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A

MEDICAL HANDBOOK:

FOR THE USE OF

Practitioners and Students.

BY

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With Numerous Illustrations.

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PREFACE.

THE plan of this Work has been suggested by the Author's own needs, both as a Student and as a Medical Practitioner. Many Students must feel — during their attendance at Lectures, and while reading the larger Treatises upon Medicine—the necessity for condensation and classification of their work; and there are few Practitioners who do not like to have in brief compass the means of occasionally, and quickly, revising the whole field of Practical Medicine.

In complying, therefore, with the request of the Publishers to prepare a short *Medical Handbook*, as a companion to Caird and Cathcart's well-known *Surgical Handbook*, the Author has kept the wants of the Student and busy Practitioner in view, and has endeavoured to make the work as condensed and as practical as possible, the chief object having been to select such matter, and to arrange it in such a way, as was deemed best in relation to the *practical diagnosis* of disease.

The Classification of the diseases is entirely *clinical*, and it is hoped that the discussion of them in groups, with the consideration of the comparative diagnosis appended (when this appeared desirable) may prove helpful. The reasons for the method used are given on page 114, in connection with the diseases of the pulmonary organs.

In the preparation of the articles upon each disease, a certain obvious order has been followed; and Standard Works and Monographs have been freely consulted, when the Author has felt it necessary to confirm or amplify his

own experience. He begs to acknowledge his indebtedness to such works—so far as the mere enumeration of facts is concerned—and trusts that he has sufficiently acknowledged elsewhere any statement which appeared to him to be original in character.

In the *treatment* of disease a difficulty has been experienced in the selection of the newer remedies. As a rule, the Author has avoided recommending them on account of the existing uncertainty as to their real value. Many, in course of time, may prove to be trustworthy; but, meanwhile, awaiting this test, it has been thought wiser only to quote the well-established drugs. The Prescriptions appended in the last chapter are in general use. When they appear special in character, and the authority has been known, the name is given—in many cases, however, this has not been possible. A few are original.

The diseases of the Integumentary system have not been included, although a classification of the Skin Diseases has been inserted (p. 305). The Author believes that a good Skin-atlas is to be preferred, when a simple revisal of the subject is required.

83 GREAT KING STREET,
EDINBURGH, *April*, 1893.

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A MEDICAL HANDBOOK.

CHAPTER I.

INTRODUCTION.

Contents.—General remarks—The constitutions and cachexiæ—Height and weight—Hints on the diagnosis of children's diseases—**Method of case-taking.**

In introducing the subject of Practical Medicine, a few remarks, by way of caution and advice, may be of service to young Practitioners. The student fresh from the medical schools is apt to forget the great object of his profession in the interest which his cases produce. In the clinical wards the absorbing pursuit of the *diagnosis*, and the satisfaction excited by the clever differentiation of obscure cases, are likely to engender a feeling that disease exists only for the pleasure of finding it out. In practice, however, our clients are *patients* and not *cases*, and the most successful practitioners are those who not only bring skill and knowledge to the case, but who are also able to look at it from the patient's point of view, and to remember that the great end of all our work is the relief of human suffering. Without sympathy there is little likelihood of success as a physician, and no class is more ready to detect its absence than those who seek relief at our hands.

It is important, therefore, in the first place, to *give attention to the patient's own statement of his complaint*. When the circumstances do not allow of his making a statement, the same attention must be given to the history of the illness, as related by the friends. The complaint may not be very well expressed; and very frequently the symptoms are ascribed to false or imaginary causes. A little experience, however, soon enables one to grasp the important points. When *pain* is present, its severity can only be estimated by the patient's own words, along with the expression of the face and aspect—due regard being given to the character and *diathesis*. To disregard the *preliminary information* may lead to awkward mistakes, and especially is this true in relation to cases of chronic disease. There is nothing to prevent a man suffering from chronic valvular

disease of the heart, or phthisis, &c., from being afflicted with a totally new and independent affection; but whether a new disease be present or not, if the patient is not allowed to give expression to his symptoms, he will not feel satisfied that his case has received careful attention at our hands.

The temperament and physical appearance of the patient are generally the first subjects to engage the attention of the physician in the examination of a case. It is while hearing the statement of the complaint that the physician's mind instinctively endeavours to know the person he is called upon to treat; but, while there is often much to be learned from simple physiognomical study, it should be noted that all such information is merely conjectural. Except in Mental cases, it is less the disposition that we seek to know than the physical conditions which certain symptoms may indicate; and, while attention should be given to the *tone of voice and manner of speaking, the attitudes and gestures, features, expression, and muscular firmness* of the patient, it is with the view rather of learning his constitution, a knowledge of which is so important in the treatment of disease.*

The term "Diathesis" (from Διατίθημι, to arrange) means the *tendency* to certain morbid states. *Dyscrasia* (from Δὺς, difficult; κράσις, temperament) and, *cachexia* (from κακος, bad; ἕξις, a habit) mean the *actual* morbid conditions themselves. The term constitution is used in a general sense, including tendencies and their results. It even, sometimes, is applied to the *temperament*. The study of "Physiognomy of Diseases," therefore, to the practical physician, means the study of the *constitutions*, including such morbid appearances and signs as are readily noticed. A short, condensed sketch of the different types is here given.

The Constitutions.—The *Sanguine* temperament is described by Laycock, who takes "John Bull" as the typical example. He is vigorous, healthy, and well-developed. The head is large, complexion ruddy, hair thick, and teeth good. His limbs are thick, feet large, and abdomen capacious. He is predisposed to rheumatic and gouty diseases —as angina pectoris, fatty heart, and apoplexy.

The *Rheumatic* constitution is a degenerate sanguine. It is less vigorous. The teeth are apt to decay, and arthritic symptoms begin to manifest themselves (see *Rheumatism*).

The *Lymphatic* temperament is also a degenerate sanguine. The patient is listless and phlegmatic. The complexion is pale. The body and limbs large and clumsy. The functions are sluggish. He easily passes into the strumous condition. The heart is often dilated, and recovery from disease is slow and imperfect. This temperament is common in women.

The *Strumous or Scrofulous Diathesis.*—Two types are described. The one has the body large and clumsy, head and chest ill-shaped, and the neck, ankles, and wrists thick. The complexion is pale, the alæ nasi are thick, and the teeth decayed. This is the degenerate *strumous*

* The student interested in Psychology should read "Laycock's Lectures," *Medical Times and Gazette*, 1862.

lymphatic constitution. The second type is the small-boned, thin, and *delicate* individual. The head may be small or large, the face generally small, and chin pointed. The complexion is finely tinted, nose small, and teeth small and apt to decay. The hair is fine in quality, and generally fair in colour. In both types the chest is badly shaped, the circulation and vitality weak, and the joints, the eyelids, and glands are apt to become affected with strumous disease. The fingers are often club-shaped. The appetite is fitful, and the mucous membranes are irritable. They are predisposed to phthisis and tubercular disease, and having little recuperative power, they recover slowly from acute affections.

The *Gouty Diathesis.*—The figure is neat and well-developed; hands and feet small. The complexion is high coloured, the nose has a tendency to redness, and the teeth are large, regular, and white. The hair tends to become early grey. The arcus senilis appears early, and is well marked. The patient looks full-blooded and robust, the heart being strong; and there is a marked tendency to gouty diseases—as angina pectoris, atheroma, fatty heart, apoplexy, aneurism, Bright's disease, &c.

The *Nervous Diathesis.*—The figure may be small. The head and face small, and chin pointed. The complexion flushed or pale. The patient has a bright look and restless manner—his nerves being "high-strung." There is liability to nervous disease—as epilepsy, hysteria, spinal disease, and insanity.

The other constitutions sometimes described require only to be named—the *bilious*, *melancholic*, *malarial*, *hæmorrhagic*, and *alcoholic*—as they either suggest the appearance, or they are mentioned in connection with their diseases. It is far more common to meet with the "mixed" varieties, and hence the *neuro-sanguine*, *neuro-vascular*, *neuro-arthritic*, *strumous-neurotic*, and other combinations, according to the more or less prominence of the different signs and symptoms present.

The foregoing types, therefore, are merely sketches of standards, by which the practical physician may classify his patients. The constitution should be kept well in view in the treatment of the case.

The Cachexiæ.—Whereas the constitutional conditions are the natural and inherited, the *cachexiæ* are acquired.

The *Cancerous Cachexia.*—Along with the debility and emaciation, there is an earthy or yellow-brown discolouration of the skin. The features are pinched and sharp, the expression anxious or pained. This cachectic appearance may be present in other painful diseases, besides cancer, as in severe hæmorrhoids, uterine and rectal tumours, &c.

The *Scorbutic Cachexia* has a somewhat similar discolouration of the skin, but there is also cyanosis, and a tendency to bleeding from the gums, &c., accompanied by the debility and other symptoms of scurvy.

The *Splenic Cachexia*, sometimes described, is the pallid appearance and other symptoms associated with Hodgkin's disease and leucocythæmia.

The *Syphilitic Cachexia.*—There is an unhealthy or dirty appearance of the skin, the bridge of the nose has fallen in, and the teeth are peg-shaped. There may be opacities of the cornea, loss of the hair, presence of nodes in the bones, fissures at the angles of the mouth, or other indications of tertiary syphilis.

The *Diabetic Cachexia.*—In this, there is a pinched or drawn appear-

ance of the face. The skin is dry, and often in the young, there is a hectic flush, suggestive of phthisis, which is frequently present. Pavy describes a "rolling and smacking of the tongue" (due to thirst) which has enabled him often to diagnose the disease.

Other cachexiæ are described as *malarial, alcoholic, tubercular, gouty, dropsical, &c.*; but these are treated in the systematic account of their respective diseases.

After the *preliminary information* comes the examination of the *general condition*, including the relations of **height and weight.**

A healthy man of

5 feet		should weigh	8 to 9	stones.
5 ,,	4 inches	,,	9½ to 10½	,,
5 ,,	8 ,,	,,	11 to 12	,,
6 ,,		,,	12½ to 13½	,,

i.e., a difference of 1½ stones for every 4 inches.

These are the weights usually given, but they are only approximate. They differ according to the development of the individual. Thus a thin, slender man, or "light weight," or the youth who has "shot up" rapidly, may be *below* the average, and the thick-set, robust, corpulent figure may be considerably *above* it—and yet the weights may not be incompatible with health.

The further examination of the case is indicated in the *case-taking chart*, at the end of the section; but in the ordinary course of practice it is not expected that such an exhaustive examination should be made in all cases. The methods used in the hospitals are intended to train the student to careful and thorough work, and to develop a good style. As much of the *method* is used in practice as is necessary for the correct diagnosis, prognosis, and treatment of the case. The *extent* of the disease should be known, and the examiner should always keep this in view. Experience will soon teach the student how much of the *method of case-taking* may be necessary, and the more obscure a case is the more thorough must the examination be. In such cases the chart may be useful to revise; and, if a consultation be necessary, the practitioner should previously examine *all* the systems again, in order to supply the consultant with the fullest information. *No* case can in any way be considered complete without an examination of the heart and pulse, and often of the urine; while inquiry should always be made as to the state of the stomach and bowels, and as to the presence or absence of cough or breathlessness—whatever organs be the seat of the primary lesion. The temperature is important in all acute cases. The diagnosis will rest upon the consideration of the history, symptoms, and physical signs. How much it may be necessary to inform the patient must be left to the judgment of the practitioner. In some cases, especially of minor affections, it is useful to explain to the patient the nature of his complaint; but in others, when the disease is a grave one, it is better to take the patient's friends into one's confidence, and to allow the patient himself to divine the gravity

of the case from the seriousness of the physician's manner. Sometimes, however, even in grave cases, it is better to inform the patient.

Hints on the Diagnosis of Children's Diseases.—Much may be learned from the *features* and *external appearances*. The cheeks are congested in feverish conditions; but transient circumscribed flushes of the face or forehead are indicative of cerebral disease. The latter may also be associated with squinting, inequality of the pupils, ptosis, &c.

Frowning is frequent in meningitis. A rapid wasting of the face, with sunken eyes and prominence of the cheek bones, points to a diarrhœal affection. Gradual emaciation may be due to a more chronic disease of the same nature—generally *tuberculosis*.

Lewis Smith considers a thick Meibomian secretion, puriform in character, and collecting between the eyelids, as a very unfavourable symptom in children. It is frequently present just before death, in severe cerebral and intestinal disease. An examination of the skin may also reveal the presence or absence of eruptions. The *attitude*, *movements*, or the character of the *voice* may be very suggestive. Irregular muscular movements, while conscious, indicate chorea. The fingers are frequently carried to the mouth, ear, or head in teething, otitis, or meningitis, &c. Rubbing the nose is common in cases of intestinal worms. In obstructive lesions of the throat, the child may clutch at the neck. In grave cerebral disease there may be a sharp piercing cry—the head being retracted, with rigidity of the limbs, &c., and light or noise may irritate or produce a convulsion. The voice may be hoarse or croupy (laryngitis). In pleurisy or pneumonia it is abrupt, or the words are uttered in short jerks. The voice becomes very feeble in abdominal affections, or in any severe wasting disease.

Circulatory System.—The pulse in children is very easily affected. It is increased in frequency—one hundred and twenty to one hundred and sixty beats per minute being very common in feverish states. Derangements of the digestive system, dentition, and the presence of intestinal worms, very readily affect the pulse in children. In meningitis it is frequently slow and intermittent. Lividity may be due to disease or malformation of the heart, &c.

Respiratory System.—The normal breathing in the young child is not only faster, but is apt to be irregular. It is frequently slow in cerebral disease, and much accelerated in capillary bronchitis. Obstructions of the larynx may cause the inspiratory portion to be prolonged. In pleurisy and pneumonia it is quick, and often accompanied by an "expiratory moan"; or abruptly terminated by pain. The breathing is also affected in abdominal disease when there is much distention.

The character of the cough may yield important information (see croup, bronchitis, pertussis, &c.). A cough may be an early symptom of measles and other fevers. It is frequent during dentition, and it may be the result of the presence of intestinal worms, irritating ingesta, &c. Such a cough is dry and painless. Dilatation of

the alæ nasi points to inflammatory disease of the respiratory organs.

Digestive System.—The ordinary examination of the mouth, tongue, and pharynx, &c., should be a matter of routine. Vomiting is not only a symptom of many diseases of the digestive system, but it is also an early symptom of cerebral disease, or it may usher in the symptoms of scarlet fever, &c.

Flatulence and distention of the abdomen, with colic pains, are very common; and in feeble, rachitic, or scrofulous children these symptoms may be more or less permanent. In meningitis, and exhausting diseases, the abdomen is often retracted. Vomiting, with *straining*, suggests intussusception.

The *stools* may be *green*, from over-feeding, irritating ingesta, cold, or inflammation. Almost all children have occasionally green stools. In enteritis the stools may be muco-sanguineous. Blood in the stools, with constipation, is suggestive of intussusception. In many diarrhœal affections the stools are acid and sour-smelling. Threadworms are common, and should be looked for by the nurse.

Nervous System.—A simple headache may be due to coryza or to commencing fever. If protracted it may indicate meningitis, &c. In acute inflammatory affections, children often have hyperæsthesia of the anterior surface of the trunk. Other symptoms of cerebral disease have already been mentioned.

Grave disease of the thoracic and abdominal organs may be present without much pain. A continuous pain, lasting for weeks, in the epigastric or umbilical regions, should suggest an examination for caries of the vertebræ. A pain in the knee should suggest disease of the hip.

The *temperature* is very easily raised in children. Two or three degrees need not excite any alarm in itself. If, however, 106° Fahr. be reached, there will, probably, be a fatal result if the fever be not shortly relieved by treatment. The temperature in meningitis rises slowly, but it never is high in the early stages. In diphtheria, the temperature often falls to the normal, during the stage of toxæmia.

By the kind permission of Dr. Wyllie, the complete method of Case-taking used in his wards in the Royal Infirmary, Edinburgh, is here appended :—

METHOD OF CASE-TAKING.

PRELIMINARY INFORMATION.

1. Name; age; married or single (if married, how many children); occupation; native place; present residence; by whom recommended. Date of admission, and No. of Ward (if in side room append letter S. to No.).

