, of effort memories necessary to speech, the difficulty being connected with the movements of the hand; but when motor aphasia exists, motor agraphia is usually also present. In sensory agraphia the patient may still be able to write by dictation, in the latter not. Agraphia also varies greatly in degree. The patient may write one or two letters, or he may be totally unable to write voluntarily or from dictation. The seat of the

lesion of motor agraphia is still unsettled. According to some authorities the graphic center is located in the *second frontal* convolution of the left side, near the ascending frontal convolution. Starr locates it in the middle of the convolution. *Paragraphia* is a condition in which one word is written when another is intended. It is a corollary to paraphasia.

Amimia is the loss or impairment of the power of expression by signs when caused by cerebral disease. *Paramimia*, the misuse of signs in the attempt to express thought, is comparable to paraphasia for speech and paralexia for reading, and is dependent on a like cause—the *destruction or impairment of commissural or association tracts between sensory and motor centers*. It is not correct to suppose that the aphasic can substitute signs for words and thus express himself, for the two defects go hand in hand, even though he retain the power of moving his hands. A patient may, however, regain pantomimic power before he regains speech. Loss of pantomimic power is found often associated with *destruction of the third left frontal convolution*, or destruction of the receptive speech centers or their connecting tracts. It may accompany verbal amnesia due to disease of these areas or disturbance of the association tracts. Just as the aphasic may say "yes" when he means "no," so he may use a sign which will be affirmative when he intends to be negative.

The following table may aid somewhat a review of the previous text, while Fig. 168, from Starr's book of "Familiar Forms of Nervous Disease," shows the situation of the lesions causing aphasia:

APRAXIA, inability to recognize the nature and purpose of an object.

		Seat of Lesion.
	Mind-blindness.	Supramarginal and angular gyri, or the white matter beneath, in the left hemisphere in the right-handed and right hemisphere in the left-handed.
	Mind-deafness, including amusia, or auditory amnesia.	Upper temporal gyrus of left hemisphere in the right-handed.
	Mind atactilia.	Gyrus fornicatus, hippocampal gyrus, precuneus, and post-parietal (Mills).
	Mind anosmia.	Uncinate gyrus (Ferrier) and hippocampal gyrus.
	Mind ageusia	Same.

APHASIA, inability to comprehend words correctly and to use them properly.

Sensory aphasia, inability to recognize word pictures and word sounds, loss of memory of word pictures and word sounds.	*Word*-blindness, in which memory of the appearance of a word is lost.	Angular and supramarginal gyrus.
	Word-deafness, in which memory of the sound of a word is lost.	Posterior part of first and second temporal gyri (Seppilli and Starr).
	Amnesia, inability to recall a word.	Disturbance of association tract.
Motor aphasia, inability to utter words, though knowing well what to say.	Including *alexia*, or inability to read aloud.	Posterior part of third left from a (Broca's convolution).
Paraphasia. A confounding of words in speaking, in which the wrong word is used instead of the right one.		Island of Reil and any disturbance of the association tract.
Amimia. Loss of power of expression by signs.		Third left frontal convolution, receiving or concept center. Disturbance of association tracts.
Paramimia Misuse of signs to express thought.		

AGRAPHIA, inability to write.

Sensory Agraphia.		*Seat.*
Inability to write because (*a*) of want of idea as to what a word is or (*b*) looks like.	(*a*) Amnesic agraphia. (*b*) A part of word blindness.	Association tract. Angular and supramarginal gyrus.
Motor Agraphia.		
Inability to write because of want of motor power of writing, although the other movements of the hand may be excellent.		Not settled, but possibly middle of the ascending frontal convolution or ascending parietal—*i. e.*, in the arm center. Possibly posterior part of second left frontal convolution.

Availing ourselves of Wernicke's condensed schema (Fig. 166) most aphasic derangements met in practice are easily explained by it by supposing lesion and interruption of conduction in certain places. According as the centripetal conduction, *m*, *a*, *C*, or the centrifugal conduction, *C*, *b*, *n*, is interrupted we have sensory or motor aphasia, while the interruption of the line, *a*, *b*, produces the conduction aphasia of Wernicke. Lesions between *a* and *b* on the one hand and *C* on the other are called *transcortical* aphasias; between *a* and *b*, *cortical*, and between *a* and *b* on the one hand and the periphery on the other, *subcortical*. These distinctions are schematic.

Derangemems of Speech of Irritative Origin.

In addition to those due to direct lesion of the speech centers there are also derangements of speech due to irritation. Such are the different kinds of stuttering, the labiochoreic and gutturotetanic stuttering and choreic speech. The first two probably reside in the cortical speech centers, but the choreic spasms not necessarily, since the function of muscles concerned in their production may be disturbed from lesions in other centers as well.

Diagnosis.—The study of derangements of speech is by no means an easy matter, but it may be facilitated by pursuing a systematic method like the following, which is that of M. Allen Starr, slightly modified:

A.	To determine whether apraxia is present.	(1) Test the power of recognizing the nature, uses, and relations of objects.
B.	To test integrity of the auditory speech area and association tracts between other sensory areas and the temporal convolutions.	(1) The power to recall the spoken name of objects seen, heard, handled. (2) The power to understand speech and musical sounds. (3) The power to call to mind objects named.
C.	To test the condition of the visual word memories in the angular gyrus and of the connections between this area and the surrounding sensory and motor areas.	(1) The power to understand printed or written words. (2) The power to read aloud and understand what is read. (3) The power to recall objects whose names are seen. (4) The power to write spontaneously and to write the names of objects seen, heard, etc. (5) The power to copy and write at dictation. (6) The power to read understandingly what has been written.
D.	To test the integrity of Broca's center and its association tracts.	(1) The power to speak voluntarily; and if it is lost, the character of its defects. (2) The power of repeating words one after another.

When aphasia is associated, as is so often the case, with paralysis of the right arm, the writing test may be made with the left hand, when the patient may produce the so-called aphasic mirror writing, which can only be read by the use of a mirror; or if he cannot write with the left hand, as also happens, he may be asked to form words by letters cut out of printed pages or with the letter blocks of children.

Recently Marie has announced some iconoclastic views as regards aphasia. He believes hearing is not localized in the first temporal convolution, and denies that impairment of hearing has ever been observed as a result of a lesion of the right first temporal convolution. In aphasia there is more or less difficulty in understanding spoken words due to defect of

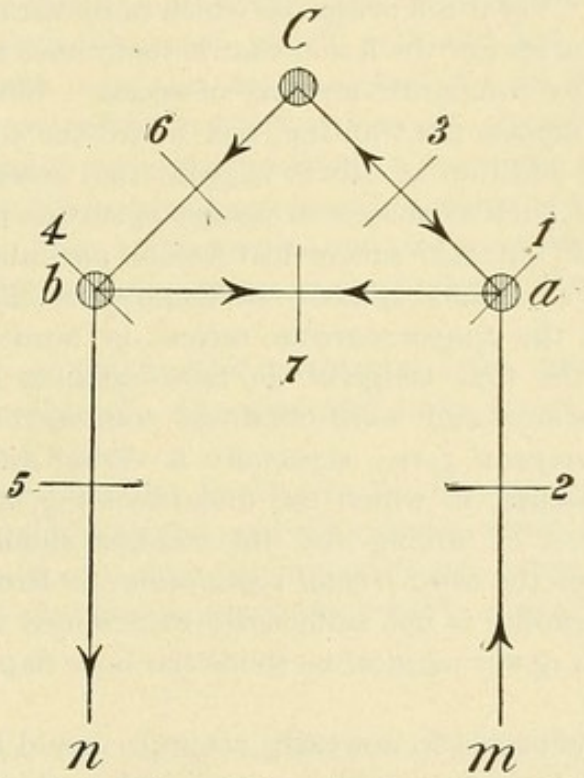


FIG. 170.—Simplification of the Schema of Conscious Speech by Reduction of the Ideas to the Point, C, and Omission of the Acoustic Center, *x*.
The letters have the same application as in previous figures.

intellect, and not to a lesion of the left third frontal convolution, since Broca's area has been destroyed without causing aphasia, and motor aphasia has occurred without a lesion in Broca's area. He declares that the left third frontal convolution plays no part in the function of language, and when a lesion occurs in this convolution the occurrence of aphasia is a mere coincidence. In motor aphasia which Marie calls the aphasia of Broca, the patient cannot read or write and understands imperfectly what is said to him. It is similar to sensory aphasia which he calls the aphasia of Wernicke, from which it differs solely in that the patient cannot speak. In what Marie calls anarthria the patient cannot speak, but understands what is said to him and can read and write. It is the condition described as subcortical motor aphasia. Everyone, says Marie, places the lesion of anarthria in or near the lenticular nucleus, it may be in the right hemisphere. When the lesion is limited to one hemisphere the anarthria tends

to disappear, but is persistent when the lesion is within or near each lenticular nucleus and is usually a part of a pseudobulbar symptom-complex.

He also says aphasia is a unit and that the aphasia of Broca is only aphasia complicated by anarthria. According to Marie, the only region capable of causing aphasia when damaged is the zone of Wernicke, *i. e.*, the supramarginal and angular gyri and foot of the first two temporal convolutions. The aphasia of Broca is caused by a lesion of Wernicke's zone, or of the fibers coming from it, associated with a lesion in or near the lenticular nucleus. The speech zone should not be divided into separate centers. The intensity of aphasia corresponds with the extent of the lesion. Dejerine denies most of Marie's assertions.

Prognosis and Treatment.—Aphasia is a symptom of a disease and not a disease itself. Yet it is a symptom which in its various phases informs us so precisely of the seat of the lesion that it sometimes suggests a point of operative interference comparatively easy of access. Where the symptoms of diffuse cerebral disease are wanting, and where the continuation of the symptoms and the addition of others suggest the presence of a possible circumscribed cause, such as abscess or tumor, operation is justified, and by it not only cortical, but also subcortical lesions and abscesses have been relieved. In cases of *sensory* aphasia the trephine should be applied over the upper part of the *temporoparietal region*, in *word-deafness* over the posterior part of the first *temporal*, in *word-blindness* over the *angular gyrus*, in word-deafness and word-blindness combined over the *inferior parietal* and *first temporal gyrus*, especially if verbal amnesia be present. In purely *motor aphasia*, in which the understanding of language is preserved but the power of talking lost, the trephine should be applied over the posterior part of the *third frontal convolution*, or Broca's center. The lesion of simple agraphia is not sufficiently determined to warrant surgical interference. With newer surgical methods the bone flap is used to expose these various areas.

When urgent symptoms do not exist, attempt should be made to reeducate the patient, and much may be accomplished in this way by perseverance, especially in the young. With adults, the prognosis is more unfavorable, especially in cases of complete motor aphasia associated with right hemiplegia. In them the patient may be taught to write with his left hand.

Cortical Areas Whose Function is Unknown or Uncertain.

After subtracting the motor, visual, and speech areas of the two hemispheres there remain extensive cortical areas, the function of which is more or less uncertain, and which are unexcitable. They include:

1. *The Frontal Region, Including all the Frontal Convolutions except the Posterior Half of the Third Frontal on the Left Side, and if Starr be correct, the Middle of the Second Frontal Convolution on the Left Side where he Locates the Graphic Center.*—Of this area the most that can be said is that, if injured, mental symptoms are quite likely to be prominent—symptoms ascribable to a loss of self-control. It is to the greater development of the region of the frontal lobes in man as compared with the lower animals that his higher mental qualities are ascribed. Various forms and degrees

of dementia have been observed after such lesions, and when such mental symptoms are present, it may with reason be inferred that there is lesion of the frontal lobes, especially of the left frontal lobe according to some investigators, more particularly when the lesion elsewhere can be excluded. It should never be forgotten, however, that the intellectual development depends on the integrity of the entire brain.

2. *The Region of the Cortex Lying between the Rolandic Fissure and the Occipital Convolutions, Including all the Parietal Convolutions except the Left Inferior Parietal Lobule.*—Recent investigations seem to show that this is the chief sensory region of the brain. The recognition of objects by contact when the eyes are closed, the sense of position, and general sensibility have all been found affected in extensive lesions of the parietal lobe.

3. *The Region Covering the Entire Temporosphenoidal Lobe on the Right Side except the First Temporal*, which probably has to do with hearing of ordinary sounds, and the *temporosphenoidal on the left side*, excluding the parts not concerned in hearing of words, as well as ordinary sounds. To the first temporal gyrus the function of hearing is assigned, but the remainder, so far as the cortex is concerned, appears unexcitable. Abscesses are common here after otitis media, and are sometimes reached with the trephine, the diagnosis being based on the presence of otitis with symptoms of brain disease.

4. The *Apex of the Temporosphenoidal Lobe*, including the uncinate convolution. To this the olfactory and taste senses have been ascribed with some show of reason.

5. *Of the Entire Median Surface of the Hemispheres*, except the paracentral lobule, which is motor, and the cuneus, which is visual, and including the gyrus fornicatus and the hippocampal cortex, the function is unknown, although the hippocampal is probably a part of the olfactory and taste area.

Tracts within the Brain—Centrum Ovale, Internal Capsule, Central Ganglia, Corpora Quadrigemina.

Centrum Ovale.—In the centrum ovale, constituting the mass of white fibrous substance beneath the cortex and above the level of the basal ganglia, the fibers of the motor paths are more or less closely associated with other systems of fibers. They include three sets—*projection*, *commissural*, and *association* systems; the first connecting the cortex with nervous structures lying below it, the second joining the two hemispheres, while the third, or association fibers, join different parts of the same hemisphere. By these fibers adjacent convolutions, alternate convolutions, and more distant regions are connected, and through these as a physical basis the activities of the various cortical areas are harmonized and the different memories united.

The *diagnosis* of lesions involving this mass is exceedingly difficult. We can only surmise in cases of *disturbance of association*, such as occur with aphasia and kindred disorders, that the *association fibers* have been destroyed. A break in the continuity of the fibers of the corona radiata must produce the same symptoms as if the corresponding center were

destroyed. Thus disease of the white substance of the *occipital lobe* may cause *hemianopia;* of the *left temporal lobe, word-deafness.* If the coronal fibers which proceed from the *third left frontal convolution* are injured, *motor* or *ataxic aphasia* occurs; yet quite extensive disease of the white substance of the frontal lobe has been found postmortem without any symptoms having been present during life.

Internal Capsule.—Since in the comparatively narrow space in the posterior limb of the internal capsule is centered the pyramidal tract on its way from the cerebral convolutions to the crus cerebri, a very limited focal disease in this locality will lead to hemiplegia on the opposite side, while clinical experience shows that almost all cases of persistent hemiplegia are occasioned by disease in this spot.

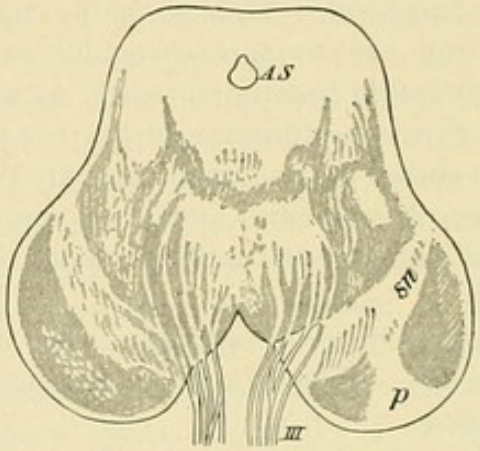


Fig. 171.—Transverse Section through the Crura Cerebri in Secondary Degeneration of the Right Pyramidal Tract—(*after Charcot*).

sn. Substantia nigra. *p.* The degenerated, and therefore translucent, pyramidal tract. *III.* Oculomotor nerves. *AS.* Aqueduct of Sylvius.

According to the views of many neurologists, a purely motor hemiplegia, unattended by impairment of sensation, implies a lesion that does not involve the most posterior portion of the internal capsule, while such involvement is probable when there is sensory disturbance as well as motor paralysis. Dejerine, on the other hand, believes that disturbance of sensation in hemiplegia indicates involvement of the optic thalamus, and that there is no distinct sensory tract in the posterior part of the posterior limb of the internal capsule.

Central Ganglia—*i. e., Caudate Nucleus, Lenticular Nucleus, and Optic Thalamus.*—It is considered that the optic thalamus may have to do with the movements of mimetic or emotional expression, such as laughing and crying, which are lost in lesion of the thalamus, but which remain when the thalamus is intact, even though the half of the face is paralyzed and cannot be moved voluntarily. It is also likely that some of the fibers of the optic tract terminate in the posterior portion of the thalamus known as the pulvinar, while most of the fibers go to the corpus geniculatum externum, and possibly some to the anterior colliculus of the corpora quadrigemina. Hence destruction of the hinder part of the thalamus produces complete hemianopia of the opposite side, usually by destruction of the optic radiations. Focal disease of the thalamus has been

supposed to cause posthemiplegic chorea and other posthemiplegic symptoms of irritation.

It would appear from recent experiments that the lenticular nucleus contains centers for regulating heat, which would explain certain temperature changes in cerebral affections. It is probable that a lesion strictly confined to the lenticular nucleus causes excessive spasticity and partial paralysis of the opposite side of the body.

Beyond this, little definite is known of the effect of lesions strictly limited to the central ganglia, while disorganization of these ganglia has been found unattended by any symptoms during life.

Corpora Quadrigemina and Crura Cerebri.—Lesions of the *corpora quadrigemina* are rare. Not much, therefore, is known of their function. The anterior tubercles are connected with fibers of the optic tract, but the extent of the connection in man is uncertain. Unilateral, or even bilateral, *paralysis of the oculomotor* nerve has been observed in connection with lesions of the quadrigeminal bodies, as have also *nystagmus* and *immobility of the pupil*. But this is because the nuclei of the motor nerves of the eyeball, except the sixth, lie very close to the tubercles, and may, therefore, be involved in such a lesion. According to Nothnagel, a staggering gait with oculomotor paralysis, associated with general symptoms of a tumor, points to the corpora quadrigemina as its site. The oculomotor paralysis is often of irregular distribution, involving especially upward and downward movements of the eye, and should be an early symptom. Tumor of the corpora quadrigemina often causes early optic neuritis.

If a *crus of the cerebrum* is diseased, there often result characteristic symptoms—viz., *paralysis of one side of the body* (arm, leg, and face), and on the side *opposite* the hemiplegia a *paralysis of the motor oculi*, or *third nerve*—crossed paralysis. An examination of Fig. 171 will explain this. A lesion on the right side at *p* in the right pyramidal tract, might involve the oculomotor nerve, III, on that side, but would produce a hemiplegia on the left side. Since the crus contains sensory fibers from the opposite side, a lesion in one crus may also produce hemianesthesia of the opposite side of the body. Tegmental lesions should also produce sensory paralysis.

CEREBELLAR DISEASE.

The cerebellar lateral lobes may occasionally be the seat of extensive lesions which do not produce symptoms. The trunk and lower extremites are chiefly affected in cerebellar ataxia. The patient may be able to lie abed and move his legs much better than when standing, but as soon as he arises he begins to sway back and forth with his whole body. This tendency is increased if he brings his feet together, but is diminished while the legs are widely separated. In this respect cerebellar ataxia does not differ from the ataxia of posterior sclerosis. Closing the eyes may occasionally increase the ataxia, but usually does not do so markedly, because the cutaneous and muscular sensibility of the lower limbs remains normal. So, too, when the patient tries to walk, he totters, but there is none of the stamping gait of tabes dorsalis. It is more the true drunkard's reel, at one

time forward, rolling now to one side and now to the other, but often with a distinct tendency toward one side or backward. Unfortunately, this gait is not so peculiar to cerebellar disease as to be pathognomonic of it, and we can only suggest that the cerebellum or its peduncles may be involved. The upper extremities are usually less affected than the lower, but Hughlings Jackson has called attention to a paresis of the trunk muscles as the result of which the movements of bending, erection, and lateral flexion of the trunk cannot be performed. The head is sometimes carried much inclined toward one side, but it is impossible to use this sign to determine with certainty the side on which the lesion is situated.

The *vertigo* of cerebellar disease, if severe, is one of the most distressing symptoms with which one can be afflicted. It varies greatly and is not constant, while it may be the only symptom. It occurs, however, under the same circumstances as the ataxia—that is, when the patient stands or moves about, disappearing when he lies down. The vertigo and ataxia are not necessarily associated, and either may be present and the other absent.

Headache is a frequent symptom in cerebellar disease, having been present in 83 out of 100 cases collected by W. C. Krauss. Most frequently it is occipital; more rarely there may be pain in the side of the head or in the forehead. *Vomiting* is also a result of chronic disorders of the cerebellum, being present in 69 of Krauss' cases. So is *visual disturbance* due to optic neuritis, which was found in 66 cases. None of these symptoms is pathognomonic, and each one may be a symptom of disease elsewhere in the brain. The most valuable, perhaps, is the cerebellar gait. It might be expected that retained reflexes would be a distinctive sign as contrasted with their absence in tabes dorsalis; but, in fact, they are sometimes absent, this being the case no less than 12 times in Krauss' 100 cases.

Other symptoms which suggest cerebellar disease, but are not distinctive, are *neuralgic pains* in the region of the neck and occiput; blocking of the venæ Galeni and dilatation of the lateral ventricles producing *hydrocephalus* in children; pressure on the medulla oblongata, causing *paralysis* of the *cranial nerves*, *glycosuria*, or even *sudden death*, incoordination and asynergy of movements; finally *bilateral rigidity* from pressure on the motor paths. On the other hand, there may be cerebellar disease without any symptoms whatever, especially as long as the middle lobe is not involved.

Form of Lesion.—By far the most frequent cerebellar lesion is *tumor*—in fact, some sort of tumor was found by W. C. Krauss in 88 out of 100 cases, of which ten were *abscess*, and there was one each of *softening* and *hemorrhage*. The remainder were: sarcoma and tubercle, each 22; glioma, 18; nature of tumor unspecified, 13; cyst, seven; and one case each of endothelioma, cyst and sarcoma, cancer, gumma, and fibroma. It is probable that some of those tumors classed as sarcoma were glioma. The tumor occupied one or the other hemisphere 32 times; the middle lobe, 17 times.

Disease of the middle cerebellar peduncles may be accompanied by the so-called *forced positions* and *forced movements*. As a result of the former, the subject may lie in bed upon a particular side, whether conscious or unconscious; and if put on the other side, may reassume his former position involuntarily. Sometimes this is accompanied by a corresponding forced position of the head and eyeballs, the extremities being seldom affected. The

forced movements are less frequent. They consist either in oft-repeated rotations of the body on its longitudinal axis or, if the patient can walk, in involuntary circular movements. There is no guide by which to determine which of the two peduncles is affected under these circumstances, while in a few cases of brain disease the same symptoms have been observed without involvement of the cerebellum.

The following very convenient summary from Strümpell's "Textbook," somewhat altered, American edition of 1910, contains the most important facts bearing on the localization of cerebral disease, and will be found useful for reference:

"1. The most frequent cause of ordinary hemiplegia is a lesion of the *pyramidal* tract in the posterior limb of the internal capsule. If the hemiplegia be persistent, then this tract is actually destroyed; if temporary, the tract has been functionally deranged for a time by focal disease in neighboring parts of the brain.

"2. Monoplegic cerebral paralysis is usually due to affections of the *cortex* of the brain—that is, the anterior central convolution and the paracentral lobule. Monoplegia of the face and tongue is the result of lesions in the lower extremity of the anterior central convolution. Monoplegia of the arm is referable principally to some lesion of the middle third of the anterior central convolution. Monoplegia of the lower extremity implies some affection of the upper portion of the anterior central convolution and the paracentral lobule.

"3. Hemiplegia or monoplegia, if associated with epileptiform convulsions affecting either one-half or one particular portion of the body, is almost always caused by cortical lesions or lesions near the cortex. These same symptoms of motor irritation without accompanying paralysis are likewise to be ascribed to some irritation of the above-mentioned regions of the cortex.

"4. Hemiplegia with crossed paralysis of the oculomotor nerve developing at the same time indicates a lesion of a crus cerebri. Co-existing tactile hemianesthesia implies that the tegmentum is involved.

"5. Hemiplegia with crossed facial paralysis implies, provided that the paralysis of the limbs and opposite side of the face occurred at the same time, that the lesion is situated in the pons.

"6. Posthemiplegic chorea (*vide infra*) seems to occur especially when there is focal disease in the neighborhood of the posterior part of the internal capsule. It is a rare phenomenon.

"7. Hemianesthesia of the skin, associated with hemiplegia on the side of the anesthesia and hemianopia developing at the same time, is due chiefly to lesions of the most posterior portion of the internal capsule.

"8. Hemianopia may be due to a lesion of the cuneus and neighboring parts in the occipital lobe. Probably, also, a lesion of the posterior extremity of the internal capsule may cause it, in which case it is usually associated with hemianesthesia. Finally, it may be produced by affections of the pulvinar of the optic thalamus, of the lateral geniculate body, or of one of the optic tracts.

"9. Genuine motor aphasia indicates disease of the foot of the third left frontal convolution. Unless we accept the teaching of Marie.

"10. Word-deafness (loss of understanding of speech) is due to disease of the first left temporal convolution or of the posterior convolutions in the island of Reil; word-blindness (loss of understanding of writing) is due to disease of the left lower parietal lobe (angular gyrus)—supramarginal gyrus also, according to Ferrier.

"11. Difficulty in articulation implies disease of the medulla oblongata, as does also dysphagia.

"12. Staggering gait and vertigo are the most constant symptoms of cerebellar disease, but they may also occur in diseases of the corpora quadrigemina and of the frontal lobe (*vide supra*). Forced positions and forced movements perhaps indicate lesions of the crura cerebelli ad pontem or of the optic thalamus.

"13. Staggering gait and ocular paralysis implicating the third and fourth nerves are indicative of lesions of the corpora quadrigemina."

DISEASES OF THE CRANIAL NERVES.

OLFACTORY NERVE.

The olfactory fibers may be affected in their intracerebral course in the rhinencephalon or in their distribution to the olfactory region of the nose. There are probably nerve cells in the frontal lobe belonging to the rhinencephalon, and nerve tracts belonging to the rhinencephalon pass through the frontal lobe.

Morbid Anatomy.—The lesions may be *tumors of the brain*, instances of which have been found in the hippocampal gyri, or disease in the hemispheres. There may be *congenital defect* of the olfactory center or atrophy of the nerve, which may explain the occasional anosmia in tabes dorsalis. There may be *inappreciable changes*, caused by injuries to the head or by concurrent disease, such as epilepsy, the aura of which is sometimes manifested by parosmia. The area of distribution of the olfactory nerve in the nose may be destroyed by *chronic nasal catarrh* or by *polypi*. *Hysterical neuroses* of the olfactory nerve are not infrequent. The sense of smell is sometimes impaired in cases of tumor situated in portions of the brain remote from the olfactory area. This possibly may be caused by increased intracranial pressure.

Symptoms.—Lesions in any of these localities may produce subjective sensations of smell, or *parosmia*, of which various foul odors are illustrations; hypersensitiveness of the normal sense, or hyperosmia, in certain highly developed degrees of which the patient, generally a highly sensitive woman, can distinguish one person from another by the sense of smell; or loss of the sense of smell, *anosmia*.

Diagnosis.—The nasal region should be carefully explored by the rhinoscope and the sense of smell should be tested. For this purpose the essential oils, such as anise-seed, cloves, or peppermint, in various degrees of dilution are employed. Cologne water, musk, or asafetida may be used for the same purpose. Pungent substances should be avoided, as they stimulate the fifth nerve in the nasal mucous membrane, and thus the subject perceives what he does not smell. By such agents the fifth nerve is tested. No

conclusion can be drawn as to anatomical differences on the two sides without a rhinoscopic examination.

Treatment is useless, unlesss the condition be due to curable or removable polypi or other nasal condition.

OPTIC NERVE AND TRACT.

There may be derangement of the retina, of the optic nerve, of the chiasm, and of the optic tract.

I. Affections of the Retina.

These may be *organic* or *functional*.

(*a*) *Organic Diseases of the Retina.*

The organic affections include hemorrhage and inflammation, or both.

Hemorrhage into the retina (arterial sclerosis) occurs as a cause or result of Bright's disease, most commonly chronic interstitial nephritis, in gout profoundly affecting the system, in leukemia, anemia, syphilis, purpura, and in ulcerative endocarditis, and other forms of septicemia. The hemorrhages are in the layer of the nerve-fibers. At first bright red, and then becoming darker and eventually lighter in color, they ultimately assume a diffuse cloudiness, owing to serous infiltration. The hemorrhages vary in extent, and often follow the course of the vessels. In septicemia they are due to capillary septic embolism, and often have white spots in the center, owing to the massing of leukocytes. Other white spots are due to fibrinous exudate, fatty degeneration of the retinal elements, or localized sclerosis of the same. Similar hemorrhages sometimes occur in the pia mater in the same cases.

Retinitis occurs under the same circumstances as hemorrhage, especially in chronic nephritis, syphilis, anemia, leukemia, and also malaria; and in diabetes mellitus and chronic lead-poisoning.

Albuminuric retinitis may occur in all forms of chronic nephritis, more frequently in the interstitial variety, of which disease it may be the earliest symptom recognized. It is characterized in general by the presence of white spots of various extent and distribution, as seen by the opthalmoscope. They are caused by degenerative processes and hemorrhages. Gowers recognizes three forms:

1. A degenerative form, which is the most common, in which there is retinal changes, but scarcely any alteration of the optic disk.

2. An inflammatory form, in which there is much swelling of the retina with obscuration of the optic disk.

3. A hemorrhagic form, in which there are many hemorrhages, but little evidence of inflammation.

In some instances of the second type the inflammatory changes in the optic nerve predominate over those of the retina, producing an *optoneuritic* form, in which the appearances are more closely allied to those of papillitis or choked disk, such as is caused by intracranial disease.

Syphilitic retinitis is a rare affection in acquired and congenital disease. In the latter it is called *retinitis pigmentosa*. *Syphilitic choroiditis* is less rare. Retinitis is not uncommon in *chronic anemia*, especially in the pernicious form. After excessive loss of blood the patient often becomes blind, either suddenly or in the course of one or two days. In such cases a neuroretinitis has been found quite sufficient to explain the blindness, which, in rare instances, may be permanent and complete. A rare variety of anemic retinitis is *malarial retinitis*, first described by Stephen MacKenzie. It may be associated with hemorrhage. In *leukemic retinitis* the retinal veins are large, and hemorrhage may also occur, with white and yellow areas. Tumor of the brain, especially of the cerebellum, has been found in some instances to cause a condition of the retina like that of abuminuric retinitis.

(*b*) *Functional Disturbance of the Retina, or Amaurosis.*

This may be *toxic*. Of this, the most striking and best known variety is *uremic amaurosis*.[1] Its suddenness is its most striking feature, and it is very frequently the forerunner of uremic convulsions, although it may occur without them. It, too, may be the first symptom noted in Bright's disease. The retina is free from any changes visible by the ophthalmoscope, and the condition is probably due to the action of the poison on the nerve centers. It generally disappears, not quite so suddenly as it comes on, but comparatively quickly, while the impaired vision of retinitis albuminurica is a more or less permanent condition. Similar are the amauroses from *lead-poisoning* and from massive doses of *quinin*. *Hysterical amaurosis* is more frequently a dimness of vision—amblyopia—but true blindness may occur in one or both eyes. *Tobacco amblyopia* is usually gradual in its appearance, and affects more especially the center of the field of vision. There may be congestion of the optic disk and if the use of tobacco is persisted in, there may be a permanent organic change, with atrophy of the disks. A scotoma for red and green is invariably present.

In *nyctalopia*, or night-blindness, objects are clearly seen by the day or by strong artificial light, but are invisible in the shade or at twilight. In *hemeralopia* the reverse state of affairs exists, objects being seen with discomfort in bright daylight or by strong artificial light, but being easily seen in deep shade or twilight. *Retinal hyperesthesia* is sometimes met in hysterical women.

2. Affections of the Optic Nerve.

Those which are of medical significance are *optic neuritis*, or choked disk, and *optic atrophy*.

(*a*) *Intracranial Trunk.*

The intracranial trunk of the nerve is rarely affected, by reason of its shortness. It may, however, be compressed by a *tumor* in adjacent parts, as of the pituitary body or of the bone; by *aneurysm of the ophthalmic artery*

[1] Amaurosis is a vague term usually defined as partial or tetal loss of vision. For partial loss of vision amblyopia is now commonly applied, while the less obscure term blindness is best used for total loss of vision.

within the orbit, or of the *internal carotid* within the skull. The trunk may also be the seat of inflammation, which may extend from carious bone or meningitis.

(*b*) *Optic Neuritis, Papillitis, Papillo-edema, or Choked Disk.*

Definition.—Inflammation of the intraocular end of the optic nerve.

Anatomical.—It will be remembered that the optic nerve pierces the sclerotic and choroid coats about 1/10 inch (2.5 mm.) to the nasal side of the center of the retina, which is occupied by the yellow spot of Sömmering. In this spot the sense of vision is most nearly perfect, while the optic papilla or disk is the only part of the retina from which the power of vision is absent. A central depression, or "cup," is due to the separation of the nerve fibers, pale because of the absence of blood-vessels, while the periphery of the disk has a rosy tint from the presence of the minute blood-vessels that lie among

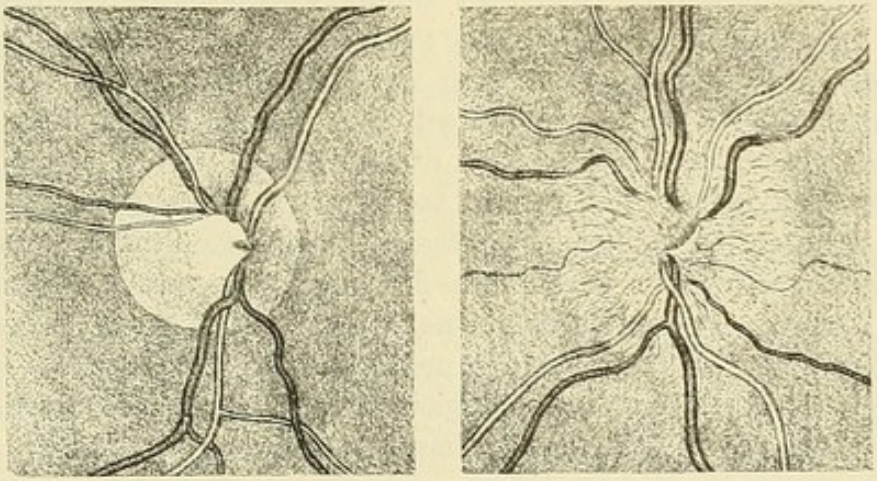


FIG. 172.—Commencing Optic Neuritis from a Case of Caries of the Sphenoid Bone with Secondary Meningitis—(*after Growers*).

The left-hand figure shows the normal right optic disk with clear outline and deep central cup. The right-hand figure of the left papilla shows well-marked neuritis; the edge of the disk is concealed by a swelling which extends beyond the normal limits of the disk. The central cup is encroached upon, but not quite obliterated. Some of the vessels are partly concealed at their points of emergence, and the veins lose their central reflection.

the nerve fibers. The "cup" varies in size, and may be absent, the vascular portion of the disk at times extending over it. The tint of the vascular portion of the disk also varies, and differences are of significance only when noted at successive examinations of the same case.

Morbid Anatomy.—It is by swelling and diminished transparency rather than by recognizable signs of congestion that the *first stage* of optic neuritis is characterized. Then there follows lessening of the sharpness of the edge of the disk, and finally its total obscuration, as seen in the right half of Fig. 172 as contrasted with the left half. It is to be remembered that the normal contrast is sometimes diminished within the limits of health, with this difference: that the pathological indistinctness is better seen with the direct method of examination, while the indistinctness sometimes normally present is more evident to the indirect. The abnormal change is earlier recognized on the nasal side, because there are more nerve fibers

there than at the temporal edge. In the *second stage* the swelling rapidly increases and the whole circumference of the disk disappears, though the cup is still represented by a slight depression. The swelling extends even beyond the normal disk, becoming two or three times as wide. The swollen disk assumes a red or grayish-red color to the indirect examination, but by the direct a fine striated appearance is noted, the striæ radiating from the center of the disk in the direction of the fibers. White spots may appear on its surface, due to degeneration of the nerve fibers, and may be seen in the illustration. As the swelling increases the retinal vessels, at first unaffected, become affected by the compression, the veins becoming wider and more tortuous, the arteries being narrowed or remaining normal, while hemorrhages may occur. The retina may also be invaded, producing a neuro-retinitis.

In very slight degrees of inflammation the swelling subsides and recovery takes place. In high degrees it remains for a long time, owing to the presence of infiammatory products, which gradually, however, undergo the usual contraction of cicatricial tissue, a condition of "consecutive atrophy" resulting, in which the disk is white and atrophied.

Etiology.—Most commonly optic neuritis is caused by intracranial disease; especially in nine-tenths of all cases tumor is said to be present. It gives no information as to the seat of the tumor. It is ascribed by some to a descending neuritis; by others, to intracranial pressure. In over 90 per cent. of cases the neuritis is bilateral, though often unequal in the two eyes. Unilateral neuritis is generally due to disease within the orbit or at the optic foramen, but may also be due to intracranial tumor. *Meningitis*, either tuberculous or simple, is the next most frequent cause. It is said to be rather more common in meningitis of the base than of the convexity. Such optic neuritis is less severe than that caused by tumor. *Cerebral abscess* may cause it; so may *diffuse cerebritis*. In *thrombotic softening* and *hemorrhage* optic neuritis is rare, but in *embolic softening* it is more common.

Optic neuritis may result from *Bright's disease*, *chlorosis*, *anemia* or *lead-poisoning*, and may occur after *acute fevers*, especially scarlet and typhoid. In the latter it may be associated with brain symptoms, especially headache. About 6 per cent. of all cases of *multiple sclerosis* are accompanied by optic neuritis, due to inflammatory or sclerotic patches in the nerve, usually slight and of short duration, often one-sided in consequence of unilateral involvement of the nerve by a sclerotic patch.

Symptoms.—Mild degrees of optic neuritis may be without symptoms, except such as are revealed by the opthalmoscope. With higher degrees, acuity of vision, color vision, and the visual field all become affected and may be lost. Its severest effect on vision may not appear until contraction sets in, because it is at this peirod that the nerve elements suffer most in integrity. The defective sight is not, however, necessarily due to changes in the disk or retina; it may be due to intense inflammation in the nerve behind the eye or to intracranial disease.

Prognosis.—Even in severe cases there may be some improvement of vision with subsidence of the inflammation. On the other hand, vision may be permanently lost.

(c) *Optic Atrophy.*

There are three varieties of atrophy of the optic nerve:

(1) Primary; (2) secondary; (3) consecutive.

1. *Primary* or *simple atrophy* is that form which is not preceded by any recognizable inflammatory change in the papilla or surrounding structures. It occurs in degenerative diseases of the brain and spinal cord, more frequently in multiple or disseminated sclerosis and tabes dorsalis. It is present in about 40 per cent. of all cases of multiple sclerosis, and, in various degrees, in at least 15 per cent. of those of tabes. In dementia paralytica it is present in about 5 per cent. Primary atrophy is sometimes hereditary, occurring in the males of a family after puberty. Other causes to which the condition has been ascribed are cold, alcoholism, lead-poisoning, sexual excesses, diabetes, and the specific fevers.

2. *Secondary atrophy* is the result of damage to the optic nerve behind the eye or at the chiasm. It is characteristic of it that demonstrable signs of atrophy follow, instead of accompany, the deranged vision; of which, too, hemianopsia may be a form.

3. *Consecutive atrophy* is that form of atrophy which succeeds neuritis or papillary neuritis. It has the same causes and the same significance. Only secondary and consecutive atrophy are the result of uncomplicated intracranial diseases; for although primary atrophy accompanies disseminated sclerosis, tabes dorsalis, and general paralysis of the insane, it is not caused by the associated brain disease, but is the result of the same widespread tendency to degeneration.

The ophthalmoscopic appearances in primary atrophy differ somewhat from those of the consecutive and secondary forms, the disk being gray-tinted—whence the name *gray atrophy*—with its edges well defined, while the arteries appear almost normal. In secondary and consecutive atrophy the disk has an opaque, white appearance, with irregular outline, and the arteries are small.

The **symptoms** of optic atrophy are the defects of vision already detailed when treating of optic neuritis.

As to **prognosis,** in primary atrophy the ultimate result is usually blindness, but in secondary consecutive atrophy some vision remains, even in severe cases, while in mild cases recovery is not impossible.

3. Lesions of the Chiasm and Tract.

Anatomical.—The decussation of the optic tracts at the chiasm is peculiar. As it reaches the chiasm each tract divides and sends a portion —the smaller—of its fibers to the temporal half of the corresponding retina, and the remaining portion to the nasal half of the opposite retina. Thus the right tract supplies the right or temporal half of the right retina and the right or nasal half of the left retina; the left tract supplies the left or temporal half of the left retina and the left or nasal half of the right retina. The decussating fibers occupy the middle of the chiasm, and the direct fibers the corresponding side. (See p. 173.)

Effect of Lesion of the Chiasm: Hemianopia.—(*a*) If the central portion of the chiasm, composed of decussating fibers only, is involved (lesions *b*

and *c*, Fig. 173), the result will be anesthesia of the inner half of each retina and blindness of the outer half of each field of vision, it being remembered, of course, that the half field which is blind is the reverse of the half of the retina which is anesthetic, since the picture formed on each half of the retina is projected from the opposite half of the field of vision. Such half blindness is known as *hemianopia*, and the form just described, in which outer or temporal half of each field is blind, is known as *bi-temporal hemianopia*.

(*b*) If the whole chiasm is involved, as is not infrequently the case as the result of pressure by tumor, there will, of course, be total blindness.

(*c*) If the lesion is intermediate, involving the direct fibers on one side of the chiasm as well as the central fibers, there will then be blindness in one eye and temporal hemianopia in the other.

(*d*) The rarest of all forms of hemianopia is *bi-nasal hemianopia*, due to a symmetrical lesion involving only the direct fibers passing to the temporal half of each retina, whence results blindness in the nasal field only. It may be caused by tumors involving the outer part of each tract, or of each optic nerve, but the more common lesion is inflammation.

Effect of Unilateral Lesion of the Tract.—If there be a lesion involving the left tract at *d* (Fig. 173), the left or temporal half of the left retina and the nasal half of the right retina become anesthetic and useless, the right half of each field of vision is blotted out, and there results a right *lateral hemianopsia* which is called *homonymous hemianopia*. The reverse is the case if the lesion is in the right tract. The number of cases involving the right side is about equal to the number involving the left. When the left half of one field and the right half of another is blind, or the reverse, the condition is known as *heteronymous hemianopia*.

In the usual forms of bi-temporal hemianopia the obscure fields are by no means always exact demi-fields, which would be the case if the dividing line passed exactly through the fixing point, or macula lutea. It may diverge to temporal side so as to leave a small area around this within the seeing half. These differences are due to peculiarities in the decussation rather than to the lesion. The half fields which remain frequently are contracted. This is usually due to an inflammatory affection of the peripheral fibers of the optic nerves in front of the chiasm.

There are other differences in the dividing-line, such as obliquity, want of sharpness, etc., due to the same cause, but minute description of these belongs to special works on nervous diseases. Since vision remains intact in the central region, equally in right and left-sided hemianopia, it follows that there must be a passage of fibers from the macular region to the optic tract of each hemisphere, else this region would be blinded by disease of one or the other tract. There is usually the same loss of vision for color in the half field, but half vision for color may be lost in central disease without any change in the field for white. This is known as *hemiachromatopia*.

4. Lesion of the Tract and Cortical Centers.

The optic tract on each side crosses the crus cerebri backward and sends fibers to the external geniculate body, to the pulvinar of the optic thalamus, and to the anterior colliculus of the quadrigeminal body. From these so-

called primary optic centers fibers pass backward through the posterior part of the internal capsule, forming the fibers of the optic radiation in the white substance of the occipital lobe, into the visual area of the cortex, of which the area about the calcarine fissure is the chief cortical center,

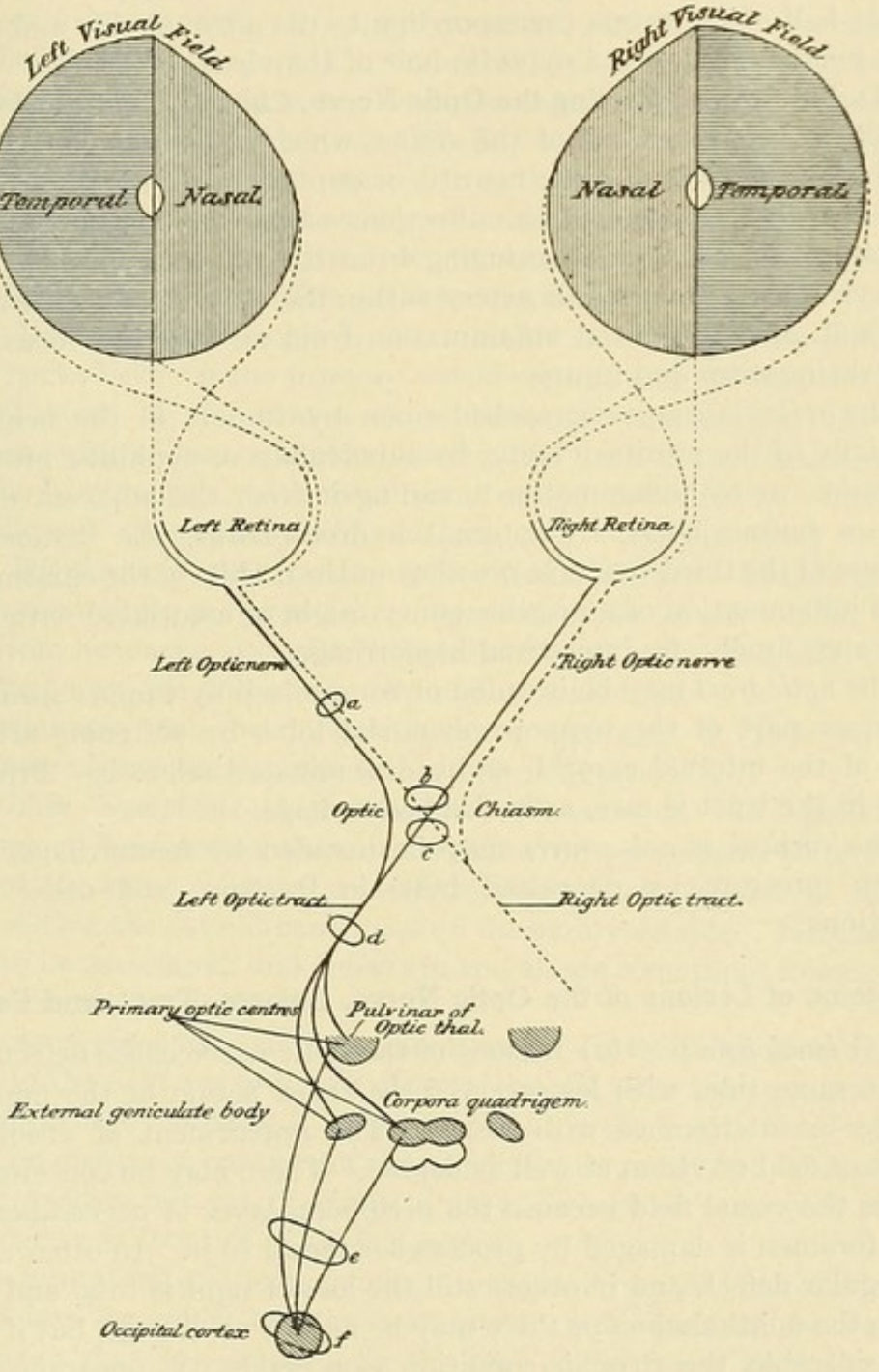


FIG. 173.—Diagram of Course of Optic Nerve-fibers from the Cortex to the Retina—(*after Sahli, Modified and Extended*).

though other parts of the occipital cortex possibly also receive and store up visual impressions.

Whence it is plain that vision may be influenced by lesions in any of the following situations:

1. In the tract itself.
2. In the external geniculate body.

3. In the pulvinar of the optic thalamus and in the anterior colliculus of the corpora quadrigemina.

4. In the fibers passing from the primary optic centers to the occipital lobe, as at *e* (Fig. 173), in the hinder part of the optic radiation.

5. In the area about the calcarine fissure.

The effect of lesion in any one of these situation is to produce anesthesia of that half of the retina corresponding to the affected side and a homonymous hemianopsia of the opposite half of the visual field.

Morbid States Affecting the Optic Nerve, Chiasm, Tract, and Centers.—Outside of the affections of the retina, which concern the ophthalmologist chiefly, and outside of optic neuritis or papillitis as a result of intracranial disease, already considered, the affections of the *optic nerve* which concern the physician are tumors springing from the pituitary body or the bone, aneurysm of the ophthalmic artery within the orbit or of the carotid within the skull, and interstitial inflammation from an adjacent focus, or rarely from rheumatism and injury.

The *optic chiasm* is encroached upon by tumors in the neighborhood, especially of the pituitary body; by tuberculous or syphilitic growths in its substance, or by inflammation invading it from the adjacent dura mater or from carious bone; by internal hydrocephalus, the distended infundibulum of the third ventricle pressing on the middle of the chiasm; by interstitial inflammation of a possible gouty origin or associated with tabes dorsalis; and, finally, by interstitial hemorrhage.

The *optic tract* may be invaded or compressed by tumors springing from the inner part of the temporosphenoidal lobe, by softening after thrombosis of the internal carotid, or by disseminated sclerosis. Primary softening in the tract is rare, as is also hemorrhage.

The *cortical visual centers* may be invaded by hemorrhage, softening, tumors, pressure by depressed bone in fracture, and other traumatic conditions.

Symptoms of Lesions of the Optic Nerve, Chiasm, Tract, and Cortex:

1. *Visual Effects.*—(*a*) Lesions of the optic nerve cause defects of vision on the same side, with lessening of the reflex action of the pupil proportionate to interference with vision. The impairment of vision includes extent of field of vision as well as degree. There may be concentric limitation of the visual field because the peripheral layer of nerve-fibers near the optic foramen is damaged by processes external to it. In other cases there is irregular defect, and in others still the loss of sight is total and lasting.

To the ophthalmoscope there may be at first no change, but if the lesion is considerable, the atrophic condition soon makes its appearance "secondary" to changes in the nerve as distinguished from "consecutive" atrophy, which succeeds papillitis. There may be slowly supervening atrophy without recognizable papillitis. Central loss of vision, due to axial neuritis, is less common, but occurs sometimes in tobacco amblyopia.

(*b*) In lesions of the chiasm the characteristic symptom is bitemporal hemianopia, or loss of the outer half of each field of vision; this is because the lesions mainly affect the chiasm at its central portion, where the fibers, after decussating, pass to the nasal half of each retina. Usually, however,

the process, be it tumor or inflammation, which causes temporal hemianopia extends laterally, involving the non-decussating fibers of one side of the chiasm, causing total blindness of the corresponding eye; or, if extending to both sides, blindness of both eyes. The different stages may often be traced in a single case as the disease progresses. The term "oscillating bitemporal hemianopia" is applied to a rapid and frequent variation of the dark fields, and is regarded as more or less clearly diagnostic of basal syphilis, such as gumma or syphilitic meningitis. More rarely we have the binasal hemianopia already described. Slight variations in the extent of the dark fields have been referred to as the result of peculiarities in decussation rather than of lesion or seat of lesion.

(*c*) In lesions of the optic tract between the chiasm and the external geniculate body there is bilateral hemianopia.

(*d*) Bilateral hemianopia is also a result of lesion of the central fibers of the nerve between the primary visual centers and the cerebral cortex.

(*e*) Lesions of the cuneus cause bilateral hemianopsia. A lesion in each hemisphere, destroying the visual paths back of the chiasm, will cause a double hemianopsia, with total loss of vision in both eyes. Such a result has followed successive lesions in the two occipital lobes.

(*f*) Hemianopia may be due to functional disease. Transient hemianopia is sometimes a symptom of migraine, either as an isolated symptom apart from headache and gastric disturbances or associated with them. It may affect now one half of the field and now another.

Hemianopia has been reported as a symptom of hysteria, but the occurrence of true hemianopia in hysteria is questioned.

2. *Other Symptoms Associated with Hemianopia.*—In about one-half the cases of hemianopia there is transient or permanent hemiplegia, the result of the same lesion, the hemiplegia being on the side of the loss of vision, so that the patient cannot see on the paralyzed side. Hemianesthesia may also be associated, and defects in speech are sometimes found when the paralysis is on the right side.

Hemiachromatopia has been mentioned. In this condition there is no change in the field for ordinary objects, but all colors appear gray as soon as the vertical line is passed. The symptoms, according to Gower, probably depend on disease of one part of the occipital lobe, and is proof of a separate center for color not yet precisely located, perhaps in some part of the occipital cortex in front of the apical region.

The limitation of the remaining functionally active half field and the isolated loss for colors are the best known differences in the features of hemianopia due to variations in the seat of the lesion in the optic path behind the chiasm; the limitation of the active half field indicating a lesion in the optic radiation near the thalamus, the isolated loss for colors pointing to a lesion in the occipital lobe.

3. *Amblyopia.*—Amblyopia is another form of sight defect due to brain disease. The term is used to indicate a *partial loss or blurring of vision.* There is concentric limitation of the visual field, different in different cases, and along with it the color fields are also reduced.

Similar eye defects, associated with hemianesthesia, occur sometimes in hysteria, with which it may be confounded. Since a simple functional

loss of vision may rarely result as a reflex from irritation of the fifth nerve or from hysteria, so a functional amblyopia, affecting both eyes, may also result from such causes—indeed, is more common than the organic form. A carious tooth may act in this way. Amblyopia from errors of refraction must not be confounded with the amblyopia due to brain disease.

Diagnosis.—How shall we interpret these phenomena of vision concerned with the optic nerve and tract? Some conclusions are easy; others are difficult, because of our limited knowledge. Accurate investigation of fields of vision, with a view to the study of hemianopia and other defects in the visual fields variously caused, is made by means of the perimeter, for directions concerning the use of which instrument the student is referred to works on ophthalmology. Herman Sahli suggests an easy, rough method, quite sufficient for recognizing marked difference in the field of vision, performed as follows: The physician seats himself opposite the patient, whose right eye—supposing this to be the one to be tested—is opposite the physician's left, the other eye of each being closed. The two open eyes being thus fixed opposed, the physician passes his finger to and fro across the field of vision exactly midway between the two eyes. In this way he can compare his own field of vision with that of the patient, noting at what moment the finger is seen approaching from the periphery of each. Care must be taken that the finger is kept exactly midway between the physician and patient, and in order to do this, the examiner may from time to time open his closed eye.

Defective sight in one eye with diminished reflex action of pupil proportionate to the defect, the function of the remaining eye being intact, usually means disease of one optic nerve. In some rare cases of functional disease in which the sight of one eye only is involved, the perfect responsiveness of the pupil distinguishes it from organic disease of the nerve. Total loss of sight in both eyes may mean chronic atrophy, damage to the chiasm, or disease of both tracts or in both hemispheres. In these cases the symptoms are at first partial, and in this way the diagnosis is aided.

Central scotoma means damage to nerve fibers in the center of the trunk of the optic nerve, either inflammatory or the result of hemorrhage. Peripheral limitation of vision means damage to fibers running in the periphery of the nerve. Sectorial blindness in one eye means disease of the nerve, decided in degree but limited in extent.

Bitemporal hemianopia means disease of the chiasm, while the combination of complete blindness of one eye with temporal hemianopia in the other means disease of the chiasm which has extended to the outer fibers, and even to the optic tract or optic nerve, on the side on which blindness is complete.

Bilateral hemianopia may be due to disease back of the chiasm, and the determination of the spot involved in the tract between the chiasm and the occipital area which is the cortical center of vision, stimulates diagnostic acumen. The most that can be attempted is the settling of the question as to whether the disease is in the tract between the chiasm and the external geniculate body or in the fibers beyond in the visual center of the hemisphere. To this end the *hemianopsic pupillary* reaction of Wernicke is sought. A perfect pupil reflex requires the integrity of the retina, of the

fibers of the optic nerve and tract, of the nuclei and fibers of the third nerve, and of the iris. When the light is thrown on the blind half of the retina the pupil contracts as much as if it is thrown on the seeing half, if the disease is in the hemisphere; but if the disease is in the tract, it does not contract because the path to the third nucleus below the corpora quadrigemina is interrupted. The employment of the test requires much care and experience. Seguin directs that the patient, being in a darkened room with a light behind his head in the usual position, be directed to look to the other side of the room, so as to eliminate accommodation movements. Then a faint light is thrown upon the eye from a plane or large concave mirror, held well out of focus, and the size of the pupil is noted. With the other hand a beam of light, focused by an ophthalmoscope mirror, is then thrown directly into the optical center of the eye, then laterally in various positions and from above and below the equator of the eye, noting the reaction at all angles of incidence. According or not as a response is obtained in the pupil the inference is drawn.

Amblyopia with concentric reduction of the field, decided in one eye and slight in the other, may be due to atrophy of the nerve, to disease of the distal visual center in one hemisphere, or to hysteria. If atrophy, the ophthalmoscope recognizes the lesion and the responsiveness of the pupil is diminished. If disease of one hemisphere, the nerve is normal to the ophthalmoscope, the pupil contracts perfectly under the action of light, and the onset is sudden or accompanied with other signs of organic brain disease. Mind-blindness, may also be a result of lesion in this locality. In hysteria the symptoms are the same as in disease of the higher visual center, and the diagnosis depends on the presence or absence of signs of organic or functional disease. In hysterical blindness the loss of sight is rarely complete. In hysterical and neurotic defects of vision there may be a derangement of the natural relation in color fields. Thus, while normally the blue field is most conspicuous in the last-named conditions, it is often overshadowed by other colors.

LESIONS OF THE MOTOR NERVES OF THE EYEBALL.

Anatomical.—The third cranial nerve (oculomotor) supplies the levator palpebræ superioris, the superior, inferior and internal recti, the obliquus inferior, the sphincter of the iris, and the ciliary muscle. The fourth cranial nerve (the trochlear) supplies the superior oblique; the sixth cranial nerve (the abducens), the rectus externus. The functions of the muscles to which these nerves are distributed are sufficiently indicated by their names.

Third Nerve.—Lesions may involve the nerve at its nuclear origin or in its course. Lesion of the third nerve *at its origin* involves also usually the origin of the fourth nerve, as a result of which the eyeball is motionless, except for outward movement, and an object moved about in front of it can be followed only by moving the entire head. The nerve may be invaded *in its course* by traumatic causes, meningitis, gummata, aneurysm, or neuritis, frequently rheumatic, and may also be affected in diphtheria, tabes dorsalis, and diabetes mellitus. The effect may be spasm or paralysis.

The results of *spasm* of the muscles supplied by the third nerve are

manifested in *nystagmus*, although nystagmus is not invariably indicative of spasm. This consists in an involuntary, clonic, rhythmical, oscillatory movement of the eyeball, usually horizontal, but sometimes rotary, more rarely vertical. It is seen in congenital or acquired brain lesions, and is often a striking feature in albinism. In meningitis and hysteria there is also sometimes spasm of the muscles supplied by the third nerve, especially the internal rectus and the levator palpebræ, the antagonist of the orbicularis.[1]

Paralysis of those muscles supplied by the third nerve, which include all the eye muscles except the external rectus and the superior oblique, results in outward squint; ptosis, or drooping of the upper eyelid; the absence of contracting power in the pupil, which remains of medium size; loss of accommodation; double vision, or diplopia. Such paralysis, involving all the branches of the nerve, may be recurrent, especially in women, rarely at the menstrual period, and usually at wider intervals.

It is sometimes associated with pain in the head and at other times with migraine. The individual attack lasts a few days, or as many weeks, Partial involvement of the third nerve may include the levator palpebræ. the superior rectus, the ciliary muscle, and the iris, while the external muscles—that is, the internal and inferior recti and the inferior oblique—may escape.

Ptosis only, due to paralysis of the levator palpebræ, complete or partial may occur under various conditions. It may be congenital and incurable, or due to cerebral lesion; or it may be hysterical, when it is apt to affect both eyes and is associated with other symptoms of hysteria. It may be caused by disease of the sympathetic nerve (pseudoptosis), and may be associated with symptoms of vasomotor palsy—viz., elevation of temperature on the affected side, redness or edema of the skin, and contraction of the pupil on the same side. Finally, it is seen in weak, delicate women as a transient event, especially in the morning. When ptosis is the result of a definite lesion of the third nerve, at its nucleus or in its course, it may also be associated with a paralysis of the superior rectus alone, or of the internal and inferior recti in addition.

Condition of the Pupil.—The condition of the pupil should be studied with light of moderate intensity, and in doubtful states the pupil under examination should be compared with that of the eye of a healthy individual about the same age.

Miosis, or contraction of the pupil, is found physiologically during sleep, especially in elderly persons; pathologically, as an early symptom in tabes dorsalis, in progressive paralysis of the insane, and as an effect of eserin, pilocarpin, morphin, and in complete chloroform narcosis.

Mydriasis, or dilatation of the pupil, occurs in deep unconsciousness, during extreme pain, in dyspnea, in peripheral blindness, especially from optic atrophy, in oculomotor paralysis, rarely in tabes dorsalis and progressive paralysis of the insane. It is also an effect of atropin, duboisin, and cocain, and of the early stage of chloroform narcosis.

The pupil may be unduly large from palsy of the sphincter (third pair)

[1] Blepharospasm is a spasm of the orbicularis muscle, which is supplied by the facial nerve. It amounts usually only to twitching of the eyelids, but may be so severe as to close them completely, so that it is not in the power of the patient to open them.

fibers or from spasm of the radiating (sympathetic) fibers; or the pupil may be abnormally small from the opposite conditions.

Other limited paralyses due to third nerve disease are cycloplegia and iridoplegia. *Cycloplegia* is paralysis of the ciliary muscle, producing loss of the power of accommodation. In this state of affairs distant vision is good, but near objects cannot be seen distinctly. It may occur in one or both eyes, being in the latter event more usually due to disease of the nuclear origin of the third nerve. It is one of the earliest manifestations of diphtheritic paralysis, and is a symptom also of tabes dorsalis. It may be corrected by the use of eye-glasses.

Iridoplegia is paralysis of the iris, and its three forms are thus classified by Gowers, one associated and two reflex:

1. *Accommodative iridoplegia* is a form in which the pupil does not diminish in size during accommodation. It is tested by having the patient look at a distant object and then at a near one in the same line of vision, so as to avoid any change in the amount of light entering the eye. It is usually associated with paralysis of accommodation, but the ciliary muscle may be efficient and yet the associated action of the iris be lost, or the reverse. This loss is less common than that of reflex action. It is the result of the same cause as cycloplegia.

2. *Reflex Iridoplegia, or Argyll Robertson Pupil.*—The path for the optic reflex is along the optic nerve and tract to the nucleus of the third nerve; thence to the ciliary ganglion, and through the ciliary nerves to the eye. In testing for this condition each eye should be tried separately, the other being covered, but not closed. The patient is asked to look toward a dark part of the room, when a bright light is thrown suddenly in front of the eye at a distance of three or four feet, so as to avoid the effect of accommodation. The pupil will not contract under these conditions. Therefore there is loss of reaction to light. If the patient is now directed to look at some object in the distance and then at an object near to the eye the pupil will react. This is the Argyll Robertson pupil.

3. *Skin Iridoplegia.*—Loss of skin reflex. If the skin of the neck is pinched or pricked, or stimulated by an electric shock, the pupil dilates reflexly. Since active dilatation of the pupil is through the sympathetic nerve, the motor path for this action must be along the cervical sympathetic, and along the fibers connecting this with the cord, at the lowest part of the cervical region.

These reactions are lost when the path is interrupted or the center is damaged. Thus, the light reflex is lost or impaired in disease of the optic nerve including the retina, or in disease of the third nerve. Disease of one optic tract does not lessen the action unless the light falls on the blind half of the retina, because, as already stated, the fibers from the central and most sensitive region of each retina pass through both optic tracts, whence disease of one does not abolish the reflex. The skin reflex is lost in disease of the cervical sympathetic and sometimes of the cervical spinal cord especially when there is associated loss of sensibility. Thus, tumor of the cord sometimes produces this symptom.

When the eye reflexes are lost without disease of the sympathetic or cervical cord, it is generally due to degenerate disease of the centers Tabes

dorsalis, in which it is a common and an early symptom, is a conspicious instance. Less frequent is general paralysis of the insane, and other degenerative processes less definite. It may occur also without other nerve symptoms. In most of the cases in which it has come under Gowers' observation thus isolated, the patient had suffered from constitutional syphilis for years. The two palsies, that of the skin reflex and that of the light reflex, are commonly associated, but not always. The pupils are often small, reduced to two millimeters, or even one millimeter, in diameter.

Inequality of pupils, or *anisocoria*, is also a symptom of progressive paresis and tabes dorsails, but occurs also in healthy persons.

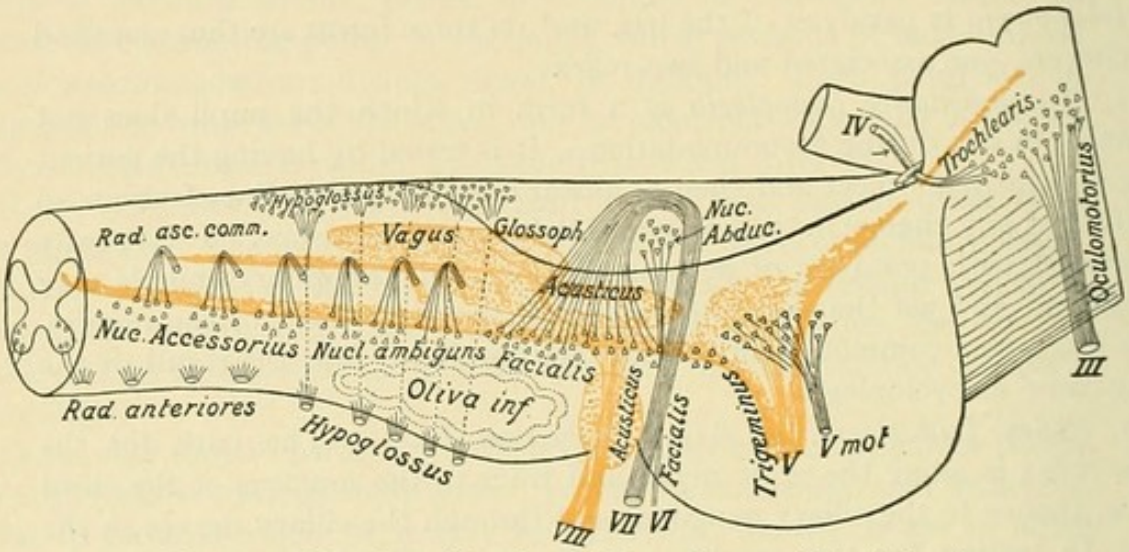


Fig. 174.—Situation of the Cranial Nerve Roots—(*after Edinger*).
Cranial nerve nuclei, oblongata, and pons represented as transparent. Motor nuclei, black; sensitive nuclei, red.

Fourth Nerve.—The fourth cranial nerve (trochlear), as it passes around the outer surface of the crus into the orbit, is liable to be compressed by tumors, by aneurysm, or by the exudation of basal meningitis. Its nucleus below the aqueduct of Sylvius may be involved in tumors or may undergo degeneration with other ocular nuclei. As the superior oblique muscle, supplied by it, acts in such a way as to direct the eyeball downward and rotate it slightly, paralysis causes retardation of downward and inward movement, often so slight as not to be noticeable. The head is inclined somewhat forward and toward the sound side, and there is double vision when the patient looks down, as in descending stairs. Paralysis of this nerve is seldom met with alone, except in nuclear disease.

Sixth Nerve.—The sixth nerve (abducens), emerging at the junction of the pons and medulla oblongata, passing forward and entering the orbit, is liable to be affected by meningitis at the base, or by tumors, especially fibromata. The external rectus being alone supplied by it, the effect of its paralysis is to produce *internal squint*, and the eye cannot be turned outward. There is diplopia when looking toward the paralyzed side. It is a frequent ocular palsy, because the nerve has a long intracranial course.

If the nucleus of the sixth nerve is affected, a very interesting condition results, which was first studied by Beevor. In consequence of paralysis of the external rectus the eye of that side is turned inward while at the

same time the internal rectus of the eye of the opposite side has lost the power to turn its eye inward. Consequently, both eyes are turned to the side opposite and away from that of the injury. Thus, if the nucleus of the right sixth nerve is involved, both the right and the left eye are turned toward the left. Such opposite deviation away from the side of lesion is known as "conjugate deviation." It is due to the fact that the nucleus of the third nerve, supplying the internal rectus, is connected by fibers with the nucleus of the sixth of the other side; whence in lesion of the nucleus of the sixth nerve there is paralysis of the internal rectus nerve in associated movements, even though the nucleus of the third nerve is not involved while the power of convergence is not affected.

In consequence of the proximity of the nucleus of the sixth nerve to that of the seventh or facial, disease of the former is likely to involve the latter. Whence, say if there is lesion of the left nerve there follows conjugate deviation of both eyes to the right, with a complete paralysis of the left half of the face.

Diabetes insipidus is sometimes associated with paralysis of the sixth nerve. Such a case was seen at the Philadelphia Hospital with J. Hendrie Lloyd; The paralysis of the sixth nerve was subsequently substituted by paralysis of the third, the polyuria remaining. A similar condition may be caused by syphilitic meningitis. Basal meningitis, involving the vascular supply to the floor of the fourth ventricle, may be suspected. Other cases of polyuria associated with paralysis of the sixth nerve are reported, notably Maguire's, but it should be recognized that both polyuria and ocular palsy may be caused by syphilis.

Phenomena in General of Paralysis of Motor Nerves of the Eye.—These include, first, limitation of movement and strabismus, referred to. In addition to these certain derangements of vision, known as *secondary deviation*, *erroneus protection*, *double vision*, occur.

Secondary deviations are thus demonstrated: After covering the sound eye, let the paretic eye fix itself upon a point which it cannot reach at all, or can reach only after extreme exertion. Then remove the covering hand from the sound eye, and it will be found that the latter has been moved much too far in the same direction, the abnormal attempt at innervation of the affected eye passing over to the associated muscle of the healthy eye and causing in it too great a contraction.

Erroneus projection furnishes the idea that an object at which we are looking is further on one side than it really is or that the movement of the eye in following it, when moving, is greater than it is. Under these circumstances, in an attempt to touch the object with the fingers the latter may go beyond it. This grows out of the fact that when the eyes are at rest in the mid position, an object at which we are looking appears directly opposite the face. Turning the eye to one side, the object appears to the side of its former position; and if the object moves, we estimate the extent of its motion by the amount of movement of the eyeball following it. Now, when one muscle is weak, the increased innervation required to contract it gives the impression of a degree of movement greater than actually takes place. This is erroneous projection. Now, as the equilibrium of the body is largely maintained by a knowledge of the relation of external objects

to it, obtained by the action of the eye muscles, the erroneous projection due to paralysis disturbs the harmony of visual impressions and may produce dizziness known as ocular vertigo.

Double vision results from the fact that if one eye is paralyzed the axes of the two eyes do not coincide, nor do the images in the two retinæ. The image produced in the sound eye is called the true image; that is the affected eye, the false image. In simple or homonymous diplopia the false image is on the same side as the paralyzed eye; in crossed diplopia it is on the other side. It is one of the most annoying symptoms of paralysis of the eye muscles.

Ophthalmoplegia.—Ophthalmoplegia is a term applied to a chronic progressive paralysis of the ocular muscles, supposed to be due to disease of the ocular nuclei, but frequently the nerves are affected at the base of the brain, especially when the sixth nerves escape. It is called *internal* when the internal muscles only are involved—*i.e.*, the iris and ciliary muscles, *external*, when the external muscles are affected more or less completely. When both internal and external muscles are involved, it is known as *total ophthalmoplegia*.

Symptoms.—These varying according to the position and character of the lesion, which may be degenerative, hemorrhagic, or the result of pressure by tumors or the product of basilar meningitis. They are bilateral, except in the instance of the sixth nerve, with resulting conjugate paralysis. Gowers describes three modes of onset—chronic, sudden, and acute.

The *chronic* form is the most common, due to nuclear degeneration, or more rarely to tumor and embolic obstruction, and still more rarely to hemorrhage. In this form there is a great variety of combination and degree. Thus, there may be internal ophthalmoplegia only, or external, or both. In the internal form there may be loss of the iris-reflex only or of the ciliary muscle action only. In the external variety the levator and superior recti are commonly first involved, the other muscles gradually. There may be loss of the upward and downward movement of the eye, ptosis, and conjugate lateral palsy. There may be double vision. In the total form the eye is fixed and immovable. Each variety may be associated with tabes dorsalis, general paralysis of the insane, progressive muscular atrophy, and bulbar palsy, often with syphilis. It is more common in males, and occurs occasionally in the young. There is a form occurring in children, known as infantile oculo-facial palsy, which may be congenital or acquired; rarely, it occurs as a sequel of diphtheria, late and permanent. The disease may be very slow in developing, and may require years. Sometimes one eye is more affected than the other.

In *sudden nuclear palsy*, the second in frequency, the onset may take but a few minutes or an hour or two. The causes in such such cases are commonly obstruction to the basilar arterial branches, rarely embolic obstruction, and still more rarely hemorrhage. The obstruction is usually bilateral. The lesions are irregular, and the symptoms are correspondingly irregular and unsymmetrical; the tendency is to recover. In these respects it differs from the chronic form also in that hemiplegia is a frequent accompaniment, generally on the side opposite the greater eye palsy. When hemorrhage

is a cause, the resulting ocular palsy lasts but a few hours, provided the hemorrhage acts on the ocular centers only by pressure, while the other phenomena of pressure by effused blood, which is apt to spread, make their appearance.

Acute nuclear palsy is rare. It develops in a few days or weeks, and is possibly of inflammatory or toxic origin whence called by Wernicke *poliomyelitis superior;* but toxic cases may occur without inflammation. This form, according to Gowers, may be due to peripheral neuritis and not to nuclear disease. Alcohol may be a toxic cause. The eye muscles are invaded irregularly, and it is common for the internal muscles to escape. In fatal cases the causal influence extends to the centers of other nerves and possibly to the cortex. In cases that survive there is improvement, various in degree.

Treatment of Ocular Palsies.—The cause should be sought and if found, treated. Although syphilis probably is the chief cause of this disease and of tabes dorsalis, with which it is so frequently associated, disappointment follows the syphilitic treatment in the majority of cases because of destructive lesions. Yet mercury or salvarsan perhaps accomplishes more than any other remedy. Arsenic, potassium iodid, strychnin, and iron are sometimes used, strychnin hypodermically. In acute cases, when there is pain, hot fomentations, leeches, and counterirritation may be used. In chronic forms electricity has been extensively used, galvanism being preferred. Benedikt recommended placing the anode, or positive pole, on the forehead and the cathode, or negative pole, on the margin of the orbit near the affected muscle. If the faradic current is used the orbital pole is held still; if the voltaic, is kept moving over the skin or the current is broken by the commutator. To overcome the ptosis, electric stimulus is applied to the third nerve, as the muscle is not accessible.

The diplopia is removed by a prism not strong enough to fuse the two images completely, but of sufficient force to approximate them, so that the fusion may be completed by muscular action. Such action may be practised for an hour each day. The dizziness due to erroneous projection can be removed only by throwing the eye out of use by an opaque glass. Operative treatment is not recommended.

Periodical Oculomotor Paralysis.—The organic lesion at the bottom of these paralyses is not yet settled. Möbius,[1] who was one of the first who have contributed largely to the subject, regarded *nuclear degeneration* as the cause, while other contributors consider that the majority of cases have a basal cause, by which is meant a *basal meningitis, tumor* or other cause compressing the trunk of the nerve at the base of the cranium. A recent paper on this subject is "Migraine ophthalmoplégique," Bornstein, Monatsschrift für Psychistrie und Neurologie, vol. xxv, No. 3, March, 1909.

Treatment.—Because of syphilitic origin, in many of these cases mercurial treatment proved promptly efficient, while at times the symptoms subsided spontaneously.

[1] Möbius, "Ueber periodische wiederkehrende Oculomotoruslähmung." "Berliner klin. Wochenschr., 1884, Nr. 30 u. 38, S. 604; and "Arch. f. Psych. u. Nervenkrankh.," xiv., S. 844.

LESIONS OF THE TRIFACIAL OR FIFTH NERVE (TRIGEMINUS).