2. Very brief note of the chief complaints for which the patient has sought admission.

3. History (*a*) of present illness; (*b*) of previous health, social condition, and habits; (*c*) family history.

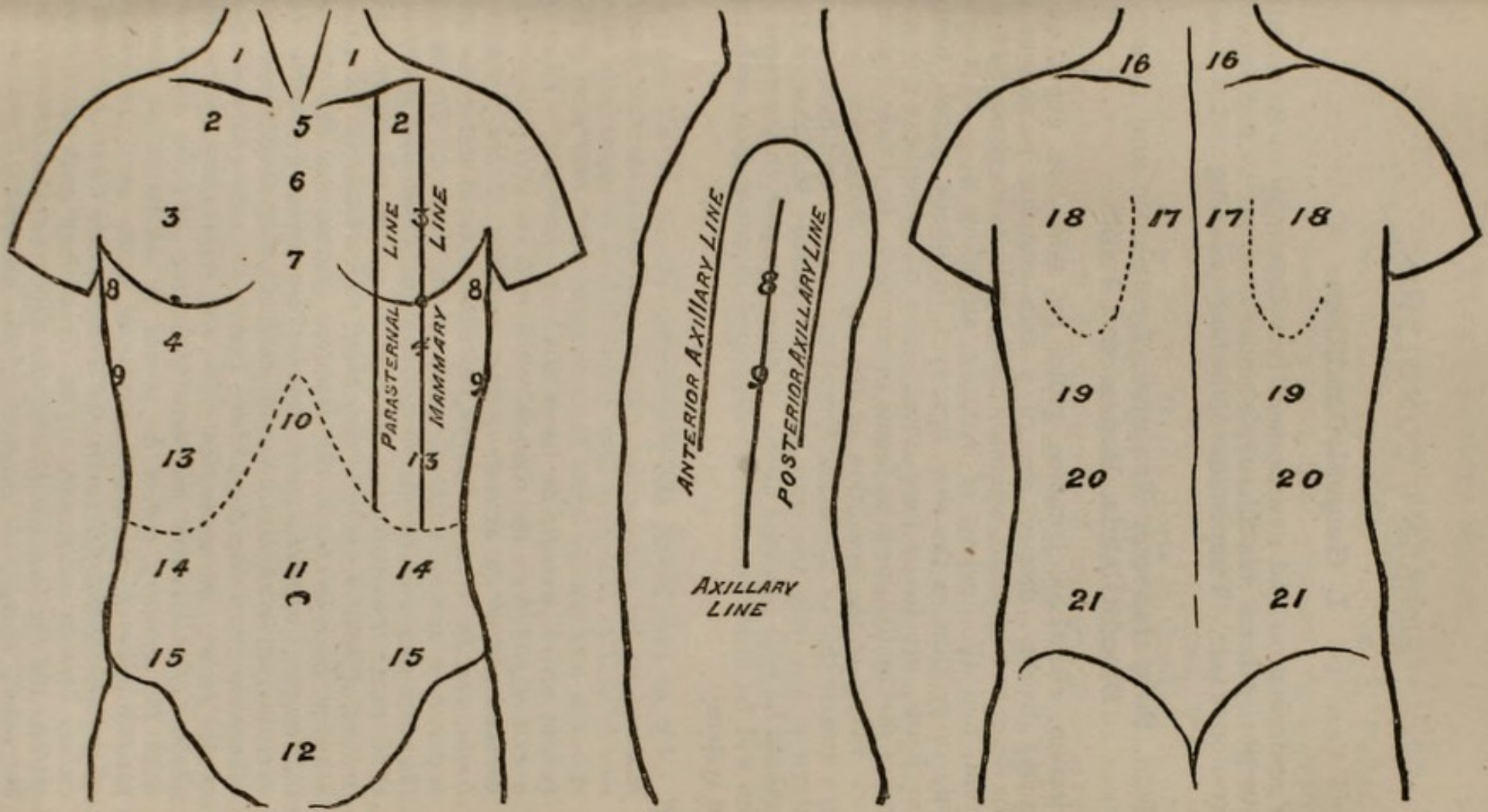


Fig. 1.—Regions of the Trunk.—1,1, Supra-clavicular; 2,2, infra-clavicular; 3,3, mammary; 4,4, infra-mammary; 5,6,7, superior, middle, and inferior sternal; 8,8, axillary; 9,9, infra-axillary; 10, epigastric; 11, umbilical; 12, hypogastric; 13,13, right and left hypochondriac; 14,14, right and left lumbar; 15,15, right and left iliac; 16,16, supra-scapular; 17,17, inter-scapular; 18,18, scapular; 19,19, infra-scapular; 20,20, 21,21, dorsal and lumbar

PRESENT CONDITION.

I. General Condition.

As to conformation and development (any deformity to be noted); height, weight, general muscularity, whether confined to bed, aspect, and expression of face. Temperature of the body (morning and evening).

II. Skin, Subcutaneous Textures, Lymphatic and other Blood-Glands, Bones, and Joints.

Perspiration, eruptions, jaundice, pallor (of skin, lips, gums, and conjunctivæ), lividity, dropsy, &c. (if a skin eruption is present it should be described in detail, attention being paid to its characters, its distribution, and the amount of irritation attending it). All marks of scrofula or syphilis in the skin, lips, eyes, lymphatic glands, teeth, bones, or joints, are to be here noted; also all articular signs and symptoms of rheumatism or gout (acute or chronic). In a case of goitre (simple or vascular) the condition of the thyroid body, in a case of Hodgkin's disease that of the whole lymphatic system, and in a case of leucocythæmia not only the state of the lymphatics but that of the spleen, should be here reported upon. (Other forms of enlargement of the spleen will be more conveniently described in connection with the digestive system).

N.B.—Up to this point, the programme should in all cases be followed in the above order, but in continuing the case it will now be well to take first that system which is apparently most diseased, and also in some cases to begin with that part of the system which presents the most striking symptoms. Thus in a case of paralysis the clinical clerk will do well to begin the nervous system by describing the condition of the paralysed limbs, and in a case of laryngeal disease he should begin the respiratory system with the state of the larynx. He should thus try to group in the foreground the prominent symptoms of the case. In treating of the various systems whose functions are not affected he may be very brief. He should in all cases, however, in referring to the digestive system, note the state of the tongue, appetite, and bowels; in connection with the nervous system the existence of good sleep or sleeplessness; in connection with the circulatory system the rate and condition of the pulse; in connection with the respiratory system the absence of all cough and breathlessness; and in connection with the urinary system he should in all cases, as soon as possible after the patient's admission, make an examination of the urine, and fill in on the Temperature Chart the particulars therein indicated. The circulatory and respiratory systems on the one hand, and the digestive and urinary on the other, should, on account of the anatomical position of their organs, be regarded as companion systems, and should always be treated of in juxtaposition.

III. Circulatory System.

1. *Subjective Symptoms.*—Cardiac dyspnœa, palpitation, pain at precordia, syncope or faintness, angina pectoris, &c.

2. *Characters of the Radial and other Superficial Pulses.*—Pulse-rate; nature of the radial pulse as regards its regularity, force, and fulness; tension of the artery from blood-pressure; rigidity, tortuosity, or calcareous degeneration of the vascular coats, from disease; equality or inequality of the two radial pulses. Condition as regards tortuosity, &c., of the temporal and other superficial arteries.

3. *Physical Examination* by inspection, palpation, percussion, and auscultation of (*a*) the Heart; (*b*) the Aorta in thorax and abdomen; (*c*) the Large Arteries and Veins in the root of the neck. (In describing a cardiac or vascular *bruit* note should be made of its acoustic characters, its seat of maximum intensity, its relation to the ventricular systole or diastole, and the extent and directions of its propagation. In describing the area of cardiac dulness the limits of both superficial and deep dulness should be given).

4. *Examination of the General Circulation.*—Varicose veins and hæmorrhoids; blueness or coldness of the hands and feet, and point of nose; lividity or pallor of skin and mucous membranes; cardiac dropsy. (If dropsy, &c., have been already described under the heading No. II., they need here only be referred to as already described.)

5. In Special Cases (anæmia, leucocythæmia, &c.), give the results of a microscopic examination of the blood, including an enumeration (by means of special apparatus) of the red and white corpuscles, and an estimate of the hæmoglobin.

IV. Respiratory System.

1. Physical Examination of Chest.

(*a*) *Inspection.*—Measurement of chest at level of nipples; general form of chest, with special note of any general or local flattening or prominence. Condition of costal parietes, as regards emaciation, distinctness of intercostal spaces, &c. Action of the chest during respiration; number of respirations per minute, their character, easy, laborious, or wheezing; note whether inspiration or expiration is the more laborious and difficult; note also how much the manubrium sterni and clavicles are drawn up during ordinary inspiration; note action of alæ nasi; also the attitude or "decubitus" of the patient, whether he is able to lie down or requires constantly to sit up, or be propped up, in bed (orthopnœa); compare by means of inspection and palpation the expansion of the two sides during inspiration. Ask the patient to cough, and note if emphysematous apices are bulged up into the root of the neck.

(*b*) *Palpation.*—Compare, as just indicated, the expansion of the two sides; also test, and compare on the two sides, the "vocal fremitus," or thrill of the voice.

(*c*) *Percussion.*—Test the characters of the percussion note over the various parts of the chest, noting the degree and limits of any local hyper-resonance, or dulness, or "crack-pot sound,"

(*d*) *Auscultation.*—Character of breath sound. Morbid accompaniments, such as ronchi, crepitation, or friction. Character of vocal resonance when patient speaks aloud and when he whispers: note any local increase, diminution, &c.

2. Subjective Symptoms.

Difficulty of breathing. Rapid or laborious breathing having been already described under "Inspection," note here if the patient suffers much distress or loss of sleep from it; also whether it is constantly present or only comes on during exertion, or paroxysmally from other causes. Cough: its character, severity, and frequency; if it deprive patient of sleep, &c. Expectoration: its amount; its physical characters, as regards viscidity, wateriness, frothiness, colour, presence of pigment, blood, pus, &c. Odour of breath and expectoration; special note to be made of any fœtor. Microscopical examination of the sputum. Pain in chest: its character, situation, and limitation.

3. Examination of the Larynx.

In describing the symptoms of a laryngeal case, bear in mind the four following functions performed, or taken part in, by the Larynx. (*a*) *Phonation.*—Is the voice natural, husky, weak, or altogether lost? (*b*) *Coughing.*—Is the cough natural in sound, husky or loudly metallic and ringing? (*c*) *Deglutition.*—Is there pain in the larynx during deglutition, or choking from the entrance of food or fluid into the air-passages? (*d*) *Respiration.*—Is there any impediment in the Larynx, as indicated by stridor in breathing; and if so, is the difficulty constant, or paroxysmal? If paroxysmal, note the time (day or night) during which the paroxysms most frequently occur, and state, if possible, by what causes they seem to be induced.

Examine the Larynx externally by palpation. Note the presence of any tenderness to touch, or of any swelling.

Results of Laryngoscopic Examination.

4. State of the Nares and Eustachian Tubes.

Catarrh, &c. Characters, microscopic and otherwise, of the secretion. Fœtor of breath.

V. Digestive System.

1. Subjective Symptoms.

Appetite, thirst, deglutition, digestion, action of bowels, &c.

Difficulty in Deglutition.—Cause the patient to indicate as clearly as possible the seat of obstruction; report result of examination with bougie, if such have been made.

Pain in abdomen.—Note situation and limits, character and degree of severity, if constantly present or intermittent, if aggravated or relieved on pressure. When pain or uneasiness is situated in the region of the stomach, enquire if it extends through to the back, if it is relieved when food is taken, or if, on the other hand, it comes on, or is distinctly aggravated, after meals; if the latter, carefully note the usual interval after meals at which the accession or aggravation of pain occurs. If *paroxysms of pain* occur only at rare intervals, note their exact situation, their severity, their mode of accession and disappearance (gradual or sudden), and their accompaniments and sequelæ (such as vomiting and

slight jaundice, &c.); and mention any cause which the patient may have observed as probably productive of them.

Vomiting.—Note its relationship to meal-time, its frequency and severity, and the characters (microscopic and otherwise) of the vomited matters. Note particularly the presence at any time of blood or coffee-ground like material. If vomiting be of acute character and of recent origin, as in a case of obstruction of the bowels, examine at once for hernia, in all the hernial localities.

Gaseous eructations, heart-burn, and water-brash.—Note their relationship to meal-times, and give the patient's experience as to the articles of food he may have found most apt to produce them.

Diarrhœa.—Note if any apparent relationship to meal-times, or to particular articles of food; character and frequency of the evacuations; presence of griping pain in the abdomen (tormina), or bearing-down pain about the rectum (tenesmus).

Constipation.—State how often the bowels are evacuated. If bowels are apparently "*obstructed*," state precisely the day and hour of the last evacuation, and give patient's statement as to its nature and amount.

2. Objective Examination.

State of tongue, teeth, fauces, and pharynx.

Physical examination of the abdomen, by inspection, palpation, and percussion.

If abdomen is *enlarged* from other causes than mere adiposity, take its *circumference* at the umbilicus, examine by palpation for fluctuation, and if this be present percuss and observe the limits of tympanitis and dulness, noting particularly how much the tympanitis and dulness remain fixed or shift about when the patient is turned from the back upon the side, and from one side to the other. If any *tumour* should be detected on palpation, state in what region of the abdomen it is situated, and describe particularly its apparent size, its hardness, and the distinctness or indistinctness of its outline. (The regions of the abdomen are nine in number, and are limited by four imaginary lines: two drawn down perpendicularly from the cartilage of the eighth rib to the middle of Poupart's ligament on either side; and two drawn horizontally across the abdomen, the upper at the level of the lowest part of the costal arches, and the lower at the level of the highest part of the iliac crests. Of the nine regions thus marked out, the three upper are the epigastric in the centre and the hypochondriac on each side; the three middle are the umbilical in the centre and the lumbar on each side; and the three inferior are the hypogastric in the centre and the iliac on each side.)

In many cases of abdominal disease, and particularly in cases of obstruction of the bowels, important information, positive or negative, will be obtained from a *digital examination of the rectum*. In all cases of anæmia inquire as to the existence of bleeding hæmorrhoids.

In dealing with a case in which one or other of the *solid abdominal organs*, such as the liver or spleen, is enlarged, ascertain and describe the limits of the enlargement, as in the case of a tumour. Note any irregularities, such as protuberances, on the surface of the organ.

VI. Genito-Urinary System.

Urine.—Amount in twenty-four hours; specific gravity; colour and transparency; acidity; odour; deposits; chemical and microscopical examination.

(In the case of every patient admitted the state of the urine should be noted upon the temperature-chart as soon as possible after the patient's admission, and a fuller account of it should be made in the general report of the case.)

Examination of the kidney, bladder, urethra, &c.

Menstruation; condition of the genital system, with note of abnormalities.

VII. Nervous System.

In cases of *Convulsive or other Seizures*, note manner of onset, and the progress of the attack. Character of the convulsions; character of the breathing; complete or partial loss of consciousness; indications of hemiplegia; localised spasms, &c.; state of pupils (contracted, dilated, unequal, insensible to light, &c.). If the patient is not seen during the attacks by the house-physician or clinical clerk, as good an account of them as possible should be obtained from the friends or others who may have seen them. Describe the mental and physical condition of the patient in the intervals between the seizures.

In examining the various functions of the nervous system in chronic or subacute cases which may admit of detailed examination without detriment to the patient, the following notes may perhaps be of use:—

1. The Motor Functions.

In cases of Paresis or Paralysis, define the limits of the condition, and indicate in what degree the motor power is lost.

In examining the *upper extremity*, if the case be one of only partial loss of power, compare the grasp of the affected with that of the sound hand. Test *co-ordination of movement* by causing the patient to pick up minute bodies, or to write, &c. Test the *muscular sense* by putting into the patient's hands, his eyes being closed, objects of the same size but of different weight, such as coins, &c.

In examining the *lower extremities*, if the patient is able to walk cause him to do so. Note how much he betrays *loss of muscular power*, in any slowness of movement, or appearance of great effort, or dragging of the feet, or scraping upon the floor with his toes. Note if he presents, as evidence of the loss of *co-ordinating power*, the high-stepping irregular action of the ataxic; if so, ascertain how much the want of co-ordination is associated with loss of muscular power by causing the patient to lie down in bed, and when he is in this position, try what resistance he is able to overcome in flexing and extending the legs. Test his *balancing power* by observing any staggering in his gait as he walks, noting particularly if he can turn round without staggering; cause him to walk along a straight line marked on the floor; and cause him to stand upright with heels and toes together and eyes closed. Examine as to the *reflex functions of the spinal cord*. Test especially the *plantar* and the *knee* reflexes, and try if you can elicit the *ankle clonus* (the plantar is a superficial, and the knee reflex and ankle clonus are deep reflexes). The other superficial reflexes which may be tested in some cases are the gluteal, cremastric, abdominal, epigastric, erector spinæ, and scapular. Inquire as to the condition of the *organic reflex centres* situated in the lumbar enlargement of the cord, namely, the centres for micturition and defæcation and the sexual centre. In morbid states of the first distinguish between

dribbling of urine from an over-distended bladder, constant dribbling from an empty bladder, and frequent involuntary contraction and evacuation of the bladder; always state in what degree the patient is conscious of the evacuations. If thought desirable, the *muscular sense* of the lower extremities can be tested by hanging weights from the feet, whilst the patient sits upon an elevated seat with the feet dependent.

In examining a case of *Facial Paralysis*, note if the patient can close both eyes perfectly, how much the mouth is dragged to one side, how much the natural wrinkles of one side are obliterated; cause the patient to whistle, and note action of lips and cheek; note any drooping of the arch of the palate on one side, and any loss of taste on one side of the tongue; observe if the patient protrudes the tongue in the middle line, or if it inclines to one side.

In *Paralysis of any kind* examine and report upon the *condition of the paralysed muscles:* whether firm and of good tone; or flaccid and deficient in tone; or rigid and contracted (spastic paralysis—when rigidity present always note attitude of the limb); or markedly atrophied (amyotrophic paralysis); or presenting an appearance of increased bulk and unnatural development (hypertrophic paralysis). In some cases report as to the reactions of the muscles when subjected to the Galvanic and Faradaic currents.

In reporting upon a case of nervous disease which presents *spasmodic* or *rhythmical movements* of the body, either general or localised, the nature, extent, and duration of those movements should be described. If they are localised to particular groups of muscles, their limits should be defined as strictly as possible, and it should be particularly stated if, in the same groups of muscles, there is any degree of paresis or paralysis.

2. Trophic Functions.

Some of the derangements of trophic functions have been already alluded to in connection with the muscles. There are many other important trophic lesions, such as those of the skin (examples of herpes and other inflammatory eruptions, certain atrophies of hair and pigment, "glossy skin," &c.), and of the joints (the arthritic changes in locomotor ataxy, &c.); but all of these will have been treated of more conveniently under the heading No. II. than under the "Nervous System."

3. Sensory Functions.

(*a*) *General Sensations of Patient.*—General feeling of well-being or exhaustion. Vertigo, constant, occasional, or occurring only when patient walks. Headache, over whole head or limited in area, frontal, vertical, occipital, unilateral, or bilateral, deep or superficial, constant or periodic, aggravated or not at night, with or without tenderness of head to touch or pressure. Backache, with any specially tender spot. Ovarian, infra-mammary, and lumbar pains. Neuralgic pains in divisions of fifth pair, in great sciatic, &c. General or wandering pains. Sudden shooting (lightning) pains about the body or in the joints.

(*b*) *Special Senses—Vision.*—Protrusion of eyeballs; strabismus; nystagmus; ptosis; lagophthalmos; spasms of the palpebral muscles. Indications of recent or old inflammation of the iris, &c. Size and equality of the pupils; contraction of pupils on exposure to strong light and on accommodation of the eyes to near vision. Double vision. Imperfect sight from errors of refraction or from disease of external or

middle parts of eye. Imperfect sight or blindness from disease of brain, optic nerves, or retina; dimness of sight, limitation of the field of vision, loss of colour sense, blindness of one half of each retina, patches of blindness in the field, spectra (such as dark spots, sparks or flashes of light, coloured spectra). Results of ophthalmoscopic examination.

Hearing.—Defect of hearing on one or both sides. Tinnitus aurium. Examination with watch or tuning fork. Results of examination with otoscope.

Taste.—Test each side of the tongue with sweet and bitter, also with acid and salt substances, the tongue being protruded during the examination.

Smell.—Test each nostril separately with various odours, as those of strong-scented flowers.

(*c*) *Cutaneous Sensibility.*—Test the *tactile* sensibility of the skin by touch with finger. If an exact estimate is required use Weber's test, the compasses, and note at each spot examined the shortest distance between the points that permits of the patient's recognition of their separate impact. Test sensibility to *pain* with the point of a pin. Test sensibility to *heat* and *cold* with two test tubes, one filled with cold and the other with hot water; or with hot and cold sponges. Note all abnormalities, such as anæsthesia, or hyperæsthesia, or creeping feeling (formication), or feeling of "needles and pins" (prickling).

Note.—In describing a case of paralysis, it is best not to take the sensory functions in the above order, but to begin with "cutaneous sensibility" immediately after having described the motor and trophic conditions of the part paralysed.

4. The Intellectual Functions.

How is the general consciousness or intelligence affected? Is it well retained, or is it obscured or perverted? Note the condition of the memory, and of the power of sustained attention. Note how the emotional nature is affected, particularly if *self-control* is maintained or impaired.

Loss of Language.—Distinguish between the loss of the power of calling up words in the memory (amnesic aphasia) and the difficulty of articulating words which may have been rightly enough chosen (ataxia aphasia). In the former case note in what degree the memory for words is obliterated or confused; and in the latter case note what consonants the patient has most difficulty in articulating. In both varieties of aphasia cause the patient to write to dictation, and preserve a specimen of what he has written.

5. The Sleep Function.

Character and amount of sleep at night, dreams, somnambulism, &c. Sleepiness during day, &c.

VIII. Treatment of the Case, and Notes from time to time of its Progress.

N.B.—The Clinical Clerk will be careful, in taking a report, not to exhaust the patient by too prolonged conversation or examination, caution in this respect being especially needed when the

patient is feverish or very ill. In cases of very severe illness, the Physician or Resident Physician will furnish what facts may be required as to the physical examination, and a history of the case may sometimes be obtained from the patient's friends.

The Clinical Clerk need not burden his report by using all the headings of the above programme. He is at liberty to select only such headings as he may think required for the particular case he may be describing, and to arrange them as may best conduce to clearness and consecutiveness.

When a patient is discharged, the Clinical Clerk will finish his notes, and report the case as finished to the Resident Physician. In the event of a patient's death, an abstract of the pathological report should be added to the notes of the case.

P.S.—In drawing up the above programme, I have in some particulars followed the "Method of Case-taking" used by the late Professor Sanders, as given in Finlayson's Clinical Manual *p. 37.*

CHAPTER II.

DISEASES OF THE CIRCULATORY SYSTEM, &c.

Contents.—*Medical anatomy.* The chronic valvular diseases of the heart; *Method of auscultation of the valves*—Dilatation, hypertrophy, and fatty disease of the heart—Endocarditis, ulcerative endocarditis, and pericarditis—Functional diseases of the heart, *viz.*, *palpitation*, *irregularity and intermittency*, *irritable heart*, *syncope*, *angina pectoris*—Exophthalmic goitre—Rarer conditions—Diseases of the blood-vessels, *viz.*, *thrombosis and embolism*, *endarteritis*, *and aneurism*—**Mediastinal tumours.**

Medical Anatomy (see Fig. 2).—The heart lies obliquely between the lungs—the base upwards to the right, and the apex downwards and forwards to the left. It extends, vertically, from the level of the second intercostal spaces to the lower part of the body of the sternum. The apex *beat* is found in healthy persons in the fifth intercostal space of the left side, between the parasternal and mammary lines—*i.e.*, *about three inches from the middle line of the sternum.* In children, it is often felt as high as the fourth intercostal space, and in aged persons it may occasionally be found as low as the sixth. If the patient be turned upon the left side it extends slightly beyond the mammary line. The *true* apex is about three quarters of an inch nearer the mammary line, at the level of the sixth costal cartilage, but here it is overlapped by the left lung. On the right side, at the level of the fourth costal cartilage, the heart—chiefly right auricle—extends about half an inch beyond the right edge of the sternum.

From these points—*viz.*, the apex, the lower part of the body of the sternum, the point half an inch from the right edge of the sternum, and the second intercostal spaces—curved lines may be drawn upon the anterior wall of the chest, indicating the area occupied by the heart. In percussion of the heart, this will be the area of *relative* dulness; but, practically, it is only necessary to percuss in the vertical and transverse diameters. The superficial, or *absolute* dulness, indicates that small portion of the heart—right ventricle—uncovered by lung, and its area forms an irregular four-sided figure having the following boundary lines—*viz.*, *the left*, from the apex beat to the lower border of the fourth left costal cartilage, about midway between the parasternal and mammary lines; *the upper*, from this latter point, runs along about the level of the fourth left costal cartilage to meet *the right* at the middle line of the sternum, extending from the fourth to the sixth costal cartilage; *the lower*,

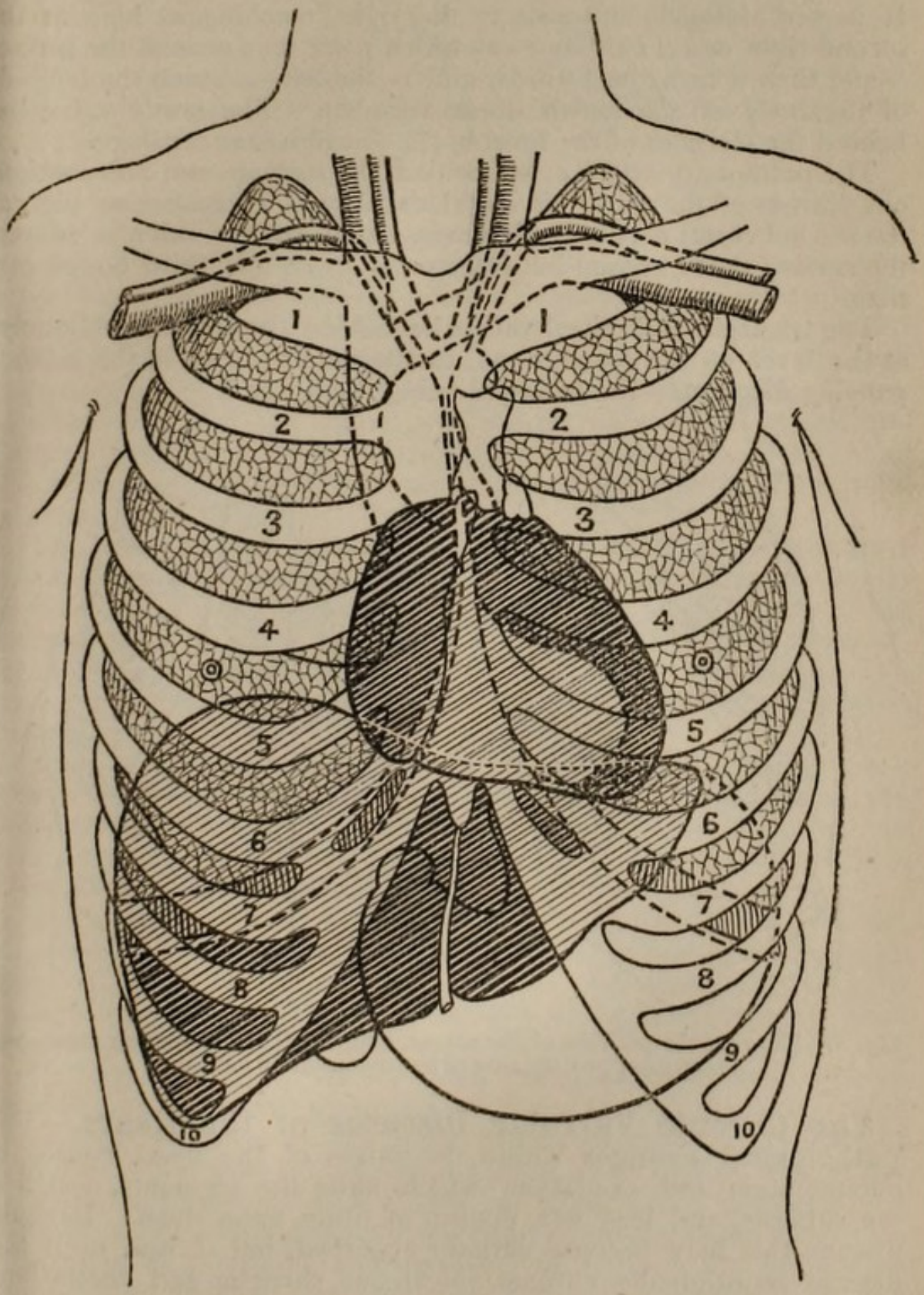


Fig. 2.—(Based upon Joessel), see Text.

from this point to the apex beat—an imaginary line, as the liver dulness does not allow of its being defined by percussion.

The aorta arises behind the sternum, immediately to the left of the mesial plane of the body, at the level of the fourth costal cartilage. It passes obliquely upwards to the right, reaching as high as the second right costal cartilage—at which point it is nearest the surface—and then it turns backwards, and to the left, to reach the left side of the body of the fourth dorsal vertebra. The aortic valve lies behind the sternum at the level of the fourth costal cartilage.

The pulmonary artery arises behind the sternum and the contiguous portion of the third left costal cartilage. It reaches as high as the second costal cartilage of the same side, but the valve is nearest the surface at the second left intercostal space and third costal cartilage.

The tricuspid and mitral valves lie behind the sternum obliquely, at the level of the fourth costal cartilages, as shown in the accompanying diagrams—the tricuspid being the lower.*

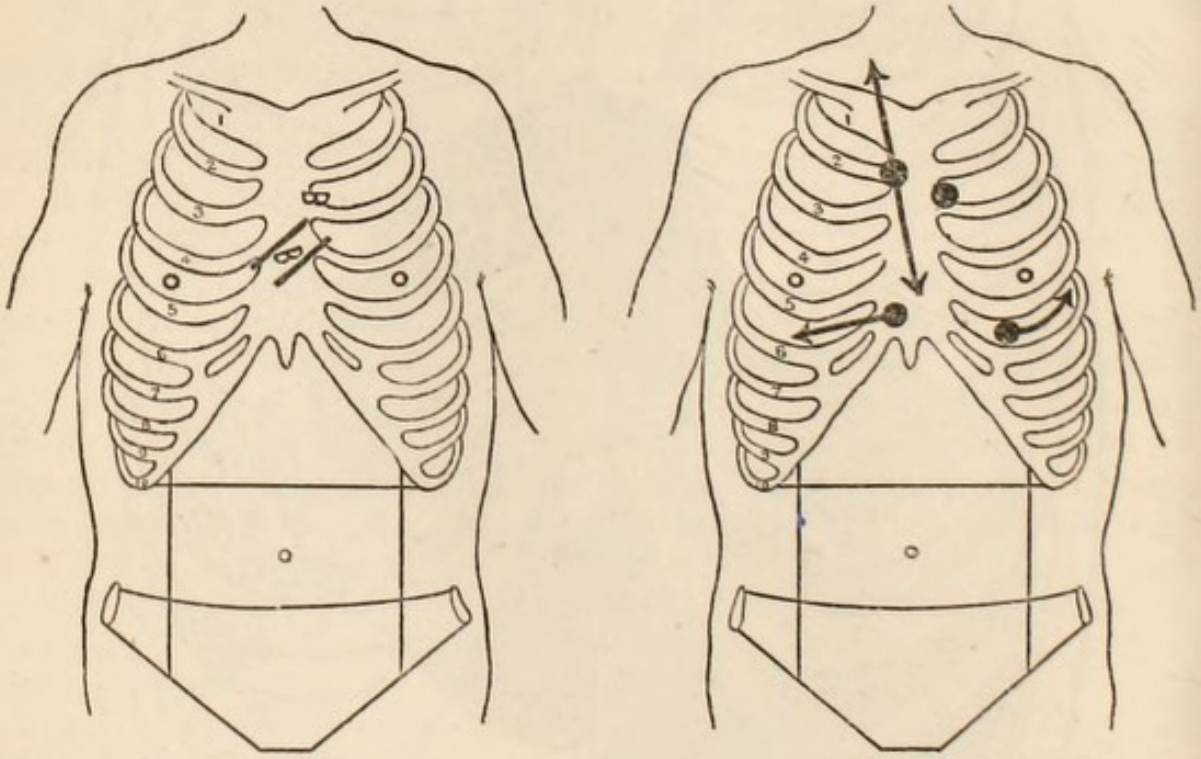


Fig. 3.—Showing the position of the valves, and also the auscultation areas, and direction of pro agation of heart-sounds or murmurs.

The Chronic Valvular Diseases of the Heart.—The pathological changes within the valves of the heart consist of inflammation and exudation, which unite the segments, roughen the surfaces, and lead to a deposit of fibrin upon them. In acute disease this may become entirely absorbed, but if not, or if the disease be originally chronic, calcareous particles and vegetations

* The anatomical relations of the heart and valves to the anterior wall of the chest, may be tested in the *cadaver*, by thrusting three or four long knitting wires between the ribs, close to the sternum, before opening into the chest. As the shape of the chest varies in individuals, there is no *absolute* precision possible in the relations of the heart and valves to the anterior wall.

are deposited within the valves, and connective tissue is developed, leading to subsequent shrinking. The shape of the valve is altered. The mitral may become a mere slit, admitting only the tip of the finger, instead of three fingers as in the healthy valve. This gives rise to the condition of mitral *stenosis*. But inflammation of the mitral with shrinking of the segments of the valve may leave a condition quite the opposite, allowing of *regurgitation* of the blood, instead of offering an obstruction to the flow. This latter condition can also arise from a dilated left ventricle without structural changes in the valve.

The aortic valve, by similar inflammatory changes—and also by the extension of atheromatous disease from the aorta—may become thickened and the segments united, the edges being sometimes ragged and shapeless, and sometimes a segment may be entirely gone. Small valvular "aneurisms" are sometimes formed, or the segments of the valve may be much thinned, and they may have slits in their substance giving them a fenestrated appearance. The result of these conditions is that the valve becomes permanently impaired, and hence aortic stenosis and incompetence. Aneurismal dilatation of the aorta may, however, give rise to incompetence without structural changes in the valve.

The tricuspid valve may be affected along with the mitral in acute disease, but this rarely occurs, and seldom leaves *chronic* valvular disease. A dilated right ventricle, secondary to obstruction of the circulation in the pulmonary vessels or at the left side of the heart, frequently gives rise to tricuspid regurgitation, but stenosis is extremely rare. Pulmonary stenosis and incompetence are rare and usually congenital.

The secondary changes following the chronic valvular lesions are—the dilatation of the heart from the overfilling of its cavities, the hypertrophy of the heart caused by the increased work necessary to carry on the circulation, and, in the later stages, degenerative changes within the muscular fibres.

The secondary effects upon the other organs are due to the diminished tension within the blood-vessels in front, and engorgement of them behind, the obstruction; and these effects occur either during the early stage of dilatation of the heart, or later, when the compensatory hypertrophy begins to fail.

If there be stenosis of the aortic valve, incompetence, or both conditions present, the result is dilatation of the left ventricle, which soon becomes hypertrophied. When this compensatory hypertrophy fails, and dilatation of the left ventricle again begins, the mitral becomes incompetent and allows of regurgitation into the left auricle, which soon becomes dilated, and thus the pulmonary veins are engorged, producing congestion of the lungs. The pulmonary arteries also become engorged, and then follow in the same way dilatation of the right ventricle, right auricle, and systemic veins, producing ultimately dropsy of the cellular tissues and serous cavities. Death may then result from gradual and sometimes sudden failure of the heart.

If the mitral valve be the seat of the disease—either stenosis or regurgitation—the left ventricle does not become dilated as it is now in front of the obstruction, but the left auricle does, and in stenosis may be enormously so, the pulmonary vessels, right side of the heart, and systemic veins becoming engorged as before.