Anatomical.—This important mixed nerve of the face supplies by its motor trunk the muscles of mastication; by its sensory portion, the skin of the face, the mucous membrane of the mouth and nasal cavity, the conjunctiva, and the cornua; also, according to some physiologists, the anterior part of the tongue with gustatory fibers. The gustatory fibers are supposed to reach the lingual fibers of the fifth nerve by the chorda tympani nerve. Recent studies of Harvey Cushing make this doubtful.

Lesions.—1. There may be lesions of the pons, especially hemorrhage, or areas of sclerosis invading the trigeminus nucleus.

2. Injury or disease at the base of the skull, especially acute and chronic meningitis and caries of the bone, tumors, syphilis, new formations compressing the trunk or the Gasserian ganglion. Fracture of the base rarely affects this nerve.

3. Tumors or aneurysms pressing on the first division (ophthalmic) of the nerve through the cavernous sinus, on the second division (superior maxillary) and on the third division (inferior maxillary) by invasion of the sphenomaxillary fossa.

4. There may be inflammation of the nerve, which is rare.

5. The sensory division may also be affected in hysteria.

6. The gustatory fibers of the trigeminus may be influenced by peripheral lesions of the facial, whence the chorda tympani is derived.

Symptoms.—*Paralysis of the Sensory Portion.*—The distribution of the anesthesia varies according as the whole trigeminus or only a part is involved. In total anesthesia there is loss of sensation in half the corresponding side of the head, including the conjunctiva and cornea, mucosa of the lips, tongue, hard palate, and nose of the same side. Hence on the tongue or mucous membrane there are often ulcers which come from unconscious lacerations by the teeth. There is, according to the views of many, loss of the senses of taste and smell. The loss of the sense of smell is probably due to drying of the mucous membrane, as it is not probable that the fifth nerve contains olfactory fibers. The so-called trophic phenomena are also observed, and among them the much-discussed *neuroparalytic ophthalmia*, an ulcerative keratitis, beginning, also, always in the lower segment of the cornea, and passing over into purulent inflammation of the whole eyeball. It seems, on the whole, more likely that the inflammation is primarily due to the action of irritants which in health are excluded by the proper closure of the eyelids, though the inflammatory process itself may be trophically influenced. The salivary, lacrymal, and buccal secretions may be diminished and the teeth may become loose. Herpes is a trophic result which may develop in the course of the nerve, is painful, and may last a long time. So, too, the anesthesia may be preceded by tingling. The skin of the face is sometimes swollen.

Paralysis of the motor portion, which supplies especially the muscles of mastication, the masseters, temporals, and pterygoids, is not common. It is most frequent in diseases of the base of the skull, compressing this branch. Difficulty in chewing is the result. If on one side, the patient can chew only

on the other; if on both sides, he cannot chew at all. The lower jaw hangs down and cannot be moved from side to side because of the paralysis of the pterygoids. If on one side, the external pterygoid cannot push the jaw toward the sound side, and when depressed, the jaw is pushed by the muscle of the sound side toward the paralyzed side. Cases have occurred associated with cortical lesion; and paralysis of the muscles of mastication occurs sometimes in hemiplegia, but is not likely to be persistent.

Spasm of muscles of mastication is found in connection with muscular cramp, the muscular contraction of tetanus (trismus), sometimes in meningitis, and reflexly through painful affections of the jaw or teeth, or from irritation near the motor nucleus. It is also sometimes hysterical. Clonic spasm occurs in muscles supplied by the fifth nerve, constituting "chattering teeth." It occurs generally in connection with general conditions, such as paralysis agitans, but it may happen as a local symptom late in life.

Diagnosis.—This is not difficult. Sensibility is tested in the ordinary way. The preliminary pain must not be mistaken for *neuralgia*. Gustatory sense is tested in the anterior end of the tongue by applying weak acid or salt solutions and comparing the effect on the two halves. The motor power is tested by biting on a piece of wood or cork or by moving the jaws against resistance.

Treatment.—This must depend upon the cause, which should be carefully sought. Syphilitic new formations are the lesions most commonly amenable to treatment. In the absence of such causes the treatment must be symptomatic. Stimulating liniments and faradization through the electric brush are often useful. Galvanism may also be used, brushing the part with the cathode. The anesthetic part should be carefully protected against irritants.

In the absence of tangible cause, systemic treatment is not indicated, except to build up the general health of the patient.

LESIONS OF THE FACIAL NERVE, OR SEVENTH PAIR.

The seventh pair (portio dura of the seventh, old classification) is the motor nerve of the face, and is subject to paralysis ofm otion and to spasm.

Paralysis of the Facial Nerve.

Synonyms.—*Mimetic Facial Paralysis; Bell's Palsy; Monoplegia facialis.*

Monoplegia facialis may be caused by lesions in the *cortical center* of the nerve, in the brain between the cortex and the nucleus, in the *nucleus itself*, and in the *nerve trunk*.

Supranuclear Paralysis.—The cortical center resides in the foot of the central convolution, probably the anterior central, from which pass out fibers along with the pyramidal fibers through the internal capsule to the facial nucleus in the tegmentum of the pons on the opposite side. Accordingly, the nerve is commonly involved in hemiplegias. Such a paralysis, due to lesion above the facial nucleus, is known as *supranuclear*.

In such a palsy the voluntary muscles of the lower half of the face are paralyzed, while the secretory and gustatory functions of the facial

are not affected; nor are the orbicularis and forehead muscles, except in some cases in the beginning of the hemiplegia, these being innervated by the upper branch of the facial. These features, together with the normal electrical excitability of both nerve and muscle, the intact reflexes and taste sense, all point to a *central* facial paralysis as distinguished from a peripheral. The limitation of the paralysis to the lower half of the face is due to the fact that the lower portion only of the face receives almost exclusively crossed innervation, while the upper part, like the ocular muscles and the motor trigeminus, is innervated more from both hemispheres, so that a lesion in one may be overbalanced by the other. This will be understood by an examination of the schematic drawing (Fig. 175), from which it is plain that a one-sided brain lesion at *a* paralyzes only the inferior and not the upper facial, because the upper facial is also innervated from the sound side. Recent investigations show that the upper and lower branches of the facial nerve have not separate nuclei, although they may be represented in different parts of the same nucleus. That they are *functionally* distinct is further shown by the fact that in bulbar paralysis, also a disease of the nerve nuclei of the medulla oblongata, usually only the inferior facial is involved. That the upper face muscles are totally uninvolved in central facial paralysis is not quite true, for careful examination will show that the function is not quite so perfect as in health; the patient cannot close the eye of the paralyzed side by itself, as in the normal state; whence it follows that the upper half of the face is innervated from both hemispheres, as is also shown in Fig. 175. The crossed influence is, however, the larger.

Cortical facial paralysis, *monoplegia facialis*, usually with some weakness of the upper limb on the side of the face paralysis, has been found associated with lesions in the center for the face muscles in the lower Rolandic region, but isolated facial paralysis due to involvement of the nerve-fibers in their path from the cortex to the nucleus is extremely rare. Cortical or capsular facial paralysis, as already explained, is on the same side as that of the arm and leg.

Nuclear Paralysis.—Paralysis may also be caused by lesions of the nucleus, but is not common. The symptoms are essentially the same as those of paralysis of the trunk of the nerve, or peripheral facial palsy, although there is no loss of taste in the nuclear palsy.

Infranuclear Paralysis, or Peripheral Facial Paralysis.—This includes all cases due to involvement of the nerve trunk. The distinctive features of this, as compared with cortical paralysis, have already been stated. It still remains, however, to determine the affected segment of the nerve, to be again referred to when treating of diagnosis.

Etiology.—*Cortical paralyses* are usually due to compression or destruction of the cortical center, as by traumatism, hemorrhage, tumor, meningitis, or embolism. *Nuclear paralysis* may be caused by tumors, chronic softening, hemorrhage, or the diphtheritic toxin, while rarely anterior poliomyelitis may involve the facial nucleus.

The most frequent cause of *peripheral paralysis*, Bell's palsy, is exposure to cold, as to a cold wind or draft from an open window. Such cases include so-called rheumatic paralysis, and may be due to neuritis. Disease of the middle ear and caries of the petrous portion of the temporal bone

are relatively frequent causes, which have evident explanation in the course of the facial through the Fallopian canal adjacent to the tympanic cavity, whence it may be invaded. At the base of the brain, tumors, syphilitic new formations and inflammatory processes also involve the facial. Rarely swelling of the parotid gland is a cause of pressure.

Symptoms.—The symptoms vary with the exact seat at which the nerve is invaded. Paralysis of the facial muscles of expression produces the most striking change of physiognomy. The homely description, understood by everyone, is that the face is drawn to one side; and it appears to be drawn to the sound side, except in old cases after contracture has oc-

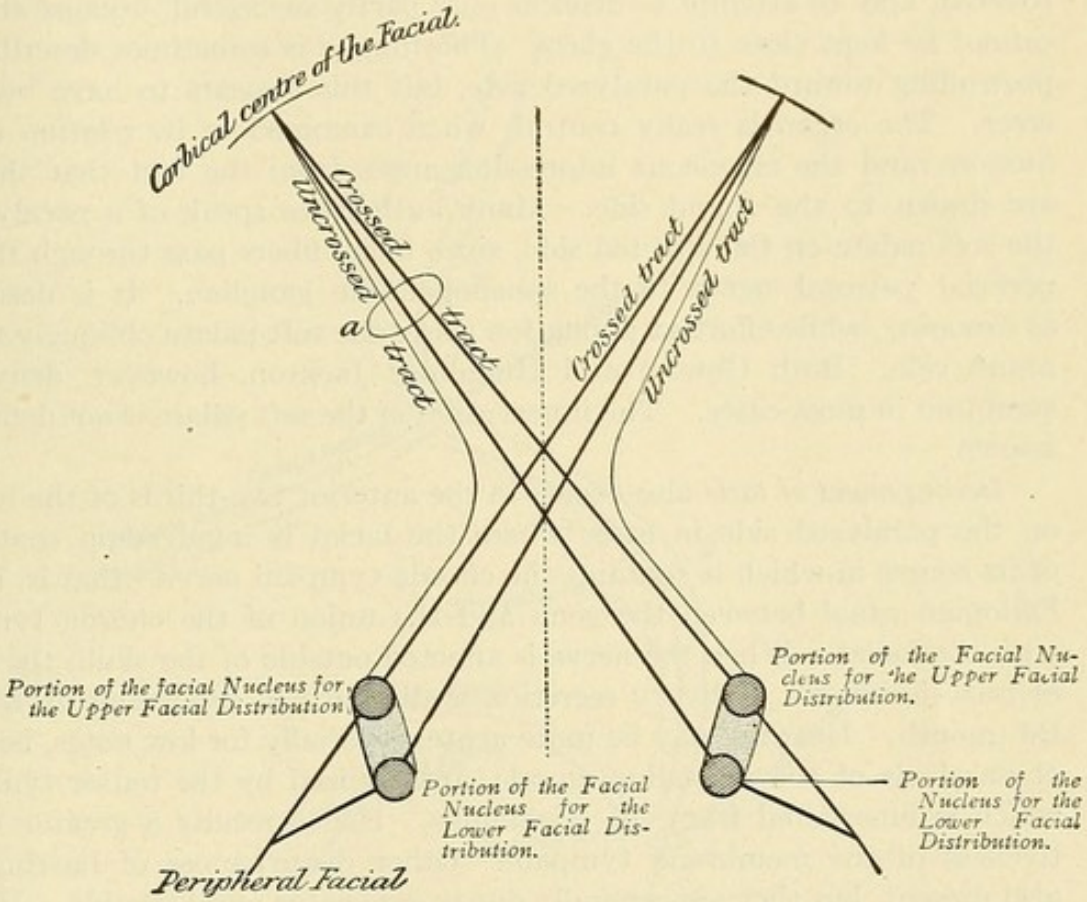


FIG. 175.—Schema and Central Innervation of the Facial Nerve—(*After Sahli*).

The nucleus of the upper branch is innervated from both hemispheres, though mostly from the opposite side, while the nucleus of the lower branch is innervated almost totally from the opposite side.

curred. The appearance of the face being drawn to the sound side is deceptive, and is caused by the flattening of the face on the paralyzed side and the contrast afforded by the two sides. Examination discloses that on the opposite and paralyzed side there is a remarkable smoothness of face, the wrinkles have disappeared from the forehead, the labionasal fold is gone, and this half of the face is quite expressionless. The corner of the mouth on the paralyzed side is lowered, while saliva frequently flows from it; the eye is wider open than natural, and can be only partly closed, even during sleep—*lagophthalmos*—and the eye waters. These symptoms are rendered still more striking on effort at smiling, talking, or whistling, at turning up the nose, wrinkling the forehead, inflating the cheeks, or closing the eyes. On attempting the latter the upper lid drops as though heavy, the eye is

turned upward, the pupil covered, but quite a space remains "uncovered." The so-called "corneal" and "optical" reflexes, by which, through closure of the lid, the eye protects itself from the entrance of foreign bodies seen approaching, are lost, and a tendency to conjunctivitis results.

In complete facial paralysis winking is impossible. Whistling is also impossible, and speech may be interfered with, owing to the difficulty in forming labial sounds. The proper muscles of mastication are not paralyzed, but, owing to paralysis of the buccinator muscle, food collects between the teeth and cheek on the paralyzed side, and an attempt to sniff reveals paralysis of the nasal muscles. The upper teeth cannot be uncovered, and an attempt to drink is only partly successful, because the lips cannot be kept close to the glass. The tongue is sometimes described as protruding toward the paralyzed side, but this appears to have been an error. The organ is really central, when examined in its relation to the incisors, and the erroneous impression arises from the fact that the lips are drawn to the sound side. Many authorities speak of a paralysis of the soft palate on the affected side, since facial fibers pass through the superficial petrosal nerve to the sphenopalatine ganglion. It is described as drooping, while effort at phonation raises the soft palate obliquely to the sound side. Both Gowers and Hughlings Jackson, however, deny this symptom in most cases. The innervation of the soft palate is not definitely known.

Derangement of taste also occurs in the anterior two-thirds of the tongue on the paralyzed side in cases where the facial is involved in that part of its course in which it contains the chorda tympani nerve—that is, in the Fallopian canal between the genu and the union of the chorda tympani with the facial. When the nerve is affected outside of the skull, the sense of taste is intact. Salivary secretion is diminished, producing dryness of the mouth. Hearing may be more acute, especially for low notes, because of paralysis of the stapedius muscle, antagonized by the tensor tympani, which is innervated from the trigeminus. Hence results a greater sensitiveness of the membrana tympani. Other disturbances of hearing are also present, but they are generally due to associated aural trouble. Herpes is also an occasional symptom, and is ascribed to the presence of trigeminal filaments among those of the facial.

Facial paralysis usually sets in suddenly, rarely gradually. Sometimes there are prodromata, consisting in abnormal sensations of taste, pain in the ear and face, and ringing in the former, all from inflammation of the nerve. With this exception, pain is not common.

Diagnosis.—The recognition of the presence of paralysis of the facial is for the most part easy. More difficult, and proportionately important, is it to ascertain the modifications due to lesions in different parts of its course. This is rendered easy by the appended schematic drawing of the distribution of the facial nerve, after Sahli. (Fig. 176.)

The phenomena vary in accordance with the following:

(*a*) Lesion at *A*, trunk of the facial, affecting only the mimetic branches. Paralysis of all the facial muscles; taste, secretion of saliva, hearing, and palate normal.

(*b*) Lesion at *B*, within the styloid foramen. Paralysis of facial mus-

cles, and auricular muscles innervated by the posterior auricular nerve.[1] Taste, secretion of saliva, hearing, and soft palate normal.

(*c*) Lesion at *C*. Paralysis of the facial muscles, derangement of taste, diminished secretion of saliva; hearing and soft palate normal.

(*d*) Lesion at *D*. Paralysis of the facial muscles, derangement of taste, diminished secretion of saliva, abnormal acuteness of hearing, and paresis of soft palate.

(*e*) Lesion at *E*, above geniculate ganglion. Paralysis of the facial muscles, diminished secretion of saliva, abnormal acuteness of hearing, paresis of soft palate, but no disturbance of taste.

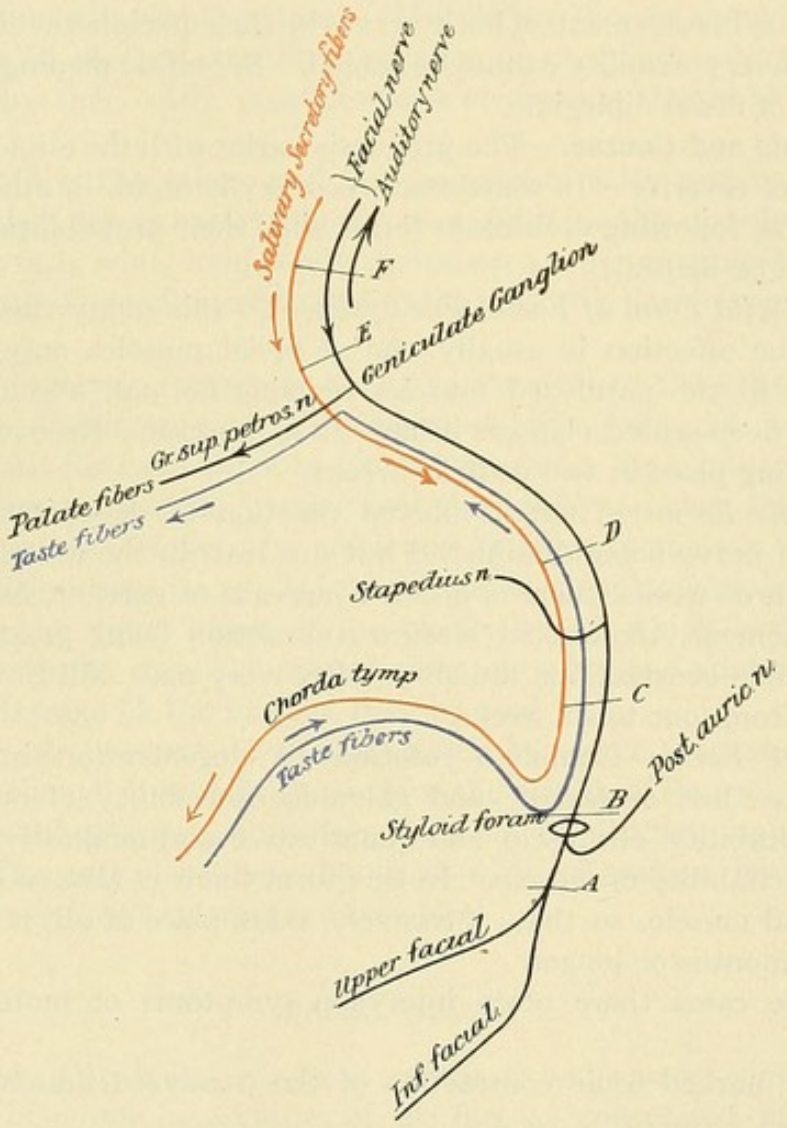


FIG. 176.—Simplified Drawing of the Peripheral Distribution of the Facial Nerve—(*after Sahli*).

(*f*) Lesions at *F*, in Fallopian canal, often associated with a lesion of the auditory nerve in consequence of its proximity to it. Paralysis of facial, diminished secretion of saliva; hearing may be influenced by common lesions to auditory; palate normal, taste normal.

The student is referred to what has previously been said as to the modification rendered necessary by the observations of Gowers and others on the non-involvement of the soft palate in facial palsy.

[1] Since the auricular muscle is in most men not under control of the will, its paralysis can be ascertained only by the electrical test (reaction of degeneration).

We are aided also in recognizing precise forms by the causes, if known, like the presence of ear disease, or a history of exposure to cold or of traumatism. Coexisting symptoms of brain or bulbar disease must also be considered. Reaction of degeneration can occur in true cerebral facial palsy, only in peripheral palsy or in such bulbar paralysis as affects the facial in or below the nucleus itself. In the cortical facial paralysis the frontal distribution of the facial nerve and the ocular muscles are not seriously affected except in the early stages; in the peripheral paralysis they usually are.

The existence of bilateral facial paralysis—*diplegia facialis*—points almost invariably to an intracranial lesion, which may be within the pons. Simultaneous involvement of both nerves in their peripheral distribution is rare, though its possibility cannot be denied. Syphilitic meningitis is a common cause of facial diplegia.

Prognosis and Course.—The prognosis varies with the etiology and with the degree of severity. In some cases recovery is rapid, in others it is only partial. The following division of forms with their probabilities, according to Erb, will be helpful:

1. *The Mild Form of Facial Paralysis.*—To this many rheumatic cases belong. The affection is usually one of facial muscles only. Electrical excitability in the paralyzed muscles remains normal, and there are no severe and deep-seated changes in nerves or muscles. Recovery is rapid, usually taking place in two or three weeks.

2. *Middle Form.*—There is partial reaction of degeneration, the excitability of nerve being diminished but not lost; in the muscles, however, in two or three weeks, there is decided increase of galvanic excitability to direct excitement, the anodal closure contraction being greater than the cathodal while contractions are slow. Recovery may still be quite rapid, usually in from four to six weeks.

3. *Severe Form.*—Complete reaction of degeneration in nerve and muscles—*i.e.*, loss of faradic and galvanic excitability of nerve, loss of faradic excitability of muscle, and quantitative and qualitative changes in galvanic excitability of muscle. In this form there is always degeneration of nerve and muscle, so that, if recovery takes place at all, it is only after two or six months or longer.

In these cases there often intervene symptoms of motor irritation, consisting:

1. In a marked tonic contraction of the paralyzed muscle, sometimes very striking.

2. Single spasmodic contraction of muscles. This is usually the movement described under the following heading.

3. Special associated movements. Thus, if the patient closes his eyes or winks, there always follows a marked distortion of the corner of the mouth, which cannot be restrained.

4. An increased reflex irritability, as the result of which, on pricking or blowing on the skin, vigorous muscular contractions follow.

These symptoms last for a long time—for years in incurable or imperfectly cured cases.

Further points bearing on prognosis have reference to the nature of

the primary disease. Paralysis caused by tumors of the base of the brain and caries of the petrous bone is almost always incurable. If the paralysis is due to middle-ear disease, the prognosis depends on the curability of the ear disease. The electrical examination affords helpful data. If at the end of two weeks electrical excitability still remains normal, a rapidly favorable termination may be predicted. If, on the other hand, the reaction of degeneration is present, a much longer course and delayed recovery, if any, may be expected. Relapses may occur, but are rare.

Treatment.—The treatment is, of course, that of the lesion which lies at the bottom of the paralysis. If it is a syphilitic, inflammatory product, mercury or salvarsan should be given or the iodids should be administered in the usual ascending doses. Middle-ear disease should receive the promptest and closest attention, as some of the most unfortunate cases are caused by this. Any possible cause of pressure should be sought and removed.

When cold is the cause, and the case comes early under observation, warmth, either dry or moist, should be applied to the distribution of the nerve in the face, while mild counterirritation at the pes anserinus is useful. Decided blistering is of questionable utility, but it is harmless and probably does good.

For the paralysis remaining after the removal of the cause electricity is indicated, and more especially the constant current. A weak current should be used for from three to five minutes at a time, interrupting from four to six times a minute, placing first the anode and then the cathode in the auriculo-mastoid fossa, the other pole in front of the ear. Galvanism and faradization may be applied to the muscles themselves, including the oribicularis, the direct effect of the electricity on which is shown by an increased power to close the eye immediately after the application of the current. Massage of the muscles may be used.

Sulphate of strychnin is a drug which has some reputation in facial paralysis, although it is difficult to trace the results of its use. Its administration by subcutaneous injection, daily or on alternate days, is recommended. The salicylate may be used with advantage in some cases. Massage of the facial muscles is advisable.

Facial Spasm.

Definition.—By facial spasm in meant a real spasm confined to the mechanism of motor innervation of the face as contrasted with mimetic facial tic or convulsive tic, all usually unilateral, sometimes bilateral.

Etiology.—No cause can be found for most cases. Possible causes are exposure to cold, lesions at the base of the skull, or irritation of the facial center in the cerebral cortex. Other cases may be explained by reflex causes, such as irritation by ocular disease, carious teeth, intestinal worms, or disease of the sexual organs. Others have been ascribed to violent mental excitement.

Symptoms.—Hugh T. Patrick[1] thus contrasts facial spasm with facial tic, *tic convulsif*, or habit spasm. First, as to points of resemblance: Both

[1] Patrick, "Journal of Nervous and Mental Disease," January, 1909.

facial tic and facial spasm are hyperkinesias. Both present intermittent painless twitching and contraction of facial muscles, both tend to become chronic and both cease during sleep with rare exceptions in case of spasm. Here resemblances cease.

On the other hand, tic is much more common than spasm, and invariably develops in a nervous or neuropathic person. Temperament has nothing to do with spasm. Second, facial spasm is a real spasm confined to the mechanism of motor innervation of the face. Tic is not a spasm, but a movement of volition.

3. Spasm is totally beyond voluntary or involuntary control, while tic is always to some extent under voluntary control and always to involuntary control as by strong emotional or mental preoccupation.

4. Spasm, according to Patrick, is an "anatomical, tic a physiological disorder." A good idea of facial spasm may be obtained by faradizing the facial nerve, and no one would mistake such resulting contraction for a voluntary movement. Tic is a reproduction of a perfectly natural and normal, though it may be unusual movement. Voluntary imitation of facial spasm is impossible, but the patient or even another person may imitate the tic movements.

5. In its incipiency spasm is confined to a part of a muscle and ultimately extends to the entire distribution of the facial nerve, no less, no more, and is, moreover, strictly unilateral. Tic never affects a part of a muscle, because voluntary fascicular contractions are impossible. On the other hand, all the facial muscles are rarely included in one movement. In tic one physiologically associated group of muscles may contract one moment, and another group the next. Tic is apt to be bilateral either simultaneously or alternately, and it is peculiarly liable to be associated with tic of adjacent or remote muscle groups.

6. The contraction of facial spasm is very like that produced by faradism when the vibrator is not running smoothly. While the general effect is tonic, there is also a flickering or quivering, or rapid slight irregular twitching such as never occurs in voluntary movement or tic and cannot be imitated. Each spasm begins with this flickering contraction, generally of a part of a muscle and most frequently in the *orbicularis palpebrarum*. The preliminary quiver may be very brief or may last several seconds, and even in a fully developed case one may see abortive spasms. This never occurs in facial tic.

7. Facial spasm may not look worse than facial tic, but it is more uncomfortable and much more disabling. The subject of tic may talk and sing as he pleases, and though his talk may be interspersed with grimaces his sentences are not interrupted by them. The speech of a person with spasm may be cut short at any time.

Blepharospasm.—A variety of the partial form is *blepharospasm*, a tonic or clonic spasm of the orbicularis muscle. In the clonic form it is apt to be associated with spasm of the lateral facial muscles, and there is constant twitching of the side of the face, with partial closure of the eye. In another clonic variety there is constant contraction of the eyelids and consequent winking.

The tonic form is usually reflex in origin, bilateral, and may last for

days or weeks, with occasional interruptions. The reflex cause is commonly some affection of the eye, producing photophobia, or it may reside in some other point in the distribution of the trigeminus. The clonic form may also sometimes be traced to a reflex act as a cause.

Very interesting in connection with blepharospasm is the discovery by v. Graefe of certain so-called "pressure points." These are points at which pressure causes the spasm to cease, so that the eyelids "fly up as if by a spring." These are commonly found at points of exit of the trigeminus, but have also been found on the vertebral column and elsewhere.

For other forms of spasm of the facial nerve see Choreiform Affections, page 1086.

Prognosis.—This in all forms is, as a rule, unfavorable. There are intervals of suspension, sometimes of considerable length, but the spasm recurs, and the disease generally remains incurable.

Treatment.—The treatment by drugs is correspondingly unsatisfactory, but a number of things may be done. Causes of reflex irritation should be sought and removed, such as carious teeth nasal polypi and ophthalmia. Paquelin's cautery may be applied to the trunk of the nerve, or to pressure points, if they exist. Nerve section of the supra-orbital nerve has been practiced in blepharospasm. Nerve stretching has been followed by relief, at least as long as the paralysis continues, which is commonly a welcome substitute for the twitching. The constant current may be used, seeking also for pressure points, to which the anode is to be applied. If there are none, this pole should be applied to the trunk of the nerve and to the different branches of the pes anserinus. Weir Mitchell recommends the freezing of the cheek every day or every other day with the rhigolene spray; at least transient relief follows. The injection of a small amount of alcohol into and about the nerve is a new and promising method of treatment. It has been successful in a number of cases. Paralysis of the facial distribution occurs, and after it has disappeared the spasm may not reappear.

As to medicines, those usual in nervous affections should be tried—bromid of potassium, strychnin by hypodermic injection, arsenic, iron, oxid of zinc, atropin, curare.

The treatment of convulsive tic is that of hysteria.

LESIONS OF THE AUDITORY OR EIGHTH NERVE.

The eighth pair (portio mollis of the seventh in the older classification of Willis) may be affected in its nucleus at the junction of the pons with the medulla oblongata, or at the base of the brain after it passes out of the pons into the internal auditory meatus to its distribution in the cochlea and vestibule. The proximity of this nerve to the facial at the base of the brain and in the internal auditory meatus is to be remembered. As indicated by its name, it is softer and more vulnerable than the facial, so that equally acting causes may affect it and leave the facial intact.

Cortical deafness from a unilateral lesion, apart from word deafness, cannot be regarded as well established.

The auditory nerve should be regarded as two nerves—the cochlear and the vestibular; the former having to do with hearing and the latter with co-ordination.

Symptoms directly due to disease of the auditory nerve are limited to some derangement of hearing, and it is their association with others which widens their significance in the study of nervous diseases. The derangements of hearing resulting from such lesion are six:

1. Loss of hearing, or deafness.
2. Increased sensitiveness, auditory hyperesthesia, or hyperacusis.
3. Symptoms of irritation, causing subjective aural sensations—tinnitus aurium and allied symptoms.
4. Disturbances of equilibrium or sensation of such, due to irritation of the fibers in the semicircular canals—Ménière's disease.
5. Certain rare instances of involuntary movements, due to disease of the nerve within the ear, as oscillatory motions of the head.
6. Purely functional derangements of hearing, occurring especially in connection with hysteria and with anemia following large hemorrhage.

1. Loss of Function; Nervous Deafness.

Etiology.—Deafness may be congenital when it is due to labyrinthine defect. According to Gowers, 80 per cent. of deaf mutes are congenitally deaf. The remaining 20 per cent. become so from disease in early life. Of congenital cases it is said that the intermarriage of relations having similar defects is responsible for some, while such intermarriage, even where there is no such defect, is held responsible for a smaller number. Partial as well as total deafness may be congenital.

Of the cases of acquired nervous deafness, disease of the labyrinth, either primary or secondary to that of the middle ear, causes most. The labyrinth is subject to inflammation, acute or chronic, to syphilitic disease, to degeneration, and to hemorrhage. It may be invaded by meningitis, cerebrospinal or tuberculous. Its membrane may undergo degeneration, due to gout or simply to old age. The product of all these may be fibrous or calcareous new formation. The deafness caused by certain drugs, as quinin, has been ascribed to congestion of the internal ear, and that by loud noise, as the explosion of artillery, to hemorrhage.

Lesions of the nerve trunks are less common causes. They may be of the same character as those of the labyrinth, except primary inflammation, although even this is said to be a cause. Primary degeneration may occur in tabes dorsalis. The nerve may be compressed by thickening of the cranial bones, calcareous nodules, tumors, or extravasated blood.

The nuclei within the pons may be damaged by hemorrhagic extravasations and tumors. Above the nuclei there may be a lesion encroaching on the superficial layer of the tegmentum.

Symptoms.—Since, as already stated, derangement of hearing constitutes the only essential symptom of nervous deafness, any enlargement of the subject can be made only by considering the modifications and conditions of this system, and by reviewing such methods of determining the precise seat of the lesion as exist.

The ability to hear through the bone while the air conduction is impaired implies that the function of the labyrinth is intact, and that deafness is due to obstruction of the meatus or to disease of the middle ear and

not to nerve deafness. This is further confirmed if the bone conduction is intensified by closing the meatus, since in this way the vibrations, which ordinarily pass out by the meatus, are retained. On the other hand, if there is diminished bone conduction, it does not necessarily follow that the labyrinth is diseased, because there may be ankylosis of the stapes, which will diminish bone conduction, although no amount of disease of the middle ear will extinguish it if the labyrinth be intact. Further, in health air conduction is heard after bone conduction ceases. This is the basis of Rinne's test, in which the vibrating tuning-fork is first placed upon the mastoid process and allowed to remain until the sound dies away to the patient, when the fork is suddenly transferred to the external auditory meatus of the same ear. If the air-conducting apparatus is normal, the vibration of the fork should again be heard. Again, there may be a moderate impairment of hearing and maintenance of the relative delicacy of the air conduction. Absence of bone conduction is, however, the characteristic symptom of nervous deafness. So, to a less degree, is deafness to short and high-pitched sounds, whence the high-pitched, short sounds of the ticking of a watch are a delicate test of the ability to hear through the bone. Simple senile labyrinthine degeneration may be responsible for inability to hear the ticking through bone in persons sixty years old or more.

Can we distinguish between labyrinthine disease and disease of the nerve before its terminal distribution? Given the absence of bone conduction, if the facial nerve is paralyzed, and there is also disease of the middle ear or of the bone, we may conclude that the nerves (facial and auditory) are affected at the base of the brain or in the internal meatus. If there is disease of the middle ear along with deafness and paralysis of the facial, it is probable that the facial nerve and labyrinth are affected by extension of the disease from the tympanum, but this is not certain. An involvement of the trunk of the nerve at the base is also probable if some other nerve near it, as the sixth, is involved. The fact that the auditory nerve is more sensitive to pressure than the facial has already been mentioned, whence an agency, such as an inflammatory product or tumor, pressing on both nerves, may affect the auditory and leave the facial intact.

No distinctive symptoms have been found associated with lesion of the auditory nuclei in the medulla oblongata. Such lesion is very rare, but has been found associated with deafness on the same side, while it has also been found when the hearing has been unaffected. Sudden deafness, associated with other symptoms of a lesion of the pons or medulla oblongata, should excite suspicion of nuclear lesion, especially if paresis of limbs on the opposite side be one of those symptoms.

The auditory fibers in their passage through the tegmentum may also be affected and may produce deafness. Such a lesion is a tumor of the corpora quadrigemina.

Lesions of the cortical center are very rare, though they have been sufficiently frequent to confirm the results of experiment on the monkey, which go to show that the first temporo-sphenoidal gyrus represents the center for hearing, since the destruction of this gyrus on the left side in man has been attended by word-deafness. It is possible that the first temporal gyrus in each hemisphere in man must be damaged in order to

produce cortical deafness for sound. Hemorrhages, softening, and pressure by fractures or tumors may be causative lesions in this situation.

Treatment.—This is for the most part unsatisfactory, at least from the physician's standpoint. Careful otoscopic examination should be made with a view to discovering the existence of disease of the external and middle ear, and the aural surgeon should invariably be consulted in derangements of hearing of more than brief duration, with a view to obtaining certainty of diagnosis between nerve deafness and disease of the middle or external ear. Suspected syphilitic tumors should be treated by iodids. A blister in front of or behind the ear may be useful, especially in acute cases; but deep blistering should be avoided in front lest it cause facial neuritis. Electricity has been employed with partly satisfactory results.

2. Auditory Hyperesthesia.

True hyperesthesia, or hyperacusis, is a condition in which ordinary sounds are heard with more than normal acuteness, and in which sounds inaudible become audible. In dysesthesia, or dysacusis, ordinary sounds, although not intensified, produce discomfort. There is generally present some pre-existing symptom, as a headache, during which sounds usually without effect intensify the headache. Both these conditions occur in functional as well as in organic brain disease. Of the former, hysteria is an instance; of the latter, meningitis.

Treatment.—The treatment, outside of the removal of the cause, is by nerve sedatives, as the bromids, preparations of valerian, and asafetida.

3. Irritation of the Auditory Nerve.—Tinnitus Aurium.

Definition.—The term tinnitus includes almost every conceivable form of auditory subjective sensation, of which the most common is ringing, roaring, or hissing. The tinnitus may inlcude humming, ticking, the sound of rushing steam, the roaring of machinery and the like, the sound of a bell, and even articulate speech, music, or the sound of voices. It may be persistent or intermittent, with rhythmical intermissions—these commonly corresponding with the beating of the pulse. The sounds may be so slight as to be forgotten when the attention is directed to something else, or they may be heard through everything, causing the sorest distress and misery. In fact, their victims have even been impelled to self-destruction. The clicking symptom, sometimes aubible to those standing near, is often very annoying, and may be due to clonic spasm of the muscles connected with the Eustachian tube or levator palati. The so-called premonitory "aura" of epileptic seizures may be a variety of tinnitus.

Etiology.—Beyond what is conveyed by the word "irritation," it is exceedingly difficult to discover the cause of tinnitus. Changes in the labyrinth appear to be the most common, and Gowers tells us that "evidence of nervous deafness, mostly due to changes in the internal ear, is distinct in four-fifths of the cases which come under the physician's notice. Disease of the middle and external ear, including inflammation and wax accumulation, is also a fruitful cause, while in a few cases the process may be wholly in the auditory centers, in the nucleus of the nerve, or in the cortical area. Blood movement, not usually audible, may become so. Inter-

nal aneurysm is a possible cause. Tinnitus is common in anemia and neurasthenia. An epileptic aura is often a tinnitus. A systolic murmur is sometimes heard over the ear in children, and even in adults.

Treatment.—This is generally most unsatisfactory. The ear should be explored and its surgical diseases treated.

The gouty diathesis must be treated by the administration of the salicylates, colchicum, and purgatives, and by regulation of the diet; anemia and neurasthenia by iron, arsenic, nutritious food, and rest. Large doses of salicylic acid and quinin, it is known, produce ringing in the ears—a fact to be remembered always.

The bromids are sometimes beneficial, and a few drops of tincture of belladonna are sometimes added. Nitro-glycerin has been highly commended. Beginning with doses of 1/100 grain (0.00066 gm.), they should be rapidly increased until the physiological effect is produced. Our experience with nitro-glycerin is that the physiological effect is often not attained in adults even by doses of 1/100 grain (0.00066 gm.).

Counterirritation is undoubtedly useful at times. It should be applied behind the ear, and actual vesication is the most efficient form. The temporary effect is sometimes striking, while permanent results may be produced by repeated blistering.

4. Disturbance of Equilibrium Associated with Defect of Hearing —Labyrinthine Vertigo. Ménière's Disease.

Definition.—The term Ménière's disease is applied to a vertigo, usually sudden, associated with deafness and noises in the ear.

Pathology and Etiology—In 1861 Ménière described some cases in which vertigo was produced by a sudden lesion of the labyrinth. Since then the term Ménière's disease has come to be applied to all cases of sudden vertigo associated with labyrinthine disease. Gowers says that "in nine cases out of ten in which there is definite giddiness, not epileptic in nature or obviously due to organic brain disease, it is due to a morbid state of the labyrinth or auditory nerve endings." Thus the vertigo becomes the result of the irritation of the nerve.

In addition to clinical sources for the confirmation of this view there is the fact that experimentally induced lesions in the semicircular canals of animals result in vertiginous movements. In point of fact, aural vertigo results from almost any one of the morbid processes possible to the labyrinth and the nerve endings it contains, but not from disease of the middle ear. The precise nature of the morbid change can only be conjectured. It is twice as frequent in men as in women, and four-fifths of all cases occur between the ages of 30 and 60. Cold, gout, and syphilis have been followed by it, probably through inflammation, and possibly resulting hemorrhage. The slower forms may be due to degenerative processes, like those of tabes or such as are due to age. Vasomotor neuroses of the vessels of the labyrinth have been held responsible.

Symptoms.—The vertigo is usually sudden and paroxysmal, though there may be light continuous dizziness between paroxysms, which occur at intervals of from a few days to as many weeks. Occasionally they occur

daily. They may be spontaneous or an exciting cause of trifling character may bring them on, such as turning, coughing, or sneezing. Gastric disturbances may excite them—a fact to be remembered in the differential diagnosis from gastric vertigo. There may be brief unconsciousness. The attacks generally pass off in a few minutes, leaving the patient pale, faint and nauseated, often in a cold, clammy sweat. Vertigo may or may not be accompanied by a tendency to fall forward, backward, or to one side, and the victim may have to grasp something to save himself from falling. External objects may appear to circle about him. The seeming movements of person and external objects are usually in the same direction.

The auditory symptoms—deafness and tinnitus—may be in one or both ears, and more marked in one side than in the other. In the latter case the sense of movement may be toward or from the ear most affected; but when the subject and objective movements coincide in direction, they are more often toward the affected side.

The deafness is nervous and always partial. The tinnitus is usually roaring or throbbing. There may be ocular symptoms; these are secondary, and include nystagmus and diplopia. Pressure on the drum or on the meatus may bring on the nystagmus, and sometimes an apparent jerky movement of objects. Diplopia, nystagmus, and jerky movements may occur together.

Diagnosis.—The essential symptoms of Ménière's disease are dizziness, tinnitus, and deafness. Gastric disturbance is not peculiar to it. The deafness must be proved to be nervous and not the result of defective air conduction. True *gastric vertigo* is not associated with deafness, while other symptoms of dyspepsia are present with it.

While the aura of *epilepsy* is sometimes accompained by giddiness, there is no impairment of hearing. Moreover, in Ménière's disease slight vertigo is more or less constant, the tinnitus is persistent, and loss of consciousness, if present, is very brief. It is the *petit mal*, with its brief unconsciousness, with which the confusion may occur.

The vertigo of *cardiac valvular disease*, especially aortic insufficiency, of *arteriocapillary fibrosis*, and of *chronic interstitial nephritis* is unaccompanied by any of the other distinctive signs of Ménière's disease. *Gelier's vertigo*, characterized by attacks of paretic weakness of the extremities, ptosis, and profound depression, but without loss of consciousness, occurring especially among laborers in the canton of Geneva, should be mentioned as a source of possible error.

Prognosis.—This depends upon the durability of the lesion causing the malady. In cases resulting from remedial causes—such as gout and even syphilis—recovery is possible, while palliation is not infrequently attained. Other cases are obstinate and incurable. Relief, however, comes to the dizziness when the deafness becomes total.

Treatment.—When traceable to gout and syphilis, the remedies appropriate to these diseases should be prescribed. The salicylates and iodids are most frequently useful, but the lithium salts and colchicum are to be remembered. The salicylates should be given in moderate doses rather than large ones, which produce the ringing in the ears. In the absence of knowledge of a definite cause the bromids are the remedies to be most relied upon.

From 20 to 30 grains (1.3 to 2 gm.) should be given at a dose, and Gowers recommends the addition of a few minims of the tincture of belladonna. Nitro-glycerin has been recommended. Suprarenal extract may be beneficial in some cases. Where the tinnitus is intense and not amenable to drugs, intracranial division of the auditory nerve may be advisable. The general health should be looked after.

Counterirritation by blistering behind the ear is sometimes promptly followed by favorable results.

LESIONS OF THE NINTH OR GLOSSOPHARYNGEAL NERVE.

Anatomical.—This triply mixed nerve supplies sensibility to the soft palate, the tonsils, the upper part of the pharynx, the Eustachian tube, and the tympanic cavity; motor impulses to the stylopharyngeus and to the middle constrictor of the pharynx; and the sense of taste to the posterior third of the tongue and to the palate.

The study of the precise pathology of this nerve is rendered difficult by its numerous communications with other nerves, notably with the fifth, the facial, and the pneumogastric, and by the fact that it is rarely involved alone. Experimental inquiry with it also is difficult.

The nerve may be invaded by meningitis, tumors, or degenerations.

Symptoms.—Symptoms of such lesion would be *difficult deglutition* and *perversion of the sense of taste—parageusia—or complete gustatory anesthesia.*

Modifications of the sense of taste are tested by means of sapid substances in solution, applied to the anterior and posterior parts of the tongue by a glass rod or a brush, suitable substances being used for each taste. Thus, for bitter a solution of quinin may be used; for sweet, a solution of sugar; dilute acetic acid or vinegar for acid, and common salt for the saline taste.

Ageusia may result not only from lesions of the glossopharyngeal nerve, but also from those of the gustatory or lingual branch of the fifth, and possibly of the fifth itself within the cranial cavity; from affections of the chorda tympani in disease of the middle ear, of the facial between the entrance of the chorda tympani and the geniculate ganglion, and in lesions of the peripheral organs of the nerves of taste. Disturbance of taste may possibly result from cerebral lesions, but the cortical area for taste is not definitely known. It is probably in the region of the uncinate gyrus.

Perversion of the sense of taste is known as "parageusia." It is a rare phenomenon, found in patients with facial palsy, in the hysterical, and in the insane, in whom, also, subjective sensations of taste may be present. The latter also occurs as an aura in epilepsy. Hyperesthesia of taste is even more rare, and is purely a hysterical symptom.

LESIONS OF THE PNEUMOGASTRIC OR VAGUS NERVE—THE TENTH PAIR.

Anatomical.—This nerve has by far the widest distribution of any of the cranial set, supplying the pharynx, larynx, lungs, heart, esophagus, and stomach, and in part also the intestines and spleen. The symptoms of its involvement are, therefore, numerous and varied.

It is a *mixed nerve* of motion and sensation, some of its most important motor functions being performed through its connection with the accessory nerve. It is the chief sensory nerve for the respiratory center in the medulla oblongata, but contains, also, accelerating and inhibitory fibers from this center. The former office preponderates, so that section of the nerve renders respirations less frequent, though deeper, while stimulation of the divided central end accelerates them, and acceleration may proceed to tetanic arrest. The inhibitory fibers are contained chiefly in the superior laryngeal nerve, stimulation of which arrests breathing with the muscles in a state of relaxation.

It is also the inhibitory nerve of the heart, slight stimulation increasing the length of diastole, while stronger stimulation arrests its action. On dividing the nerve cardiac contractions become more frequent. It is also inhibitory for the vasomotor center, and its stimulation produces relaxation of the arteries throughout the body. It is the motor and sensory nerve for the esophagus, sensory nerve for the stomach, and partly the motor nerve for the stomach and intestines.

Lesions Involving the Nucleus and Trunk of the Pneumogastric and Branches.

The *nucleus* in the medulla oblongata may be involved in softening, hemorrhage, or slow degeneration, but adjacent nuclei are also affected at the same time, whence resulting effects are associated and are especially seen in bulbar palsy.

The *trunk* of the nerve near its origin may be compressed by thickened meningitis, tumors, or aneurysm of the vertebral artery. In its course it has been implicated in incised wounds, and tied in ligation of the carotid. Neuritis and fibroneuromata are possible. The results of such lesions of the nerve are commonly paralytic, rarely irritative. The paralytic symptoms, if total, are diminished breathing-rate, "suffocation," frequent pulse-rate, and death. According to Traumann and others unilateral division of the vagus in experiments on animals caused few pulmonary symptoms. One vagus seems to be sufficient for the function of both lungs. The results of partial paralysis are better considered in connection with lesions of the separate branches of the pneumogastric, some of which are also invaded separately.

Lesions of the Pharyngeal Branches.—These branches of the pneumogastric, together with branches of the glossopharyngeal, form the pharyngeal plexus, from which the muscles and mucous membrane of the pharynx are innervated.

Etiology.—Nuclear disease is a most common cause of paralysis of the pharynx. It shares with disease involving adjacent nuclei, constituting bulbar palsy, already considered; but it may also be caused by meningitis or bone disease at the base of the skull, or it may form part of the lesion of diptheritic paralysis.

Symptoms.—The results are mainly paralytic, occasionally irritative, producing spasm. The symptoms of *paralysis* are difficulty in swallowing, food lodging in instead of descending into the esophagus. A most frequent

consequence is the entrance of food into the larynx, causing spasm and even choking. Pulpy food is better swallowed than liquids, the latter passing easily into the posterior nares when there is paralysis of the soft palate, and even when the paralysis is limited to the superior constrictor of the pharynx owing to contraction of the middle constrictor. When the nerves on one side only are involved, the difficulty is much diminished. Should there be a doubt in diagnosis between paralysis of the pharynx and obstruction or morbid growth, the passage of a bougie will clear it up.

Spasm of the pharynx is always functional in origin, chiefly hysterical. The so-called "globus hystericus," or sensation as of a ball in the throat which has to be swallowed but immediately arises again, is one of its manifestations; so is eminently the spasm in hydrophobia. Extreme degrees are those in which persons cannot swallow their food in the presence of others.

LESIONS OF THE LARYNGEAL BRANCHES.—The laryngeal branches are two, the superior and inferior or recurrent laryngeal. The former supplies the mucous membrane above the vocal cords, the cricothyroid, and the depressors of the epiglottis. The inferior or recurrent laryngeal on the left side winds around the arch of the aorta; on the right, around the subclavian. The nerves then pass up to the larynx between the trachea and the esophagus, supplying all the laryngeal muscles except the cricothyroid and epiglottic, and the mucous membrane below the cords; also that of the trachea. It has been supposed that the motor fibers in these nerves come from the spinal accessory nerve, but this is now doubted. Their nucleus is probably a part of the pneumogastric nucleus. The sensory filaments of laryngeal branches pass to the medulla oblongata in the roots of the pneumogastric.

In order to appreciate the phenomena of paralysis of the larynx it should be remembered that the glottis is opened or closed only by the movement of the posterior extremity of the cords, the anterior remaining fixed, and that this movement is effected chiefly by the arytenoid cartilages attached to the cricoid cartilage by an articulation which permits free movement. Each arytenoid is shaped like an irregular pyramid prolonged at the base into two processes—an anterior or vocal, from which the cord passes to the thyroid cartilage, and an external or muscular, to which the muscles are attached. When the latter, which is at right angles to the vocal process, is moved back, this process moves outward from its fellow, the cord is abducted, and the glottis opened. If the muscular process is moved forward, the vocal process is moved inward toward its fellow, the cord adducted, and the glottis closed. These movements are further aided by movements of the arytenoids away from or toward each other.

Symptoms.—These are phonic and respiratory, together with altered position of the cords, as recoginzed by the laryngeal mirror. The voice may be changed or lost, the entrance of air in breathing impeded, while the closure of the glottis, necessary to coughing, is usually imperfect. The voice and respiratory functions of the larynx are regulated by the same muscles and nerves, but by centers that differ in anatomical connection, if not in position.

In breathing the cords are abducted or separated during inspiration, the extent being proportionate to the force of inspiration. During expiration they are a little nearer than in inspiration. In phonation they are made tense and brought together, the degree of adduction and tension varying with the note produced. After death the vocal cords assume a position of slight abduction from the middle line, a little nearer than during ordinary breathing, known as the cadaveric position. The position is one of partial relaxation, complete relaxation being never fully attained during life.

The symptoms of deranged function of the laryngeal nerves admit of classification into those of *paralysis* and *spasm*.

1. *Total Paralysis of Both Cords or of One.*—In what is known as *complete paralysis* of the laryngeal muscles—which does not, however, usually include the cricothyroid—the vocal cords assume the cadaveric position previously mentioned, from which they cannot be moved. Hence vocal sounds cannot be produced. In deep inspiration the current of air may bring them a little closer, and there may be slight stridor, and instead of the natural explosive cough, there is only a sudden rush of air through the glottis. If *one cord is paralyzed*, it alone is motionless in the cadaveric position. Phonation may still be possible, because the unaffected cord may be overadducted beyond the middle line, but the voice is low-pitched and often hoarse. During inspiration the abduction of the healthy cord prevents stridor, while an explosive cough is impossible because the glottis is not closed with sufficient firmness to produce it, unless the paralysis is very slight.

The *causes* of complete paralysis are central disease and disease of the trunk of the vagus or of the recurrent laryngeal.

2. *Bilateral Abductor Paralysis.*—In abductor paralysis involving the posterior crico-arytenoids the cords are near together—in the position of phonation—and cannot be abducted even as far as the cadaveric position. They can, however, be brought together in phonation and in coughing, at the cessation of which they recede a little, but the normal wide abduction of inspiration does not take place. This slight recession is due to the elasticity of the attachment of the cords. The adductors, unopposed, undergo secondary contracture, so that if the paralysis is of long duration, the chink of the glottis becomes pemanently narrower. The tensors are still active, as well as the adductors, hence the voice is little affected The chief difficulty is in breathing, since the normal recession of the cords essential to inspiration does not take place,while they are even brought closer together by the pressure of the entering air. Hence inspiration is accomplished with stridor, and the obstruction to the entrance of air brings into play the extraordinary muscles of respiration, the effect of which is to prolong the inspiratory act. Expiration is unimpeded, the current of outward air tending to open the cords. The absence of voice involvement and of cough may cause the obstruction to be referred to the trachea, but the absence of the expiratory stridor excludes this, while the movement of the larynx up and down during breathing is greater than in tracheal stenosis. The added urgent dyspnea, the loud inspiratory stridor, livid features, and cold extermities furnish an unmistakable picture; so that a laryngoscopic examina-

tion is therefore not necessary to complete the diagnosis. In bilateral palsy there is even great danger, as a slight catarrhal swelling may close the larynx nad tracheotomy may be necessary to save life.

The *causes* of abductor paralysis are central disease and local influence such as laryngeal catarrh and degeneration of the posterior circothyroids, possibly of toxic origin. Disease of the recurrent laryngeal has produced such paralysis, although this nerve supplies fibers to the adductors as well as abductors. On the other hand, the abductors have been found degenerated when the other muscles were found normal. Paralysis of both cords is generally due to disease of both nerves, and may be produced by pressure on both vagi or both recurrent laryngeal nerves. Central causes are tabes dorsalis and bulbar palsy. Abductor paralysis is also a rare symptom in hysteria, when it is bilateral, with characteristic symptoms, and has caused death.

3. *Unilateral Abductor Paralysis.*—In this the affected cord is near the middle line, and it does not move in inspiration. There are hoarseness and roughness of voice and sometimes dyspnea, but the mobility of the other cord permits the function of the larynx to be carried on with tolerable comfort. If the adductors become involved, as is sometimes the case, phonation is still more impaired.

The most frequent cause is *aneurysm*, and *the left cord* is most frequently involved—though other tumors may cause it—and on the right side the nerve may be involved in a thickened pleura.

4. *Adductor Paralysis (Phonic Paralysis; Hysterical Paralysis).*—In adductor paralysis due to involvement of the lateral crico-arytenoid and the arytenoid muscles the cords are apart and cannot be approximated. In true adductor paralysis there is still the power of separating the cords on deep inspiration, but no power to bring the cords nearer than in the cadaveric position.

The *causes* of adductor paralysis are rarely organic diseases of the nerves or centers. It is the condition causing the oft-quoted *hysterical aphonia*, and may be brought on by overuse of voice and catarrhal laryngitis. The patient with hysterical aphonia can sometimes sing, though she can only talk in a whisper. It is most common as a partial paralysis. While the cords cannot be approximated for phonation, they can be in coughing. Hence it was called by Türck "phonic paralysis." Another partial adductor paralysis is due to the loss of power in the arytenoid muscle, resulting in defective closure of the posterior part of the glottis and hoarseness or loss of voice.

5. *Tensor Paralysis.*—Little is known of this except that palsy of the internal fibers of the thyro-arytenoideus causes the edge of the cord to be concave.

Diagnosis.—The laryngoscope is necessary to a proper diagnosis of laryngeal palsies, but symptoms are also useful. The inability to produce explosive cough is of great value in pointing to palsy or organic orgin, if there is no local lesion to prevent it.

(*a*) Absence of cough with entire loss of voice points to bilateral palsy of organic origin.

(*b*) No cough, voice low-pitched and hoarse, paralysis of one cord.

(*c*) Loud inspiratory stridor without loss of voice, total abductor paralysis.

(*d*) Little change of voice or cough, unilateral abductor paralysis.

(*e*) Perfect cough, no voice, no stridor, unimportant adductor palsy.

The following table from Gowers contains in separate columns the symptoms, laryngoscopic picture, and lesions:

Symptoms.	Signs.	Lesion.
(*a*) No voice; no cough; stridor only on deep inspiration.	Both cords moderately abducted and motionless.	Total bilateral palsy.
(*b*) Voice low-pitched and hoarse; no cough; stridor absent or slight on breathing.	One cord moderately abducted and motionless, the other moving freely and even beyond the middle line in phonation.	Total unilateral palsy.
(*c*) 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.
(*d*) Symptoms inconclusive; little affection of the voice or cough.	One cord near the middle line, not moving during inspiration; the other normal.	Unilateral abductor palsy.
(*e*) No voice; perfect cough; no stridor or dyspnea.	Cord normal in position and moving normally in respiration, but not brought together on an attempt at phonation.	Adductor palsy.

Spasm of the Larynx.—In spasm of the larynx the adductors are alone concerned. The closers of the glottis are stronger than the openers, while reflex mechanism is connected chiefly with those muscles because of the importance in guarding against the entrance of foreign bodies into the larynx. Spasm is quite common in children, especially in the rickety, and is not rare in adults under the name of laryngismus stridulus. It is generally reflex, although the reflex cause is not always discoverable. The patient commonly wakes up at night in an attack of intense dyspnea; but it may occur at any time. The symptoms are like those of ordinary croup.

The paroxysm differs from that of abductor paralysis in that stridor accompanies expiration as well as inspiration. The attacks occur in the so-called laryngeal crises of tabes dorsalis, in tetany, in the paroxysms of hydrophobia, sometimes in alteration with attacks of migraine, and in hysteria.

Spasm is also sometimes excited by attempts to speak, when aphonia results. The condition is the reverse of phonic paralysis, in which the cords cannot be brought together in speaking, while in spastic aphonia they come together too forcibly.

Disturbances of the sensory innervation of the larynx are chiefly confined to the irritation which causes cough and spasm.

Lesions of Cardiac Branches.—The cardiac plexus is made up of fibers derived in part from the pneumogastric and in part from the sympathetic. The vagus fibers are motor, sensory, and probably trophic.

The *motor fibers* include those which inhibit, control, and regulate the cardiac action. Their *irritation* inhibits the heart's action and causes slowness of the pulse, or bradycardia. In *complete paralysis* of the vagi the inhibitory action is abolished and the accelerator influence is unham-

pered, producing rapid pulse, or tachycardia. Yet it sometimes happens that complete paralysis of one vagus is followed by no cardiac symptoms.

The causes of these effects are, unfortunately, not always discoverable. Pressure of a tumor, accidental ligation of one vagus, irritation of its nuclei, anginal attacks, in one instance associated with a small tumor of the vagus, have all been followed by bradycardia. Toxic blood states are also held responsible for it. Some persons are able to control the action of their own hearts, notably a Colonel Townsend, who could control the action of his heart at will. The heart may sometimes be slowed by pressure against the pneumogastric in the neck

The opposite condition, tachycardia, has been produced by diphtheritic neuritis, tumors of the vagus, or accidental removal of the vagus.

Sensory phenomena in connection with parts supplied by the cardiac branches of the pneumogastric are unusual, but any uncomfortable sensations arising from palpitation or irregularity are conveyed by branches of the pneumogastric.

Trophic influence in the pneumogastric is inferred from the fact that the heart has been found in a state of fatty degeneration after injury to the nerve.

Lesions of Gastric and Esophageal Branches.—Among phenomena ascribed to effect on these branches are spasm of the esophagus and difficulty in swallowing. The vagus is also the sensory nerve of the stomach, and pain in this organ is felt through this nerve. The severe gastric crises which occur in tabes dorsalis may be caused by irritation of the vagus nuclei. The senses of hunger and thirst are also believed to be conveyed through it, and have been lost in disease involving the root, but appetite is not always lost after section of the nerve, while in some case of disease of the nerve there has been excessive appetite. On the other hand, loss of appetite is from so many causes that it cannot be ascribed to pneumogastric lesion without careful investigation.

The pneumogastric is also the motor nerve of the stomach, though motion of the organ is not entirely arrested after its section. Vomiting is probably produced through its agency, and is excited by central and reflex irritation. Meningitis, which so frequently excites vomiting, does so through it; the pressure of a tumor on the nerve has had a similar effect, also direct pressure on an exposed nerve.

Lesions of Pulmonary Branches.—While the vagus sends branches to the lungs, little is known of their office. They are supposed to go to the bronchial muscles. Irritation of the afferent pulmonary fibers certainly produces spasm. Stimulation of the respiratory center also causes energetic respiratory movements, while rapid congestion and even hemorrhage have been noticed after section, though these effects may possibly be of reflex origin excited through the sympathetic, since the vasomotor fibers in the vessels of the lungs are derived from the sympathetic. After section of the vagus animals die of broncho-pneumonia. This has not been considered the result of trophic influence, but because of the entrance of foreign particles into the bronchi in consequence of paralysis of the larynx and esophagus;

this was shown by Traube as far back as 1871, and confirmed by Frey by numerous experiments in 1877. Such broncho-pneumonia has also been ascribed to paralysis of the bronchial musculature and of the vaso-constrictor fibers which causes neuroparalytic hyperemia of the pulmonary tissue. Spiller's case[1] has some bearing on the subject. This patient suffered an injury of the left glossopharyngeus and vagus by a fracture of the base of the skull. He died 46 days after the accident and at necropsy numerous areas of bronchopneumonia were found. It seemed improbable that the pulmonary condition was caused by the entrance of foreign bodies into the lungs in this case, because the patient was unable to swallow. Saliva, however, doubtless passed into the trachea and carried with it micro-organisms. The patient was tested with a glass of water and his choking was so alarming that the attempt was not repeated. He was nourished by rectal enemas and the stomach tube was passed only on the day before his death. The choking was probably the result of impaired function of the epiglottis. Spiller thinks it reasonable to attribute the pulmonary condition to the paralysis of the vagus nerve, although the pulmonary lesions were not recognized until the necropsy was made.

The phenomena of hiccough may be the result of disease of this nerve, as it is also the result of disease of the respiratory center.

Prognosis in Pneumogastric Lesions.—This varies greatly. In central and nuclear disease it is unfavorable; it is unfavorable also when it is the result of pressure from intrathoracic tumors, especially aneurysm. In hysterical and purely local affections the prognosis is more favorable.

Treatment.—This is, of course, that of the casual lesion, if it can be discovered. Syphilis is the more curable of the central causes. Other causes of central disease are not removable.

Of diseases of the trunk, neuritis of the vagus is as amenable to treatment as the polyneuritis of which it is a part. The laryngeal symptoms due to involvement of the recurrent laryngeal are as remediable as the causes which produce them. If they are caused by aneurysm of the aorta or cancer, treatment is useless; if caused by syphilitic and scrofulous growths, the prognosis is more hopeful.

In the paralyses of more purely local origin, especially the hysterical, phonic, and diphtheritic forms, electricity offers the most promising results. The method of its employment will be found detailed under diseases of the larynx. Either form of electricity may be used. Strychnin is a useful remedy. The method preferred is by hypodermic injection, the nitrate being employed in doses of from 1/60 to 1/30 grain (0.0011 to 0.0022 gm.) daily.

In additon to strychnin, other tonics should be used to restore the general health of the patient. Laryngeal gymnastics have been recommended and used with some success. They consist in pressing firmly with the thumb and forefinger on each side of the upper and hinder part of the thyroid cartilage, the patient being requested to make a simple sound during the compression.

The treatment of laryngeal spasm demands also the removal of the cause if possible, in addition to which sedatives, local and general, especially

[1] "Univ. of Penn. Med. Bull.," March, 1903.

the bromids and cocain, may be used. Chloral, chloroform, and nitrite of amyl by inhalation may be necessary to break up the spasm.

LESIONS OF THE ELEVENTH PAIR OR SPINAL ACCESSORY NERVE

Anatomical.—This nerve, purely motor in its function, has two portions—an internal, which passes to the pneumogastric and innervates the laryngeal muscles, and an external or spinal portion. The former has been considered. It should be regarded as probably a part of the vagus, and the eleventh nerve is called by some the vago-accessory nerve. The latter, *i.e.*, the spinal portion, is essentially a set of motor fibers from the cervical spinal cord, which ascends into the cranial cavity and passes out again as one of the cranial nerves to be distributed to the sternocleidomastoid and trapezius muscles, whose innervation they share with the spinal nerves. The purpose of the trapezius is chiefly to raise the shoulder; that of the sternocleidomastoid is to assist in turning the head to the opposite side, the chin being at the same time raised. This is accomplished by drawing the occiput toward the side of the muscle acting.

Lesions.—The nuclear origin of the nerve may be involved and contribute to the phenomena of bulbar palsy, or it may share in progressive central degeneration, causing wasting in the muscles supplied, which may be a part of a more general muscular atrophy. The trunks of the nerve or both nerves may be compressed in the foramen magnum by meningitis or tumor. Outside the skull there may be wounds, tumors, caries of the vertebrae, and resulting abscesses, and sometimes abscesses springing from the cervical glands.

The resulting conditions are *paralysis* and *spasm*. Those of the internal or accessory portion have been described under lesions of the pneumogastric. It remains to consider only those of the external branch.

Symptoms of Paralysis of the External Branch of the Spinal Accessory Nerve.—The seats of the paralysis are the sternomastoid and trapezius muscles. When one sternomastoid is involved, the head may still be moved to the opposite side, and there is no wry-neck, or torticollis, though in some cases the head is held obliquely. The trapezius is not so much involved because it is well supplied with cervical and thoracic nerves, but a portion which passes from the acromion to the occipital bone is motionless. The middle portion of the muscle is also weakened, the shoulder droops downward and forward, and the inferior angle of the scapula is rotated inward by the action of the rhomboids and the levator anguli scapulæ. Elevation of the arm is also partial, because the trapezius does not fix the scapula at a point whence the deltoid can work. The paralysis is well seen when the patient takes a deep breath or tries to shrug his shoulders. Wasting almost always accompanies the loss of power, and there is usually reaction of degeneration.

In bilateral paralysis the power of holding the head in the upright position is impaired. If both sternocleidomastoids are affected, the head tends to fall backward; if both trapezii, it falls forward so that the chin rests on the sternum. The latter is the characteristic position of the head in progressive spinal muscular atrophy, and in children who have chronic menin-

gitis about the foramen magnum, pressing on both nerve trunks, and in cervical meningitis the result of caries. A peculiar drooping of the head is sometimes seen during the first year of life in children, which Gowers says may be due to injury to the spinal accessory nerves in difficult labor. In recent cases the nerves may give characteristic reaction of degeneration. In central disease the reaction varies, as it does in progressive spinal muscular atrophy.

Treatment.—This must have for its object, first, the removal of the cause, or the morbid process which produces it. After this the weak muscles are to be treated by massage and electricity. Faradization is, perhaps, most efficient for this purpose, and either form of current will answer.

Symptoms of Accessory Spasm (Torticollis; Wry-neck).—Though the muscles supplied by the spinal accessory are not the sole ones responsible for these conditions, they are the ones chiefly concerned. The terms are applied to unnatural positions of the head resulting from contraction of these muscles. There are two principal varieties:

1. Fixed wry-neck, or congenital torticollis.
2. Spasmodic wry-neck.

These two may be regarded as *true* torticollis, and are to be distinguished from two somewhat similar states which may be called *false* torticollis. The first of these is the ordinary "stiff neck," which is really a condition due to exposure to cold, and characterized by pain and tenderness, for the relief of which the position is assumed, and should not be called wry-neck. The second is a twist-neck, not due to muscles, but to some other cause, most frequently disease of the cervical vertebræ. This deviation puts the sternocleidomastoid muscle on the stretch, and thus may give rise to the impression that it is responsible.

1. Congenital Torticollis, or Fixed Wry-neck.—This depends on the shortening of some muscle, commonly the sternocleidomastoid, which is also often atrophied, hard, and firm. It is met most frequently in children, and is thought to be due, in some cases at least, to injury of the muscle produced by traction during birth. In others it is ascribed to developmental shortening of the muscle, due to the inclined position of the child's head in the pelvis. It is not always noticed immediately after birth because of the natural shortness of the child's neck. A similar condition may result from injury to the muscle during life, producing inflammation and tricial contraction. It affects the right side almost exclusively. It is more or less constantly associated with facial asymmetry, first noticed by George Wilks and further studied by Golding Bird, who suggested that the two conditions are parts of one affection which has a central origin. In fixed wry-neck the face is turned toward the side opposite to that of the contracted muscle, which stands out conspicuously, and cannot be turned toward the latter. While the sternocleidomastoid is the muscle almost invariably responsible in these cases, the trapezius is occasionally the seat of similar atrophy.

Treatment.—The treatment is by section of the contracted muscle. Some appliance may be necessary for a time to keep the head in proper

position, especially when secondary changes in the articulations have taken place. In simple rheumatic wry-neck Tyson has used an appliance consisting of webbing or "saddle girth" about three inches wide, stretched from side to side of the bed and raised a few inches above the mattress—the distance to be regulated by circumstances—on which the patient lay at night, instead of on a pillow on the side to which the head is drawn. This expedient may be used after operation. The facial asymmetry is likely to remain after the wry-neck is cured, and may even become more conspicuous.

2. SPASMODIC WRY-NECK.—This is a condition analogous to the facial spasm, occurring as a symptom of disease of the facial nerve. There are two forms, the tonic and the clonic, which may alternate in the same case or, as is most usual, occur separately and remain so.

Etiology.—It is for the most part an affection of adults, and, according to Gowers, is more common in females—that is, in 22 out of 32 cases. While this must be true of England, the opposite seems to be the case in this country, since of eight or ten cases observed by Osler in Philadelphia and Montreal, all were men. It is more common in middle life, two-thirds of all cases occurring between the ages of 30 and 50. In women under 30 it is likely to be of a hysterical origin; rarely it is ascribable to the same cause in boys. It is prone to occur in neurotic families and often has the characteristics of tic. Very rarely it occurs in the first year of infantile life, ceasing after a few months. Cold has been assigned as a cause; also traumatism.

In the *tonic* form, when the sternocleidomastoid is responsible, the face is continually turned to the opposite side, the chin is raised, and the occiput is drawn down toward the affected side—the *caput obstipum spasticum*. When the trapezius is involved, the head is still more depressed toward the same side. In combined and bilateral spasm of these muscles the head is drawn backward, producing the *retrocollic spasm*. In prolonged cases the muscles involved are prominent and rigid, and there may be spinal curvature with the convexity toward the sound side.

In the *clonic* form there are paroxysmal twitchings of the head, which may be very severe and correspondingly distressing. When there is predominating unilateral spasm of the sternocleidomastoid, the head is turned to the opposite side and the chin is raised with every contraction of the muscle. In unilateral spasm of the trapezius the head is drawn more backward with each contraction and toward the shoulder of the affected side. In bilateral and combined spasm there is clonic retrocollic spasm, with shaking and nodding movements—the so-called "salaam convulsions" sometimes seen in children. They may be produced also by contractions of the other muscles of the neck. Tonic and clonic spasm of the splenius may occur either alone or in combination with that of the trapezius and sternocleidomastoid. In splenius spasm the head is also drawn backward and toward the affected side, and there will be noticed muscular swelling to the outside of the cervical portion of the trapezius. The splenius is, according to Gowers, associated with the sternomastoid about half as often as the trapezius. The retrocollic spasm is commonly associated with a wrinkling of the forehead in both the tonic and clonic forms.

In the clonic form the contractions may come on suddenly or be preceded by stiffness and irregular pain. The movements occur every few minutes, and the head cannot be kept still, although the movements cease during sleep. They are increased by emotion, excitement, or fatigue. Sometimes there is pain, but at other times there is merely a sense of fatigue. The muscles in time may become hypertrophied, but never waste.

Pathology.—This is very obscure. Reasoning, rather than demonstration, leads to the conclusion that the muscular contractions probably depend on the overaction of nerve cells, and not on irritation of nerve fibers; the movement usually involves the deep rotators on one side of the neck and the sternocleidomastoid muscle on the opposite side. It is therefore a movement of associated muscles, and this suggests a cortical origin, at least in many cases.

Diagnosis.—The distinction lies between true and false torticollis, in which there is deviation of the head from some other cause than muscular contraction, and it is only the form of true torticollis due to shortening of one sternocleidomastoid which is likely to be confounded with the false. In the spurious form the sternomastoid is tense on the side toward which the face is turned, and in the true form the tension is on the side opposite. In retrocollic spasm the invariable association of contraction of the frontalis muscles, producing the peculiar wrinkling of the forehead, distinguishes it from simple tremor. The hysterical form occurs in women under 30, and this fact is presumptive evidence of its presence, while hysterical spasm is also apt to spread from the neck to the trunk; in the true form of torticollis it is limited to the neck.

Prognosis.—The prognosis is always grave, and the more severe and extensive the spasm, the more unfavorable. Relief is more possible in the first half of life than in the second. Cases do, however, occasionally get well, and temporary relief is more frequent.

Treatment.—If the cause can be found which is responsible, it ought to be removed. If discovered in an acute stage, absolute rest in bed and fomentations or dry heat are indicated. Electricity has, perhaps, more reputation than any other remedy. The faradic brush may be applied over the skin of the affected muscles and to the swelling. Gradually increasing faradic currents may be used. If the galvanic current is used, a weak one is preferred, and the anode, or positive pole, is placed below the occiput or highest accessible part of the nerve, and the negative on each contracting muscle, for 10 minutes at a time.

Sedatives and narcotics have also some reputation. Among these the bromids and cannabis indica in large doses are included. Five-minim (0.3 c.c.) doses of the fluid extract of cannabis indica may be given, rapidly increased. The drug is proverbially unreliable. The hypodermic use of morphin is of undoubted value in relaxing the spasm, but the dangers of its protracted use almost preclude it. It would be unfair to the drug, however, to omit the statement of Gowers that, "continued for several months in doses increased gradually to 1/2 or 1 gram a day, it has entirely removed the spasm." Naturally, such persons are weaned from the drug with difficulty. The hypodermic use of atropin in the affected muscles has also been recommended.

Mechanical supports for fixing the head are recommended, but are not well borne. Surgical measures have been employed—such as section, exsection, stretching of the nerve, and section of the muscle—with, at best, but temporary results. Mention should be made, however, of the deep-seated operation of W. W. Keen and Noble Smith, which consists in dividing the spinal accessory nerve and the posterior branches of two or three cervical nerves which also supply the splenius and complexus. This reduces the spasms that reside in these muscles to a slight degree, while the otherwise paralyzing effect of the division of branches of the spinal nerves is comparatively unimportant.

LESIONS OF THE TWELFTH PAIR OR HYPOGLOSSAL NERVE.

Anatomical.—This is the motor nerve of the tongue, and supplies also the depressors of the hyoid bone and the hyoglossus and geniohyoid of the elevators. It arises from the medulla oblongata behind the olivary body. Its cortical center is probably the lower part of the ascending frontal gyrus. It is subject to paralysis and spasm.

Etiology.—1. *Cortical disease* is frequently responsible for paralysis of the tongue on the opposite side, as is seen in the numerous cases of hemiplegia associated with this condition. The same paralysis occurs when the fibers between the cortex and the nucleus in the medulla oblongata are invaded, and probably this is the most frequent cause of paralysis of the tongue. Apoplexies and other causes of compression, softening, thrombosis, and embolism, are agencies operating to this end.

2. *Nuclear disease* is another cause. It is usually degeneration, rarely sudden softening: the former as a part of bulbar palsy and tabes dorsalis, and the latter from vascular obstruction. The effect is almost always bilateral, the nuclei being so close together that it is scarcely possible to involve one only, although such isolated result has occurred in sudden cases and, rarely, in slow ones, as in tabes dorsalis and general paralysis.

3. *Infranuclear disease* may operate at various sites—

(*a*) Within the medulla oblongata the root fibers may be invaded by a tumor or by softening.

(*b*) Outside the medulla oblongata the fibers may be damaged by the products of meningitis, simple or syphilitic, and by new formations. The nerve may be compressed in its foramen by outgrowth of bone. Outside the skull the nerve is compressed by tumors, by inflammatory products, or injured by disease communicated from caries of the upper cervical vertebræ and by penetrating wounds. Hence the spinal accessory and vagus nerves are often implicated coincidently and there is paralysis of the palate, occasionally of the vocal cords, with or without wasting of the trapezius and sternomastoid. The hypoglossal may be the seat of neuritis.

Symptoms. 1. *Of Hypoglossal Paralysis.*—These are motor only. When there is supranuclear disease in addition to the palsy of the tongue, there is hemiplegia, but no wasting of the tongue, nor change in electrical reaction. The tongue is usually but not always protruded toward the affected side. In nuclear disease the lesion is apt to be bilateral palsy. The tongue lies motionless in the floor of the mouth, and is usually drawn

away from the paralyzed side and speech and deglutition are seriously impaired. Mastication is interfered with mainly because the tongue cannot regulate the position of the food, the proper muscles of mastication being intact. There are atrophy and reaction of degeneration. The mucous membrane is thrown into folds. The condition is likely to be a part of bulbar palsy. In infranuclear disease only one nerve is affected, there is wasting with reaction of degeneration and fibrillary twitching. Speech is not much impaired, nor is swallowing.

2. *Of Spasm.*—Spasm of the tongue as an isolated event is very rare. It may be unilateral or bilateral. It commonly occurs as a part of some other convulsive affection, as epilepsy or chorea, or spasm of the facial muscles. It may occur also in hysteria. In the biting of the tongue in epilepsy the organ is thrust between the teeth by spasmodic contraction of the genioglossus and caught by the jaws through a spasm of masseters. Spasm of the tongue occurs in some forms of stuttering, the spasm often preceding the explosive utterance of words. In other cases there are various protrusions and deviations of the tongue, produced in some instances by irritation of the fifth nerve, variously induced, as by a carious tooth. The spasm may be clonic, the tongue being thrust in and out many times in a minute, at others more slowly. It may be associated wth facial spasm. It may occur during sleep.

Diagnosis.—This is generally easy. If there are hemiplegia and palsy, but no wasting of the muscles of the tongue, no reaction of degeneration, the lesion is supranuclear. If there is paralysis of the tongue on the one side and of the limbs on the opposite, there is probably a unilateral lesion in the medulla oblongata, involving the nucleus or the fibers arising from it. When the disease is on the surface of the medulla oblongata, the paralysis is commonly unilateral, and is associated with paralysis of the corresponding half of the palate and vocal cord, because of the involvement of the vagus fibers to the spinal accessory nerve.

Prognosis.—The prognosis is unfavorable because the lesion is incurable.

Treatment.—The treatment embraces that of the disease producing it. The lingual paralysis may be treated with electricity—with an electrode in the shape of a tongue depressor.

The treatment of spasm has been by sedatives, including bromids, by iodid, and by electricity.

DISEASES OF THE SPINAL NERVES AND BRANCHES.

CERVICAL PLEXUS.

Affections of the Phrenic Nerve.—Paralysis of this nerve may be the result of a lesion in the anterior horn of the gray matter of the cord, at the level of the third and fourth cervical nerves; of a lesion to these nerve-roots in disease of the membranes of the cord or of the vertebræ; or by compression by aneurysms or other tumors. Exposure to cold, producing neuritis, may cause it, and it may be a part of a diphtheritic palsy.

Symptoms.—The result is *paralysis of the diaphragm*, which is com-

plete if both nerves are involved, as is the case in disease of the cord or its membranes; partial when a tumor or other cause affects one nerve. Respiration is still carried on by the intercostals, and when the victim is quiet, there is little or no embarrassment, but examination shows the abdomen to be retracted in inspiration and protruded in expiration. In other cases, in consequence of increased movement of the thorax, the upper abdominal walls are drawn outward with inspiration—a movement not to be mistaken for movement of the diaphragm. On exertion, however, there is *dyspnea*, which is also observed if the paralysis is sudden. The effect of paralysis of a single phrenic, involving one-half of the diaphragm, is scarcely noticeable.

A further effect is to aggravate any lung affection, as bronchitis or pneumonia. There is difficulty in coughing effectually, and, therefore, of emptying the lungs of mucus, accumulation of which may result in impairment of resonance at the base of the lungs in bronchitis and in the physical, signs of edema.

Diagnosis.—Nervous breathing resembles the breathing of paralysis of the diaphragm in that this muscle is used very little, while the upper thorax is freely used. If, however, the attention of persons thus breathing is distracted, or they are watched when not conscious of observation, the diaphragmatic breathing will at once become apparent.

The diaphram does not move when it is inflamed or when there is diaphragmatic pleurisy, but it is because of the extreme pain which its motion causes under these circumstances.

The diaphragmatic palsy from diphtheritic neuritis is only a part of the symptoms due to such neuritis. In diaphragmatic paralysis due to spinal disease there is usually atrophy of other muscles, together with other symptoms of that disease.

Prognosis.—This depends upon that of the disease of which it is a part, except in diphtheritic neuritis, in which it is the direct result of the disease, and where the prognosis is unfavorable.

Treatment.—The treatment is that of the disease of which it is the result. If there is neuritis, effort may be made to galvanize the nerve by pressing one pole outside the clavicular portion of the sternomastoid, and the other pole over the epigastrium or the corresponding half of the diaphragm. Counterirritation may also be applied in the triangle of the neck outside the clavicular portion of the sternomastoid. These measures however, do not promise much relief.

BRACHIAL PLEXUS.

Of the Combined Plexus.—This may be affected above the clavicle by causes producing pressure on the nerve trunks—the lower four cervical and first thoracic—after they leave the spine and before they unite to form the plexus. Such causes are tumors and other morbid processes in the neck. More frequently, causes operate below the clavicle, of which the most frequent is prolonged luxation of the humerus, especially under the coracoid process. One or more branches may be thus involved, producing a corresponding degree of paralysis, to which is added wasting of muscles, with reaction of degeneration and trophic changes in the skin. Fracture

of the humerus is another cause. Blows or falls on the shoulder and injuries in the neck may produce the same results, as may also compression during birth. The muscles involved may be the deltoid, supraspinatus, infraspinatus, biceps, and brachialis anticus. A rather frequent cause is pressure from a cervical rib. This condition may be recognized by physical examination or by use of the X-ray.

Neuritis of the brachial plexus also occurs rarely as a primary inflammation. The result ultimately may be complete loss of power in the arm. A still rarer disease is neuro-fibroma of the plexus.

Lesions of Individual Nerves.—*Of the Long Thoracic or Posterior Thoracic (Serratus Palsy).* This nerve is particularly subject to pressure through its long course and position, especially in the posterior triangle of the neck. Such pressure may be direct, as by carrying heavy burdens on the shoulder, or as the result of severe muscular effort in carrying or wielding a hammer, or long exertion with the arm raised, as in whitewashing a ceiling. The result may be a neuritis. Neuritis may also be caused by cold. The same nerve may be involved in progressive spinal muscular atrophy or poliomyelitis anterior. From natural causes it is more common in men.

The result is a dislocation of the scapula of the corresponding side, which presents a winged appearance in consequence of projection of its angle and posterior border, rendered especially distinct when the arm is moved forward, since the scapula is no longer held to the thorax by the serratus. In severe cases faradic irritability is lost, though voltaic excitability may remain. Severe neuralgic pain may precede the paralysis.

The course of serratus palsy is slow, and the paralysis is sometimes permanent.

Treatment.—The treatment consists in maintaining the nutrition of the muscles by electrical stimulation. Counterirritation may be applied over the scalenus muscle, because it is in it that the nerve is most frequently injured. Among the conditions to which the now popular vibratory stimulation has been applied with more or less success is brachial neuritis. The arm should be kept at rest, and to this end should be carried a sling, embracing the elbow in such a way as to raise the shoulder.

Nerves of the Arm.—1. *Of the Circumflex Nerve.*—This rises from the posterior cord of the plexus and supplies the deltoid and teres minor, and the skin over the deltoid. It may be injured by dislocations, blows, bruises, pressure by a crutch, or position long maintained, as during illness. Neuritis may result from these causes and from cold, or by extension of inflammation from the joint.

There is loss of power in the deltoid and the arm cannot be fully raised, also a loss of sensation in the skin over the lower part of the muscle. The muscle wastes and the shoulder becomes flattened. The joint may relax and a space arise between the head of the humerus and the acromion. On the other hand, adhesions may form, partly trophic, since the articulation is supplied by the same nerve. Movement may be further imparied by thickening of the ligaments.

Paralysis of the deltoid is to be distinguished from ankylosis, in which the scapula moves with the arm, which it does not do in palsy.

2. *Suprascapular Nerve.*—This nerve rises from the trunk formed by the union of the sixth, fifth, and a branch of the fourth cervical, but its own fibers are derived from the fifth and partly from the fourth cervical. It is occasionally injured alone or with the circumflex in dislocation of the humerus, and by falls on the shoulder, or by carrying heavy weights. The result is palsy of the supraspinatus and infraspinatus muscles. The first is of little significance, but the latter causes a defect of rotation outward of the humerus, interfering with many movements, of which one is carrying the hand along in writing. The scapula is rotated so that the lower angle is rotated upward and inward.

3. *Musculospiral Paralysis.*—The musculospiral nerve arises from the posterior cord of the brachial plexus, and apparently derives its motor fibers

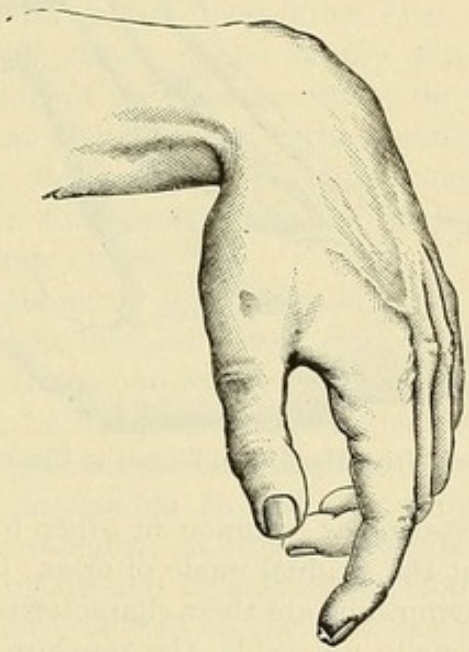


FIG. 177.—Wrist-drop in Musculospiral Paralysis—(*Leube*).

from the nerve-roots forming the plexus except the first thoracic. With its branches the muscular, cutaneous, the radial and posterior interosseous, it supplies the triceps, all the muscles of the back of the forearm, the extensors of the wrist and fingers, both the supinators, as well as the skin on the radial side of the back of the hand, back of the thumb, index-finger, and half of the middle finger. As the musculospiral nerve is called the radial by the Germans, its paralysis is described in German literature as radial palsy.

It is more frequently paralyzed than any single nerve, because of its position—winding around the humerus after it leaves the plexus. It is often bruised by crutches, producing the so-called "crutch palsy," by blows and fractures, and especially by pressure when sleeping with the arm over the back of a chair or with the arm under the head. Even a sudden and violent contraction of the triceps, as in pulling on a tight boot, or forcible extension of the forearm as in throwing a ball, may bruise it. More rarely it is the subject of a neuritis from cold.

In a lesion of the nerve high up all the muscles previously named are involved; when near the middle of the humerus, the triceps generally escapes. The supinator longus and extensor carpi radialis longior usually are involved, but escape if the lesion is below the origin of the branches supplying them, and sometimes in partial injury of the nerve higher up. A characteristic symptom of extensor palsy is the "wrist-drop," while the inability to supinate is also striking. Sensation is rarely lost, though there may be tingling without loss of sensibility.

Paralysis of the musculospiral is to be distinguished from the wrist-drop of lead palsy, which is bilateral, while the supinators usually are unaffected and the onset is gradual. However, in lead palsy the supinator longus may be affected, and in wrist-drop from pressure this muscle may

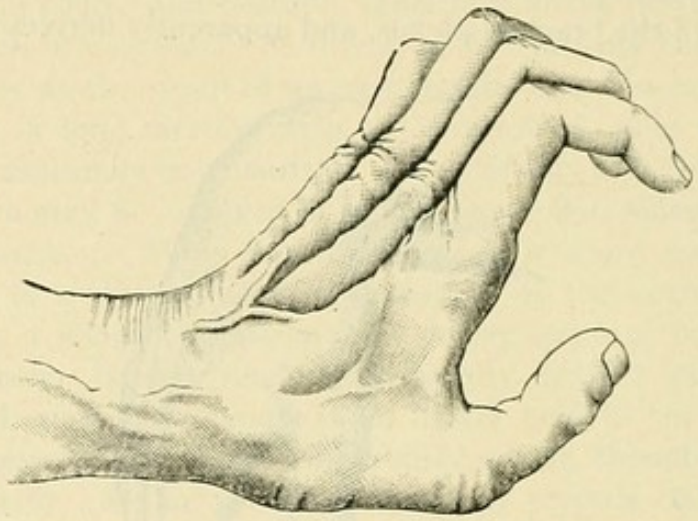


FIG. 178.—Position of Wrist, Hand, and Fingers in Ulnar Paralysis—(*Leube*).

escape. Bilateral wrist-drop is common in other forms of neuritis, especially the alcoholic, but the gradual mode of onset, the involvement of the legs, and the sensory symptoms are their characteristics.

The prognosis is usually favorable, the pressure palsy disappearing in a short time, while recovery is the rule even when delayed.

Erb's rules as to prognosis apply as follows: If both faradic and galvanic irritability are maintained, recovery may be expected in from 14 to 20 days; if these are lessened for the nerve and increased for the muscle, while An C > Ca C, with contraction sluggish, recovery may take place in from four to six weeks, sometimes in from eight to ten weeks. When there is evidence of degeneration of the nerve, the prognosis is more unfavorable, so that recovery may be delayed for from two to fifteen months.

4. *Ulnar Nerve.*—This comes through the inner cord of the plexus from the last cervical and first thoracic. It is the first of all the brachial nerves to be affected by disease ascending from the thoracic to the cervical part of the cord. It supplies the ulnar flexor of the wrist, the ulnar half of the deep flexor of the fingers, the muscles of the little finger, the interossei, two of the lumbricales, the adductor, and the inner head of the short flexor of the thumb. Its sensory portion supplies the ulnar side of the hand, back and front—more of the back—the little finger and the adjoining sides of the little and ring fingers. It communicates with the posterior branch of the internal cutaneous nerve and sends a communicating branch to that

branch of the radial nerve which supplies the adjoining sides of the middle and ring fingers. (Gray.)

The course of the nerve, superficial behind the elbow and at the wrist, makes it vulnerable. It may be injured in wounds of the forearm and about the elbow, in dislocations and fractures about the shoulder and elbow, and continued flexion of the elbow. Neuritis is a possible cause. The most common cause is probably a blow upon the arm.

The hand moves toward the radial side because of paralysis of the ulnar flexor, and adduction of the thumb is impossible, the first phalanges cannot be extended, and in long-standing cases the "claw-hand" may be produced, consisting in overextension of the first phalanges and flexion of the others. There may be wasting of the muscles supplied by the nerve. There is loss of sensation in the sensory distribution.

A similar condition of the ulnar nerve may be produced by lesion of the lower cervical portion of the cord.

5. *Median Nerve.*—Its motor fibers arise from all the cervical roots that enter the brachial plexus. They supply the pronators, the radial flexor of the wrist, flexors of the fingers—except the ulnar half of the deep flexor—the muscles that abduct and flex the thumb, and two radial lumbricales. The sensory fibers supply the radial side of the palm and the front of the thumb, the first two fingers, half of the third finger, and the dorsal surface of the same fingers.

Isolated palsy of this nerve is not frequent, but it may be caused by wounds or fractures of the forearm, rarely from injuries of the upper arm. There may be neuritis from compression.

The wrist can only be flexed toward the ulnar side, and the thumb is in a state of persistent extension and cannot be opposed to the tips of the fingers. Pronation is impossible beyond the midposition to which the supinator can bring the forearm; an attempt is made to supplement this by rotating the humerus inward and separating the elbow from the side. The second phalanges cannot be flexed on the first, nor the distal phalanges of the first and second fingers, while in the third and fourth fingers this action can be performed by the ulnar half of the deep flexor. There is conspicuous wasting of the thumb muscles, which gives a characteristic appearance.

There may be complete or partial loss of sensibility. If there is anesthesia, it is on the palmar surface.

Treatment of Lesions of Nerves of the Arm.—The first principle of treatment is the removal of the cause, whatever it may be, as determined from the etiology. If neuritis is present, it must be treated. Rest by supports or splints, electrical stimulation and massage may be necessary.

LUMBAR AND SACRAL PLEXUSES.

THE LUMBAR PLEXUS.—This is sometimes damaged by growths in the abdomen, especially of the lymph glands, by inflammatory process, by psoas abscess, and by diseases of the bones and vertebræ affecting the nerve-roots. The obturator nerve may be injured during parturition; the anterior crural nerve by the same cause, by wounds of the groin or thigh, by dislocation of the hip, and sometimes by growths about the spine.

Symptoms.—In paralysis of the *obturator*, adduction of the thigh and crossing of the legs are impossible, while outward rotation is also deranged.

In paralysis of the *anterior crural* extension of the knee is impossible; there is wasting of muscles, with anesthesia of the anterolateral part of the thigh and of the inner side of the leg and big toe. There may be pain in the area of distribution.

Paralysis of the *superior gluteal nerve*, which is rare in the isolated form, causes loss of the power of abduction and circumduction of the thigh from paralysis of the gluteus medius and minimus.

The Sacral Plexus.—This suffers from compression by growths in the pelvis, pelvic inflammations, and compression during labor. In addition to spontaneous neuritis, there may also be a neuritis ascending to it from the sciatic nerve. The sciatic may be affected by wounds, dislocation of the hip, disease of the bone, and morbid growths. It is also occasionally the seat of fibroneuroma.

The result of lesions of the *sciatic* varies with its seat. If near the sciatic notch, there is paralysis of the flexors of the leg and all the muscles below the knee, while injury below the middle of the thigh involves only the latter muscles, the flexors of the leg escaping. There is anesthesia of the outer half of the leg, of the sole and greater portion of the dorsum of the foot, but the leg may escape, perhaps through the intermediation of other nerves. Frequently there is wasting of the muscles, with other trophic symptoms. In lesion of one sciatic the leg is fixed in extension by the action of the quadriceps extensor, and the patient can walk, even when all the muscles below the knee are paralyzed, the foot being raised by overflexion of the hip.

The *small sciatic* is implicated only when the pelvic plexus is impinged upon, and it rarely suffers alone. The effect is palsy of the gluteus maximus with difficulty in rising from the sitting posture, and a strip of anesthesia along the back of the middle third of the thigh and upper half of the calf.

Injury to the *external popliteal* or *peroneal* nerve results in paralysis of the tibialis anticus, long extensor of the toes, peronei, and extensor brevis digitorum. There results inability to flex the ankles or extend the first phalanx of the toes, or to raise the foot from the ground in walking—there is foot-drop. Talipes equinus ultimately results, and may be attended with persistent flexion of the first or proximate phalanges from contraction of the unopposed interossei. In walking the whole leg must be lifted, and there is the steppage-gait of neuritis. In old cases there may also be wasting of the anterior tibial and peroneal muscles. There is also anesthesia in the outer half of the front of the leg and on the dorsum of the foot.

Lesion of the *internal popliteal* produces paralysis of the popliteus, calf muscles, tibialis posticus, long flexors of the toes, and muscles of the sole. The symptoms are loss of plantar flexion, inability to extend the ankle-joint, and, if the disease is high enough to involve the branch to the popliteus, loss of power to rotate the flexed leg internally; the foot cannot be adducted, nor can the patient rise on tiptoe. Talipes calcaneus results, and the toes may assume a claw-like position from secondary contraction, due to overextension of the proximal and flexion of the second and third pha-

langes. There is also loss of sensation on the outer lower part of the back of the leg and on the sole of the foot.

Treatment.—The treatment of lesions of the nerves of the legs is similar to that of lesions of nerves of the arms. Secondary contractures are to be guarded against, being favored by position. Fatigue and exposure to cold should be avoided, as they favor fresh attacks of neuritis.

EFFECT OF SECTIONS OF SENSORY NERVES. SENSORY MECHANISM OF PERIPHERAL NERVES.[1]

The studies of Head and Sherren on the effect of nerve action go to show that the sensory mechanism of the peripheral nerves includes three systems: 1. protopathic sensibility; 2. epicritic sensibility; 3. deep sensibility. When a nerve is cut only a small area supplied by it becomes totally analgesic, the remainder being variously modified as to sensibility through the distribution to it of adjacent nerves giving rise to the systems named.

By protopathic sensibility is meant altered sensation in which stimulation by the prick of a pin is felt more severely than over healthy skin, the pain is more widespread and the site of stimulation is not always accurately localized. The patient cannot tell the two points of a compass when separated by less than two centimeters. The power of recognizing differences of temperature is so modified that he cannot recognize cold above 22° C. or heat below 40° C. In considering the peculiarities of this area it is not the fibers of the cut nerve that show this modified sensibility, but the fibers of neighboring nerves that supply certain forms of a sensation to this impaired area.

The epicritic sensibility presents well-marked and definite qualities, such as greater sensitiveness to touch as shown by the recognition of gentle stimulation, as by cotton wool, more precise and definite sense of localization, the perception of the two points of the compass as distinct when separated by less than two centimeters, and the discrimination of fine grades of temperature. Epicritic sensibility is not only an addition to the protopathic sensibility, but it has a remarkable inhibitory or modifying effect on the protopathic sensibility, inasmuch as the pain felt by a prick is less severe and a radiation of pain and of cold is much less extensive than when protopathic sensibility is alone present.

The deep sensibility which responds to pressure and to movements of joints is capable of evoking pain when pressure is excessive or when a joint is injured. It is best demonstrated in an area where the skin is totally analgesic through division of a purely sensory nerve. Because it is thus present, where the skin is totally insensitive it is inferred that the sensory nerves supplying deeper structures, like muscles, tendon, bones, and joints, reach their destinations not by the purely sensory nerves, but by the nerves supplying the muscles. Support for this inference is found in the facts that Sherren has found afferent fibers in the nerves supplying the muscles and that section of motor nerves deprives the muscles and deep structures of all

[1] "The Afferent Nervous Systems from a New Aspect." Henry Head, "Brain," part xi., Nov., 1905. "The Consequences of Injury to the Peripheral Nerves in Man." *Ibid.* Abstracted in "Review of Neurology and Psychiatry," vol. iv., p. 47, 1906. This abstract is so admirably condensed that it has been necessary to follow it quite closely.

sensation. If an injury severs the ulnar nerve at the elbow before the muscular branches are given off, then deep sensibility of certain fingers is destroyed. But if the ulnar is cut at the wrist, then the deep sensibility of the fingers persists. If the injury severs the tendons as well as the ulnar nerve at the wrist then the deep sensibility of these fingers is lost. Hence, it is argued that the fibers supplying the deep sensibility of those fingers reach their destination by running along the tendons.

Much information is gained by studying the phenomena attending the gradual repair of the nerve. Some time elapses before sensation begins to be restored, and the restoration takes place in an orderly manner. The first sign is a diminution of the analgesic area due to the gradual spreading over it of protopathic sensibility, which proceeds until the whole analgesic area is covered. The time required for recovery is from two to three months, and more than six months may elapse before complete restoration of protopathic sensibility is restored. After this there is often an interval of two or three months before improvement takes place in epicritic sensibility. When it begins it appears as a blurring of the margin separating the protopathic from the epicritic sensibility. There is a simultaneons return of all the forms of sensation by which epicritic sensibility is recognized—of light touch, better localization, appreciation of finer grades of temperature, etc. Recovery is gradual, and usually more than a year is required to complete it.

If the nerve has been bruised or incompletely divided, it may fail to conduct impulses, and the resultant loss of sensation may at first resemble that which follows complete division. Recovery, however, in such cases pursues a different course from that following a complete division of the nerve. Thus at the end of a period varying with the extent of the injury, a sensibility to prick and light touch return simultaneously, and as recovery progresses protopathic and epicritic sensibility return together. By observing the form of recovery one can tell whether the nerve is completely severed or merely injured.

"A difference in the distribution of protopathic and epicritic sensibility is also observed, depending partly on the distance of the section from the spinal cord. Thus section of a peripheral branch of a nerve near its final distribution presents a different result from section of a nerve near its exit from the spinal cord. If the forearm and hand be divided into a pre-axial (radial) and postaxial (ulnar) half, it is found that the nerves supplying one of these halves overlap only to a slight extent the areas supplied by the nerves of the other half, while the peripheral branches that supply one of these areas overlap among themselves to a very considerable extent. Thus section of the internal cutaneous high in the arm produces an area of total analgesia embracing the greater portion of the ulnar half of the forearm and hand, while at the same time the epicritic sensation is lost over the remaining portion of the ulnar half of the forearm and hand. This shows that there is very little overlapping of the nerves supplying the radial half of the forearm with the field supplied by the internal cutaneous. Division of one of the two branches of the internal cutaneous presents a very different result—the overlap being so great that little or no analgesia results from section of one branch only.

"Injury to the cords of the brachial plexus produces not only very considerable changes in the sensibility of the parts supplied by the nerves constituting the cords, but they sometimes show a great difference in the relationship of the protopathic and epicritic area. Here the areas of protopathic and epicritic sensibility are nearly co-extensive.

"A further difference in the relationship of these two forms of sensibility is shown when the posterior nerve roots are cut. In two cases division of several posterior nerve roots resulted in the loss of protopathic sensibility over an area greater than that of epicritic sensibility; that is to say, there was an abolition of the sensation to prick over an area larger and more sharply defined than that which became insensitive to light touch. Moreover, this insensibility to prick was accompanied by an inability to appreciate temperature below 15° C. and above 60° C., although 40° C. and 23° C. appeared definitely warm and cool."

The practical applications of the above in interpreting phenomena of deranged innervations is evident. Seats of lesions and progress in improvement may be inferred by the study the phenomena observed.

DISEASES OF THE MEMBRANES OF THE BRAIN.

Although, anatomically considered, the brain is enveloped by three membranes—the tough dura mater, the delicate arachnoid, and the highly vascular pia mater—the diseases of the membranes are practically confined to the dura on the one hand, and the arachnoid and pia conjointly on the other, the last two being always affected together. The dura is, however, separable into two layers—a thin internal layer with its endothelial lining, and a looser external layer which serves as a periosteum to the bones; these two layers may be affected separately.

The term pachymeningitis is applied to inflammation of the dura mater, and leptomeningitis to that of the pia and arachnoid; the latter is commonly meant when the word meningitis is used alone.

PACHYMENINGITIS.

SYNONYM.—*Inflammation of the Dura Mater.*

EXTERNAL PACHYMENINGITIS.

Etiology.—External pachymeningitis is *always acute* and is commonly circumscribed. It usually results from injuries to the head, especially *fractures;* from *caries* of the petrous portion of the temporal bone, caused commonly by middle-ear disease; or from *syphilitic disease* of the bone with pus formation. Sometimes no cause is discoverable. Rarely pus infiltrates between the two layers of the dura mater. More frequently there is pus between the dura and the bone. This may occur in syphilis, which, too, may cause thickening of the bone.

Symptoms.—These are indefinite and are often obscured by those of its causal disease. They are pain, delirium; sometimes, but not always, fever; sometimes convulsions, and signs of pressure. Such pressure may or may not be sufficient to cause paralysis of the opposite side.

Treatment.—The treatment is that of the causal disease, with surgical interference to remove pressure and give vent to pus.

Internal Pachymeningitis.

This is *usually chronic*. Three forms are commonly noticed—purulent, pseudo-membranous, and hemorrhagic.

Purulent and pseudo-membranous internal pachymeningitis are not recognized before death. The former may follow an *injury primarily*, but commonly it is an *extension from* inflammation of the *pia*. Pus between the dura and arachnoid is rare. Pseudo-membranous internal pachymeningitis may occur as a secondary process in *infectious diseases*.

Internal Hemorrhagic Pachymeningitis.—Hemorrhagic pachymeningitis, or *hematoma* of the dura mater, is a rare, but well-recognized condition; it is much more common in infirmaries and hospitals connected with almshouses and asylums. It occasionally occurs in children.

Etiology.—It is probably most frequently a result of chronic alcoholism, though it has been found in chronic insanity without association with alcoholism, especially in general paralysis of the insane; also in acute fevers, when it is associated with profound anemia. Syphilis is a possible cause; in like manner, tuberculosis. It occurs chiefly in males over 50, but also in those between 30 and 40. In mild degree it is sometimes found in chronic cardiac, renal, or pulmonary diseases, when it is commonly first recognized at necrospy.

Pathology and Morbid Anatomy.—The original dictum of Virchow continues for the most part to be held—viz., that it begins as a hyperemia in the area of the *middle meningeal artery*, extending thence forward, backward, and downward. The arteries become tortuous, dilated, and surrounded by thickened adventitia, while the capillaries, being overfilled, produce a rose-colored flush on the under surface of the membrane. To this succeeds a delicate web-like tissue containing wide, thin-walled capillaries three or four times the natural width, between which is a delicate reticulum of spindle cells extending over the greater part of one or both hemispheres. This becomes afterward paler and firmer. Upon this succeeds another delicate vascular layer, succeeded by another and even another. From three to seven layers are thus superposed until a product of from 1/8 to 1/5 inch (3 to 5 mm.) in thickness results. The delicately walled capillaries, however, easily give way, causing hemorrhages which vary in extent from mere points to large collections of blood—the smaller being interstitial and the larger between the youngest vascular layer and the next older. The proportion of blood and organized membrane varies greatly, now one predominating and now another. At times there seems to be blood only. The hemorrhage is believed by some to be the initial event.

Both products are subjecty to degenerative changes, the effused blood being disintegrated and partially absorbed, while the blood-vessels become obliterated and substituted by lines of pigment deposit along their course. There may also be serous infiltration, cystic degeneration, and even diffuse suppuration.

Symptoms.—The symptoms are indefinite. There may be *apoplectiform seizures* coincident with fresh hemorrhages, *drowsiness*, or *coma*. *Muscular weakness* was very marked in a case under Tyson's observation. *Headache* in the region involved, *vomiting*, *nystagmus*, *convulsions*, generally unilateral, and even hemiplegia may be present, and, toward the close, *optic neuritis;* extensive disease may, on the other hand, exist without any symptoms whatever.

Diagnosis.—In the absence of distinctive symptoms the possibility of the presence of hematoma should be remembrered when there are other signs of general paralysis or chronic alcoholism. If to such symptoms great muscular weakness is added, further suspicion is justified.

Prognosis.—This is absolutely unfavorable so far as recovery is concerned.

Treatment.—This consists only in the relief of symptoms as they arise. Indications of hemorrhage should be treated by rest in bed, elevation of the head, and an ice-cap.

LEPTOMENINGITIS.

SYNONYM.—*Inflammation of the Pia Mater.*

Of leptomeningitis there may be an acute and a chronic variety. In addition, other adjective terms are used to indicate its seat and the nature of its cause; such as basilar meningitis, meningitis of the convexity, tuberculous meningitis, etc. Epidemic meningitis has received separate consideration.

ACUTE LEPTOMENINGITIS.

Definition.—An acute inflammation of the pia and arachnoid membranes, attend by exudation between two membranes.

Etiology.—All ages are subject to meningitis, that of the convexity being possibly more frequent in adults because they are more subject to traumatic agencies which cause it, while the basilar form is more common in children. It is rather more frequent in males, and there is a hereditary tendency to one form—tuberculous meningitis.

Of the direct causes—

1. An eruption of *miliary tubercles* is the most frequent. This cause may operate at all ages, but is most active in children. In adults it generally starts from a recognized tuberculosis elsewhere; in children the process is almost always part of a general tuberculosis. Tuberculous meningitis takes place generally at the base of the brain, constituting the chief form of *basilar meningitis*.

2. *Adjacent disease*, which may be outside of the dura mater, such as caries, especially in the petrous portion of the temporal bone. Even disease outside the skull, like erysipelas or suppurative disease of the scalp, may be a primary focus. In these cases it is usually unilateral, and may be accompanied by thrombosis of the sinuses and abscesses; or the disease may result in abscess within the brain.

3. The bacterium or toxin of the acute *infectious diseases*—pneumonia, ulcerative endocarditis, measles, scarlet fever, smallpox, typhoid fever, acute rheumatism, and septicemia. Care must, however, be taken not to confound the simple intense delirium in some of these affections with meningitis, remembering, too, that the latter complication is, under any circumstances, a rare one. The toxin of pneumonia is the most common cause, and perhaps after this that of smallpox. The inflammation thus caused is chiefly of the *convexity*, except in septicemia, when it is general.

4. Chronic Bright's disease and other cachectic conditions. In these the inflammation is commonly basilar.

5. Sunstroke.

6. Mental excitement and brain work—doubtful causes.

7. Rarely in acute inflammation, syphilis, whose product is also basal.

8. Finally, unknown causes may produce meningitis of the convexity or of the base. Possibly, as Gowers suggests, organisms otherwise powerless become sufficient causes during ill health. Thus may be caused some undoubted though rare cases of non-tuberculous basilar meningitis of children—*leptomeningitis infantum*.

In tuberculous meningitis, which is chiefly basilar, the eruption of tubercles precedes the inflammation. There may even be tuberculosis of the pia without inflammation. In tuberculous meningitis the inflammation is never actually purulent, though the lymph has often the appearance of pus. The tubercles are most abundant about the optic chiasm, over the pons, and in the fissure of Sylvius, but the cortex is often affected. According to Spiller's experience the upper part of the cerebral hemispheres contains more tubercles than are found at the base.

Morbid Anatomy.—The early results of leptomeningitis are the same in all varieties. They consist, first, in a hyperemia of the capillaries producing a diffuse pinkish tinge. The next visible changes are a turbidity and an opacity of the arachnoid which extend to the pia, where opacity is especially distinct along the blood-vessels, consisting, in fact in an infiltration of the lymph spaces and lymphatic sheaths with leukocytes. As the cellular accumulation increases the exudate beneath the arachnoid assumes a yellowish-white, creamy appearance. The subarachnoid fluid increases, constituting *hydrocephalus externus*. In suppurative cases it becomes pus, which forms a greenish-yellow layer at the convexity or base, or both.

Ventricular effusion is present in the majority of instances—about four out of five—constituting *hydrocephalus internus*, generally associated with closure of the openings of the fourth ventricle. The effusion is usually limited to a few ounces, but it may be large in quantity, distending the ventricles and compressing the cortex. The walls of the ventricles and the choroid plexuses may be inflamed, and the ventricular effusion may be the result of such inflammation.

In all varieties of meningitis, and especially in the tuberculous, the superficial layer of the cortex is also involved, being at least hyperemic, and sometimes softened; it may also be the seat of punctiform hemorrhages, constituting *red softening*. This is especially prone to occur in tuberculous meningitis, because of the extension of the tuberculosis along the blood-ves-

sels which dip into the cortex. In pulling off the pia these blood-vessels are dragged with it, leaving a ragged appearance of the cortex.

Leptomeningitis infantum presents an appearance similar to that of tuberculous meningitis. It involves chiefly the posterior part of the meninges and cerebellum, closing sometimes the foramen of Magendie, whence the term *occlusive meningitis*. It may also cause an acute, sometimes purulent, hydrocephalus.

Symptoms.—These are varied and not always distinctive of the different forms. First, it is important to remember that all except those which are peculiar to inflammation of the base may be present in any of the serious infectious fevers without meningitis, especially pneumonia, typhoid fever, and smallpox; but in some cases of typhoid fever the typhoid bacillus has been found in the cerebral membranes. When secondary to these affections, they are accompanied by the symptoms of the disease which they succeed.

Meningitis is usually ushered in by *premonitory symptoms*, which, again, are not distinctive, being those usual to acute disease. Perhaps *irritability* is more constant than in other acute diseases. In case of children, *vomiting* with a slight cause, or without discoverable cause, is a symptom of more suspicious nature. It is especially frequent in basilar meningitis, of which it is more or less charactertistic. It has this peculiarity, that it is not usually accompanied by nausea and retching. Generally there are *high fever*, *coated tongue*, and *constipation*, although fever is not invariable. The usual temperature is from 103° to 104° F. (39.5° to 40° C.), but it may reach from 105° to 106° F. (40.5° to 41.1° C.), and toward the close of fatal cases, 108° F. (42.2° C.). It is especially likely to be mild or absent in the meningitis of Bright's disease or of debilitated children. The *pulse* is increased in frequency at first, but later may be slow and irregular.

Of the symptoms the direct result of the disease, *pain in the head is* the most constant. Commonly frontal, it may be general. Its constancy and severity are characteristic. Yet it is subject to such exacerbations as may cause the patient to cry out, constituting the *hydrocephalic* cry of children. The headache is invariable, followed sooner or later by *unconsciousness*. Delirium is an early symptom and soon follows the headache; at first wandering, it soon becomes active, and may alternate with drowsiness or stupor.

General *convulsions* are also another symptom, occurring in all forms and at all ages, but more frequently in the tuberculous meningitis of children. When the inflammation is at the base, *rigidity of the neck* with retraction of the head is very marked, especially when the inflammation extends down the membranes of the spinal cord. *Optic neuritis* is another symptom, usually late in occurrence—at the end of the first week—and possibly due to involvement of the sheath of the optic nerve within the skull. *Strabismus* is also common. There may be weakness of the eye muscles and slight *ptosis*. The *pupils* are usually *contracted* in the early stage from intolerance of light; later, they are *dilated*. *Inequality* of the *pupil* is even a more characteristic symptom, though transient and variable. It occurs in connection with inflammation of the convexity as well as of the base.

The *facial nerve* may be involved in basilar cases, producing slight paralysis, as may also be the *fifth nerve*, producing anesthesia and trophic changes

in the cornea. On the other hand, *hyperesthesic* skin is often present; also *hyperesthesia* of the *special senses*, especially hearing and sight.

Symptoms in the limbs may present themselves, such as *muscular rigidity*, *unilateral convulsions*, and even *hemiplegia*, but the last is rare. When they occur, they are late symptoms.

Diagnosis.—The diagnosis is not always easy, because so many symptoms may be simulated by simple congestion due to the poison of *the infectious diseases*. The *basilar symptoms* are the most distinctive, and it is a real help to know that a possible cause is present, either predisposing or exciting; such, for example, as the tuberculous taint, or tuberculous disease, or middle-ear disease. Retraction of the head, so characteristic of this form, may result from myositis affecting the muscles of the back of the neck. Sir William Jenner pointed out a difference between the relation of headache and delirium in general disease and meningitis: In general disease the headache ceases when the delirium begins; in meningitis the headache continues and coexists with the disease. Convulsions, too, when present, occur at the beginning of a general disease, particularly in scarlet fever; while they occur late in meningitis. Optic neuritis and other eye symptoms are common in meningitis.

A rapidly growing *intracranial tumor* often gives rise to difficulty in the diagnosis between it and meningitis. In tumors which may be tuberculous or gliomatous, symptoms in the extremities, such as weakness, hemiplegia, and convulsions, are manifested only after the tumor begins to interfere with function, which it may not do at first; the loss of power, moreover, comes on gradually, while in meningitis all these symptoms are rapidly developed. Higher degree of optic neuritis, as observed by the ophthalmoscope, are found in connection with tumor than with meningitis. The duration of the disease will settle the question ultimately, as meningitis is of short duration—from two or three days to as many weeks—while tumors last for months or years.

Meningitis, especially tuberculous, is sometimes mistaken for *hysteria*, but the almost invariable presence of fever in meningitis and its total absence in most cases of hysteria should prevent error. In children the symptoms even of tuberculous meningitis are sometimes closely simulated in bad *cachectic states*, in which there is no meningitis whatever. What is regarded as meningitis after *sunstroke* is a prolonged state of mental hebetude with symptoms usually aggravated on slight exposure to the sun.

Prognosis.—The prognosis in leptomeningitis is unfavorable, although not necessarily hopeless. In meningitis of the convexity recovery is possible; in undoubted tuberculous meningitis it is very rare, and yet it may occur. Especially in general tuberculosis should we avoid too unfavorable a prognosis, because mistakes here are quite frequent. In meningitis from adjacent bone disease much depends on the accessibility of the bone lesion, but as this is generally difficult of access, the prognosis is correspondingly serious. This is especially the case in ear disease. In syphilitic meningitis if the diagnosis is made early, chances of recovery or improvement are better.

Treatment.—The *treatment of adjacent* disease which may cause the meningitis is of the first importance. Surgical interference should be promptly resorted to in middle-ear disease. In the absence of such disease the treat-

ment is mainly symptomatic. The utmost *quiet* and the avoidance of all causes of excitement are paramount. It is the one disease, outside of ophthalmia, in which the darkening of the room may be justified. The *head* should be *raised*. *Leeching* is a most valuable measure toward cure, when possible, and temporary relief when cure is impossible. Leeches should be applied to the back of the ear and to the temple. *Ice* should be kept applied to the head. *Counterirritation* by blisters to the back of the neck is also very useful, and not so painful or annoying as its appearance suggests. It has even been applied to the whole scalp after shaving the head, but there is no justification in this, especially when the diagnosis of tuberculous disease is quite clear. The *bowels* should be kept free.

The *diet* should be liquid—milk and animal broths of a light kind are the best food. Such drugs as meet the symptoms should be given. *Acet phenetadin* to relieve pain in the head if the ice and abstraction of blood do not do it. The temperature is kept down by *sponging* and even by cool *bathing*. *Mercury* is still an acknowledged drug in meningitis not tuberculous; and as chances of error of diagnosis always exist, it may be employed in any case. It should be administered to the production of slight salivation, preferably by inunction because the effect is more rapidly produced. The mercurial ointment should be used. Lumbar puncture has therapeutic as well as diagnostic value.

Chronic Leptomeningitis.

Etiology and Morbid Anatomy.—This comparatively rare disease affects chiefly the convexity of the brain, and is the result of alcoholism, syphilis, or tuberculosis.

In milder degrees, seen in alcoholics, the pia arachnoid is opaque, as seen over the sulci, the opacity and thickening being more marked along the borders of the blood-vessels. In syphilis there are often foci or thickened patches, thickest in the center and receding toward the edges. These may reach dimensions to justify the term gummatous outgrowth or tumor. The blood-vessels are the seat of endarteritis. In the tuberculous forms in children the base of the brain is affected, as in acute tuberculous meningitis. Internal hydrocephalus may be a consequence when there is obstruction of the orifices of the fourth ventricle.

Symptoms.—These are those of the acute form in a milder and more prolonged manner—headache, vomiting, mental symptoms, sometimes convulsions, rigidity, retraction of the head, optic neuritis, more rarely strabismus, and nystagmus. They may last from a month to a year or more. Fever is more frequently absent in chronic meningitis, but careful observation will generally find some elevation of temperature.

Diagnosis.—It is, in fact, the chronic variety of leptomeningitis which is separated from *tumor* with the greatest difficulty. Loss of motor power is more characteristic of tumor. Optic neuritis is also a more decided symptom in tumor, and goes on increasing, while it seldom reaches an advanced stage in chronic meningitis. Other eye symptoms—strabismus, irregularity of pupil—are more distinctive of meningitis. Strabismus occurs in *hysteria*, but it is always convergent and there is total absence of fever, as shown by the absence of elevation of temperature.

Prognosis.—This is not so unfavorable as in the acute variety. The syphilitic form is quite amenable to treatment, the alcoholic less so; the tuberculous is almost always sooner or later fatal. Caution in prognosis is demanded by occasional error in diagnosis.

Treatment.—The cause must be carefully sought. If syphilitic, iodids mercurials and salvarsan must be used, as for this disease. In alcoholism and tuberculosis the symptoms must be treated by measures already indicated.

AFFECTIONS OF THE BLOOD-VESSELS OF THE BRAIN.

HYPEREMIA.

SYNONYMS.—*Cerebral Hyperemia; Congestion of the Brain.*

Definition.—A condition of the brain in which the blood-vessels are surcharged with blood. The congestion is *active* as the result of increased flow of blood to the brain, as in alcoholic hyperemia; *passive* when there is obstruction to its outward movement, as in constriction of the vessels in the neck.

Etiology.—The causes of *active* hyperemia are prolonged mental activity, excitement, and overwork, pre-eminently alcohol and the causes of the acute fevers; the hypertrophy and overaction of the heart which attend aortic regurgitation may be causes. The causes of *passive* congestion are mainly mechanical, including mitral valvular heart disease, emphysema, straining, or other cause obstructing the return of blood from the brain—such as tumors pressing on the vessels of the neck, or tight clothing.

Morbid Anatomy.—While, from the standpoint of morbid anatomy, our ideas may be very definite as to what should constitute active and passive hyperemia, it cannot be said that a definite set of symptoms is associated with either in the case of the brain. In the first place, the amount of blood in the brain varies greatly within the limits of health, and while it might be said that physiological hyperemia ends where abnormal mental phenomena present themselves, it is undoubtedly true also that an overfullness of the vessels of the brain may exist for some time without the symptomatic expression which finally appears. With the appearance of such symptoms we commonly date the clinical beginning of the pathological state known as chronic hyperemia.

The difficulties are increased by the fact that in acute active and passive hyperemia, at least, no postmortem evidences of it remain, the congestion having disappeared with death, although an unusual distinctness of the puncta vasculosa has long been regarded as postmortem evidence. The difficulty of recognizing such condition makes this sign an unreliable one. In chronic hyperemia there result, sometimes at least, a turbidity and even an opacity of the pia mater, with slight thickening, together with elongation and tortuosity of the vessels, which are regarded as characteristic.

Symptoms.—These are not very distinctive. The symptoms of active hyperemia, so far as recognizable, are a sense of fullness or pressure, headache, mental excitement, irritability, confusion of ideas, insomnia, vertigo, ringing in the ears, and, in extreme cases, hallucinations, delirium, and

mania. These symptoms are increased when the head is held downward or there is straining. The phenomena of so-called "rush of blood to the head" are probably the result of active hyperemia. They include a suffusion of the skin of the face and head and a feeling of warmth in these situations, strong beating of the carotids, headache, tinnitus aurium, spots before the eyes, vertigo, and sometimes actual falling.

It is not easy to separate the phenomena of passive hyperemia from those of active congestion. They are, however, less pronounced and slower in their development.

Treatment.—The indications for treatment are, nevertheless, plain. The *head* is to be kept *raised*. *Purgation* is the first measure to be thought of. The saline and hydragogue cathartics are especially indicated, because of their depleting effect. The *ice-cap* should be used. In extreme cases even *blood-letting* may be necessary, the efficiency of which is sometimes seen in the relief afforded by bleeding of the nose. Leeches applied behind the ears often afford magical relief to the symptoms commonly ascribed to congestion of the brain. Wet cups may be placed upon the back of the neck for the same purpose. The diet should be spare and easily assimilable, in acute cases liquid only.

Of medicines, the *bromid of potassium* theoretically fulfills the indications, and in full doses of from 15 to 30 grains (1 to 2 gm.) every three hours to adults is often useful, though it should not be allowed to substitute the other measures mentioned. *Acetphenetadin* is an admirable remedy for the headache, a single dose of 10 grains (0.66 gm.) being often sufficient. It may be repeated if necessary, or smaller doses may be given more frequently.

ANEMIA OF THE BRAIN.

Definition.—The more usual application of the term anemia of the brain is to conditions in which the quantity of blood in the organ is diminished, although depraved states of the vital fluid without loss of bulk may also produce the same symptoms.

Etiology.—The causes leading to this condition are for the most part those which withdraw blood from the brain, but they include also such as prevent its access. Among the former are hemorrhages, profuse and rapid; bowel fluxes, such as those of cholera in adults and cholera infantum in children; and the opening of vascular areas by the removal of pressure caused by large tumors or ascitic fluid. Thus is explained the fainting which sometimes succeeds the removal of a large abdominal dropsy. In the second set of causes are feeble action of the heart, ligation of the carotid artery, or other obstruction in vessels carrying blood to the brain. Such obstructions are thrombi and emboli. The brain substance adjacent to the dilated ventricles in hydrocephalus internus is anemic from compression. The fainting due to sudden emotion, such as fright, is ascribed to a withdrawal of blood from the brain.

Morbid Anatomy.—This is more distinctive than in hyperemia. The membranes are pale, the blood in their vessels, except the larger ones, is scanty, and over the convolutions the vessels are quite empty. The gray and the white matter are both pale on section, and the puncta vasculosa are less distinct and less numerous. The cerebrospinal fluid is increased.

Symptoms.—Some of these are definite and the direct result of loss of blood to the brain. Such are the dizziness, confusion of ideas, flashings of light, roaring in the ears, nausea, and ultimate loss of consciousness and even death which succeed hemorrhages or emotion. In other cases the skin is cold and clammy, and a cold perspiration starts to the surface. Other symptoms are less distinctive. They are ascribed to chronic anemia, but may result also from other causes. Such are mental apathy, disinclination to work, a sleepy feeling during the day, and insomnia at night. Nausea, headache, tinnitus, vertigo, hallucinations, and delirium are also consequences more particularly of lowered composition of the blood, of anemia, in fact, the result of prolonged illness, like pulmonary consumption and Bright's disease. The convulsions characteristic of the latter disease have been ascribed to anemia and also to edema of the brain.

The hydrocephaloid symptoms, described by Marshall Hall as the direct results of prolonged diarrhea and of cholera infantum in children, are regarded as results of anemia. They include semistupor with eyes unclosed, later, dilated pupils, strabismus, convulsions, rigidity, and death.

Treatment.—The immediate consequences of the acute form of anemia are diminished or averted by placing the patient on the flat of the back with the head low; by diffusible stimulants, of which ammonia is the type; also cardiac stimulants, and nourishing and easily assimilable foods. The chronic forms of brain anemia are treated by nutritious, easily assimilable foods, and tonics, especially iron and arsenic. In the hydrocephaloid condition in infants alcohol is the pre-eminent remedy, associated with warm baths and general restorative measures.

EDEMA OF THE BRAIN.

Definition.—The term includes two conditions, the most definite and easily recognizable of which is an abnormal accumulation of cerebrospinal fluid within the pia arachnoid. In the second condition there is added to the first an abnormal moistness of the substance of the brain.

Etiology.—The most common cause is mitral stenosis, although any cause obstructing the return of blood from the brain as well as recurring irritative hyperemias, such as are produced by alcoholism and the psychoses, are also causes. Bright's disease is a cause of edema of the brain, local or general.

Local edemas of the brain are also caused by obstruction of single sinuses of the dura mater, or compression by tuberculous or other tumors of the veins of the velum interpositum, known as the *venæ Galeni*.

Morbid Anatomy.—The membranes are turbid, their vessels are distended and serpentine in their course, and the subarachnoid space is filled with clear fluid. The substance of the brain is anemic, moist, and glistening. In extreme cases there is compression of the cortex, with resulting flattening of the convolutions and widening of the sulci. The fluid in the lateral ventricles may also be increased.

Symptoms.—These are ill defined. There may be *hallucinations* and even *mania*, very similar, in fact, to those of anemia. Traube and Rosenstein ascribed the *convulsions of Bright's disease* to edema of the brain, while certain *unilateral* convulsions and *paralysis* in connection with this disease

have been assigned to the same cause. Even death has been ascribed to sudden serous effusions of this kind, constituting acute edema of the pia mater, or *apoplexia serosa*. In recent years much has been written on cerebral edema under the name of meningitis serosa.

Treatment.—The treatment is that of the conditions to which the symptoms are secondary. The effects of cardiac stenosis must be overcome by cardiac stimulants; Bright's disease must receive appropriate treatment. Thrombosis of the sinuses admits of no treatment, though its effects may diminish by gradual contraction and possible liquefaction and removal of the thrombus. The psychoses shoud receive treatment appropriate to them.

APOPLEXY.

Definition.—The term apoplexy is applied to a sudden loss of motor power with or without loss of consciousness due to cerebral hemorrhage, or the sudden plugging of a blood-vessel. Laceration of the brain without hemorrhage produces a like effect. In point of fact, when the term apoplexy, is used, cerebral hemorrhage is commonly intended.

Unconsciousness may also be produced by simple congestion, and it was formerly thought that a simple serous transudate could produce similar symptoms in a milder form and of shorter duration; whence the term "serous apoplexy." Concussion of the brain, also, causes similar symptoms.

I. Cerebral Hemorrhage.

Arterial Distribution.—In the first place hemorrhage is *meningeal* or *central*. Meningeal hemorrhage may be outside of the dura mater between it and the bone, or between the dura and the arachnoid, or within the pia arachnoid. The extradural and subdural meningeal hemorrhages are both traumatic, one variety of which is produced during birth, but those in the pia arachnoid are due to the causes to be considered below. Central hemorrhages may also burst into the membranes as well as into the ventricles of the brain and in some instances the hemorrhage is almost entirely intraventricular. Meningeal hemorrhage may occur in the infectious fevers, in leukemia, and in anemia.

It is a rare event to find a rupture in any of the large arteries of the circle of Willis, although white patches of atheroma are often seen upon them at autopsy. But the free anastomosis of this circle scarcely allows of increase of intravascular pressure sufficient to cause rupture. Further, it is the "central" rather than the "cortical" branches of this circle which rupture, and especially the central branches of the *middle cerebral*, which, entering the brain at the anterior perforated space, pass to the corpus striatum and internal capsule.

Etiology.—Disease of the artery involved is responsible for the vast majority of cerebral hemorrhages. Indeed, except in the case of traumatic hemorrhages either with or without fracture of the skull, it is very doubtful whether hemorrhage ever occurs without such disease. The simplest form is the fatty degeneration and "erosion" of the intima, characteristic of advanced age. *Endarteritis*, however produced, is perhaps the most fre-

quent cause. Its ultimate result, as shown by Charcot and Bouchard as far back as 1868, is the *miliary aneurysm* which very frequently precedes the rupture. It is a spindle-shaped, rarely lateral, dilatation, from 1/25 to 1/5 inch (1 to 5 mm.) in diameter. The inflammatory process preceding it consists in a proliferation and degeneration of the intima cells, followed by atrophy, which extends also to the muscular layer and the scanty adventitia. These, yielding to the intravascular pressure at the weak points, dilate to form the little aneurysm, which is later ruptured by some further increment of pressure. Embolism is also a cause of endarteritis which may result in aneurysm.

The "fatty erosion" of the intima which is the next most frequent cause of vulnerability is favored by age, by chronic interstitial nephritis, and the overstrain of the vessels due to hypertrophy of the left ventricle, so often associated with that disease as well as with valvular heart disease.

While by far the larger majority of hemorrhages are preceded by miliary aneurysm or fatty erosion—fully nine out of ten—there still remain a number of instances in which careful search fails to find anything but *diffuse degeneration;* whence the miliary aneurysm and fatty erosion cannot be regarded as indispensable conditions. The *infectious fevers*, *leukemia*, and *anemia* are also causes of hemorrhage which is independent of miliary aneurysm.

Age is also a predisposing factor, most ruptures occurring after 50, although apoplexy has occurred under ten; while the occupations and dissipations of men furnish additional predisposing elements which accounts for its greater frequency in the male sex. Other predisposing causes are those usually responsible for endarteritis—viz., gout, alcohol, syphilis, Bright's disease, the apoplectic habit, as seen in the stout, short-necked, full-blooded individual; and, finally, heredity, which is, strictly speaking, a hereditary tendency to the favoring diseases.

The exciting causes are such as temporarily increase intravascular pressure, as violent exertion, straining, debauch in eating and drinking, and mental emotion.

Morbid Anatomy.—The *large central ganglia* in the *neighborhood of the lateral ventricles—i. e.*, the optic thalami, the caudate and lenticular nuclei, and the adjacent white matter of the internal capsule and centrum ovale—are the favorite seats of miliary aneurysm and consequent hemorrhage. These aneurysms are found also, but much more rarely, in the smaller branches of the *cortical vessels*, in the *pons*, *cerebellum*, *crura cerebri*, or *medulla oblongata*. On section of the large ganglia these may be seen as small dark points, as large as a pin's head, and are often very distinct in arteries drawn out of the substance of the brain, especially the anterior perforated space. Larger aneurysms are also found on the branches of the circle of Willis.

Given a massive hemorrhage, what is its effect on the brain substance, and what are the changes in the extravasated blood? The former varies somewhat with its situation. If extradural, the dura mater is torn away from the bone to a varying extent. If subdural or beneath the pia arachnoid, it separates these membranes from the brain substance, but in either event the convolutions are more or less flattened and the sulci more or less obliterated.

As already stated, central hemorrhage most frequently occurs in the neighborhood of the corpus striatum, through which, if large, the blood finds it way toward the outer section of the lenticular nucleus, pushing inward the optic thalamus and bursting into the lateral ventricle or into the white matter of the centrum ovale. The pressure exerted is often such as to flatten the convolutions, empty the parietal veins, and press the falx aside, sometimes even to produce a sense of fluctuation over the membranes. Hemorrhages may occur rarely in the crura or pons or fourth ventricle, and also in the cerebellum, occasionally from the superior cerebellar artery. Osler mentions two cases of death in women of twenty-five from cerebellar hemorrhage. Very rarely hemorrhages into the ventricle may start in the choroid plexus or the ventricular walls. Blood in large quantities may be poured out at the base of the brain, and it may flow down upon the cord from a rupture of any of the arteries going to or from the circle of Willis.

If the patient survives, changes take place in the extravasated blood, which promptly coagulates into a dark red mass. This almost immediately begins to contract, permitting often the return of a certain degree of function by removing pressure. As time elapses the dark red mass passes into a chocolate-brown pulp, composed of liquefying blood-clot and disintegrated nervous matter. The microscope, at this stage, reveals numerous hematoidin crystals and granular fat-cells which are probably fatty by imbibition of fat-granules. The adjacent nervous tissue is stained yellow by the absorbed hematoidin. The clot itself becomes encapsulated by fibrin and gradually absorbed, being often substituted by a semitransparent or completely transparent fluid, forming the apoplectic cyst. If smaller, the walls approach and unite, leaving only a linear pigmented scar. Especially is this the case with small clots on the surface of the convolutions, which may leave only a staining of the membranes. In other cases of abundant cortical effusion, especially in infants, there may be circumscribed wasting of the convolutions and a cyst of the meninges or brain. The position and extent of the permanent lesion determine the presence of secondary descending degeneration. If the motor cortex or motor tract is involved, there may be found, in persons dying some years after a stroke of apoplexy with hemiplegia, degeneration in the pyramidal fibers of the pons and medulla oblongata, in the direct pyramidal fibers of the cord of the same side, and in the crossed pyramidal fibers of the opposite side, and to some extent in the crossed pyramidal fibers of the same side.

Symptoms.—*Premonitory* signs are rarely present. There may be a feeling of fullness in the head, headache, tinnitus, vertigo, or numbness, tingling, pains in the limbs on one side, loss of memory of words or choreiform movements—prehemiplegic chorea—possibly due to miliary aneurysm or otherwise diseased vessels.

With the bursting of a vessel of sufficient size there occurs the *apoplectic "stroke,"* or apoplectic shock. Its most striking feature is *sudden loss of consciousness*. If complete, the patient falls heavily to the ground, and there may be slight convulsive movement, but it soon ceases. More rarely a true convulsion ushers in the attack. The patient cannot be aroused, the face is suffused, cyanotic—sometimes, however, pale; the *breathing* is slow, noisy,

stertorous often attended with a puffing sound during expiration; corresponding with a blowing out of the relaxed cheek on the paralyzed side; it may also be of the Cheyne-Stokes type. In contrast with the foregoing, the development of unconsciousness is sometimes much more gradual, requiring several hours or a day, corresponding to which it is presumed that the hemorrhage is slow, constituting the "ingravescent form."

The second major symptom of apoplexy is *motor paralysis*, of which hemiplegia is the most conspicuous form. In most cases the motor pyramidal tract, as it descends in the internal capsule, is either directly destroyed or indirectly affected. Hence most patients who survive the primary shock present a hemiplegia—paralysis of half the body opposite that of the hemorrhage, and most frequent on the right side. It is most noticeable in the arms and legs. These are thoroughly relaxed, falling limp when allowed to drop, as the limb of one thoroughly etherized. More rarely there is early *rigidity*, especially on the paralyzed side. *Reflex action* is early either totally suspended or only brought out in response to a deep pin thrust or severe pinching.

The signs of hemiplegia are not always easily elicited at first, because a certain degree of consciousness is necessary to stimulate attempt at motion, but it may be that the angle of the mouth hangs down lower on one side—the paralyzed side—while the puffing of the cheek alluded to may be present on the same side, or the limbs of one side may be appreciably more flaccid than those of the other, or a small amount of reflex response may be elicited on the sound side. The *pulse* is usually slow, full, strong, and tense. The *temperature* may be subnormal at first, rising to normal and even above, and in basal hemorrhage may be higher. In a rapidly fatal case it remains subnormal to the end. The *pupils* are *irregular—i.e.*, sometimes contracted, at others dilated, unequal. They respond to light either slowly or not at all. If the hemorrhage is where it can irritate the nucleus of the third nerve, the pupil is contracted. This may occur with hemorrhage into the pons or ventricles.

In *cortical lesions* quite often one of the early symptoms in hemiplegia is *conjugate deviation* from the paralyzed side and toward the side of lesion, from which we have the expression that "the patient looks at the lesion;" that is, in right hemiplegia the head and eyes look toward the left side. This symptom usually passes away, but sometimes continues for weeks, and, as Gowers suggests, is perhaps occasionally represented by nystagmus or movement in the direction concerned. Should, however, convulsion, or spasm, or early rigidity develop, the head and eyes are rotated toward the paralyzed side—*i. e.*, away from the side of lesion. This is true only of cortical lesions.

In lesions of the *pons*, on the other hand, where the conjugate deviation may also occur, the phenomena are reversed—the patient looks away from the lesion, in the absence of spasm—but if the convulsion or spasm or rigidity occur, the eyes and head look toward the lesion. These facts are a little confusing at first and may be expressed in the following.

In lesion of the cortex—

Without spasm, conjugate deviation is *toward* the side of lesion.

With spasm or convulsion or early rigidity, *from* the side of lesion.

In lesion of the pons—

Without spasm, *from* lesion.

With spasm, etc., *toward* lesion.

This may be due to the fact that these movements in health are innervated from both sides, and when a lesion occurs on one side of the cerebrum, the innervation is given over to the other side until the injured one resumes its function. In pontile lesions the destruction occurs possibly below the decussation of the fibers innervating the parts affected in the conjugate deviation and the symptoms are reversed. Conjugate deviation in lesions of the pons is, however, a rare phenomenon.

Where unconsciousnes exists the *feces* and *urine* are passed involuntarily, and the latter is sometimes slightly albuminous.

As to further progress in a few cases there is no reaction from the previously described condition. The symptoms all deepen, the breathing becomes rapid and rattling, the skin cool, the pulse weak and rapid, and the patient dies. In most cases, however, there is a certain abatement of the symptoms, even if the patient does not recover more fully. Consciousness returns partially or completely, the patient can be aroused by a loud voice, and one can recognize which side is paralyzed. There may, at this time, be a febrile movement, due to cerebral inflammation or disruption of heat-regulating centers, during which the patient may die; or there may be another hemorrhage which carries him off.

On the other hand, improvement may continue to a further degree. The consciousness and intelligence may return completely, and the signs of paralysis may gradually grow less, *more rapidly in the legs than in the arms*. They, however, almost never disappear completely, the patient continuing lame and requiring the use of a cane for the rest of his life. In severe cases a remnant of paralysis of the face can almost always be recognized, while articulate speech may also continue defective.

Such marked improvement is, for the most part, reserved for the milder attacks, in which there is great variety as to degree. In such the loss of consciousness is of short duration, or it may not occur at all. Such attacks are not infrequently ushered in by nausea, vomiting, vertigo, or sudden headache. The paralytic symptoms may still be marked, and permit a study rather more satisfactory than the fulminating cases. In such study it will be found that all muscles are by no means equally paralyzed. Thus it will be seen that the lower division of the facial nerve, which supplies the muscles of the cheek, nose, and mouth, is plainly paralyzed; while the upper division, distributed to the muscles of the eyes and forehead, is almost, if not entirely, intact. The forehead may be wrinkled with equal ease on the two sides, but an attempt to draw up the nose or purse the mouth fails, while one labionasal fold may be obliterated and one angle of the mouth lower than the other. The natural wrinkles of the forehead are commonly less distinct on the paralyzed side than on the other. This event—the comparative freedom from paralysis in the upper part of the face—may be explained by the fact that while both sides of the face receive fibers from each cerebral hemisphere, this is especially true of the muscles of the upper part of the face, which are always exercised bilaterally.

The *tongue* may not be paralyzed, but when it is, if protruded, it usually

but not always goes toward the paralyzed side, being pushed out by the geniohyoglossal muscle of the other side, the innervation being by the hypoglossal nerve. Occasionally paralysis of the tongue contributes to difficulty in articulation. The motor branch oft he fifth nerve is sometimes involved on the hemiplegic side, and there is paralysis of the pterygoid, temporal, and masseter muscles.

Of the *trunk muscles*, the trapezius is most involved, and that but slightly, permitting the shoulder to drop a little, and the paralyzed side of the chest may expand more than the normal side in ordinary breathing, while in voluntary deep breathing this is not the case. The reason of this possibly may be found in the exaggeration of the reflexes on the paralyzed side; ordinary breathing being a reflex action.

Sensation is but slightly impaired in most cases of hemiplegia due to cerebral hemorrhage, and such impairment usually grows rapidly less as time elapses, unless the optic thalamus is seriously damaged. It is hemianesthesia when anesthesia exists, and it is on the side opposite that of the lesion. There may also be trifling paresthesia at first. Any marked disturbance of sensation means that the posterior extremity of the internal capsule is involved, or, according to some authors, it indicates that the optic thalamus is invaded. Distinct impairment of the deep sensibility—the so-called muscular sense or sense of position—may indicate a lesion of the parietal lobe. There is sometimes temporary and even permanent *hemianopia*, which implies some lesion of the fibers of the optic radiation posterior to the internal capsule or in the posterior portion of the optic thalamus—the pulvinar.

Astereognosis or the inability to recognize objects by touch is sometimes a symptom.

The *tendon reflexes* are increased in nearly all cases on the paralyzed side, though at the very beginning of a severe shock they may be abolished, and if this abolition of the reflexes persists, it is regarded as a serious sign. In cases of any duration even the periosteal reflexes are increased, and to a less degree the reflexes of the sound side are increased, because each side of the body is innervated from both sides of the brain, although the number of fibers passing to the same side of the body is considerably less than those passing to the opposite side. There is even, at times, ankle clonus, and, more rarely, wrist clonus. These events are explained by supposing a suspension of the inhibitory reflex cortical centers, due to the cerebral lesion. The *skin reflexes*, on the other hand, are diminished on the paralyzed side, remaining normal on the sound side.

The *rapid improvement* mentioned as occurring in some cases is usually confined to a few weeks or days, after which improvement goes on more slowly, the lower extremities recovering more completely than the upper. The *gait* resulting from partial recovery is peculiar. Short steps are taken by the affected leg, and the toe is dragged more or less, while locomotion is sometimes accomplished by sweeping the leg around in a semicircle by the iliacus and psoas and the vastus externus, while it is held stiff, as in a splint, by the quadriceps extensor muscle. In the upper limb the hand muscles are the last to recover.

Later in the history of the case *contractures* may come on in the para-

lyzed muscles, shown especially in flexion of the fingers, contracture of the forearm in a position of pronation, and partial flexion, with the upper arm adducted. The lower extremity is usually in the position of extension. This contracture is explained by some, and notably by Strümpell, as a "passive contracture," the position assumed being the natural one in a state of rest. On the other hand, Charcot and his pupils hold that the contractures are due to secondary degeneration of the pyramidal tract. It is very doubtful whether secondary degeneration produces symptoms.

There are also sometimes *associated movements* of the paralyzed muscles, to which Hitzig has called attention. In these, movements of the sound side excite associated movements in the corresponding muscles of the other side, and attempts to move the affected side result in motion of corresponding muscles of the sound side. Sometimes, also, involuntary movements of the lower extremity occur when the patient attempts to move the corresponding arm. A *posthemiplegic chorea*, first described by Weir Mitchell, should also be mentioned. It is seen not so much in the hemiplegia resulting from cerebral hemorrhage as from focal disease of the posterior end of the internal capsule and optic thalamus. A form of *hypertonia* has recently been described in which the muscles are in a state of exaggerated tonicity without much paralysis. In this condition the position of the spastic limbs varies from time to time. It is seen in some cases in which a cerebral lesion has occurred early in life.

Trophic symptoms may appear late in the disease, seen at first in elevation of temperature, increase of color on the paralyzed side of the face, swelling of the eyelids, and contraction of the pupil; also swelling of the hands. It is to be remembered, however, that slight swelling may result from sluggish circulation of blood and lymph, contributed to by diminished muscular contraction and absence of use. In a more advanced stage the extremities become cooler and are often constantly moist. Among these vasomotor events Charcot has placed what he calls *acute malignant decubitus*—a disposition to rapid gangrene of the tissues over the sacrum. It may appear in a few days after the shock, beginning with a circumscribed redness and formation of vesicles, succeeded by deep-reaching necrosis. While this is probably, as Charcot regards it, a vasomotor phenomenon, it is also invited by the usual causes of gangrene in dorsal decubitus, such as irritation by urine, feces, and even inequalities in the bed-clothing. Charcot also considers an occasional arthritis, acute or chronic, a neuropathic event.

General nutrition is well maintained, the patient even gaining in flesh at times. More rarely there is rapid wasting.

The *mental condition* of patients who recover partially from the effects of hemorrhage is, for the most part, good, but it not infrequently happens that after a time mental weakness manifests itself in loss of memory and defective intellection, while imbecility sometimes ultimately supervenes.

Diagnosis.—The greatest difficulty lies in the differential diagnosis between cerebral hemorrhage, *embolism*, *thrombosis* and *cerebral sclerosis*. We will, however, defer its consideration until cerebral embolism and thrombosis are treated.

In *fulminating cases* the coma is sometimes so profound that it is difficult or impossible to ascertain the presence of hemiplegia. The symptoms

which aid in determining this have been mentioned on page 1038. To these may be added the increase of reflexes on the affected side, present in an early stage of the paralysis, conjugate deviation of the head and eyes, and rigidity of limbs on one side. It is this condition that is sometimes confounded with *epilepsy, opium poisoning, acute alcoholism, or uremia.* In *epilepsy* there is the history of previous convulsions, and it is only when this has been overlooked that mistakes occur. In *opium poisoning* the coma is slow in its onset, the pupils are uniformly contracted, and the odor of laudanum is often on the breath. But here, too, the victim is often only discovered after coma has thoroughly developed. In *alcoholism* there is the odor of whisky, but many an innocent person has been treated as a drunkard on whose brain lay a clot pressing him to death. The young ambulance or police surgeon is wise who defers his opinion. Sometimes alcoholism and apoplexy are combined, in which event a conservative course will be no less astute. The coma of *uremia* in Bright's disease very strongly simulates that of apoplexy, especially in the rare cases of the latter in which there are convulsions. The presence of dropsy, or, in its absence, of the peculiar anemia of Bright's disease, and the finding of albuminuria and casts should suggest this disease, but albumin may be found in hemiplegia not of renal origin. It is to be remembered, too, that uremic convulsion may terminate in hemorrhage, while Bright's disease is also associated with a state of the arteries which disposes them to rupture. The use of the phthalein test will make for or against a kidney lesion. Coma in a puerperal woman, associated with high blood pressure dropsy and albuminuria, means taxæmia of pregnancy.

Prognosis.—To have had a stroke of paralysis is justly regarded as having received a blow which marks the beginning of inevitable decline in health and usefulness, though cases are constantly occurring in which a "slight stroke" is followed by complete recovery. Some of these are probably errors of diagnosis, many being temporary loss of power caused by cerebral sclerosis, yet all are not. Minute areas of softening and cortical hemorrhages are the lesions most frequently followed by recovery. After these come a large number of cases of first attack, from which the patient recovers quite a considerable degree of health. Second attacks are prone to occur, which usually are more severe, and few survive a third serious attack.

The unfavorable cases are those in which the coma is profound and lasting. Such are hemorrhages into the ventricles and corona radiata, which are rapidly fatal. Meningeal hemorrhages are serious, but less so when traumatic than when due to diseases of the vessels. Cases attended by early and persistent fever and delirium are unfavorable, as are also cases complicating renal disease and alcoholism. Hemorrhages into the corpus striatum and internal capsule produce persistent hemiplegia, followed by contracture. When cases survive the primary stroke and improvement sets in, this is much more rapid in the first few weeks than later. In explanation of this it has been held that the symptoms thus rapidly removed are indirect focal symptoms, due to pressure of the clot on adjacent nervous tissue, while those more slow to yield are the result of destructive lesion.

Treatment.—The patient should be promptly placed in a horizontal position *with the head raised.* This is of the greatest importance, as it con-

stantly happens that a patient in whom consciousness is returning immediately becomes comatose when the head is lowered. *He should then be bled* if the blood pressure is high or rising. The bleeding should be accompanied by a *laxative*, which should be given alone if there be any reason why phlebotomy should not be practiced. In view of the unconscious state of the patient the best laxatives are croton oil and elaterium. Two drops of the former should be mixed in a little glycerin or oil and carried to the back part of the throat, or 1/4 grain (0.0165 gm.) of elaterium, dissolved in a small quantity of water, may be given in the same way. The rectum should be at once cleaned out by an enema of warm water. An ice-bag should be placed on the top of the head, hot water and mustard to the feet, while counterirritation may also be applied to the back of the neck. The patient should be kept in bed as long as there is any evidence of bleeding.

Compression of the carotid artery, formerly recommended and practiced on empirical grounds, has received the indorsement of Horsley and Spencer, these experimenters having found that bleeding from the lenticulostriate artery ceases when the carotid is compressed. It is especially in the ingravescent form that it has been recommended.

If, after bleeding and purgation, the pulse continues bounding, the *tincture of aconite, veratrum viride* or nitro-glycerine may be given in doses of a minim every half-hour until the pulse is influenced. *Iodid of potassium* can hardly be expected to promote absorption of the clot, but may be given if syphilis is suspected. It may, however, facilitate circulation by lowering peripheral pressure.

The foregoing treatment is for the period immediately succeeding hemorrhage. The remainder of treatment consists in measures to protect the patient against the effect of decubitus if this is prolonged, and in maintaining the nutrition of muscles and protecting against contractures. The former is accomplished by attending to the secretions, preventing the irritation of the body by putrid urine and feces or foreign substances like bread-crumbs, by bathing and drying the body thoroughly, by frequent changes of posture. The latter will also guard against pneumonia, which is rather prone to *occur* on the paralyzed side. This last disease may also be caused by the inspiration of particles of food, liable to happen if there is paralysis of the muscles of deglutition. The second indication is met by massage, faradization, and gymnastics, but they should be deferred for two or three weeks. *Warm salt baths* three or four times a week are useful to the same end. Tonics in the form of iron in small doses, quinin, and strychnin may be given, but alcohol in more than very moderate amounts is contraindicated.

Operative treatment has been suggested to relieve the pressure of a clot in cerebral hemorrhage. When the clot is meningeal, especially after fracture, operation is imperative. Cushing has shown that a rising blood pressure is due to an attempt to counteract the intracranial pressure. He advises decompression to relieve pressure and removal of the colt if practicable. Careful attention should be paid to the facts mentioned under topical diagnosis with a view to determining the seat of hemorrhage and the place to trephine.

II. Embolism and Thrombosis of the Cerebral Vessels.

A. Of Cerebral Arteries.

Synonyms.—*Cerebral Softening*; *Acute Softening*.

Definition.—By *embolism* is meant the plugging of an artery by a foreign body carried by the blood-current from some point in the vascular system to a situation beyond which it cannot pass. By *thrombosis* is meant plugging of an artery or vein by a clot formed *in situ*.

Etiology.—*Nature and Source of Embolism.*—The embolus is most frequently a vegetation from a diseased valve in the left ventricle. Less commonly it is a fragment of a clot in the same ventricle or in the auricular appendage or in an aneurysm, or it may be a calcareous particle from an atheromatous vessel or a piece of thrombus from the same. Even the territory of the pulmonary veins may contribute an embolus. Embolism is very much more frequent in chronic valvular disease than in primary acute endocarditis. It is prone to occur in recurring valvulitis, and especially in malignant mycotic endocarditis. Pregnancy with or without heart disease, the infectious fevers, and blood dyscrasiæ may be predisposing causes.

The embolus commonly enters the brain by the carotid, especially the left—which furnishes the most direct course—thence through the internal carotid to the *left middle cerebral* in the fissure of Sylvius; more rarely by the *vertebral* and the *posterior cerebral artery*.

Thrombosis.—In thrombosis there is also plugging of a vessel, but by a clot formed *in situ*, which is either primary at the point plugged or secondary about a previous embolus. Some favoring cause commonly exists. This is most frequently roughening due to endarteritis, with or without atheroma. Weak heart and blood dyscrasiæ are also predisposing causes. Ligation of the carotid artery is sometimes followed by thrombosis of cerebral vessels.

The vessels most frequently affected in thrombosis are the *middle cerebral* and the *basilar* in its course or at its bifurcation; but the vertebral, the posterior cerebral, and the branches of the circle of Willis may be plugged.