The accompanying scheme of the circulation shows how congestion of the other organs takes place wherever the obstruction exists; and explains the apparent irrelevancy of the symptoms of patients

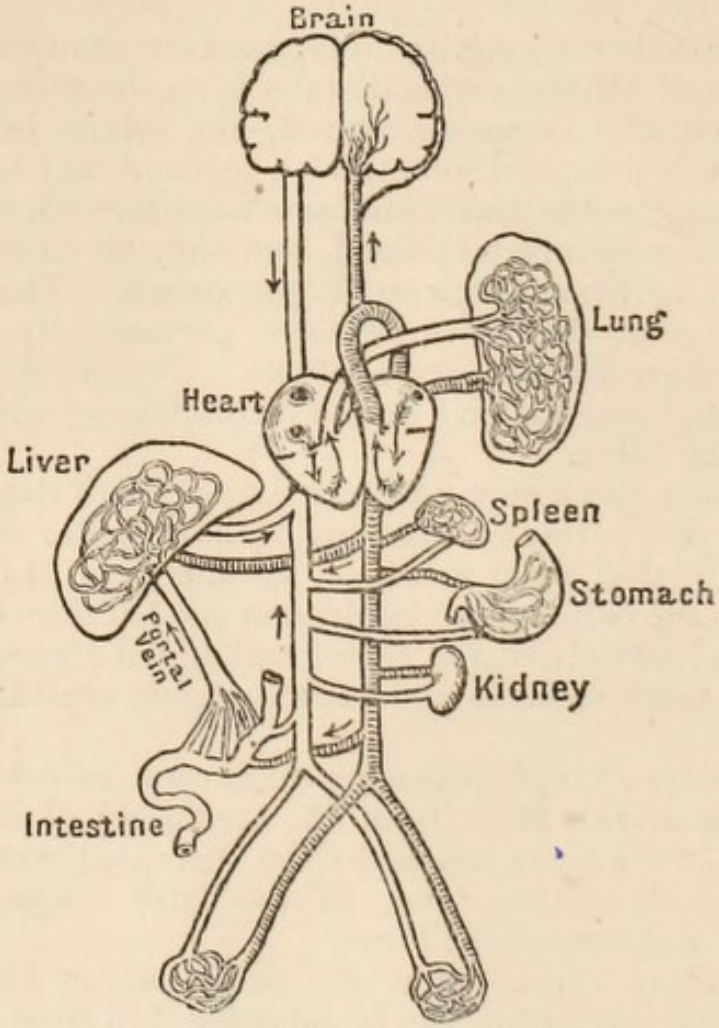


Fig. 4.—Scheme of the Circulation.

suffering from heart disease. The conditions produced in the lung are congestion, brown induration, and œdema. Hæmorrhages (pulmonary apoplexy) often occur within the lung tissues, and small patches of pneumonia may develop, either the result of inflammation around infarctions, or the effects of cold upon the congested lung. Bronchitis is very commonly combined with heart disease, and the two affections re-act upon each other, the heart disease producing congestion of the lung and bronchi, while bronchitis (especially when associated with emphysema) produces an obstruction to the circulation within the pulmonary vessels, and hence dilatation of the right heart and systemic veins, with the resulting dropsy, as in valvular lesions. Congestion and enlargement of the liver (nutmeg liver) giving rise to hepatic symptoms, and congestion of the stomach, spleen, kidneys, uterus, &c., with hyperplasia of these organs, are

all consequences of the heart's failure to maintain the circulation. Thrombi may form in the stagnated blood, and emboli from them or from the diseased valves may pass into the circulation, producing infarctions in other organs. An embolus in the middle cerebral artery is a frequent cause of hemiplegia, often with aphasia; but it should be noted that atheromatous disease of the cerebral vessels often co-exists with the aortic disease, and hence the hemiplegia, in such a case, may be due to cerebral hæmorrhage and not to embolism.

The **causes** of chronic valvular disease of the heart are endocarditis, whether rheumatic, simple, or choreic in origin—the rheumatic being the commonest; scarlatina, diphtheria, and pyæmia; and atheromatous disease of the aorta—which may appear early in gouty and rheumatic subjects, and in cases of lead poisoning, syphilis, and chronic alcoholism. Also, laborious occupations which throw extra stress upon the valve favour the development of aortic disease.

The **symptoms** and **physical signs** vary with the condition of the heart. Should the extent of the valvular disease and the amount of compensatory hypertrophy be nearly balanced, there may be no symptoms complained of by the patient, and the lesion is then said to be *mute*. But when the heart fails to respond to the extra call that is made upon it, the symptoms present will depend upon the extent, seat, and stage of the disease. The patient may complain of *palpitation*, directly due to the valvular lesion, or to the secondary consequence of a congested stomach, giving rise to dyspepsia with flatulence, and thus interfering with the heart's regular action. It is common for patients suffering from heart disease to refer their symptoms to the stomach, and *vice versâ*. *Pain* is often complained of, from dull stretching pain or uneasiness in the precordial region or left side, to the severe agonising pain known as angina pectoris. The latter usually indicates degenerative change (fatty disease) within the fibres of the heart.

Breathlessness is marked in all stages of valvular disease when compensation is incomplete. It is most obvious upon making exertion of any kind, and thus differs from the dyspnœa of lung disease, which may be apparent while the patient is at rest. *Cheyne-Stokes'* breathing is a peculiar form of respiration, beginning with shallow breaths, gradually increasing in depth and rapidity, and as gradually fading until the patient seems to have ceased to breathe; but in a few seconds the cycle is repeated. It may indicate fatty degeneration of the heart; but it occurs also in cerebral hæmorrhage and other brain diseases. *Syncope*, *headache*, *giddiness*, and *tinnitus aurium* are frequent symptoms of valvular disease, all due to disturbance or failure of the circulation. *Sleeplessness* is also a common symptom. *Cyanosis* and *dropsy* are marked signs in mitral disease, and in the later stages of aortic disease when the ventricles become dilated. The cyanosis may exist from the faint blueness of the lips and dusky look of the lower eyelids, to the blue-black appearance noticed in extreme cases. The tip of the nose, the ears, and finger nails are the special sites, and often the superficial veins are prominent. The

dropsy generally begins on the dorsum of the foot, and extends upwards. Dropsical patients have frequently attacks of *diarrhœa*. The characteristic *facies* of mitral or aortic disease may be present—a flushed look over the malar bones in the former, and a pale anæmic condition in the latter. There is *mental weakness* in advanced and long-continued valvular disease, the brain being poorly nourished. This symptom is more obvious when emboli pass into the circulation and block the cerebral arteries. *Hemiplegia* is then usually present, and if the clot should be on the left side and cut off the blood supply from Broca's convolution, *aphasia* also results. Emboli may be the cause of *epileptiform convulsions*, but this symptom may also arise from simple and sudden disturbances of the circulation.

The congestion of the lungs with *cough*, and often *hæmoptysis*—the expectoration being usually pink in colour—and the symptoms and physical signs of *œdema* of the lungs, and of *bronchitis*, are described under diseases of the respiratory system. Should a large *hæmorrhagic infarction* be produced in the lung, there will be hæmoptysis and dyspnœa, perhaps produced suddenly, and if large and superficial, the physical signs of consolidation—as bronchial breathing, increased vocal resonance, and dulness on percussion—may be made out. Sudden death may take place from these large infarctions; or they may end in gangrene of the lung, and subsequent *pneumothorax*. Smaller infarctions may heal, leaving cicatrices, and, if superficial, they may light up *pleurisy* with pain and fever.

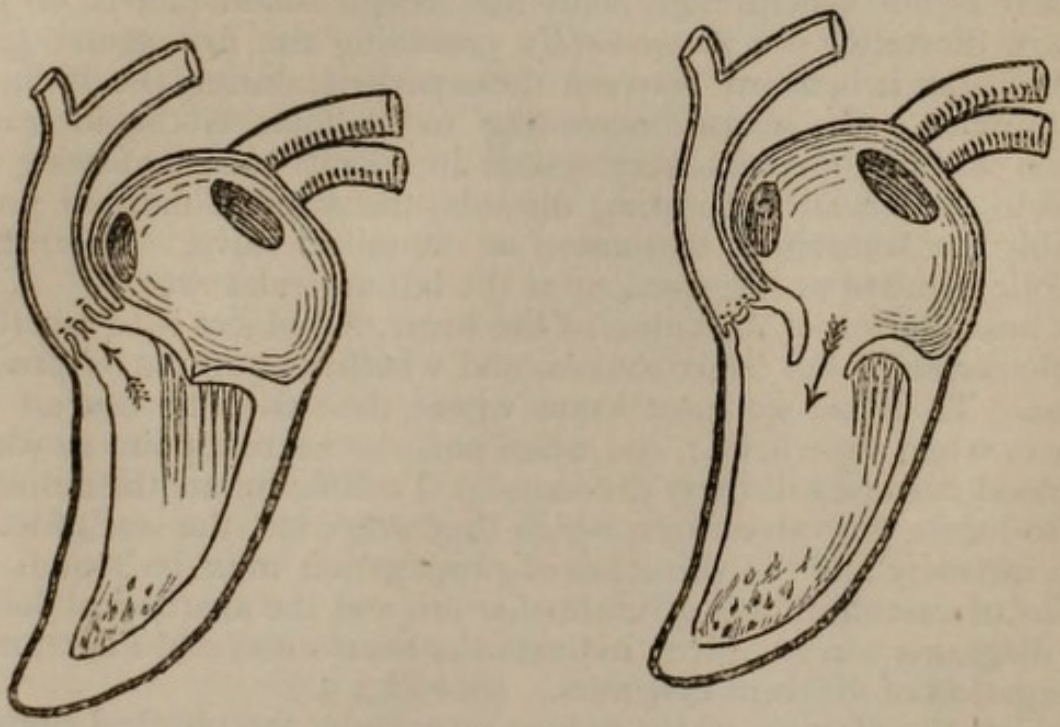
Should the liver be deeply congested, slight *jaundice* may be present, and percussion may reveal the liver to be enlarged.

The presence of *albuminuria* and tube casts, with diminution in the quantity of urine, will indicate congestion of the kidneys; while *hæmaturia*, if present, may be due to the congestion, or be the result of embolic infarction. It may be sometimes difficult to say in a case of albuminuria, with bronchitis and a dilated heart, whether the disease is originally one of heart disease or Bright; but an enlarged liver, in this respect, points to heart disease. In the female the uterus may be congested, and *menorrhagia* may be present as a symptom.

To explain the *murmurs* heard in valvular disease of the heart, and to locate their seat of origin, the normal sounds must be understood. The heart may be looked upon as a specially altered blood-vessel, dilated and surrounded by muscular fibres, to form the propelling organ. It is a hollow muscle, with four chambers and four valves. The right auricle, receiving the blood from the venæ cavæ, communicates with the right ventricle through the *tricuspid* valve; and the right ventricle sends on the blood through the *pulmonary* valve into the pulmonary arteries. The left auricle, receiving the blood from the pulmonary veins—having circulated through the lungs—communicates with the left ventricle through the *mitral* valve; and the left ventricle propels the blood into the aorta through the *aortic* valve.

The contraction of the heart is called the systole, and it begins

with the silent contraction of the auricles, and is immediately followed by the contraction of the ventricles, corresponding with the first sound—the mitral and tricuspid valves being closed, the aortic and pulmonary valves being open. The first sound is longer and duller than the second, which is sharp and short. The sounds are represented by the syllables *lubb'—tup*. The second sound indicates the closure of the sigmoid valves in the aortic and pulmonary arteries. It marks the end of the systole, and the beginning of the diastole—*i.e.*, the dilatation of the heart—the aortic and pulmonary valves being now closed, the mitral and tricuspid valves beginning to open.



A. Contraction of the Heart (systole), the mitral valve being closed, and the aortic valve open.

Fig. 5.

B. Dilatation of the Heart (diastole), the mitral valve being open, and the aortic valve closed.

The following are the areas at which these sounds (or murmurs in relation to them) are best heard, viz. :—The *mitral area* at the apex beat ; the *tricuspid area* at the foot of the body of the sternum, about the level of the fifth costal cartilage ; the *aortic area* at the second right costal cartilage, at its junction with the sternum ; the *pulmonary area* at the sternal end of the third left costal cartilage and second intercostal space (see Fig. 3). The *left auricular area* lies just outside the pulmonary area, in the second intercostal space. If the auricle be dilated it may extend a little lower. It is about an inch to an inch and a half from the left edge of the sternum.

Murmurs are due to oscillations of the blood in passing through vessels of different calibre, and not due to friction in passing over roughened and diseased valves. They are described as being harsh, grating, soft, and musical.

Any murmur heard *immediately* before the first sound would be auricular-systolic in rhythm ; but clinically—taking the first sound as the starting point—this murmur is called *presystolic*, and it originates at the mitral or (very rarely) at the tricuspid valve. The cause of

the murmur is stenosis of the valve; and it is the only murmur which alone indicates with certainty the presence of valvular disease. Any murmur heard during or through the first sound is *systolic* in rhythm, and its origin may be at any of the four valves. It is caused by mitral or tricuspid regurgitation, and by stenosis at the aortic or (rarely) at the pulmonary valve. A murmur heard immediately after or replacing the second sound is *diastolic* in rhythm, and it originates at the aortic or (very rarely) at the pulmonary valve. The cause is aortic incompetence or, possibly, the rare condition of pulmonary incompetence. The *mitral diastolic* or post-diastolic murmur is not synchronous with the second sound (aortic or pulmonary diastolic) nor *immediately* preceding the first sound (presystolic), but it is heard between these periods, during the filling of the auricles. Its cause, according to Balfour, is the increased tension within the pulmonary system in mitral stenosis, forcing the blood to the left auricle during diastole, the musical murmur being possibly due sometimes to tension of the mitral valve. The mitral diastolic is heard at the apex, or at the left auricular area.

In auscultation of the valves of the heart, the object is to ascertain the character of the heart-sounds, and whether murmurs be present or not. To do so, we must know where the valves lie nearest the surface, when superficial; and when not, the nearest point to which the blood current will carry the sound. To differentiate the murmurs and to locate the valves from which they originate, the seat of maximum intensity and the direction of propagation must be found. A *method* of auscultation is given further on, and the anatomical details and diagrams already given indicate the heart areas and direction of propagation of different murmurs. (See Fig. 3.)

Taking the diseases of the valves separately, the physical signs of each—in addition to what has been already noticed in connection with valvular disease generally—may be thus shortly stated:—

Mitral Stenosis.—The murmur characteristic of this form of valvular disease is generally *presystolic;* but there may be also a diastolic (mitral) present, and it is sometimes combined with the presystolic murmur. If there be regurgitation through the stenosed valve, a *systolic* murmur may also be heard ("double mitral" murmur). The presystolic is best heard at the apex; the mitral diastolic is usually loudest at the left auricular area. The first sound is peculiarly altered and "thumping" in character. Sometimes no murmur is heard, or some exertion requires to be made by the patient in order to develop it. Very frequently there is accentuation of the second sound in the pulmonary area, caused by the increased tension in the pulmonary vessels; and often there is reduplication of the second sound, due to the same cause. The apex beat is in its normal position, and a second impulse or undulation may generally be noticed just above the fourth left rib. A presystolic *thrill* (*frémissement cataire*) may be felt upon placing the hand over the precordial region. Dulness is increased in the transverse diameter. The pulse is small and low in tension, and in extreme stenosis it may be very irregular.

Tricuspid stenosis is a very rare condition, but when present, the presystolic murmur would be loudest in the tricuspid area, and the superficial veins would be dilated. It is almost always congenital.

Mitral Regurgitation.—The murmur is systolic in rhythm, occurring with the first sound and heard in the mitral area over the apex beat. It is propagated towards the left axillary line, and, if loud, may be heard at the angle of the left scapula. Sometimes it is best heard at the left auricular area. It is generally blowing or soft in character. Frequently *no* murmur can be heard. The heart is irregular in its action, and the systole may require to be timed with a finger upon the carotid artery. There may be accentuation and reduplication of the second sound. Inspection may reveal a diffuse impulse; and a systolic *thrill* may sometimes be felt. Percussion may show that the heart is enlarged, chiefly in the transverse direction. In the later stages when the right heart becomes dilated, the apex beat is pushed back and may not be felt. There is distension of the veins of the neck, epigastric and sometimes hepatic pulsation, with other signs of tricuspid regurgitation. The pulse is irregular, small, and rapid. The other signs, as enlarged liver, dropsy, albuminuria, &c., have been described with the general symptoms and signs of valvular disease.

Tricuspid regurgitation may be due to congenital conditions, but it is usually a result of dilatation of the right ventricle, and caused by valvular lesions of the left side of the heart; or by obstructions in the pulmonary system, as chronic bronchitis and emphysema. The murmur is loudest in the tricuspid area, and it is propagated over the liver, and systolic in rhythm. The pulmonary second sound is generally weak, except when there is co-existing mitral disease. The apex of the heart is pushed back by the dilated right ventricle, but a diffuse impulse is seen, and felt, extending upwards and to the right. There is also epigastric and sometimes hepatic pulsation, and true venous pulsation at the neck. By compressing the jugular vein about the middle the following should be made out:—If the venous pulsation continue above the finger it is due to the carotid pulsation, if below, it is due to the heart movements, and on emptying the vein by pressure from below upwards it will refill (regurgitation), and the pulsation continues when the patient holds his breath. There will be no true venous pulsation unless the regurgitation is sufficient to dilate the jugular veins so as to render their valves incompetent. Percussion reveals dulness in the transverse diameter and to the right of the sternum. Venous stasis, with dropsy, albuminuria, &c., occurs early.

Aortic Stenosis.—The murmur is systolic in rhythm, and is best heard at the junction of the second right costal cartilage with the sternum, and propagated up the carotid artery. It is rasping in character, and it is often combined with the diastolic ("see-saw" murmur). The apex beat is lower and nearer the mammary line, percussion revealing enlargement of the heart in its vertical diameter. The pulse is very small, slow, and regular. The condition of

stenosis seldom exists alone, but is almost always associated with incompetence, although the latter may be slight, and the diastolic murmur more difficult to hear.