Relative Frequency of Thrombosis and Embolism.—Embolism has been thought to be more frequent in women, but of 79 cases collected by Newton Pitt at Guy's Hospital, 44 were in men and 35 in women. Thrombosis is considered more common in men. Embolism is rare in children, being more frequent at from 20 to 50; thrombosis in older persons at from 50 to 70.

Morbid Changes Due to Thrombosis and Embolism.—*Degeneration* and *softening of the brain* are the direct result of obstruction of its arteries, and occur sooner or later when the shutting off of the blood-supply is sufficiently complete. The process generally begins within 24 hours and the minimum time required to complete it is from one to two days. The local anatomical product of embolism is much less distinctive in the brain than in the lungs or spleen. Thus, there is almost never a distinct hemorrhagic infarct, though there is often a condition resembling it, the area cut off being infiltrated with blood. At other times the region is paler than in health and slightly softer. In either event the area becomes gradually infiltrated with serum and a more or less complete liquefaction results,

presenting a reddish, yellow, or white color, whence the terms *red* softening, *yellow* softening, or *white* softening. These variations are not the result of any essential difference in the nature of the process, as was formerly thought, but are rather accidental. In red softening the softened focus happens to contain an unusual amount of extravasated blood, due to punctiform hemorrhage or capillary bleeding. This blood melts away and stains the softened mass. In yellow softening the proportion of fatty degenerated cells is larger, and it is found, therefore, chiefly in the cortex, where cells prevail. In white softening there are few or no cellular elements, hence the white softening is found in the white nervous matter. It is most characteristically seen about tumors and abscesses. As the gray matter of the cortex is also the most vascular part of the brain, it is here also that we find red softening. Certain superficial yellow spots known as *plaques jaunes* are found at times on the surface of the cortex in old persons. They are sharply circumscribed, measure from 2 to 4 centimeters (0.8 to 1.6 inches), are made up of a yellow, turbid material sometimes crossed by trabeculæ, and are the result of fatty degeneration of peripheral cortical arteries.

Minutely examined, the softened areas consist of fatty granules and oil drops, myelin drops, fragments of swollen nerve fibers, fatty granular cells representing fatty neuroglia and nerve cells, or leukocytes and neuroglia cells, and perhaps endothelial cells which have *imbibed* the oil drops, arising probably from disintegrated nervous matter. In the yellow softening these constitute the sum of altered materials. In red softening there are added in the early stages blood-disks, later pigment granules or hematoidin crystals, or there is general staining by dissolved hemoglobin. In the white softening the fragments of nerve fibers together with myelin drops make up the chief bulk, as already stated. If collateral compensatory circulation is set up within two days, the destruction may not go so far, and the nervous elements may resume their function; or if this does not occur and the patient lives, the dead and disintegrated tissue may be gradually absorbed and eventually be replaced by a cyst, while a minute focus of softening may be replaced by indurated cicatricial tissue. If the embolus is derived from an infective focus, as ulcerative endocarditis, an abscess may result.

Symptoms.—Neither thrombosis nor embolism of the cerebral arteries is always followed by recognizable symptoms. All the large arteries of the base and the smaller arteries of the surface anastomose so freely that the effects of obstruction are promptly equalized. Nay, more; it is not unusual to find at the necropsies of elderly persons yellow spots of fatty degeneration; the *plaques jaunes* referred to, scattered over the convolutions where nothing was suspected before death. Moreover, softening may take place in the "silent regions" without exciting suspicion.

Very different is it with obstruction of the middle cerebral artery—the artery of the fissure of Sylvius. The clinical aspect differs, however, according as this vessel is plugged at its origin or a little further on in its course. Allusion has already been made to the two separate systems with which the brain is supplied—the "cortical arteries" (Duret), passing to the cortex, and the "central" arteries, passing to the central ganglia. The central arteries are the first given off by the cerebral branches of the circle of Willis, and are terminal arteries, unprovided with anastomoses. The

cortical arteries spring from a network of branches of the cerebral arteries in the pia mater, in which tolerably free communication exists between the tertiary branches of the same trunk, and even between the branches of different trunks. These two systems are, however, altogether independent of each other, and no anastomosis takes place between them, the zone at which they meet within the cerebral substance being situated about an inch and a half below the cerebral convolutions. In the case of the middle cerebral artery, when it is obliterated beyond the point at which its "central," branches come off, the superficial parts of the brain are alone affected, and since its branches in the pia mater anastomose with those of the anterior and posterior cerebrals, there may be no softening at all, and but a temporary loss of function. At other times softening does occur, the exact situation and extent of which vary with the arteries plugged. The blood-supply of the central, frontal, and parietal convolutions being more or less cut off, there is motor paralysis of the opposite side of the body, and as the lesion is most frequent on the left side, there are right-sided hemiplegia and aphasia; the same phenomena, in fact, as follow hemorrhage, and which may be permanent or transient; or the lesion may be still more limited. The embolus may lodge in the artery passing to the third frontal convolution, or in that of the ascending frontal or ascending parietal. It may lodge in the branch passing to the supramarginal or angular gyrus, or to the lowest branch, which is distributed to the upper convolution of the temporosphenoidal lobe. If, on the other hand, the seat of the lesion is at the point where the Sylvian artery arises from the internal carotid, the central ganglia are involved, and there is almost certain to be softening of the corpus striatum and optic thalamus, because the arteries have no anastomoses, while the cortex escapes entirely because its vessels are distinct.

Diagnosis.—It has already been said that the chief difficulty lies in the differential diagnosis between cerebral hemorrhage, on the one hand, and embolism and thrombosis on the other. Sometimes, indeed, at first it is impossible. As to *embolism*, both it and hemorrhage are sudden. In embolism the patient is commonly younger, but not always so, and we look for valvular heart disease. According to Charles L. Dana, even in patients between the ages of 30 and 50, when there is no heart disease, the chances are six to one in favor of hemorrhage. *An apoplectic seizure after parturition is likely to be embolic.* In embolism, too, there is less disturbance of temperature, the paralysis is more likely to precede the coma and convulsions if the latter are present; the turgid face, hard pulse, loud breathing, and greater general disturbance of a serious stroke of apoplexy from hemorrhage are wanting.

In *thrombosis* the difficulty in diagnosis may be even greater. The symptoms of thrombosis are slower in their development, but in the "ingravescent" form of apoplexy, in which the hemorrhage is gradual, requiring sometimes a day or two, the development of symptoms is correspondingly slow. In thrombosis there are more frequently prodromata in the shape of slight seizures, quickly recovered from. Such events occurring in the aged, when there is evident atheroma of the blood-vessels and weak heart, point to thrombosis, in which, too, there is absence of stertorous breathing, of variations in temperature, and of pupillary disturbance.

It is also important to be able to decide whether the obstruction is embolic or thrombotic. In the former the onset is sudden, without premonitory symptoms; in the latter it is gradual, and there are often premonitory symptoms. In embolism there may be convulsive twitchings, but hemiplegia quickly follows, without or without temporary loss of consciousness. In thrombosis the patient has previously complained of headache, vertigo, or tingling in the fingers; then paralysis may begin in one hand or foot and extend slowly, the hemiplegia often remaining partial. Speech may have been embarrassed for some days previous, and the memory defective. In thrombosis due to syphilis, especially, the hemiplegia may come on gradually without loss of consciousness. The same is true of the so-called senile softening, which is generally due to thrombosis after atheroma of the cerebral arteries. In a few cases the onset is more sudden, and may happen during sleep. The temperature usually has a slight initial fall, followed by rise, as in hemorrhage. In embolism aphasia is quite a characteristic symptom, as it seems to occur more frequently on the left side than on the right.

In both embolism and thrombosis the hemiplegia tends to improve rapidly unless the vessel obstructed be a large one or there be rupture of a collateral branch. It is true that acute softening may terminate fatally within 24 hours, but usually the patient survives the onset, and at the worst dies after several weeks, the phenomena of the chronic stage being almost identical with those of that stage after hemorrhage. Spastic symptoms may also occur, and there is a tendency to the characteristic mobile spasm.

Prognosis.—A patient rarely dies of a first attack of cerebral embolism, unless a very large vessel is obstructed, such as the internal carotid or basilar, whose occlusion is usually fatal; next in seriousness after these is plugging of the middle cerebral and vertebral, while obstruction of the two vertebrals is always fatal. Every succeeding attack increases the danger. Embolism is less serious than thrombosis. Sudden severity in thrombosis is serious, and deranged breathing is an unfavorable symptom. Convulsions may be a result of syphilitic thrombosis. When the embolism is due to valvular heart disease, the condition is likely to recur; when due to other causes, not. Thrombosis is prone to recur, especially when caused by atheroma.

Treatment.—Neither thromobsis nor embolism demands blood-letting. Indeed, it is strongly contraindicated. *Rest in bed*, with head raised, is important. If syphilis is the cause of thrombosis, it should receive the usual treatment—the iodid of potassium in ascending doses until doses of a dram or more are reached, or mercury or salvarsan. There is no treatment for atheroma. Attention should be paid to the heart, kidneys, and bowels. The heart is commonly feeble, and digitalis and strophanthus are needed to keep its action uniform and strong, by which one condition resulting from thrombosis is removed. The urine is scanty and highly colored, but the treatment for the heart is also the treatment for the scanty secretion, which calls also for diluents. The bowels should be kept freely open. The circulation is aided by nitroglycerin, which may be given in doses of 1/100 grain (0.0066 gm.) every two hours. The iodid of potassium is useful also for this purpose. Its effects are more permanent than those of nitroglycerin. From

5 to 15 grains of the iodid (0.33 to 0.99 gm.) should be given three times a day.

Moderate stimulation is beneficial. The aromatic spirit of ammonia and alcohol are the most useful for this purpose. Mental excitement is to be especially avoided after a return to consciousness, and physical rest should be continued. Stimulants are then best discontinued, or continued in great moderation. Care should be taken to protect against the effects of decubitus

Unfortunately there is no treatment which will restore softened brain matter, although a certain amount of function may be vicariously assumed. The same measures calculated to maintain nutrition and muscular integrity as are recommended in the treatment of hemorrhage should be taken.

Thrombosis of the Cerebral Sinuses and Veins.

Description.—Thrombosis occurs in the sinuses rather than veins and is *primary* or *secondary*. Primary thrombosis is the result of a state of the blood and circulation; secondary, a consequence of disease adjacent to the sinuses. The former is much the rarer, occurring half as often.

Primary thrombosis is met in the longitudinal sinus, more rarely in the lateral, sometimes in the cavernous. It is found associated with general malnutrition and prostration, more frequently in children during the first six months of life as the result of exhausting maladies, especially diarrhea. It is met also in older children. Brayton Ball and others have shown its association in young girls with chlorosis and anemia. It occurs in the aged also as the result of exhausting disease, like pulmonary tuberculosis and cancer.

Coagulation is favored by the trabeculæ which cross the cavity of the sinus, and by irregularities in the shape and lining of the latter. It may or may not be associated with phlebitis.

Very little is known of primary thrombosis of the cerebral *veins*, except that it may occur in veins of the convexity as the result of meningitis, and from other causes that produce thrombosis of sinuses.

Secondary thrombosis occurs at any age, and is the result of disease adjacent to a sinus, commonly caries of bone, and is especially frequent as the result of disease of the internal ear. It spreads more frequently from the posterior wall of the middle ear, but also from the mastoid sinuses. Fracture, suppurative disease outside of the skull, especially erysipelas, and tumor compressing the sinus may produce it.

Symptoms.—There may be no symptoms in primary thrombosis, or there may be *nausea* and *vomiting*, *headache*, and *hebetude* increasing to *coma*. Dilatation of the pupils, choked disks, and paresis have been reported.

Secondary thrombosis is a septic process. It is commonly announced by a *chill*, followed by *fever* and *occipital pain*, succeeding on earache with suppurative otitis. The sinuses occluded are those near the ear, but the blood escapes by other channels, and the brain substance is not seriously invaded. The *symptoms of meningitis* are soon added. They are headache, somnolence, and stupor, or there may be active delirium and convulsions, rigidity, or optic neuritis, all the results of meningitis. Death is most frequently caused by *suppurative pulmonary pyemia*, as was the case in 70 per cent. of

Newton Pitt's cases, and the appearance of the latter disease under the circumstances is almost conclusive evidence of previous sinus thrombosis.

Prognosis.—This is always grave. The average duration of the secondary disease is about three weeks, and its termination is almost always fatal. Pitt reports a case of recovery in a boy of ten who had otorrhea for years, after removal by operation of a foul clot from the lateral sinus.

Treatment.—For primary thrombosis there is no treatment except that for its cause. For secondary, operative treatment is indicated by trephining or other measures to give exit to pus. Quinin and restorative measures are indicated. Gowers lays particular stress on the use of the tincture of the chlorid of iron.

INTRACRANIAL ANEURYSM.

Definition.—Intracranial aneurysms are of two kinds, miliary and those of larger size. The former have been considered when treating of hemorrhage. The latter vary in size from that of a pea to a walnut.

Distribution.—Larger aneurysms affect the larger arteries at the base of the brain in the following order:

1. Middle cerebral.
2. Basilar.
3. Internal carotid.
4. Anterior cerebral.

The anterior or posterior communicating and vertebral arteries are also occasional seats; the posterior cerebral and inferior cerebellar rarely. William Osler found 12 of these aneurysms in 800 autopsies, and Newton Pitt 19 in 1900.

Etiology.—Intracranial aneurysms are found rather more frequently in the male sex, and most frequently between the ages of 10 to 60. Osler and Pitt each found one at the age of six. Heredity exercises some influence. Endarteritis and embolism, both of which weaken the vessels, are the chief causes. The former may be syphilitic or simple. The presence of endocarditis should especially invite examination for aneurysm at autopsies.

Symptoms.—*Death* from *apoplexy*, owing to rupture of the aneurysm, may be the first intimation. Not only are there often no symptoms, but when present they are vague. They may be those of tumor at the base of the brain, including optic neuritis and paralysis of the third and other cranial nerves. There are rarely *convulsions*. There may be *headache*, *vertigo*, *nausea*, *hebetude*, and even *coma*, *hemiplegia*, and *hemianopia*. A *murmur* may be heard on auscultating the skull, while occasionally the patient himself is conscious of a murmur or recognizes the pulsations in his head.

Diagnosis.—This is usually impossible, but the foregoing symptoms, associated with endarteritis, may excite suspicion. *Syphilitic disease* being as likely to produce tumor, the history of its presence gives no assistance in diagnosis.

Treatment.—None exists which can be specifically directed to the disease.

THE CEREBRAL PALSIES OF CHILDREN.

Definition.—Referring to the division already made of the motor path into an upper cortico-spinal segment, extending from the cells of the cortex to the gray matter of the cord, and a lower spino-muscular, extending from the ganglia of the anterior horns to the motorial end-plates, the diseases now to be considered have their anatomical seat in the former, and are characterized by paralysis, with spasm or disordered movements, exaggerated reflexes, normal electrical reactions, without rapid or extreme wasting. They result from a destructive lesion of the motor centers, or of the pyramidal tract in the hemisphere, internal capsule, crus, or pons. The condition is hemiplegic, diplegic, or paraplegic.

Spastic Infantile Hemiplegia.

Synonyms.—*Hemiplegia spastica cerebralis* (Heine); *Hemiplegia spastica infantilis* (Bernhardt); *Acute Encephalitis der Kinder* (Strümpell); *Die atrophische Cerebrallähmung* (Henoch); *Agénèse cérébrale* (Cazauvieilh); *Sclérose cérébrale atrophie partielle* (other French writers). *Hemiplegia in infants and children with spastic symptoms.*

Distribution.—The disease is somewhat more common in girls than in boys, 63 out of 120 cases studied by Osler at the Nervous Infirmary in Philadelphia being of this sex. Of these cases 15 were congenital, 45 began in the first year, 22 in the second, 14 in the third, one in the fourth, three in the fifth, sixth, and seventh, one in the eighth, ninth, tenth. In ten the age of onset was not given. The hemiplegia was right-sided in 68 and left-sided in 52 cases.

Etiology.—Among the causes may be mentioned abnormal conditions of the mother during pregnancy, including accidents, possibly disease, especially syphilis, in a few cases fright or distress, the effect of the last two being doubtful. Especially frequent causes are difficult or abnormal labor, injury with forceps producing depressions and fractures of the cranial bones during delivery. After birth are penetrating wounds of the head, ligation of the common carotid, and infectious diseases, including whooping-cough, diphtheria, scarlet-fever, measles, meningitis, typhoid fever, vaccinia, and mumps. Previous convulsions may cause the lesion on which the paralysis depends, and in a few cases embolism may be responsible.

Morbid Anatomy.—The morbid states of the brain found at autopsy are mainly *sclerosis* and *porencephalia*, the latter a defect consisting in arrest of development of the brain shown by absence of convolutions or even lobes, causing irregular subpial cavities. *Embolism* and *thrombosis* of vessels, especially of the Sylvian artery, and *hemorrhage* into the ventricle and substance of the brain, have been found in a few cases. The sclerosis involves groups of convolutions, an entire lobe, or even an entire hemisphere. The *skull* may be flattened on the affected side, broad and prominent above the mastoid processes, sometimes thickened. The *dura* may be thickened and adherent; the *arachnoid* turbid and thickened and the amount of cerebrospinal fluid increased. The *pia mater* may be thickened and adherent, and portions of the cortex may be torn away when the pia is removed, leav-

ing a roughened surface, while there may be nodular projections of sclerosed tissue. The *reduction of weight* of the sclerosed hemisphere may be very considerable; in one case, referred to by Osler in his monograph, the atrophied hemisphere weighed 5 1/2 ounces (169 gm.), the normal being 20 ounces (653 gm.). The lateral *ventricle* may be greatly *dilated*, and the brain tissue over it very thin, while *cysts* have been found in the sclerosed areas—the remnants of old hemorrhages. The Rolandic area is that most frequently involved.

In 90 cases studied by Osler the lesions in 50 were atrophy and sclerosis, in 24 porencephalia, and in 16 embolism, thrombosis, or hemorrhage.

Symptoms.—The symptoms are complex and varied, but may be divided into three classes: those of the *onset*, those pertaining to the *paralysis*, and the *residual* symptoms.

The most important symptoms of the onset are *convulsions* and *coma*, although the hemiplegia may come on suddenly, without spasm or loss of consciousness, in children apparently healthy. In the majority of cases, however, the disease begins with the convulsions, partial or general. Loss of consciousness almost always accompanies the convulsions, and may last from a few hours to many days. Rarely coma occurs without convulsions. Among other symptoms may be mentioned *fever*, transient or persistent; according to Strümpell and Guadard, it is an invariable accompaniment of the convulsions. *Delirium* is a common symptom, as is also *soreness* of the general surface. *Vomiting* and *screaming spells* are also noticed.

The *hemiplegia*, which is noticed as soon as the child recovers consciousness, is usually complete. Less commonly there is first *paresis*, which gradually extends to complete loss of power; and in some instances a total *paralysis* is established after repeated convulsions. The *face* is not always involved, and, as a rule, in facial paralysis of cerebral origin the superior muscles are intact, and the child can close the eyes and elevate the brows. The facial palsy usually disappears rapidly and completely.

As to residual symptoms, the *residual paralysis is most marked* in *the arm*, which is subject to slow wasting, and is commonly useless for the ordinary purposes of life. The *atrophy is moderate*, but there may be arrested development, leaving a wasted and withered member. In extreme cases the arm is held close to the side, the forearm strongly flexed at right angles and in a semiprone position, the hand flexed and the fingers contracted, the palm usually embracing the thumb. Motion may be almost lost in the arm and completely in the fingers, though in most cases there is considerable power of movement, the patient being able to lift the arm above the head, while flexion and extension can be made at the elbow and wrist. The finger and more delicate movements of the hand are rarely recovered. The *leg*, as a rule, *recovers* more *rapidly* and completely than the arm, and the palsy may disappear entirely in it, while it rarely does in the upper extremity. In the leg the wasting is also less pronounced, while arrested development is also less frequent. A persistent halt is apt to remain—indeed, almost always does—as evidence of impaired power; this may consist in simply favoring the affected side, noticeable only on rapid walking. A decided dragging of the limb is, however, nore usual, and there may be tremor of the leg while moving.

The frequency with which *rigidity* is present has given rise to one of the names of the disease, *spastic infantile hemiplegia*. It is not, however, an invariable symptom, and the paralyzed limbs may be relaxed a long time after paralysis sets in. When rigidity is present, it is lessened during sleep, and is increased by emotion and forcible attempts to overcome the spasm. Contracture may ultimately result, after which relaxation is no longer possible. A form of rigidity with partial paralysis is known as *postapoplectic hemi-hypertonia*, and has been previously referred to.

The *reflexes* are almost always *increased* in the affected limbs, ankle clonus being often obtainable in addition to exaggerated knee-jerk. The reflexes may even be increased on the sound side. Rectus clonus and clonus of the flexors of the fingers are rarely present, while in a very few cases the reflexes are absent.

Sensation is rarely affected, but vasomotor derangements are sometimes present. *Electrical reactions* are *normal*, as a rule.

Posthemiplegic *chorea*—hemiataxia—is not infrequent. More uncommon are *mobile spasm* and *athetosis* and posthemiplegic *tremor*. These interesting symptoms were first described by S. Weir Mitchell and Hammond in a study of cases of cerebral palsy.

Aphasia is present in a majority of cases and is almost invariably transitory, is associated most commonly with right hemiplegia, very rarely with left.

Defects of intelligence are very common, the degree of feeble-mindedness ranging from low-grade imbecility to total idiocy. Psychoses may occur late in life, even when there have been no defects in childhood.

Epilepsy is very frequent, and is sometimes confined to the paralyzed side, but also tends to become general. The attacks usually begin within two or three years, sometimes within a few weeks, after the onset of the hemiplegia, but may be delayed from eight to ten years, or even longer. The seizures may present *three well-defined degrees*—the first, in which the child is simply dazed for a moment or two, or longer, without any motor involvement; second, Jacksonian epilepsy, without loss of consciousness, in which the spasms are confined to the affected side, and third, general convulsions, beginning in the paralyzed limbs, and usually accompanied by loss of consciousness. The Jacksonian epilepsy is most common, but all forms may occur in any one case. Jacksonian epilepsy is usually without loss of consciousness, unless the convulsions are very severe or involve a large portion of the body.

Diagnosis.—Infantile *spinal* paralysis (anterior poliomyelitis), most frequently must be excluded, usually without difficulty. The history of the case, including the presence of some of the causes named, the frequent onset with convulsions, the hemiplegia, the absence of rapid wasting of the affected muscles, the retained electrical reactions, are characteristic of infantile cerebral hemiplegia in its early stages; while rigidity of muscles, increased reflexes, the peculiar gait, and residual palsy, with mental impairment and epileptic seizures, distinguish the later stage.

Tumor of the brain sometimes produces similar symptoms. Tuberculosis and glioma are the forms most common in children. *Pressure paralysis* by obstetrical forceps affects the face and upper extremities, but other

symptoms are wanting, and it is scarcely likely to be confounded with infantile hemiplegia.

Prognosis and Treatment.—The prognosis is favorable so far as life and the recovery of considerable locomotive power are concerned; unfavorable as to recovery from mental defect and epilepsy. An institution for feeble-minded children, in which the subjects have the benefit of training and watching, is the safest permanent home for them.

Bilateral Infantile Spastic Hemiplegia.

Synonyms.—*Spastic Rigidity of the New-born* (Little); *Tonic Contraction of Extremities; Essential Contractions; Permanentes Kinder-Tetanus* (Stromeyer); *Spastic Diplegia; Spastic Paralysis of Children* (Adams); *Spastic Diplegia* (Gee); *Spasme Musculaire Idiopathique* (Delpech); *Birth Palsies* (Gowers); *Little's Disease.*

Definition.—Double hemiplegia in infants and children with spastic symptoms.

Etiology.—Most cases of bilateral hemiplegia in children date from birth, and are the result of *injury during birth.* Many are caused by premature birth. The infectious fevers are responsible for a certain number, and a few are direct results of convulsions. In a word, the causes are those of infantile hemiplegia. J. H. W. Rhein[1] reports a case of spastic diplegia following pertussis.

Morbid Anatomy.—As may be inferred from the name, the lesions are bilateral and involve motor areas of the cortex almost solely. They consist in *sclerosis* or *porencephalous* defect, of which the most frequent primary cause is compression by a blood-clot from meningeal hemorrhage from the veins or longitudinal sinus. A meningo-encephalitis may, however, be responsible for the sclerosis. In cases of premature birth there is arrest in the development of the motor system in the brain and cord.

Descending degeneration of the pyramidal tracts or imperfect development of these tracts has been found in a few cases.

Symptoms.—These are to be distinguished from those of the next form, cerebral spastic paraplegia, which the disease closely resembles when the arms are so slightly affected that the palsy is scarcely appreciable. The cerebral spastic paraplegia of childhood is due to lesions similar to those of the bilateral spastic hemiplegia. In the diplegic state *all the extremities* must be more or less *spastic*, although the legs almost always are more so than the arms. These cases are further characterized, as are those of spastic paraplegia, by their occurrence *at* or *very soon after birth.*

There may be *convulsions* or a prolonged succession of convulsions immediately after birth. After this or without it there may be noticed a *limpness* or flaccidity of *muscles*, an expression of *paresis*, often overlooked, because present at a time when the muscular development of the child is so slight that little is expected of it. Soon, however, the *inability to hold up its head* may be observed, and when the time comes for it to walk, it is noticed that

[1] Rhein, J. H. W. "Spastic Diplegia Following Pertussis," "Journal of Amer. Med. Assoc.," March 4, 1905.

the limbs are clumsily used, and when examined, they are found to be *stiff*. As the child grows older it slowly acquires some power so as to be able to sit, but the *legs* are *crossed* and the head is not well supported by the neck muscles. If it is held up, the legs are extended and strongly adducted, and crossed with the feet in the pes equinus or equino-varus position. Occasionally the legs are partially *flexed*, while stiffness varies greatly, involving, in extreme cases, the whole body, sometimes one side more than the other. It is sometimes constant, at other times not. It may be greater on one side than another. The *arms* are usually *stiff* in flexion.

To the spastic symptoms described are added, in certain cases, *spasm* and certain movements known as *athetoid*. In the former, in an attempt at voluntary movement, as taking hold of an object, the fingers are thrown out in a stiff, spasmodic, or irregular manner, or there may be constant irregular movements of arms and shoulders, movements which are usually characterized as choreic. In fact, such cases have been named *chorea spastica*, being differentiated from the congenital choreas by the spastic feature. Spasm rarely affects the muscles of the face, though it does occasionally, causing a continual *grimacing*, which does not always disappear during sleep.

The *athetosis* is double or bilateral, resulting in the most grotesque and distorted movements. They consist in a constant flexion and extension of muscles, more particularly of those of the fingers of one hand and forearm. Flexion of the fingers of one hand may take place, while those of the other may be extending, and the same may be true of different fingers of the same hand. The shoulder and trunk muscles may be also affected, producing a rhythmical and orderly twisting and bending of the body; or those of the neck, producing a turning of the head from side to side. These movements are all increased under excitement or with the effort to do anything.

Mental defect, consisting in imbecility and various grades of idiocy, is more or less characteristic of these cases, but is commonly less than in infantile hemiplegia.

The form resulting from premature birth should be distinguished from that caused by injuries at birth, or by lesions acquired later, as in the former convulsions and athetoid movements do not usually occur, mentality may not be affected, or only slightly so, and improvement may be slowly progressive even after many years.

Infantile Spastic Paraplegia.

Synonyms.—*Paraplegia cerebralis spastica* (Heine); *Tetanoid Pseudoparaplegia* (Seguin); *Spastic Spinal Paralysis* (Erb); *Tabes dorsalis spasmodique* (Charcot).

Definition.—Spastic paralysis of the legs in infants and children.

Etiology.—The causes are those of spastic diplegia and infantile hemiplegia, and also premature birth, the child being born at a period when the central motor tracts are very imperfectly developed; premature birth causes an arrest in the development of these tracts.

Morbid Anatomy.—This is less known than the morbid anatomy of the other forms of cerebral palsy. It may be due to cerebral lesion involving

especially the centers for the lower limbs, to imperfect development of the motor tracts, or to other causes. A few cases with necropsy are recorded.

Symptoms.—These are almost *identical* with those already described as belonging to the *spastic paraplegia of adults*, with which the earlier writers classed it. Spastic paralysis of the lower extremities, dating from birth or appearing within the first few years of life, with talipes equinus or equinovarus, adductor spasm, rigid stiff gait, the patient walking on his toes or by crossed-legged progression—*all without wasting*—these are, in a word, the symptoms. The order of sequence of events is very similar to that described under spastic diplegia. In attempting to walk the heels are everted and knees approximated, because of spasm of the adductors, which may be so strong as to make it impossible to separate the thighs. The spastically extended legs may, however, be gradually forced into flexion after the manner of the "lead-pipe" contraction. If, however, the attempt be made to extend the leg, the spasm returns. If the extension be gradually insisted upon, it often happens that when the extension is nearly complete, the spasm suddenly completes it, as the spring acts on the blade of a pocket-knife, whence the name "clasp-knife" rigidity.

Mental imbecility is not so serious as in spastic diplegia or even as in infantile hemiplegia, and may be entirely absent, especially in those cases resulting from premature birth.

Diagnosis.—The distinction between *spastic diplegia* and *paraplegia* is not a very important one. The two conditions are probably the results of different degrees of similar lesions having different locations. There is an affection of children known as *pseudoparalytic rigidity*, idiopathic contraction with rigidity, or tonic contraction of the extremities, with which it is sometimes confounded, but the following table of differences from Osler's monograph will aid in separating the two conditions.

Pseudoparalytic Rigidity.	Spastic Paralysis; Diplegia and Paraplegia.
Follows a prolonged illness. Is often associated with rickets, laryngismus stridulus, and the so-called hydrocephaloid state.	Usually exists from birth. History of difficult labor [or of premature labor], of asphyxia neonatorum or of convulsions.
Begins in hands and feet as carpopedal spasm; often confined to hands and arms.	Arms rarely involved without legs and not in so marked a degree.
Spasms painful and attemps at extension cause pain.	Usually painless.
Intermittent and of transient duration.	Variable in intensity, but continuous.

The spasm in the pseudo cases is altogether more severe and difficult to overcome. The disease is associated with rickets and other constitutional diseases.

Tetany is characterized by a different history and causation. Bilateral rigidity may also be produced by *tumors* of the pons and cerebellum.

Treatment.—The treatment varies with the stage existing at the time the physician is called. If in the stage of initial convulsion, there the best remedy is chloral, which should be given in doses by mouth or rectum sufficient to control the fits. In the mild degrees, or with a view to keeping up an effect first obtained by chloral, the bromids may be used. If chloral fails, chloroform may be inhaled.

In established paralysis medicines do not avail much and recoveries are rare. Hygiene and good food, gymnastics, manipulation, massage, passive motion, and surgical appliances may be used. Baths and electricity should not be forgotten.

The epileptic convulsions should be treated as when occurring under other conditions, though the cortical lesions occasioning the disease preclude any expectation of permanent relief. Operative procedure has been suggested in certain selected cases and carried out, but with results which have been disappointing.

The mental deficiencies are best treated in an institution for feeble-minded children, where all such cases should be taken, whatever the circumstances of the parents.

Herpes Zoster.

Synonyms.—*Acute hemorrhagic inflammation of the dorsal root ganglia; zona; cingulum; ignis sacer; zoster; shingles.*

Definition.—Herpes zoster, as shown by the studies of Head and Campbell, is an acute inflammation of the posterior ganglia of the spinal nerve-roots associated with extravasations of blood and destruction of the ganglion cells and their axis cylinder processes.

Etiology.—There is usually a specific cause. In some cases there is a prodromal period of illness and neuralgic pain, on the third or fourth day of which the eruption makes its appearance. Atmospheric influences, such as severe cold and dampness, may act as causes. So may checking of profuse perspiration and mechanical violence producing injury to nerves, all favored by lowered resisting power from mental and physical fatigue. Arsenic is occasionally a cause.

Symptoms.—The chief symptom is a painful vesicular eruption, along the course of the afferent spinal nerves, covering their respective skin fields, and is therefore correspondingly irregular. It is, as a rule, confined to one side of the body, though in rare instances, especially about the head, it invades both sides. According to the locality, it is called *zoster capitis*, *z. faciei*, *z. orbicularis*, *z. nuchæ*, *z. brachialis*, *z. pectoralis*, *z. abdominalis*, and *z. femoralis*, and in these different varieties corresponding ganglia are involved; the Gasserian ganglion in zoster of the face, the otic ganglion in *z. orbicularis* which may be complicated with transient paralysis and sometimes severe auditory and visual symptoms. On the head it may invade the scalp as well as the forehead, appearing in the course of the supraorbital nerve and upward over the scalp. The circular involvement of the chest, zoster pectoralis, whence the name herpes zoster, is rarely complete, indeed seldom embraces half the circumference.

Everywhere there is the same severe burning pain in the region of the eruption which is on an angry red surface. While rupture of the vesicles is infrequent, their contents may sometimes become purulent and result in chronic ulceration. There may be an extension of the process from the posterior ganglia to the adjacent meninges, causing pains down the spine, girdle pains, and exaggerated knee-jerks with leukocytosis.

It is met in both sexes, in children as well as adults. In the case of persons past middle life, the disease is often very exhausting, being sometimes the beginning of a decline in health which is permanent. Among the sequelæ is a most inveterate form of neuralgia.

Prognosis.—The prognosis is most favorable in children, and in ordinary circumstances ten days to three weeks suffice to cover its duration, although it may be prolonged much beyond this period. Subjects rarely have more than one attack, although Duhring refers to a case of Kaposi in which nine relapses occurred all on the right side of the body but not in exactly the same region. The disease commonly runs an acute course, terminating spontaneously in recovery except in those rare cases followed by neuralgia.

Treatment.—This appears to be without effect in cutting short the disease and is commonly limited to soothing local applications. The pain is often so severe as to require the hypodermic injection of morphine. Morphine may be employed locally in the shape of a dusting powder. Solutions of carbolic acid 10 to 15 grains to an ounce of water (0.3–0.5 grm. to 30 c.c.) may be used locally, and it is generally comforting to have the parts protected from the atmosphere by a light covering. Dusting with stearate of zinc is a soothing treatment.

MULTIPLE SCLEROSIS OF THE BRAIN AND SPINAL CORD.

Synonyms.—*Insular Sclerosis; Disseminated Nodular Sclerosis; Sclérose en plaques.*

Definition.—A chronic affection of the brain and spinal cord, consisting in the presence of numerous sclerotic patches scattered through the nerve centers, characterized especially by intention tremor, scanning speech, and nystagmus.

Etiology.—Its precise cause is unknown. The infectious diseases, especially scarlet fever, are alleged causes; so are cold, exposure, mental emotion, and syphilis, but without definite foundation. Hereditary predisposition has been noticed. The disease is more common between the ages of 18 and 35, though Strümpell reports a case which came to autopsy at 60. Both sexes are equally subject. Prichard states that more than 50 cases have been reported in children, but it is doubtful whether the diagnosis was invariably correct. It has been thought that the disease depends on anomalies of the vessels, but this view is not held by all.

Morbid Anatomy.—The sclerosed patches are widely scattered through the brain and cord, rarely in the cord alone. They may generally be recognized by their gray color and unnatural firmness. On section, they appear as grayish-red areas. Histologically they consist of thickened neuroglia traversed by a few healthy nerve-fibers. In the vessels there is an increase of the nuclei and, later, a thickening of the walls. Fatty granular cells are present in fresh cases. Many of the axis-cylinders are preserved in the sclerotic patches for quite a long time after destruction of the medullary sheaths. The favorite seats of the plaques in the brain are the centrum ovale, the walls of the lateral ventricles, the corpus callosum, and the cerebellum; while they are quite numerous in the pons, less so in the medulla

oblongata, but numerous in the cord, especially the white substance. The brain cortex is not often invaded.

Symptoms.—By no means can every case of multiple sclerosis be recognized, so often are the symptoms united with those of other lesions whose effects predominate, while the slowness of the onset necessitates delay in the recognition of even typical cases. Typical cases do, however, occur, and they present a set of symptoms whence their recognition is more or less easy.

One of the most important of these symptoms is *tremor*, known as "*intention* tremor," because it is associated with any voluntary effort to perform an act, as picking up an object, raising a glass of water to the lips, or opposing the ends of the fingers of the two hands. This does not prevent the ultimate attainment of purpose. When the patient is quiet, the tremor ceases, and in this respect it can be differentiated from the trembling of paralysis agitans. It is not confined to the arms, but occurs also in the head and trunk, so that the head trembles when it is raised from the pillow. It is increased by excitement.

Another characteristic symptom is what is known as *scanning speech*, a slow, measured, yet indistinct and obscure utterance, depending upon disturbances in the innervation of the tongue and larynx, probably caused by sclerotic patches in the pons and medulla oblongata. There may be tremor in the tongue and lips when speaking. The third symptom is *nystagmus*—oscillatory or lateral movements of the eyeballs when the eyes are directed to an object. In addition there may be *spastic symptoms* manifested chiefly in the presence of *increased reflexes*—including periosteal as well as tendon reflexes—in both upper and lower extremities, but the skin reflexes remain normal. There is ankle clonus, and the gait is often spastic. *Paresis*, at first absent, ultimately appears, amounting at times to complete *paralysis*. Indeed, spastic rigidity and paresis may be among the earliest signs of the disease. The sphincters remain intact, at least until toward the close. There are *no disturbances of sensibility* in the majority of cases. *Optic atrophy* is present in 40 per cent. of cases and in 15 per cent. in tabes dorsalis, and is associated with such *derangements of vision* as amblyopia, achromatopia, and even blindness. *Optic neuritis* may occur with subsequent atrophy, especially in the temporal halves of the optic nerve. There may be also derangements of innervation with diplopia.

Mental weakness and *imbecility* are sometimes present, more rarely *melancholia or exaltation*. *Apoplectiform* attacks also occur, following prodromal symptoms, such as *vertigo* and *headache*, and succeeded by hemiplegia, which, however, subsequently disappears.

Diagnosis.—This is not difficult in typical cases. The intention tremor, the scanning speech, and nystagmus are characteristic, and when associated with spastic weakness, the diagnosis of multiple sclerosis is probably correct. The apoplectiform seizures and mental weakness are also valuable signs. When the symptoms are mixed with those of other nervous lesions, diagnosis is not so easy. In *paralysis agitans* tremor occurs during rest as well as in motion; in multiple sclerosis only when motion is attempted. Strümpell says: "The circumstance, indeed, that the anomalous cases will not properly fit the molds of any other form of disease should make us think of the possibility of multiple sclerosis."

The disease known as *pseudo sclérose en plaques*, described by Westphal, seems to have most of the symptoms of multiple sclerosis except nystagmus. The tremor movements are said to be more violent. Strümpell has found slight degeneration of the pyramidal tracts in a few cases of this kind. It is probably a diffuse sclerosis which is not always easy to detect with the microscope, but may be sufficient to cause a peculiar hardness of the brain and cord before they have been put in any hardening fluid.

Prognosis.—This is unfavorable after a long and tedious course, terminating in the bedridden state.

Treatment.—This is unavailing. The end possibly may be delayed by galvanism and tepid bathing.

PARETIC DEMENTIA.

SYNONYMS.—*Chronic Diffuse Meningo-encephalitis; Dementia Paralytica; General Paresis; Progressive General Paralysis of the Insane.*

Definition.—A chronic progressive meningo-encephalitis, or meningo-rachitis, with resulting mental and motor derangements, terminating in dementia and paralysis.

Etiology.—At least 75 per cent. of all cases are caused by syphilitic infection, and observations reported by Krafft-Ebing seem to indicate that the proportion is much greater. Starting out with this assumption, we have at once an explanation of its greater frequency in the male sex, though many women have it; while it is rather a sad commentary on the fidelity of man that it is much more frequent among married men. It is possible, however, that syphilis was contracted before marriage, as the development of paretic dementia may be delayed for a number of years after a syphilitic infection. The fact that it occurs most frequently between the thirtieth and fiftieth years, that it is a disease of the better classes—especially army officers and artists—and that it is pre-eminently a disease of the cities, should be added. Although other factors apparently enter into the causation of general paresis, those who have most closely studied the subject are disposed to assign to them a predisposing rôle. Such influences are heredity and exhausting mental work, such as comes of public political life and ambitious financial ventures. Intemperance, chronic lead-poisoning, and traumatism are included among causes.

Morbid Anatomy.—An *atrophy* of the brain, and especially of the frontal lobes, may be set down as the most important morbid change. The convolutions are wasted and pale in color, the fissures are wider, and the weight of this portion is reduced to one-fourth or one-third the normal, while the consistence is firmer and more resisting to section. Other macroscopic changes are a *thickening of the dura mater, pachymeningitis interna, edema* of the pia with thickening, opacity, and adhesion to the cortex. Minute examination of the cortex recognizes *thickening of the vessel-walls* and cellular infiltration of the adventitia of the arterioles and lymphatic sheaths—in other words, the effects of mild inflammation. To these are added demonstrable *destruction of nerve elements*, especially of the fine medullary nerve fibrils known as "tangential fibers" in the frontal convolutions, island of Reil, and elsewhere; also *atrophy* of the *ganglion cells*. Associated with this are neuroglia proliferation and numerous Deiters' spider cells. Here

enters a contested question as to whether the nerve changes are primary, or secondary to an interstitial encephalitis. Tuczek, Wernicke, and Strümpell hold to the former view; while Rindfleisch and Mendel adopt the latter, making the destruction of nervous tissue secondary to the overgrowth of neuroglia.

The white matter is also involved, the central ganglia as well. Coincident *changes in the spinal cord*—first described by Westphal—consisting in fascicular systemic degeneration of the *lateral columns* and *posterior columns*, either alone or jointly, are quite constantly present. To these is ascribed a large part of the ataxic and spasto-paralytic symptoms. From this brief statement of the character and situation of the morbid changes it will be seen that they are widespread, while they are also degenerative.

Symptoms.—So widely scattered a distribution of morbid changes naturally brings about corresponding differences in the variety and severity of the symptoms. As further characteristic, no absolute constancy is observed in the order of their development. As a rule, however, the *first stage* is characterized by abnormal mental processes, and these are at first what may be comprehended under the single expression *peculiarity* or "*queerness*" *of conduct*. The patient will perform acts wholly unnatural to him, and will surprise his friends and family by breaches of decorum and morality. An *apathy* and *loss of memory*, causing the omission of obligations, are also constant. At first these may pass unnoticed as temporary, but their permanence is gradually established. In lieu of this may be present an *irritability* and intense *restlessness*, so that the patient cannot remain in one spot, but walks constantly to and fro. Not often in this stage is there much volubility, but rather a *morose silence* is observed. In this stage, too, the patient may make rash and ruinous financial ventures, and lose his own money and that of his friends, or he may become very generous, giving away freely all he possesses, and more, too. The power of arithmetical calculation is defective or gone. He may be self-satisfied and intensely egotistical. On the other hand, he may be conscious of these ills and be anxious about them, as well as experience a discomfort or malaise, for which he may consult the physician.

Nor are *motor disturbances* wholly wanting in the first stage. They are chiefly *derangements of speech and handwriting*, and are of no small diagnostic value. The speech is slow and hesitating, yet the patient stumbles over syllables, especially when the word is complex or rather difficult to enunciate. As to the handwriting, it is tremulous, characterized by the omission of letters and substitution of wrong ones, as well as erroneous spelling—all motor defects.

Other symptoms of the first stage are *inequality of the pupils, ocular paralysis*, in tabetic cases often reflex immobility of the pupils. There may be absence of the patellar reflexes, and in spastic cases increase of reflexes. There may be *neuralgic pain* and attacks of *migraine*.

The *second stage* is characterized by more *exalted mental symptoms* and excitement, with a *higher degree of motor disturbance*. The former consist in exaggeration of all previously maintained mental symptoms, amounting to noisy, boisterous, and maniacal excitement, and even uncontrollable violence. In this stage belong, too, those extraordinary delusions of

grandeur—expansive delirium—in which the patient imagines himself or herself to be a person of great consequence and unlimited wealth. This is not, however, invariable, and there may be an exaggerated degree of the opposite condition of melancholy sometimes present in the first stage, or the two conditions of delirium and depression may alternate or may be absent. *Sleeplessness* may be added to restlessness and mental excitement, causing rapid decline of strength.

Motor disturbances are greatly increased in this stage, but a uniform order of invasion is by no means always observed, while remissions and temporary improvement are often noticed. *Speech* becomes almost impossible and incomprehensible. There is paraphasia—persistent repetition of words—and reading and writing are impossible. The voice can no longer be modulated, and is weak and rough from imperfect innervation of the vocal cords.

The *gait* becomes defective, and the patient often trips in walking. There may be *ataxia* and other tabetic symptoms; apoplectic seizures with *paralysis;* or *epilepsy* with grand or petit mal and aura, sometimes one-sided and followed by monoplegia or hemiplegia. There may be loss of *sensibility* with *bladder* and *rectum paralysis*. The *tendon reflexes* may be lost and the pupil be immobile, or the opposite condition of spasm with increased tendon reflexes prevails. The paralytic attacks may occur in the earlier stages, though in mild degree, manifested by vertigo or obscuration and loss of consciousness, lasting for a short time and then passing away. There may be local twitching in the face and extremities and even typical Jacksonian epilepsy. Finally, *bulbar* symptoms may appear with invasion of the medulla oblongata. Ultimately, the patient becomes helpless, bedridden, and completely demented, dying from exhaustion or intercurrent disease. In a few cases none of the mental symptoms described are present, but a gradual decline of mental power takes place until complete dementia supervenes.

An *acute variety* is also sometimes met, properly termed "galloping," in which the disease runs its whole course in a few months, and is especially characterized by emaciation and rapid loss of strength due to restlessness, sleeplessness, and insufficient food. The *pulse* and *temperature* are essentially normal, or at least there are not characteristic variations.

Diagnosis.—To recognize paretic dementia *ab initio* is perhaps impossible, but to watchful observation the disease commonly reveals itself after the symptoms have existed for a short time. The early symptoms resemble those of *neurasthenia*, but differ from those of the latter disease in their steady progression. Other affections possibly mistaken for it are *cerebral syphilis*, *tumors of the brain*, and *multiple sclerosis*. In *cerebral syphilis* the onset is usually more sudden, and paralytic symptoms appear earlier. Headache is more frequent and severe, and there may be convulsive seizures; affections of the tongue and speech are wanting, while the train of mental symptoms is less complete and characteristic, and expansive delirium, as a rule, does not occur. The epilepsy is more commonly Jacksonian. It is to be remembered that the syphilitic virus produces both, and it is not unnatural that the two should sometimes merge. *Tumors of the brain* frequently, but not always, produce symptoms more localized, and often also

optic symptoms, including choked disk. The symptoms of *insular sclerosis*, which include dementia, are often identical with those of paralytic dementia, and the two diseases cannot then be differentiated. Intention tremor is more characteristic of sclerosis. The cerebral symptoms of some forms of *plumbism*, it is said, also sometimes closely resemble those of paralytic dementia.

Prognosis.—The prognosis is almost always unfavorable, although the course of the disease varies somewhat. The most rapid cases of the galloping form may terminate in a few months, but two or three years is the more usual duration; sometimes much longer, it may be 10 years or more. Death ensues from exhaustion, hastened by the complications and secondary conditions which naturally supervene on an illness so prolonged and in which nutrition is so interfered with; or it may be due to intercurrent disease.

Treatment.—In view of the general acknowledgment of the syphilitic origin of chronic diffuse meningo-encephalitis, antisyphilitic treatment has been employed but with little success, as it cannot restore degenerated tissue and probably cannot arrest active degenerations. The treatment is confined mainly to iodids, mercurials and salvarsan. Mercurials are best used by inunction or hypodermatic injections, and the iodids in ascending doses.

As to the rest, treatment must be symptomatic. The bromids and chloral, with quiet, hygienic surroundings, and sometimes enforced retirement, are measures demanded for the relief of the nervous excitement.

For the opposite condition of depression and melancholia change of scene by travel and residence in different localities should be enjoined. Further than this the use of a proper hygiene, with bathing, frictions, wholesome outdoor life, and an abundance of nourishing and easily assimilable food constitute about the sum of the means we can bring to bear against the disease.

PARALYSIS AGITANS.

SYNONYMS.—*Chorea scelotyrbe sive festinans* (Sauvages); *Chorea procursiva* (Bernt); *Shaking Palsy; Parkinson's Disease.*

Definition.—A chronic nervous disease characterized by muscular weakness, tremor, or shaking in the extremities, muscular rigidity, and forward-bent gait.

Etiology.—Shaking palsy is commonly a disease of the second half of life, but occasionally occurs between 30 and 40, and has been observed as early as the twentieth year. It is a little less frequent among women than men—11 to 14. Among the causes held responsible for it are fright, mental excitement, business worry, injury—whether to nerves or other parts of the body—alcoholism, sexual excesses, and the infectious diseases, including malaria, while heredity is said to have a slight influence. The etiology of the disease is largely a matter of conjecture and inference.

Morbid Anatomy.—This is unknown so far as essential lesions are concerned. Various lesions have been described, while the brain, spinal cord, and peripheral nerves of the most typical cases have been examined with results not entirely satisfactory.

As the phenomena are similar in kind, if not in degree, to those of senility,

it is held by Dubief, Borgherini, Koller, Sass, Jacobson, Ketscher, and Sanders that they have for their anatomical basis the lesions of senility somewhat intensified, and that the disease differs from true senility only in its earlier onset. Other investigators conclude that this is not the case, and that paralysis agitans is a disease *sui generis*, although there are many changes in the spinal cord and brain, which are common to the two affections, consisting essentially in increase in interstitial tissue and proliferation of neuroglia cells in the spinal cord, medulla oblongata, pons, and the motor cortex in a less degree. Some investigators attribute paralysis agitans to abnormal conditions of the ductless glands, but, the pathology of this disease is unknown, and is at present a subject for speculation.

Symptoms.—The disease is not a very rare one in this country, and the county almshouses almost always contain one or more cases—easily recognized by the characteristic shaking or tremulousness of the hand. Though commonly gradual in onset, the symptoms may come on quite suddenly, and at first only after exertion. Indeed, there may even be a *prodrome* in the shape of neuralgic pains, paresthesia, dizziness, and the like. The more sudden cases follow fright or trauma. The *tremor* is most marked in the fingers and hands, where it commonly begins, and whence it extends to the arms and lower extremities. The upper arm muscles are rarely involved. It most frequently passes from the right arm to the right leg, thence into the left arm, and thence into the left leg; or the course may be crossed—that is, from the right arm to the left leg. It may remain in one limb to the exclusion of the others. In the fingers the movements between the thumb and index-finger is frequently that of rolling pills, but the movement may not always be characteristic. At the wrist it is one of pronation and supination. In the feet it is most marked at the ankle-joint. It affects the writing, making it trembling, as in the aged, and ultimately it becomes impossible to write. The muscles of the head and face are last involved, sometimes not at all, and when present, the motion is vertical and quite rhythmical, usually about five times in a second. At first the tremor ceases during sleep, but continues during the waking state even when the muscles are at rest, but ultimately it continues even during sleep—in fact, sleep is sometimes prevented thereby. It frequently is partially arrested by voluntary motion and is increased by emotion. Should rigidity become excessive, the motion may cease.

The rate of tremor varies greatly, being at first slower, and increases in rapidity as the disease advances. Roughly, it may be put down at from three to five times a second. There may be intermissions of the tremor of days and even weeks.

Muscular weakness is a less striking symptom, but may be estimated by the dynamometer, and increases with the duration of the disease and the intensity of the tremor. It is most striking at least in the extensor muscles, the *flexors* being disposed to *rigidity* and *spasm*, which early produce a slowness and stiffness of motion which is characteristic. It is this flexor spasm which brings the thumb and forefinger into the writing or pill-rolling position. At other times, hyperaction of the interossei muscles over that of the common extensors of the fingers results in the position so characteristic of arthritis deformans—that is, with the first phalanx bent, the second extended, and the terminal phalanx also bent. Ultimately extension is impossible. Occasionally the opposite state of fixed extension exists.

The *attitude* and *gait* ultimately assumed by the subject of shaking palsy are also the result of rigidity, which sooner or later affects most of the muscles. The head is bent forward, the back is bowed, the arms are held away from the body and flexed at the elbows, and the knees are approximated so that they are often rubbed in walking; while the general appearance is that of a man in danger of falling forward. The position of the body due to flexion also gives rise to a "propulsive" gait, caused by carrying forward the center of gravity, so that, when started, the patient is likely to "get a-going" and cannot stop until he comes up against some object. On the other hand, a push backward, bringing the center of gravity behind the point of support, is apt to make the patient fall, because be cannot move back fast enough to save himself by "retropulsion." Charcot regards both these phenomena as "forced movements," but Strümpell prefers to explain them by simple physical laws, as previously described. Sometimes the characteristic position of the patient exists without the shaking, and for this the name "*paralysis agitans sine agitatione*" has been employed.

The similarity in the rigid bent condition of the vertebral column and the deformities of the hands in this disease and rheumatoid arthritis, together with other points of resemblance has suggested to W. G. Spiller a similar origin of the two diseases.

The *facial expression* is also very strikingly altered. The face is indeed without expression, stiff and mask-like, giving rise to the name "Parkinson's mask." There is often a dribbling of saliva from the partially closed mouth. On the other hand, sometimes the mouth is kept closed, and is found full of saliva—a condition ascribed to delayed deglutition rather than to increased secretion. The speech is slow, hesitating, and monotonous, and the voice may be piping and shrill. On the other hand, if the lips and tongue share in the tremor, the speech is stuttering, as though the patient were in a hurry to speak—quite different from the scanning speech of insular sclerosis.

The remaining nervous and organic functions are essentially normal. Sensation is usually unaltered, and the bowels and bladder are usually unaffected, as is also the temperature, although it is said that the surface temperature is sometimes elevated. Charcot has noticed an alteration of the temperature sense. There is sometimes a tendency to unnatural perspiration.

Diagnosis.—This is usually very easy, and can generally be made at a glance. *Multiple sclerosis* resembles it in some respects. Both have tremor, but in multiple sclerosis this is shown more particularly when the patient attempts to do something, as to bring a glass of water to his lips or approximate his fingers. The speech is rhythmical, "scanning," instead of stuttering, as in shaking palsy; there is nystagmus, and the disease begins almost invariably in the lower extremities, while the attitude is not that of paralysis agitans. *Chorea* is characterized by movements, but these are irregular and more intermittent.

Prognosis.—A well-established case of paralysis agitans is not curable by medicines. On the other hand, the disease lasts indefinitely, the patient getting slowly worse, with perhaps the intermissions alluded to, until he dies of some intercurrent disease or from the effects of some accident growing out of his condition.

Treatment.—Under the circumstances this must, for the most part, be by tonics and general hygienic measures. As the disease advances the patient should be guarded against accident; and especially when in bed his position should be changed for him if he cannot change it himself, as is often the case.

Cases have improved under the use of the iodid of potassium and arsenic, and hyoscin has been especially recommended by Erb—hypodermically, in doses of from 1/150 to 1/100 of a grain (0.00044 to 0.00066 gm.) of the hydrobromate, but it may be given by the mouth. Good results have also been reported from the use of atropin, of which from 1/100 to 1/60 grain (0.00066 to 0.0011 gm.) may be used subcutaneously or by the mouth.

Measures calculated to improve the general health are indicated, such as sea-bathing, massage, electricity, fresh air, and outdoor life.

Other Forms of Tremor.

Synonym.—*Ballismus.*

In addition to the tremor in paralysis agitans, a similar tremor occurs under other circumstances, sometimes without assignable cause, when it is known as *simple* tremor, or it may be induced by fright or overexertion. A *hereditary* tremor has been described by C. L. Dana. *Senile* tremor is the well-known form of tremor which comes on with advancing years, at times earlier than others, but usually not until after 70 years. The existence of a tremor due to senility was denied by Charcot, but is accepted by most neurologists.

Toxic tremor is due to a number of toxic agents, among which tobacco and alcohol are the most frequent. Lead and excessive drinking of tea or coffe are other causes. Finally, *hysterical* tremor occurs as a part of hysterical phenomena in women. *Asthenic* tremor is due to simple weakness, and is especially seen in exertion during convalescence from acute disease.

TUMORS OF THE BRAIN.

Synonyms.—*Neoplasmata cerebri; Intracranial Tumors.*

Definition.—Cerebral tumors, clinically considered, include not only tumors of the meninges and substance of the brain, but also all intracranial and even such extracranial tumors as ultimately invade the brain. Among the latter are tumors of the orbit or nasal cavity, of the antrum, and of the sphenopalatine fossa.

Varieties.—The principal varieties of cerebral tumor, approximately in the order of frequency, are:

1. Glioma. 2. Tyroma, or tuberculous tumor. 3. Sarcoma. 4. Carcinoma. 5. Cystic, including parasitic cysts and cysts arising in sarcomata and gliomata. 6. Gumma. 7. Histioid tumors. Among these occur in irregular order, cholesteatoma, lipoma, myxoma, angioma, fibroma, psammoma. Even dermoid cysts, as well as parasitic cysts—including the echinococcus or hyatid cyst and the cysticercus cellulosæ, are met. Of these tumors, psammoma and glioma are peculiar to the brain. According

to M. Allen Starr's tables, gliomata and gliosarcomata practically equal in number the sarcomata, but the term gliosarcoma is regarded by many unfavorably.

Etiology.—Tubercle is more common in childhood; parasites, glioma, sarcoma, and gumma in early and middle life, and cancer in middle and late life, but is rare even then. Brain tumors of any kind are rare after 60. Heredity appears to have slight, if any, influence. A few brain tumors are metastatic, especially carcinoma, and to a less degree sarcoma. Eichhorst relates several remarkable cases in which trauma seemed to be the exciting cause.

Distribution.—Certain tumors seek by preference special localities. Thus, *tuberculous* tumors are most numerous in the cerebellum and about the base of the brain. *Glioma* starts from the neuroglia in any part of the brain, but more frequently the cerebrum or pons, and may also attain a large size —larger than any other brain tumor; it is further characterized at times by its great vascularity, leading sometimes to rupture and apoplectic symptoms. Glioma may also occur in the eye. *Sarcoma* develops most frequently in the membranes of the brain and sheaths of the vessels; it may be primary or secondary; it is often encapsulated and often appears as endothelioma. *Myxoma* and *fibroma* occur in the same localities. *Carcinoma* is usually secondary, but may be primary; it arises more frequently in the membranes or pituitary body, but may be found in the substance of the hemispheres; it is especially secondary to primary cancer of the breast, lungs, or pleura. *Syphiloma* elects the cerebral hemispheres or the pons and vicinity; it is generally superficial, grows from the meninges, or is attached to arteries, attaining sometimes a large size. It may be multiple. *Parasitic tumors* are found in the membranes, the substance of the brain, and the ventricles. The hydatid cysts developed by the echinococcus are usually on the surface of the brain; the cysticercus, usually multiple, on the surface or in the ventricles. *Psammoma*, or sand tumor, is found commonly in the neighborhood of the pineal gland.

Symptoms.—The symptoms of cerebral tumor are in no way specialized by the kind of tumor present, and depend entirely upon the effect exerted on the surrounding brain substance, chiefly by pressure. They do, however, vary somewhat with the part of the brain involved. It occasionally happens that a brain tumor may produce no symptoms whatever, being thoroughly latent, and disclosed only by the autopsy. On the other hand, apparently insignificant tumors cause very decided symptoms. Such differences may depend in part on the location of the tumor, and in part on the rapidity of its development.

As in all local diseases of the brain, two sets of symptoms usually present themselves: (1) Diffuse and (2) focal symptoms.

1. *Diffuse or General Symptoms.*—These are symptoms which may be associated with various forms of nervous disease. The most constant of these is perhaps *headache*, which varies in intensity and constancy. Probably the severest headaches human beings suffer are caused by brain tumors, exhibiting every variety of pain—sharp, cutting, shooting, boring, or dull and pressing. At times it is moderate, producing a sense of discomfort only. It may be intermittent or constant. It may be over the entire head, or

half of it, or be still more localized in the forehead or back of the head, extending also from the former over the face and the latter down the neck. It may be increased by mental excitement of any kind, by noise, or by alcoholic drink or strong light. There may be tenderness on pressure, or pain in percussing the head. The seat of pain is, however, for the most part, no indication of the seat of the tumor, though the presence of pain limited to the occiput and back of the neck suggests a tumor in the posterior fossa of the skull, the occiput, or the cerebellum. Localized pain on tapping the skull is a more reliable index.

Astereognosis is an occasional symptom.

Vomiting is another characteristic symptom of brain tumor. It may occur independent of headache, but is often associated with it. It is further characterized by being independent of food ingestion, may be without nausea, and is likely to be worse in tumors of the cerebellum and pons.

Dizziness is also a very frequent symptom, and often an early one. It is at times intermittent, at others constant, and it may be so severe as to make it impossible for the patient to walk. It is most serious in tumors of the posterior fossa and of the cerebellum. Along with vertigo may be *slowing of the pulse.*

Mental symptoms may be present. They may be intermittent, and variously manifested in peculiarities of temper, such as sullenness, indifference, absent-mindedness, and loss of memory; or the opposite condition of maniacal excitement or delirium; or there may be drowsiness and even coma. Such mental states may, indeed, be the only manifestations of tumor.

Speech.—The patient may *talk slowly*, and the facial expression is sometimes altered.

Apoplectic seizures and *epileptiform attacks*, especially of the Jacksonian variety, are distinctive symptoms. The former may be due to hemorrhages in the tumor or around it, and may be followed by transitory paralysis and paresis. Epileptic convulsions, especially if unilateral, point, though not unmistakably, to tumors in the cerebral hemispheres impinging on the cortex. *Choreiform movements* are sometimes present.

Choked disk, *papilloedema* or *papillitis* and *optic neuritis* are the most constant and most valuable diagnostic symptoms of brain tumor. Choked disk consists, in brief, in a swelling of the optic nerve, with overdistension and congestion of the retinal veins, and narrowing of the retinal arteries. It is usually bilateral, rarely unilateral. There is still much difference of opinion as to the mechanism of choked disk, but it is thought by many to be the result of intracranial pressure forcing the cerebrospinal fluid from the subarachnoid space into the lymph sheath of the optic nerve, causing compression of the nerve and the vessels within it. The vision is not necessarily deranged in choked disk, and its defects are not uniform, varying from slight amblyopia to total blindness. The swelling may diminish and improvement in vision ensue, but retinitis or neuro-retinitis may set in with consequent nerve atrophy, producing permanent impairment of vision. The choked disk is sometimes the only symptom of brain tumor, and its subject first consults the oculist for relief. On the other hand, it is not caused by brain tumor alone, but it may result from meningitis or abscess, in fact anything which produces intracranial pressure. Papilloedema occurs

in from 80 to 90 per cent. of all cases of intracranial tumor. It may be absent, even though a brain tumor of considerable size exists. By optic neuritis a milder degree of papilloedema is generally understood.

The *senses of smell and hearing* may be impaired by tumors impinging on the olfactory or auditory nerves, and there may be modifications of cutaneous sensibility; also *neuralgic pains*. If the tumor is on the floor of the fourth ventricle, there may be *polyuria* and *glycosuria*. Finally, sooner or later the *appetite* may fail and the *nutrition* suffer, although the opposite condition of large appetite and good nutrition may obtain. In the terminal stage there may be *irregularity* of *breathing* (Cheyne-Stokes) and slowing of the pulse, while the final issue is often preceded by a *febrile movement*. The pulse rate falls with increasing intracranial pressure often as low as 48 and less. It is not permanent and may alternate with frequent pulse.

2. *Focal Symptoms.*—These are symptoms peculiar to the seat of irritation or destruction, and become, therefore, of value in diagnosis. They are the results either of irritation or destruction of nervous tissue, irritation causing contraction or spasm, while destruction causes paresis or paralysis.

Tumors of the prefrontal area, especially on the right side, often give no localizing symptoms whatever, motor or sensory, while general symptoms may also be absent and the tumor truly latent. Then, again, general symptoms may be well marked, including mental torpor and imbecility, childishness, irritability, and emotional phenomena. These symptoms occur whichever side of the brain is affected, but possibly are more pronounced in tumors of the left frontal lobe. If the tumor extends downward into the inferior frontal convolution, it may cause aphasia; or if backward, it may occasion irritative spasm or destructive paralysis. Involvement of the optic tract may cause hemianopsia and optic neuritis; of the olfactory system, anosmia; if the tumor invades the orbit, oculomotor paralysis and protrusion of the eye. Percussion tenderness may aid in localizing the tumor.

Tumors in or *near the central* or *motor region* (possibly true only of the precentral convolution) may cause irritative lesions, resulting in spasm. If the tumor is in or near the upper third of this area, the spasm may begin in the toes, in the ankles, or in muscles of the leg; if in or near the middle third, spasm beginning in the fingers, in the thumb, in the muscles of the wrist or shoulder; if in or near the lower third, in the muscles of the face, the angle of the mouth, or tongue. In a word, the phenomena of Jacksonian epilepsy are present. All of these may be preceded or associated with sensory disturbance, such as numbness and tingling, and may be limited to one muscle group before extending to another, constituting the "signal symptom" of Seguin. There may be an aura, and the muscular sense is also sometimes affected.

Destructive lesions cause paralysis, and this may have the same distribution as the convulsions which sometimes precede. If on the left side in right-handed persons, aphasia and agraphia may result.

3. *Tumors of the parietal area* may produce no symptoms of sensory or motor phenomena, but there may be impairment of stereognostic perception, and often of ordinary sensation and of the sense of position with ataxia. With the involvement of the angular gyrus and lower parietal lobule may

come word-blindness and mind-blindness. If the tumor is upon or near the central area, spasms and paralysis of the various muscular groups described under 2 may develop. Paralysis of the third nerve has occurred in connection with tumors in the neighborhood of the angular gyrus; no satisfactory explanation for this has been offered—possibly it is due to pressure at a distance.

4. *Tumors of the occipital lobe*, if in the cuneus or neighboring parts, may produce homonymous lateral hemianopia; and if double, total blindness; if elsewhere on the left side, there may be mind-blindness; and if the tumor extends also into the angular gyrus, word-blindness, along with hemianopsia; if obtruding further forward into the parietal lobe, hemianesthesia, hemiataxia, and perhaps some hemiplegia from involvement of the internal capsule may occur.

5. *Tumors of the temporosphenoidal area on the right side* rarely produce symptoms; on the left side, in the posterior part of the first and upper posterior part of the second gyrus, they cause word-deafness. Disturbances of the senses of smell and taste may result from involvement of the hippocampal convolution.

6. *Tumors of the pons and medulla oblongata* produce two sets of phenomena by:

(*a*) Irritation or destruction of fibers in the pons and medulla oblongata.

(*b*) Pressure on the nerves emerging in this region.

Either may occur alone or both jointly. Lesions here are especially likely to produce alternate paralysis: that is, involvement of certain of the cranial nerves on one side and the limbs on the opposite side.

If the tumor is in the cerebral peduncle, there may be a palsy of the third nerve on the same side and a hemiplegia on the opposite side; if lower down and in the pons, a palsy of the fifth on the same side and hemiplegia on the other; if still lower down, it may involve the sixth nerve, producing internal strabismus, the seventh producing facial paralysis, and the eighth causing deafness. If the tumor is very large, it may produce a hemianesthesia as well, and there may be forced movements of the body, either toward or from the side of lesion. Conjugate deviation of the eyes away from the side affected may also occur. This is in direct contrast to the conjugate deviation sometimes noticed in cerebral lesions, in which the head and eyes are turned toward the side of the lesion.

Tumors of the medulla oblongata may produce hemiplegia and hemianesthesia, and, if the tumor is large, symptoms of bulbar paralysis. From irritation of nerves on the same side, the ninth, tenth, eleventh, and twelfth, difficulty in swallowing, irregular action of the heart, irregular breathing, and vomiting may arise. Sometimes also there is retraction of the head, or sensory symptoms including numbness and tingling and finally convulsions. If the cerebellum is impinged upon, there may be unsteadiness of gait, but this is frequently caused by implication of the cerebellar peduncles without involvement of the cerebellum.

7. *Tumors of the cerebellum* produce very characteristic symptoms, though here, too, there may be latency if the growth is limited to the hemispheres. If the middle lobe is invaded, vertigo, vomiting, headache, papilloedema, with blindness and cerebellar ataxia, are present. *Papillo-*

edema is more common in cerebellar than in cerebral tumors and is usually an early sign. The pressure causing papilloedema is not directly on the occipital lobe or optic tract, but is generally on the cranial contents, and possibly interference with the circulation of fluid in the ventricles causes pressure on the optic chiasm by an excess of fluid in the third ventricle. More rarely nystagmus and neuralgic pains in the neck and occiput occur. The irregular and staggering gait of cerebellar ataxia is very striking, the patient reeling like a drunken man, or he may be thrown sideways or forward, rarely backward, by forced motion.

If the medulla oblongata is compressed by the tumor, vomiting from this cause may ensue, also bulbar symptoms and glycosuria.

8. *Tumors of the corpus callosum* are rare. The symptoms are similar to those of tumors in the third and lateral ventricles of the brain, extending peripherally. They cause general symptoms of brain tumor, with gradually developing hemiplegia, and later paraplegia. With this there are mental dullness and drowsiness and indisposition to speak. The cranial nerves are not involved.

9. *Tumors of the basal ganglia and the internal capsule* produce symptoms similar to those that occur in the corpus callosum. They are partly pressure symptoms. There is progressive hemiplegia, with which there is likely to be hemianesthesia. Sometimes there are choreic and athetoid movements if the tumor involves the optic thalamus and adjacent parts of the internal capsule. Tumors of the caudate nucleus alone, or of the lenticular nucleus alone, are generally latent or not recognizable; so are those of the anterior three-fourths of the optic thalamus, except that choreic and athetoid movements referred to may be noticed, due to irritation of fibers of the internal capsule, or, as supposed by some, to irritation of the anterior cerebellar peduncle. Tumors of the basal ganglia are often infiltrative (glioma) and therefore cause few symptoms at first. Tumors in these areas are very likely to give pressure symptoms. A large tumor of the thalamus may involve the fibers of the optic radiation and cause hemianopia or sometimes hemianesthesia. This may be differentiated from hemianopia due to lesions of the occipital lobe by the presence of the hemianopic pupillary reaction, in accordance with which a ray of light thrown on the insensitive part of the retina will not produce a reflex contraction of the pupil. Papilloedema is likely to be an early symptom of tumors in this vicinity.

10. *Tumors of the corpora quadrigemina* usually involve the crura as well. They are characterized by inco-ordination, forced movements, and oculomotor palsies, to which may be added hemianopia, or blindness due to destruction of the primary optic centers; the pupillary reflex is lost and there is nystagmus.

11. *Tumors of the crus* from involvement of the third nerve are especially characterized by oculomotor paralysis on one (the same) side and hemiplegia on the other. Tumors of the crus are, however, rare.

12. *Tumors of the base*, if of the anterior fossa, produce symptoms much like those of tumors of the prefrontal area, adding, however, anosmia from destruction of the olfactory lobe; while there may be also involvement of the optic and oculomotor nerves and of the orbital contents. Tumors of the middle fossa and of the interpeduncular space produce pressure on the optic

chiasm with consequent neuritis and bitemporal hemianopsia, by which lesions of this area are distinguished from those in the anterior fossa.

Diagnosis.—This consists first in the recognition of the presence of tumor from the general symptoms, and then the determination of its location in either hemisphere from the focal symptoms. The same symptoms may be produced by any agency causing pressure on these structures. *Papilloedema*, which is so constant a symptom of tumor, may be caused by *Bright's disease*, *cerebral syphilis*, *lead encephalopathy*, and *anemia*. The albuminuria, hypertrophy of the right ventricle, polyuria, and tube-casts usually help to recognize the first. Other symptoms of lead-poisoning indicate that disease, and the usual symptoms of anemia point to it. *Meningeal thickening*, *hemorrhage*, *aneurysm*, and *abscess* may also produce pressure symptoms.

The nature of the tumor may be determined in part by what has been said of the preference for certain localities and the age of the patient, and in part by the history, say of tuberculosis or syphilis or primary growths elsewhere. The surface temperature is of uncertain value in diagnosis. Death may be sudden, especially from growths near the medulla oblongata. It is usually the result of increasing pressure. The X-ray has recently been applied to the diagnosis of brain tumor with uncertain results in most cases; a change in the percussion note over a tumor is also of doubtful value.

Prognosis.—This is generally unfavorable. It is true that in some rare instances the brain tumors cease to grow after a time. Various observers find the ratio of removable tumors from 5 to 10 per cent. Of 1121 cases collected from different authors by M. Allen Starr in his article on "Tumor of the Brain" in Dercum's "Nervous Diseases," 80, or 4.25 per cent., were regarded as operable, but four-fifths of all persons operated on perish. Calcification is a rare, but happy, termination of tuberculous growths. The duration of tumor averages two or three years; the extremes range from a month to many years.

Treatment.—This is *medicinal*, *hygienic*, and *operative*. The first is limited in its purpose to the cure of syphilitic conditions, simulating tumor and, perhaps, in a slight degree, to tuberculosis. The astonishing effect of the mercurial and iodin treatment upon syphilitic new formations is nowhere so well shown as upon cerebral gumma, but probably only in its early structure. Unless syphilis can be excluded with absolute certainty, the *iodid of potassium* should be given in any case in ascending doses, limited only by their effects. In the absence of syphilis the larger doses are not well borne. In addition, *mercury* should be used, at first preferably by inunction until the specific effect is produced, after which it may be discontinued, to be renewed as indicated. Instead of inunction, the *bichlorid* may be given internally in doses of 1/12 grain (0.005 gm.) three times daily, or until the physiological effects are produced, or the salicylate of mercury may be given hypodermically. When the tumor is once under control, it is still necessary to keep up the treatment in such doses as experience may determine to be necessary. Usually the iodid of potassium is sufficient for this purpose. When, however, the symptoms of tumor disappear and remain absent many years under iodids, the diagnosis of tumor may be doubtful. This would seem to be verified by the following case: Tyson had under observation

for 30 years a patient in whom the disease has been kept in check by a dose of 60 grains (4 gm.) a day, which had occasionally to be doubled for a time. The evidence of a syphilitic lesion in this case seemed conclusive, since following acknowledged infection there occurred secondary symptoms of syphilis, the full train of classic symptoms of brain tumor, including ophthalmic symptoms studied by an experienced ophthalmologist. The case, however, came to necropsy and only meningitis was found. If mercury is necessary in this stage, the *biniodid* may also be used in doses of from 1/24 to 1/12 grain (0.0025 to 0.005 gm.), as required, though I have not the confidence in it that I have in the separate use of the iodid of potassium and the bichlorid of mercury.

In tyroma the usual constitutional treatment of tuberculosis by cod-liver oil, iron, and other tonics, with nourishing food and healthful indoor and outdoor life, is to be carried out.

The usual remedies indicated to relieve pain are to be used, bromids, if necessary, in large doses, phenacetin, antifebrin, and antipyrin, and, if necessary, morphin. The ice-cap may be used. Other symptoms should be treated by appropriate remedies.

The hygienic treatment is of the greatest importance. Excesses of every kind should be avoided, alcohol should be rigidly excluded, as well as all sexual excitement and mental excitement of any kind, for a slight increment of blood in the brain may bring on a convulsion and cause death.

Exploratory operation being much less dangerous than formerly, with the aseptic precautions of the present day, should be made whenever the tumor can be localized with any approach to accuracy. Although cerebral localization has been developed to a very high degree, it must still happen that we frequently fail to locate a tumor accurately. Cerebral decompression is important for relief of symptoms, especially when the location of the tumor cannot be determined, and if performed early may save the eyesight as well as remove some other symptoms.

SUPPURATIVE ENCEPHALITIS.

SYNONYMS.—*Suppurative Inflammation of the Brain; Cerebritis; Abscess of the Brain.*

Definition.—By encephalitis is meant inflammation of the substance of the brain as contrasted with inflammation of its membranes. What is spoken of as inflammation of the brain in popular parlance is really inflammation of the membranes of the brain, or meningitis. A literal application of the term encephalitis is here intended.

Etiology.—The causes of cerebritis are: (1) Traumatic; (2) and adjacent focus of inflammation extending to the brain substance; (3) pyemia.

Under traumatic causes are included blows upon the head and falls, more commonly those attended by fracture or punctured wound; although it is not necessary that there should be even a scratch upon the skin.