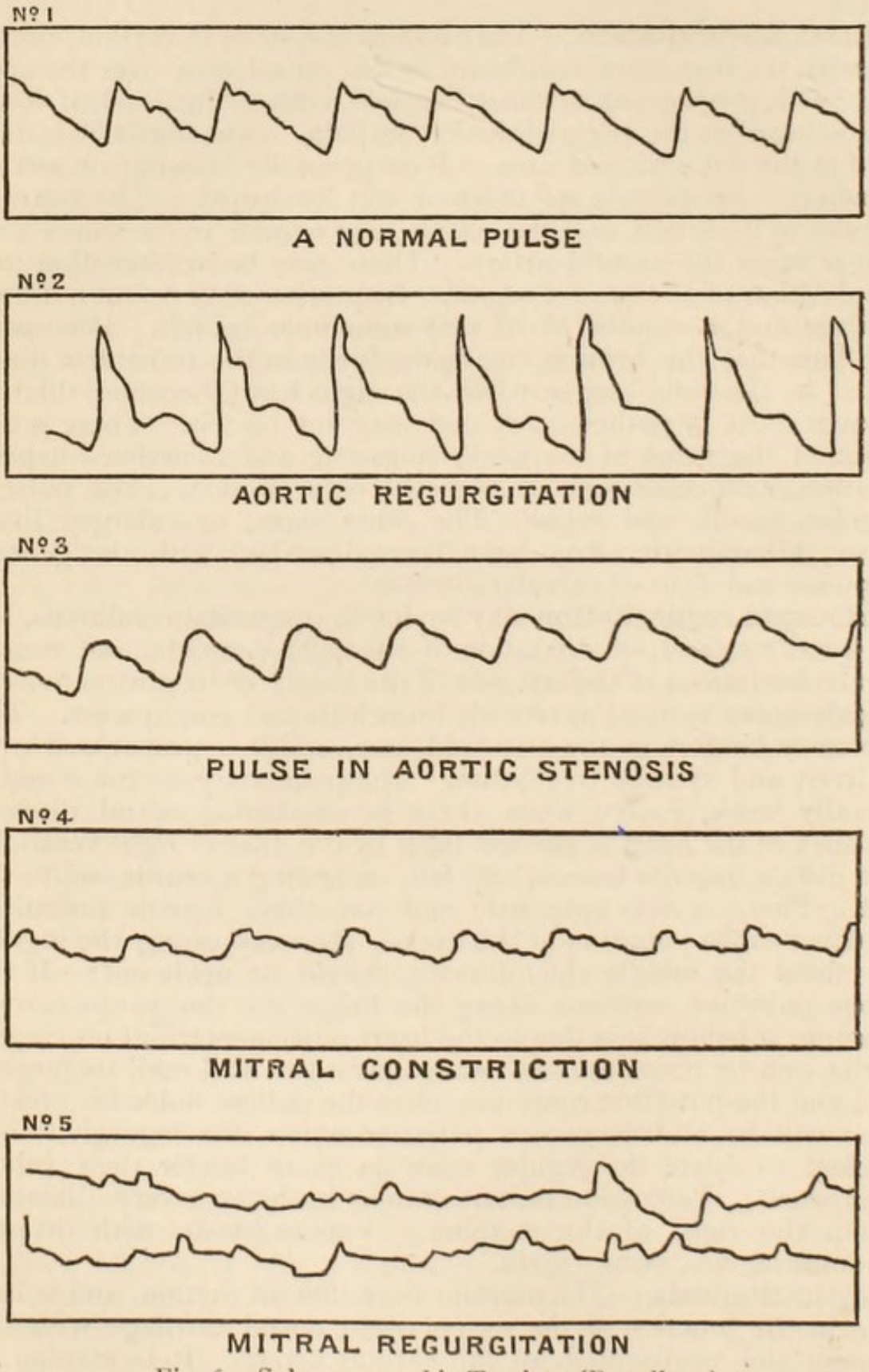


Fig. 6.—Sphygmographic Tracings (Broadbent).

Aortic Incompetence.—The diastolic murmur which characterises this form of valvular disease is heard at the aortic area, or down the left edge of the sternum as low as the ensiform cartilage, and

it is sometimes heard at the apex. If the incompetence be great it is also heard in the carotid artery when pressure is made with the stethoscope upon it, and then it is accompanied by a systolic murmur, artificially produced by the pressure. The apex beat may be as low as the sixth or seventh left interspace, and it is forcible if the heart be much hypertrophied. Percussion reveals enlargement, chiefly in the vertical line. Sometimes a diastolic thrill is felt. The pulse (known as the "water-hammer" or Corrigan's pulse) rises to the finger sharply, and as suddenly collapses. It is more markedly jerky on raising the arm, and it may be felt and seen to pulsate in the smaller arteries if the incompetence be great.

The two conditions—aortic stenosis and incompetence—almost invariably exist together, but often they differ in degree. A "see-saw" murmur at the aortic area is not always heard, although stenosis and incompetence be present. If the stenosis be greater than the incompetence, the systolic murmur is loud in the aortic area because directly conducted to the stethoscope, and it is heard well in the carotid artery because propagated in that direction, while it becomes

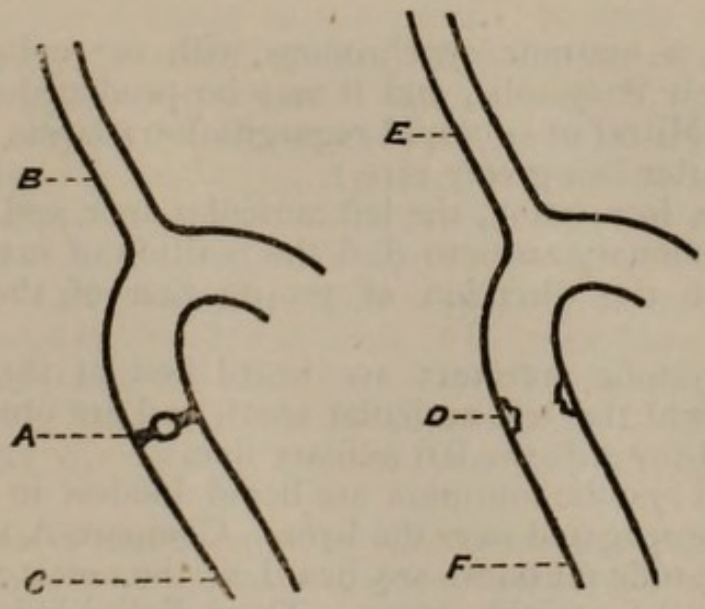


Fig. 7.—A represents a calcified aortic valve, at which there is great obstruction, but trifling incompetence. In such a case there is a loud systolic murmur at A; a systolic murmur, but no diastolic, at B; and at C a slight diastolic may be heard. D represents the aortic valve almost gone, and there is no obstruction, but great incompetence. There is a diastolic murmur usually heard at D, but no systolic; at E there is both a systolic and a diastolic: and at F there is a loud diastolic murmur (see Text).

diminished in its intensity as the stethoscope is carried down the left edge of the sternum, where the feeble diastolic murmur may now be heard. If the incompetence be greater than the stenosis no systolic murmur may be heard in the aortic area, but the diastolic may be heard and propagated loudly down the left edge of the sternum and to the ensiform cartilage. It is also heard in the carotid artery, along with a systolic, on pressure being made upon it with the stethoscope —if the incompetence be great (Fig. 7). It is well to note that the absence of the second sound does not indicate that the valves

are entirely gone, but simply that there may be diminished elasticity, and hence no accentuated sound. The secondary effects in the later stages, when the mitral valve becomes incompetent, have already been described.

Pulmonary stenosis and incompetence are extremely rare, and then generally congenital. If murmurs exist, they are best heard in the pulmonary area, and if a diastolic murmur be present it may be propagated, like the aortic diastolic, down the left edge of the sternum, but it would be accompanied by signs of dilatation of the right ventricle, with tricuspid incompetence, &c.—the signs of aortic disease being absent.

Method of Auscultation of the Valves.* — Place the stethoscope on the mitral area, and concentrate the attention upon the *first* sound of the heart, with a finger upon the carotid artery if necessary.

(1) Is there a murmur *immediately* preceding the first sound? If so, it is presystolic, and it may originate at the mitral or tricuspid valve. Compare the mitral and tricuspid areas for the maximum intensity. (Mitral or tricuspid stenosis, the latter being extremely rare.)

(2) Is there a murmur synchronous with or replacing the first sound? If so, it is systolic, and it may be produced at any of the four valves. (Mitral or tricuspid regurgitation; aortic or pulmonary stenosis, the latter being very rare.)

(3) Examine, in rotation, the left auricular area, and the tricuspid, aortic, and pulmonary areas to find the position of maximum intensity, and trace the direction of propagation of the murmur or murmurs.

A. Mitral systolic murmurs are heard best at the mitral area (sometimes also at the left auricular area), and are propagated from the apex round towards the left axillary line.

B. Tricuspid systolic murmurs are heard loudest in the tricuspid area, and are propagated over the liver. Compare A with B.

C. Aortic systolic murmurs are heard at the aortic area, and are propagated up the carotid artery. They diminish in intensity as the stethoscope is carried from the aortic area, inch by inch, down the left edge of the sternum, curving towards the apex of the heart, where the diminishing aortic systolic murmur may be found, by the pitch and character of the sound, to be the same originally heard at the mitral area. If not, compare C with A, as both may be present. Also, while carrying the stethoscope down the sternum, if the attention be transferred to the *second* sound for a moment, a feeble diastolic murmur may be heard, as *some* incompetence almost invariably exists with the stenosis.

D. Pulmonary systolic murmurs, seldom due to valvular disease, are best heard in the pulmonary area, and are not propagated in any definite direction.

* This method only relates to the systematic examination of the murmurs, and the result of the examination should be considered along with the signs and symptoms already described.

(4) Place the stethoscope on the second right costal cartilage at its junction with the sternum, and carry it down, inch by inch, the left edge of the sternum, concentrating the attention now upon the *second* sound. Is there a murmur synchronous with or replacing the second sound? If so, it is diastolic, and it may originate at the aortic or pulmonary valve. (Aortic or pulmonary incompetence, the latter being very rare.)

A. Aortic diastolic murmurs may not be heard in the aortic area, but lower down the sternum and at the apex of the heart. If the incompetence be great, they are heard in the carotids by pressure being made upon the artery with the stethoscope, and then accompanied by a systolic. The "see-saw" murmur (aortic stenosis and incompetence) is often heard at the aortic area.

B. Pulmonary diastolic murmurs should be heard best in the pulmonary area and over the right ventricle. Compare the pulmonary and aortic areas; and note any accentuation or reduplication of the second sound.

(5) Place the stethoscope again upon the mitral area, but concentrate the attention upon the interval between the second sound and the first—*i.e.*, the diastole of the heart. Is there a murmur, not with or obscuring the second sound, but following it, and not *immediately* preceding the first sound? If so, it is the "mitral diastolic" murmur. It may be louder in the left auricular area, and it may occur along with, or run up to, a presystolic murmur, as it is most common in mitral stenosis. Compare it with the aortic and pulmonary areas, and note the absence of other signs of aortic disease.

Diagnosis.—The valvular diseases having already been placed before the reader in synoptical form, it is unnecessary to repeat the symptoms with the object of further contrasting them. An endeavour should always be made to trace the disease to its pathological source, and to do this the physical examination and history are most important.

The student is apt to consider the presence of a murmur *alone*, as pathognomonic of valvular disease, and to take its loudness as indicative of its seriousness; but this is not so, and the *presystolic* murmur is the only one which alone is a certain sign of valvular disease (mitral stenosis). Alteration in the size of the heart, as revealed by palpation and percussion, is just as important an indication as the presence of murmurs; but in giving an opinion of so grave a disease, no source of information should be neglected, and hence the history of the case, antecedent illness—especially of acute rheumatism—together with the symptoms and physical signs, all require to be carefully considered.

A question which should always be kept prominently in view, is whether the disease is *organic* or *functional*. The diagnosis of valvular disease is sometimes very easy; but when the symptoms of heart failure are present, and the physical signs, including murmurs, have all been made out, it has often still to be decided whether the disease is valvular in origin or due to simple anæmia or dilatation, the result of relaxation and debility. Dyspnœa, pallor, dropsy, &c.,

are symptoms common to these affections; and there may be a combination of these present. The diagnosis is then difficult, and a very cautious opinion should be given; for although all or many of the signs of grave disease of the heart seem to be present, proper treatment may result in a complete cure, should the disease prove to be simple dilatation or anæmia. In connection with the latter disease, hæmic murmurs are generally systolic, and they are often heard loudest in the left auricular area. They are not propagated like the murmurs of valvular origin, and the age, sex, and history may help one to an accurate diagnosis. In simple dilatation, the flapping character of the first sound, and the history of the case with the presence of a *cause*, are the most important indications. It should be remembered that murmurs vary in intensity, and that they sometimes vanish altogether, especially those originating at the mitral valve. A basic systolic murmur is sometimes produced by excited action of the heart under medical examination, and it is probably due to a narrow aorta. In phthisical consolidations, the pulmonary artery, or subclavian, may be involved, and murmurs may be heard over these vessels—the subclavian murmur being sometimes present in the healthy. Pulmonary murmurs in phthisical patients may also be due to the congenital alterations sometimes present in these cases.

With regard to exocardial murmurs they are more superficial, and they are not associated with any valve nor propagated in any definite direction, like the endocardial murmurs. It is better to first consider all murmurs endocardial, and then exclude them.

Prominence of the chest wall may be due to enlargement of the heart, aneurism, or tumours. Increased dulness upon percussion over the precordial region may be due to these causes, and also to dilatation of the heart; or diseases in which there is retraction of the lung may leave more of the heart uncovered (see *Aneurism and Mediastinal Tumours*, p. 47).

The **prognosis** in all the chronic valvular lesions is grave, as regards both the health and duration of life. It must be estimated in relation to the circumstances of the patient, the nature of the lesion, the signs and symptoms to which the lesion gives rise, and to the effects of the treatment.

The regurgitant diseases are more serious than the obstructive, the order of importance being as follows, viz. :—Aortic incompetence, very grave, as sudden death may take place from dilatation of the ventricle; mitral regurgitation, less grave, but death may also take place suddenly, although it is far more usual by the slow development of the secondary effects leading to dropsy, &c.; mitral stenosis comes next; and aortic obstruction is the least important. When unfavourable symptoms manifest themselves in aortic incompetence, the danger is great, and the end may take place—if not suddenly—in about three months (Balfour). In mitral regurgitation, as the attacks become more frequent, and the symptoms and signs disappear more slowly under treatment, the prognosis becomes more and more serious, and death may take place from failure of the

heart or from a secondary complication. The loudness of a murmur is no test of the extent of a valvular disease; but a "see-saw" murmur at the aortic area, in which the diastolic is louder than the systolic, is more serious than the reverse. It is far more important to consider the prognosis in relation to the probable state of the *fibres* of the heart, as revealed by the persistency of the physical signs and symptoms, and the presence of secondary complications.

The **treatment** of the chronic valvular diseases. When the lesion is "mute" no direct treatment, and certainly no digitalis, is required Beyond advising the patient to throw no stress upon the heart by climbing or running, &c., to avoid excitement, and to take all the rest that he can, no treatment is necessary. When the heart begins to dilate, or the compensation to fail, then digitalis is indicated. To avoid repetition a short synopsis of the physiological action and uses of digitalis is here given.

Digitalis has (1) a direct action on the heart muscle, (2) it stimulates the cardio-inhibitory fibres of the vagus and thus holds the heart in check, and (3) it stimulates the vaso-motor centre and thus raises the arterial tension. The direct action upon the heart may be *tonic*, or it may be that the coronary arteries are better filled and the heart thus better nourished, by the increased tension in the smaller vessels. The stimulation of the vagus renders the beats of the heart *slower*, and stimulation of the vaso-motor centre by increasing the tension, induces the heart to act more *forcibly* to overcome the increased resistance. When digitalis is long continued, the pneumogastric may lose its control and the heart may become rapid and feeble. The appetite and stomach may become disordered. Patients under the full influence of the drug should be kept *at rest*, as exertion may produce syncope and other alarming symptoms. A high temperature seems to lessen the power of digitalis over the cardio-inhibitory centre, and in these cases its action should be carefully watched. It is not considered to be a true "cumulative" poison, but after prolonged use, if there should be defective elimination, alarming symptoms may suddenly occur. Digitalis should not be used in chronic Bright's disease.

Digitalis is used in palpitation with irregular action of the heart, whether due to valvular disease or not. Intermittence of the pulse is always an indication for the use of digitalis, but it must never be used when fatty degeneration of the heart fibres is suspected. It is indicated in dilatation of the heart. In mitral stenosis, by the prolongation of the diastole, it allows time for the better filling of the ventricle through the stenosed valve. In mitral regurgitation, with feeble and irregular action of the heart, the lungs becoming engorged and lividity with dropsy setting in, it is most strongly indicated. The increased tension in the blood-vessels produced by digitalis lessens the transudation and promotes the absorption of the serum in dropsical cases. In aortic stenosis it should only be used in the later stages when the heart is dilated and feeble; but when hypertrophy exists, or the vascular system is atheromatous, it may be dangerous. In aortic regurgitation it is of great value when the

compensatory hypertrophy begins to fail. It is also used in bronchitis with a dilated heart; and it acts as an indirect diuretic when the kidneys are healthy. Digitalis has also some other uses, but these need not be considered here in relation to the valvular diseases and their effects. The doses of digitalis are five to ten, and even thirty minims sometimes, of the tincture; two to four fluid drachms (or more) of the infusion; one-fiftieth to one-twentieth of a grain of digitalinum, hypodermically. Bartholow states that the English or German wild plant of the second year's growth is the most active digitalis.

The drugs of a similar character which may be used when digitalis is not suitable, are the tinctures of cimicifuga or convallaria—the dose of each being five to ten minims, given in water three times a day. The tincture of strophanthus has lately acquired a great reputation in the treatment of heart disease. It differs in one respect, at least, from digitalis, in not raising the tension within the blood-vessels. The dose is three to ten minims thrice daily. The patient while under digitalis treatment should be kept in the recumbent position if taking full doses—rest being a very important factor in the treatment of heart disease. It is advisable to suspend the use of digitalis occasionally for a day or two. *Tonics* may be given, as arsenic and iron (℞ 1), quinine and mineral acids (as ℞ 2), Easton's syrup, &c., when the patient is fairly well and able to take moderate exercise. Tonics may be given early with the digitalis—either separately or together, in the form of a pill (as ℞ 3).

The diet should be at all times nutritious and easily digestible, care being taken not to overload the stomach. Small doses of whisky may be necessary; but strong tea, coffee, and tobacco are to be avoided.

The treatment of secondary congestion, œdema, and infarctions of the lung with pneumonia, bronchitis, &c., are described in diseases of the respiratory system. Frequently in these cases the digitalis is prescribed with carbonate of ammonia and other expectorants, as senega and squills (℞ 4). Poultices or turpentine stupes are also necessary; and wet or dry cupping of the chest is sometimes practised to relieve the symptoms. Hæmoptysis may be treated by turpentine in capsules, ergot, or by strong doses of acids and morphia. Rest, ice to suck, and poultices on the chest to relieve the internal congestion will generally suffice. Congestion of the stomach and liver are best treated with occasional doses of podophyllin, or by iridin, euonymin, or bluepill, followed in a few hours by a saline purge. Pepsin with a mineral acid may be necessary for the stomach—with attention to the diet. The kidneys may be occasionally flushed with draughts of cream of tartar in water—a drachm or more for a dose—in those cases where there is a tendency to œdema; but if the dropsy be great in quantity, the compound powder of jalap, forty grains or more, and combined with a sixteenth to a half grain of elaterium if necessary, should be used regularly twice or thrice a week. The dropsy may also be treated by occasional vapour baths and by the use of diaphoretics. When the legs and

scrotum become extremely tense it may be necessary to puncture them to relieve the pain, but to do this with a sharp tenotomy knife is much less likely to produce erysipelas than by using Southey's tubes. *Paracentesis abdominis* may be performed when the ascitic fluid embarrasses the breathing of the patient ; or when existing in large quantity, it hinders the process of absorption. A pill containing digitalis, mercury, and squills (℞5) is useful in heart disease with dropsy. Diuretin—fifteen to thirty grains thrice daily—has proved to be a useful drug, and a powerful diuretic in dropsical conditions. Pain and severe dyspnœa are best treated by the hypodermic injection of morphia—one-twelfth to one-fourth of a grain. This may also be used for sleeplessness. The inhalation of nitrite of amyl or chloroform may be necessary in the severe forms of angina pectoris. Arsenic should be given in the chronic forms. A mixture of ether and morphia (℞ 6) is useful to relieve breathlessness. Convulsions should be treated by bromides and rest.

Dilatation, Hypertrophy, and Fatty Disease of the Heart.—Dilatation of the heart has already been considered in relation to valvular disease as a cause, but it exists also as a disease *per se*. The walls, especially of the auricles and right ventricle, become thinned, and there may be fatty degeneration of the muscular tissue. The valves are also thinned, and the orifices may be unable to close.

Dilatation may be caused by over-exertion in feeble persons, or it may be the result of myocarditis associated with antecedent endocarditis and pericarditis. Fatty degeneration of the fibres of the heart may lead to dilatation, and so may fevers and debilitating diseases. Chronic pulmonary disease (bronchitis, emphysema, pneumonia, &c.), by obstructing the circulation within the pulmonary blood-vessels, is a cause of dilatation of the right side of the heart.

The **symptoms and signs** of dilatation of the heart—whatever the cause—are the same as when the dilatation is the result of valvular disease. They are all caused by the failure of the heart to maintain the circulation, and hence the congestion of the lungs, liver, stomach, kidneys, &c., and the other changes leading to dropsy and death. Diffuse impulses may be seen, and felt, over the precordial area corresponding to the dilated cavities. The apex beat is lower and its impulse feeble. Percussion reveals enlargement *transversely* if the right side of the heart, and *vertically* if the left side, be dilated. Some hypertrophy may exist with the dilatation, especially when the left ventricle is affected. The first sound of the heart is feeble and flapping in character (lap′ tup, instead of lubb′ tup) and if the dilatation be sufficient to prevent the closure of the valves, soft blowing systolic murmurs may be heard at the mitral or tricuspid areas. The pulse is irregular, feeble, and quick. (See *Diagnosis of Chronic Valvular Disease*, p. 29).

Hypertrophy of the heart has also been referred to as a compensatory change in valvular disease. It may exist, however, without valvular disease being the cause, and it consists of increase of the

muscular fibres of the heart, which are firmer than normal, unless fatty degeneration be also present, a change which may occur in the later stages. One or more of the cavities may be affected, and sometimes the heart may be enormous in size (*cor bovinum* or ox's heart). The causes of hypertrophy are over-action of the heart muscle, whether due to laborious occupations or to the abuse of such stimulants as coffee, tea, and alcohol, or by excessive smoking. Bright's disease and aneurism, as well as the chronic valvular diseases, are also causes of hypertrophy. The first group of causes gives rise to *general* hypertrophy.

The **symptoms and signs** of excessive hypertrophy are palpitation and an uneasy or oppressive feeling in the chest, with headache, dizziness, and ringing in the ears—all being due to the increased action of the heart. Bleeding at the nose is a common symptom. The chest wall may be prominent, especially in young patients, and the impulse of the heart is seen, and felt, to be markedly *heaving* in character. Percussion reveals enlargement extending in the vertical axis of the heart, but also transversely if the right auricle and ventricle be affected. The heart may extend as low as the sixth, seventh, or eighth intercostal space, and round as far as the left axillary line. The area of absolute dulness is increased when percussion is made with the patient in the erect position, and if the condition of the lungs be normal. The first sound is dull, muffled, or "booming," and the second sound clear and accentuated. The pulse is strong, slow, full, and bounding.

Hypertrophy is to be distinguished from dilatation of the heart, pericardial effusion, and tumours of the mediastinum—the latter displacing the heart.

Dilatation and *hypertrophy* are chronic diseases, the dilatation being the more serious affection, liable to return, and ultimately ending in dropsy and death unless the cause be removable. Hypertrophy, even when excessive, may exist for years; but if the arteries become atheromatous there is great danger of cerebral hæmorrhage.

The **prognosis**, therefore, in both diseases is grave, and it should be guarded; but many cases of dilatation are curable, and this depends upon whether the cause be removable or not.

The **treatment** of *dilatation* consists of rest, digitalis, and iron tonics, as already indicated in the treatment when it is the result of chronic valvular disease. *Hypertrophy* may in itself be curative when associated with the dilatation; but when excessive, then rest, purgatives to lower the blood pressure, and tincture of aconite (three or four minims in water, thrice daily) become necessary. The aconite may be given in drop doses, frequently repeated. A low diet is indicated in hypertrophy, and iodide of potassium (ten or twenty grains thrice daily) is also useful.

Fatty disease of the heart may co-exist with, or be the result of, dilatation and hypertrophy, and the chronic valvular lesions. It also arises as an independent disease which has dilatation as a secondary effect.

Fatty *degeneration* means a replacement or change in the struc-

ture of the fibres; while fatty *deposit* implies a displacement with atrophy of the cardiac muscle by pressure. In the latter case the whole heart may be enveloped by a dense layer of fat. In fatty degeneration the disease may be general or may exist in patches, the muscular substance being pale-yellow, or speckled, and easily torn. The speckled appearance is best seen in the papillary muscles and trabeculæ. The early microscopical changes consist of cloudy swelling and granular deposit within the fibre cells. Ultimately the deposit is replaced by oil globules, and the striæ disappear.

The causes of fatty degeneration are pericarditis and myocarditis, atheroma of the coronary arteries, and prolonged anæmia; wasting diseases, fevers, and certain poisons as phosphorus, &c.; and the chronic valvular diseases with dilatation and hypertrophy.

The **symptoms** are due to the weak action of the heart, the most significant being attacks of angina pectoris, Cheyne-Stoke's breathing, and a weak, slow, intermittent pulse. The apical impulse is feeble or absent, and there is increased dulness if the heart be dilated, and perhaps also a systolic murmur. The heart sounds may be reduplicated or almost inaudible. A waxy-looking skin may sometimes be observed in cases of fatty disease. The other physical signs are due to the dilatation of the heart, viz. :—congestion of the lungs, liver, stomach, kidneys, &c., terminating in dropsy and death.

The **diagnosis**, in most cases, can only be surmised; and often it is only by the summation of the probabilities, with the presence of one or more of the causes, that fatty degeneration of the heart is suspected.

The **prognosis**, of course, is very serious, as cardiac failure or rupture may take place at any moment.

The **treatment** consists of good nourishment, rest, and tonics—as Easton's syrup, iron, and arsenic, &c. Digitalis should not be used.

Endocarditis and Pericarditis.—Endocarditis may be acute, chronic, or ulcerative. The chronic form has already been described along with the *valvular* diseases of the heart, and hence only the acute and ulcerative forms remain to be considered.

The *acute* form of endocarditis begins with hyperæmia of the endocardium. The cells proliferate and become cloudy, and soon minute capillary extravasations take place into the membrane, which loses its smooth character and becomes rough and opaque from inflammatory exudation. The fibrinous deposit upon the roughened valves leads to the formation of vegetations. If not excessive this condition may be recovered from by the absorption of these inflammatory products, but more usually it passes into the chronic form (see *Chronic Valvular Disease*, p. 18). It is almost always the mitral and aortic valves which become affected; the tricuspid and pulmonary valves are only very seldom the seat of endocarditis, and then, generally, along with the valves of the *left* side of the heart.

The **causes** are rheumatism and other acute fevers. Endocarditis frequently accompanies pericarditis and myocarditis; and it may occur, occasionally, as a secondary change in cases of pleurisy and

pneumonia. It may (*rarely*) be primary or idiopathic, but in this respect it should be noted that frequently the valvular disease precedes the pain and swelling of the joints in acute rheumatic fever. Bright's disease is also a cause of endocarditis.

The **symptoms** of acute endocarditis are fever, with nausea, vomiting, and general *malaise.* When endocarditis occurs with rheumatism, or other fevers, the patient complains of uneasiness at the heart, or palpitation, and there may be exacerbation of the temperature. The pulse is more rapid, and the general condition worse. There may, however, be no new symptoms, and, as the physical signs are distinctive, they should be carefully looked for in every case. The physical signs consist of the sudden development of these murmurs which have been described in chronic valvular disease of the heart; but it should be carefully noted that the presence of a soft-blowing systolic murmur at the apex does not necessarily mean endocarditis. It is not until dilatation of the heart takes place, with increase of the cardiac dulness, and engorgement of the pulmonary vessels with accentuation of the second pulmonary sound, that the diagnosis can be safely made. If the endocarditis be slight, the patient may entirely recover; but it is more frequent for it to become chronic. The duration of the acute attack is short; and the prognosis is not generally dangerous to life, but it must be guarded as regards the future health.

The **treatment** of acute endocarditis depends upon the causal disease (see *Rheumatism*). Absolute *rest* is necessary, and iodide of potassium is useful to reduce the tension within the blood-vessels. Counter-irritation over the precordial region with strong iodine liniment, is useful. Digitalis should not be given unless the heart be very rapid and feeble. The diet should be non-stimulating.

Ulcerative Endocarditis.—The **pathological changes** are at first similar to those of acute endocarditis, but the soft inflammatory swelling increases, and then breaks down, leaving ragged ulcers, which become infiltrated with micrococci. The valves are broken down, and infective emboli pass into the circulation, producing infarctions in the brain, lung, spleen, kidneys, &c.

The commonest **causes** are diphtheria and puerperal fever, but it may occur during the course of acute rheumatism, or of chronic valvular disease. Bad hygienic conditions seem to favour its development in weakly persons. It is sometimes called *diphtheritic endocarditis.*

The **symptoms** begin with severe rigors, or a "chill," with headache, nausea, and vomiting. The tongue is dry and furred, and the bowels may be constipated. Sometimes there is diarrhœa. There is great prostration, with delirium, stupor, and ultimately coma, the stools and urine being passed in bed. The spleen is enlarged, and jaundice may be present. Irregular exacerbations of the temperature, with increase in the rapidity of the pulse, and severe sweating, indicate pyæmic infection. Pyæmic infarctions within the lung give rise to dyspnœa, and frequently they light up pneumonia with its physical signs. Infarctions of the brain cause hemiplegia

and convulsions; of the kidney, hæmaturia and albuminuria. Painless abscesses form rapidly in the joints. A loud systolic murmur is heard at the apex and base of the heart. The case may terminate fatally in less than a week, or it may run on for three or four weeks.

The **diagnosis** is not difficult when it occurs as a complication of acute rheumatism, but otherwise, it is extremely like typhoid fever, and it can only be differentiated by the history and the physical signs.

In the **treatment**, salicylic acid or benzoate of ammonia may be given, along with stimulants and strong nourishment. The cases of ulcerative endocarditis are very hopeless.

Pericarditis may be acute or chronic. The initial changes are the same as in other serous inflammations. The exudation begins at the base of the heart about the large blood-vessels. It may be tough, but sometimes it is soft, and it may affect a small area or be diffused over the whole pericardium. The lymph may present a honeycombed appearance, arranged in concentric rings which may be stripped off. The effusion is usually serous, but it may be a mixture of blood and serum, and only very rarely is it purulent. The quantity may be from three to eight ounces, but sometimes it is as much as three pints. Gas may be present in severe cases attended with decomposition. Complete absorption may take place; or the lymph may, ultimately, cause adhesion of the visceral and parietal layers of the pericardium. These changes may terminate in calcification; or an effusion may remain and give rise to the chronic condition.

The **causes** are chiefly Bright's disease, rheumatism, gout, and fevers. It may occur as the result of injury, or by the extension to the pericardium of other diseases—as pleurisy, pneumonia, chronic cardiac disease, aneurism, &c.; or by irritation produced by abscesses, caries of ribs, tumours, &c. It is doubtful if it ever occurs as an idiopathic disease, although, sometimes, it may appear to be so, and it may precede, by a few days, the joint symptoms in acute rheumatism.

The **symptoms** are sometimes not very evident, especially when the pericarditis is associated with other diseases which cause it. There is frequently pain or uneasiness over the precordial region, and rapid action of the heart. There may be slight rigors in the apparently idiopathic form of the disease, or when the pericarditis arises in connection with non-febrile causal affections. When effusion takes place, and especially if large in quantity and suddenly, there is syncope, severe dyspnœa, and cough—the face being pale and anxious. The pulse is small, rapid, and in extreme cases often very irregular. Sometimes there is delirium and other cerebral symptoms, but these are due rather to the primary or other co-existing disease. Death may result from syncope, or by interference with the respiration—the lungs becoming œdematous, and dropsy setting in.

The earliest physical sign is *friction* at the base of the heart, which may, however, be absent if the exudation be soft. It is superficial

in character, and it is not synchronous with the valvular sounds; and it is sometimes increased by pressure with the stethoscope. After effusion of serum, if the quantity be large, the heart sounds are feeble and distant. There is sometimes an undulatory impulse to be observed, and the apex beat may appear to be higher. The diaphragm is sometimes pushed down, and the epigastric region may appear prominent. The chest wall, especially in young persons, may bulge out, and the intercostal spaces become widened from the second rib to the level of the sixth or seventh. The apex beat is very feeble, if felt at all. The dulness is much increased, and when mapped out upon the anterior wall of the chest, the area of dulness assumes a pyramidal form, the apex being upwards. The dulness begins at the base of the heart, and it may extend to the clavicle, and sometimes even above it, but it seldom extends lower than the sixth rib, except in extreme cases. Transversely, it may extend from the right border of the sternum to beyond the left mammary line, and changes of posture alter its limits. The effusion may affect the lungs by pressure, and the physical signs of consolidation, due to collapse of the lung, may be made out, generally at the base of the left. Over the cardiac area the breath-sounds are distant, and the vocal resonance and fremitus are diminished. After absorption of the fluid the friction rub may return, and it may be sometimes felt (*friction fremitus*) now, or in the early stages.

Acute pericarditis may terminate by complete recovery in about three weeks; or in sudden death by syncope; or it may become more or less chronic in character, with adhesions to the chest wall. The chief signs of adhesion are depression of several intercostal spaces over the cardiac area during the heart's systole, and the fixed position of the apex-beat when the patient is turned over upon the left side. The obliterated pericardial sac may become calcified, and secondary changes—as dilatation and hypertrophy of the heart, with fatty degeneration—may ultimately result.

The **prognosis** must, therefore, be guarded. Pericarditis is a grave symptom in Bright's disease, generally ushering in a fatal termination. It is not so serious in acute rheumatism; but when the effusion is great in quantity, whatever be the cause, the patient is in great danger.

The **diagnosis** depends chiefly upon the physical signs, and, therefore, in all cases where pericarditis is known to be a possible complication, the chest should be carefully and frequently examined. Pericarditis and endocarditis require to be distinguished from each other, and both diseases are frequently present, as in acute rheumatism. Precordial pain indicates pericarditis, but it may be pleuritic in origin. An inflammatory pericardial effusion is to be differentiated from cardiac dilatation and hypertrophy, and from dropsy of the pericardium (hydropericardium). A pleuritic effusion of the left side may extend over the precordial region: and consolidation of the left lung may also simulate a pericardial effusion.