Under adjacent disease, whence extension of inflammation is especially frequent, is to be included caries of the petrous portion of the temporal bone due to disease of the middle ear or labyrinth, the most common of all

causes of abscess of the brain. Disease of the orbit or of the nasal passages is another focus of the same kind. The route of such a communication may be through either the sinuses of the brain or the lymph paths.

Pyemic abscess of the brain is rare. Causal foci are malignant endocarditis, gangrene of the lung, chronic bronchitis with bronchiectasis, bone disease, suppuration of the liver, and the specific fevers, among which may be included *la grippe*.

Encephalitis occurs most frequently between the ages of 10 and 40, and about three times as often in the male sex as in the female.

Morbid Anatomy.—Abscesses of the brain are usually solitary, though there may be two or three, or even more. The abscesses may be from one-half to three inches (one to eight cm.) in diameter, rarely more, though an entire lobe has been involved. The abscess itself is a very interesting product. Unless very recent, it is surrounded by a distinct wall which is composed of *three layers*. The inner is smooth, made up for the most part of granular fatty cells. Outside of this is a layer of germinal tissue containing spindle cells and more perfect fibrillated tissue. Externally again is another layer of fatty cells. The pus within the abscess is usually greenish-yellow in color and acid in reaction, while its corpuscles are distinctly nucleated. The *zone outside* of the *abscess* is *edomatous*, the cells are swollen, sometimes disintegrated, with blood points scattered throughout, becoming sparser as the periphery is extended.

The locality of the abscess may be preceded by the condition known as *red softening*, which is often spoken of as the first stage of the inflammation, but it is most important to remember that red softening is not peculiar to abscess. It consists simply of brain substance broken down into a reddish, blood-stained pulp. In this substance are found fragments of nerve-fibers, drops of myelin, pus-corpuscles, and granular fatty cells. The termination of cerebritis is not always in abscess. It is barely possible, before the stage of abscess is reached, for a condition of *yellow softening* to supervene, and the so-called apoplectic cyst may be the final result, or even cicatricial tissue may develop.

The *cerebrum* is involved *four times as often as the cerebellum*, the *left hemisphere* more frequently than the right, and the temporo-sphenoidal lode more than any other. The cause has something to do with the location: Ear disease places the abscess in the temporal lobe or cerebellum; if in the tympanum, the cerebrum rather than the cerebellum; if the mastoid cells and labyrinth, the cerebellum.

Symptoms.—While inflammation of the brain is spoken of as acute and chronic, more strictly speaking it is rather primary and delayed, the symptoms of the so-called chronic form being essentially the same as those of acute cerebritis, but characterized by their late appearance after the cause which precedes them. In acute cases the symptoms develop rapidly and may run their course in a few days, while in the forms known as chronic the symptoms are scarcely less rapid after they once set in, which may be weeks, months, and even longer, after the operation of the cause.

These symptoms are the result of pressure—direct or indirect—of destruction of the brain substance, or of poisoning by absorbed putrid matter. They are much the same as those of meningitis, with which, indeed,

abscess is often associated, especially if there is injury. The most striking are *headache*, often severe and persistent; *vomiting; vertigo; mental dullness*, succeeded sometimes by *delirium* and sometimes by *coma*. *Convulsions* are often present, and are epileptoid in character. *Optic neuritis* is also one of the symptoms. Other cranial nerves beside the optic are sometimes involved.

There is usually *fever*, as shown by elevation of temperature. At other times the temperature is normal or subnormal. The *pulse* is usually slow—from 60 to 70. The symptoms may set in with a *chill* after the latent period. The toxic symptoms are those usual to toxic states—viz., chill, irregular fever, prostration, emaciation, exhaustion. *Paralysis* in the form of hemiplegia sometimes occurs. The paralysis, however, is not always hemiplegic, and may be limited to the arm and face, especially in abscess of the temporo-sphenoidal lobe, which may compress the internal capsule. If on the left side, there may be *aphasia*.

When the abscess is in the parieto-occipital region, there may be *hemianopia*. It is especially in abscess of the cerebellum that *vomiting* occurs, and *staggering* if the middle lobe is affected.

Of the *chronic* form it has already been said that the symptoms, though long delayed, are the same as those of the acute form. Such delay, however, does not always cover all symptoms, since during the latent stage the patient may have headache or vertigo in a mild degree, and especially may be irritable and depressed, while he may even have a convulsive seizure during this preliminary period. It occasionally happens that there are no symptoms at all, and cases have occurred, more particularly of abscess in the frontal lobe, in which there were no signs or symptoms before death.

Phlebitis of the superior petrosal and lateral sinuses is especially common when the abscess is caused by disease of the ear, since the former receives a vein from the internal ear, and the latter receives the mastoid veins. *Edema about the ear and neck* and *hardness of the jugular veins* should suggest phlebitis, while *rigidity* of the neck and *cranial nerve paralysis* even more unerringly point to meningitis.

Diagnosis.—This is easy in acute cases, being substantiated by the history of injury, rigor, and fever, followed by the brain symptoms described. Almost as certain is the diagnosis when such symptoms follow chronic ear disease or localized putrid lung disease. It is to be remembered, that general cerebral symptoms may be produced by pus in the middle ear. These should be treated by puncture of the tympanum, and should the symptoms persist, after puncture abscess may be suspected. In like manner *meningitis* and *abscess* may be confounded, and with reason, because meningitis may be produced by the causes that produce abscess; and may be caused by abscess, and both may occur together. Meningitis, however, affects the cranial nerves more than abscess, unless the abscess is seated in the pons, and usually meningitis succeeds more promptly upon its cause. It is to be remembered that *tumor of the brain* may produce symptoms identical with those described. The chief distinctive symptom in abscess is the presence of fever.

Prognosis.—This, unless we admit a curable form described by Strümpell, is always ultimately fatal unless we have the rare good fortune to reach the abscess by operation.

Acute cases last from eight to 14 days, rarely 30 days; the delayed cases may not show their first symptoms for months. In the curable form referred to, Strümpell says pronounced symptoms of focal disease exist for a time and suggest a tumor, but after some months or even a longer time they gradually abate, and recovery is complete. The nature of the symptoms is such as to suggest a seat in the cortex, for there is usually paresis of some part of the body, often associated with symptoms of motor irritation and impairment of speech.

Treatment.—A certain prophylaxis may be exercised in the proper treatment of disease of the ear, for it is often the neglect of this which leads to the abscess. Such prophylaxis includes measures which secure free discharge and antisepsis. Beyond this the only treatment for abscess which promises anything toward a favorable result is operation, on which account the surgeon should be promptly associated in the treatment of the case. The use of the trephine has saved a few cases. For the details of the operation the student is referred to text-books on surgery.

Encephalitis without Abscess.—When, on the one hand, inflammation of the surface of the brain accompanying meningitis is eliminated, and, on the other, softening of the brain, formerly thought to be the result of inflammation, but now known to be due to the arrest of blood-supply, a number of cases of encephalitis without abscess remain, in some of which a necropsy was obtained.

CHRONIC HYDROCEPHALUS.

Definition.—A collection of serous fluid either between the meninges or in the ventricles of the brain. The former constitutes intermeningeal hydrocephalus, or *hydrocephalus externus*, or *hydrocephalus ex vacuo*. The latter is ventricular hydrocephalus, or *hydrocephalus internus*. The seat of effusion in hydrocephalus externus may be either in the subdural space—*i. e.*, between the dura mater and the arachnoid—or in the subarachnoid space. The first was formerly regarded as the most frequent; later its occurrence came to be denied, but more recently, by means of frozen brain sections, it has been demonstrated. Since the subarachnoid space communicates with the ventricles of the brain, the two forms of hydrocephalus may coexist. Both external and internal hydrocephalus may be diffuse or circumscribed. When circumscribed there result in the case of the forner cystic spaces in the membranes, and in the latter distention of portions of the ventricles.

External Hydrocephalus occurs in connection with atrophy of the brain, and is not of much clinical importance.

Internal Hydrocephalus.

This is divided into congenital and acquired.

Congenital Hydrocephalus.

This develops before birth, and may be present to such a degree as to retard the birth of the head. More frequently it is not recognized until some time after birth.

Etiology.—This cannot be said to be certainly known. Virchow early ascribed it to inflammation of the ependyma; Rindfleisch rather to an obstruction to the circulation in the choroid plexus. Drunkenness and syphilis in parents, and accidents in pregnancy, are held responsible; occasionally, also, tumors of the brain. More than one child in a family is sometimes affected.

Morbid Anatomy.—The head is characterized externally by its spherical shape and large size, its smooth eyebrows and protruding eyes, the last being due to depression of the orbital plate of the frontal bone. The protrusion is often so great that the eyelids cannot close over the eyes. The size of the head thus obtained is often enormous—from eight to ten inches (20 to 25 cm.) in diameter in a child of three or four years. On the other hand, the face appears very small. On closer examination the cranial bones are found separated and exceedingly thin, at times almost as thin as paper. In the membranous interspaces are often found Wormian bones. The veins may be seen beneath the skin, and fluctuation may sometimes be obtained through the scalp. On incising the brain a variable quantity of limpid fluid passes out. The quantity is sometimes enormous, reaching 20 pounds (40 koils) or more. The cerebral cortex is greatly thinned, the thickness on the convexity being reduced to but a few millimeters. The gyri and the basal ganglia are compressed, and the ventricles are dilated. The commissures are stretched and even torn. The foramen of Monro is a wide opening, and the third ventricle is dilated and sometimes also the fourth. The ependyma is thickened, the choroid plexuses are vascular, sometimes little changed.

Symptoms.—These consist largely of the external morbid states just described, but in addition there is *slowness of physical and mental development.* The child learns to walk late and is very feeble and likely to be mentally deficient, although it is sometimes bright. The weight of the head is sometimes so great that the head inclines to fall to the side or backward or forward, and must be supported by the hands of the patient. The fontanelles are wide open; other symptoms may, at times, be decidedly delayed, and the child may make some progress in studies. Signs of *mental imbecility* sooner or later make their appearance, manifested first, perhaps, by absence of development, but progressing until the child lives an almost vegetative existence, having to be fed and cared for like an infant, even though several years old. At times there is early headache. There may be *convulsive contractions*, *tremors*, *ataxic gait*, *paresis*, and *paralysis;* in fact, all the symptoms which succeed on irritative and destructive lesions of the nervous system. So, too, if life is sufficiently prolonged, the symptoms of tumor of the brain may be quite closely simulated, especially when the cranium does not enlarge with the growing distention of the ventricles. There may be *choked disk*, *atrophy of the optic nerve*, or *total blindness*. This is more true of acquired hydrocephalus. There may be prolonged attacks of *drowsiness*, or *coma*, with *slow pulse*, while *sudden death* is not uncommon during epileptiform convulsions or apoplexy.

Diagnosis and Prognosis.—The rachitic head may be mistaken for the hydrocephalic, but the latter has not the broad forehead with prominent

frontal eminences; it is rather spherical and smooth. A child with congenital hydrocephalus raraly lives to be more than four or five years old, though it may attain adult life.

Acquired Hydrocephalus.

Etiology.—This is also commonly ascribed to some inflammatory process, although it is said to be sometimes idiopathic. Especially is it a consequence of suppurative and tuberculous meningitis, when it is spoken of as acute acquired hydrocephalus, though chronic inflammatory processes may also cause it. Derangements in the circulation in the choroid plexus and changed in the ependyma of the ventricles may, however, be responsible. Especially may a tumor in the third ventricle, at the base of the brain, pressing upon the venæ Galeni or on the straight sinus of the dura mater, be a cause; or closure of the foramen of Monro or of the aqueduct of Sylvius. Even lung or heart affections and growths in the mediastinum and neck may produce the needed obstruction.

Morbid Anatomy.—In cases of acquired hydrocephalus, even though beginning tolerably early in life—say the seventh year—as well as in adults, the skull does not necessarily expand, and the head may not enlarge. Indeed, the head may even be smaller than natural, as in cretins. In these instances the brain substance must yield, and is reduced in thickness, at times to a few millimeters only. In other cases the skull yields, its plates become thin, the fontanels grow larger, and an appearance like that of congenital hydrocephalus may result.

Symptoms.—The symptoms of acute acquired hydrocephalus are never distinctive, on account of the rapidity in the course of the disease which produces and obscures it.

Of chronic acquired hydrocephalus as of congenital the most striking symptom is, as a rule, the *marked distortion in the size and shape of the head* already described.

Other symptoms are those of congenital hydrocephalus influenced by the greater age the patient may attain.

Spontaneous evacuation of the fluid sometimes takes place by the nose, mouth, ear, or orbit.

Diagnosis.—This is commonly easy. It is only in cases in which the cranium does not expand that the symptoms of *brain tumor* may lead to a diagnosis of the latter condition instead of hydrocephalus.

Prognosis.—This is usually unfavorable. Generally the child lives from two to five years, though it may perish in a few months or live for 10 to 15 years, or, as in a case of Bright's, to 29 years, or even longer. It has happened that spontaneous recovery has followed the evacuation of fluid previously described. The absorption of small amounts of fluid is also possible.

Treatment.—This consists primarily in the treatment of the disease which is responsible for the hydrocephalus if it can be discovered; secondly, in the treatment of the symptoms which may arise, and next, in attempts to cure the malady. Some favorable results have followed the removal of the fluid by puncture of the ventricles, although there has been failure in the majority of instances. Measures should be taken to make

the removal gradual, if possible, thus attempting to imitate the spontaneous efforts of nature, which have occasionally been followed by recovery. To this end the slow removal of the fluid—by puncture of the subarachnoid space between the third and fourth lumbar vertebræ—has been recommended and practised by Quincke. At this point, too, the spinal cord is not very likely to be injured. It is more particularly in congenital hydrocephalus that operation may be employed, but operation has not resulted in much improvement.

If operation is deemed undesirable, attempts may be made to ged rid of the fluid by diuretics and purgatives, although with little prospect of success. Iodid of potassium may be tried, with the faint hope that the hydrocephalus is due to a syphilitic tumor which might thus be melted away. Blisters may also be applied.

GENERAL AND FUNCTIONAL DISEASES—NEUROSES.

The term neuroses is applied to nervous affections in which there are functional disturbances corresponding to which there is no known anatomical lesion.

ACUTE CHOREA.

Synonyms.—*Chorea minor; Mild Chorea; Sydenham's Chorea; St. Vitus' Dance.*

Definition.—A disease chiefly of the young, characterized by irregular, involuntary muscular contractions, associated at times with psychical disturbance, often with rheumatism and endocarditis. The term chorea is derived from the Greek χορεια, dancing.

Etiology.—The disease, though not confined to children, occurs far more frequently among them, notably from the time of the second dentition—the sixth or seventh year—to the 15th year. More than three-fourths of the entire number of cases occur during this period. Among adults it is relatively more frequent from the 15th to the 24th year. Occasionally it occurs in old age, when it is known as *chorea senilis*. Chorea is about twice as frequent in the female sex as in the male if all periods of life are considered, but below the period of puberty the difference in the sexes is not so striking. Heredity has always been an acknowledged factor in its causation, but is probably less significant than was once supposed. It has even been claimed that the disease is sometimes congenital in the offspring of a choreic mother. It is more frequent in neurotic families. As to temperament, it is well known that high-strung, excitable, nervous children, as contrasted with the dull and phlegmatic, are especially liable to the disease. It is principally in these that overstudy is seen to have a predisposing effect. Psychical influences are undoubtedly potent; thus, fright causes a large number of cases, while grief causes many, and even joy some.

The so-called Huntington's chorea, which is hereditary, is not the same as Sydenham's chorea, although Charcot did not make this distinction. Sydenham's chorea affects children of all social grades. It is rare in the negro. Wharton Sinkler, who has especially investigated this point, has seen but

one case in a full-blooded negro, while William Osler, at the Johns Hopkins Hospital, out of 175 cases found five in the negro race. It is apparently unknown among Indians in their natural state.

The season of the year appears to have an undoubted influence. Morris J. Lewis, whose studies have been most thorough in this direction, finds that the fewest attacks occur in October and November and the greatest number in March and April. Hermann Eichhorst, on the other hand, says that the greatest number of cases occur in the autumn and winter months. The disease prevails more generally in towns than in the country.

Imitation, commonly regarded as an exciting cause, has been shown by modern studies to play a less important rôle than was thought, many cases described as thus originating being really hysteria. Trauma precedes a certain number of cases. Reflex irritation, especially digestive disturbances, and intestinal worms were regarded as potent causes by the older observers; but here again Osler's studies have failed to find any causal relationship. The chorea of pregnant women has been referred to this category. The causal relation of eye-strain to chorea has been emphasized by Stevens, but is practically denied by George de Schweinitz, who concludes, from an examination of more than 100 cases, that, while ordinary chorea and many forms of facial spasm—habit spasm, etc.—are materially benefited by correcting refractive errors and anomalies of the ocular muscles, he does not believe there is any proof to show that eye-strain is of itself responsible for their origin, with perhaps the single exception of habit spasm affecting the orbicularis and adjacent facial area. It may be such chorea which Howard F. Hansell cured in Da Costa's clinic[1] by atropin, paralyzing the ciliary muscle and preventing the effort at accommodation until the habit was broken up.

The association of arthritis and chorea was observed by the earliest students of the subject, and was distinctly recognized in England as early as 1802, but the exact causal relation of the two diseases has, perhaps, not yet been made out. That they are frequently associated and that there is close connection between the two affections is admitted by English and French writers, but the Germans find the association much less frequent. Steiner, for example, found only four cases of rheumatism in 252 cases of chorea. English observers find from 20 to 70 per cent. of cases of associated joint affection, while in this country, where rheumatism is apparently less frequent in children, the range of percentage found by various observers is from 15.5 to 54 per cent. That the arthritis precedes the chorea in a large number of cases is generally conceded, the latter disease developing with the subsidence of the former, or not until convalescence has been well established. The authors believe that while the infectious nature of chorea is not established, there is enough evidence to justify the belief that it is the result of some unknown infection, possibly the same as acute rheumatism. This theory is further sustained by the fact that the infectious diseases play an acknowledged rôle in the etiology of chorea. Scarlet fever, diphtheria, measles, typhoid fever, gonorrhea, secondary syphilis, puerperal fever, pyemia, multiple suppurative polyarthritis, have all been followed by chorea; but with the exception of acute rheumatic

[1] Da Costa's "Medical Diagnosis," eighth ed., p. 221, 1895.

polyarthritis and some forms of septicemia, the number of cases thus associated is not large. On the other hand, acute exanthemata developing in the course of chorea usually check the disease. Anemia has been held to be a cause, and probably is a predisposing cause, although frequently also a result. In fact, the studies of Charles W. Burr and others go to show that anemia is less frequently associated with chorea than has been commonly supposed. The relation of hysteria to chorea is interesting from the close resemblance, at times, of the two conditions. It has already been said that the cases of so-called imitation chorea are often examples of hysteria, and, on the whole, the association of the conditions is rather coincidental than causal, but some cases may be truly imitation in children not hysterical. Poisons are acknowledged causes in a few instances. Carbon dioxid and iodoform are among those which appear to have caused acute attacks of chorea of short duration.

Morbid Anatomy.—There is no definitely ascertained morbid anatomy for chorea, and the lesions which have been found are the result of the complications or are incidental. The most constant of these associated lesions are endocarditis, in 85 per cent. of Osler's cases; pericarditis, 26 per cent.; combined heart lesions, 90.4 per cent.; pneumonia, 12 per cent.; less numerous were acute pleurisy, pyemia, and phlebitis, also noticed. As to the nervous system, the symptomatology would lead us to expect the essential lesions in the cortex of the brain, and C. L. Dana has analyzed the recorded autopsies, of which there were only 39 in which the state of the nervous system was accurately described. In 16 there were intense cerebral hyperemia, periarterial exudation, erosions, softened spots, minute hemorrhages, and occasional emboli. The changes were most marked in the deeper parts of the motor tracts, particularly in the lenticular nuclei and the thalami. These changes are the same as those described by W. H. Dickinson in 1876. Essentially similar were the lesions found in two of Osler's cases. In two reported by Bevan Lewis there was apoplexy, one cerebellar and one cerebral and extraventricular. The so-called chorea corpuscles described by Ellischer are in no way characteristic. The same may be said of the swelling and turbidity of certain of the large pyramidal cells in the deeper layers of the cortex in the Rolandic region described by F. C. Turner. The changes in the ganglion cells of the spinal cord described by H. C. Wood in canine chorea have been found also by Triboulet, but he agrees with others who hold that canine chorea is a very different disease from human chorea.

Nature of Chorea.—This, it must be admitted, is as yet unknown. It has been intimated that the symptoms are of a kind which would naturally result from lesions in the motor cortical area. No constancy in such lesions is demonstrable. A cerebral seat for chorea is rendered likely by the existence of hemichorea, the association of chorea with mild psychical derangements, and by the fact that choreiform movements are sometimes symptoms of undoubted brain lesions—*posthemiplegic hemichorea*. The embolic theory which was suggested by Senhouse Kirkes, and supported by him, Hughlings Jackson, Broadbent, Tuckwell, and others, was based upon the presence of foci of embolic softening found in a few instances in connection with endocarditis, but has gained few supporters.

The theory which is at the present day naturally attracting most attention is the infectious theory, but the limits of a text-book do not permit its developmental consideration. Suffice it to say that Pianese, of Naples, has apparently isolated from the nervous system of a choreic patient a bacillus which he was able to cultivate successfully, and the cultures from which caused death in animals; also that while the acuter forms present many, if not all, of the conditions necessary to the conception of an infectious disease. The views of the authors have been briefly set forth on the previous page.

Symptoms.—*Premonitory* symptoms, both motor and psychical, usually precede the onset of chorea. They include *restlessness* and inability to sit still, and an *altered disposition*, manifested by irritability and perversity. These symptoms, often misunderstood by parents, are sometimes the occasion of reproof and even severe punishment to the child—a course which accelerates and aggravates the disease.

A close study of the symptoms permits of their division into three separate groups, determined chiefly by their severity:

1. A *mild* form, including the majority of cases in which the affection of the muscle is slight, the speech scarcely involved, and the general health slightly disturbed.

2. The *severe*, in which the choreic movements are general, power of speech is lost, and the patient is unable to go about and help himself.

3. The *maniacal*, or *chorea insaniens*, characterized by intense cerebral excitement.

It is, however, unnecessary to separate the symptoms of each variety.

The *motor phenomena* are those first observed. They consist in peculiar jerky movements which begin most frequently in the upper extremities, especially in the right hand, rarely in the legs. They may even be general from the first, though the earliest symptoms often escape notice. *Speech* is affected, sooner or later, in one-fourth of the cases. The extent varies greatly from slight hesitancy to incoherency—the difficulty being in the muscles of articulation rather than in phonation. As a rule, the movements cease during sleep, though they sometimes persist even then. It is not generally believed that the movements extend to the muscles of organic life, though associated irregular and rapid action of the heart has been ascribed to choreic spasm of the papillary muscles. As the disease continues *muscular weakness* becomes manifest in a general want of strength rather than paralysis, though the weakness may affect both limbs of the same side, or only one limb. It may even precede the jerking movements. Very rarely the pulse may be slow in the feeble state that follows chorea.

Sensory symptoms are less conspicuous than motor. Pain is rare, though its presence has been characteristic enough in some cases to obtain the name "painful chorea" from Weir Mitchell. Painful points over the sites of emergence of spinal nerves have been pointed out, though they must be rare. *Numbness*, *tingling* and *prickling* sensations are occasionally met, and may be a part of the phenomena of multiple neuritis sometimes present. *Headache*, sometimes very severe and paroxysmal, may occur, while *epileptiform seizures* are also a rare symptom, and when they occur are probably not a part of the chorea. The *reflexes* are variously affected, the knee-jerk being normal in about half the cases, in the remainder increased

or absent. Trophic lesions are almost unknown. *Mental symptoms*, in the majority of cases, are not very conspicuous, though there are in some severe cases extreme manifestations, including melancholia, hallucinations, and even mania, which have their climax in *chorea insaniens*.

Most important are the *symptoms of cardiac disease*, in regard to which William Osler makes the startling statement: "There is no disease in which endocarditis is so constantly found postmortem as chorea. It is exceptional to find the heart healthy." The symptoms which are, therefore, to be always carefully sought include a *systolic apex murmur, palpitation, and irregular heart action*, although the child rarely complains of the latter or of pain about the heart. It is further important to note that in a majority of these cases the endocarditis is independent of acute arthritis, unless we hold with Bouillaud that in young subjects the heart acts as a joint. Organic *murmurs at the base* are very much more uncommon, most of the murmurs here being functional. They are heard with greatest intensity in the area of the pulmonary artery, but are audible sometimes in the aortic area as well. In a large proportion of all cases in which a murmur is heard at the base or along the left margin of the sternum in the second, third, and fourth interspaces it is functional, but a soft systolic murmur in this area with systolic pulsation in the cervical veins may be caused at the tricuspid orifice.

On the other hand, *endocarditis* sometimes occurs *without symptoms* or physical signs, while the disappearance of physical signs does not prove that endocarditis was not present. A *presystolic murmur* is also at times present, indicating mitral stenosis—in 19 per cent. of Osler's cases. On the other hand, the comparative rarity of simple aortic valve involvement is conspicuous, this being more uncommon than combined aortic and mitral disease, or even combined mitral and tricuspid disease. The tricuspid valves may alone be attacked.

A to-and-fro murmur, indicating *pericarditis*, may be present in from 8 to 25 per cent., and in more than half of these it is associated with endocarditis. It is to be remembered that both forms of organic heart disease, and especially endocarditis, may occur in chorea without rheumatism—*e. g*,. in 66 per cent. of Osler's cases—also that such endocarditis may lay the foundation of permanent organic disease.

W. S. Thayer, in his studies of 689 cases at Johns Hopkins Hospital or Dispensary, found evidence of cardiac involvement in 25.4 per cent. of the cases, and in the wards of the Hospital 50 per cent. The cardiac involvement occurred with somewhat greater frequency in cases where there was acute polyarthritis than where such history was absent.

Cardiac involvement was commoner in cases of chorea with frequent recurrences than in those in which there was but a single attack.

In 110 cases treated in the wards there was fever of moderate degree in almost every instance, and where there was high fever there was evidence of cardiac involvement. There is good reason for the belief that the presence of fever in otherwise uncomplicated chorea is in a large proportion of cases associated with complicating endocarditis.[1]

[1] "Journal Am. Med. Assoc.," Oct. 27, 1906.

Occasional *skin affections* make their appearance in chorea, the larger proportion being due to the prolonged administration of arsenic, so much used in the treatment of this disease. The forms for which the arsenic treatment is more or less responsible are erythematous and papillary eruptions, herpes, and the pigmentation frequently resulting from the prolonged administration of this drug. Eruptions also occur independent of arsenic administration. They are usually purpuric and associated with arthritis, similar in form to the purpura so often associated with rheumatism, and include some of the forms of multiple erythema—as erythema nodosum, purpuric urticaria, or simple purpura. C. H. Brown[1] has reported a remarkable case of subcutaneous nodules composed of young granulating tissue in a case of chorea in a boy of 11.

Fever is a rare symptom in chorea, except as the result of complications, of which arthritis is the most common, but endocarditis and pericarditis may also cause fever. The rare instances are cases of *chorea insaniens*, in which the temperature may rise to 105° F. (40.5° C.).

Diagnosis.—This is usually easy. *Simple tremor*, *athetosis*, *paralysis agitans*, as well as *alcoholic*, *senile*, *saturnine*, and *mercurial tremor*, are not likely to be confounded with the movements of chorea. The symptomatic choreiform movements due to cortical irritation by *meningitis*, *tubercle*, *hemorrhage*, *softening*, *tumor*, or *parasites*, are attended by other symptoms which distinguish them from chorea. *Friedreich's ataxia* might be mistaken for chorea, but it is easily recognized by the lost knee-jerks, the slowness and inco-ordination of movements, talipes, nystagmus, and family distribution. Huntington's chorea is characterized by its heredity, its limitation to adult life, and ultimate gradually developing dementia.

Prognosis.—Except in *chorea insaniens*, which is always fatal, recovery is the rule in from eight to ten weeks. It happens, too, sometimes that the severest cases of the ordinary forms are intractable, and rarely that they terminate fatally after a few days' illness, it may be from exhaustion or it may be from the complicating heart disease. Chorea of the pregnant woman is more serious than the chorea of children.

The *duration* of the disease may be from eight to ten weeks for the ordinary cases and from three to six months for the very severe ones. *Remissions* occur, and *relapses* as well, pointed out by Sydenham. A disposition to vernal recurrence has been noticed.

Treatment.—All cases should be carefully examined for causes of reflected *irritation*, which should be *removed;* then *rest* is essential. It is not necessary that in the mildest cases the patients be put to bed, but they should be withdrawn from school and guarded from excitement and the curious gaze of friends and strangers, for the movements almost invariably increase when the patient is under observation. In more serious cases confinement to bed should be employed—a more thorough exclusion as well as rest is thus secured. Not only is recovery thus facilitated, but a diminished liability to heart complication is also attained.

Of drugs, *arsenic* and *iron* hold the first place. The former is given in slowly ascending doses of Fowler's solution until its physiological effect is produced, after which the dose should be gradually diminished. Some one

[1] "Journal of Mental and Nervous Disease," August, 1893.

of the preparations of iron should be given continuously in moderate doses. The bromids are also indicated, especially when there are restlessness and want of sleep, when chloral may also be added, and in severe cases may be given continuously. Opiates should, however, never be employed. An old remedy in this country is black snakeroot or cimicifuga racemosa, first recommended by the late Hiram Corson, who wrote Tyson that he had used it for 50 years without a failure. Tyson has sometimes used it in the shape of the infusion in mild cases, with apparently satisfactory results, in doses of 1 or 2 fluidounces (30 to 60 c.c.). Modern remedies are aspirin, antipyrin and physostigma. The former is given to adults in doses of from 7 to 15 grains (0.5 to 1 gm.), much reduced for children. Physostigma has been given in doses of from 1/70 to 1/35 grain (0.0094 to 0.0188 gm.) hypodermically. Hyoscyamin in doses of 1/100 grain (0.00065 gm.), three times a day, has apparently been followed by good results. The oxid of zinc, valerianate of zinc, nitrate of silver, and sulphate of copper, formerly much recommended, have fallen into disuse. In consequence of the close relations between chorea with its attending arthritis and rheumatic arthritis it is reasonable to expect that the salicylates might be useful, but such expectation has not, as yet, been realized.

Montrose Graham Tule attaches great value to apomorphin. He administered in a desperate case, a girl of 15, hypodermically 1/40 grain (0.0016 gm.) which allayed the spasms in three minutes. This was followed up by 1/20 grain (0.0033 gm.) by the mouth every three hours, followed by prompt recovery. Chloralose has also controlled the spasms in an acute case in my hands.

CHOREIFORM AFFECTIONS.

There remain to be considered some forms of convulsive contractures several of which are included under the term "habit spasm" or "habit chorea," and "tic." The term *tic*, as originally understood, means *facial spasm*. It has, however, been extended by the French school (whose lead in these affections seems at present acknowledged) to include "an habitual, conscious, convulsive movement, resulting in the contraction of one or more muscles of the body, reproducing, most frequently in an abrupt manner, some reflex or automatic action of common life" (Guinon). It is characteristic of these motions that they are more or less under the control of the will, and the tic movements have a purposeful character, in which respect they differ from the contractions of chorea minor.

I. Simple Tic.

Tic should not be confused with habit spasm. Tic is a quick movement, of a purposeful character, usually in the same group of muscles, and occurring in a neurotic individual; its prognosis is often bad. Habit spasm is often in different groups of muscles, is not purposeful, is slower, is often sign of nervous child and often outgrown. Oppenheim gives description.

Simple tic may be localized or general.

Localized tic begins usually in young persons, most frequently in girls from seven to 14 years of age, and may persist through life. The spasm

is confined to a single muscle, a group of muscles, or a group of associated muscles, *most frequently the muscles of expression*. The mild forms are looked upon as simply peculiarities of the individual; but the more severe forms, in which nearly all the muscles of the face are affected and even the depressors of the jaw and the tongue are often thrown into action while speaking, are manifestly pathological. It differs from the *idiopathic facial* spasm of adults in that the latter is rarely seen until after the 40th year, and is, moreover, slower than the habit spasm of the facial muscles. It is possible for the simplest forms of habit spasm to be a childish trick perpetuated; such may be a blinking of the eye or the act of sniffing. In other simple forms there is a drawing aside of the mouth or a jerking of the head to one side, or a simple shaking of the head, while the eye is winked at the same time; or there may be shrugging of one shoulder. More rarely the contraction occurs in the legs, as in the very characteristic "string-halt" like in which at times the leg is suddenly drawn up. Localized tic may be transient, gradually disappearing after a few months. The French school has devised a method of treatment of these localized tics, consisting of educational movements of the affected muscles.

Generalized Tic, Electric Chorea (Henoch).—In this there is sudden electric-like spasm of the muscles of the trunk and limbs, but especially of the neck and shoulders, causing an instantaneous start, which affects the patient for an instant only, when it passes off and leaves him quiet and motionless. The contraction is like that produced by a galvanic shock. It may be associated with facial spasm. It occurs especially in children, but also in adults, particularly in women, and may persist for years.

Paramyoclonus Multiplex; Myoclonia.—This term was applied by Friedreich in 1882 to a disease first observed by him, in which there are clonic convulsions in symmetrical muscle groups in the arms and legs without loss of consciousness. It occurs usually in males, and follows emotional disturbances like fright. In addition there is a considerable increase in the tendon reflexes. In order that a case may be one of true paramyoclonus it is necessary that the contractions in the single muscles should be sudden—lightning-like. The muscles affected are commonly those of the trunk and extremities. The contractions are usually bilateral, and vary from 50 to 150 a minute. There are no sensory symptoms. Between the attacks there may be tremors. These cases are allied, on the one hand, to the electric chorea just described, and, on the other, to the different forms of convulsive tic, clonic facial cramp, and clonic cramp of the neck muscles. Some cases of so-called paramyoclonus are really cases of hysteria. This view is sustained by Arthur Conklin Brush,[1] who reports three cases and reviews several.

Dubini's Disease.—The term electric chorea is applied to an acute infectious disease occurring in Lombardy, and known as Dubini's disease, in which there are sudden contractions, first usually in the arm, but passing thence into all the extremities, followed in several weeks or months by paralysis and muscular atrophy, occasionally by epileptiform convulsions and fever. No morbid anatomy has been determined.

[1] "The Nature of Paramyoclonus Multiplex," "American Jour. of the Medical Sciences," December, 1899.

II. Tic with Explosive Utterances, Coprolalia, Echolalia, Fixed Ideas, etc.

Synonyms.—*Maladie de la tic convulsif; Gilles de la Tourette's Disease.*

Definition.—In addition to motor spasm, this form of tic is characterized by explosive utterances of certain words and sounds, such as "fire," "murder," "hah," "bow-bow"; or profane words, such as "God damn," "Jesus Christ"; or filthy and obscene words, when it is known as *coprolalia.* There may also be mimicry of words, when it is called *echolalia*, or mimicry of action, *echokinesis;* or the patient may be possessed of a fixed idea of the variety known as *arithmomania*, *délire du toucher*, *onomatomania*, and *folie pourquoi.* In *arithmomania* almost every action is preceded by performing a certain number of acts, as in a patient of Osler's, who before she went to bed had to tap her heel upon the bedstead a given number of times; before drinking a tumbler of water, to rotate the glass nine or ten times, and the same thing when sitting it down; before opening a door a certain number of knocks had to be given, and the greatest difficulty was experienced in getting her to brush her hair, as it took so long to count before she began. In the *délire du toucher* there is the constant fear of contamination from contact with objects; in *onomatomania* to repeat over and over again names which arise, and in the *folie pourquoi* to demand a reason for every one of the simplest acts. In other instances the patient imagines that some one is talking to her. All these are in addition to the convulsive acts.

The involuntary movements themselves vary greatly from trifling tic in any one or more of the muscles of the face to contractions involving all the muscles of the body. This condition, which is neither chorea nor habit spasm, is at times mistaken for both. It is commonly easy of recognition, and although of uncertain prognosis, recoveries take place.

III. Complex Co-ordinated Tic.

Definition.—This includes a number of forms of habit movement differing from ordinary tic in the more complex nature of the actions performed. It includes tricks and habits, such as those of one who in writing stops at every few words and looks intently at his finger tips; the "head nodding" of children (not to be confounded with the *epilepsia nutans* of children), "thumb sucking," "rocking in bed," and similar actions. Of the same nature is the so-called "head-banging," in which the child, asleep or awake, while in bed, will turn over and bang the head violently into the pillow, repeating this act five or six times or for two or three hours at a time; or the child may strike the head repeatedly with the fist—*krouomania;* or it may rotate the head violently from side to side, balancing or gyrating the body with great rapidity. This practice is sometimes communicated from one child to another. These movements are met especially in feeble-minded children, in whom it may be accompanied by nystagmus, and is sometimes the result of injury after birth. When these phenomena do not occur in the feeble-minded or after injury early in life, the prognosis is said by Gee and Haden, who have especially studied the subject, to be favorable.

IV. Spasm of the Muscles of Respiration and Deglutition.

Definition.—The spasm affects the muscles of respiration and phonation, the muscular contraction being accompanied by more or less noise, as a "sniffle" or "hiccough" during inspiration, or some noisy or explosive sound during expiration. Such spasms are sometimes part of a hysterical state. Among those described as thus occurring is a sort of rumbling which comes from low down in the abdomen, passes up the stomach, and out of the mouth as an explosive loud noise—something like belching, but louder. In another instance there was a peculiar clucking noise in the throat accompanying motions, particularly those of swallowing, which disappeared only during sleep. Again, there may be a loud inspiratory cry preceded by three or four deep inspirations and followed by a deep, hoarse, expiratory sound.

V. Chronic Progressive Chorea.

Synonyms.—*Huntington's Chorea; Chronic Hereditary Chorea.*

Definition.—A disease of adult life, commonly hereditary, characterized by irregular movements, deranged speech, and ultimate dementia gradually developing.

Etiology.—Its frequent hereditary origin has been mentioned. Indeed, heredity is one of its most striking features, 25 per cent. of certain families having been victims, and even more than 50 per cent. of the adults in families. It is especially when both parents were affected, and seriously, that one or more of the offspring almost invariably have the disease if they live to adult age. The two sexes are about equally affected, though in some families males are oftener affected. It is further characteristic that if a generation is skipped, the disease never manifests itself again in that family, and that it *rarely* presents itself *before 30 years* of age. Huet has, however, collected seven cases of earlier onset. It is said, also, that it is not invariably hereditary, being sometimes due to emotional causes. In all the families in which this choreic tendency has been found the nervous temperament prevails.

Morbid Anatomy.—This is somewhat more definite than that of chorea minor. At least, there has been found at necropsy quite frequently a condition of *pachymeningitis* and *hematoma* of the dura mater with atrophy of the cortex, and less frequently a disseminated *encephalitis*, evidenced by subcortical foci of round cells. Nothing has, however, been found which can in any way be regarded as peculiar or as accounting for the disease occurring at a certain age or for its affecting certain individuals, though the lesions do explain the motor phenomena. It should be stated that Charcot and his pupil Huet do not separate this chronic progressive chorea from chorea minor, but all other writers do.

Symptoms.—The *onset is gradual* in hereditary cases, although it may be sudden in cases arising otherwise. As in chorea minor, *motor symptoms* are the first to appear: first usually in an unsteadiness of the gait or slightly irregular movements of the hands. Occasionally only the *mental symptoms* are the first to appear, not usually manifesting themselves until the motor

are well established. Motor symptoms include also *spasm* of the muscles of the face. The movements differ from those of chorea minor in being *slower* and by *absence of co-ordination*, strikingly manifested in walking. The station may be good, except for a slight swaying of the trunk, but an attempt to walk is followed by an unsteadiness characterized by marked lateral deviation from the straight line, by swaying of the body, and sometimes by precipitate falling movement from which the patient may, however, recover himself—in brief, a typical drunkard's gait. This unsteadiness ultimately makes locomotion impossible, and the patient takes to his bed. Yet before this stage is reached, although ataxic, he may be able to walk long distances. While at rest the movements cease altogether. They are aggravated by emotion and excitement, while in the beginning they may to a degree be influenced by the will. Thus, a patient said lately: "If I put my mind to it, I can stop it."

Speech is affected in most instances, being at first slow and hesitating and interrupted by interjections; later it is indistinct. The handwriting is likewise involved, the letters being irregular and badly formed, running into one another and off the line, and ultimately writing becomes impossible. Sensation and the special senses remain intact, as does the muscular sense, until the disease is advanced. The reflexes are usually increased.

The *tendency to insanity and suicide* has been referred to as an acknowledged symptom. Beginning as a simple irritability or moodiness with depression, it passes slowly over into feeble-mindedness. The suicidal impulse is sometimes carried out.

Diagnosis.—This is easy in the hereditary cases only. *Friedreich's ataxia* resembles it slightly, but begins earlier. *Idiopathic double athetosis* also occurs in elderly persons, but in it the movements are associated with rigidity and are of a peculiar character, and the gait is also spastic, while neither rigidity nor spastic gait plays any part in progressive chorea.

Prognosis is ultimately fatal. The progress of the disease is progressively and irresistibly from bad to worse.

Treatment is of no avail.

VI. Chorea Major.

Synonyms.—*Pandemic Chorea; Automatic Chorea; St. Vitus' Dance; Rhythmical or Hysterical Chorea; Lata; Miryachit; Jumpers; Jerkers; Holy Rollers.*

We prefer to include under this heading all the different varieties of saltatorial spasm of which the historical St. Vitus' dance of Paracelsus, prevalent in the fourteenth, fifteenth, and sixteenth centuries, is the most familiar illustration.

All are neuroses, in which strong contractions take place in the leg muscles when the patient attempts to stand, causing a jumping or springing action, and are illustrated by the "jumping Frenchmen" of Maine and Canada, the subjects of which are liable, on any sudden emotion, to jump violently and utter a loud cry or sound and obey any command or imitate any action without regard to its nature. The jumping prevails in certain

families. Similar were the "jerkers" who appeared during the religious revivals in Kentucky in the early part of the present century; and the "holy rollers," in New Hampshire and Vermont. The disease known as *lata* among the Malays, and *miryachit* in Russia are similar. In the true St. Vitus' dance, chorea major, or *chorea Germanorum*, the paroxysm arises spontaneously; so, also, in the salaam convulsions of children, in which the muscles of the abdomen are affected, and in which there is a bowing forward of the head and body as many as a hundred times or more. The paroxysms may occur several times a day, lasting from a few seconds to as many minutes. In the others, as the American jumpers, etc., it is in response to some external impression. During the paroxysm the affected person sings, dances, jumps from the ground, rolls from side to side, hammers with his hands, stamps with his feet, or whirls madly around until he falls exhausted to the ground.

VII. Postparalytic Chorea and Postchoreal Paralysis.

Synonym.—*Posthemiplegic Mobile Spasm* (Gowers).

Definition.—By this are meant choreiform movements which are the result of cerebral disease, most frequently hemorrhage. They may immediately precede or follow the stroke.

Posthemiplegic chorea, on the other hand, ordinarily appears in the limbs previously paralyzed, at the time when they again begin to be capable of motion. It is generally sudden, and either continues throughout life or disappears gradually. Often it is associated with contractures. Not infrequently the affected side of the body is anesthetic, and even the organs of special sense may take part in the hemianesthesia, in which cases it is probably a hysterical hemianesthesia.

The movements are more frequent in the hand than in the leg, though sometimes they occur in both, most marked in the fingers and toes, and diminish toward the shoulders and hips. They are really more athetoid than choreic, but quicker, consisting mainly in inco-ordinate gyrations of the fingers and thumbs, flexion and extension of the wrist and elbow, shrugging and other movements of the shoulder. They always cease during sleep. Carcot considers posthemiplegic chorea as identical with athetosis.

Symptoms.—The prehemiplegic form is rarer and more serious in significance. The movements vary greatly, and the milder degrees can be recognized only on close examination. In this form the symptoms precede, usually by a few days, the apoplectic stroke, and cease as soon as paralysis appears.

The lesion causing these symptoms is regarded as cerebral, and in that portion of the cerebrum within the internal capsule in which the fibers of the pyramidal tract pass between the lenticular nucleus and the optic thalamus. Sometimes, however, similar phenomena are associated with disease elsewhere, as in the pons or even in the spinal cord; but under any circumstances it would seem to be necessary that there should be irritation of the pyramidal tracts somewhere in their course.

EPILEPSY.

SYNONYMS.—*Morbus caducus sive sacer; Morbus divinus; Falling Fits.*

Definition.—Epilepsy is a chronic paroxysmal disease, characterized in its typical form by sudden loss of consciousness and by violent general convulsions (grand mal); but both unconsciousness and convulsions may be so fleeting as to be barely recognized (petit mal); while convulsions may be localized and unattended by loss of consciousness (Jacksonian or focal epilepsy); finally, seizures may be substituted by conditions of uncontrollable violence or somnambulastic acts (psychical epilepsy).

Epilepsy is, strictly speaking, a syndrome or group of symptoms of which the morbid basis is not always the same. Formerly it was considered essential to the diagnosis of epilepsy that the convulsions should not be toxic, reflex, traumatic, the result of previous brain disease, or heart failure. At the present day toxic convulsions, which are essentially covered in actual practice by uremic convulsions, are not regarded as epileptic, nor are pure reflex convulsions which are due to such causes as teething, constipation, worms, and other forms of peripheral irritation. On the other hand, certain convulsions due to cortical brain lesions, which will be further considered, are acknowledged to be epileptiform. For most cases of epilepsy no anatomical basis has as yet been discovered.

Etiology.—From one to six persons out of every 1000 have epilepsy. The tendency of modern studies is to diminish the importance of heredity, formerly so conspicuous as a supposed cause of epilepsy. Gowers' statistics, which may still be regarded as representing the older pathology, drawn largely from his own practice, ascribe to heredity a percentage of 35, while the range in the older statistics is from 9 to 40. Osler's observations, on the other hand, on cases at the Infirmary for Nervous Diseases in Philadelphia, and in the Institution for Feeble-minded Children at Ewlyn, Pa., give the percentage in the two institutions as a little over 1 per cent., and in five cases out of 435 in which the epileptics were children of epileptic parents it was traceable to the mother in every instance. The comparative unimportance of heredity as a cause is upheld by the modern French school, notably by Marie. On the other hand, the disease is of frequent occurrence in neurotic families, including those subject to insanity, hysteria, and neuralgia. So, too, vices of constitution and vicious habits in parents, especially alcoholism and syphilis, are acknowledged causes. The intermarriage of relatives is also an element. More certainly responsible is local disease of the brain *cortex*, including tumors and traumatic disease, such as are produced by fractures, and the conditions described as causing the cerebral palsies in children.

All of these are causes which may be both essential and exciting. Among the more purely exciting causes are fright, irritation by worms in the intestinal tract, dentition, constipation, and the like, all of which may provoke attacks in an epileptic. Some would regard the reflex epileptiform attacks excited by these causes as true epilepsy, and call it reflex epilepsy. But at present these cases should not be called epileptic, since they do not recur after the exciting cause is removed. These are very different from

others in which attacks are brought on by like exciting causes, but occur also independently of these causes.

True exciting causes are infectious diseases, alcoholism, and syphilis. The influence of infectious diseases is thus shown: Given an epileptic who is subject to seizures once a month, who acquires typhoid fever, the prodromal symptoms are almost always sure to include frequently recurring epileptic seizures. *Masturbation* is included among the true causes, but is probably only an exciting cause. *Ocular* and *aural irritations* are exciting causes. *Cardiac epilepsy* is a variety in which there is disturbance of the heart's action, either palpitation or slowing, prior to attacks; but such derangements are symptoms rather than causes, or they may be a mode of manifestation of the aura to be presently described.

Epilepsy is pre-eminently a disease of childhood and youth, and after 20 it is most unlikely to arise. Most cases begin between the ages of 10 and 16. Yet idiopathic epilepsy may occur after 60. It seems to be slightly more frequent in boys than girls, although all statistics do not point this way, whence it may be concluded that the numbers in each sex are nearly equal.

Morbid Anatomy.—The cortical lesions described as causing the cerebral palsies of children and some resulting from trauma are found in connection with most cases of Jacksonian or focal epilepsy. *Tumors*, especially those involving the motor layer of the cortex, are among these causes; so are localized *syphilis*, *pachymeningitis*, and *tyroma* or tuberculous tumor, and sometimes tuberculous meningitis, pointed out by J. Hendrie Lloyd. Lloyd would, however, exclude the gross deformities, such as porencephalia; and diffuse processes, such as lobar sclerosis, which manifest themselves by idiocy and arrested development, and are not infrequently provocative of epileptic seizures.

Sclerosis of different parts of the brain and medulla oblongata is also found in cases of epilepsy. This is especially true of the hippocampus major, this being probably a conspicuous local focus of a more diffuse lesion. Similar sclerosis is sometimes found in the cerebellum. A *nuclear degeneration* and vacuolation of the cells of the second layer of the cortex has been claimed by Bevan Lewis as a distinctive lesion of epilepsy. Many cases of so-called idiopathic epilepsy are still without a demonstrable morbid anatomy.

Mechanism of the Convulsion.—The epileptic seizure itself is regarded in the light of our present knowledge as an explosion or *discharge of nerve force*, the seat of discharge, in the severe seizures, at least, being the large motor cells in the deeper layers of the cortex, the function of which is to store up and discharge nerve force. The same mechanism exists in sensory and psychical epilepsy. The explanation is not entirely satisfactory.

Symptoms.—These vary in the four varieties known as grand mal, petit mal, Jacksonian, and psychical epilepsy.

1. *Grand Mal.*—In a large number of cases the epileptic attack is preceded by what is known as the *aura*, a peculiar sensation which differs greatly in different individuals. Occasionally it is like what the word literally means, a breath of air, which starts from a particular part of the body, as the extremities or a single finger or toe or a part of the surface of

the body, such as the nieghborhood of the stomach or the heart. At other times the aura is a simple epigastric sensation, a sense of discomfort or uneasiness emanating from the stomach or the feeling of a ball arising therefrom, and this is not a very uncommon form. It may be a flash of light, which may be of different colors; an object, as a face or faces, and even a coffin—as in one of M. Allen Starr's cases. Auditory auræ are manifested through the sense of hearing, and may be subjective sounds of any kind, including musical tones or even voices. Gustatory and olfactory auræ include subjective tastes and smells, mostly of an unpleasant character. Auræ are represented also by tingling, numbness, or simple flushing or chilliness anywhere in the body. "Intellectual auræ" so called by Hughlings Jackson, are certain mentla conditions, such as the "dreamy state," and the consciousness of a certain algebraic formual, which always presented itself to a patient of Starr.

In other cases there is a more prolonged *prodrome*. For several hours or for a day the patient may be the subject of sensations. He may feel generally miserable, dispirited, timid, irritable, or dizzy, or he may be pale or quiet, and wait patiently for the dreaded event, known to him rather by its consequences than its phenomena, of which he is unconscious. There is pathetic sadness often in this patient expectation. *The aura is by no means always present*, indeed, perhaps in the majority of cases of epilepsy there occurs no warning of the attack. The aura may be substituted by certain movements, such as running rapidly for a few minutes either forward or in a circle—the so-called *epilepsia procursiva*—or the patient may stand on his toes and rotate with great rapidity.

Following the aura or independent of it occurs the *convulsion* or "*fit*," of which the initial event is often the *epileptic cry*. This is succeeded by the *fall* which may be sudden, as if the patient were shot, while serious injury may be a consequence. Following this the phenomena of the fit may be quite sharply divided into *three stages*, that of tonic spasm, of clonic spasm, and of coma.

(*a*) *The Tonic Spasm.*—In this the head is drawn back or to the right or left and the jaws are fixed; the arms are flexed at the elbow, the hand is flexed at the wrist, and the fingers are clinched into the palm, while the legs and feet are extended. The muscles of the chest are involved and respiration is suspended, and the face becomes dusky, livid, and swollen, contrasting with the initial pallor. The muscles of the two sides are not equally affected, so that the neck is twisted and the spine curved. This stage lasts but a *few seconds* and is succeeded by clonic spasm.

(*b*) *The Clonic Spasm.*—Now the muscular contractions become intermittent. At first tremulous and vibratory, they soon become strong and general, until the arms and legs are thrown about in the most violent manner, sometimes so violently as to produce dislocation, usually of the shoulder. The muscles of the face are also involved in distorting contractions, while the eyes roll and the lids open and close. The jaw muscles contract violently and the tongue is apt to be caught and bitten. A frothy saliva, often blood-stained, escapes, and the patient is said to "froth at the mouth." There may be voluntary discharge of feces and urine. The lividity supervening in the first stage diminishes somewhat during this stage. The temperature

rises 1/2° to 1° F. (0.28° to 0.55° C.). Very soon the contractions become less violent, finally abate, and this stage terminates, in *one or two minutes*, in the stage of coma.

(*c*) *Coma.*—In this the limbs are relaxed and there is profound unconsciousness, but the breathing is noisy and stertorous. The face remains congested, but is no longer cyanotic. The patient may, after a time, be aroused, but if left alone, commonly sleeps several hours, awaking after a time in a remarkably natural state, feeling bruised and aching, but otherwise quite himself; or there may be some mental confusion and even headache.

These are the phenomena of the attack in the vast majority of cases of grand mal. There may not be another attack for several days or a month or more. In severe cases, on the other hand, there may be daily recurrence, though not until the disease has lasted for several years. In a few instances the attacks may follow one another in rapid succession without a return of consciousness, lasting from 12 hours to a day or more, producing the *status epilepticus*, in the course of which the patient may die from exhaustion. In this state there is often decided fever. In some instances the Jacksonian form of epilepsy may appear as status epilepticus.

After the attack the *reflexes* may be increased and ankle clonus may be obtained; at other times the reflexes are absent. The urine is also often increased, and a small amount of albumin is quite common after the fit. There is also sometimes an increase in the amount of uric acid in the urine *after* the convulsion in *grand mal*.

Inequality of pupils (anisocoria) has been considered a symptom of epilepsy. This symptom,[1] however, occurs in healthy individuals, and too much value should not be attached to it.

2. *Petit Mal.*—The symptoms in minor attacks vary somewhat, but commonly the patient stops in the midst of what he may be doing, the eyes become staring and fixed, the pupils dilated, the countenance pale, there may be some twitching of the facial muscles or the limbs, and consciousness is lost, but there is no convulsion. Anything that is in the hand may be dropped, but in a minute or two consciousness returns and the patient resumes what he has been doing as though nothing had happened. Here, too, though rarely, there may be auræ of various kinds and even an epileptic cry; also forced movements—procursive epilepsy. There may be dizziness without unconsciousness, and the patient may fall. An increase of uric acid in the urine is said also to be quite frequently associated with this form of epilepsy. As the disease continues the attacks of petit mal generally become grand mal, or the two forms of attack may alternate.

3. *Jacksonian or Partial or Focal Epilepsy.*—In this, *consciousness is retained*, though it is thought by some that there is always a momentary period of unconsciousness while convulsions occur, though circumscribed to a single group of muscles or to a single limb. It is almost always symptomatic of some focal lesion in the cortical motor area, which may be a tumor, an injury or inflammatory process in the membranes or brain substance, softening, hemorrhage, abscess, or sclerosis. It is especially likely to be a sign of a growing tumor. Hence it is also called symptomatic epilepsy. Previous to the twitching there may be a numbness or *tingling in the part to*

[1] See a paper by Wendell Reber, "The Pupil in Health and Epilepsy," "Med. News," August 24, 1895.

be involved, which has been called the "signal symptom" by Seguin, because it ushers in the attack. It may remain during the attack, and is of value in determining the seat of the lesion, and therefore the place for operation. Its seat is usually the same in the same patient in all the attacks.

The spasm or convulsion begins uniformly in one part—it may be the face, the thumb, the toes—thence slowly invades an entire limb. It continues sometimes for three or four minutes or longer. The movement is tonic and clonic, extending from the part in which it begins to other parts in a definite order of extension. Thus, if it begins in a part of the face, it extends thence to the whole face, then to the shoulder, arm, forearm, and hand, and possibly the leg from the trunk down to the toes; or it may start in the fingers and go in the opposite direction. Jacksonian epilepsy also occurs in uremia and progressive paralysis of the insane, and it has already been spoken of as following the hemiplegia of children. After the convulsion, the parts convulsed and especially that in which the spasm begins may be partially paralyzed and awkward in movement, and quite often the numbness and palsy continue for some time, with a moderate degree of tactile or thermal anesthesia. More rarely this paresis is permanent, when it is evidence of changes in the cortex such as may be caused by a growing tumor. The opposite side of the body may also be affected, and if this occurs, consciousness may be finally lost.

4. *Psychical Epilepsy.*—This occurs either as a later symptom following the more common forms of *grand mal* and especially *petit mal*, or as an independent state or as what is known as a "psychical epileptic equivalent," where the usual seizure is substituted by a somnambulistic state in which the patient performs various acts, sometimes of great complexity, including driving, walking, and the like, of which he is totally oblivious after he passes into the natural condition. Some striking instances of psychical epileptic equivalent are related by M. Allen Starr in his book on "Familiar Forms of Nervous Disease." Other manifestations of psychical epilepsy are represented by violent maniacal excitement and uncontrollable violence, in which criminal acts, including even homicide, are committed. See also under Prognosis.

Relative Frequency and Time of Attacks.—The major form of attack is the most frequent, after this, mixed forms of major and minor, and then minor and Jacksonian; the most infrequent are the psychical forms.

Two-thirds of the attacks occur between 8 A. M. and 8 P. M.; many attacks occur early in the morning after awaking, some between 3 and 5 A. M., and others in the night at unknown hours—nocturnal epilepsy. In true epilepsy the patient generally feels perfectly well between the attacks—indeed, he not infrequently feels better for a time after the spell.

Diagnosis.—The epileptic fit is of itself in no way characteristic of the disease. The uremic convulsion is identical, as is also the reflex convulsion due to teething and other causes. Even hysterical convulsion closely resembles it, but there are points of difference. Something more, therefore, than the convulsion is necessary to prove the presence of the disease. The *aura* is distinctive, and when present, is almost conclusive. Scarcely less so is the *epileptic "cry"* although it is less constant than the aura. The *relaxation of the sphincters* belongs rather to the epileptic fit, while the bitten tongue,

the dilated pupil, and sudden unconsciousness belong to uremia as well; and it is from *uremia* that it is most important to distinguish epilepsy. The occurence in the midst of apparent health of a convulsion with the features described, followed by prompt recovery without albuminuria or casts, can hardly be anything but epilepsy. Here the use of the phthalein test described in another place will help to make the diagnosis. At other times, when other signs of Bright's disease are absent, it may be necessary to defer the diagnosis a little longer in order to examine the urine. Finally, epileptics may have *Bright's disease*, when errors are still more likely and sometimes unavoidable.[1]

The *reflex convulsion* in children is likely to be repeated until the cause is removed, and in this respect the condition resembles the *status epilepticus*, but in the former a little careful searching will probably discover the cause. The isolated reflex convulsion may be more difficult to account for at first, but in these cases immediate decision is less important. The very short duration of the *petit mal* separates it sharply from the uremic fit. Nocturnal convulsions, occurring as they do often without the knowledge of the patient, are usually epileptic.

The *hysterical convulsion* sometimes simulates closely the epileptic. But the hysterical patient rarely loses consciousness completely, the fall is not so sudden, the victim rarely if ever hurts herself, and never bites her tongue; nor is there any rise of temperature, while even the pulse and respirations commonly remain quite normal. There is rigidity, but it is unlike that of epilepsy—it is not more conspicuous in the beginning of the attack. Opisthotonos, or arching of the back, does not occur in the epileptic convulsion. Finally, the hysterical convulsuon is of longer duration, lasts 10 minutes or more, while the duration of the epileptic fit is not usually more than three or four minutes.

The *petit mal* is most frequently mistaken for *fainting*, but after two or three occurrences it should be recognized. The vertigo of *Ménière's disease* and of attacks of *indigestion* resembles it, but in the former there is deafness, while in neither is there actual unconsciousness, as is always the case in *petit mal*.

Jacksonian epilepsy is *sui generis* and is not simulated by any except the rare instances of *localized uremic convulsions* and similar spasms in *general paresis;* however, it has been described as occurring in hysteria. A further study of each instance must quickly dissipate the error. While the approximate *seat* of the lesion may be inferred in many cases of Jacksonian epilepsy, the precise *cause* cannot generally be determined, because all sorts of lesions produce the same symptoms. Recurring epilepsy in persons over 30 is probably due to organic causes, and in nine cases out of ten, according to H. C. Wood and also Fournier, is due to syphilis.

The highest refinement of diagnosis in the study of epilepsy attempts to determine from the character of the aura the seat of beginning cortical irritation. Thus, a visual aura, it is claimed, might indicate that the nervous discharge began in the occipital lobes; a vertigo might indicate that it began in the cerebellum; a sense of numbness, the sensory area of the

[1] See two interesting cases reported by me in the "Transactions of the Association of American Physicians," vol. vi., 1891.

cortex. The "intellectual auræ," as they are called by Hughlings Jackson, are regarded by him as affording evidence of a nervous discharge from the highest cerebral centers.

Prognosis.—The true epileptic rarely gets well. Tyson has seen two cases of recovery in his experience. In such statement epileptiform attacks due to peripheral irritation are rigidly excluded as not being true epilepsy. These invariably get well with the removal of the irritation, while true epilepsy, in which attacks are readily excited by such irritation, is benefited but not cured. The chances of recovery are said to be greater in the young, and in the male sex than in the female. One of the cases of apparent recovery was a man who had his last fit after 40; the second a woman who had no attack after 14. Both live. C. L. Dana places the recoveries at from five to ten per cent., which appears to me large. Even in cases of combined petit mal and grand mal, in which the prognosis is most unfavorable, recovery is said to occur. The prognosis of petit mal is more unfavorable than that of grand mal; of the mixed forms still more unfavorable, and posthemiplegic epilepsy most unfavorable of all.

On the other hand, an epileptic rarely dies of his disease. He may fall in the water during an attack and drown, may choke to death if attacked while eating, or may be smothered by the bed clothes. Death sometimes occurs from exhaustion in the *status epilepticus*, but this is not frequent. The health of epileptics usually deteriorates slowly, and life is shortened accordingly, few surviving the age of 40 or 50. They rather frequently die of tuberculosis. Especially frequent is mental deterioration; indeed, it may be said to be the rule when the patient lives long enough, and about ten per cent. become demented or insane. Changes begin with loss of self control succeeded by confusion of intellect. Delirious and passionate outbreaks precede and follow the convulsive seizures during which criminal acts are committed. Much may be done by treatment to control the number of attacks, and the less numerous they are, the less serious is the effect upon the health. Many epileptic persons earn a living, and more could if properly helped.

The more infrequent the attacks, the better the prognosis. Pure nocturnal epilepsy and the pure diurnal form are each more easily cured than the mixed forms. Cases, too, which arise after 20 years of age are more likely to get well.

Treatment.—No fact in therapeutics is better established than that the *bromids* control epilepsy in varying degree—it may be completely, it may be simply to render infrequent the seizures. There is probably no important difference in the efficiency of the various preparations, but the bromid of potassium has been most extensively used. The bromid of sodium is preferred on account of its greater solubility. Bromid of ammonium is slightly more stimulating. More recently bromid of strontium has been highly recommended. Causes of *peripheral irritation* should first be sought, and if possible *eliminated*. Gastro-intestinal irritation should be removed. Phimosis should be cured. The possible practice of masturbation should be inquired into. These eliminated, the bromid treatment may be commenced. The doses required vary greatly and must be determined by trail. Scarcely less than 15 grains (1 gm.) four times

a day are required for adults, and from this point the dose may be increased until the desired effect is produced. The massive doses sometimes given, amounting to ounces in a day, are ultimately harmful, but doses of a dram (4 gm.) are sometimes necessary and well borne, but if long continued are likely to produce bromism. It is sometimes of advantage to combine the various bromids of sodium, potassium, and ammonium. Greater efficiency is secured if the drug is given on an empty stomach, half and hour before meals or two hours after, and smaller doses suffice when thus administered, and the omission of sodium chlorid from the diet is believed to lessen the amount of bromid necessary and to increase its efficiency. Bromism, shown by drowsiness, mental torpor, gastric and cardiac distress with acne, sometimes results. It is doubtful whether it can be obviated in any way except by omitting the drug. The bromid eruption may sometimes be averted by combining arsenic, but this does not always suceed. In a few cases the bromids are absolutely useless, more especially in cases in which they produce gastro-intestinal derangement, perhaps in about five per cent. of cases. *Chloral* adds to the efficiency of the bromids, and is sometimes necessary to produce the desired effect. It may be given in doses of from 10 to 30 grains (0.66 to 2 gm.).

Of late dechloridation or elimination of chlorine from the food has been recommended as an adjuvant to the treatment of epilepsy by the bromids. It is accomplished by substituting sodium bromid for sodium chlorid in the food of epileptics. Sodium chlorid is not wholly eliminated but enough bromid is added to the food to make each patient take about 15 grains a day. It is claimed that the bromid is rapidly absorbed and becomes part of the body tissue when thus given, and that only one-half the usual quantity is necessary to produce the sedative effect.

Flechsig claims for the associated use of opium and bromid superior results, but the possibility of an opium habit will restrain the cautious physician from adopting its use.

To treatment by the bromids should, of course, be added proper hygienic measures. Suitable food, especial attention to the bowels, fresh air, and outdoor life are indispensable. It is of importance that the patient be put upon milk diet at first—later, the diet can be carefully adapted to each individual case. Bathing is important, and cold baths—particularly douches and shower-baths, cold sponge-baths or wet packs should be judiciously used. Vasomotor tone and circulation are thus strengthened.

Of other remedies recommended may be mentioned *antifebrin* and *antipyrin*. A trial of the former in the Vanderbilt Clinic in New York, by M. Allen Starr, was unsatisfactory. On the other hand, in the hands of Charles S. Potts, at the Dispensary of the University of Pennsylvania, it was apparently useful. Especially efficient at the latter proved a combination of antipyrin and bromid of ammonium, suggested by H. C. Wood. For adults a dose of 6 grains (0.39 gm.) of the former and 10 grains (0.66 gm.) of the latter, three times a day, in a number of cases averted the seizure for months. Continuous exhibition seems necessary. These drugs at least merit a trial where the bromids are for any reason unsatisfactory. *Monobromated camphor* has been recommended by Hasle. The best mode of administration appears to be in a capsule or emulsion, the dose being 2 to 5 grains (0.13 to 0.32 gm.).

Starr has also used the *tincture of simulo* (*Capparis coriacea*) at the Vanderbilt Clinic with the effect of reducing the number of attacks in *grand mal*, but to no purpose in petit mal. It was used in doses as large as 1/2 ounce (13.5 c. c.) daily. In *petit mal* the same observer found *nitro-glycerin* the only remedy of any service. He appears to have used it in doses of 1/100 grain (0.00065 gm.) three times a day. In my experience this regulation dose fails in a large number of cases to produce the physiological effect, and the larger doses—from 1/50 to 1/25 grain (0.0013 to 0.0026 gm.)—may be given. It is to be remembered that epilepsy is one of the diseases which are nearly always influenced for a time by new remedies. The preparations of *valerian* may also be tried in the event of failure with the bromids. Others which have been used are borax, iodid of zinc, and sulphonal. Chloretone is sometimes of great service in doses of 3 grains once or twice daily.

The *nitrite of amyl* has been employed to abort the attack in cases where there was an aura, and in a certain number of cases—about 25 per cent. in Starr's experience—has proved efficient.

Operation, usually trephining, is increasingly practised, and many successful cases have been reported, chiefly of Jacksonian epilepsy. When a well-defined lesion can be located, operation should promptly be done. Even in doubtful cases operation may be justified, as with modern surgical precautions it is attended with much less risk. It should be remembered, too, that operation, *per se* has proved curative—that is, cases have apparently recovered after trephining where no lesion was found after removing the disk, and that practically all cases are temporarily improved.

Food should be simply and easily assimilated, overeating should be especially avoided. Stale bread, wheaten grits, and similar foods, rice, potatoes, fresh succulent vegetables like string beans, peas and tomatoes, with an abundance of milk, are suitable. Water should be freely drunk by the patient, and a glassful is advised between meals and at bedtime. Constipation must be avoided.

Asylum Provision.—It is exceedingly important that some systematic provision should be made for epileptics either by the State or by private charity. They are, as a rule, unwelcome inmates of hospitals because of their incurability and the disturbance they occasion. Doubtless the neglect to which they are subjected at home aggravates in many instances their condition, while it makes even their lot more unhappy. Provision should be made to enable them to pursue some vocation, the tendency of which has been shown to be curative. A hospital with such provision has been inaugurated near Philadelphia, and similar institutions exist in some other States. The mind should be kept occupied; nothing is more baneful to the epileptic than idleness, and it is said that cures have been effected by giving the patient something to do.

Treatment of the Convulsion.—Of no small importance is the treatment of the eclamptic attack. The first measure is to secure protection against biting the tongue. Unfortunately, this is often the initial event in the convulsion. The end of a towel may be twisted and inserted between the teeth, or a suitable piece of wood or a clothes-pin may be similarly used. A small object like a cork is unsafe, as it may be swallowed or drawn into

the larynx and cause death by suffocation. Some patients carry such an appliance ready for use. The patient should be controlled sufficiently to protect him from injury.

Given a case that under the bromids has yielded to treatment, what course shall be pursued as to its interruption? The most experienced clinicians urge that the drug should be continued at least two years after the fits have disappeared, and Seguin even advises that there should be no reduction in the bromids until three years have elapsed without symptoms. My own practice has been to continue a dose of from 15 to 20 grains (1 to 1.32 gm.) at bedtime for an indefinite period after cessation of the fits. The friends of the patient should be impressed with the importance of such a course, as he himself is almost sure to grow indifferent after the long absence of attacks.

REFLEX CONVULSIONS OF CHILDREN.

SYNONYMS.—*Infantile Convulsions; Epilepsia Acuta.*

Definition.—Convulsions in children due to peripheral irritation.

Etiology.—Eichhorst says: "Epileptiform convulsions, which have the same genesis as true epileptic attacks, are excited by irritation of the cortical motor brain areas." He then names, among the causes of these, toxic agencies, including uremia and lead-poisoning, but also says he will treat only under eclampsia of the convulsions of infants (5 to 20 months), among the causes of which he names heredity; psychical causes, as fright or anger; but most frequently reflex irritation, as of the skin or gastro-intestinal tract (dentition, intestinal worms, inflammation, and the like); foreign bodies; fecal accumulation; stone in the bladder, etc; and finally, the infectious fevers and rickets. This class of cases we have taken great pains to exclude from the epilepsies, and prefer to include at present under the heading of Reflex Convulsions of Children. The convulsions which attend diseases of the brain are a part of the symptomatology of these affections, and do not require separate consideration.

Debility and malnutrition may be considered as predisposing causes of the form of convulsion under consideration.

Symptoms.—These demand no detailed consideration, since the convulsion is epileptiform and has been described. It is much oftener partial than the typical fit of true epilepsy, but it has the same stages of the tonic and clonic spasm followed by drowsiness. It is most frequently single, but the fits may follow one another in rapid succession, and though rarely, terminate fatally. As in epilepsy, the temperature rises slightly during the fit. It may come on suddenly without warning, or be preceded by restlessness and fever. It not infrequently occurs during sleep.

Diagnosis.—This is usually easy, the convulsion coming on suddenly in the midst of health, yet traceable to some such event as the ingestion of some indigestible food, to teething, or to some other source of peripheral irritation.

The convulsion is distinguished from that of *infantile hemiplegia* by

the absence of hemiplegia. A transient paresis does, however, sometimes follow the reflex convulsion.

These convulsions most frequently occur between the fifth and twentieth months, and toward the end of the second year, though they may occur as late as the fifth year. Convulsions occurring after this period are more likely to be true epilepsy.

Prognosis.—Cases of infantile convulsions are always alarming, yet most get well, and doubtless many cases among the poor recover which do not come under the notice of the physician. On the other hand, not a few deaths are caused by them—according to Morris J. Lewis, 8.5 per cent. of all deaths in children under 10; and according to West, 22.35 per cent. of all who die under one year. Cases of infectious disease ushered in by convulsions are almost always serious, but the convulsions themselves are rarely fatal. Convulsions due to gastric derangement are generally followed by recovery.

Treatment.—The first step in the treatment must always consist in finding and *removing the cause*. If it be undigested food, an emetic and an enema indicated; if dentition is at fault, the lancet should be promptly applied to the gums. The next step is immersion in a *warm bath*, at 95° F. (35° C.), increased to 100° F. (38.8° C.), to which mustard may be added. At the same time *cold* should be applied to *the head* by means of an ice-bag or cold water. To control the convulsion, *chloral* is the remedy *par excellence*, but while waiting for its effect, it may be necessary to permit the child to inhale a few drops of chloroform. The dose of chloral should be sufficient—2 1/2 to 5 grains (0.165 to 0.33 gm.) to a child of one year, frequently repeated until the effect is produced. It may be given in enema in double this dose, the buttocks being compressed until it is absorbed. The *bromids* may be given in combination with chloral, but they are altogether too feeble to be relied upon alone. Should these measures fail, *opium* may be used and even morphin, hypodermically, in minute doses; but these drugs should be used only as a last resort. Generally, the attack is relieved the moment the peripheral irritation is removed.

MIGRAINE.

SYNONYMS.—*Sick Headache; Bilious Headache; Hemicrania; Megrim; Migran; Paroxysmal Headache.*

Definition.—Migraine is an intermittent, sensory neurosis, of which headache, commonly hemicranial, is the most invariable symptom. Almost as constant are aggravated nausea and vomiting, to which may be added other sensory symptoms, especially deranged vision. Ophthalmoplegic migraine is a rare form, in which paralysis of ocular muscles occurs.

Etiology.—This is obscure. The disease is more common in females—apparently three times as frequent as in males. It begins early in life, commonly at puberty, and even earlier—as early, in fact, as at two years, It affects vigorous and strong as well as nervous and anemic subjects. Exciting causes are fatigue, mental and physical, including eye-strain,

digestive derangements, and menstrual disorders. What is known as the uric acid diathesis plays an undoubted rôle in certain cases. As often none is discoverable.

It is usual to speak of migraine as a vasomotor disturbance, because there are symptoms which point to involvement of the sympathetic system, but this is a matter of inference rather than demonstration. The attacks are characteristically paroxysmal. It appears to be more frequent in the winter season in this climate, when it is not infrequently associated with a gouty or rheumatic attack. Caries of the teeth and nasal troubles are a cause in children.

Morbid Anatomy.—No lesions other than those described as causal are found. The precise seat of the pain is not known, but is believed to be in the meninges of the brain.

Symptoms.—The attack is often ushered in without any warning, at others with *prodromal* symptoms familiar to the patient. They are various and not distinctive of the disease, but so characteristic for each case that the individual foretells the attacks on their approach. They include general discomfort, vertigo, a sense of pressure, tinnitus, spots before the eyes, chilliness, and the like. Hemianopia and scotoma may be among them.

Then the *pain* starts in suddenly and is continuous, usually in one side of the forehead, but it may also be in the occiput, whence it extends to the half or whole head. It is extremely severe, sometimes described as blinding, at others sharp and boring or shooting. It is sometimes attended by flashes of light. Light and noise aggravate it, and a darkened room is always sought. Hemianopia is not infrequent.

Along with pain there is generally total want of appetite, and intense *nausea* succeeded by *vomiting*. The vomited matter includes first the contents of the stomach (if the stomach is empty, mucous matter), and later yellow and bitter bile, whence the term "bilious headache." If the stomach happens to be full, the pain may be relieved by the vomiting.

The *vasomotor* symptoms are conspicuous in some cases, and are assigned by some an important rôle in the causation of migraine. From this standpoint two subdivisions are made, *angiospastic hemicrania* and *angioparalytic hemicrania*. In the first form, described by Dubois-Reymond from observations on himself—some of the best descriptions have been by sufferers—the forehead and ear on the affected side are pale, the skin is cool, the temporal arteries are contracted, the *pupil is often dilated*, and the secretion of saliva is increased—in a word, there are the symptoms of irritation of the sympathetic. In hemicrania angioparalytica, described by Möllendorff, also from observations on himself, the face is reddened on the affected side, it feels warm, the temporal arteries are dilated and pulsate strongly, there is sometimes unilateral sweating of the face, with the *pupils contracted*—symptoms suggestive of paralysis of the sympathetic. By no means all cases are capable of being thus classified, and mixed forms are met.