The **treatment** of acute pericarditis consists of keeping the patient entirely at rest; giving opiates or chloral for pain, or to

calm the excited action of the heart; and continuing to treat the primary disease which is its cause. Opiates should not be given in Bright's disease. In the early stage, twenty grains of quinine may be given, and three or four leeches may be applied over the precordial and epigastric regions. Hot linseed poultices should then be continuously used. After exudation, or even earlier, strong iodine liniment should be painted over the precordia, and the chest thereafter protected with a layer of wadding or warm flannel. Should the breathing become embarrassed, *paracentesis* of the pericardium is indicated. The chest should be thoroughly washed with carbolic lotion or other antiseptic, and the apparatus thoroughly cleansed by running strong (one in twenty) carbolic lotion through it, the aspirating needle being allowed to lie in carbolic lotion for ten minutes before using. A small incision is then made through the skin in the fifth left intercostal space, one inch from the left edge of the sternum, and the needle should be introduced slowly and firmly at that point. Only two or three ounces should be removed, unless the fluid be purulent, when more should be drawn off if possible. A preliminary puncture with a thoroughly clean hypodermic needle, may be made. Iodide of potassium with diuretics may be given in the later stages (℞ 7); or steel drops and other tonics (℞ 8). The diet should be light, and fluid nourishment only should be allowed. Brandy may be necessary if there be any tendency to syncope or collapse.

Functional Diseases of the Heart.

Palpitation—Irregularity and Intermittency—Irritable Heart—Syncope—Angina Pectoris.

These morbid conditions have all been referred to as symptoms of grave organic disease of the heart, but they exist also as diseases *per se*, the diagnosis being made by the process of exclusion, after a careful *physical* examination.

Palpitation of the heart has the following additional *causes*, viz.: — nervous exhaustion—the result of anæmia, debilitating diseases, and venereal excesses, &c.; the abuse of tea, alcohol and tobacco; and reflex disturbances from flatulent distention, indigestible food, pregnancy, &c. It occurs also as a symptom in Bright's disease, gout, hysteria, and exophthalmic goitre; and it may be produced by cerebral excitement, prolonged mental work, emotions, &c. The sensations in the neurotic forms may be due to palpitation of the aorta—the abdominal portion usually—the heart being unaffected.

Irregularity and intermittency of the heart's action may also be due to grave organic disease, or they may exist as functional disorders. They indicate either cardiac failure, or an interference with the nervous apparatus of the heart, and they are frequently found along with palpitation as symptoms. In gouty subjects, irregularity may exist for a long period of years without materially affecting the health, and, therefore, in such cases the prognosis is not

so grave. It is well to note that the radial pulse does not *always* correspond with the heart's beat.

Irritable heart is a condition described by Da Costa, characterised by palpitation and irregular heart's action, due to excitement and hard physical work, in individuals who have not had any preliminary training.

The *symptoms* of palpitation, &c., consist of uneasiness, "fluttering" at the heart, and sometimes actual pain. The patient looks anxious and complains of a feeling of insecurity; and there may be occasional fainting fits, with symptoms as described under *syncope*. The pulse is rapid and it may be irregular. The heart-sounds are accentuated and louder than normal, with sometimes reduplication; the impulse seems diffused and in prolonged cases the dulness may be increased in the transverse diameter.

The *treatment* of the foregoing group, apart from cardiac disease, depends entirely upon the cause. Remove all sources of reflex disturbance. During an attack brandy or sal volatile should be given, and mustard may be applied to the precordial region. If due to shock, give a hypodermic injection of morphia—one-fourth of a grain—and repeat the dose in a few hours if necessary. The diet should be light, and four or five hours should intervene between meals. Strong soups, beef-tea, and milk should be ordered, and only limited quantities of solid food allowed. Pepsin and mineral acids may be given when palpitation occurs as a symptom of dyspepsia. Attention to the bowels, and regular exercise, should be encouraged. Digitalis and caffeine are useful remedies; and so also are arsenic and iron when the palpitation is the result of nervous exhaustion or debilitating disease. Bromides should be given when due to cerebral irritation and overwork, and the patient should be warned to avoid all sources of excitement and strong emotions. A belladonna plaster may with benefit be applied to the precordial region.

Syncope.—Fainting may be a symptom of organic disease of the heart, but it also occurs as a functional disorder. The patient becomes pale, faint, and giddy, the breathing is sighing or gasping in character, and there is dimness of vision, ringing in the ears, and mental confusion. In extreme cases there is insensibility more or less complete, the pupils being dilated; and when the patient is unconscious the sphincters may relax and the urine and fæces be passed involuntarily. The pulse is rapid, feeble, and often irregular. The cardiac sounds and impulse are weak, and the attack may terminate in profuse perspiration or vomiting. There may be slight convulsive movements during the attack. Unless due to grave heart disease, or the result of severe hæmorrhage or prolonged wasting diseases, as cancer, phthisis, &c., recovery under proper treatment is the rule; but in some cases there may be a fatal termination. The commonest *causes* of syncope are loss of blood, anæmia, and wasting diseases; heart disease; poisons, as aconite and tobacco; strong emotions in nervous individuals; pain, unpleasant sights, shock; and hot rooms, or warm baths, in weakly patients, &c. It also occurs as a result of pressure of fluids upon the heart,

as in pericardial, pleuritic, or abdominal effusions; or by their too sudden removal by aspiration.

The *treatment* when a fainting attack is threatened is to bend the head forward as far as possible, and to remove any obvious reflex cause. The clothing should be loosened around the neck, and cold applied to the face. Admit fresh air, and give brandy or sal volatile. Should the patient become insensible *keep the head low* and raise the limbs. Ammonia may be inhaled, but care should be taken that it be not strong, as, otherwise, bronchitis and pneumonia may be set up. Friction over the heart, slapping the hands, and rubbing the limbs will help to restore the patient; and in severe cases with complete unconsciousness, stimulating enemata should be given, and artificial respiration should be carried on if the breathing be embarrassed. Ether may be injected hypodermically if there is continued failure of the heart's action; and if the cause be great loss of blood transfusion may be necessary.

Angina pectoris, or breast-pang, is that severe agonising pain in the precordial region, shooting up the neck or down the arm, and which is generally associated with grave organic disease of the heart. The pain is described as burning, boring, or gnawing; and sometimes it is confined to the side of the chest, or it may shoot through to the back. There is no tenderness upon pressure. The attack may consist of only one paroxysm, lasting for some few minutes and leaving suddenly, or sometimes there is a succession of spasms, the patient remaining quite conscious unless syncope supervene. The seizure may terminate in general convulsions. Cold sweat breaks upon the brow, the face is deadly pale, and the expression terribly anxious. The pulse is feeble and often irregular. Angina pectoris occurs chiefly in those who have long suffered from chronic valvular disease of the heart and dilatation; and especially liable are those who suffer from fatty degeneration, atheroma of the arch of the aorta, or calcification of the coronary arteries. The *cause* seems to be of the nature of a spasm or paralysis of the heart, and in many cases there is a rise of blood pressure, which is supposed to be produced by general vaso-motor spasm, the left cavities of the heart being suddenly distended. The exciting causes are—strong emotions, excessive smoking, cold, physical exertion, and reflex causes, as indigestion, &c. The patients suffering from angina pectoris have sometimes warnings of an attack, consisting of slight or indefinite pains in the precordial region. Several forms are described, as the *neuralgic*, where there is no evidence of organic heart disease; *angina sine dolore*, when all the symptoms are present but pain; and that form associated with atheroma of the arch of the aorta is sometimes regarded as distinct. In the latter case the pain radiates from the top of the sternum, and it is apt to be excited by active exercise, walking against a high wind, or by ascending a hill.

Pseudo-angina is a minor form of pain, occurring sometimes in the healthy, and of short duration; it is more frequently a symptom in gouty, anæmic, and hysterical patients.

Diagnosis and Prognosis.—True typical angina pectoris is generally obvious; but the differentiation of the several forms is not so easy, while it is very important to recognise the cause. The *neuralgic* form is the most hopeful, and it occurs in patients of the gouty and nervous temperaments. It can only be diagnosed by excluding the forms due to organic disease of the heart and aorta, and by watching the results of treatment, a cure being possible. The pain produced by pressure of a thoracic aneurism may sometimes be regarded as a *true* angina (see *Aneurism*). The *spurious* forms may be diagnosed by considering the age and sex, and whether the patient be anæmic, hysterical, or suffering from nervous debility. The pain is not so commanding as the true angina; and neither do intercostal neuralgias, pleurisy, pericarditis, nor pleurodynia resemble it in this respect. Cramp and neuralgia of the stomach or œsophagus sometimes resemble angina pectoris very closely; but the history of the case as regards the digestive powers, and whether a previous sufferer from flatulent dyspepsia or not—the seat, mode of onset, and character of the pain, and whether relieved by pressure or not—will generally serve to distinguish these disorders after a careful examination of the heart has been made. Should there be convulsions, the case may resemble an epileptic seizure.

The prognosis, when due to organic disease, is very grave, and especially so when fatty degeneration of the heart is the suspected cause. A cautious prognosis should be given in all the forms. Sudden death during an attack of angina is always possible, and it is not uncommon.

The *treatment*, when called to a severe attack of *true* angina pectoris, is to give (1) nitro-glycerine, either two or three tablets, or four to eight drops of an alcoholic solution (1 per cent.). (2) If not relieved, cautious inhalation of nitrite of amyl may be tried—four to six drops upon a handkerchief. (3) Remove all possible reflex or exciting causes, rub the limbs, apply mustard to the chest, and keep the lower limbs and body warm. Should the nitro-glycerine or nitrite of amyl still fail to relieve, then give (4) an inhalation of chloroform, and while soothed by the vapour, inject, hypodermically, as much as one-half of a grain of morphia. After the attack the patient should take, for a considerable time, arsenic and iron (℞ 1); and the diet should be regulated as in *palpitation* of the heart. Quinine, cod-liver oil, and mineral tonics are useful, and galvanism is also recommended. The pseudo-forms do not require such powerful remedies; but for cramp in the stomach, hot brandy and water, ether, or sal volatile may be given; and morphia should be administered, either by mouth or hypodermically, if the pain be severe. The nitro-glycerine tablets may be carried and used by the patient—two or three daily—when threatened with angina pectoris.

Exophthalmic Goitre, Grave's or Basedow's Disease.—Although sometimes classified with the nervous diseases, exophthalmic goitre is best considered here, as palpitation is generally the first, as it is the most important, symptom calling for treat-

ment. The *pathology* is not clear, but there is found an increase of the size and thickness of the blood-vessels of the thyroid gland, and an increase in the quantity of fat behind the eyeballs. The *cause* is also indefinite, but there is some disturbance of the nervous regulating apparatus of the heart, and it occurs chiefly in anæmic and chlorotic patients. Exophthalmic goitre is generally attributed to moral emotions, fright, &c., and it is more common in women than in men. It develops generally before the age of thirty.

The **symptoms** are severe palpitation, at first in paroxysms, with intervals, but ultimately constant, the heart beats being from 120 to 200 in the minute. A basic murmur is generally present, and there is pulsation of the blood-vessels in the neck. The thyroid becomes enlarged, first one lobe and then general enlargement, and frequently it disappears; or it may be altogether normal throughout. The eyeballs protrude and are staring, and Grave's symptom—a want of co-ordination of the eyelid and eyeball—is an early sign. The patient should be directed to look at his feet, when the upper eyelid will be seen to lag behind the movement of the eyeball, the sclerotic being still perceptible when the movement is completed. Sometimes there is also a spasmodic retraction of the upper eyelids, known as Stellway's sign. Recently, Charcot has described a new symptom of Grave's disease, which is highly important in the diagnosis of doubtful cases. This consists of *rhythmical vibratory tremors* which are very rapid, and usually affect the muscles of the upper and lower extremities. Other secondary nervous symptoms are described. There is pallor, with frequent flushing of the face, throbbing of the head, and sometimes slight fever. The disease runs, occasionally, an acute course of a few months, but more generally it is chronic, and lasts for years, with improvement and exacerbations. Death may ultimately ensue from heart complications or from tubercular disease.

The **treatment** consists of giving iron and digitalis; and the best results are produced by galvanism—the anode being placed under the ear, and the cathode upon the epigastrum. Bromides may be used, and the patient advised to avoid all sources of excitement—a quiet country life being the best, with fresh air, gentle exercise, and good nourishing diet.

The following group of heart diseases need only be shortly noticed:—

Cardiac aneurism is a localised dilatation of the walls of the heart, almost always secondary to structural changes, as fatty degeneration, &c. A pulsating prominence may be observed, and auscultation may reveal the presence of a murmur. Death may take place from rupture.

Malformations consist of incomplete development of ventricular and auricular septa, patent foramen ovale, constrictions of pulmonary artery, &c. The symptoms result from the admixture of venous

and arterial blood—dyspnœa, cough, lung symptoms, and cyanosis, being the most frequent. Pulmonary murmurs may be present. The treatment consists of careful hygienic measures, and iron, &c.

The *new formations* and *degenerations* are fibroid degeneration (cirrhosis), atrophy, calcification, cancer, syphilitic growths, and tubercle; and sometimes parasites are found.

Heart clots are sometimes found as large, loose, black coagula, extending into the pulmonary vessels or venæ cavæ. They occur after severe hæmorrhage and septic diseases, or during the course of pneumonia. Sudden symptoms arise, as palpitation, embarrassed breathing, "air hunger," deep cyanosis, convulsions, and death. These symptoms, in a lesser degree, may come on more slowly. A loud systolic bruit is heard at the base of the heart on auscultation. The treatment is carbonate of ammonia in small doses—two to three grains—with the cautious use of digitalis and stimulants. Small clots are also found associated with valvular disease. Soft, yellowish, translucent clots occur as a *post-mortem* change in chronic wasting diseases.

Rupture of the heart occurs as the result of chronic structural changes, death being generally instantaneous.

Myocarditis consists of inflammation of the muscular substance of the heart, and it exists, more or less, with pericarditis and endocarditis. The symptoms of myocarditis are obscure. The heart's action is weak and irregular. The patient passes into the *typhoid state*. Pyæmia and septicæmia, puerperal fever, and diphtheria are causes; and if abscesses form and discharge their contents into the cavity of the heart, multiple embolisms, with rigors, sweats, delirium, &c., may be the result. The treatment consists of free stimulation.

Hydropericardium or *pericardial dropsy* exists usually as a part of general dropsy; or it may occur as the result of pressure by aneurisms, mediastinal tumours, pneumothorax, &c.; or it arises secondary to Bright's disease. There is no fever, friction, nor bulging of the thorax; and the dulness is less, and more easily altered by change of posture. Paracentesis may sometimes be necessary.

Pneumopericardium may result from decomposition of the pericardial fluid, or by the entrance of air from without. It may give rise to tympanitic resonance over the heart.

Hæmopericardium may result from rupture, injury, or hæmorrhagic effusion, scorbutus, and purpura. The symptoms are those of severe collapse if a large quantity of blood be effused, and the signs are those associated with accumulation of pericardial fluid.

Adherent pericardium is the result of chronic pericarditis. There is dull pain over the heart, palpitation and breathlessness upon exertion. The impulse is increased in extent, and there is depression of the intercostal spaces, synchronous with the contraction of the heart. The apex beat is generally higher, and change of posture does not alter it. There may be dulness on percussion; and friction may be heard. The pericardial sac may be completely obliterated

and calcified, giving rise to serious disturbance of the circulation, especially when increased work is required of the heart.

Diseases of the Blood-vessels.—**Phlebitis and Varix** are considered in surgical works.

Thrombosis and Embolism.—*Thrombosis* is a local coagulation of the blood within the heart or a blood-vessel, the clot thus formed being a *thrombus*. *Embolism* means the plugging of a vessel by a solid fragment, or *embolus*, from some distant part. Any condition which tends to render the blood stagnant, favours the formation of a thrombus. Cardiac disease, pulmonary affections and wasting diseases, aneurism, and varicose veins ; morbid conditions of the blood, as in diabetes, gout, typhoid, and especially puerperal fever, pyæmia, &c. ; diseases of the coats of the blood-vessels and endocardium, as atheroma and acute inflammation roughening the surfaces of valves—are all *causes* of thrombosis. The irritation produced by the clot may set up endarteritis or endophlebitis ; and disease in the immediate neighbourhood of a vein may cause thrombosis—*e.g.*, disease of the temporal bone, producing thrombosis in the jugular vein ; cancer of the liver, affecting the portal vein ; and aneurism, pressing upon the vena cava. Thrombi in the iliac veins and inferior vena cava are dangerous, as an embolus may break off at any moment, and passing through the right side of the heart, may plug the pulmonary vessels (see *Heart Clots*, p. 44). The puerperal state favours this latter condition, the clots within the uterine sinuses spreading to the iliac veins. **Phlegmasia dolens**, or white leg, is caused by thrombosis of the iliac or femoral veins.

Upon the arterial side the most common sources of embolism are vegetations from the heart valves, morbid products of atheromatous disease, and emboli from clots within the systemic veins or from aneurisms. Other sources are of pathological interest. An embolus may plug the middle cerebral artery, generally the left, and produce hemiplegia ; but there may be embolism of the arm or leg, characterised by sudden pain in the limb, the arm or leg becoming cold and pulseless, and it may ultimately become gangrenous. Emboli—simple or infective—may be carried throughout the whole system, producing infarctions of the lung (pulmonary embolism), kidney, spleen, &c.

The *symptoms and treatment* of thrombosis and embolism vary according to the organ affected, and they are described with the diseases of these organs. Venous thrombosis is characterised by pain and swelling of the parts behind the obstruction, as in phlegmasia dolens, where the pain and tenderness is referred to the thigh, the vein is felt to be thickened and cord-like, and the lymphatics are visible as superficial red lines. Venous congestion is followed by œdema, giving the leg a *white*, glazed, and stretched appearance.

The *treatment* consists of, at first, absolute rest, with hot fomenta-

tions of opium or belladonna applied to the limb. Morphia may be necessary for the pain. Later, *tonics*, and friction of the limb to induce absorption, may be ordered.

Endarteritis, or inflammation of the arteries, may be *acute*, with pain referred to the part, as the aorta; or more frequently it is *chronic*, and associated with atheromatous changes (see *Angina Pectoris*). The aorta and cerebral arteries are the favourite seats. The aortic valves are frequently affected (see *Aortic Heart Disease*). The second sound may be accentuated, and the heart hypertrophied, when the disease is in the ascending portion of the aorta. The radial and temporal arteries may be rigid and tortuous, and they may be rolled under the skin like whip-cord. It occurs in advanced age, but it may appear in middle life, especially when the patient suffers from gout, rheumatism, syphilis, lead poisoning, or chronic alcoholism.