The frequency of the attacks varies greatly; usually they do not occur oftener than once in two weeks or once a month. They may, however, occur every ten days or even weekly.

The duration of the attack varies. Very often the patient goes to bed at night, and in the morning, or at the end of 12 hours, is relieved; or the attack may last 24 hours or even two or three days. The attacks continue over a period of many years, sometimes ceasing in women after the climacteric is passed, and in men after 50.

Further speculation as to the true nature of migraine would be unprofitable here, though mention should be made that the arteries on the affected side sometimes become the seat of arteriocapillary fibrosis, a condition giving some force to the view of vasomotor origin.

Diagnosis.—The symptoms of *brain tumor* sometimes closely simulate migraine. Ophthalmoscopic examination may discover papilloedema in cases of brain tumor and thus settle the diagnosis. Such examination should always be made.

Prognosis.—This is favorable so far as life is concerned, but it is not always easy to prevent the attacks or diminish the frequency of their occurrence. It often happens that they cease after middle life.

Treatment.—Before treatment is instituted every case should be thoroughly investigated with a view to discovering causal conditions. Should such search be successful, their elimination may result in a cure. Such accessible causes are eye-strain, affections of the nose, mental and physical fatigue, and indiscretions in diet.

The attack itself is more likely to be warded off the earlier the treatment is instituted. Sometimes a dose of salts, taken as soon as the first symptoms appear, wards off an attack, or the attack may be relieved by vomiting. *Acetphentadin* in from 10 to 15 grain (0.66 to 1 gm.) doses relieves some attacks. Byrom Bromwell in a very impressive paper recommends 30 grain (2 gm.) doses of this drug as curative. After the first dose it may be continued in smaller doses. *Antipyrin* and *antifebrin* are similarly successful, and I am informed by apothecaries that many women purchase these drugs regularly to relieve their attacks. Such practice should, however, be discouraged.

Sometimes a hypodermic injection of *morphin*, even so small a dose as 1/8 grain (0.011 gm.), acts magically, and on the whole it is the most reliable remedy. This, however, should practically never be given, better attacks of migraine which are painful but which do not kill, rather than a morphin habit which is death dealing. *Caffein* is a less efficient remedy, but may be used in conjunction with morphin or immediately after it to counteract the unpleasant effect of this drug. It may be given in 3 to 5 grain (0.2 to 0.33 gm.) doses, and is sometimes administered hypodermically in the shape of caffein-sodio-benzoate in the same dose. *Salicylate of caffein* is also recommended in like doses. *Cannabis indica* is a remedy much recommended, but is unfortunately of uncertain strength. We may begin with 1/4 grain (0.016 gm.) and increase rapidly. *Bromids* may be tried. *Guarana* is more efficient in from 30 to 60 grain (2 to 4 gm.) doses of the powder and similar doses of the fluid extract.

If the spastic form can be distinctly recognized as present, *nitrite of amyl* may be expected to be serviceable—3 to 5 drops by inhalation. In the opposite or paralytic form *ergot* has been advised, and may be given in doses of from 10 minims to 1 dram (1 to 4 gm.). *Nitroglycerin* in doses of

from 1/100 to 1/50 grain (0.00066 to 0.0013 gm.), and *nitrite of sodium* in doses of from 3 to 5 grains (0.2 to 0.33 gm.), may be useful in the class of cases benefited by nitrite of amyl. *Cold* to the head is sometimes grateful, and when there is nausea, *cracked ice* or cold carbonated or apollinaris water or small doses of iced champagne are sometimes efficient.

Electricity is said to have been useful in a few cases. It is recommended that in the spastic form the anode should be applied to the sympathetic, and in the paralytic form the kathode, the other pole being applied to the cervical cord as high as possible on the occiput.

Preventive Treatment.—General treatment between attacks should not be neglected. When there is anemia, the judicious use of *iron* and *arsenic*, continued for some time, has occasionally been followed by a disappearance of the tendency to the disease.

The urine should be carefully examined, and if concentrated and tending to deposit uric acid or oxalates, diluents and the alkaline mineral waters are indicated. In a few instances in the daily use of natural Vichy water, to the extent of a bottle a day, had the effect of diminishing, and in one instance of eliminating, the attacks. The conditions of a healthful life, bathing, fresh air, and simple wholesome food, should be observed. Many persons are totally free from attacks while traveling. A course at Contrexville, Vichy, or Carlsbad may be of service in averting attacks.

OCCUPATION NEUROSES.

SYNONYMS.—*Professional Spasm; Copodyscinesia.*

Definition.—A term applied to a group of diseases characterized by symptoms excited by an effort to perform some oft-repeated muscular act, commonly one involved in the occupation of the patient. The most usual symptom is cramp or spasms in the muscles concerned, whence this word is preceded by that of the various occupations, to indicate its special variety. Thus we have writer's cramp or scrivener's palsy, telegrapher's cramp, pianoforte-player's cramp, typewriter's cramp, seamstresses' cramp, milker's cramp, etc.

WRITER'S CRAMP.

SYNONYMS.—*Graphospasmus; Cheirospasmus; Mogigraphia; Scrivener's Palsy.*

Definition.—The professional neurosis of clerks and scriveners. It is the most frequent of the occupation neuroses and may serve as the type for all.

Etiology.—There is no predisposition to sex, the disease being more frequent in men in occupations where more men are employed, and more frequent in women in occupations where more women are employed; and it is likely that since an increasing number of women have become telegraph operators, more cases may be expected among them, in whom, perhaps, also, the neuropathic temperament may favor it. The majority of all cases occur between 20 and 50—154 out of 177 cases collected by Berger from Gowers, Poore, and his own cases. Predisposition is caused

by previous injury and a neurotic disposition, while even heredity is said to predispose. An especially important factor is a faulty method of writing, while cases have occurred which were apparently independent of the usual exciting cause. Steel pens are said to be responsible for an increased number of cases since their introduction. The disease is becoming less frequent as clerical exactions grow less.

Morbid Anatomy and Pathology.—No distinctive anatomical changes have ever been discovered in writer's cramp. Three theories are held regarding its nature. According to the *first*, it is essentially a local disease: weakness in certain muscles permitting overaction on the part of their antagonists, an overaction which increases to spasm. According to a *second* theory, the spasm is reflex and due to an irritation of the sensory nerves concerned in the act of writing. The *third*, and usually accepted theory, makes the affection primarily and essentially central, due to deranged function in the centers concerned in the act of writing, and therefore in the central nervous system.

The only discoverable mobid change is an occasional atrophy of muscles concerned.

Symptoms.—*Spasm* is almost always the initial disturbance, commonly affecting the forefinger and the thumb; but the onset is gradual, and the first effect is an awkwardness in which the pen does not move quite as intended. It is irresistibly grasped too tightly, yet the forefinger has a tendency to slip off, the pen passing between it and the middle finger, while an attempt to mend matters by taking a new hold only increases the difficulty, and the hand labors as if tied down. It feels tired, and there is often an aching pain throughout, extending even to the arm. The writing is irregular and uneven. These symptoms may continue, with more or less difficulty in writing, lasting for weeks or months, coming earlier, however, after each effort, and with gradual increasing severity until the intolerable spasm sets in. This may be so violent in a combined movement of flexion and adduction in the thumb that the pen may be wrested from the grasp and thrown to a distance, or there may be a lock spasm, described by S. Weir Mitchell, in which the pen is firmly locked between the fingers. The spasm is almost always tonic in character, although it may now and then be varied by a slight start or jerk. It is sometimes associated with *tremor*. Rarely tremor occurs alone, and it may be the premonitory symptom of *atrophy*. The spasm may be limited to the act of writing, while other actions are well performed; but absolute limitation to this act is seldom met in severe cases.

Special difficulty attends the performance of acts requiring delicate co-ordination of the muscles. Sometimes a patient can write with a pencil, but not with a pen. *Paresis* and *paralysis* may occur with spasm or alone. On the other hand, the strength of the hand may be quite unimpaired. Such loss of power varies greatly, being sometimes trifling, at other times considerable.

Sensory symptoms are almost always present in various degrees. They may even exist alone, producing a sensory form. They are manifested at first by the distressing fatigue alluded to, or by dull pain often referred to the bones or joints, very often to the metacarpal bones or to the wrist,

ceasing with cessation of writing. Sometimes there is local tenderness or a tingling sensation. Again, the pain is more severe, neuralgic in character, and distributed along the course of the nerve, induced at first by the act of writing, later by any muscular act of the part. There may also be tenderness in the course of the nerve.

Vasomotor disturbances are seen in severe cases, manifested by hyperesthesia, a glossy, shining skin, or a cyanosed, chilblain-like appearance; or the hand may become blue and hot on attempting to write.

In the beginning the *electrical reactions* are normal, but in advanced cases there is a diminished faradic and sometimes increased galvanic irritability of the motor nerve endings ditributed to the muscles. It is to be remembered that the radial, ulnar, and median nerves all supply muscles employed in writing.

Diagnosis.—This is usually easy, the initial limitation of the symptoms to the act of writing sufficiently indicating the nature of the case. More frequently other paralytic and painful affections of the arm and hand are mistaken for writer's palsy. Among these may be included *hemiplegia* of gradual onset, *commencing insular sclerosis*, early *tabes dorsalis* affecting the arms, or *pressure palsy of the musculo-spiral nerve*.

In most of these cases, however, other symptoms are present or are soon added. More frequently nervous persons *imagine* they have writer's palsy. In some cases the condition is really one of *myositis*.

Prognosis.—A well-established case of scrivener's palsy rarely gets well. There are, however, exceptions, even under the most unfavorable conditions. The prognosis is more favorable when sensory symptoms predominate. Relapses are prone to occur when the patient returns to work.

Treatment.—*Prevention*, as usual, is much more effectual than curative treatment. The disease is confined almost exclusively to those who write in a cramped manner, and is said to be unknown in those who write from the shoulder. The curative treatment consists essentially in *rest* promptly adopted—a long rest being often sufficient to effect a cure, while no other treatment can take its place. Various *mechanical devices* to aid in writing while the cure is going on have not accomplished much, and the patient may learn to write with the left hand, although the disturbance may occur in his hand also. Typewriting is, as a rule, as easily learned with the affected hand as before disability and should be practiced. The devices referred to may be such as a very thick penholder which can be directed by the whole hand; or a pen attached to a ring, which is slipped over the index or middle finger, and the thumb is thus permitted to rest. The typewriting machine has, however, rendered all such devices of less consequence. The usual nerve tonics, such as strychnin, may be given. Hygiene of the part, including hydrotherapy, frictions, especially massage, and sometimes electricity are useful.

The important position assigned by all neurologists to the *electrical treatment* of writer's cramp demands some special consideration, especially as the methods advised are by no means uniform. The preference given to the galvanic current over the faradic is, however, almost unaminous, and unless the latter is especially mentioned, the former is intended. Berger recommends a stabile current—*i. e.*, a current in which the elec-

trodes are not moved about—with the positive pole in the neck and the negative partly in the fossa supraclavicularis, partly on the affected nerves and muscles of the arm; the length of sitting, from five to ten minutes daily, or every other day.

Benedict recommended galvanization along the spinal column, with especial reference to sensitive vertebræ, but also localization of the galvanic current, as recommended by Berger; duration of sitting, three to four minutes, current strong enough to be easily felt. He also found subsequent faradization to the affected muscles useful. Eulenberg also advised galvanization of the muscles affected with chronic cramp and of the involved nerve-trunk with the positive pole. In cases with tremor and rapid exhaustion the negative pole is to be applied to the spinal column and the positive on the peripheral nerve-trunks and muscles affected. Erb advised galvanization of the cervical vertebral column, with ascending stabile and labile currents combined with peripheric galvanization. In several cases it appeared to him that transverse and longitudinal currents through the head were followed by favorable results.

Onimus used an ascending current through the affected arm, with the negative pole in the neck and the positive pole upon the muscles of the forearm, especially the ball of the thumb, in addition to a current of moderate strength along the cervical vertebræ. M. Meyer employed a stabile galvanic current with the anode to the tender spots on the vertebral column when these were present, and the kathode on the sternum. Althouse sought to reach the cervical cord by placing the anode upon the cervical spine and the kathode on the depression between the angle of the jaw and the sternocleidomastoid muscle—a position corresponding to the superior cervical ganglion of the sympathetic. The current should be mild, uniform, and uninterrupted for from three to five minutes at a time. The method should not be reversed. In cases of paresis of certain muscles it is sometimes of benefit to have the patient make voluntary movements of these muscles simultaneously with the closing of the galvanic current applied to the nerve innervating these muscles.

Testimony is united to the effect that the galvanic treatment must be kept up for a long time, even for months continuously, with a current of moderate strength, say a maximum of four milliamperes, and section electrode of about three cm. in diameter.

Faradization is recommended only in cases where there is demonstrable paresis and anesthesia, and then in weak currents. In anesthesia the brush may be used. Erb found that many of his patients were benefited by wearing on the arms, for several hours daily, a simple galvanic element, such as a zinc and copper plate, united by wire, and under it a moist piece of linen.

Gowers has much less confidence in electricity, especially in the spasmodic form of the disease, and is probably right when he says if the patient goes on writing electricity has not the slightest influence on the disease. Tyson's experience with electricity has not been very encouraging.

The position according to *gymnastic exercise* of the arm and hand muscles is scarcely second to that of electricity—indeed, it is preferred by some. Especially efficient appears to be that of a German writing-master, Julius Wolff. The gymnastics are of two kinds: First, active, in which the

patient moves the fingers, hands, forearms, and arms in all directions possible, each muscle being made to contract from six to twelve times with considerable force, and with a pause after each movement, the whole exercise not exceeding 30 minutes, and repeated two or three times daily. Second, passive, in which the same movements are made as in the former, except that each one is arrested by another person in a steady and regular manner. This may be repeated as often as the active exercise. Massage is practised daily for about 20 minutes, beginning at the periphery; percussion of the muscles is considered an essential part of the massage. Combined with this are peculiar lesions in pen-prehension and writing. Priority for this method is claimed by Roman Vigoroux and Th. Shott. The testimony of some of the best authorities in Europe is given in behalf of this method. Poore secured good results by combining gymnastic exercises with the use of electricity. Tenotomy and nerve-stretching have been attempted and abandoned as useless.

HYSTERIA.

Definition.—Hysteria is a morbid state of the nervous system in which may be manifested every variety of nervous symptom due to deranged function of the cerebral, basal, and spinal centers, associated with lowered will-power and exaggerated emotional tendencies.

Etiology.—Hysteria is a disease of civilization and of certain races. It is unknown in the barbarian, and is more rare in Northern races, while the volatile Southern temperament favors its development. Thus, the French and Italians of the Latin race furnish many subjects, while it is rarer among Germans, English and Americans. The disease is also frequent among Hebrews.

The sexual organs of women have been held responsible for hysteria in the female, and the name hysteria is derived from ὑστέρα, a womb; but this conception is erroneous. In males the disease assumes more the form of hypochondriasis, but in them also convulsions, contractures, and paralysis occur. It is found in boys as well as in adult males, especially in alcoholic males. About half of all cases occur in the second decade, especially after puberty, though it may also occur earlier; one-third between 20 and 30; while boy subjects are commonly under the age of puberty. Masturbation is held responsible for many cases in boys. Heredity plays a certain part, while the neurotic constitution especially favors hysteria.

Among the exciting causes are included diseases of the generative organs in women, especially deranged menstruation. Ovarian disease has been held responsible, and tenderness in the ovarian region is undoubtedly a frequently associated symptom, but it is questionable whether this tenderness is of ovarian origin. Association with others similarly affected is an undoubted factor, and it is not unusual for the disease to spread itself from one to a number of girls living under the same roof. Various diseases other than those mentioned also predispose to hysteria. Even local affections, including injuries, may thus operate, and hysterical joint affections may follow trauma of a joint. Strümpell relates an instance of a girl who, from having inhaled smoke, acquired hysterical paralysis of the vocal cords. General disease of an ex-

hausting kind, such as fevers, nervous diseases, functional and organic, act as exciting causes in hysterical subjects. Hysteria is common in prostitutes. Among psychical nervous causes are fright, such as attends a runaway or a fire; an angry scene; the constant operation of trifling mental causes, including worry and anxiety, but such causes act only on a person predisposed to hysteria.

Explanation of Symptoms.—Always a favorite subject of theory and speculation, modern times are as rich as older dates in developing theories in explanation of the varied symptoms of the disease. As the French nation furnishes the largest number of cases of hysteria so the French physicians have given correspondingly of their time to the study of the subject. The first and greatest of these was the great master Charcot who defined hysteria as a psychic malady in which morbid states are excited by ideas, and as an imitator of all sorts of organic disease, so closely associated with "suggestion" that the capability of responding is the test of the presence of hysteria.

Janet of the College of France in his psychological theory of hysteria says "it is a form of mental depression characterized by the retraction of the field of personal consciousness and a tendency to the dissociation and emancipation of the systems of ideas and functions that constitute personality." On the other hand, Babinski of *la Pitie* amplyfying the dictum of Charcot that "to be hypnotizable is to be hysterical" has elaborated a definition of hysteria to the effect that it is a "special state capable of giving rise to certain symptoms that have features of their own. It manifests itself by primary and by secondary disturbances, the former being characterized by this, that it is possible to reproduce them by suggestion and to make them disappear under the sole influence of persuasion." He therefore suggested the name pithatisme, from two Greek words πειθα meaning persuasion and ἰατος, curable. The primary symptoms are anesthesia, paralysis, contractures, crises, mutism, etc. The secondary are those strictly subordinal to the primary. The muscular atrophy in the hysterical person is the type of these.[1] It never appears primarily. Suggestion cannot cause it. It is secondary to hysterical paralyses and never precedes them.

Sigmond Freud of Vienna, during the last 15 years has developed another psychical hypothesis of hysteria whose contention is that "in a normal *vita sexualis* no neurosis is possible" and that he who can interpret the language of hysteria can understand that the neurosis deals only with repressed sexuality."

Seeking from the physiological side to devise a pathogenic theory, "at once comprehensive and satisfying," Sollier of Paris defines hysteria as "a condition of cerebral torpor or cerebral inhibition a special sort of sleep analagous to what has been called vigilambulism."

On the other hand, Dubois of Berne, says "it is useless to make an effort to give hysteria the character of a morbid entity," in other words it is only a symptom, in which he is seconded by Steyerthal.

In an admirable review entitled *Some Modern French Conceptions of*

[1] J. Babinski, Ma Conception de l'Hysterie et de l'Hypnotisme (Pithiatisme). Conférence facte à la Societe de l' Internat des Hôpitaux de Paris, June 28, 1901.

Hysteria, by S. A. K. Wilson in Brain,[1] the author well says, "The mere enumeration of these conflicting hypotheses may overwhelm the reader with a deep sense of despair at their hopless dissimilarity, and he may reasonably fear that finality is as far off as ever. But let him not be unduly distressed. Odd as it may appear, many of these contradictory opinions have one feature in common. Not only are the respective originators alike in the earnestness of their advocacy of them, but there is a curious similarity in their appeal to their own clinical experience subsequent to the adoption or enunciation of their own particular theory, and in their resort for substantiation of its virtues to the results of treatment." . . . . .

"To the individual who is not so absorbed in science as to lose his sense of humor it may seem that hysteria, the while she responds so nobly to the appeals of the advocates of these various theories, is quietly smiling in her sleeve. As of old the ascetic and the epicurean, the celibate and the polygamist, the socialist and the monarch by divine right turned alike to the pages of Holy Writ for support of their particular ways of living and views on life, and found it therein, so the exponents of the sexual theory, the suggestion theory, the sleep theory, and the "hysteria-only-a-symptom" theory, alike appeal to experience for confirmation of their opinion, and find it. It seems to us that not only does the mere juxtaposition of the above-selected passages furnish a self-evident proof of the fallaciousness of this appeal to the results of the treatment, as Sollier himself has clearly recognized; it also affords a significant illustration of what appears to be as unfortunate a tendency in the study of modern hysteria as in the study of the ancient, viz., the introduction, consciously or unconsciously, of the subjective element on the part of the physician." Wilson says moreover that more than one speaker at a recent Discussion on Hysteria held by the Neurological and Psychiatrical Societies of Paris made allusion to this fact, but it has not been emphasized at all adequately. It is not merely that his hypothesis is apt to color the physician's way of looking at a case, but also that in some obscure and little understood manner the patients come in a sense to respond to his hypothesis, so that the wider his experience the greater is the apparent confirmation of its truth. How else can we explain the facts so familiar to the student of the history of the disease? The clinics of Paris and Vienna have shown us how hysteria can be cultivated; the hysterical patients of the *Salpetriere* differ from their fellows of *la Pitie*. In a hundred consecutive cases Babinski has failed to discover a single instance of hemianesthesia, and says moreover, "as for constriction of the visual fields, dyschromatopia, ovarian tenderness, and so on, the hysteriques in my wards simply do not have them." "Heureuses hysteriques!" says Sollier in an aside. Wilson continues

"There is I think a profound truth in the remark of the late Professor Raymond, that the patients do not change so much as is thought; it is rather that the *observer* changes his point of view. Any one reads the contributions of Freud will be struck by the way in which, in his earlier communications, he describes his open-mindedness in approaching the problems of hysteria, and how the juvenile sexual trauma eventually forced itself on his consideration till he became convinced of its essentialness. Wherever he looks now, it

[1]Vol. xxxiii, 1910–11, p. 295.

meets his gaze. We may say if we will, that there are differing hysterical types but the interesting thing is that these types appear wherever the disease is cultivated, and that in a matter of this kind the subjective element must enter largely. Nor can the influence of environment, circumstances and that indefinable something which is usually called the "atmosphere" of a place be ignored. Let the reader turn to the fascinating essay on "The Wandering Jew" from the pen of Henry Meige, which appeared long ago in the *Nouvelle Iconographic de la Salpetriere*, to appreciate what the reputation of Charcot and the atmosphere of the Salpetriere meant to the world of hysteria."

We have ventured to make this long quotation because we think it will convey to the reader in the most satisfactory exposition possible of the theories of hysteria and their significance together with their weaknesses.

Symptoms.—An idea of the number and variety of the symptoms of hysteria has probably been obtained from the definition given—a variety which belongs to no other disease, and which may include almost all symptoms excited by any of the numerous nervous diseases. The hysterical patient is, however, characterized by certain general, corporal, and mental peculiarities which should be first considered. Such persons are emotional, irritable, capricious, sensitive, often willfull, sometimes because of indifferent early home training and overindulgence. They exaggerate every illness and demand an inordinate amount of sympathy. If women, they are at times disagreeable and petulant or doggedly silent, while at others they are charming and fascinating. They are often intellectually bright. Hysteria does occur, however, among intellectual degenerates. Other hysterical cases present no mental peculiarities. As to physical development, the hysterical patient is by no means always delicate; indeed, some of the most stubborn cases are those which appear in blooming health, rosy, and well nourished.

The symptoms of hysteria are conveniently arranged in five divisions: 1. Derangements of sensation. 2. Derangements of motion. 3. Vasomotor derangements. 4. Visceral derangements. 5. Convulsive seizures. 6. Joint symptoms. 7. Mental symptoms.

Some of these symptoms are so common in hysteria and so peculiar to it, that of themselves they are of decided diagnostic value, and as such have received the name of "hysterical stigmata." Among the most important of these are:

1. Derangements of Sensation.—The symptoms in this category are, as a rule, only elicited by the special examination of the physician, being rarely discovered by the patient. They include, especially, alterations of cutaneous sensibility, manifested by anesthesia or hyperesthesia. Most striking is insensibility to painful impressions, known as *analgesia*. It is usually tested by thrusting a pin deeply into the flesh—an act which is often totally unfelt. Less invariably is there failure to appreciate the sharp irritation of the electric current. Such analgesia may be confined to definite parts of the body, half the body, or may be general. It may extend to the mucous surfaces as well, and even to the deeper tissues, as those of the muscles and joints. While analgesia is the most common manifestation of de-

ranged sensibility, there may be absence of the sense of temperature, of pressure, and even of the muscular sense.

Hyperesthesia is almost equally characteristic. The areas involved may be exquisitely sensitive or but slightly so, requiring, sometimes, considerable pressure to develop the tenderness, while at other times it is elicited by the slightest touch. The hyperesthesia is especially noticeable when the attention of the patient is directed to it by such remarks as, "This will hurt you very much when I touch you." The sensitive areas may also be limited or extended and anywhere—on the head, thorax, limbs. Inguinal tenderness is especially frequent on the left side. Even more characteristic is the hyperesthesia of the spinal column—the so-called "hysterical spinal irritability"—which effects the column as a whole or in segments, not infrequently a single vertebra. The sensitiveness may be so extreme that the slightest contact may cause the patient to cry out, while strong pressure may be necessary to cause it. Of special interest also are the hysterical zones, to be again referred to.

The *special senses* are variously involved. There may be simple dimness of vision or narrowing of the field, due to anesthesia of the peripheral part of the retina. There is often total amblyopia, but never hemianopia. The cases of so-called hysterical hemianopia are rejected by some competent observers. Hysterical achromatopia is not infrequent. According to Charcot, the loss of the appreciation of violet is the most common, then of green, and, lastly, of blue and yellow. Loss of hearing is not infrequent and still more frequent is anesthesia of taste and smell, even bitter substances, like quinin, or pungent ones like vinegar, producing no impression or but a trifling one.

2. Derangements of Motion.—The most striking of these is *paralysis*. It commonly comes on suddenly, apparently as a result of fright or other suddenly acting cause. It may, however, be gradual, and take weeks for its development. It is most frequently hemiplegic, but may be monoplegic, rarely diplegic, while every form of organic paralysis may be simulated. Hemiplegia is more usual on the left side—according to Weir Mitchell, four times as frequent as on the right. The face is not usually affected, the neck and arms rarely, the legs oftenest. The patient can sometimes move the legs in bed or even when sitting up, while all attempts at walking are unsuccessful; or she may be able to move the arms when the eyes are open, but not when they are shut. It is a paralysis of the will. Sometimes one leg only is paralyzed, giving rise to a peculiar gait, the free leg making long strides while the paralyzed one is dragged along with a shuffling noise, and not swuug outwardly in a circle as in true hemiplegia. Sometimes there is ataxia with paresis. Paralysis may be either flaccid or spastic. Though far more frequently a symptom of hysteria in women, it may be as striking in men. Paralysis of the vocal cords is one of the most frequent symptoms of hysteria, giving rise to *aphonia*. The paralysis is easily demonstrable by laryngoscopic examination, because of anesthesia of the pharynx. It may be so marked that the vocal cords acutally open with an attempt at phonation. Anesthesia and motor paralysis are commonly associated.

Contractures and *spasms* are a form of motor derangement; they may occur alone or with anesthesia and paralysis. They exhibit every variety,

and may attack any group of muscles; they may be tonic or clonic, and sudden or gradual in development. The tonic contracture is most usual in the arm, which is flexed at the elbow and wrist, while the fingers grasp the thumb in the palm tightly. In the feet, also, flexures predominate, the feet being inverted and the toes flexed. In the larger joints, on the other hand, the extensors are involved, as a rule. Rarely extensor contractures occur in the small joints; all disappear with chloroform narcosis unless they have persisted a long time and shortening of the muscles, ligaments, etc., has occurred. The reflexes may be very much exaggerated and the condition closely resemble spastic paraplegia. Extreme emaciation may occur in connection with these contractures, as witness a remarkable case related and illustrated by Weir Mitchell in the "Medical News," August 24, 1895.

Even hysterical trismus may occur, and a very striking result of abdominal contracture is the *phantom* tumor, which is found usually just below and in the neighborhood of the umbilicus, often simulating a firm and solid growth. The mechanism of its production, according to Gowers, is a relaxation of the recti and spasmodic contraction of the diaphragm, together with inflation of the intestines and an arching forward of the vertebral column. Women have even been prepared for surgical operation on such tumors when the delusion was dissipated by the anesthetic, and the abdomen has been opened for purely hysterical conditions. Such tumor is not infrequently associated with symptoms of spurious pregnancy—pseudocyesis. Visible tremor may be present, rarely hysterical athetosis.

3. Vasomotor Derangements.—A striking *pallor* is often present, at other times hyperemia, and even a hot skin. *Hemorrhage* from internal organs, especially the stomach and lungs, often alleged, is usually at least apocryphal. Commonly, the blood is derived from the gums, and its amount is never considerable. Yet such symptoms have been the basis of a diagnosis of pulmonary disease or gastric ulcer. Hemorrhages into the skin are also alleged, but are very rare.

Hysterical fever belongs also to vasomotor symptoms. A temperature of 115° F. (46.1° C.), and even more, has been reported. Such temperatures are characterized by their irregular occurrence. Actually they are extremely rare, being in most instances traceable to deception, and many doubt their existence.

Anomalies of secretion include profuse and scanty perspiration, the latter resulting in a peculiar dryness of the skin; the salivary secretion is similarly influenced, and modifications in the urinary secretion are some of the most characteristic phenomena of hysteria. They include ischuria, but especially polyuria, the patient passing a large amount of very light-colored urine of low specific gravity. *Excessive thirst* is also frequent, further augmenting the polyuria. The chemical composition of the urine is altered in many severe cases; thus, the phosphates and urates have been found diminished, while the ratio of earthy to alkaline phosphates may be changed to one to two or one to one, instead of one to three. Such changes are held by Charcot's school to be diagnostic of convulsive hysteria as contrasted with epilepsy.

4. Visceral Derangements.—The *digestive system* is especially disturbed by simple indigestion, depraved appetite, flatulence, and gastric pain.

Not infrequently there is spasm of the esophagus, causing difficulty in swallowing: in some instances expulsion of food before it reaches the stomach. Hysterical vomiting is very common, and alleged vomiting of impossible substances is one of the most characteristic symptoms. An antagonism to food is sometimes present, so extreme that death by starvation has been barely averted; indeed, is said to have occurred. Constipation is a frequent and troublesome symptom. Much more rare is the opposite condition of diarrhea.

Cardiovascular and *pulmonary* symptoms exhibit every variety, including irregularity of the heart's action, tachycardia and bradycardia, precordial oppression and sense of suffocation, with extreme frequency of breathing and deranged rhythm. Laryngeal spasm, hysterical cough, and hysterical hiccough are frequent symptoms. *Hysterical cries* with inspiration or expiration, and imitation of the sounds produced by various animals are described by the French neurologists.

5. JOINT AFFECTIONS, purely hysterical, were early studied by Sir B. Brodia, and later by Sir James Paget. They involve the knee and hip and consist of fixation, tenderness, and even swelling,

6. The MENTAL SYMPTOMS are a prominent feature of hysteria, and vary greatly in their manifestations. Irritability and capriciousness of temper, maniacal excitement, hallucinaitons, and even insanity may occur. The hysterical trance is a well-known condition. It may come on spontaneously, but more frequently it follows one of the forms of hysteroid attacks to be later described. The cataleptic state may be associated with this symptom.

7. CONVULSIVE SEIZURES.—Hysterical convulsions are a recognized symptom, while in some they are the only manifestation of the disease. Their severity varies greatly; but two degrees are described, a milder or minor, and a severer or major.

(*a*) *Minor Form.*—This may come on suddenly or be preceded by a *prodrome*, including hysterical behavior, such as laughing and crying; a sense of constriction about the throat, or that of a ball rising in it (the so-called globus hystericus); a feeling of anxiety with shortness of breath with pain and discomfort in the chest or abdomen (pseudo-angina).

In the *actual seizure* the *patient falls*, with this striking feature: that she rarely fails to find a soft spot, such as a sofa or bed, to receive her. The convulsion consists in clonic contractions of a disordered and irregular kind, in which all four extremities and even the trunk may take part. Though seemingly unconscious, the patient still gives to the careful observer the impression of a certain method in her madness. The convulsion lasts usually a few minutes, when it passes off spontaneously, or the patient may be aroused by some powerful impression, such as the dashing of cold water in the face, or by a sharp galvanic shock. She may remain emotional for a time, but the period of torpidity, so characteristic of the epileptic fit, is rare.

(*b*) *Major Form* (*Hysterical Epilepsy*).—This has become widely known, more particularly from the graphic descriptions and vivid pictures furnished by the French school of neurology. It is much less common in this country; indeed, it is rare outside of hospital walls, where prostitutes are the usual subjects. The attack may be preceded by prodromata similar to those that

precede the milder attacks. The convulsion is described by French writers as having four distinct stages:

1. The epileptoid state, closely simulating a true epileptic attack, with apparent unconsciousness, tonic spasm, even opisthotonos, grinding of the teeth, livid face, succeeded by clonic convulsions, relaxation, and coma; lasting rather longer than the true epileptic attack.

2. The period of "contortions and grand movements," called by Charcot "clownism," characterized by emotional display, striking contortions, or cataleptic poses.

3. The period of spastic positions and passionate attitudes, including those of ecstasy, fright, beatitude, or eroticism.

4. The return to consciousness and a stage characterized especially by manifestations of delirium with extraordinary hallucinations, and by hypnotic "suggestibility." In it visions are seen, voices heard, and conversations carried on with imaginary persons. Imaginary events are related as actually true. These hallucinations sometimes persist even after recovery. These periods are not sharply separated from one another.

Suggestions and Hypnosis.—At this point it is suitable to say something of these conditions, so closely associated with the hysterical state and which have attracted much attention. By suggestibility is meant the susceptibility of a person to the production of a definite psychical or physical state dependent upon the arousing of corresponding ideas in the mind. It is really a further development of the hysterical mental constitution already referred to, in which the patient permits himself to be dominated by his imagination. Suggestion is merely the artificial fostering of the psychical peculiarity. It is most easy during the part of the hysterical attack when the patients speak, hear, and answer. At such times a definite direction may be given to the patient's ideas. If he is told in an emphatic, convincing manner that he is in a certain situation, be it one of a pleasurable kind or a state of suffering or danger, he believes it, and at once, by behavior or expression, shows that he believes it, and is actually experiencing the conditions named. Physical states may be similarly suggested, such as paralysis, contractures, and anesthesias, while severe pain may be inflicted without exciting sensibility. After the attack is over the subject is totally ignorant of what has transpired, but during another attack may remember the events of the previous one, or what is still more strange, supposed events, furnishing thus an instance of double consciousness.

Hypnosis is closely allied to suggestion. It is regarded by many as nothing more or less than the intentional production of a hysterical attack, or a hysterical psychosis by suggestion. As Strümpell graphically puts it, "Hypnosis is an artificial hysteria." This view, however, is not held by all. The French school makes four principal forms of the hypnotic state with many transitions:

1. The cataleptic state, in which the limbs retain all the positions artificially given them.

2. The state of suggestion or artificial hallucination, in which patients are induced to eat tasteless and unnatural food with a gusto.

3. The lethargic state: a state of apparent unconsciousness, with the eyes closed, the muscles relaxed, yet with a markedly increased excitability

in the muscles and nerves, in which a light tap on a nerve like the facial is sufficient to put all the muscles supplied by it into a tetanic contraction far outlasting the irritation.

4. A state of hysterical somnambulism, in which the patient, while remaining half unconscious, still answers automatically questions put to her, obeying orders or giving them, and sometimes exhibiting certain sensory hyperesthesias (vigilambulism). It will be seen that each of these corresponds with one or another of the different manifestations of the hysterical attack.

Hysterogenous Zones.—In this connection some further reference should be made to the so-called hysterogenous zones already alluded to. These are hyperesthetic areas especially studied by Richet, on which persistent pressure will sometimes excite a hysterical attack. While the submammary areas, especially the left, and the inguinal region are favorite hysterogenous zones, the zones may be in any part of the body: as, for example, the sides of the trunk. Pressure in such a zone may cause an existing attack to subside. Hysterical spasm may be localized or limited to groups of muscles.

Diagnosis.—This is not usually difficult. There is something indescribable in the bearing and appearance of a hysterical patient which enables the experienced physician often to recognize the disease at a glance. While, as stated, many phenomena of any organic nervous disease may be present, yet the essential symptoms of organic lesion are still wanting and there are symptoms which are peculiar to hysteria alone. The anesthesias are peculiar in their area of distribution, and hysterogenous zones are nowhere else found. The hysterical convulsion is quite *sui generis*, the throat and pharyngeal symptoms are not found elsewhere, and the emotional symptoms are tell-tale. Cases occasionally occur in which the diagnosis between hysteria and organic disease is very difficult.

Prognosis.—This is very rarely serious, though the course and duration of the disease vary greatly. The milder cases may be of very short duration, while the more serious may last for weeks or years, often, however, with intermissions and changes. Only in very rare instances does a fatal result occur, and reports of death from hysteria demand very critical examination.

Treatment.—A proper prophylactic treatment, so commonly overlooked, would prevent many cases of hysteria. The counteracting of all that is mentioned under the head of predisposition constitutes such treatment. Wholesome discipline or training in youth, the inculcation of self-denial as contrasted with overindulgence and the gratification of fancy, and careful exclusion from the companionship of hysterical persons make up the sum of these.

The successful curative treatment of hysteria also more frequently depends upon the individuality of the physician than on the remedies employed. Indispensable, however, is the removal of the causes which predispose to the disease, whether they be of the nature of moral influences or bodily ailment. Among the most difficult to eliminate of the former are those which arise from the fondness and sympathy of relatives who have, from long habit, become almost slaves to the fancies of the hysterical subject, and with whom, in consequence, firmness has become impossible. It is in consequence of such difficulties that the isolation plan of treatment,

which has become inseparably associated with the name of Weir Mitchell, has been so successful. Originated for neurasthenic cases, it is as applicable to the hysterical, in whom neurasthenia is likewise often present, while hysteria also often forms a large factor in the neurasthenic state. Whenever possible, the patient must be removed from her previous surroundings, her family, and even her friends. This accomplished, the details of management largely depend upon the peculiarities of the case, but in a general way the Weir Mitchell plan may be said to be as follows:

First, and indispensable, is the care of an intelligent and sensible nurse. Under her charge the patient is put to bed and kept in a condition of absolute rest, even reading being prohibited, and also at first self-feeding. Massage is used daily, at first for short periods, which are gradually lengthened, until an hour is thus consumed. With massage is associated electricity, the faradic current with slow interruptions being usually preferred. Thus, with a small electrode, the motor nerve points can be picked out, and the contraction of individual muscles produced. Massage and electricity both have for their purpose the substitution of exercise, to which end the former is by far the most useful and important. Both are discontinued during menstruation. The food at first is milk, which has been usually skimmed, but in my own experience good milk unskimmed and diluted with one-fourth its bulk of water, or aerated water, answers the purpose better. The proportion of casein is less, and the oil, which is so valuable for the nutrition of the patient, is retained in more nearly its normal quantity. At first from four to six ounces of milk are given every two hours. After a week or ten days a chop or a few raw oysters are added at luncheon, with a cup of coffee or tea, and later at breakfast an egg, bread and butter or biscuit with the milk, the latter being continued at two-hour intervals. The patient should have a thorough sponge bath daily at the hands of the nurse. It is convenient to make out a schedule including the hours for nourishment, massage, and electricity, of which the last two should be separated by several hours. Massage should be followed by a full hour's rest. Under this forced feeding the patient gradually fattens, and concurrently with this the excitability of the nervous system usually grows less. In a month or six weeks the patient is allowed to sit up, at first for a few minutes only, but each day a little longer, until the whole day is thus spent, interrupted by periods of rest. Later she is taken out to drive, and then to walk for gradually increasing distance, until, in the vast majority of instances, she is enabled to perform enormous amounts of physical exercise without fatigue. Many patients who have bedridden for months and even years, women whose relatives had been worn out with nursing, who, after a few weeks of this treatment, acquired the most vigorous health, walking many miles a day and presenting an appearance of health and strength which would be considered absolutely impossible by one unfamiliar with the results of this mode of treatment. As a rule, three months should be asked for its fulfillment. As has already been said, the individuality of the physician has much to do with the success of the method. One who has a firm, earnest, yet gentle manner will do more with such cases than one who is vacillating and disposed to yield to the caprice of the patient. An element of "suggestion" must perhaps be acknowledged in the power of the physician thus constituted,

yet the full application of this principle of treatment by hypnotic suggestion is to be deprecated. The nurse in charge must be similarly constituted, and it not infrequently happens that a nurse otherwise excellent is totally unadapted for the management of a case of this kind.

As to medicines, the number that are useful are few. Iron and arsenic, in very moderate doses, are the only ones which are actually curative. The various nervous sedatives, including valerian, asafetida, the bromids, the milder hypnotics, such as phenacetin, rarely chloral, may be used as occasion requires; morphin should never, or almost never, be given. A convenient form in which to use asafetida is the suppository; 10 grains (0.66 gm.) may be put in a single one.

The paralysis and contractures generally require some time to overcome, and in some cases are persistent in spite of all treatment. Cure is accomplished mainly by manipulation aided by electricity, under the use of which the symptoms gradually disappear and the patient, induced at first to walk for a few steps, will slowly acquire full power of locomotion. Anesthesia is best treated by faradization and the electrical brush. Paralysis of the vocal cords is also best treated by electricity, suitable electrodes having been devised for that purpose.

Allusion should be made to metallotherapy, a treatment instituted by a French physician named Burq, who years ago ascertained that by laying plates of metal upon a cutaneous surface affected by hysterical anesthesia, sensation is sometimes restored at once not only in the immediate region, but also sometimes in a much larger area. The cases so treated were mostly hysterical hemianesthesias. Iron is the metal most frequently efficient, but sometimes copper, zinc, or gold. The selection of the metal essential to each case was called *metalloscopy*, and Burq held that this metal would also have the same effect if given internally. A committee of the Paris Society of Biology confirmed these statements in 1876, except as to the internal administration of the metal. A similar discovery of Charcot showed that the return of sensation to an anesthetic area after applying a metal plate, is accompanied by a simultaneous development of anesthesia upon the opposite side and in an exactly corresponding place. This is known as transfer. Other hysterical symptoms than anesthesia have been found to exhibit analogous phenomena. Thus, transfer can sometimes be observed in hysterical amblyopia, achromatopia, deafness, loss of the senses of smell and taste, contractures, and paralysis, while such transfers may be induced by other means than metal plates, known as esthesiogenous remedies. They include large magnets, feeble galvanic currents, static electricity, vibrating tuning-forks, and sinapisms. It must be plain to any thinking person that these phenomena are merely the result of suggestion produced by ideas similar to those already described. Their career will doubtless end like that of Perkins' tractors. Hypnotism has also been employed for the treatment of hysteria, and has acquired some popularity in France, where it has been especially practised by the school at Nancy. Wonderful cures have doubtless thus been accomplished, but based as it is upon mysticism and imagery, and being already much abused by charlatans, it is to be hoped that its fate will be that of metallotherapy and Perkins' tractors.

NEURASTHENIA.

SYNONYMS.—*Nervous Exhaustion; Nervous Weakness; Encephalesthenia; the American Disease.*

Definition.—A term originally suggested by George M. Beard, in 1879, for a symptom complex without anatomical basis, in which muscular weakness, nervous irritability, and pain are variously manifested. Beard defined nervousness as "Deficiency of nerve force, manifested chiefly by undue sensitiveness to external impressions," and neurasthenia as "A sign and type of functional nervous disease" evolved out of this general nervous sensitiveness. The line of demarcation between neurasthenia and hysteria is not always definite. Not only do the two conditions sometimes merge, but certain cases of neurasthenia are in no way distinguishable from the minor forms of hysteria. The condition is called *spinal, cerebral, cardiac*, or *gastric*, according as the symptoms dependent on one or the other of these systems predominate, but the line of demarcation is not sharp.

Etiology.—The same class of persons who are predisposed to hysteria are predisposed to neurasthenia, and such predisposition may be inherited or acquired. So, too, many of the exciting causes of the former become the exciting causes of the latter. Among these are overstrain of mind and body, overwork, especially overwork associated with care and anxiety. It is distinctive of neurasthenia as contrasted with hysteria that it is more frequent among men, on whom business care and financial worry fall more severely. It is well known that men differ greatly in their power to bear the mental strain incident to the struggle for existence or business success. From the special prevalence of this disease in America it has been called "the American disease," and is reasonably ascribable to the fact that mental and physical strength in this country is more taxed than in any other.

Morbid Anatomy.—Although Beard took great pains to prove that neurasthenia is a physical and not a mental state, and that these phenomena do not come from emotional causes or excitability, but from nervous debility and irritability, there has been found no distinctive morbid change associated with its complexus of symptoms any more than with hysteria. It is barely possible that the investigations of C. F. Hodge and others, demonstrating changes in nerve cells during functional activity, may result in some further knowledge in this direction.

Symptoms.—It has already been said that the symptoms of the minor forms of hysteria are the symptoms of many cases of neurasthenia. The *appearance* of the patient may be that of perfect health; less frequently he looks worn and worried. In the *spinal* form motor phenomena are the most conspicuous. Of this and, indeed, of all forms, the most constant symptom is *muscular weakness*, as a result of which the patient complains of being tired and weary, even too weak at times to keep out of bed. Such weakness may affect the gait, making it uncertain and trembling, and the acts performed by the upper extremities may be similarly embarrassed. There may be *hyperesthesia* and *paresthesia*, and even the special senses may be affected, especially *vision* and *hearing*. The latter is more frequently oversensitive, and vision may be obscured by the presence of scotomata or muscæ volitantes. In the *cerebral* form especially characteristic is a low-

spiritedness or *despondency*, often painful to witness, and which may alternate with *irritability* or *moodiness*. Another symptom is *sleeplessness*, though many patients sleep well; indeed, there is occasionally an irresistible disposition to sleep. A disposition to seek *solitude* is characteristic, while at other times the patient fears to be alone. Again, he is *restless*, unsettled, and impelled to move about from place to place, while there is sometimes a pronounced disposition to *suicide*. *Confusion* of *mind*, and especially a difficulty in dealing with figures, is a very common symptom, sometimes an initial symptom, the simplest arithmetical problems being quite impossible with one so affected.

The *cardiac* form is characterized by *palpitation* and frequent irregular action of the heart and *precordial pain*, which give rise to the belief in the patient's mind that he has cardiac disease, arteriocapillary fibrosis, or "hardening of the blood-vessels," as it is called by the laity. Of vasomotor phenomena there may be *flashes* of heat, *sudden sweats*, even night-sweats, and a relaxed state of the peripheral blood-vessels to an extent which may cause the "*water-hammer*" and even *capillary* pulse, similar to that of aortic regurgitation. *Epigastric pulsation* is often an annoying symptom in women. In the *gastric* form are, especially, *gastric pain*, the distinctive symptom of "nervous dyspepsia," but there are also distention and discomfort after eating, or a constant noisy motion of gases—*borborygmus*. *Polyuria* is a conspicuous symptom. A slight degree of glycosuria and even intermittent albuminuria have been reported. The opposite condition of urine—a dark hue and high specific gravity—is more rarely present. *Hoarseness*, *aphonia*, and very frequent breathing are regarded as symptoms of neurasthenia as well as of hysteria.

Diagnosis.—This is generally easy, and is arrived at by the exclusion of the objective symptoms of organic disease and by the etiology, for it will be observed that all of the symptoms which have been narrated are subjective in character. The so-called *spinal irritation* is a condition which resembles neurasthenia, and probably some of the cases so named which are not hysteria are cases of nervous exhaustion. Sensitiveness of the vertebræ is not apt to be present in neurasthenia, whereas it is the most distinctive symptom of spinal irritation. No case should be declared neurasthenic without the most searching physical examination. Many organic conditions (tuberculosis, brain tumor, etc.) have as their first manifestation, neurasthenic symptoms.

Prognosis.—Recovery from neurasthenia may be confidently promised to almost every patient who is in a position to meet the indications of a successful treatment, which, unfortunately, are likely to be expensive, though the modern hospital affords to even the poorer classes an asylum where the treatment may be successfully carried out.

Treatment.—The first essential condition of a successful treatment is *removal of the causes* which are responsible for the illness. To this, in the case of women, and sometimes of men, the most successful adjuvant is the *rest treatment* of Weir Mitchell, the technic of which has been already described under the treatment of hysteria. After this and, in the case of men, often even before this, *removal from the scene* and surroundings which attended the development of the disease is most useful. *Travel* away from home,

especially in foreign countries, a sojourn at a sanitarium or health resort, the seaside, the woods, the mountains, for prolonged periods, sooner or later been followed by recovery in the majority of cases. For the poor, the rest-cure as carried out in hospitals may be substituted for the more expensive methods of home treatment.

The treatment of the *insomnia* of neurasthenia calls for brief special consideration, and what is said here may apply to the treatment of any form of simple insomnia, by which is meant insomnia not the result of pain. Modern therapeutics has added to our resources a number of drugs which are more or less efficient to this end. The best of these, considered from all standpoints, are *sulphonal, trional* and *veronal*. Not less than from 10 to 15 grains (0.66 to 1 gm.) of any of these should be given to an adult, while twice as much may be given if needed. We prefer to give this dose and repeat it in an hour if no effect follows. They are bulky, soluble with difficulty in cold water, but readily so in any hot menstruum, and especially suitable is hot milk. They should be given an hour or two before sleep is desired, but associated quietude is necessary to secure its effect. *Paraldehyd* is an excellent remedy, but very disagreeable, and is more prompt in its action than sulphonal or trional, or veronal and should be given in dram (4 gm.) doses. *Chloralamid* is also a good hypnotic; its dose is 30 grains (2 gm.). It should be dissolved in alcoholic menstruum diluted to 1/2 ounce (13.5 c.c.). *Chloral*, as a simple hypnotic, is better, perhaps, than any of those named, although it has yielded its former high place to those just mentioned because of their harmlessness. All of these have the disadvantage of sometimes causing drowsiness the next day. Chloralose, a modified chloral, is often efficient in doses of 5 grains (0.33 gm.) to an adult. It often acts magically but sometimes a second dose excites. Hydrobromate of *hyoscin* may be used in doses of 1/100 grain (0.00066 gm.) if the drugs named fail. Sometimes it acts like a charm, at other times it produces the opposite effect —exciting the patient. One trial suffices to settle the question.

It is true of all the drugs named that their effect is apt to wear off, and increasing doses must be used, and the hypnotic habit is easily formed. It is, therefore, desirable to obviate the necessity of their use as early as possible, and, if possible, substitute other measures. Often the patient simply needs a start to put him in the way of sleeping, while sometimes the simple feeling that there is something at hand which he can use if he wishes gives him the needed confidence and he goes to sleep at once. A warm *bath* before retiring, or even at times a cool bath or cool sponging, and again a hot bath, promote sleep. To persons residing in cities sleep is often favored by a sojourn at the seaside, many being able to sleep there when they cannot do so at home. The same is true of the country or the mountains.

It is important, too, in our efforts to secure sleep for our patients to investigate the various functions, derangement of any of which may keep a neurasthenic patient awake. Irregularities of digestion and circulation should receive attention. An undigested meal or a loaded bowel often keeps one awake, while an excited heart, by its ceaseless beating, repels the restful sleep without which life is wretched. Often a light meal or a single glass of wine seems to furnish the brain-cells the right amount of stimulus.

TRAUMATIC NEUROSES.

SYNONYMS.—*"Railway Brain"; "Railway Spine"; Traumatic Hysteria; Erichsen's Disease.*

Definition.—A neurasthenic or hysterical state, the result of shock from railroad accident or accident of similar alarming character.

Etiology.—Profound nervous shock, however induced, by railroad accidents, shipwreck, boiler explosions, and the like, even when the sufferer himself is not a victim, but is profoundly impressed by it, is capable of producing this nervous state.

Morbid Anatomy.—In the vast majority of cases, anatomical changes are not discoverable; in fact, as most cases recover, there is little opportunity to seek them. In a few, however, morbid alterations have been found in the brain and spinal cord, including *degeneration* of the pyramidal tracts of the cord, demonstrated by Edes in four cases; *multiple sclerotic areas* in the white matter, and *arteriosclerosis* in the vessels of the brain, with scattered areas of degeneration, but the study of concussions in man has not led to very definite results. The effects of concussions of the brain and spinal cord have been studied in animals, and changes in the nerve cells and nerve fibers have been found.

Symptoms.—These are not essentially different from those of neurasthenia from other causes. The most remarkable fact with regard to them is that they do not necessarily immediately follow the accident, and there may be some interval of time between the two events—the accident and its results. In some cases the symptoms appear suddenly, in others they are gradual in their invasion. All the symptoms detailed under neurasthenia may be present, especially spinal tenderness and pain in various parts of the body, principally in the back and head; there may be numbness and tingling in the extremities, increased muscular irritability, and increased knee-jerk. The latter varies from day to day, and may be exhausted by repeated stimulation. Extreme depression of spirits is another symptom. Other patients exhibit active hysterical symptoms, including modifications of sensation and motion, hemianesthesia, anesthesia, paresis, and even paralysis.

In the more severe cases in which there is actual concussion the symptoms suggest organic changes, which are, indeed, actually found at times in the shape of pachymeningitis. Such cases exhibit diminished superficial reflexes, with exaggeration of the deep ones. There may be severe pain, variously distributed. Other symptoms are alterations in the temperature sense and in the muscular sense, both of which may be bilaterally distributed. There may also be modification of the special senses, including those of smell, taste, and vision, with inequality of pupils. There may be monoplegia with or without contracture. Symptoms which imply true organic change are optic atrophy, bladder-symptoms, paresis, and exaggerated reflexes. Such cases are sometimes, though rarely, fatal.

Prognosis.—Most cases get well. The effect of delayed litigation is often to delay recovery, while successful litigation does not always relieve the symptoms, and when it does, it is by no means always speedily—months and even years elapsing before the cases recover. A few cases, where there is true organic disease, perish.

Treatment.—Rest, mental and physical, is the first essential condition of recovery. It may be aided by the measures useful in other forms of neurasthenia, such as massage, electricity, and proper feeding. Medicines avail little, except for their moral effect. Narcotics should be avoided.

OTHER FORMS OF FUNCTIONAL PARALYSIS.

Abasia-astasia.

Definition.—Abasia (α privative; βασις, a step) is a term given by P. Blocq, in 1888, to a difficulty in starting the act of walking from a state of previous rest. Astasia (α privative; στασις, a standing) is an inability to stand, contrasted with integrity of sensation, muscular strength, and co-ordination of other movements of the legs.

Nature.—The phenomena are thus far inexplicable in the absence of discoverable lesions, and are usually regarded as hysterical. It is a condition occurring in adults, equally frequent in men and women—as determined by Knapp's study of 50 cases, of which half were in either sex.

Symptoms.—These occur in connection with a variety of morbid states, and a large majority of them are doubtless hysterical.

In the "unconscious" variety the patient is without any idea that he cannot walk or stand, when he suddenly finds that he cannot do either. Another variety of abasia-astasia is the "hypochondriacal," in which the patient acts under "conscious" erroneous impression that he cannot walk or cannot stand. It is sometimes associated in the hypochondriacal paranoiac with paresthesia, and in the neurasthenic with abnormally increased sense of fatigue. A third form is associated with some suddenly acting "shock," as fright, which acts inhibitorily on the motions of the patient. Finally there is the "coercion" variety of abasia-astasia, in which the patient, while in the act of walking or standing, is suddenly seized with the idea that he cannot walk or shall not walk. This differs from the hypochondriacal form in that the patient is conscious of the erroneousness and absurdity of the idea, but is nevertheless coerced by it.

These different forms are not always sharply defined. Suddenness is especially characteristic in the "unconscious" form. In other cases the patient may walk a few steps and then suddenly break down. Sometimes he stands rooted to the ground, as it were. At other times the development is slow, requiring even years to reach its acme. Sometimes it is preceded by trembling or staggering, as associated symptoms, the result of the effort of the patient to stand or move forward.

Closing the eyes usually increases the difficulty. On the other hand, sometimes, with the eyes closed the patient can walk in the normal manner, when it is impossible to do so with the eyes open. The latter is especially true of the hypochondriacal variety. In these cases, too, the natural gait is sometimes restored after attempting an unusual method of walking, as walking backward or with the legs crossed or by leaping or in military step. So, also, abasics can walk on all-fours. The morbid state is also influenced by certain surroundings, as broad open surfaces or long narrow corridors or standing without special support. Th. Ziehen refers to a case in which

abasia came on when the patient walked under a tree, the moving leaves of which produced moving shadows. There is sometimes associated tachycardia; at other times evident hysterical symptoms, such as tender spots, hemianesthesia, and the like. In other cases there is epilepsy, paralysis agitans or chorea.

Diagnosis.—This is based upon the retention of absolute integrity of sensation, of muscular strength, and of co-ordination of the legs, demonstrable in the recumbent position. From *hysterical paraplegia* it differs in that the power of motion is intact in the recumbent position. From *intermittent lameness* it is distinguished by the fact that in intermittent lameness the inability to walk comes on after the patient has been walking a while, and the power of locomotion is restored after rest. Abasia-astasia has been observed in tumor of the frontal lobe of the brain.

Prognosis.—This is regarded as favorable, though relapses occur.

Treatment.—The evident hysterical nature of the affection, in the majority of cases, suggests the treatment for such cases. The rest-cure, massage, gymnastics, electricity, gradually increasing practice in walking, are measures which are likely to be useful. Th. Ziehen, to whose article in Eulenberg's "Real-Encyclopädie" we are indebted for much of the information in this section, recommends "suggestion without hypnosis," especially in the hypochondriacal and hysterical forms, as a reliable means of rapid cure; and in the cases in which fear or terror is conspicuous, small doses of opium,

Family Periodical Paralysis.

Definition.—A rare form of hereditary or family paralysis of the voluntary muscles, usually general, except those of the face, recurring at intervals of from one or two weeks to three months, and confined principally to children. It is attended with a loss of reflexes and electrical reaction, but no mental or sensory disturbance.

The disease is rare. It was first described by Cavaré in 1853 and by Romberg in 1857. Edward Wyllis Taylor[1] collected 25 cases, including two of his own, up to September, 1898, to which John K. Mitchell[2] added a twenty-sixth in 1899.

Etiology.—The disease is hereditary and is transmitted through the mother. As many as 12 members of a single family have been affected, though it does not usually affect all the children. Goldflam suggests that the paralysis is due to autointoxication, the poison acting upon the nerve endings in the muscles, while he also found that the urine secreted during the attacks was more toxic than at other times. The view of autointoxication is not accepted by all, and J. J. Putnam has advanced a theory of inhibition. The recent studies of John K. Mitchell on the case referred to, for some time under his observation, tend to confirm Goldflam's view, and also to show that there are two poisons, one of which predominates in one case and the other in another, and according as one or the other predominates the effect is greater on the peripheral nerves and muscles or the spinal

[1] "Journal of Nervous and Mental Diseases," vol. xxv., p. 637, 1898. Dr. Taylor alludes to 53 cases, but does not include cases inadequately reported: as, for example, cases merely mentioned by writers, but not reported in detail.

[2] "Amer. Jour. of the Med. Sci.," December, 1899.

center. It should not be omitted that some clinicians, including C. L. Dana, consider the majority of cases hysterical, though he says some may be cases of recurring poliomyelitis.

Symptoms.—The disease occurs in *youth* and in the *midst of health, even during sleep.* Beginning as a weakness or weariness in the arms and legs, it is *usually complete in 24 hours.* It is rarely confined to the legs, and may also involve the muscles of the neck, and even those of the tongue and pharynx, while those of the head and face remain intact.

Sensation for the most part is *unaffected*, as are also the special senses. The deep reflexes are diminished, sometimes abolished, while the superficial reflexes are feeble. Faradic sensibility of nerves and muscles is greatly lessened, sometimes absent. There is no fever, and sometimes the temperature is below normal, while the pulse is slow. Nothing abnormal has been found in the blood or urine, though the breath is heavy, the tongue is coated, and the urine is relatively diminished during the attack and increased after its termination, as happens in migraine.

The attack recurs at intervals of from one to two or more weeks, in some instances daily. It *begins to abate* usually *in a few hours or after a day or two*, and ultimately disappears completely, and the patient remains well until another attack sets in.

Treatment.—None is of any service, though some of the earlier cases seem to have yielded to quinin, while it is more than likely that these were in some way complicated with malaria.

AMAUROTIC FAMILY IDIOCY.

Definition.—A rare and generally fatal disease affecting several children of the same family, characterized by a feeble mental development, by progressive weakness of all the muscles of the body and by failing vision terminating in complete blindness depending on optic nerve atrophy and changes in the macula.

Etiology.—This is obscure. Singurlarly the disease is apparently almost confined to Hebrews, and Sachs tells us that he knows of no undoubted cases in other races. It cannot be ascribed to syphilis though the nerve atrophy resembles that due to hereditary syphilis, with which it may therefore be confounded. Its duration is usually less than two years. The disease is regarded by some as acquired, by others as congenital; the latter is more likely.

Morbid Anatomy.—The morbid changes are essentially those first described by Sachs who found them similar to those in brains of arrested development. There was confluence of the median and Sylvian fissures and complete exposure of the island of Reil. The cortex was hard and grating to the knife. Microscopic examination found destruction of the brain cells whose contour was rounded or elongated and the cell protoplasm was variously degenerated. The nucleus and nucleolus were sometimes wanting. Very few pyramidal cells were left. Hirsch found these same changes in the gray matter of the entire central nervous system, including the spinal cord and spinal ganglia.

Retinal changes are thus described by Tay:[1] "The optic disks were apparently healthy, but in the region of the yellow spot of each eye there was a conspicuous, tolerably diffuse, large white spot more or less circular in outline, and showing at its center a brownish-red fairly circular spot contrasting strongly with the white patch surrounding it. This central spot did not look at all like a hemorrhage, nor as if due to a pigment, but seemed a gap in the white patch through which one saw healthy structures."

Symptoms.—The child is well nourished at birth and the disease does not usually set in until the third to sixth month of life, when the mental and physical defects begin to be noticed. The child becomes quiet, listless and apathetic, and visual disturbance makes its appearance. As time passes muscular weakness occurs, the child is unable to hold up its head or sit up, the muscles are soft and flabby though they may be spastic. The reflexes may be normal, slightly subnormal or exaggerated. There is sometimes hyperacusis to sound and touch; on the other hand, a loss of hearing has been noted. Convulsions may occur but are not essential. Bodily functions and vitality are lowered resulting in susceptibility to bronchial and gastro-intestinal derangement. Retinal changes occur as described by Tay. (See morbid anatomy.) The symptoms gradually increase in severity and terminate in ultimate complete mental imbecility, marked palsy and total blindness. The child wastes and dies usually before the end of the second year.

Treatment.—None has ever proved of use. Proper and sufficient nourishment with wholesome hygienic surroundings naturally suggest themselves.

VASOMOTOR AND TROPHIC DERANGEMENTS.

Acute Angioneurotic Edema.

Synonyms.—*Giant Urticaria; Acute Circumscribed Edema of the Skin; Quincke's Disease.*

Definition.—Edematous swelling occurring suddenly in various parts of the body, disappearing in a few hours, perhaps to recur again.

Etiology.—Heredity is sometimes observed, but any other cause is unknown.

Pathology.—The condition is regarded by Quincke as a vasomotor neurosis producing sudden dilatation and increased permeability of the vessels. It is, however, one of the derangements which may be said to be of mixed vasomotor and trophic origin.

Symptoms.—The *face*, especially the eyelids and nose, is the most usual site, but the swelling may affect any part of *the body*, as the hands, face, or genitalia, including the penis. It may be painful. Even the *mucous membranes* may be invaded, especially the lips, mouth, and pharynx, while a fatal edema of the larynx has occurred. The *onset* is *sudden* and the patient's previous health may have been excellent. *Gastro-intestinal disturbances*

[1] For a fuller presentation of this subject the student is referred to the paper by B. Sachs in Posey and Spiller's book on "The Eye and the Nervous System," 1906.

manifested by vomiting, colic, diarrhea, and gastralgia, are sometimes associated while they sometimes alternate. They are ascribed to an edema of the mucous membrane. This has been demonstrated from sections of fragment of mucous membrane of the stomach removed by stomach-tube by Roger S. Morris.[1] There are also at times *heat*, *redness*, and *itching*. While the symptoms arise suddenly, subsiding often as quickly, the disease is likely to be prolonged.

Treatment.—Remedies calculated to increase muscular and nervous tone, such as strychnin, quinin, and iron, are indicated. In other respects the treatment is symptomatic, and directed to whatever symptoms demand attention. Morphin is sometimes necessary to relieve pain. Tracheotomy may be necessary to escape death by edema of the glottis.

INTERMITTENT HYDRARTHROSIS.

Intermittent hydrarthrosis is an affection allied to angioneurotic edema, being characterized by periodic swelling of one or several joints, without fever. The swelling may occur rapidly, sometimes, it is said, so rapidly as to be accompanied by a sensation as of water rushing into the joint. There is also pain and stiffness. The intermissions vary from 10 days to three months. It is sometimes associated with hysteria in women. To constitute the affection no other local symptom than those mentioned should be present and it should not be primary or secondary to any other condition. Nor should it develop into any other articular disease. It is to be distinguished from hydrarthrosis, the result of gout and rheumatism and traumatism. The prognosis is unfavorable and the disease appears to resist treatment.

The condition was described by Perrin in 1845 and 70 cases, according to Garrod, were reported up to the time of his writing.

Raynaud's Disease.

Synonyms.—*Local Asphyxia; Symmetrical Gangrene of the Extremities.*

Definition.—A vasocontractile disease characterized by three stages, more or less complete—viz.:

1. Local syncope.
2. Local asphyxia.
3. Local gangrene.

Symptoms.—The disease is more frequent in women—Raynaud's cases including 20 women and 5 men. It is also a disease of early life; the majority of Raynaud's patients were between the ages of 18 and 30, while five were between three and nine. The first phenomenon noticed is an unusual *pallor or anemia of the part*, resulting in marble-like whiteness and loss of sensation. This is the *local syncope*. Affecting, as it often does, the fingers and toes, these have been called dead fingers and toes. It follows exposure to cold, and to comparatively slight degrees of cold in those predisposed. The condition may disappear under warmth, and then only does pain manifest itself—when the parts are being thawed out, as the saying is. *Local*

[1] "Angioneurotic Edema," "American Journal of Medical Sciences," November, 1904

asphyxia follows, consisting in *engorgement*, the parts previously pale becoming purple and livid. The change is not simultaneous in all the fingers, some being still white while the others are livid.

The local asphyxia may succeed the local syncope, or it may come on independently of it. The tip of the nose and helices of the ears are the parts prone to cyanosis, but in addition to the fingers and toes the hands, feet, and arms and legs may be involved. A peculiar and striking mottling is the result on these large surfaces, produced by an alternation of various shades of purple with intervening lighter-hued spaces. In the darkest areas the capillary circulation is quite stagnant. There are also *swelling*, resulting *stiffness*, and *pain*, the latter often extreme and associated with an intense itching. But in Raynaud's disease there is perhaps more frequently *anesthesia* than pain. These are the phenomena, too, of chilblains, with which so many suffer in this climate with the approach of cold weather. In Raynaud's disease, as in chilblains, these symptoms may pass away in time under the influence of warmth; in fact, for a long time they occur only during the colder weather. A reaction takes place, and the parts assume a bright, red color in which the circulation is very active, and the anemia produced by pressure is rapidly replaced by an active hyperemia. The attacks may keep recurring for years without further effect, though in extreme cases there may be *loss of substance* in the ear-tips and fingers' ends, which in time may become indurated, uneven, and scarred from this cause.

The third stage of *local* or symmetrical *gangrene* is reached in a few cases only. In these the parts affected remain asphyxiated, and the phenomena of dry gangrene make their appearance. The fingers or toes, one or more, become black, dry, and cold, while gangrenous blebs appear in the parts adjacent to the sound tissue, a line of demarcation occurs, and the dead part sloughs away less extensively than at first seemed likely to be the case. Rarely, and only in cases of young children, does a fatal termination occur.

The symptoms that have been described may be said to be essential, but others also may be added of great clinical interest. One of these is *hemoglobinuria*, which is, of course, associated with a corresponding albuminuria. There are, at times, a few blood disks in the urine. Hemoglobinuria, when present, generally occurs at the same time with the cyanosis, and the attack has frequently been preceded by a chill. Other associate symptoms, less common, are scleroderma and edema, probably angioneurotic.

At other times *cerebral symptoms*, including torpor and partial loss of consciousness, are present; at others, epilepsy, mania, delusions, and even temporary hemiplegia. Dimness of vision is a symptom easily explained if we suppose there is a spasm of blood-vessels producing local retinal syncope. Other associated symptoms are peripheral neuritis with tingling and formication—neuritis being regarded as one of the causes of the disease; arthritic swelling; urticaria; erythema; also colicky pains, nausea, vomiting, and diarrhea.

Pathology.—Three chief theories have been brought forward to explain Raynaud's disease:

1. That it is due to endarteritis obliterans.
2. That it is caused by peripheral neuritis.
3. That it is the result of vascular spasm.

The intermittent nature of the disease is quite incompatible with its causation by endarteritis, which is progressively increasing in its effects.

It is true that some of the results of peripheral neuritis are similar to those of Raynaud's disease, but the frequency of the former affection as contrasted with the rarity of the latter militates, also, against this view.

The theory of arteriole spasm, suggested by Raynaud himself, best explains the symptoms. It is possible that endarteritis may be associated with spasm in advanced cases. The frequency of the disorder among women and children, whose vasomotor system is so impressible; its occurrence under the influence of cold, which is one of the most powerful exciters of vasomotor spasm; the frequent dimness of vision, which has been shown by ophthalmoscopic examination to be associated with contraction of the central artery of the retina; the occasional precedence of a chill; and the phenomena of hemoglobinuria, all go to show the probability of vasomotor spasm. Since the hemoglobinuria is likely to be associated with hemoglobinemia—which probably arises from the solution of hemoglobin liberated in the asphyxiated parts—such an origin for the hemoglobinuria must be admitted. The relation of Raynaud's disease to chilblains also affords an interesting field of investigation—in fact, has already been studied by Legroux.

Diagnosis.—It is not unlikely that Raynaud's disease and gangrene from endarteritis obliterans have been confounded. Raynaud's disease is limited to smaller areas, as the ends of the fingers, the tip of the nose and the helices of the ears. It is preceded by local pallor. Obliterating endarteritis affects larger vessels and limbs, especially the lower limbs, is less apt to be symmetrical, and is more likely to be fatal while Raynaud's disease is rarely so.

Prognosis.—This is not altogether unfavorable. Only delicate and feeble children, as a rule, perish, while it is quite possible, under favorable circumstances, to outgrow the tendency.

Treatment.—Persons subject to local syncope and local asphyxia should be protected from cold, and when the attack comes on, they should be kept warm, if necessary in bed, the parts being wrapped in wool and subjected to artificial heat. Friction may with advantage be associated. Galvanism and faradism are recommended.

B. B. Cates, of Knoxville, Tenn., has reported the successful treatment of Raynaud's disease by nitroglycerin in doses of 1/100 grain (0.00065 gm.) increased to 1/50 grain (0.0013 gm.) three times a day, and Harvey Cushing has reported recovery by the application of the tourniquet or rubber bandage to the affected limbs repeated frequently during the day.

Progressive Facial Hemiatrophy.

Synonym—*Unilateral Progressive Facial Atrophy.*

Definition.—A gradual progressive wasting of the bony, muscular, integumental, and adipose tissue of half the face.

Etiology.—That it is a trophic neurosis can scarcely be doubted. In one case—that of Mendel—which came to autopsy there was the terminal stage of a neuritis in all the branches of the trifacial. In Homen's case,

an acute one, and perhaps not strictly to be regarded as an instance of true facial hemiatrophy, a tumor was found pressing on the Gasserian ganglion and trigeminal nerve, but in similar cases of tumor of the ganglion facial hemiatrophy has not occurred. It has been observed in syringomyelia.

The disease usually begins in youth, but in a few cases it did not make its appearance until middle age. It is rather more common in the female sex. Sachs has collected 97 cases.

Symptoms.—The atrophy is much more frequent on the left side than on the right. It may begin as a circumscribed spot on the cheek or chin, or diffusely, involving first the subcutaneous tissue, the muscles, chiefly those of mastication, and finally the bones, especially of the upper jaw. In the cases which begin in early youth the muscles remain intact. The tissues of the orbit take part in the atrophy, and the eye appears sunken. The corresponding halves of the tongue and of the soft palate are sometimes involved. The hair on the same side may fall out and appears thin. The line of demarcation is sharp in the median line. In a few rare instances the disease is bilateral, and in a few cases also the atrophy involves the corresponding shoulder and arm. Sensibility is intact.

Diagnosis.—The disease, though very rare, can scarcely be confounded with anything else. The facial asymmetry associated with *congenital wry-neck* alone resembles it. Strümpell mentions a case of facial *hemihypertrophy* in a boy of 10 under his observation. Hypertrophy of one side or of one limb is also a rare condition.

Treatment.—A suitable treatment is the application of electricity to the atrophic side, alternated with massage, but it does not promise much.

Scleroderma.

Synonyms.—*Cutis tensa chronica; Sclerema; Dermatosclerosis; Glossy Skin.*

Definition.—A chronic, somewhat diffuse, indurated, hide-bound, and pigmented condition of the skin, trophoneurotic in origin.

Etiology.—This is obscure. It is more common in women than in men, and is most frequent in early adult and middle age.

Pathology.—The identity of scleroderma and morphea is claimed by some. We follow Louis A. Duhring in separating them, because both are capable of assuming a variety of forms which present entirely different clinical features at various stages. Scleroderma is much rarer than morphea. In the matured forms, while the epidermis is unaltered, there is increase of pigment in the lower layers of the rete, with a distinct overgrowth of connective tissue in the corium and subcutaneous connective tissue. Contrary to what would be expected, the sweat and sebaceous glands appear to be normal.

Symptoms.—The disease appears first in the neck, shoulders, back, chest, arms, and face. It begins usually as a stiffening of the *skin* which passes over into a *hard, tense, unyielding tissue*, resisting motion, and causing fixation and flexion. The patient is literally "*hide-bound.*" The hand, with its smooth, glossy surface, utterly without wrinkles, is striking

and distinctive. The change may involve the greater part of the body and even the whole of it. When less general, it is symmetrical. The condition passes insensibly into that of the surrounding healthy tissue. *Pigmentation* is usually a later symptom, but may be an early one.

There is generally *no constitutional disturbance* or other local symptom, such as pain, burning, and tingling, but more rarely these are present. The evolution of the condition is generally slow, requiring weeks and months, and when completed, it is likely to remain unchanged for months or years, or slowly passes away, leaving the skin normal. Rarely, however, an atropic state may succeed, producing such a shrinking or contraction that the integument is apparently bound to the bones, while over the joints the skin may become so fixed and immobile that ulcers and excoriations are easily produced.

Diagnosis.—The diagnosis rarely furnishes difficulty. In some stages it resembles morphea, from which it will be distinguished when that subject is considered.

Prognosis.—This should always be guarded, as the disease is often intractable, though recovery sometimes occurs.

Treatment.—Treatment of a curative kind is unknown. The patient should be thoroughly protected against cold, as he is exceedingly sensitive. Friction with oil is a rational means for softening the skin, and may give comfort, but does not check the spread of the disease. Cod-liver oil, iron, and arsenic are indicated. The constant electrical current has been recommended in the local forms. X-ray has given sume benefit in one of our cases.

Morphea.

Synonym.—*Keloid of Addison.*

Definition.—A trophic, asymmetrical neurosis of the skin, characterized by patches of skin firm in texture, white, pale pink, light yellow, or waxy hued, sometimes elevated, at other times depressed.

Etiology.—More common than scleroderma, it is also found more often in women, and at all ages. Its etiology is unknown, but its trophoneurotic origin is more than likely.

Symptoms.—The *patches* occur more frequently about the breasts and neck and sometimes in the course of nerves, such as the intercostal or lumbar, or on the face along the branches of the fifth pair. They range from 2/5 inch (1 centimeter) to four inches (10 centimeters) in diameter. There may be a *preliminary hyperemia* with itching of the skin and increased pigment deposit, or a milk-white *leukoderma from the beginning. The spots are dry*, without perspiration, sometimes scaly. Ultimately there may be *anesthesia*, in pinkish or purplish hyperemic spots or in small linear cicatricial-like areas which grow rapidly. In fact, the *rapidity of spread* of the spots is one of the most interesting clinical features. In the later stages there are often distinct *atrophy* and *cicatrization* with *pigmentation*. The spots may persist for months or disappear in a few weeks, and though more frequently persistent for a long time, they ultimately disappear spontaneously. The spots seem to be the *direct result* of a *cutting off of the circulation by a narrowing of the blood-vessels*. This may be by

compression by an inflammatory exudate, but is more likely to be a vasomotor constriction, probably due to irritation of the vasoconstrictor nerves.

Histologically there is a condensation of the connective tissue of the corium with a shrinkage of the papillary layer.

Diagnosis.—Morphea differs from scleroderma in that its lesions are more circumscribed, and in an absence of sclerodermic hardness. Pigmentation and cicatrization usually appear only in the later stages of morphea, while they are seen in the early stages of scleroderma before there is change in structure. Scleroderma is symmetrical in distribution; morphea is not. The atrophic striæ seen in one form of morphea closely resemble the lineæ albicantes of pregnancy or other cause of distention.

Treatment.—That recommended in scleroderma may be expected to be useful in morphea, especially arsenic, which is recommended by Louis A. Duhring. Here, too, the constant galvanic current is held to be of service, an extended trial being, however, necessary.

Ainhum.

Synonyms.—*Ainham; Quigila; Suhka Pakla, or Dry Suppuration; Pityriasis æthiopius; Scleroderma annulare.*

Definition.—A trophic disease resulting ultimately in the spontaneous amputation of one or more toes, especially the little toe, confined almost exclusively to male negroes.

Etiology.—It would seem that a moist, sandy soil and warm climate must have some influence in its etiology, but nothing definite is known. Its practical limitation to the colored race has been referred to. The operation of a pathogenic organism has been suggested, and the disease as an amputating leprosy. Traumatism has undoubtedly been associated with it.

Symptoms.—Ainhum begins as a furrow or crack at the digitoplantar fold, seen first on the inner side. In a few days the toe will swell and become the seat of a burning, shooting pain, which may extend into the foot and leg, though pain is not constant. The furrow increases laterally and in depth until finally the toe is constricted and the distal end becomes ovoid. The swelling subsiding, spontaneous amputation ultimately takes place, a dry scab forms at the furrow, and the case ends. It is not always confined to one toe, though it is as a rule. Sensation is not usually destroyed though it may be, and the nail remains unchanged. There are no constitutional symptoms.

The histology of the process has been studied by C. H. Eyles, who concludes that there is an ingrowth of epithelium with corresponding depression of surface, due to a hyperplasia that strangles the papillæ and cuts off the nourishment of the epithelium and causes it to undergo horny change. The bone changes are those of a rarefying osteitis, proceeding from the periosteum inward. According to Collas, it is an amputating leprosy.

The **diagnosis** is easy. There is no disease which resembles it.

The **prognosis** is favorable as to any danger to life. Its duration is from two to four years.

Treatment is unnecessary.

SECTION X.

DISEASES OF THE MUSCULAR SYSTEM.

MYOSITIS.

Infectious Myositis.

Definition.—A rare form of acute or subacute inflammation of striated muscle, due to unknown infectious agencies.

Morbid Anatomy.—Several cases have come to necropsy. The conditions found have been firmness, fragility, and fatty degeneration of the muscle substance, with serous infiltration and hyperplasia of the interfascicular connective tissues. In another case there was hyaline degeneration in varying degree without involvement of the intermuscular tissue.

Symptoms.—The parts usually involved are the extremities, but the disease may also invade the trunk-muscles and heart. There is swelling with slight edema, hardness, and stiffness, making motion painful and difficult. Instead of pain there is rarely paresthesia. The symptoms resemble those of trichiniasis, insomuch that it has been called pseudo-trichiniasis. In addition to the symptoms named an erythematous rash, irregularly scattered over the trunk and extremities, is regarded by Löwenfeld as characteristic. It is sometimes followed by slight pigmentation. There sometimes succeeds an atrophy of groups of affected muscles, and Wagner suggested that some of the cases may be examples of acute progressive muscular atrophy. Such cases are hardly fair examples of infectious myositis. The duration of the disease is from three months to three years.

Another form of infectious myositis is acute purulent myositis, sometimes associated with pyemia.

Progressive Ossifying Myositis.

This is a rare form of myositis, in which the muscles undergo progressive calcification, localized or extending over widespread areas. The disease is more common in males, and usually begins about puberty. It occupies many years in development, and consists in a preliminary inflammatory process, followed by more or less extensive deposits of bony plates throughout the muscular system, and at times in ossification of entire muscles, with fixation of joints and vertebræ.

Treatment.—No treatment has availed in any of these forms of acute inflammation.

PROGRESSIVE MUSCULAR DYSTROPHIES—PRIMARY MYOPATHIC FORMS OF MUSCULAR ATROPHY.

In addition to the spinal or myelopathic forms of muscular atrophy described under nervous diseases, there are several varieties of muscular wasting which apparently reside in the muscles themselves, and which are therefore strictly idiopathic. These forms occur in the young, and follow decidedly upon hereditary disposition. They are all probably the result of a congenital tendency to defective development.

There are several clinical types of primary muscular atrophy, of which the principal are:

1. Pseudo-hypertrophic muscular paralysis.
2. Erb's form of juvenile muscular paralysis or the scapulo-humeral form.
3. The facio-scapulo-humeral type of Landouzy and Dejerine.

These are all forms of one disease, called by Erb progressive muscular dystrophy. In very rare cases the atrophy has begun in the distal portion of the limbs.

1. Pseudo-hypertrophic Muscular Paralysis.

Synonyms.—*Pseudo-hypertrophy of Muscles; Lipomatosis luxurians muscularis* (Heller); *Atrophia musculorium lipomatosa* (Seidel).

Definition.—A state of muscular paresis associated with an atrophy of the muscles involved—an atrophy obscured by interstitial fatty overgrowth.

Etiology.—This is especially an affection of childhood, and heredity is an important causal factor, many members of the same family being sometimes affected through several generations. Boys are more frequent subjects than girls, though the disease is more likely to be transmitted through the mother, even though she may not herself be a subject. Heredity is not invariable. The disease usually begins before puberty, though sometimes as late as the 20th or 25th year or even later. Hysteria, epilepsy, feeble mindedness, with an occasional anomaly of the skull, have been observed in the same families.

Morbid Anatomy.—The nervous system is not involved except in rare cases. Minutely examined, the muscles exhibit marked differences in the size of the muscular fasciculi, some being wider, many narrower than normal, while there is considerable increase in the adipose and connective tissue between the fasciculi. The fibrillæ themselves are not fatty.

Symptoms.—The disease begins gradually with paretic symptoms, without the hypertrophic appearances which are later so pronounced. A child previously healthy exhibits clumsiness in its movements and insecurity on its legs, being especially awkward in jumping and running upstairs. Then close examination discovers that certain muscles or groups of muscles are enlarged, the calves of the legs being especially conspicuous. The extensors of the leg, the glutei, the lumbar muscles, the deltoid, triceps. and infraspinales next become enlarged, while the hands, arms, and neck are rarely involved, in strong contrast to the spinal atrophies. *Walking*

becomes more and more *difficult*, until finally a diagnosis may be made from the gait alone, which becomes waddling, while the shoulders are thrown back, the belly is thrown forward, the vertebral column being also arched forward in the lumbar region. The buttocks stand out, and the legs are far apart. In walking the legs are raised slowly, the toes dropping from paresis of the dorsal flexors. Especially characteristic is the child's method of rising from the floor. He first gets on all-fours, and raises his trunk by moving the arms along the floor. The arms are then drawn toward the legs until the knees can be reached, when, with one hand on the knee, he pushes himself up, then grasps the other knee, and completes the act of raising himself to the erect position. Late in the disease the same paretic condition may extend to the upper extremities, making it impossible to rise.

The enlargement of the muscles is due to an interstitial deposit of fat, and as a consequence the muscles are soft and flabby instead of hard and firm, as in true hypertrophy. Thus the hypertrophy is truly a pseudo-hypertrophy, the condition being really one of atrophy of muscular substance. Along with this may be associated a genuine atrophy of other muscles, with loss of substance unassociated with fatty infiltration, especially in the upper extremities. Very rarely there is a true hypertrophy, except of individual muscle-fibers.

Fibrillar twitchings are rarely present. *Electrical excitability* is diminished in proportion to the destruction of muscular tissue, but there is never a reaction of degeneration in a typical case. *Sensibility* remains normal, and the sphincters are intact. The patellar *reflex* is sometimes absent. The skin, especially of the legs, sometimes presents a peculiar bluish mottling. As a rule, the *intelligence* of the child *is preserved*, though sometimes there is mental and moral obliquity.

2. Erb's Juvenile Form of Progressive Muscular Dystrophy.

Synonym.—*Scapulo-humeral Form of Muscular Dystrophy.*

This type is also commonly found before the age of 20, usually between 15 and 20, but its subjects are not so young, as a rule, as those of the pseudo-hypertrophic form. It is, like all the forms of muscular dystrophy, hereditary in families of which female members are affected, while the boys may have pseudo-hypertrophic paralysis. It starts rather more frequently in the upper extremities, the upper arms and shoulders, but may begin also in the back and legs. The following are the *muscles involved*, according to Erb: In the upper extremities the pectoralis major, latissmus dorsi, and later the triceps; while there remain normal, at least for some time, the sternomastoid, the levator anguli scapulæ, the coracobrachialis, the teres major and teres minor, the deltoid, the supraspinatus and infraspinatus, and the small muscles of the *hand*, which, it will be remembered, are remarkably wasted in myelopathic atrophy. The muscles of the forearm, except the supinator longus, remain exempt for a long time, if not altogether. In the lower extremities the glutei, the quadriceps, the peronei, and the tibialis anticus are affected, while the sartorius and calf muscles are spared for a long time.

Very characteristic is the marked projection of the scapula, due to paralysis of the serratus. The gait in this form becomes waddling, and walking is ultimately impossible, although, like its congeners, the progress of the disease is slow, 23 to 28 years being the range of duration of cases described by Erb. Bulbar symptoms are rare, but the diaphragm may atrophy and death be due to respiratory deficiency.

The muscular changes are essentially atrophic, though in the beginning a few of the muscular fibers may be hypertrophied. The interstitial connective tissue is increased, its nuclei proliferated, and there is no interstitial fat. The number of muscle nuclei is also increased, and vacuoles may be seen in the individual fasciculi.

3. The Facio-scapulo-humeral Type.

This is also a family form. Duchenne called attention to the fact that in certain children's palsies the muscles of the face are involved in the atrophy, but the fact was overlooked until Landouzy and Dejerine opened the subject anew, and showed that this event is not infrequent—indeed, may be the first symptom. This atrophy may begin later in life—say the twentieth to thirtieth year. In these cases the eyes can no longer be completely closed, and whistling, laughing, and talking become difficult. An appearance characteristic, even diagnostic, known as the *facies myopathique*, results, to which the half-closed eyes, the sunken cheeks, and the tapir mouth contribute. The muscles of mastication, the internal ocular, and those of the forearm and hand remain normal. Fibrillary contractions are absent, and there is no reaction of degeneration. In other respects it resembles the juvenile form of Erb's palsy, with which it is closely allied. From what has been said it is evident that the three forms just described are modifications of one variety, a view strengthened by the fact that two or more of the types may be present in the same family.

THE PERONEAL TYPE OF PROGRESSIVE MUSCULAR ATROPHY.

Synonym—*Progressive Neural Muscular Atrophy.*

This form of atrophy, described by Charcot and Marie, and independently by Tooth, is met in the second half of childhood, seldom after 20. It occurs also in families, more frequently in males. It begins in the peroneal muscles, involving also the intrinsic muscles of the foot, and may lead to club-foot, of the variery pes equinus or pes equinovarus. The upper extremities may be affected after many years, and rarely it begins in the hands. It differs from the other forms of juvenile atrophy in the presence of fibrillary contraction and the occasional presence of the reaction of degeneration, while vasomotor and sensory disturbances may also be present.

Degeneration of the peripheral nerves has been found with ascending degeneration of the posterior columns, and some change in the lateral columns. Both the symptomatology and morbid anatomy of this, so far as known from a limited number of autopsies, go to show that it is a combination of neuritis and alteration in the spinal cord.

Prognosis and Treatment.—These are also essentially identical with those of progressive muscular atrophy.

AMYOTONIA CONGENITA.

GENERAL OR LOCALIZED HYPOTONIA OF THE MUSCLES IN CHILDHOOD.

SYNONYMS.—*Oppenheim's disease; Myatonia Congenita; Congenital Myohypotonia.*[1]

Definition.—A congenital affection consisting in atrophy with corresponding weakness of muscles (hypotonia and atonia), especially of the extremities and more of the lower; associated with loss more or less complete of the tendon reflexes. In advanced cases the weakness resembles paralysis, but closer observation discovers feeble contractions in the muscles but not sufficient to move the limbs. The muscles of the trunk and the neck are most rarely affected, while those of the eye, tongue and throat escape, as does the diaphragm, while the intercostal muscles may be invaded. The electrical reaction is affected proportionately to the hypotonia. Intelligence and sensation are undisturbed. Neither heredity nor family tendency seems to play any part. Although always congenital the symptoms are not always noticed immediately after birth. Oppenheim, who was the first to investigate it, believes the morbid change is in the muscles which are arrested in their development, and thinks it has no relation to *muscular dystrophy*. He admits the possibility of disease in the cells of the anterior horns of the cord whence it is, however, distinct, the latter developing acutely in a previously normal child.

Morbid Anatomy.—No case came to necropsy until one described by Spiller[2] in 1905 in which, however, no changes in the nervous system were found, but the muscles were wasted, in places almost absent, while a large amount of fatty connective tissue was present with increase of its nuclei. The muscles presented also in places a hyaloid appearance. Postmortem rigidity was delayed. The microscopic examination showed arrest of development of the muscular fibers, but no change in the central nervous system or peripheral nerves. Later necropsies have revealed alteration of the nerve cells of the spinal cord but in a few cases the lesions were purely muscular.

Diagnosis.—Congenital amyotonia differs from *progressive muscular dystrophy* especially in the absence of family tendency, in being congenital, and in the absence of progressive increase in the symptoms; from *amaurotic family idiocy* in that in the latter the symptoms increase and the ophthalmological changes are pathognomonic. It occurs in more than one member of a family.

Improvement has been observed in some instances.

MYOTONIA CONGENITA.

SYNONYM.—*Thomsen's Disease; Myohypertonia.*

Definition.—A hereditary affection, characterized by overdevelopment of muscles and by tonic cramp on attempt at voluntary motion.

Etiology.—The disease is always congenital, the symptoms making their appearance in early childhood and in family groups, more frequently

[1] Oppenheim suggested the name myatonia, but this word is so like myotonia (Thomsen's disease) that confusion has occurred. Amyotonea is used by English authors.

[2] Contributions from the Laboratory of Neuropathology, University of Pennsylvania, for the year 1905.

in men. Cases of acquired myotonia have been observed, but these are regarded as somewhat different from Thomsen's disease. A few isolated cases presenting the same symptoms have been described. It is to be regarded as a congenital anomaly of the muscular system.

Morbid Anatomy.—The muscles are characterized, especially in the extremities, by voluminous development in strong contrast to their power. In addition to an obvious macroscopic enlargement there is also found histologically an evident increase in the volume of the muscular fasciculi, recognized by Erb and confirmed by Hale White, together with intermuscular proliferation of the muscle nuclei and moderate increase of the connective tissue itself. The heart is exempt, but the diaphragm may be involved. There is no lesion of the spinal cord. The only necropsy in a case of Thomsen's disease ever observed was reported by Dejerine and Sottas. The muscles were altered, but the nervous system was normal.

Symptoms.—The disease manifests itself at first in childhood by a *stiffness* or "mild tetanus," in which the relaxation which necessarily precedes each muscular act is delayed. Voluntary contraction takes place slowly and with difficulty. The arm and leg muscles are involved, and thus the child's play is interfered with. There is, however, *no paralysis*, and after motion is started, it proceeds with facility. Prompt, rapid, and precise muscular movements are, however, difficult, and military service, for example, becomes impossible. Rarely facial, ocular and pharyngeal muscles are involved. *The condition is aggravated by cold and emotion.* Sensation and the reflexes are normal. Rarely there is mental weakness.

A peculiar reaction of muscle and nerve to *both* currents is developed, called the *myotonic reaction* of Erb. The motor *nerves* show quantitatively a normal faradic and galvanic excitability, and all briefly acting stimuli give short contractions; but with continuous irritation by both currents the contractions attain their maximum slowly and relax slowly, while vermicular wave-like contractions pass from the kathode to the anode. The *muscles* are also faradically easily excited, responding to a fairly strong current always with the above-described prolonged contraction. To galvanic irritation of muscle there is a slight increase of excitability, and to somewhat strong currents the contractions are sluggish, tonic, and continued. They occur only with current closure and not with current opening. The mechanical irritability of the muscles to strokes from the percussion hammer is also increased.

Diagnosis.—If more is needed than the peculiarity of the muscular phenomena, the electrical and mechanical muscular reactions described are characteristic.

Prognosis.—The disease is incurable, but patients become accustomed to the defect and conceal it as much as possible.

Treatment.—Nothing specific is known. Friction and massage, with muscular gymnastics, are rational measures to be recommended.

SECTION XI.

THE INTOXICATIONS.

The intoxications constitute a group of diseases caused by the action of certain foreign substances introduced from without, through the digestive or respiratory tracts or through the skin. They differ from the infections in that the toxic agent does not increase after entering the blood.

ALCOHOLISM.

Definition.—The effect on the human economy of the intemperate use of alcohol in some of the forms in which it is used as a beverage. Such effect is either acute or chronic.

Acute Alcoholism.

Definition.—This is the condition known as inebriety or drunkeness. Varying amounts of alcohol are required to produce it, very small quantities sufficing to intoxicate those unaccustomed to its use, while the habitual drinker may consume large quantities without effect.

Dipsomania is a term applied to a state in which there is an inherited immoderate desire for alcohol at times, followed by long periods in which there is no such desire.

Morbid Anatomy.—There is no permanent anatomical change in acute alcoholism, the congested condition of the entire body passing away with the drunken debauch.

Symptoms.—The order of symptoms is not always the same. More frequently the primary effect is one of excitement, associated with flushed face, bright eye, and loose tongue. To this succeeds the well-known staggering gait of drunkenness, which increases until its subject is unable to walk and finally falls to the ground. The ready speech, at first coherent, now wanders at random, and finally ceases altogether. The stage of narcosis is reached, and the drunken man breathes stertorously in his sleep, his face being congested and his breath alcoholic. He may, perhaps, be aroused, and may respond vaguely and incoherently to a question, but soon drops off to sleep again.

Another subject in the first stage is much more excited and even violent, and he may cry out boisterously, and either spontaneously or upon the slightest provocation inflict injury or even commit murder. In other subjects, again, there is no stage of excitement, and they are morose, or pass gradually and directly into stupor. The stage of inco-ordination and ultimate stupor comes, however, invariably if the quantity of alcohol drunk is enough to bring it about. The effect is upon the cortical nerve cells of the brain.

Other less conspicuous features are a lowered temperature—96° to 90° F. (35.6° to 32.2° C.), or even lower—involuntary evacuations of the bowels and bladder, dilated pupils, and muscular twitchings. The breathing may be slow and the pulse correspondingly slow.

Diagnosis.—The diagnosis of drunkenness is usually easy, yet mistakes are not infrequent; it has been mistaken for *apoplexy* or *apoplexy with fracture of the skull*. In the latter case stupor is usually deeper, and the patient cannot be aroused, while the breathing is more stertorous. The subject should always have the benefit of the doubt, and resident physicians in hospitals will often save themselves and the institution they serve much opprobrium if they will remember this. *Uremic coma* developing with convulsions also simulates drunkenness, and when the existence of Bright's disease is unsuspected, may cause error. In such the odor of alcohol in the breath is wanting, while that of urine is sometimes present, although, of course, a person with nephritis might have an attack of uremic coma after he had been drinking alcohol. In acute alcoholism the pupil is commonly dilated. In uremia it is variable, being sometimes dilated and sometimes contracted. In all doubtful cases the urine should be drawn by the catheter and tested for albumin and the phthalein test used. In *opium posioning*, which may also be confounded with alcoholism, the pupils are contracted.

Chronic Alcoholism.

Definition.—This is a condition of degenerative tissue metamorphosis more or less general which supervenes sooner or later in who use alcohol habitually and intemperately. Intemperance does not always imply the consumption of the same amount of alcohol, smaller quantities producing harmful effects in some persons while large quantities are apparently harmless in others.

Morbid Anatomy.—If we include under this the numerous morbid states which are directly or indirectly ascribed to the long-continued use of alcohol, such as cirrhosis of the liver, gastritis, low grades of meningitis, and the arterical changes so frequently ascribed to it, a large amount of space would be consumed. Fortunately, these conditions have already been described as separate entities, and their relation to alcohol as a cause has been discussed.

A few words may, however, be devoted to the consideration of the effect of alcohol on the cellular elements, since it is through such effect that its consequences are produced. A good while ago Lionel S. Beale called attention to the destructive effect of alcohol on protoplasm. More recently, in 1894, Obersohn, working in the laboratory of Gaule, in Zürich, demonstrated not only that alcohol, ether, and chloroform destroy cellular protoplasm, but also that the cells which are the most complicated, so far as function is concerned, such as nerve-cells, are the most vulnerable. These conclusions were confirmed by other experiments, among them Wilkins, in this country, in 1895, and the whole tendency of experiment and observation at the present day is to show the degenerative effect of alcohol on elementary histological units.

Chronic alcoholism, like acute, predisposes to other diseases. Its direct effect is, as already stated, mainly on the protoplasm of cells, modifying or impairing their normal metabolism, at times destroying cells and substituting them by fibroid material, at others inciting to inflammatory action; at others still simply delaying oxidation, as in the case of the a dipose vesicle, whose fat remains unoxidized because its congener, alcohol, is more easily oxidized. In some instances, as in the case of the liver-cells, fat is deposited in new situations because it cannot be sufficiently burnt up. Different kinds of alcoholic beverages also seem to act differently, some, as gin, producing *destruction* of liver-cells and cirrhosis, while others, as malt liquors, produce *fatty* livers. Friendwald in Welch's laboratory caused typical cirrhosis in rabbits by feeding them alcohol with degeneration of the liver cells. See also section on cirrhosis of the liver. It is also true that persons addicted to intermittent debauch are less liable to inflammatory lesions than constant consumers.

As a consequence of irritation of the intima by the alcohol, arise endarteritis, sclerosis, and thickening followed by atheroma and fragility. Irritation of nervous tissues results in meningitis neuritis and cerebritis.

Thirty years ago Lancereaux announced that alcoholic excesses are one of the principal causes of tuberculosis, affecting by preference the back of the right lung, while disease of the left in front is the result of insufficient aeration or defective alimentation; also that such disease is characterized by improvement and general arrest if the patient leaves off his habit, and by recurrence if he relapses.

The effect of alcoholism on the *kidney* is also two-fold in the direction of contraction and enlargement, the former due to gradual destruction of renal cells and tubules with substitution of interstitial tissue, the latter to fatty infiltration and hypertrophy. Tyson has often expressed the belief that alcohol is a less frequent factor in causing interstitial nephritis than was formerly supposed, because of the facilities for its elimination in its long journey from the stomach through the liver and lungs to the kidney.

Symptoms.—These may be classified to the systems they invade. Thus we have the effects of alcohol on the—

Nervous System.—The most constant of these is the well-known unsteadiness—especially of the hands in the performance of muscular actions. It is also apparent in an attempt to protrude the tongue. Gradual mental deterioration is an inevitable consequence, sooner or later, of chronic alcoholism. It is manifested in sluggishness of intellect, in weakness of resolution, a loss of moral character, in irritability, restlessness, and occasional dementia and insanity. Yet it is surprising how some enormous consumers of alcohol maintain their mental acumen and ability to manage large financial interests, while their vascular and digestive apparatus is evidently the seat of advanced degeneration. When dementia and insanity are present, they are probably due to vascular degeneration and consequent secondary changes in the brain structure. The tendency of such insanity is toward delusions, including suspicion, distrust, fear of impending evil, and, more rarely, delusions of grandeur, as in general paralysis of the insane.

Multiple and simple neuritis is a well-recognized and almost characteristic symptom of chronic alcoholism, and has already been considered.

Pachymeningitis hæmorrhagica is sometimes met. More frequent are slight thickening and turbidity of the pia arachnoid membrane. But this is not peculiar to alcoholism, being the same as that found in the neuroses of insanity.

A gradually deepening drowsiness culminating in coma, sometimes presents itself terminating fatally without the symptoms of kidney disease. There may be no changes in the brain of one thus dying or there may be a slight edema known as "wet brain" or in protractive cases, there may be encephalo-meningitis with adhesions of the membranes.

Digestive Apparatus.—This is a favorite point of attack in alcoholism. Chronic gastric catarrh is one of its most frequent consequences, producing loss of appetite, nausea, constipation, coated tongue, and foul breath, symptoms which are always worse in the morning, and are temporarily relieved by the dram which the habitual drinker is apt to seek at this time of day. Autopsy in such cases may be negative as to the stomach, or reveal the changes described under chronic gastric catarrh.

Symptoms due to Liver Changes.—From these arise the symptoms due to cirrhosis and contraction, fatty infiltration, and enlargement. The interstitial overgrowth so charactertistic of cirrhosis is probably secondary to a primary poisonous and destructive effect of the alcohol on the cells, as confirmed by the experiments of Weigert, and later by those of Obersohn and Wilkins, previously referred to. The compression of the cirrhotic liver on the portal vessels produces secondary effects, viz., hyperemia of the stomach, causing gastric catarrh; hyperemia of the rectum, producing hemorrhoids; and of the esophagus, pharynx, and nasal mucous membrane, resulting in hemorrhage in any one of the localities; in dilatation of the venulæ of the face and nose, and eruptions on the latter, constituting the acne rosacea or "blossoms," by which the toper is so often marked. In many cases, on the other hand, the livers of hard drinkers have been found normal.

From vascular changes result cardiac and renal disease, and their symptoms, unequal distribution of the blood in the brain, and consequent symptoms, viz., dizziness, thrombosis, apoplexy, softening.

Delirium Tremens, or Mania a Potu.

Definition and Symptoms.—This is a special manifestation of chronic alcoholism, ascribed to the long-continued action of alcohol on the brain, though its occurrence coincides rather with the sudden withdrawal of the drug. On the other hand, a debauch, however prolonged, by a person previously temperate, is rarely followed by mania a potu, so that the relation of the illness to the withdrawal of alcohol may be more apparent than real. Purely accidental circumstances may determine the cessation from drinking. It is very frequently an attack of acute illness, especially pneumonia, to which drunkards are especially predisposed. The first symptom is usually sleeplessness associated with intense depression, or there may be intense restlessness, during which the patient, unless restrained, will go out of the house on some imaginary business. To this succeed hullucinations of visions as the result of which he imagines he is pursued by monsters, serpents, rats, mice, and other vermin. The intense shivering terror of the victim under

these circumstances is pitiable, and the "horrors"—a term applied to the disease—is but a feeble expression of the terrors of the patient. Frequently in his attempts to escape these objects, he is unmanageable, and must be confined. Suicide is not infrequent with such patients. At other times the eager though misguided intelligence displayed in watching the imaginary objects is amusing. Auditory hallucinations may be present, and unusual noises be complained of. At the same time, even though the patient is violent, the pulse will be found frequent feeble, and often irregular. There is great muscular weakness, as evidenced by the tremor which accompanies all muscular acts. There is slight fever, 102° to 103° F. (38.9° to 39.4° C.), which is increased if there is intercurrent inflammatory disease.

Diagnosis.—This is never difficult. The symptoms somwhat resemble those of *meningitis*, and meningitis is also sometimes present, but with the history of the case and the general appearance of the patient a mistake is not likely to be made. It is most important, however, to examine each case thoroughly, as pneumonia is so frequently associated with delirium tremens and constitutes its most serious danger. Again *pneumonia of the apex* is sometimes accompanied by delirium similar to that of delirium tremens.

Prognosis.—If there is pneumonia, recovery is a rare event, but if delirium is uncomplicated, recovery generally takes place, certainly from the first attack, and generally even after one or more attacks and a duration of from three or four days to a week. If recovery does not take place, the adynamia increases, the pulse grows increasingly feeble, the tongue dry, the delirium becomes muttering, and the patient dies with the usual symptoms of the typhoid state. The event is, of course, more common in hospital practice.

Prophylaxis.—Alcohol is a poison. It is true that a certain amount may be burned as fuel when taken without harm to the individual. There is no measure to that amount however until mischief is done. The only safe rule is absolute abstinence. If this were followed, the directions given below, would be unnecessary.

Treatment of Alcoholism.

Acute alcoholism rarely requires any treatment except restraint from the further use of alcohol and opportunity to sleep off the debauch. A full dose of chloral—from 15 to 30 grains (1 to 2 gm.)—may be necessary when there is extreme excitement. Morphin is indicated, but caution should be exercised in its use. In cases where the subject is not to drunk too swallow, a dram (3.7 c.c.) of aromatic spirit of ammonia often acts happily. When there is reason to believe that alcohol or undigested food is in the stomach, the stomach should be washed out, preferably with a stomach tube. Should it be deemed desirable that an emetic be given to one unconscious, apomorphin hypodermically administered is the best—from 1/15 to 1/10 grain (0.0044 to 0.0066 gm.).

The first step in the treatment of *chronic alcoholism* is the withdrawal of the poison. Except when mania a potu is present, this may be total. Nothing is to be gained by gradual withdrawal, while it only prolongs the struggle. No drugs like morphin or chloral or cocain should be used in the treatment of chronic alcoholism, as to do so is simply to substitute one

evil for another and to weaken the resolution of the victim. The bromids may, however, be availed of, and trional, chloralamid, and sulphonal may be employed to procure sleep. Not less than 15 grains (1 gm.) of any of these drugs should be administered for an adult, while twice the dose may be necessary. Hydrobromate of hyoscin is often an admirable remedy to quite excitement. It may be given in doses of 1/96 grain (0.0007 gm.). Attempts at reformation are rarely successful, but success is not impossible. The first and most important element of the treatment is a conviction on the part of the patient that alcohol in the very smallest amount is harmful to him; this must be accompanied by a firm resolution to resist the sometimes well-nigh overwhelming desire for drink, and his willingness to be truthful and open with his medical advisers and his friends; without this firm resolution almost any treatment is useless. Some means of restraint is usually indispensable, and as a rule can only be secured in an institution. Unfortunately, relaxation of this is apt to be followed by a relapse. The difficulties increase in the presence of hereditary tendency. An abundance of nutritious food should be insisted upon, as it is found to be the best substitute for alcohol, while tea and coffee may be allowed freely, having the advantage of being stimulating without intoxicating. Tonics, such as strychnin 1/30 grain (0.0022 gm.) three or four times a day, or quinin, should be administered, none of the specific treatments.

As to the remainder of treatment, it must be mainly symptomatic, directed to the symptoms as they arise. Neuritis, one of the most important of these, has been elsewhere considered.

Still another drawback is the intense depression which succeeds the exciting effect of alcohol and often impels to a return to its use.

Treatment of Mania a Potu.

Alcohol should as a rule be immediately withdrawn. There are, however, certain cases where a weak pulse and dilated heart are improved by a continuation of the alcohol.

The first indication after withdrawal of the alcohol is to secure sleep. For this purpose the soporifics previously named scarecely suffice, though they may be tried in the full doses specified. Especially may we hope to obtain some results from the hyoscin in doses of 1/96 grain (0.0007 gm.) given hypodermically. In many cases of delirium tremens it is scarcely possible to do without morphin, which may be given hypodermically in 1/4 grain (0.0165 gm.) doses, caution being observed not to repeat too often. Chloralose may be given in from 5 to 10 grain (0.33 to 0.66 gm.) doses, dissolved in warm water; it has the advantage of small doses, equal in effect to the largest of chloral, while in also diminishes tremor and has no harmful secondary effects. 20 grains (1.32 gm.) of trional, mixed in water, with 10 minims (0.62 c.c.) of tincture of capsicum, after a calomel purge is useful. A very hot bath is given, of which the temperature is gradually lessened. If in 30 minutes the delirium shows no signs of abatement, 10 grains of trional (0.65 gm.) are again given. In all cases forced feeding in small quantities often repeated is practised, the diet consisting of milk, eggs, and soups. Paraldehyd in fluidram (3.7 c.c.) doses is a remedy which may be expected to be of service. A cold bath sometimes has a tranquilizing

effect, especially if there is fever, or sponging the body may suffice. Many things must be done to keep the patient occupied, because, after all, the treatment amounts for the most part to a conflict between the patient and faithful attendants and the irrepressible and, at times, almost maniacal desire of the patient to get away. In preventing this it may sometimes be necessary to confine him to bed, but all gentleness should be exercised in carrying out this measure. It is much better to use a folded sheet than the unsightly straps which are sometimes used in hospitals.

With the idea that the symptoms are due to toxic substances produced within the body by the disassimilation of alcohol, purgation has assumed greater importance with a view to elimination. For the same reason diuretics are advised, diuresis as well.

As a rule it is much better to stimulate with the aromatic spirit of ammonium, digitalis, and strychnin, one dram (4 gm.) doses of the first, 10 minims (0.62 c.c.) of the second, and 1/30 grain (0.00022 gm.) of strychnin being given every three hours to overcome such weakness. Even larger doses may be demanded by emergencies. Nourishing food in easily assimilable shape, repeated at short intervals, should be insisted upon as the best subsititute for alcohol. With the first sound sleep comes, usually, relief and the patient awakes convalescent, unless, as already said, the mania is accompanied by acute disease, like pneumonia, when death is apt to be the termination, whatever our efforts.

A failing heart and insufficient kidney secretion may be overcome by hypodermic injections of spartein and caffein.

The treatment of dipsomania is most difficult because the conditions is less tangible. It is the previous condition that requires treatment.

THE MORPHIN HABIT.

Synonyms.—*Morphinism. Morphinomania.*

Definition.—An irresistible craving for morphin, which is commonly used in gradually increasing daily doses to meet the demand. Periodic attacks, or "morphin sprees," comparable to alcohol sprees, during which large quantities are used also occur.

Etiology.—The morphin habit is most frequently acquired as the result of long-continued administration of morphin, to relieve some suffering due to a painful or incurable malady or for insomnia. The influence of heredity in favoring the formation of the habit is acknowledged. Neurotic persons are more apt to become its victims. The victim of alcohol often becomes a morphin fiend, being deluded by early experience with the drug to believe that he can thus overcome the previous more disgusting, if not more terrible, habit. The same is true of cocain. Opium smoking such as is practised in the orient appears to be less harmful than the ingestion of opium by the mouth or hypodermically.

The quantities consumed are often enormous, as much as 400 grains of opium (25.92 gm.) as a daily dose being reported.

Symptoms.—The chief symptom is, of course, the *craving for morphin*, but it brings with it others which are more or less temporarily relieved by a dose of the drug. Among these are *irresolution* and *loss of self-*

control, and a moral obliquity similar to that induced by alcohol, especially in women, who are the most frequent subjects. *Untruthfulness*, especially with regard to the drug and the quantities used, is habitual. Epigastric *pain* or *nausea*, or both, are frequently complained of toward the time when another dose is due, though whether this is actual or feigned is not always easily determined. *Mental depression* is a more constant and characteristic symptom, associated with *intense anxiety*, *restlessness*, and a *sense of impending evil*, both relieved for a time by the dose. All of these symptoms are increased by a more prolonged withdrawal of the drug, when the mental depression becomes intense, sometimes impelling to suicide So far from the usual constipating effort of morphin being produced by the drug thus used, diarrhea is not infrequent. So too the contraction of the pupil usually produced by opium is substituted by dilatation.

As the habit is prolonged *tremor*, *paresis*, and more rarely *ataxia* are superadded, while diffuse and neuralgic *pain* is complained of. *Sleep* is irregular, digestion is bad, and appetite and nutrition fail, the pulse becomes feeble and rapid, vasomotor derangements appear, as shown by a tendency to sweating and by dilatation of the pupils. Except when under the direct influence of the drug the patient grows weak and becomes a ready victim to acute disease.

On the other hand the opium eater sometimes attains old age, presenting a wizened, sallow appearance quite characteristic. The pleasurable effect so often ascribed to opium is rarely realized, though it is not unlikely that a certain amplification and distortion of actual facts which may arise in the dreamy state may form the basis of such weird and beautiful fancies as are pictured by DeQuincey.

Diagnosis and Prognosis.—With an accurate history the diagnosis is easy, otherwise it is difficult; but the prognosis is exceedingly uncertain because of the difficulty in carrying out treatment.

Treatment.—Successful treatment is scarcely possible outside of an institution, and even within one serious difficulties beset the way, the chief of which is the deception practiced by the patient. Patients should be divested of their own clothing and put to bed in hospital garb, because in this way alone can we be sure that morphin is not concealed about the person. Whenever possible, a special nurse should be assigned to each case. The latest testimony favors complete and sudden withdrawal of the drug as furnishing a short struggle, though a severe one. Such treatment is usually followed by diarrhea, vomiting, and insomnia. In most cases it is impossible to secure the consent of the patient to sudden and complete withdrawal, when the gradual plan must be adopted. The success of either plan depends on securing effectual control of the patient, and if this cannot be obtained, all efforts fail. Some counsel even that no adjuncts should be employed, but certainly there can be no harm in the employment of general tonic treatment and remedies directed to the irritability of the stomach and torpor of the liver. A calomel purge is useful at the start. It is a well-established fact that, as in alcoholism, the patient should be well nourished, given such food as milk, cream, beef-juice, or beef peptonoids, rich broths, and beef-tea. When there is great asthenia, aromatic spirit of ammonium, strychnin, and digitalis may be given as directed under

alcoholism. If possible an occupation of an absorbing kind should be furnished.

To promote sleep, one of the numerous hypnotics in which the present day is rich should be given. Chloralamid is probably the best of these. It is not easy of administration, because of its pungent taste and difficult solubility. Twenty grains (1.32 gm.) or 30 grains (1.98 gm.) are a moderate dose, and are easily soluble in a fluiddram (3.7 c.c.) of a mixture of two parts alcohol and one part glycerin. Of such solution two teaspoonfuls should be given in a glass of sherry wine or four tablespoonfuls of milk at the ordinary temperature. Trional, sulphonal or veronal may be given in from 15 to 20 grain (0.99 to 1.32 gm.) doses dissolved in hot water. Hyoscin in doses of 1/100 grain (0.0007 gm.) may also be tried. Chloral may be used in doses of from 10 to 30 grains (0.66 to 1.98 gm.). If there is cardiac weakness, the dose should not exceed 10 grains (0.66 gm.). Chloralose may be given in 5 grain (0.33 gm.) doses in wafers or in hot milk.

Too much carelessness is practiced by physicians in placing morphin in the hands of patients to be used at their pleasure. The hypodermic syringe has wrought untold mischief, and should never be placed in the hands of patients. On the other hand, when morphin is judiciously ordered for patients suffering extreme pain only, it is very rarely the case that a habit is established.

CHLORALISM.

Definition.—The chloral habit or the habitual use of chloral.

This habit is sometimes acquired when the drug is used to obtain sleep or prescribed by the physician for any purpose.

Symptoms.—For symptoms and treatment of *acute chloral poisoning* see page 1176.

The presence of the *chloral* habit is characterized by nervousness, mental weakness, and depression of spirits, even to a degree of melancholia. There may also be general weakness, characterized by muscular tremor and cardiac palpitation. Lowered temperature is characteristic. These symptoms are aggravated by sudden withdrawal of the drug. There is sometimes dyspnea, aggravated at meals or after exertion. Mania and dementia are reported.

Various skin eruptions or a tendency toward them are a symptom. Though there may be no eruption, the slightest exertion or a glass of wine will produce an intense erythematous redness on the face and elsewhere on the body. This erythema, which may also extend to the mucous membranes is ascribed to vasomotor weakness. There may be diarrhea from the same cause.

Treatment.—Treatment requires the gradual withdrawal of the drug and cardiac stimulation by ammonia and digitalis, the use of nutritious food, tonics, massage, and electricity. For insomnia if needed, sulphonal or trional, administered as previously directed, are more suitable than chloralamid. In extreme cases morphin may be used. It is not usually difficult to master the habit.

COCAINISM.

Cocainism has become a comparatively frequent modern habit. It is especially common among physicians, some of whom acquire the habit in tentative local applications to their own mucous membranes in the treatment of patients. We have known three successive chiefs of clinic in throat and nose dispensary service to acquire the habit. Cocain is also taken as a substitute for some other drug, and its subjects are very apt to be those with neuropathic tendencies. It is usually snuffed, frequently in the form of one of the powders on the market for the relief of coryza.

Symptoms.—The effect is a total demoralization of the individual, who loses all moral responsibility, delaying and neglecting appointments in the most remarkable manner. There is volubility of tongue, suggesting alcoholism, and the presence of hallucinations, which also resemble those of the alcoholic effect. The eyes are bright, and the pupils are dilated. The subject becomes suspicious, charging his wife with infidelity, and his best friend with persecuting him. Hallucinations of hearing, sight, and smell are sometimes present, including tinnitus aurium. Mild epileptoid seizures, with partial loss of consciousness, may occur, limited to muscle groups, as about the eyes. Nystagmus is also a symptom. The pulse becomes weak and feeble. The symptoms are often associated with those of alcoholism and opium.

A symptom to which a certain amount of diagnostic value has been attached is a sensation of foreign bodies under the skin. In one case observed by Rybakoff of Moscow, was a sensation as of worms beneath the skin. The recognition of the symptom is ascribed to M. Magnan of Paris.

Treatment.—The sale of combinations of cocaine to the public should be prohibited. If the habit be uncomplicated, treatment is promising. It mainly requires withdrawal of the drug, which should be total. The assistance of a trusty nurse or friend is essential, but it is not often necessary to remove uncomplicated cases to a sanatorium. Cases complicated with alcoholism or the opium habit are more difficult to handle, and incarceration in an institution becomes necessary.

Tonics of the usual kind—strychnin, in full doses, and quinin—should be ordered. Non-intoxicating stimulants, like ammonia and coffee, should be given to counteract the depressing effect, while good, nourishing, easily assimilable food is necessary.

LEAD-POISONING.

SYNONYMS.—*Colica pictonum; Plumbism; Saturnism; Devonshire Colic.*

Definition.—A disease of manifold symptoms resulting from the toxic effect of lead on the system, having its subjects mainly among workers in lead-works, and among painters, glaziers, and plumbers.

Etiology.—The lead enters the system by inhalation, through the digestive tract, or by the skin. Almost without exception the cases we have had in hospital were from the lead-works in the neighborhood of Philadelphia. The Philadelphia Hospital is almost never without one or

more such cases. Water which has been kept in lead tanks, or even painted tanks, or water passed through lead pipes, has produced the disease. It must, however, be very pure water, such as rain water, and it is the very impurities of our drinking-waters which protect us. Almost all drinking waters contain sulphate of lime, the sulphuric acid of which combines with the superficial layer of lead and forms an insoluble coating of sulphate of lead which prevents further solution.

Accidental contamination has been caused by the use of cosmetics and hair dyes. To the use of chrome yellow as a substitute for eggs for coloring were traced a number of cases occurring in Philadelphia in a very interesting study by D. D. Stewart.[1] Even vegetables canned in tin vessels are held to have produced lead-poisoning. But the best manufacturers now up safe canned goods.

Among the more rare cases of lead-poisoning may be named materials used in making rag carpets,[2] cooking in badly glazed crockery-ware, beer drawn through lead pipes, or beer, cider, and wine from bottles which have been washed with shot of which some have been left behind, the use of snuff packed in spurious tin-foil containing lead, and from sleeping on mattresses the hair in which was dyed black by some lead-containing substance; and one, a most incredible case, mentioned by Naunyn, is that of a proof-reader who was poisoned after many years' reading of printed proof. Notwithstanding the solubility of the acetate of lead so much used in medicine, it is very rare that poisoning has resulted from its administration, and there need be no fear of using it for the purposes in which it is indicated until at least 2 drams (7.4 gm.) have been given. Cases of poisoning by lead administered as a medicine are reported by Taylor and other toxicologists. Osler tells of four cases at Johns Hopkins Hospital following the use of lead and opium pills for dysentery. To these J. Milton Miller has added two interesting instances and reviewed others.[3]

That the lead itself lodges in the tissues is easy of demonstration, and analysts have gone so far as to determine the exact quantity in the different tissues of animals poisoned by lead; which, by the way, is surprisingly small, the largest amount found being 1/4 of one per cent. in the bones, while that in the muscles was but 2/1000 to 3/1000 of one per cent. On the other hand, it would seem that lead is contained in the tissues of many persons who are healthy—according to J. J. Putnam, in 25 per cent.

Most cases occur among adults, usually between the ages of 30 and 40, but in children occasionally. Women are said to be more predisposed than men, as four to one, and to be more readily brought under its influence. Yet one seldom sees a case of lead-poisoning in women, because they are less frequently exposed to the cause.

The period of exposure necessary to produce lead-poisoning varies greatly, from a month or less to many years.

Morbid Anatomy.—This is not striking. Tissue may contain a considerable amount of lead without exhibiting changes. Fatty degeneration and fibrosis are, however, characteristic. Thus, the muscles become fatty and fibroid. The kidneys gradually lose their parenchymal cells

[1] "Philadelphia Med. News," June 18 and December 21, 1887.

[2] A very interesting case thus caused is reported by J. Milton Miller and G. Oram Ring in the "Amer. Jour. of the Med. Sciences" for February, p. 193, 1896.

[3] Lead-poisoning from the therapeutic use of lead-acetate in capsules with a report of two cases. "Therapeutic Gazette," Aug., 1904.

and become fibroid, while nerves exhibit fatty degeneration. In the spinal cord are found in chronic lead-poisoning the changes characteristic of anterior poliomyelitis—*i. e.*, sclerosis of the anterior cornua, with atrophy of the cells and nerve fibers, but the remainder of the cord and nerve-roots are not altered. Demonstrable changes in the central nervous system, even when there are symptoms of lead encephalopathy, are not numerous. In 32 out of 71 cases Tanquerel found none. Von Monkalow discovered a high degree of atrophy of the cortex, especially marked over the frontal region at the vertex, and in the crura cerebri. Small hemorrhages in various parts of the brain and atheroma of the arteries have been noticed; also overgrowth of connective tissue. Severe enterocolitis has been found in acute cases. Arterio sclerosis is common.

Symptoms.—While the symptoms which make known the presence of lead-poisoning are at times rapid in their development and at others slow to appear, there seems on this account scarcely sufficient reason for dividing them into two classes of acute and chronic.

The most striking of the symptoms, and often the first to which attention is called, is *colic*. Indeed, it, with constipation, next to be considered, is often the sole manifestation of the disease, and from these two alone a diagnosis may be considered, after exposure to lead absorption. The term *lead colic* has long been a recognized term in medical terminology. The pain is most frequent in the region of the umbilicus, and is often relieved by pressure. It varies greatly in degree, being sometimes a simple grumbling pain, at others of extreme severity, the patient writhing in the paroxysm. This, as a rule, does not last long, but is soon followed by another. On the other hand, it may continue for hours or until relief is afforded by treatment. It is probably due to powerful contractions of the muscular wall of the intestine, by which the nerve filaments distributed through it are compressed. As contrasted with flatulent colic, the abdomen is not distended, but flat, and may even be contracted, sometimes so much so that it is said that the vertebræ may be discerned through the abdominal walls. Yet distention of the abdomen is occasionally present. The pulse during the attacks of colic is often strikingly slowed, having been noticed as infrequent as 30 beats in a minute.

Groups of muscles anywhere, and especially the flexor muscles, as of the arms and legs, become involved in cramp, the latter more frequently. There may also be cramps in the fingers and toes. In addition to these painful cramps, which, like the colic, are intermittent, there is pain in the neighborhood of the joints. The sum of these painful joints and muscles has received the name *arthralgia saturnina*. They are quite frequent, occurring, according to statistics of Tanquerel, in 755 out of 2151 cases.

Constipation is very common, even more commonly present than the colic, and yet it is not invariable, and may even be substituted by *diarrhea*.

A *blue line* on the patient's gums is a very characteristic symptom, and appears at the border of contact of the gums with the teeth, or just above it. As a rule, it is easily recognized when present. It is caused by the presence of sulphuret of lead, produced by the action of sulphureted hydrogen upon the lead in the tissue of the gums. Hence the line is more common and distinct on the gums of those who take no care of the mouth,

and in whom sulphureted hydrogen is generated in the decomposition of the food. This line often remains after all other symptoms have subsided, and although it is not invariably present, its disappearance may be considered as quite a certain sign that the lead has been practically eradicated.

Anemia is a very constant symptom in lead-poisoning, and its higher degrees are attended by a sallowness which early gave rise to the term *icterus saturninus*, but which is in no way due to a deposit of bile pigment. In more serious cases, too, the impaired nutrition results in an *emaciation* which is sometimes extreme. Along with the anemia there is often *loss of appetite*, and frequently a *sweetish* taste and *fetid breath*.

Degeneration of the Red Cells.—Comparatively recent studies have found associated with lead-poisoning in common with other toxic conditions a granular degeneration of the erythrocytes. The granular change which responds to the basophilic stains was first investigated by Geelmyden, Hansemann, Von Noorden and others, but Grawitz was the first (1889) to lay particular stress on the condition as evidence of a special form of degeneration. It would appear from the recent studies of Alfred Stengel, C. Y. White and Wm. Pepper, 3d,[1] that no poison thus far studied is as regular in its production of degeneration or as prompt in its action as lead. Cadwallader has also shown the occasional occurrence of nucleated red corpuscles in the blood of lead-poisoning.

Another symptom of great importance is *muscular paralysis*, which may be *localized or general*. The localized palsies, in contrast with muscular cramp, are more likely to involve extensor muscles than flexors, and especially those of the wrist, giving rise to the very characteristic symptoms known as "wrist-drop," which, in Tanquerel's experience, occurred in 107 out of 2151 cases. Usually it is not until the colic and arthralgia present themselves that the paralysis appears. On the other hand, it has been the first symptom observed. It may last but a few days, or it may resist all treatment. It may affect a single muscle or groups of muscles. It is further characterized by the fact that the muscles affected are subject to rapid and extreme *atrophy*, so that they seem almost to disappear. Dislocations of the more movable joints, as the shoulders and phalanges, may occur in consequence. While sensibility is but slightly impaired, *electro-muscular contractility* rapidly *disappears*. The muscles cease to respond to the faradic current, while the reaction to galvanism is unchanged or slightly increased at first.

The palsies of lead-poisoning are due to *neuritis* which may also be acute.

The localized forms of lead palsy are divided by Madame Dejerine-Klumpke in her masterly monograph[2] into the following groups:

1. The *antibrachial type*, the most frequent of all forms, and in which the musculo-spiral nerve is involved, producing paralysis of the extensors of the fingers and the characteristic wrist-drop, the supinator longus usually escaping. As the result of the prolonged flexion of the wrist there may be slight displacement backward of the ends of the bones with distention

[1] "Further Studies of Granular Degeneration of Erythrocytes," "American Journal of the Medica Sciences," May, 1902.

[2] Des Polynéorites en general et de Paralysies et Atrophies Saturnines en particuliar par Madame Dejerine Klumpke, Paris, 1890.

of the synovial sheaths producing the so-called Gruebler's tumor over the wrist.

2. The *superior* or *brachial type* involving the deltoid, the biceps, the brachialis, the supinator longus and rarely the pectoralis. It is much rarer than the anti-brachial type. The atrophy is of the scapula-humeral type and is commonly bilateral. It may be primary or secondary to the first form.

3. The *Aran-Duchenne* type in which the small muscles of the hand and the thenar and hypothenar muscles are involved producing a paralysis like that of the early stage of polio-myelitis anterior. This group seems to be always primary and may be the first manifestation of lead intoxication.

4. The *peroneal* type in the lower extremities producing foot-drop and steppage gait, due to paralysis of the peroneal muscles, of the common extensor of the toes and of the extensor proprius of the great toe.

5. *Laryngeal* form invading the adductor muscles of the larynx as noted by Morell MacKenzie.

In the generalized palsies the invasion may be gradual, beginning in the wrist and ankles and extending gradually over the body, or it may extend rapidly becoming complete in a few days.

The *central nervous system* may also be invaded by lead-poisoning. Occurring usually in those who are peculiarly exposed. The symptoms come on in from eight days to 50 years, the majority showing themselves, according to Tanquerel, within the first nine months. The most frequent mode of manifestation is in *eclampsia* independent of Bright's disease. True epilepsy may follow these convulsions. But there may be headache or amaurosis, optic neuritis, apathy, stupor, or the opposite condition of maniacal excitement or melancholia and hallucinations. In a few cases of lead-poisoning the symptoms are limited to the central nervous system—in 72 out of 1390 cases observed by Tanquerel. *Tremor* of the paralyzed muscles is a frequent nervous system.

A frequent complication, more especially when it has been present for some time, is *interstitial nephritis*, and its resulting morbid product, the contracted kidney, as shown by the presence of a small degree of albuminuria and hyaline tube-casts; and as this is the form of kidney disease in which uremic convulsions are most frequent, it is evident that these must be distinguished from the convulsions just referred to as part of saturnine encephalopathy. Hence an examination of the urine in every case should be early made in the study of the case. *Arteriosclerosis* is often a direct result as well as *hypertrophy of the heart.*

Prognosis.—As to prognosis, it depends largely upon the degree of saturation of the system with lead. Ordinary lead colic is commonly followed by recovery. As a rule, therefore, persons who respond most quickly to the action of the poison are those who most promptly recover, provided, of course, they are removed from the influence of the lead, for such persons, too, being most suspectible, are in great danger from prolonged exposure. We are enabled to infer something of the prognosis from the symptoms which are present. If the attack be ushered in by a colic, and there be no other symptoms except constipation, and a lead line on the gums we may confidently expect our patient to recover completely.

If there be arthralgia and palsy, the prospect is less certain, still less so if there be atrophy, and least of all if there be encephalopathy, though even here recovery may take place. Contracted kidney due to lead-poisoning is also usually incurable. No favorable prognosis should be given when the patient is unable to remove himself from the cause. It must be remembered, too, that relapses occur, often at long intervals, even when the patient is removed from exposure, and that the primary disease has been known to make its appearance a long time after exposure. As a matter of fact Wickham Legge collected 264 cases of persons who died with symptoms of plumbism in 32 of whom the cause of death was some excephalopathy, in 43 Bright's disease, in 47 cerebral hemorrhage, in 43 paralysis, 44 lead-poisoning, 38 phthisis and 40 pneumonia, heart disease, aneurysm also occurred. There is an absence of precision in the above as where deaths are said to have been due to encephalopathy, cerebral hemorrhage and paralysis, but the data help us to a conception of the prognosis.

Treatment.—Much may be done to guard against the occurrence of lead-poisoning by *proper precautions* on the part of those exposed to it, and those employed in lead-works may do much to protect themselves, or rather their employers may do it for them. Such persons should keep themselves scrupulously *clean* by frequent *hot baths* and frequent changes of clothing, which should never be allowed to become saturated with lead. Mehu recommends that *hypochlorite of sodium* be added to the hot baths. It is made by mixing in 2 1/2 gallons (10 liters) of water 13 ounces (400 gm.) of chlorinated lime with 11 drams (43 gm.) of sodium carbonate. *Sulphur baths* were recommended by Todd, it being thought that sulphur has the power of neutralizing lead by forming insoluble compounds with it. From 2 to 4 ounces (62.5 to 124.5 gm.) of sulphuret of potassium are mixed in from 20 to 30 gallons of water (75.5 to 113.4 liters). Above all, the employees in lead-works should *not* be allowed to *eat meals* in the lead factory, as the metal is often introduced with food. Finally, the *ventilation* of the factory should be of the best. Experience has shown that much may be done to arrest the dangers of lead-works by such precautions. The same remarks as to cleanliness, bathing, and change of clothing apply to painters, and indeed to all who have to do with lead in any shape or degree. It is evident that lead-lined and painted cisterns should never be used in houses, that cosmetics and hair-dyes are dangerous, and that care should be taken in the selection of canned foods not to use those which have been too long canned.

The curative measures may be divided into those for the immediate relief of urgent symptoms and the removal of the lead from the system. It is scarcely necessary to say that the patient should be promptly removed from the influence of the lead. The extreme pain of the lead colic requires to be relieved by the hot bath or poultice, and an opiate, of which the best mode of administration is by the hypodermic syringe, 1/4 to 1/3 grain (0.016 or 0.02 gm.) of sulphate of morphin being required for the purpose. Identical treatment is required for the arthralgia. The accompanying constipation is best relieved by sulphate of magnesium, the sulphuric acid of which, on theoretical grounds, at least, aids in render-

ing inoperative the lead which entered the system by forming an insoluble sulphate.

These more urgent symptoms being relieved, measures directed to the *elimination of the lead* should be taken. The hot baths already referred to fulful this purpose as well as prophylaxis, while *purgatives* and *diuretics* may aid elimination. The *iodid of potassium* is the remedy most relied upon to eliminate lead. It is believed that after its absorption the lead becomes intimately united with the albumin of the tissues, forming an insoluble compound; that the iodid of potassium, after its absorption, combines with the lead and forms a soluble iodid of lead, which is dissolved out, re-enters the circulation, and is passed out with the urine and feces. It is evident that elimination by these channels will be encouraged by purgatives and diuretics. Iodid of potassium should be given in ten grain doses three times a day is the proper dose and this dose should be kept up until the patient is relieved. Iodid of potassium is more efficient when given fasting and freely diluted. The use of *Fel Bovis* in 3 grain doses is valuable.

For the paralyzed muscles *faradic electricity* is indicated and should be daily applied, both to resist the tendency to atrophy and to overcome it.

That *restorative* and *blood-making* remedies, in the shape of nutritious, easily assimilable food, together with iron, should also be given to antagonize the cachexia which is always a part of plumbism is evident. In view of the nervous and muscular symptoms which enter so largely into the disease *strychnin* may be expected to be a useful adjunct or our treatment, and it is generally so considered. It should be given in full doses, 1/30 grain (0.0022 gm.) three times a day, and increased to 1/20 grain (0.0033 gm.), which should be kept up. *Ergot* is said to have been useful in restoring the power of muscles involved in the palsy.

ARSENICAL POISONING.

Acute Arsenical Poisoning.—Acute arsenical poisoning is usually the result of accidental or intentional ingestion some commercial form of arsenic such as Paris green or "Rough on Rats," prepared and sold for the destruction of rats, mice, vermin, and insects. Occasionally also it is taken with suicidal intent.

Symptoms.—These are intense *abdominal pain*, at first gastric, with *vomiting;* later intestinal, twith *diarrhea* and *tenesmus*, which may be followed by collapse and death. The symptoms are not unlike those of cholera, including rice-water stools, cardiac weakness, and cyanosis. Sometimes a *skin eruption* makes its appearance, and sometimes *blood* and *albumin* appear in the *urine*. Fatal cases terminate in one or two days.

Recovery from these acute symptoms may be followed by paralysis.

Treatment.—The ingestion of a poisonous dose of arsenic is apt to be followed by free vomiting. But even in the event of emesis, the stomach washed out with draughts of warm water. The best antidote is freshly precipitated sesquioxid of iron, which forms, with arsenic, an insoluble compound. It must be freshly prepared, taking any of the sesqui solutions of iron, preferably the chlorid, and neutralizing it with sodium carbonate or magnesia. The precipitate, being hastily washed by emptying it on muslin

or a filter, pouring water on it and allowing it to drain, should be freely administered. Dialyzed iron may be used, but it is best also precipitated with ammonia or other alkali before using. In extreme cases the tincture of the chlorid of iron, Monsel's solution, or any of the sesqui preparations may be substituted for the precipitated sesquioxid.

After the emetic has acted, and while the antidote is being given, castor oil should be administered to carry off the poison from the bowels.

Chronic Arsenical Poisoning.—This is ascribed to wall-papers covering occupied apartments, to artificial flowers, to carpets and clothing fabrics colored or dyed with arsenic. The glazed green and red papers are those especially dangerous. The arsenic emanations may be in the shape of small particles or gaseous volatile bodies. Occasionally, arsenic medicinally administered may produce the symptoms of slow arsenical poisoning, Fowler's solution being the drug usually responsible. A widespread epidemic occurred in Manchester, England, due to contaminated glucose used in making beer.

Symptoms.—Chronic arsenical poisoning may be suspected in the presence of unexplained *anemia* and *debility, irritation* of the conjunctiva, mouth, pharynx, and stomach producing gastralgia; and of the lower digestive tract. From *neuritis* there may result *numbness, tingling*, and pain in the fingers succeeded by paralysis and localized wasting. All these symptoms may, however, be produced by other causes. The *paralysis* resembles that of lead palsy, but affects rather the lower extremities, especially the extensors and peroneal group, whence may arise the characteristic *steppage gait* of peripheral neuritis. These symptoms have been ascribed by some authorities, as in the case of lead-poisoning, to central lesions rather than to alterations of the nerves. *Deranged electrical reaction* may be present before any loss of power, but on differential examination a weakened power of wrist extension and feeble power to spread the fingers may be detected. Pigmentations, flushing and redness of the skin are common, even keratosis and rarely epithelioma.

Treatment.—The patient should be removed from the exposure and the symptoms be treated as they arise. The iodid of potassium may be used.

BISULPHID OF CARBON POISONING.

Symptoms.—The acute symptoms are those of exhilaration followed by depression, excitement or taciturnity, loss of appetite and headache. Overwhelming doses cause great weakness. Mania and hysteria have been reported. The chronic symptoms are those of peripheral neuritis resembling those caused by alcohol, great muscular weakness, followed by wasting, sluggish reflexes, tremor. Absence of sexual desire is a characteristic symptom. There may be foot-drop, wrist-drop and finger extension, or the fingers may become stiff and numb. There may be scotoma and limitation of the field of vision with hyperemia of retinal vessels. These symptoms may occur after short exposure or only after long periods. The urine has been affected in some cases, shown by the presence of hematuria, albumin, indican, and hydrobilirubin. Blood changes are not marked or numerous but the hemoglobin may be reduced.

Prognosis.—Death never occurs from the poisoning alone, though it may be caused by resulting cachexia. Recovery is more or less incomplete, some claiming that it is never complete. Muscular weakness is often permanent in mild degree.

Treatment.—Prophylaxis should be observed to protect the workmen. The work-room should be on the ground floor with vents next to the floor to carry off the gas which is heavier than air. Such removal can be facilitated by the air-pump. The muscular weakness is best treated by electricity and massage; general weakness by strychnin and nourishing food.

FOOD POISONING.

Ptomain and Leukomain Poisoning.

Ptomain, from Greek πτώμα, a cadaver, is a word suggested by the Italian toxicologist, F. Selini, for substances generated in the decomposition of organic matter, which more recent studies have shown to be the result of bacterial action. Ptomains are basic, uniting with acids to form salts. Leukomains are similar basic substances formed in the living body. Ptomains differ greatly in their character and properties, certain ones being intensely poisonous, others harmless. For the former L. Brieger suggested the name toxins, retaining that of ptomains for the non-poisonous basic products; but, as Victor C. Vaughan suggests, there are difficulties in the way of such classification, because a ptomain may be poisonous under certain conditions and harmless under others.

Meat Poisoning.

This succeeds the eating of various kinds of meat in which poisons have developed either through bacterial agency or chemical change.

Of the cases due to bacteria several groups may be made:

1. *Poisoning due to the colon bacillus or the typho-coli group* intermediate between the typhoid group and colon bacillus group. The symptoms of this group exhibit a wide range of symptoms, being limited in certain mild cases to headache and moderate fever, while certain severe cases begin suddenly with vomiting, purging and severe pain, terminating in collapse and death. Some cases resemble in their symptoms the paratyphoid infection, while others are discoverable only by the presence of the typhoid (Widal) reaction. Very interesting and important is the fact that in this type the appearance of the meat is unaltered. Beef and veal are most liable when the animals are the subject of some diarrheal or septic state. The last has been noted in Germany. In England pork is a frequent seat of the poison served in the favorite pork pie.

2. *Poison Due to Putrefaction.* Both smell and taste proclaim the danger from this source. The poisonous products are the result of protein hydrolysis and include putrescin, cadaverin and sepsin. There are, however, also found associated organisms, especially proteus and colon bacilli, which are held responsible by some, so that it cannot be regarded as settled whether they or the aromatic compounds named are responsible.

3. *Poisoning Due to Bacillus Botulinus.* This is a rare form of bacillus discovered by von Ermengem in ham which was held responsible for an outbreak involving 50 cases. The symptoms are like those of atropin poisoning, dryness of the throat, dilated pupils and cardiorespiratory paralysis.

Ham poisoning not due to trichina has ocurred in England, Germany, and Switzerland, while poisoning has also been traced to beef, mutton, less frequently veal, turkey, and goose, and in America to canned meats. Some of these last must be ascribed to muriate of zinc and tin, but others are doubtless due to the meats. Poultry, especially if kept too long, and game birds also prove poisonous at times.

Symptoms.—The symptoms of various epidemics of meat poisoning vary, but the following are more constant, after a period of incubation of from one to 48 hours: *nausea, vomiting, cramps,* and *diarrhea*—in a word, acute gastro-intestinal irritation. To these may be added *dryness of the mouth, constriction of the throat, difficulty in swallowing, vertigo, indistinctness of vision, dilatation of pupils,* while sometimes *constipation* substitutes diarrhea. *Thirst, headache,* and *muscular weakness* may also be present.

The symptoms may begin at once without incubation in a feeling of languor and general malaise, loss of appetite, nausea, and griping pain in the belly.

In fatal cases the symptoms of cholera are simulated, such as cramps in the legs or arms, or both, muscular twitchings, stiffness of the joints, drowsiness, coldness of surface, pinched features, blueness of fingers and toes and around the sunken eyes—in a word, the symptoms of collapse. On the other hand, the temperature sometimes rises to 101° to 103° F. (38.3° to 39.4° C.), with a pulse of from 100 to 128.

Poisoning by Milk and its Products.

The causes of poisoning by cheese claimed attention as far back as 1827 when analyses of poisonous cheese were made by Hunnefeld. The older view that the poisons are fatty acids has been refuted, and Vaughan isolated a ptomain in 1884 which he has called tyrotoxicon (τυρός, cheese, and τοξικόν, poison). Tyrotoxicon was not, however, always found by Vaughan in cheeses of acknowledged poisonous properties; nor was it the most virulent. In 1885 he found it in milk which had stood in well-stoppered bottles for about six months, and in 1886 Newton and Wallace obtained it from milk which had poisoned a number of persons in a hotel at Long Branch, N. J. Since then tyrotoxicon has been isolated many times from poisonous milk. Finally, in 1886, Vaughan obtained tyrotoxicon from ice-cream which had proved poisonous, and since then it has been frequently found in such cream. A number of cases of poisoning after eating "cream puffs" have been reported in Philadelphia and elsewhere, in which doubtless the same ptomain is responsible. A family under Tyson's observation was poisoned by *blanc mange,* of which all had eaten freely, and which had been made for several days.

Symptoms.—The symptoms of milk and cheese poisoning are those of gastro-intestinal irritation, comparable in various degrees to those described as due to meat poisoning, etc.

Poisoning by Shell-fish and Fish (*Ichthysmus*).

The mussel furnishes the most frequent source of poisoning from this cause, instances of which were reported as early as 1827 by Combe. A ptomain was isolated by L. Brieger in 1885, from poisonous mussels, at Wilhelmshaven, where numerous instances occur. Brieger has called it from *mytilis*, a mussel. It is found chiefly in the liver of the mussel, but whether in a special poisonous mussel or a mussel which becomes poisonous under certain circumstances is not settled, though the latter would seem to be true, since Schmidtmann found that non-poisonous mussels placed in the waters of Wilhelmshaven Bay became poisonous, and poisonous mussels from the latter became harmless after being placed in the open sea.

Symptoms.—Both cooked and raw mussels may produce the poisonous symptoms. Three sets are described:

First, those of gastro-intestinal irritation, similar to those described as due to meat poisoning, and which may terminate fatally within two days, the autopsy revealing inflamed stomach and intestines.

In a *second* set of symptoms the nervous system seems to bear the brunt of the poison, and these cases are said to be the most frequent. The symptoms include a sense of heat and itching, usually beginning in the eyelids, but soon extending over the whole face and sometimes over a large portion of the body. An eruption, vesicular and papular, makes its appearance and intensifies the itching. The eruption is often followed by asthmatic breathing. Sometimes the dyspnea precedes the eruption, the face becomes livid, the patient unconscious, and there are convulsive movements of the extremities. In other cases there are delirium, convulsions, coma, and death within three days. In other nervous cases there are numbness and coldness, frequent pulse but no fever, the pupils are dilated, and death takes place in a couple of hours with symptoms of collapse.

In a *third* set of cases a symptom like intoxication by alcohol is present, followed, by paralysis, coma, and death.

Treatment of Ptomain and Allied Poisoning.

This is mainly symptomatic—the *purgative* and *emetic* effect of the poison generally promptly gets rid of any residue which may be in the stomach or intestinal canal. But if there is any reason to believe that these are not emptied, purgatives should be administered, and of these calomel is probably the best because it is less apt to be rejected. The stomach should be washed with a tube.

In addition counterirritation by mustard, hypodermic injection of 1/3 grain (0.0165 gm.) *morphin*, repeated if necessary, to relieve pain, digitalis from 10 to 30 minims (0.66 to 2 gm.), and strychnin 1/30 grain (0.0022 gm.) administered in the same manner to counteract collapse may be given. *Stimulants* by the mouth should be given if retained, and to this end champagne becomes very suitable, or milk mixed with carbonated water may be given in small quantities. Hexamethylenamine, salol and sodium sulphocarbolate should be used as intestinal antiseptics to complete the detoxication.

GRAIN POISONING.

For a century or more districts have been subject to ailments which have been traced to the use of certain grains as food, some of which have been found to be spoiled or diseased. Residents in some parts of France, Germany, Switzerland, Italy, Spain and India and the United States have been thus affected.

1. ERGOTISM.—Ergotism is one of these ailments. It is a disease found to succeed upon the use of meal contaminated with the *sclerotium*, an intermediate stage of development of the *claviceps purpura*, a fungus which infests the rye grain. Ergot is a sclerotium, which appears at the base of the grain as a hard, dark-hued "spur," which, as it grows, lifts up the diseased and withered mass of the original grain. Wheat, barley, and rice may also become spurred. The growth of the fungus is favored by wet seasons. The disease prevailed in France, Switzerland, and Germany much more commonly from the 10th to the 18th century than at present. The cause of ergotism was discovered in 1830 by Thuillier.

Two forms of chronic ergotism are recognized, one convulsive or spasmodic, the other gangrenous.

Spasmodic Ergotism.—This is believed to be due to cornutin. In this form there is a prodromal period of from 10 to 15 days, during which there are a peculiar sense of weariness and anxiety, a tingling and sense of formication in the skin, especially of the fingers and toes, gastro-intestinal irritation manifested by vomiting, purging, and colicky pains, accompanied sometimes with slight fever. Then *spasmodic symptoms set in.* These consist at first in involuntary twitchings, which soon pass into painful continuous contractions, the arms being flexed and the legs and toes extended. The cramp lasts for an hour or more, followed by a period of exhaustion, which may be succeeded by another painful convulsion. There may be delirium, melancholia, or dementia. The urine may be suppressed or violent dysuria may be present from spasm of the bladder. Pustules, boils, whitlows, and other evidence of deranged nutrition may appear. Cardiac contractions are slow and feeble, the arteries are constricted and contain little blood. Death may occur from cardiac paralysis, and is often preceded by convulsions or paralytic symptoms. The duration of the illness is from four to eight weeks or longer.

Sclerosis of the posterior columns of the cord was found in some of the cases which came to necropsy. Thus, Tuczek and Siemens found it four times in nine autopsies, which represented, also, the deaths in a group of 29 cases.

Gangrenous Ergotism.—This is believed to be due to sphacelinic acid. It is ushered in by the same prodrome as that described for the spasmodic. On this succeeds, from the third day to the fourth week, an erysipelatous redness in some peripheral locality, as in the toes and fingers, ears, and nose. This is followed usually by dry gangrene, but the moist form,' which may be confined to a finger or toe or may involve the whole hand or foot, may also appear. The disease may not go beyond the erysipelatous redness.

For acute ergot poisoning see concluding section.

2. LATHYRISM, or LUPINOSIS.—This is a condition resulting from the use of meal made from the chick-pea, or grain of a variety of vetches, more particularly the *lathyrus sativus* and *lathyrus cicera*. It is used in admixture with barley and wheat in India, Italy, and Algiers. According to James Irvine, the symptoms supervene in India when the proportion exceeds 1/12.

The symptoms are, first, gastro-intestinal irritation, then a condition of spastic paralysis, which may pass on to complete paraplegia. The arms are rarely, if ever, affected.

No associated *morbid change* has been discovered.

Treatment of Grain Poisoning.

This consists, primarily, in the removal of the cause and the substitution of wholesome food; in removal, also from the district, if possible, and suitable treatment of symptoms.

BERI-BERI.

Definition.—Beri-beri is a disease of the Malay Peninsula, Philippine Islands, South America, Japan, China, and some parts of Africa, characterized in its end-results at least, by edema, wasting of the muscles, and of polyneuritis. It is a disease of great antiquity.

Morbid Anatomy.—In the acute cases, there is general edema; there is postmortem ecchymosis; the mucous membrane as the pharynx is red; there is excessive liquid in the abdominal, pleural, and peritoneal cavities. The gastro-intestinal mucous membrane may be congested or actively inflamed with hemorrhagic erosions; there may be hemorrhagic extravasations. Microscopic examination of the mucous membrane of the gastro-intestinal tract shows active, congested, small-celled infiltration necrosis of the epithelium.

There is a bacillus which Wright considers pathognomic, found between the epithelial cells. The muscle of the heart shows fatty degeneration. The kidneys are usually congested.

According to Wright, the changes of the nervous system are as follows: In the earliest stages the neurons are found to be "spotted with black altered myelin. Nissl's bodies of the process have disappeared. Later in the nerve cells the nuclei are swollen; the peripheral cells are rarified. It is not the degree of neuro-atrophy in the acute pernicious beri-beri which causes death, but it is its extent. The second stage is seen in the more subacute stage of beri-beri. The cells are rarefied. Nissl's bodies have disappeared or broken down; the termination of the dependent fibers are in an early and distinct stage of atrophy. In the third stage there is vacuolation of the cells; rupture of its membrane and that of its nucleus; sometimes exclusion, either wholly or partially of the nucleolus." The last stages are accompanied by complete degeneration of the peripheral nerve fibers.

Etiology.—The disease certainly has a direct relation to the kind of rice used as a diet. Takiki banished beri-beri from the Japan navy in 1884 by the correction of diet and the substitution of other foods for rice. Bratton, Fraser, Stanton and others have established beyond question that in-

dividuals fed upon polished rice develop beri-beri, while those fed upon parboiled rice do not develop beri-beri. Two hundred and twenty persons were fed upon polished rice; of those, twenty developed beri-beri. Of 223 persons under exactly the same conditions, except they were fed parboiled rice, none developed beri-beri.

Later the same observers made some experiments upon fowls, and found they could develop beri-beri at will by feeding them upon polished rice.

Beri-beri patients were in contact with persons fed upon parboiled rice, but did not contract the disease. No organisms were found in the blood or urine of beri-beri patients.

The Philippine statistics go to show certainly that a diet without polished rice does not cause beri-beri, while one with polished rice does cause it. On the other hand Wright believes the disease is an acute infectious disease due to a specific organism, the paralysis so characteristic of the disease being simply the result of a toxin.

Symptoms.[1]—Wright divides the disease into

1. *Acute pernicious beri-beri*, where there is more or less sudden onset with gastro-intestinal symptoms, cardiac failure, with varying degrees of sensory motor paresis and pulmonary failure. These cases end fatally.

2. *Acute Beri-beri.*—Here the onset is sudden but not so fulminating as in the pernicious cases. Here too there is weakness of the heart and signs of peripheral nerve involvement, but the cases are not so surely fatal, some of them recovering and passing over into the terminal stage.

3. *Subacute Beri-beri.*—Here the onset is more gradual; the gastro-intestinal symptoms may escape notice. Clinically the heart does not show any involvement, and there is but slight paresis of the sensory motor nerves.

4. *Beri-beri Residual Paralysis or Neuritis.*—This is the state usually recognized and long described as endemic neuritis. This includes the cases of acute and subacute beri-beri, which have recovered from the first effects of the poison leaving behind a peripheral neuritis.

Here the symptoms are those of chronic peripheral neuritis. This is weakness, lassitude, followed by languor. There is formication of the legs and thighs. The body then becomes edematous, there is dyspnea, cardiac distress. This stage is followed more or less suddenly with great muscular atrophy, the patient becomes much emaciated and entirely helpless, all these symptoms being the result of the general multiple neuritis. Some of the cases are chronic and relapsing in character.

Prognosis.—The disease is a serious one; the acute pernicious cases all die after a few days. Those of the acute cases which drift over into the other stages, have a mortality of about 20 per cent.

Prophylaxis and Treatment.—From the more recent studies, especially those of Fraser and Stanton, attention to the diet is the all important prophylactic agent. Polished rice should not be used. Barley or beans or parboiled rice may be substituted for this universal diet of the East. Chamberlain's experiences in the Philippines bears out this statement. All the well-known sanitary regulations should be carried out. In the early stages of the disease parboiled rice, the hulls from the rice, barley or beans may be used and some of the cases recover.

[1] Studies from the Institute for Medical Research, Federated Malay States, 1902—1904.

In the stage of paralysis, rest in bed, purgation for the edema, massage and electricity for the atrophy, with strychnine as a tonic, is of value. Iron also in the form of Blaud's pills for the anemia is useful.

PELLAGRA.

By Edward Jenner Wood, M. D.

Definition.—Pellagra is a disease of unknown cause, occurring usually in the temperate and subtropical sections, characterized by symmetrical skin lesions largely confined to the uncovered portions of the body, by gastro-intestinal disturbances, by changes in the nervous system of a heterogeneous nature, and by definite seasonal variation.

Historical—It has been suggested that Hippocrates was referring to pellagra when he described *solsido*. This idea was warmly opposed by Strombio who considered it a new disease never known until after the introduction of Indian corn from America. The first definite description was given by Gaspar Casal, a physician of the Asturias in 1735. It is not improbable that pellagra had existed for many years before in an unclassified state. It is not improbable further that it was confused with syphilis, leprosy, scurvy, and even such conditions as some of the purpuras. It is a matter of more than passing interest to fix the time of the first appearance of this disease in Europe as it would be of great aid in a study of the etiology.

The first known appearance of pellagra was in Spain. In 1755 Strombio recorded its appearance in Italy and from that time to now that country has been considered the natural home of the disease. It was known as alpine scurvy when it first appeared in Lombardy and about the year 1755 this section was ravaged by it. Somewhat later France became a victim and in recent years Roumania has been one of the chief scenes of its activity. In the last decade Egypt has furnished many cases which have been carefully observed by a number of English physicians. Sporadic cases have been reported from Tyrol, Servia, Bulgaria, Greece, Asia Minor, and a few from Great Britain.

It seems probable that pellagra has existed in the United States in sporadic form for many years. Babcock recently found among the case records of the South Carolina hospital for the insane an account by Dr. James Davis of a definite case occurring in 1834. It was not until 1907 that the first account of a definite outbreak was reported. For this report we are indebted to George H. Searcy of Alabama.

At this time pellagra has been reported either in endemic or sporadic form in all but nine states of the Union. It is more prevalent in the south Atlantic states. Lavinder claims that 30,000 cases have occurred with a fatality of over 39 per cent. This is a low estimate. As pellagra is a disease of rural sections and occurs chiefly in states outside the registration area no definite idea of its extent can be secured at this time.

Etiology.—Since the time of Casal, pellagra has been connected with the consumption of Indian corn. In every period, however, there have arisen zealous opponents to this theory and recently this number has materially increased. The difficulty in the way of the solution of the problem is that the disease cannot be reproduced in any of the laboratory animals.

Among the zeists or advocates of the corn theory were two schools. One taught that corn was injurious through a deficiency in food value. The other school considered it a toxic process due to verderame or verdet which had resulted from the action of a mold, sporisorium maidis. Chickens were fed on this mold and developed a number of symptoms which at the time were thought to be pellagrous. Lombroso, after many years of study, concluded that the disease was an intoxication process produced by the action of certain micro-organisms on Indian corn. In themselves these organisms were thought to be harmless but to have the property of producing a poisonous ptomaine.

Tizzoni claimed to have isolated a "strepto-bacillus pellagræ" from the blood, the feces, the cerebro-spinal fluid, and the organs at autopsy. This or-

ganism, while not a spore-bearer, was supposed to resist very high temperatures accounting for the fact that it was not killed by cooking. It has been shown that this organism is not the specific etiological factor.

Recently L. W. Sambon of London has advanced the hypothesis that pellagra is due to an animal parasite yet undiscovered which is transmitted by a biting fly of the Simulium group. The chief ground for this view is the geographical distribution. Pellagra usually occurs in the foothills or the so-called sub-alpine regions. It does not usually occur on the sea coast nor in the high mountains. The Simulium lays its eggs in rapidly running water and without this aeration the eggs die in a few hours. Wherever Sambon found pellagra he found the stream and the fly. The geographical findings in Italy conform exactly to those in North Carolina and it seems more than coincidental that the disease should so often select the very banks of the streams. The seasonal variation and the pathologic changes further suggest a disease of animal parasitic origin, but until the animal is found that can be successfully experimentally inoculated all views are speculative.

Pathology.—Some observers find an analogy between pellagra and ergotism, while others find it between pellagra and such diseases as kala-azar and syphilis. Like the latter pellagra is characterized by a perivascular infiltration of the tissues and by a mononuclear increase in the elements of the blood. It may be argued that syphilis as well as ergotism produces tract degenerations in the cord but it cannot be denied that there are many points of similarity between the nervous changes in pellagra and ergotism.

Increase of pigment and atrophy of the visceral organs have been often mentioned in connection with pellagra, but cannot be counted distinctive.

Atrophy of the muscular coat of the intestinal tract and ulceration as low down as the rectum occur. Chronic enteritis with cicatricial constrictions, thickening of Peyer's patches and cystic degeneration of the submucous glands have been found at autopsy.

In the nervous system pigmentation is especially marked and suggests a senile state. Hyperemia, anemia, and edema of the nervous system occur but are not distinctive. Obliteration of the central canal of the cord with general deformity of the various elements frequently occurs, but cannot be counted as distinctive. It is commonly acknowledged that the brunt of the affection falls on the *lateral* tracts of the cord in the form of definite tract degeneration. The crossed pyramidal tracts are usually affected but the direct cerebellar tracts always escape. The writer has had a preponderance among his cases of degeneration of the *posterior* tracts. Muscular atrophy has been noted but does not select definite groups of muscles. Lesions of the posterior columns are more frequent in the cervical and upper dorsal regions.

The skin changes in pellagra are not distinctive. They follow either the changes of erythema or dermatitis. Atrophy is always the end-result. Hyperkeratosis and parakeratosis are both mentioned and the former is counted of considerable importance.

Symptoms.—Pellagra affects all ages with almost perfect impartiality. The writer's youngest case was twenty-two months and his oldest seventy-five years.

All observers are agreed that the female sex is more often affected. In

Tennessee the proportion was 214 females and 102 males. In both Tennessee and North Carolina the negro was much less frequently affected than the white race. A part of this may be due to the comparative infrequency of uncinariasis in the negro, while in the poor white of the South it forms the chief source of general lowered resistance to all infections.

The symptoms of pellagra arrange themselves into three definite groups: the gastro-intestinal, the skin, and the nervous. The gastro-intestinal symptoms usually appear about Christmas time and are followed by the outbreak of erythema with the first warm weather of late spring or early summer. The gastro-intestinal symptoms are at first indefinite partaking of the nature of a neurosis. The mouth symptoms are present in a large majority of all cases. General redness of the whole mucous membrane of the mouth is usually seen. The epithelium of the tongue is often denuded and small ulcers and vesicles may be found. Congestion of the fungiform papillæ especially about the tip and edges is recorded by Manson. Babcock found small black or bluish spots on the dorsum which was called by Lavinder "stipple tongue." Salivation often occurs. The pain in the mouth and æsophagus is often so great as to interfere with the taking of food.

The gastric symptoms are indefinite. Nausea frequently is a distressing symptom. Diminution of hydrochloric acid, the presence of lactic acid and mucus are found in some cases and it is not remarkable that the cachexia of pellagra added to the gastric findings should have caused the erroneous diagnosis of gastric carcinoma.

One of the most constant symptoms is a diarrhea which is distressing in the extreme. The number of stools daily is often very great and the consequent weakness adds greatly to the suffering. This symptom is rarely absent and a careful history-taking will almost certainly disclose its past occurrence. It is no unusual thing to learn that for a number of consecutive seasons this symptom without other pellagrous manifestations had occurred and for this reason it is often left out by the patient in recounting the symptoms. It is a common sight to find a wasted pellagrin wearing a diaper, but incontinence will sometimes occur when there is no diarrhea. Occasionally there is an alternation with constipation. Fatal intestinal hemorrhage has occurred.

The most important symptom of pellagra is the symmetrical erythema or dermatitis occurring on those parts which are exposed to light and air. This symmetry is most definite and includes symmetry of shape, size, and location. Without this appearance or a definite history of it a diagnosis is never justified. The atypical locations of skin lesions are numerous and may occur on any portion of the skin surface, but should never be counted pellagrous without coexisting symmetrical lesions of the exposed portions. The most usual location of the skin lesion is the backs of the hands and the forearms. The next most frequent is the neck above the collar and to this is given the name Casal's collar. The feet in children and in negroes who go barefooted are commonly affected. In the beginning it is extremely difficult to distinguish the erythema of pellagra from simple sunburn. Later the lesion assumes a brown or chocolate color before exfoliation. After exfoliation a soft smooth skin is left. Just about one-fourth of an inch within the line of demarcation of the skin lesion is to be found a brown line which is

known as the hyperkeratotic border and is important because it persists long after the disappearance of the erythema and is often the means of making a diagnosis. There are two types of skin lesion commonly recognized. The dry type is a simple dry erythema which undergoes changes of color, and final exfoliation. The moist variety is characterized either by bleb formation or by weeping surfaces caused by crevices in the skin. The final result of both is crust formation. In the moist variety the condition is often loathsome in the extreme. It would suggest the appearance produced by extensive burns. The skin lesions of pellagra recur year after year always at the same season. It should be borne in mind that the skin lesions are similar to skin lesions in syphilis in that they are present only during a short period of the affection and that the disease is not eradicated because of the disappearance of these symptoms.

The nervous and mental manifestations of pellagra are almost as varied as in syphilis and occur with almost as much certainty as in this disease when it goes untreated. Tract degenerations are usual. As a rule pellagra must exist for at least three years before the impression on the nervous system becomes sufficiently indelible to produce these tract degenerations. It is usually stated that the lateral tracts are more frequently affected. In the writer's experience the columns of Goll and Burdach have suffered oftener. In the event of degeneration of the posterior tracts the root zones and the posterior roots of the spinal nerves are not affected. There is more hope of restitution when there is marked exaggeration of the reflexes and a spastic tendency. In an absence of reflexes resulting from posterior column affections the outlook is less favorable for a return to normal. Amyotrophic lateral sclerosis of pellagrous origin has been reported. Symptoms corresponding with the description of Landry's paralysis have occurred in a case of the writer's and death resulted from glosso-labio-laryngeal paralysis. Neuritis especially involving the nerves of the hands and feet is frequent and is a most difficult condition to relieve.

The time of appearance of mental symptoms of pellagra is most variable. It has been reported that even in the first outbreak of the disease insanity occurred before the erythema or digestive disturbances. This insanity is usually of the depressive type and melancholia is frequently diagnosed. Delusions of persecution are frequent, but delusions of grandeur unknown. Suicidal tendencies are often manifested and death from suicide is a considerable factor in increasing the death rate.

Diagnosis.—In typical cases diagnosis offers no difficulties. With the triad of symptoms—stomatitis and diarrhea, symmetrical erythema of the exposed portions of the skin, and nervous and mental changes—there can be no error. The diagnosis is never justified in the absence of skin symptoms or a definite history of such. The diagnosis may be made in the absence of nervous or mental changes, having in mind the fact that the disease may exist for many years without the development of either. Either one or the other group of symptoms of the mouth or intestinal tract may be wanting but practically never both. The only malady with which the disease may be reasonably confused is sprue in the South. This disease is identical with pellagra without skin symptoms, *i. e.*, the so-called "pellagra sine pellagra."

Prognosis.—The prognosis is more encouraging than in 1907. The

fulminating type is seldom seen at this time and it was this class of cases which produced the high death rate in the first years of its observation in this country. However, Lavinder places the death rate at above 39 per cent., but this is certainly too high for private practice. The prognosis is materially affected by the time of diagnosis for neglected cases just as in syphilis are discouraging. The means of the patient are important, for in no disease is change of climate and environment of greater importance. A death rate of 10 per cent. at this time would be conservative estimation.

Treatment.—In the absence of a known cause treatment must of necessity be empirical. In spite of this fact much progress has been recently made along this line. The only group of drugs worthy of mention are the arylarsonates. Atoxyl is probably the best, but owing to its toxicity and the fact that it is not dispensed in tablet form it has been superseded in the hands of the writer by soamin, which is less toxic and can be had in 1-grain or 5-grain tablets. In either instance the drug is given in dose of from 5 to 7 1/2 grains (0.3 to 0.5 grams) by hypodermic injection deep into the gluteal muscles. The dose is repeated about every fourth day. This plan of treatment is especially effective when begun one month before the time of the expected outbreak in the spring. This anticipatory plan is of the greatest importance. Rest in bed during the acute symptoms is as important as rest in tuberculosis during the stage of fever. The best diet is milk and it should be persisted in until all diarrhea ceases. To the milk may be added a raw egg. It is useless to give drugs to control the diarrhea. The mouth is best treated with simple cleanliness and such deodorant preparations as Dobell's solution.

Removal from unhygienic surroundings and correction of such resistance-lowering conditions as uncinariasis cannot be too strongly emphasized.

SECTION XII.

EFFECTS OF EXPOSURE TO HIGH THOUGH BEARABLE TEMPERATURE.

Such effects are easily separable into two groups, covered by the terms heat exhaustion and thermic fever.

HEAT EXHAUSTION.

Definition.—A condition of syncopal exhaustion with vasomotor paralysis and lowering of body-temperature, caused by exertion under high temperature. Such condition may arise quite independently of the direct rays of the sun. The heat may be that of confined rooms and may be artificial heat.

Symptoms.—The sense of *great weakness*, often experienced in hot weather after some unusual exertion, exhibits the mildest degree of this condition. In the more severe forms a sense of faintness, associated with *pallor*, *dizziness*, at times *blindness*, and the starting of cold *perspiration* are the first symptoms. Sometimes the victim can get to a place where he may sit or lie down; at other times he faints away before assistance can reach him. Then follows a condition of *unconsciousness* or semi-consciousness, whence, under favorable circumstances, he may respond to simple stimulus by ammonia or wine and then fall into a sleep, from which he will awake in an hour revived.

In more severe cases the *collapse* is more permanent, the *pulse* is extremely feeble and frequent, the *skin* continues leaky, while there may be great *restlessness* and *muttering delirium*. It is characteristic of this form of heat affection that there is extreme *adynamia* with *lowered body-temperature*. H. C. Wood, whose name is inseparably associated with the subjects of heat exhaustion and thermic fever, reports a case with a temperature as low as 95° F. (35° C.), with complete collapse.

Diagnosis.—Heat exhaustion is characterized by *lowered temperature* and feeble pulse, as contrasted with the opposite in *thermic fever*. It is important that the two conditions should not be confounded, because of the widely different treatment required. The syncopal attack from *cardiac failure* or from *concealed hemorrhage* much more closely resembles heat exhaustion, being associated also with feeble pulse and lowered temperature, but as the treatment is identical, the distinction is less important. The fall in temperature is, however, less decided in *syncope*.

Treatment.—The patient should be put to bed at once with his head horizontal or slightly raised. When possible, stimulants should be administered moderately by the mouth—brandy, whisky, or ammonia with digitalis. If this is not possible, digitalis and strychnin should be given hypodermically from 10 to 30 minims (0.66 to 2 gm.) of tincture of the former and 1/30 grain (0.0022 gm.) of the latter. Friction should be applied, and dry heat by hot-water bags or cans.

THERMIC FEVER.

SYNONYMS.—*Heat Fever; Sunstroke; Coup de soleil.*

Definition.—A state of high fever induced by exposure to heat, natural or artificial.

Etiology and Pathology.—In this country the majority of cases occur in the summer season in those exposed to the direct rays of the sun, though they occur also among those exposed to high temperature within doors, as in sugar refineries, fire-rooms of ocean steamers, laundries, and the like. A heated atmosphere charged with moisture, impeding, therefore, evaporation, produces fever much more rapidly than a dry heat, which is in fact slow to produce it. The habitual use of alcohol is found to be a potent predisposing cause—at least alcoholics succumb very much sooner to the influence of overheat than temperate persons.

The pathology of the two conditions of heat exhaustion and thermic fever is thus explained by H. C. Wood: "There is in the pons or higher portion of the nervous system a center whose function it is to inhibit the production of animal heat, and in the medulla oblongata a center (probably the vasomotor center) which regulates the dissipation of bodily heat. Fever is due to a disturbance of these centers, so that more heat is produced than normal and proportionately less thrown off. Let it be supposed that a man is placed in such an atmosphere, that he is unable to get rid of the heat which he is forming. The temperature of the body will slowly rise, and he may suffer from a general thermic fever. If early or late in this condition the inhibitory heat center becomes exhausted by the effort which it is making to control the formation of heat, or becomes paralyzed by the direct action of the excessive temperature already reached, then suddenly all tissues will begin to form heat with the utmost rapidity, the bodily temperature rises with a bound, and the man drops over with one of the forms of coup de soleil.

"Heat exhaustion," on the other hand, "with lowered temperature, represents a vasomotor palsy—*i. e.*, a condition in which the existence of the heat paralyzes the center in the medulla oblongata, and the heat is dissipated more rapidly than it is produced." It must be admitted that the explanation of heat exhaustion is less satisfactory than that of thermic fever.

Morbid Anatomy.—The high temperature characteristic of heat fever remains a long time after death. Hence putrefaction sets in early. Rigor mortis also occurs promptly. The blood remains liquid. There is general venous engorgement, especially of the lungs and cerebrum. In early autopsies the left ventricle is found contracted, the right dilated.

Symptoms.—A sense of uncomfortable *burning heat* and feeling of *oppression* may precede the "stroke" which fells its victim, who quickly becomes *unconscious* and comatose, perishing sometimes instantly, at other times in a few hours. In other cases there are intense *headache*, *dizziness*, *oppression*, *nausea*, and *vomiting*, occasionally *diarrhea*. *Chromatopia*, or colored vision, may be present. Sooner or later unconsciousness sets in, and may be associated with *muttering delirium* and intense *restlessness*. In this condition the patient is commonly admitted to hospital with face flushed, eye suffused, skin hot and dry, temperature from 107° to 112°

F. (41.6° to 44.4° C.), the *breathing* labored, and sometimes *stertorous*, the *pulse* frequent and full. The *pupils* at this stage are usually contracted, though at first dilated. The urine is scanty, sometimes albuminous. Usually there is relaxation of the muscles, but at times there is a *convulsive tendency*, shown by twitching and jactitation, and occasionally by epileptiform convulsions. The *skin*, usually dry, may become moist and bathed with perspiration, which does not, however, reduce the temperature. Wood speaks of a peculiar *odor* exhaled by the entire body as characteristic.

Attention has been called by C. F. Close[1] to cardiac dilatation as a symptom of thermic fever.

In fatal cases the stupor deepens, the pulse becomes more frequent and loses even its seeming strength, then becomes irregular; the breathing is labored and irregular, and toward the last, shallow, or assumes the Cheyne-Stokes type previous to death. Death does not usually take place for several hours. In favorable cases improvement is indicated by a falling temperature and a return to consciousness.

Iron and steel workers, ships' stokers, and a variety of other persons, whose occupation exposes them to very intense heat, frequently develop attacks of muscular spasm, which have been studied by David L. Edsall. The spasms may be mild or violent and intensely painful and in rare cases even fatal. The spasms especially affect the flexors of the forearms, legs, hands and feet, and at times have a superficial resemblance to tetany; but any or all the muscles of the trunk and extremities may be affected and Chvostek's and Trousseau's phenomena are absent. Signs of involvement of the cerebrum, spinal cord and nerve-trunks are usually absent also, and the disorder seems to be resident chiefly in the muscles themselves. The temperature may be somewhat elevated, normal or, especially in the severe cases, subnormal. There is likely to be marked and at times dangerous general collapse in the very bad cases. In the cases studied by Edsall there were very remarkable disturbances of metabolism, showing severe tissue destruction, probably chiefly in the muscles. Elliott has reported practically negative postmortem findings.

Recovery may be complete, but more rarely a permanent condition results in which there may be more or less constant *mental weakness*, as evidenced by incapacity for sustained mental effort, while exposure to moderate degrees of temperature produces great excitement or headache or pain in the upper cervical region. *Epileptic* convulsions sometimes occur. In these cases there is probably a certain degree of meningitis.

Mention has already been made, when treating of fevers, of the form of continued fever occurring in the south of the United States, where it is known as "Florida fever" and "country fever," and in India and the West Indies as *fièvere inflammatoire*, for which John Guitéras proposes the name *continued thermic fever*, but which more recently he is inclined to ascribe to a septic origin.

Diagnosis.—The diagnosis of heat fever presents no difficulties. The distinction between it and *heat exhaustion* has been alluded to.

Prognosis.—The prognosis depends partly upon the severity of the case and the promptness and thoroughness of treatment. A few cases

[1] "Journal of the Am. Med. Assoc.," March 1, 1901.

are almost instantly fatal. If the cooling treatment can be applied properly, a decided majority—fully 60 per cent.—recover. A temperature of 110° F. (43.3° C.), though indicating gravity, should not discourage.

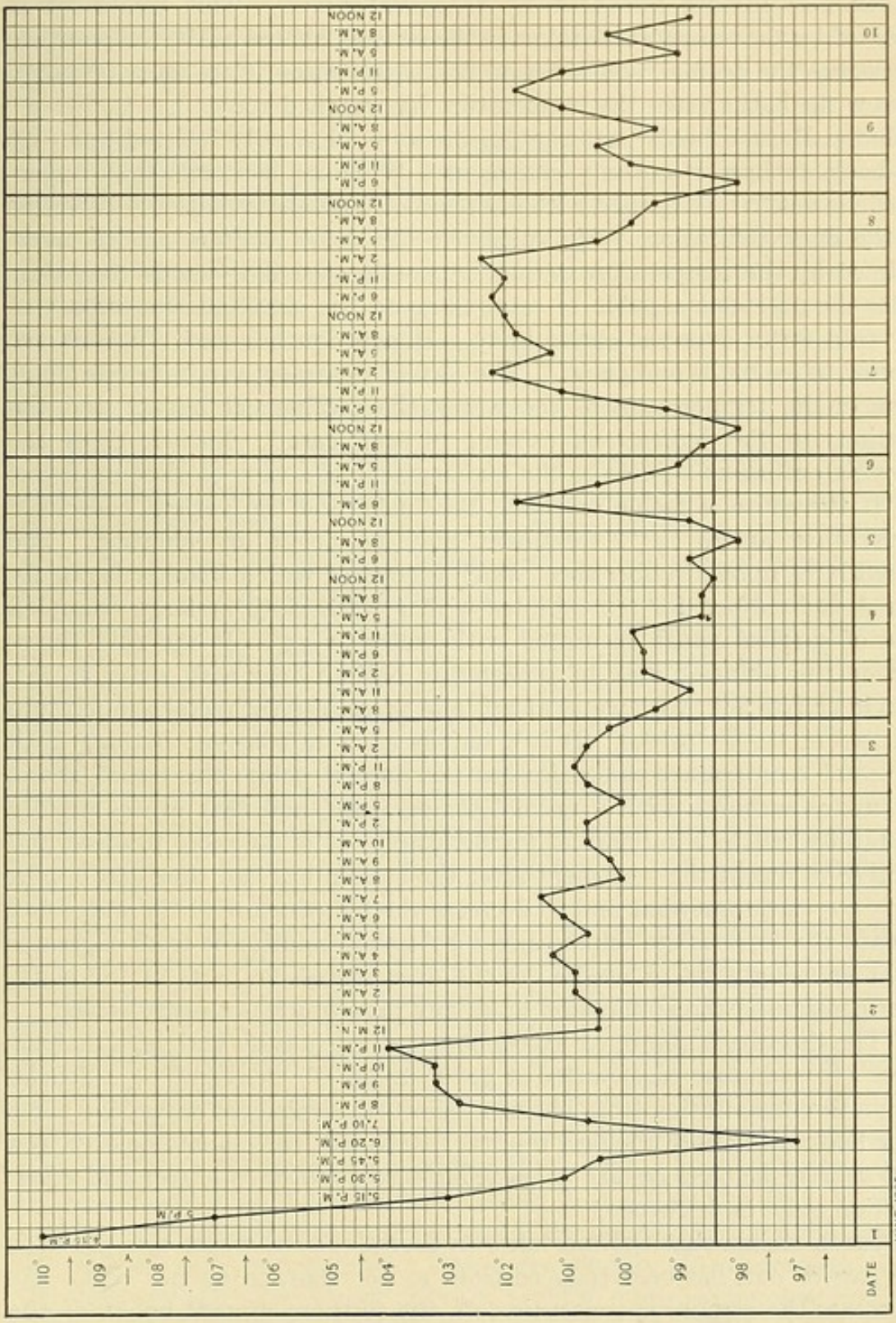


Fig. 179.—Temperature chart from a case of suntroke, treated by ice water baths and frictions. Recovery.

Treatment.—By many the success of treatment of thermic fever is thought to depend altogether upon our ability to lower the temperature of the victim. To this end it is directed the patient be placed in a *bath of water* to which ice is freely added to keep the temperature down as low as

possible, which in summer is not likely to be below 60° F. (15.54° C.). It is also directed that the surface of the body be vigorously *rubbed with ice.* In the absence of bathing facilities the patient was placed on a mattress covered with a mackintosh and rubbed with pieces of ice. The refrigerating effect is further increased by *ice-water enemas.* This treatment is regulated by the temperature taken in the rectum, abated as the temperature approaches the normal, and renewed as it again rose. Care must be taken to cease the baths when the temperature has reached 100°.

Simon Baruch holds that the dogma so emphatically insisted upon by text-book authors is entirely erroneous. While he contends that hyperpyrexia is not to be regarded with indifference, nerve-stimulating procedures are the chief indication. "In sunstroke the nerve centers are so overwhelmed by the temperature acting on predisposed individuals that they render the patient incapable of responding to the simultaneous impact of very cold water over the entire body. Hence douches and affusions are superior to ice baths and packs. The correct treatment is a judicious adaptation of the temperature of the water, the mechanical impact and the duration, to the indications existing in each case." Baruch gives the following outline as a guide to the management of a case of thermic fever:

"If the pulse is feeble or rapid, skin pale, and the patient conscious, temperature above 103°, treatment may be begun by ablution. The patient lying upon a rubber sheet which is covered with a blanket is rapidly bathed and rubbed for ten minutes with a wash cloth or large crumpled piece of surgical gauze saturated with water at 75°. If the skin shows reaction this ablution may be repeated in half an hour with water at 70°, again in half an hour with water at 65°, and so on until the rectal temperature falls two or three degrees. It is a serious error to continue bathing until the rectal temperature approximates the normal point; the record shows that many succumb with a normal temperature, and that others rapidly and fatally fall to a subnormal temperature. It is far more prudent and effective to repeat the treatment after the system has had time and opportunity to react somewhat and to decrease the temperature of the water and increase the force of its impingement by slowly pouring it from a greater height or using a jet or needle douche over successive parts of the body, never continuing the application upon any part which is already cooled.

"The sheet bath affords an excellent procedure in cases with high temperature. In comatose cases water at 50° F. to 40° F., delivered by a douche or by a forcible affusion from basins at short intervals over successive parts of the body, will afford the best results. Here, too, care should be exercised *not to depress the nerve centers by long-continued application of very cold water.* Reaction must be induced."

Baruch claims that the mortality in thermic fever, where this plan has been carried out, has been reduced from 40 per cent. to 60 per cent. He deprecates the erroneous conception of the authors of text-books of the present day that reduction of temperature is the chief therapeutic aim and that the coldest bath is the most efficient antithermic bath.

The experience of one of the authors in three of the hospitals of Philadelphia during an extremely hot summer would go to show that criticisms

of Baruch are not justified. The number of heat cases was large. The treatment was by ice baths or rubbing with ice. The mortality was almost zero.

After this, or in addition to this, *treatment* should be *symptomatic*. For convulsions, chloral or chloroform; for heart failure, digitalis and strychnin hypodermically; for asphyxia, bleeding. Mild cases or recurrent fever may be treated with the antipyretics represented by antifebrin, phenacetin, and antipyrin.

In the cases associated with muscular spasm, treatment consists of the use of laxatives and sedatives, though in the bad cases the effects of even morphin are unsatisfactory. Normal salt solution by enema or subcutaneously, and freely used, may prove valuable, since chlorids are absent from the urine; and some cases seem to have been benefited by this.

CAISSON DISEASE.

(The Bends, Diver's Disease.)

Definition.—Caisson disease is a condition occurring in divers and caisson workers who are compelled to spend some time under high degrees of atmospheric pressure. It is the result of the saturation of the tissues with nitrogen, followed by too rapid decompression. Bubbles of nitrogen are liberated in the blood, causing emboli and the symptoms of the disease.

History.—The first important observations upon the disease were made in 1820 by Howth of Dublin. Dr. Jaminet made important observations upon the condition during the building of the St. Louis bridge over the Mississippi. Dr. A. H. Smith of New York in 1886 made important observations during the building of the Brooklyn Bridge. In 1912 Leonard Hill published a monograph upon Caisson Sickness, which brings up to date all of the experiments which have thus far been made, and places upon a sure foundation the actual cause of the condition. From this monograph we have made many quotations.

Etiology.—The cases occur exclusively in caisson workers and in divers. It is the direct result of high pressure followed by too rapid decompression. According to P. Bert, who proves his contention by many animal experiments, all the effects consequent upon high pressure are due to liberation of bubbles of nitrogen in the blood. It is neither CO^2 or O^2, but nitrogen dissolved, which once set free upon decompression may in one case block the pulmonary circulation, in another cause embolism of the spinal cord and so produce softening and paralysis there, and in another produce swelling and emphysema in the tissues. It has been discovered both by animal experimentation and with workers, that if the decompression is done slowly, the disease does not appear. The more rapidly decompression occurs, the more certainly the disease appears, and the more serious the symptoms. It has been discovered by actual experiments made by Hill and his co-workers that fat increases the susceptibility to caisson disease. This is due to the fact that fat individuals take longer to saturate and desaturate the nitrogen than more spare individuals. Both Smith and Jaminet noticed this fact clinically. "Compression up to 4 1/4 atmospheres is not in itself

to be dreaded. It is endured without any trouble and infinitely better, to rarification in a far less proportion or degree. The return to the normal pressure is alone to be dreaded. The risk of this is proportionate to the period and degree of compression, and the rapidity of decompression. It is necessary to make the last much slower." (Pol & Watelle.) Coolness and fatigue are predisposing causes of the attacks.

Morbid Anatomy.—In postmortem, lesions are found in the central nervous system, which consist of bubbles which are more or less abundant in the white matter than in the gray matter, the bubbles which occur in the gray matter are nearer the edges. The bubbles are more frequent in the dorsal and upper lumbar cord than in the lumbar enlargement. This is probably due to better circulation in the lumbar and cervical region than in the dorsal region. Following the gas embolism in the vessels, necrosis of the cord constantly follows, which is the cause of the paralytic symptoms. Massive embolism both in the lungs and liver occurs. Thecases of sudden death are probably due to air embolism in the lungs.

Symptoms.—The symptoms of the disease are varied. Pains in the various portions of the body; myalgia and arthralgia occurred in 18 per cent. and 32 per cent. of the cases observed and quoted by Hill. Paraplegia occurred in 8.1 per cent., monoplegia 5.3 per cent. Paralysis of the bladder occurs usually accompanying paraplegia. Paraplegia may last a few minutes, hours, or even days. It may disappear or may persist, and is a spastic condition. Often marked itching and formication of the skin occurs, probably due to formation of bubbles underneath the skin.

In 110 of Smith's cases there were three deaths.

Prophylaxis and Treatment.—Apparently the disease can be practically prevented by careful decompression. There are two methods of doing this, the interrupted adopted by Haldane and one supported by Hill, called the continuous method. According to Hill the latter method is preferable and much less trying to the workers. This is practically done by allowing the workers to pass through air locks, of which the pressure in the East River Tunnel was as follows: the inner chamber was forty pounds pressure; the intermediate 29 pounds; the outer was 12 1/2 pounds. The men were ordered to take five minutes in the first lock, eight minutes in the second, ten minutes in the third lock, there being an approximate distance of 1,000 feet between each pair of locks, so that in all forty-eight minutes were taken to decompress from 40 pounds to atmosphere. No serious or fatal cases resulted, and but little time was lost by the men through caisson disease. Hill believes that if a single lock had been used at 12 1/2 pounds, and very active exercise been carried out, fifteen minutes would have sufficed for decompression.

For the painful condition—friction, massage, hot baths may be tried. Oxygen may be given in severe cases, but the most effecacious method is recompression. As soon as the symptoms appear, the individual may be sent either back into the locks, or into a special recompression apparatus. According to Keays, recompression relieved 90 per cent. of 3,967 cases of pain. When treatment is delayed more than six hours, medical means are as valuable as recompression. Hot fomentations, massage, hypodermics of morphine may be used to relieve pain.

Mountain Sickness.

Definition.—This is a condition due to low atmospheric pressure encountered at high altitudes.

Symptoms.—The symptoms are vertigo, palpitation of the heart, dyspnea, and faintness. The symptoms are due, according to Holdane, Douglass and Higgison, to want of oxygen, produced by the diminished pressure of the atmosphere. There is also an increase in the hemoglobin. The red cells in persons residing in high altitude are increased. In persons with weak hearts there may be syncope and intermittent heart. These symptoms disappear when the patient remains at these heights after a long time, provided there is no organic disease of the organs. The treatment is obvious. If the symptoms are severe, the patient should go to a lower altitude.

SECTION XIII.

SUMMARY OF SYMPTOMS FOLLOWING OVERDOSES OF POISONS.

(Alphabetically Arranged.)

ACONITE (*Monkshood; Wolfsbane; Blue Rocket*).—All parts poisonous. The tincture may be mistaken for sherry or whisky; it has an exceedingly acrid taste.

Symptoms.—These appear quickly, and consist of an acrid taste in the mouth, a feeling of warmth in the stomach, followed by a tingling sensation throughout the body; muscular weakness, pulse weak and slow at first, later rapid and running; respirations slow and shallow; vomiting may be present, but is rare; the skin is cold and clammy and exceedingly pale; death may be gradual, from respiratory failure, or it may be sudden as a result of any movement of the body which throws any strain upon the heart. The mind is clear to the last.

Treatment.—Stomach-tube or emetics if seen early; recumbent posture, with feet elevated, not allowing the patient to arise for any cause. Stimulants freely, such as ammonia, ether, digitalis, atropin, and strychnin. External heat about the body, and artificial respiration for two hours.

ALCOHOL.—Taken in the form of spirituous beverages. Acute alcoholic poisoning. A brief period of excitement, with flushing of the face, followed by unconsciousness, stertorous breathing, rapid and, finally, weak pulse, vomiting, a subnormal temperature, delirium, complete muscular relaxation, at times convulsions; the pupils are usually dilated. Recovery commonly takes place in a day or two, but remissions may occur. Odor of alcohol on the breath.

Treatment.—Evacuation of stomach by pump; emetics, like apomorphin, 1/10 grain (0.0064 gm.); washing out the stomach. If coma and total relaxation are present, use external heat and stimulation by ammonia, atropin, caffein, digitalis, strychnin, or faradic current to muscles of respiration applied by passing an electric brush rapidly over the surface.

DELIRIUM TREMENS.—Delirium, with hallucinations; great restlessness and insomnia; slight fever, pulse rapid and soft.

Treatment.—Withdrawal of alcohol; hyoscin hydrobromid 1/100 grain (0.00064 gm.) hypodermically every two hours until patient is fully under its influence; bromids in full doses, or chloral, provided that the heart is not weak; aided by a cold bath to induce sleep; nourishing food and stimulation if the condition demands it, even by alcohol.

AMMONIA—Taken by mistake or with suicidal intent in the form of "household ammonia," water of ammonia, spirit of hartshorn, and in liniments.

Symptoms.—At once, burning pain in the mouth, throat, esophagus, and stomach; the lips and tongue are intensely swollen and inflamed; vomiting of blood-tinged mucus, suffocative cough, with rapidly increasing dyspnea.

The face is pale, pulse is rapid and thready, and collapse soon develops. Death may follow at once as the result of spasm or edema of the glottis, or some days later, from the violent gastro-enteritis and stricture of the esophagus.

Diagnosis.—Odor of ammonia on the breath, vapors of the corresponding salt when a rod dipped in hydrochloric acid is held before the mouth, together with the sudden onset of the symptoms.

Treatment.—Neutralization with vinegar, orange-juice, lemon-juice, or some other dilute acid, as soon as possible. If the patient lives, administer milk and bland oils to soothe the inflamed mucous membrane. Give morphin to relieve pain, stimulants to overcome depression, and apply external heat to maintain bodily temperature. Tracheotomy should be performed if there is danger of death from edema of the glottis.

ANTIMONY.—Taken as a tartar emetic, the tartrate of antimony and potassium. A heavy, white, odorless, slowly soluble powder having a sweetish, metallic taste; charring to redness upon heating.

Symptoms.—Metallic taste in the mouth, muscular relaxation, skin moist and relaxed, severe nausea and gastric distress followed by violent vomiting, and purging of, first, the normal contents of the bowel and later of serous material containing small shreds of mucous membrane. Cramps in the abdomen and in the muscles of the arms and legs occur as the result of the abstraction of water from the tissues by the violent purging. At the same time, symptoms of collapse appear—cold, clammy skin, feeble, shallow respirations, weak, thready pulse, subnormal temperature, coma and death.

Treatment.—Tannic acid as the chemical antidote, followed by washing out the stomach; recumbent posture with the head low, not allowing the patient to raise the head; external heat, stimulants, opium for the pain and demulcent drinks, such as bland oils, mucilages and albumin water, to soothe the inflamed mucous membrane.

ARSENIC.—Used in the form of arsenic trioxide in rat-poisons, in fly-paper, and to preserve stuffed birds and animals. Paris green, used as potato-bug poison, is an arsenite of copper, hence the symptoms are similar. Arsenous acid, or white arsenic, is an odorless, tasteless, white powder, quite heavy, and but slowly soluble in water.

Symptoms.—These appear usually in the course of an hour, and are those of violent gastro-enteritis, so severe as to suggest Asiatic cholera. Burning pain in the throat and stomach, persistent vomiting of brown matter streaked with blood, though the vomited matter may be green from bile. Purging is very severe; the stools, after the passage of the normal contents of the bowel, are serous as in antimonial poisoning, but contain larger flakes of mucous membrane and blood. The urine is scanty, concentrated, and contains albumin. Great prostration accompanies these symptoms and the patient dies in collapse in one or two days. If the patient survives so long, there is usually an intermission in the symptoms about the third day which in a few hours is followed by a return of the symptoms. Nervous symptoms may appear. The poisoning resembles cholera morbus and Asiatic cholera.

Treatment.—An emetic if seen early, wash out the stomach, external

heat and stimulants. The best antidote is the official ferri hydroxidum cum magnesii oxido, or ferri hydroxidum. These should be freshly prepared. In the absence of these official preparations, any liquid preparation of iron may be used by diluting with water, then adding a dilute solution of ammonia, strain out the precipitate, wash it free of ammonia while on the strainer, then dilute with fresh water and administer freely.

Atropin (*Belladonna*).—The deadly nightshade. Used as a mydriatic and in liniments. The leaves impart a narcotic odor to the tincture, but recognition depends upon the physiological effect.

Symptoms.—Face flushed, skin hot and dry, throat dry, pupils widely dilated, pulse rapid and bounding, respirations quickened and deepened; if the dose has been large there may be active, talkative delirium. An erythematous or scarlatiniform rash is sometimes present. The urine contains the alkaloid, hence it will cause dilatation of the pupil if dropped into the eye of an animal.

Treatment.—Tannic acid as a chemical antidote, followed by the stomach-tube or emetics. Morphin, physostigmin or pilocarpin as physiological antagonists. External heat and stimulants if the patient goes into collapse.

Belladonna.—See Atropin.

Bromin.—A dark red, very heavy liquid, emitting reddish vapors resembling chlorin.

The fumes, when inhaled, cause convulsive cough, bloody expectoration, dyspnea, and spasm of the glottis.

Treatment.—Fresh, moist air and cautious inhalations of ammonia.

Bromism.—The symptoms of chronic gastro-intestinal disturbance, such as fetor of the breath, anorexia, diarrhea; great depression of all the functions, especially the sexual function, with languor and mental apathy. A general eruption of acne is an early sign.

Treatment.—Stop the administration and aid elimination.

Carbonic Acid Gas.—The choke damp or after-damp of miners. May be accidently inhaled in overcrowded rooms, in fermenting vats, over lime-kilns, or wherever the products of complete combustion cannot escape.

Symptoms.—Headache, dizziness, noises in the ears, a sense of tightness across the chest, great drowsiness, loss of muscular power, followed by symptoms of asphyxia; coma, tumultuous heart action, stertorous breathing, cyanosis, possibly convulsions and sometimes delirium.

Treatment.—Fresh air, if need be artificial respiration, kept up steadily and unceasingly; ammonia by inhalation; oxygen, if obtainable; cold douche to the head and chest, with external heat and stimulation as occasion requires. Use of pulmotor.

Carbonic Oxid (*Carbon Monoxid*) is formed during the incomplete combustion of carbon, and is a direct poison, while carbonic acid gas, the product of complete combustion, kills merely by exclusion of oxygen.

Symptoms.—The same as in carbonic acid poisoning.

Treatment as for carbonic acid gas.

Caustic Potash or Soda.—Taken in the form of "lye."

Symptoms.—An acrid, burning taste, the burning extending down to the stomach, followed by vomiting, purging, and collapse. The mucous membrane of the mouth shows evidence of corrosion. Convulsions may occur. Stricture of the esophagus may follow recovery from the acute symptoms.

Treatment.—Dilute vegetable acids, such as vinegar, lemon-juice, or orange-juice, to neutralize. Demulcent drinks, such as bland oils, mucilages, milk, or white of egg. Opium or morphin for the pain. External heat and stimulants. The stomach-tube should not be used. If the patient lives, the resulting stricture of the esophagus may require dilatation.

Cheese Poisoning.—Decayed cheese owes its poisonous properties probably to tyrotoxicon.

Symptoms.—Violent vomiting and purging; pain in abdomen; tongue first coated white, later red and dry; pulse weak and irregular; face is first pale, later cyanotic. The poisoning, while severe, rarely causes death.

Treatment.—The stomach-tube may be used if vomiting has not been free; subsequent lavage; opium and demulcent drinks to relieve pain and irritation; stimulants.

Chloral.—A popular somnifacient and sedative. Occurs in deliquescent crystals with characteristic odor and acrid, burning taste.

Symptoms.—Shortly after swallowing a poisonous dose of chloral, the patient becomes drowsy and gradually passes into a state of coma, from which he cannot be aroused. Respirations are slow and labored at first, later shallow and feeble; the pulse is feeble and shallow; the face may be white, livid, or cyanotic; the skin is relaxed, cold, and clammy; the pupils, at first contracted, later become widely dilated; there is complete muscular relaxation and abolition of reflexes; the temperature is lowered more than by any other toxic agent.

Treatment.—An emetic, if seen early, or use the stomach-pump. The maintenance of bodily temperature is of the utmost importance. Rouse the patient, and employ artificial respiration with the faradic current and by other methods usually employed. The head should be kept lower than the feet, and the patient should not be allowed to raise the head or body for any reason. Stimulants should be given in full doses.

Chloroform.—Identified by its peculiar ethereal odor and sweet, pungent taste; a heavy, volatile, non-inflammable liquid, not miscible with water.

Symptoms.—First stage of narcosis, excitement, struggling, a flushed face, contracted pupils, lessened sensibility to pain. Second stage, muscular relaxation, loss of sensibility, unconsciousness, reflexes abolished, temperature subnormal, respiration slow, pupils dilated, pulse slow. Third stage, that of paralysis, in which the pulse is irregular and weak, respiration fails, skin becomes cyanotic. Death, which results primarily from vasomotor paralysis with secondary respiratory paralysis and cardiac failure, may occur in any stage.

If the drug has been swallowed, the patient experiences an intense

burning pain in the mouth, throat, and stomach, vomiting and purging, followed by the symptoms of narcosis enumerated above.

Treatment.—In case of accident during anesthesia, the patient should be held head downward; employ artificial respiration; rhythmical traction of the tongue by grasping it with forceps and pulling it out of the mouth and upward about 14 times a minute; bandages to the extremities, and compression of the abdomen by means of a compress and bandage to confine the blood to vital centers; external heat; strychnin and other stimulants hypodermically; adrenalin chlorid intravenously or by hypodermoclysis. The faradic current may be used by sweeping the electrode over the chest to promote respiration. When swallowed, the stomach-tube or emetics should be used prior to the treatment above.

Cocain.—Solution used as a local anesthetic, particularly in eye surgery.

Symptoms.—Vertigo, headache, paroxysmal dyspnea, rapid weak pulse, elevated temperature, mental excitement, blindness, delirium, coma, and convulsions. Some of these may be caused by the local application of solutions to mucous membranes. The pupil is dilated, but the power of accommodation remains in part.

Treatment.—Nitrite of amyl, stimulants, atropin, caffein, and ammonia. Wash out the stomach or administer an emetic if the drug has been swallowed. Death is unusual.

Conium (*Poison Hemlock; Common or Spotted Hemlock*).—Not a native of this country, hence cases of poisoning must be restricted to the use of the preparations. The plant and some of the preparations have a peculiar odor, resembling the urine of mice.

Symptoms.—Loss of muscular power, first felt in the legs, going on to complete paralysis which creeps up toward the trunk, the arms being less rapidly affected. The muscles of respiration become involved eventually, the patient becomes cyanotic, and death results from asphyxia. The pupils are dilated, the skin is relaxed and clammy and ptosis is a prominent symptom. Sometimes delirium, coma and convulsions are prominent from the first.

Treatment.—Stomach-pump or emetics; tannic acid; stimulants, particularly strychnin; external heat; artificial respiration.

Copper.—The sulphate, blue stone or blue vitriol, is used in the arts. Recognized by its crystalline shape, blue color, and acrid, metallic taste. Articles of food are frequently prepared in imperfectly cleansed copper kettles; a bright piece of steel, such as a knife, will show a deposit of metallic copper a few minutes after immersion in a liquid containing copper. Verdigris (subacetate) is another source.

Symptoms.—An astringent, metallic taste in the mouth; pain in the stomach and abdomen; violent vomiting of a greenish or bluish liquid; profuse purging of the normal contents of the bowel, later mucus and blood, accompanied by tenesmus; jaundice, respiratory failure, collapse, coma, and death.

Treatment.—Stomach-pump and emetics if vomiting has not already emptied the stomach; yellow prussiate of potassium (potassium ferrocya-

nid) as the chemical antidote; demulcents, such as bland oils, white of egg, and milk; opium for the pain; external heat and stimulants.

DIGITALIS (*Foxglove*).—A native of Europe. The tincture has a distinct odor of tea, the drug itself lacking a narcotic odor.

Symptoms.—Nausea, vomiting, pain, with or without diarrhea; headache, often severe, occasionally delirium and convulsions; the skin is cold and clammy; the pupils are dilated; the pulse at first slow and full, later becomes dicrotic or shuttle-like and difficult to count, while the heart-beat is irregular and tumultuous; the patient may show an inclination to somnolence, which may deepen into coma; death may not result for several days or it may occur suddenly following a muscular exertion.

Treatment.—Emetics or the stomach-pump may be used early; tannic acid as the chemical antidote; aconite may be used early as the physiological antagonist; external heat; horizontal position for several days after active symptoms have subsided; stimulants, such as ammonia, ether, atropin and strychnin, may be used in the later stages to combat collapse.

ERGOT.—Used to produce abortion.

Symptoms of Acute Poisoning.—Giddiness; pain in the stomach; thirst; nausea; vomiting; cardiac oppression; numbness and tingling of fingers and toes, extending along the limbs; cramp; dyspnea; coldness of the body, especially the limbs; great anxiety; delirium; coma and convulsions are among the symptoms which may be produced.

Treatment.—Emetics or stomach-tube. Stimulants and external heat as necessary. Nitroglycerin may be administered by the mouth.

Chronic ergotism occurs as a result of eating bread made from grain contaminated with the fungus. *Early symptoms* are gastric pain, general depression, nausea, occasionally vomiting; sometimes diarrhea, at others constipation, dizziness, insomnia, and lack of energy. *Subsequent symptoms* may take one or both of two courses: (1) Gangrenous ergotism characterized by patches of anesthesia, with a sensation of coldness of the parts affected, or by a burning sensation with redness of the skin. This usually occurs in the fingers and toes, and is followed by dry gangrene which may advance as far as the elbows or knees, but rarely affects the trunk. (2) Spasmodic ergotism characterized by paresthesias of various kinds; sometimes there is complete anesthesia. Motor disturbances follow: twitchings, spastic muscular contractions attended with great pain, paralyses, and disturbance of the special senses.

FISH-POISONING.—Tainted fish probably contains ptomains, while several kinds of fish are constantly poisonous.

Symptoms.—Vomiting, profuse purging, thirst, great pain in the abdomen and in the head, with subsequent collapse and death in some instances.

Treatment.—Emetic or stomach-pump if vomiting has not occurred, stimulants, external heat, and artificial respiration if required.

HYDROCHLORIC ACID.—See Mineral Acids.

HYDROCYANIC ACID (*Prussic Acid*).—The pure acid is an exceedingly poisonous gas. In medicine it is employed as a 2 per cent. aqueous solution.

Contained in oil of bitter almonds distilled from the seed; not found in the artificial or the purified natural product. Its salt, the cyanid of potassium, is employed as a quickly acting poison for the destruction of animals. Contained also in cherry-laurel water.

Symptoms.—The patient may drop dead almost immediately after a large dose of the poison has been swallowed. When smaller doses have been taken there is first difficult respiration, slow pulse, dizziness, impeded locomotion, a sense of constriction and heaviness in the head, progressive intellectual confusion, followed by tetanic convulsions, involuntary evacuations of urine and feces, with ejaculations of semen, dilated pupils, in turn succeeded by asphyxia, collapse, general paralysis, and death.

The odor of hydrocyanic acid about the body, the wide-staring eyes, the clinched teeth covered with froth, and the cyanotic face are diagnostic of death by this poison.

Treatment.—Stomach-pump or emetics, stimulants, artificial respiration, rapidly interrupted current applied by means of an electric brush swept quickly over the chest, alternate hot and cold douche. If the patient survives for 20 minutes or half an hour, the chances are in favor of his recovery.

Iodin.—May be taken by mistake; the tincture has the odor of iodin, which somewhat resembles chlorin.

Symptoms.—Metallic taste in the mouth, salivation, great pain in esophagus, stomach, and abdomen, followed by violent vomiting and purging. Face pale, pulse rapid, running, and feeble; urine suppressed; death occurs from respiratory paralysis. The presence of iodin is detected by the blue color of the vomited material if starch has been present.

Treatment.—Boiled starch (arrowroot or flour), as a chemical antidote; emetics and stomach-pump; opium or morphin for the pain; external heat, stimulants, and demulcent drinks.

Iodoform.—Rarely taken internally, but toxic symptoms may appear when used freely as an antiseptic dressing.

Symptoms.—In mild cases, malaise, nausea, vomiting, headache, anorexia, followed by cerebral excitement, insomnia, fever, erythematous rashes on the skin; small, rapid pulse; hematuria, or retention of urine, and in severe cases maniacal delirium, coma, and at times death.

Treatment.—Soullier advocates sodium bicarbonate to unite with the iodin and so aid in its elimination. External heat, stimulants. Emetics may be used if the drug has been swallowed.

Lead.—Taken most frequently as sugar of lead (acetate), or in the form of Goulard's solution (subacetate), lead water (subacetate), white lead (carbonate).

Symptoms of Acute Poisoning.—After the acetate has been swallowed, the patient complains of a sweet, metallic taste in the mouth, a feeling of constriction in the esophagus, followed by vomiting of white, opaque masses; great thirst; cramps in the calves of the legs, occasionally paralysis; constipation or diarrhea, the feces being black, due to the formation of lead sulphid; great prostration, coma, and death.

Treatment.—Emetics and the stomach-pump. Magnesium or sodium sulphate in large quantities as chemical antidotes. Opium or morphin for the pain. External heat. Stimulants. Demulcent drinks.

Chronic Poisoning.—Obstinate constipation, with colicky pains centering around the umbilicus; abdominal walls retracted and hard; a blue line on the gums, due to a deposit of lead sulphid; the tongue is coated; the appetite diminished; pulse hard and tense; bilateral wristdrop, due to peripheral neuritis, with paralysis of the extensor muscles of the forearm; arthralgia, especially of the knee-joints, less frequently involving the elbows and shoulders; cerebral disturbances—headache, vertigo, insomnia, delirium, epileptiform convulsions.

Treatment.—Remove the cause; promote elimination by the administration of potassium iodid in full doses, and by the use of hot baths; opium or morphin for the pain and to relax the spasm of the intestine caused by the lead; strychnin in full doses with electricity, massage, fresh air, and good food to overcome paralysis and increase general nutrition.

Meat Poisoning.—*Vide* Ptomain Poisoning.

Mercury (*Corrosive Sublimate; Bichlorid of Mercury; Mercuric Chlorid, or the Perchlorid*).—Used in aqueous solution as bed-bug poison, as an insecticide, to preserve specimens, and as an antiseptic surgical dressing.

Symptoms of Acute Poisoning.—Acrid, metallic taste in the mouth, followed by burning pain in the esophagus, stomach and abdomen; the mucous membrane of mouth and pharynx is white and swollen; severe vomiting of the stomach contents mixed with shreds of mucous membrane and blood; violent purging accompanied by severe tenesmus, the stools being watery and blood-streaked; urine scanty or suppressed; profound prostration, collapse, and death.

Treatment.—White of egg as the chemical antidote; wash out the stomach or give emetics if vomiting is not profuse; external heat; opium for the pain; demulcent drinks; stimulants.

Chronic Poisoning.—**Symptoms.**—Increased salivation; swelling and tenderness of the gums when the jaws are snapped together; fetid breath; nausea, vomiting and diarrhea may be present at intervals; loss of flesh and strength; anemia; skin eruptions; nervous disorders—tremors, psychical disturbances, choreic movements, hallucinations, mania, and paralyses as a result of peripheral neuritis.

Treatment.—Remove the cause; aid elimination; administer stimulants, good food and tonics; combat other symptoms as necessity arises.

Mineral Acids.

Hydrochloric Acid (*Muriatic Acid; Spirit of Salt*).

Symptoms.—Similar to those mentioned under sulphuric acid, though the acid is not so powerful and leaves no distinctive stain. It may be recognized by its odor and by the white fumes formed when the gaseous acid comes into contact with ammonia. Medicinally and in the arts hydrochloric acid is used in aqueous solution, the commercial variety tinted yellow from a trace of iron.

Treatment.—Same as for sulphuric acid.

Nitric Acid (*Aqua fortis*).—A colorless, moderately heavy liquid of peculiar and characteristic odor, staining organic tissues yellow.

Symptoms.—The same as those mentioned under sulphuric acid, though the characteristic yellow stain may be found on the lips.

Treatment.—As for sulphuric acid.

Sulphuric Acid (*Vitriol; Oil of Vitriol*).—A very heavy, colorless, odorless liquid, having a very acid taste and mixing with water with the production of great heat. Used largely in the arts. Turns organic matter black.

Symptoms.—Intense burning pain in mouth, stomach, and abdomen; mucous membrane of mouth is swollen, corroded or excoriated, sometimes white; lips swollen and excoriated; vomiting of a blackish fluid, containing shreds of mucous membrane and coagulated mucus; intense thirst with great dysphagia; bowels usually constipated, occasionally diarrhea, the stools containing altered blood and shreds of mucous membrane; urine scanty or suppressed; great feebleness and collapse.

Death may occur in the stage of collapse. It often occurs suddenly either from edema of the glottis, from pulmonary embolism or thrombosis, from perforation of the stomach, or later by the secondary effects resulting from esophageal stricture and destruction of the gastric mucous membrane.

Treatment.—Neutralize by giving oxide of magnesia, chalk, lime-water, soap and water, and water in large quantity; demulcent drinks; opium or morphin for the pain; external heat; stimulants. The stomach-pump should not be used in sulphuric-acid poisoning.

Morphin Poisoning.—See Opium Poisoning.

Mushroom Poisoning.—Harmless varieties may prove poisonous to some individuals. *Agaricus muscarius* is the most poisonous variety, containing the active principle muscarin. The fungus is bright red, with yellow spots. As a rule, highly colored fungi, with an astringent, styptic taste and a pungent odor should be avoided; they frequent especially dark and shady places.

Symptoms.—These may be divided into gastro-intestinal and nervous.

Gastro-intestinal symptoms appear six to ten hours after the fungi have been swallowed. Pain in the stomach and abdomen, nausea, vomiting, and diarrhea. Great thirst; small, weak pulse; skin cold, clammy and livid; great prostration which may result in death.

Nervous symptoms consist of muscular twitchings; convulsions; delirium; disorders of special senses, especially of vision; stupor or profound coma.

Treatment.—Emetic; castor oil to evacuate the bowels; atropin as the physiological antidote; external heat; stimulants.

Nicotin.—The liquid, volatile alkaloid of tobacco. An acrid, oily liquid of amber color, smelling of tobacco. A very deadly and quickly acting poison.

Symptoms.—Burning in throat; dizziness; muscular weakness; nausea; vomiting; skin cold, clammy; pulse rapid and feeble; pupils at first con-

tracted, later dilated; mental confusion, delirium, and convulsions. Death may occur within a short time after the poison has been taken.

Treatment.—Emetics or the stomach-tube, followed by tannic acid as an antidote; stimulants; external heat; artificial respiration, if necessary; strychnin.

Nitrobenzol (*Nitrobenzene; Oil of Mirbane; Artificial Oil of Bitter Almonds*).—Used in the preparation of anilin dyes and in the arts on account of its flavor (soaps, etc.). Recognized by its highly characteristic odor.

Symptoms.—Characteristic intense cyanosis extending over the whole body; headache; dizziness; muscular weakness; breathing irregular and stertorous; vomiting may occur, the vomited matter having the odor of the poison, as does the breath; mental confusion, stupor and coma; pulse weak; temperature lowered; skin relaxed; the pupils may be dilated or contracted, usually dilated.

Treatment.—Evacuate and wash out the stomach thoroughly; external heat with friction; artificial respiration and faradism, if necessary; stimulants as required; alcoholic stimulants should not be given until after evacuation of the stomach because alcohol renders the poison more soluble.

Opium Poisoning (Acute).—Taken accidentally or with suicidal intent in the shape of morphin or laudanum.

Symptoms.—The first stage, which is of short duration, is marked by restlessness; increased mental activity with a feeling of exhilaration; cardiac stimulation and flushed face. This is succeeded by a second stage of stupor, ushered in by drowsiness, followed by deep sleep; slow, full pulse; respirations slow and full; contracted pupils; warm, dry skin. During this stage, the patient can be aroused, but goes to sleep again when he is not disturbed. The patient passes insensibly into the third stage which is characterized by inability to arouse the patient; cyanosis; respirations very slow and shallow; muscular relaxation; the skin becomes relaxed, cold and clammy; the pupils dilate just before death occurs.

Treatment.—Wash out the stomach thoroughly and frequently; solution of potassium permanganate or tannic acid as chemical antidotes; strong, hot coffee by enema; strychnin hypodermically with atropin; keep the patient awake by mild flagellation, shaking, shouting, or by the use of the electric brush swept quickly over his chest, using the faradic current; artificial respiration, external heat and stimulants in the later stage.

Oxalic Acid.—Mistaken for Epsom salt; taken with suicidal intent. Occurs in prismatic, colorless, odorless crystals, with a very sour taste. Volatile without charring at a red heat.

Symptoms.—After large amounts have been swallowed there is pain in the mouth, stomach, and abdomen; persistent vomiting of altered blood; violent purging of a bloody material; cramps in the legs; the skin is cold, clammy and cyanotic; collapse follows; more rarely convulsions occur.

Treatment.—Calcium carbonate in the form of chalk, lime-water, whiting, marble dust, plaster from the walls, or egg-shells powdered and

suspended in a little water. Calcined magnesia may be given. Follow these by an enema or castor oil to sweep out the bowel.

PHENOL (*Carbolic Acid*) AND CREOSOTE.—Phenol is a white crystalline solid when pure, very deliquescent, and when liquefied in a colorless or reddish colored liquid, having a characteristic odor. Impure phenol is a dark colored liquid.

Symptoms.—When pure phenol is swallowed death may follow almost at once from sudden respiratory failure. If the patient does not die at once, he suffers intense burning pain in the mouth, esophagus, and stomach; the mucous membrane is stained and swollen, the eschar being white and surrounded by a zone of inflammation. The face is ghostly; the skin cold and clammy; violent vomiting and purging may be present, although vomiting may be absent. The urine is diminished or suppressed, that which is passed being dark colored; collapse follows with a small, imperceptible pulse, low temperature, and extreme dyspnea. The odor of phenol on the breath and the white eschar on the mouth are characteristic of this poison.

Treatment.—Any soluble, non-poisonous sulphate, such as magnesium, or sodium sulphate, as the chemical antidote. Wash out stomach, use emetics or the stomach-pump. Morphin for pain, demulcent drinks, such as albumin, mucilage of acacia, but *no oils*. External heat and stimulants to overcome collapse.

PHOSPHORUS.—Employed in the form of a paste as a rat and roach poison: matches are sometimes sucked for suicidal purposes. The paste is recognized by its peculiar garlicky odor, and by the luminous fumes it emits in the dark.

Symptoms.—These usually do not appear until eight or ten hours after the poison has been swallowed, and are manifested by a peculiar taste in the mouth; the odor of phosphorous on the breath; burning pain in the alimentary tract; vomiting, the vomit being luminous and consisting of food, mucus, bile, and perhaps blood; purging, the stools being also phosphorescent in the dark; constipation may be present. At the end of 24 or 48 hours a cessation of the primary symptoms occurs which may last two or three days or longer. At the end of this time, jaundice develops and the primary symptoms return with renewed vigor; the vomit is like "coffee-grounds," due to altered blood; obstinate constipation may exist; the fecal masses, if passed, are white and clay colored; the urine is scanty, albuminous and contains casts, sarcolactic acid and bile pigments; the nervous symptoms consist of headache, vertigo, delirium, convulsions, and unconsciousness, followed by death. If the patient survives the acute symptoms, he may die later as the result of fatty degeneration of vital organs.

Treatment.—Wash the stomach freely with a 1 per cent. solution of potassium permanganate as a chemical antidote; hydrogen peroxid may also be given; demulcent drinks. *No oils* should be given, as they increase the solubility of the phosphorus. Stimulants. External heat.

POTASSIUM NITRATE (*Nitre; Saltpetre*).

Symptoms.—After a large dose, the patient experiences violent pain; in the stomach and abdomen; vomiting and purging, the ejected matter sometimes containing blood; occasionally convulsions, labored respiration, cramps in the legs, paresthesia, paralysis of the limbs and aphonia have been observed; collapse occurs, followed by coma and death.

Treatment.—Emetics, and wash out the stomach; opium for the pain: demulcent drinks; external heat, with counterirritation over the stomach; stimulants.

PTOMAIN POISONING.—Ptomains are alkaloidal bodies, the products of the decay of animal tissues. These are probably the cause of the posionous action of tainted meat and fish, cream-puffs, ice cream, *blanc mange*, and cheese.

Poisonous Fish.—Vomiting, profuse purging, thirst, great pain in the abdomen, headache, itching of the skin, collapse, and death in some instances.

Shell Fish, Mussels.—Erythematous or urticarial eruptions on the skin with itching; abdominal pain, thirst, vomiting, diarrhea; dyspnea; pupils dilated; collapse; paralysis and death.

Poisonous meat contains ptomains, those produced when stale meat is just beginning to decay being more virulent than others which replace the first when decomposition is well under way.

Symptoms.—Headache, anorexia, rigors, thirst, vomiting, purging, pains in the back or limbs, skin eruptions, delirium, collapse. The temperature is usually subnormal.

Ices, Ice-cream.—The cheap varieties are colored with anilin dyes, which may not be free from arsenic. Again, the highly poisonous alkaloid, tyrotoxicon, may have formed in the milk used, and to this principle most cases of poisoning of this kind are traced.

Symptoms.—Nausea, vomiting, cramps in the abdomen, collapse; diarrhea may be present.

Treatment.—Emetic or stomach-pump if vomiting has not occurred, stimulants, external heat, and artificial respiration if required.

SILVER NITRATE (*Lunar Caustic*).—Used in the form of the fused nitrate as a caustic. Otherwise in colorless, rhombic crystals, freely, soluble, with astringent metallic taste. Yields a white precipitate with chlorids and stains organic matter black.

Symptoms.—Violent pain in the stomach and abdomen, soon followed by vomiting and purging; the vomited matter is white at first, turning dark later; the lips are at first white, but soon turn brown, then black; the skin is cold and clammy; cramps in the legs may occur; cardiac depression and collapse. Nervous symptoms, consisting of epileptiform convulsions, and delirium are sometimes severe.

Treatment.—Common salt and water, followed by an emetic or stomach-pump; opium for the pain; oils and demulcent drinks; external heat; stimulants.

STRYCHNIN.—An alkaloid occurring in nux vomica and ignatia. Appears in the form of white, prismatic, odorless crystals which have an intensely bitter taste. Used as a vermin killer.

Symptoms.—These may come on suddenly or gradually. If suddenly, the patient may be thrown into tetanic convulsions without warning. If they develop gradually, the patient experiences stiffness at the back of the neck, and muscular twitchings, with a feeling of anxiety and impending suffocation, followed by violent tetanic convulsions in which the body assumes the position of opisthotonos, occasionally emprosthotonos or pleurosthotonos, the eyeballs are prominent, the pupils dilated, and the corners of the mouth drawn into risus sardonicus. Respiration is much impeded or entirely arrested, producing marked cyanosis. The patient is conscious and experiences the most acute physical pain with mental anguish at the prospect of death which he believes to be imminent.

Intervals of relaxation occur in which the patient lies exhausted, the respirations are more rapid and full, and the cyanosis clears up. Any external impulse is sufficient to cause another convulsion, and these may succeed each other so rapidly that the patient dies of cramp asphyxia or from exhaustion.

In tetanus there is usually the history of a wound; symptoms develop much more gradually; the muscles of the jaw are early involved and trismus is much more marked than spasm of the respiratory muscles, and the convulsions are tonic.

Treatment.—Emetics or wash out the stomach, if seen early. Amyl nitrite to relax the spasm, followed by chloroform. During anesthesia, the stomach may be washed out and a solution of tannic acid given to act as a chemical antidote, while hydrated chloral with bromids in full doses may be given by enema to act as physiological antagonists. Artificial respiration if necessary.

Sulphureted Hydrogen has the characteristic odor of rotten eggs. It forms the bulk of the gas emanating from sewers and cess-pools, some ammonium sulphid and nitrogen occurring with it.

Symptoms.—If inhaled in small quantity, irritation of the eyes, nose, and throat occurs, with headache, vertigo, muscular relaxation, nausea, and diarrhea.

If inhaled in large quantities, unconsciousness rapidly follows, with cyanosis, dilated pupils, collapse, and fatal coma, sometimes interrupted by violent convulsions.

In a state approaching purity, sewer gas kills almost instantly; in moderate amount it frequently causes death in 24 hours, all efforts to restore consciousness proving useless.

Treatment.—Fresh air; artificial respiration for many hours; ammonia by inhalation; stimulation.

Zinc.—The chlorid, in solution, is used as a disinfectant (Burnett's Disinfecting Fluid). A very heavy, corrosive, colorless liquid, of astrigent taste. It partakes of the nature of a corrosive poison.

Symptoms.—Burning sensation in throat and stomach, perhaps signs of local corrosion. Nausea, vomiting, purging, dyspnea, collapse, coma, death.

Treatment.—Carbonate of potassium or sodium in water; milk; eggs; tannic acid; opium as a sedative; demulcent drinks; external heat, and stimulants.

APPENDIX.

TABLES FOR REDUCING THE METRIC SYSTEM INTO THE ENGLISH.

(Troy Weight.)

Grains to Grams.

$\frac{1}{200}$	=	0.00033
$\frac{1}{195}$	=	0.00034
$\frac{1}{190}$	=	0.00035
$\frac{1}{185}$	=	0.000357
$\frac{1}{180}$	=	0.00036
$\frac{1}{175}$	=	0.000377
$\frac{1}{170}$	=	0.000388
$\frac{1}{165}$	=	0.0004
$\frac{1}{160}$	=	0.000413
$\frac{1}{155}$	=	0.000425
$\frac{1}{150}$	=	0.00044
$\frac{1}{145}$	=	0.000455
$\frac{1}{140}$	=	0.00048
$\frac{1}{136}$	=	0.00049
$\frac{1}{130}$	=	0.0005
$\frac{1}{125}$	=	0.000528
$\frac{1}{120}$	=	0.00055
$\frac{1}{115}$	=	0.000574
$\frac{1}{110}$	=	0.0006

Grains to Grams.

$\frac{1}{105}$	=	0.000628
$\frac{1}{100}$	=	0.00066
$\frac{1}{94}$	=	0.00694
$\frac{1}{90}$	=	0.0073
$\frac{1}{85}$	=	0.0077
$\frac{1}{80}$	=	0.0082
$\frac{1}{75}$	=	0.0085
$\frac{1}{70}$	=	0.0094
$\frac{1}{65}$	=	0.001
$\frac{1}{60}$	=	0.0011
$\frac{1}{55}$	=	0.0012
$\frac{1}{50}$	=	0.00132
$\frac{1}{45}$	=	0.00146
$\frac{1}{40}$	=	0.00165
$\frac{1}{35}$	=	0.00188
$\frac{1}{30}$	=	0.0022
$\frac{1}{25}$	=	0.00264
$\frac{1}{20}$	=	0.0033
$\frac{1}{15}$	=	0.0044

Grains to Grams.

$\frac{1}{12}$	=	0.0055
$\frac{1}{10}$	=	0.0066
$\frac{1}{8}$	=	0.0082
$\frac{1}{7}$	=	0.0094
$\frac{1}{6}$	=	0.011
$\frac{1}{5}$	=	0.0132
$\frac{1}{4}$	=	0.0165
$\frac{1}{3}$	=	0.022
$\frac{1}{2}$	=	0.033
1	=	0.066
2	=	0.132
3	=	0.198
4	=	0.264
5	=	0.33
6	=	0.396
7	=	0.462
8	=	0.528
9	=	0.594
10	=	0.66

Grams to Grains.

1	=	15.43
2	=	30.86
3	=	46.29
4	=	61.72
5	=	77.15
6	=	92.58
7	=	108.01
8	=	123.44
9	=	138.87
10	=	154.3
1 pound avoirdupois	=	453.5925 gm.
1 ounce avoirdupois	=	28.3495 gm.
1 grain avoirdupois	=	0.0648 gm.

Grains to Milligrams.

1	=	64.8
2	=	120.6
3	=	194.4
4	=	259.2
5	=	324
6	=	388.8
7	=	453.6
8	=	518.4
9	=	583.2
10	=	648
1 dram or 60	=	3.89 gm.
1 ounce or 480	=	31.1 gm.[1]

FLUID MEASURES.

1 teaspoonful distilled water	= 1 fluidram	=	3.7 c.c.
1 dessertspoonful distilled water	= 2 fluidrams	=	7.4 c.c.
1 tablespoonful distilled water	= 4 fluidrams	=	14.8 c.c.
1 wineglassful distilled water	= 2 fluidounces	=	59.14 c.c.
1 fluidounce distilled water		=	29.57 c.c. (*circa* 30 c.c.[1])
16 fluidounces distilled water	= 1 pint	=	473.11 c.c. (*circa* 480 c.c.)

[1] A fluidounce of water which measures 30 c.c. does not weigh 31.1 gm., because an ounce of water really weighs but 455.7 grains Troy, and not 480 grains.

TABLES FOR REDUCING THE METRIC SYSTEM INTO THE ENGLISH FLUID MEASURES—(*Continued*).

Minims to Cubic Centimeters.	Cubic Centimeters to Minims.	Fluidrams to Cubic Centimeters.	Cubic Centimeters To Fluidrams.
1 = 0.06	1 = 16.2	1 = 3.7	1 = 0.27
2 = 0.12	2 = 32.4	2 = 7.4	2 = 0.54
3 = 0.18	3 = 48.6	3 = 11.1	3 = 0.81
4 = 0.24	4 = 64.8	4 = 14.8	4 = 1.08
5 = 0.31	5 = 81	5 = 18.5	5 = 1.35
10 = 0.62	6 = 97.2	6 = 22.2	6 = 1.62
15 = 0.92	7 = 113.4	7 = 25.9	7 = 1.89
16¼ = 1	8 = 129.6	8 = 29.6	8 = 2.16
20 = 1.23	9 = 145.8	9 = 33.3	9 = 2.43
30 = 1.85		10 = 37	10 = 2.7
40 = 2.46			

Fluidounces to Cubic Centimeters.	Liters to Fluidounces.	Liters to Pints	Pints to Liters.
1 = 29.57	1 = 33.8	1 = 2.1	1 = 0.473
2 = 59.14	2 = 67.6	2 = 4.2	2 = 0.946
3 = 88.71	3 = 101.4	3 = 6.3	3 = 1.419
4 = 118.28	4 = 135.2	4 = 8.4	4 = 1.892
5 = 147.75	5 = 169	5 = 10.5	5 = 2.365
6 = 177.42	6 = 202.8	6 = 12.6	6 = 2.838
7 = 206.99	7 = 236.6	7 = 14.7	7 = 3.311
8 = 236.56	8 = 270.4	8 = 16.8	8 = 3.784
9 = 266.13	9 = 304.2	9 = 18.9	9 = 4.257
10 = 295.7	10 = 338	10 = 21	10 = 4.73
12 = 354.84			
16 = 473.12			

LINEAR MEASURES.

Centimeters to Inches.	Inches to Centimeters.	Inches to Millimeters.	Millimeters to Inches.
1 = 0.3937	1 = 2.54	1 = 25.4	1 = 0.03937
2 = 0.7974	2 = 5.08	2 = 50.8	2 = 0.07874
3 = 1.1817	3 = 7.62	3 = 76.2	3 = 0.11811
4 = 1.5784	4 = 10.16	4 = 101.6	4 = 0.15784
5 = 1.9685	5 = 12.7	5 = 127	5 = 0.19685
6 = 2.3622	6 = 15.2	6 = 152.4	6 = 0.23622
7 = 2.7559	7 = 17.78	7 = 177.8	7 = 0.27559
8 = 3.1496	8 = 20.32	8 = 193.2	8 = 0.31496
9 = 3.5433	9 = 22.86	9 = 288.6	9 = 0.35433
10 = 3.9370	10 = 25.4	10 = 254	10 = 0.3937

Feet to Meters.	Meters to Feet.
1 = 0.3048	1 = 3.28
2 = 0.6096	2 = 6.56
3 = 0.9144	3 = 9.84
4 = 1.2192	4 = 13.12
5 = 1.524	5 = 16.4
6 = 1.8288	6 = 19.68
7 = 2.1336	7 = 22.96
8 = 2.4348	8 = 26.24
9 = 2.7432	9 = 29.52
10 = 3.048	10 = 32.8

A micromillimeter = 0.001 millimeter. Symbol μ.

TO CONVERT DEGREES OF FAHRENHEIT'S THERMOMETER TO CENTIGRADE, AND VICE VERSA.

Centigrade to Fahrenheit.

1	=	1.8
2	=	3.6
3	=	5.4
4	=	7.2
5	=	9
6	=	10.8
7	=	12.6
8	=	14.4
9	=	16.2
10	=	18

To use this table, convert the given number of degrees Centigrade into degrees Fahrenheit, and add 32°.

Fahrenheit to Centigrade.

1	=	0.555
2	=	1.11
3	=	1.665
4	=	2.22
5	=	2.775
6	=	3.33
7	=	3.885
8	=	4.44
9	=	4.95
10	=	5.55

To use this, table subtract 32° from the given number of degrees Fahrenheit and convert the remainder into degrees Centigrade.

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