The *treatment* requires the cause to be considered and corrected, if possible. Cod-liver oil, quinine, and iron, with good nourishing diet, are general indications.

Aneurism—Thoracic and Abdominal.—A true aneurism is a localised dilatation of an artery, involving all three of its coats. A bulging of one side, or even a uniformly dilated artery which does not give rise to pressure symptoms, is often termed an "aneurismal dilatation"; but should this dilatation extend further it becomes the spindle-shaped or fusiform aneurism. There are also the cylindrical-shaped, sacculated, dissecting, and varicose forms—the latter being when the aneurism communicates with a venous cavity or vessel. The term *false* aneurism is sometimes applied to the sacculated form when the coats of the artery are incomplete. A ruptured blood-vessel with condensation of the tissues around a clot is also a false aneurism. *Miliary* aneurisms are considered with cerebral hæmorrhage. The other forms are described in surgical works.

The commonest seat of aneurism is the aorta, especially the ascending portion and the arch, and in these situations the sacculated form is the most frequent. The sac may be composed of all three coats, especially when *fusiform*, but it may consist of only the outer coat—the two inner having undergone more or less morbid softening, and the outer having become much thickened by calcified and inflammatory deposits and is sometimes adherent to the adjacent structures. When the aneurism is fusiform in shape the walls of the sac are usually smooth, and there is no tendency to coagulation of the blood; but when sacculated, the walls are rough, and fibrin is deposited in layers. The slower circulation within the sacculated form also favours coagulation. Dissecting aneurisms occur in the aorta, and are produced by the rupture of the inner and middle coats, the blood being effused under the external coat. Sometimes there is a sac, but usually there is little or no swelling, and hence no marked pressure symptoms in such cases. Aneurisms may press upon the neighbouring organs producing inflammation and atrophy, and they may ulcerate into the bronchi, œsophagus, or thoracic duct, &c. The bones of the spinal column may be eroded, and nerve trunks irritated

or even absorbed. Thrombi may form in the venæ cavæ. Rupture is the commonest termination; but recovery sometimes takes place.

The **causes** of aneurism are chiefly chronic arteritis and atheroma; and laborious occupations which throw strain upon the aorta (blacksmiths, sawyers, &c). Soldiers are frequently sufferers from aneurism. Gout, rheumatism, the abuse of alcohol, lead poisoning, and probably syphilis, all predispose to the development of atheroma. Aneurism is more frequent in males, about the middle period of life.

The **symptoms** of thoracic aneurism may be divided (1) into those associated with aneurism *proper*, (2) those the result of a circumscribed tumour containing fluid or solid blood and giving rise to *pressure* upon the neighbouring organs, and (3) *special* symptoms according to the seat. The pressure symptoms, especially in the early stages, are sometimes not very well marked owing to the soft and yielding character of the thoracic structures.

1. Inspection may reveal a pulsating tumour which may be felt to expand and communicate a thrill to the hand, the pulsation being *at least* as forcible as the heart (Balfour). Auscultation of the tumour, or over a circumscribed area of dulness, may reveal a distinct jar or throb, and sometimes a murmur. The second sound of the heart is accentuated and "booming" in character; but should the aneurism involve the aortic valves, a diastolic murmur is present, due to the regurgitation. The cardiac sounds or murmurs, if they exist, are propagated over the aneurism; and accentuation of the second sound heard over a dull patch outside the aortic area is very characteristic of aneurism. The radial pulses may be delayed or may show differences in time or strength, due to diminished tension, or to occlusion by fibrin (in fusiform aneurisms of the arch); or sometimes it is the result of a sacculated aneurism of the aorta pressing upon the subclavian vessels.

2. The *pressure symptoms* depend upon the seat and direction of development of the aneurism. The thoracic aorta comes into close relation with the trachea, bronchi, and lungs, the pulmonary vessels and nerves, the œsophagus, superior vena cava, thoracic duct, and the pneumogastric, phrenic, recurrent, laryngeal, and sympathetic nerves.

Pain is generally an early symptom, and it may be referred to the front or back of the chest, and frequently shoots up the neck or down the arm, and thus it resembles the pain of true angina pectoris. It may be absent for months at a time. Pressure upon the trachea gives rise to breathlessness, often relieved by bending the head forwards; and in pressure upon the bronchi the entrance of air is impeded, and hence diminished breath-sounds on the right or left side according to the bronchus affected. Sibilant and sonorous rhonchi may be present, and sometimes the breathing—instead of being weakly vesicular on the affected side—is bronchial in character. The affected lung may also yield a higher pitched note. Should ulceration follow, then hæmoptysis may be present as a symptom. The attacks may consist of the slow and occasional oozing of blood—sometimes these attacks extending over a period of years—or there may be sudden rupture and death.

Pressure upon the lungs—or upon the pneumogastric and pulmonary plexus—gives rise to cough and dyspnœa, with the ultimate development of a low form of pneumonia with fever, terminating with all the symptoms of phthisis. The pulmonary vessels may be involved, and the proper aëration of the blood interfered with; and should ulceration take place, a varicose aneurism may be established. The heart may be displaced and the liver depressed by large aneurisms. Irritation of the phrenic nerves sometimes gives rise to hiccough, and pressure upon the œsophagus to dysphagia or difficulty in swallowing. Œdema of the head and arms may be produced by pressure upon the superior vena cava, and pressure upon the subclavian vessels may give rise to pain and swelling of the arm. Pressure upon the thoracic duct produces rapid emaciation. Pressure upon the spine sets up severe pain, generally long continued, and it often leads to ulceration of the bone, irritation of the nerves, and sometimes to paraplegia.

The dilatation of the pupil (with sometimes unilateral sweating of the face) is the result of irritation of the sympathetic, but should the nerve be destroyed the pupil is contracted.

3. These pressure symptoms are more or less common to aneurism of any part of the thoracic aorta; but the signs characteristic of the part involved may be shortly stated:—

Ascending Aorta.—An aneurism of this part usually extends to the right and behind the arch. There is more or less dulness, according to the size, at the level of the second and third rib of the right side. Pulsation may also be felt here; and sometimes the aneurism ulcerates through the ribs. The aortic valves are usually affected. Pressure upon the right auricle, or vena cava, may give rise to cyanosis, enlargement of the veins of the neck and chest, and sometimes to venous stasis and dropsy. The pressure may affect the right bronchus, pulmonary vessels, lung, subclavian vessels, and sometimes the sympathetic nerve, with the results stated in the foregoing. Death may result from rupture of the sac into the pericardium.

The Arch.—An aneurism of the arch of the aorta usually affects the trachea and œsophagus, and it may involve the bronchi. Pulsation may be felt at the sternal notch, and sometimes dulness is increased at the level of the left second costal cartilage, and a bruit is heard there. The pupil symptoms are often present. A characteristic symptom of aneurism of the arch is the loud, ringing cough, due to interference with the recurrent laryngeal nerve—almost invariably the left. The breathing may be embarrassed from the same cause, and the voice may be affected (*vox anserina*). The laryngoscope reveals the paralysis of the left vocal cord when sound is attempted. Complete aphonia is rare. The pressure may also affect the pneumogastric and pulmonary plexus of nerves, the lung, pulmonary vessels, œsophagus, or spine.

Descending Aorta.—An aneurism of this part usually develops to the left of the vertebral column, and dulness may, sometimes, be made out in the left interscapular region, from the level of the fourth dorsal vertebra downwards. Pulsating tumours are sometimes felt,

and a bruit may be heard in this region. They usually give rise to much pain, and the spine is involved sooner than with aneurisms of the arch. Dysphagia is a common symptom. They also may compress the trachea, left bronchus, and lung, and death may result from pneumonia or rupture into the pleura or œsophagus.

Abdominal Aneurism.—An aneurism of the abdominal aorta is usually situated at or near the cœliac axis. Should it develop posteriorly the vertebræ are soon involved and pain is the prominent symptom, referred to the back, and shooting down the sciatic nerves. Sometimes the pain is felt in front radiating up into the hypochondriac regions, or down into the iliac. In addition to these occasional paroxysms dull local pain is almost constantly complained of, and it is frequently referred to the stomach and associated with the symptoms of indigestion. A tumour may be felt to the left of the epigastrium, communicating an impulse to the abdomen, and felt to expand in all directions. The patient should be turned over on his front in the palpation of such a tumour. A bruit may be heard over the tumour, and sometimes at the back.

Aneurisms of the *hepatic*, *mesenteric*, and *coronary* arteries are of rare occurrence. *Innominate*, *subclavian*, and *iliac* aneurisms are considered in surgical works. The symptoms of aneurism of the innominate are like those of the first part of the arch.

The **diagnosis** of aneurism is sometimes easy and sometimes very difficult, and often it can only be surmised. There may only be some ill-defined sensations complained of, or the pain may be very severe, yet unaccompanied by any other pressure symptoms. At other times pressure symptoms are present without the signs proper to aneurism. Pulsations from the heart and aorta may be transmitted to swellings, and bruits may be heard over them, but they do not expand in every direction. The chief morbid conditions which may simulate aneurisms are mediastinal tumours or abscesses, pulsating empyemata, tumours or suppurations of the chest wall, pericardial effusions, and cardiac disease. In the latter case aneurisms may displace the heart and suggest that there is only enlargement, but there may be a second impulse felt, and pressure symptoms present; or, on the other hand, there may be signs of heart disease, as dropsy, albuminuria, &c., helping to a correct diagnosis. A phthisical consolidation of the left apex of the lung, associated with the pulmonary or subclavian murmur, may sometimes simulate aneurism. The age, sex, occupation, history, and seat of the pulsations are all important considerations in doubtful cases. With regard to the form of aneurism, a diffuse impulse, thrill being marked, and the presence of a *whirring* murmur with few or no pressure symptoms, all point to dilatation or fusiform aneurism. A persistent rheumatism of the shoulder, especially of the left, should always suggest an examination for aneurism. Abdominal aneurism may require to be distinguished from cancerous and simple tumours, floating kidney or spleen, and from simple nervous pulsation (*neurotic aortæ*). The symptoms are very often attributed to chronic dyspepsia and the paroxysmal pain ascribed to gastralgia, sciatica, &c.

The **prognosis** is always very grave. The disease is very chronic, and the termination may be by exhaustion, pneumonia, or by sudden rupture and hæmorrhage. Under good treatment life may be prolonged, and sometimes cures are effected.

The medical **treatment** of aneurism may be very shortly stated, as iodide of potassium has practically displaced all other remedies. Solidification of the sac is the object of treatment, and the iodide, by a peculiar action upon the fibrous tissues whereby the walls become thickened, has proved the most efficient remedy. It also lowers the blood pressure, and hence favours coagulation within the sac, while checking further tendency to dilatation. Its use will relieve the pain and other symptoms, sometimes within three or four days. The dose is ten grains or more, thrice daily. Rest in the recumbent position is also a most important aid to the treatment; but if the aneurism be not large and the symptoms not urgent, *absolute* rest is not necessary. The diet should be restricted, and very light and digestible; but some cases do better with a full diet. Alcohol is contra-indicated. A cure may take place in three months. External remedies may be used—as belladonna and opium—and the tumour, if bulging, should be protected with cotton wool or a cage. Should the heart be affected, digitalis may be necessary. To relieve pain, a hypodermic injection of morphia should be given. For hæmoptysis give acetate of lead (see ℞ 9); or ergotine may be injected. Cough and breathlessness may be relieved by fifteen minim doses of chlorodyne, or by ℞ 10. The treatment of abdominal aneurism is the same; but here pressure is possible, and in suitable cases it may be the best. Aneurisms may also be treated by surgical methods.

Mediastinal Tumours.—These are best considered here in relation to aortic aneurism, which is by far the most frequent mediastinal enlargement. *Cancer* arising from the œsophagus, glands, and root of the lung; *tubercular* masses of glands or *lymphadenomatous* tumours; simple *fibrous and fatty* tumours; inflammatory *exudations and abscesses*—may all originate within the mediastinum. The symptoms may be very indefinite. Pain, breathlessness, and difficulty in swallowing are common. "Currant-jelly" expectoration is said to occur frequently in cancerous growths. Inspection may show a local bulging, the respiratory movements being deficient or absent. Interference with the superior vena cava and innominate veins, producing dilatation of the superficial veins, and œdema of the arms and face, is far more common from the pressure of mediastinal tumours than from pressure of aneurismal growths. Percussion reveals complete dulness when the tumour is large, and the outline of its limits is important in its differentiation from pleuritic and pericardial effusions. The sense of resistance is increased. The vocal fremitus is usually absent. Auscultation over the tumour may reveal diminution of the breath-sounds, or the contrary, according to its size and its relation to the bronchial tubes; and the vocal resonance may be altered from the same cause. Moist sounds may be present. The heart and other organs may be displaced.

Diagnosis.—Mediastinal tumours require to be distinguished from pleuritic and pericardial effusions, enlargement of the heart, and from chronic pneumonia. The history of the case is important in the differentiation of these, and it should be noted that a pleuritic effusion may arise, secondarily, when the pleura is reached by a new growth. (For the differentiation of aneurism, see p. 49.) If a cancerous cachexia be present it is an important indication.

The *prognosis* in all cases of mediastinal tumour is grave; and the *treatment* can only consist of relieving symptoms.

CHAPTER III.

DISEASES OF THE RESPIRATORY SYSTEM—Section I.

Contents.—Coryza—Epistaxis—*Laryngoscopy*—*The anatomy of the larynx*—Acute laryngitis—Œdematous laryngitis—Chronic laryngitis, phthisis, and syphilis of the larynx—Perichondritis—Morbid growths within the larynx—Laryngeal paralysis—Laryngismus stridulus—Croup—Spurious croup—**Percussion**—*Medical anatomy of the lungs, liver, and spleen*—**Auscultation.**

Catarrh—Coryza.—"Catching cold" is of the nature of a fever or febricula when acute. The quantity of blood within the cutaneous capillaries is controlled by the vaso-motor nerves to the smaller blood-vessels, and these nerves, by their contracting and dilating fibres, regulate the supply. The external temperature, through the sensory nerves of the skin, acts upon the vaso-motor centre, and reflexly contracts or dilates the blood-vessels according to the stimulus—cold or warmth. Any *sudden* change from warm to cold, by the exposure of a larger quantity of blood to the cooling process, probably sets up chemical change within it, and at the same time the sudden reflex contraction of the blood-vessels produces congestion of the internal organs. There is increased tissue metabolism, especially in the muscles and red blood corpuscles, as shown by the increased excretion of potash. The heat-regulating mechanism is also disturbed. The organs which suffer will be those which are weakest in vitality, either from constitutional tendencies or the result of previous attacks. Careless exposure will produce catarrh in the most robust; but it is far more frequent in the delicate or strumous, or in those who may be temporarily in a low state of health. The nervous diathesis is an important predisposing factor in the tendency to catarrh, some people being morbidly sensitive to changes of temperature. When the two constitutions—strumous and nervous—are combined, as they frequently are, the

tendency to catarrh is very great, and it often becomes more or less chronic, with subacute attacks.

The **symptoms** begin with a feeling of chilliness, perhaps a rigor, and the temperature rises to 101° or 102° Fahr. The pulse is quick, the skin dry, the tongue furred, and the urine is thick with urates, which are deposited on cooling. Sometimes there is much depression, and often there is a tired or aching feeling in the limbs. These symptoms are accompanied by frontal headache and dryness of the nasal mucous membranes, followed soon by increased mucous secretion. The congestion may extend to the pharynx, larynx, and trachea—pain upon swallowing, hoarseness, and a tickling cough being the results. The Eustachian tubes may be affected, and partial deafness may be noticed in some cases. Taste and smell may be impaired. The nostrils become sore from the irritating discharge, and herpes may affect the lips. The fever, under treatment, may disappear in a few hours, but the cough accompanied by at first mucus and then muco-purulent expectoration, may remain for several days, and if chronic, for a considerable time longer. In the chronic form, the mucous membranes become hyperæmic and hypertrophied, sudden changes in the atmospheric conditions being apt to produce subacute attacks.

The **diagnosis** of an acute catarrh is obvious, but *measles*, *influenza*, *hay asthma*, and the coryza produced by *iodide of potassium*, must be remembered. The chronic form may suggest an examination for such surgical diseases as *polypi* of the nose, *ozæna*, strumous and syphilitic *ulcerations*, *hypertrophy* of the mucous membrane, and sometimes *chronic abscess of the antrum* with a muco-purulent discharge from the nostril.*

A cold may extend to the bronchial tubes, especially in the young and in the aged; but in some cases it attacks the stomach and bowels, a chill producing gastric symptoms and often a sharp attack of diarrhœa. Recovery from an acute cold generally takes place within a few days.

The **treatment** of catarrh depends upon the stage. If taken very early, a Turkish or hot vapour bath may cut it short. The latter may easily be taken at home by means of Allen's spirit kettle, a simple and safe apparatus which may be used in any bath-room, and its use has the advantage of allowing the patient at once to proceed to bed without risk of a second chill. Five or ten grains of quinine, fifteen grains of antipyrin, or ten grains of Dover's powder, may be taken at bedtime—hot milk or gruel being allowed for supper. This treatment may be sufficient, and it may enable the patient to go about next morning with care; but if the cold be a severe one, he should certainly keep his bed for a few days. Tincture of aconite—drop doses in water every ten minutes for two hours—is a useful remedy when the fever is high and the pulse strong and bounding.

* Apart from nasal catarrh altogether, it should be noted that "many cases of megrim, asthma, nightmare, nervous cough, supra-orbital neuralgia, swelling of the face, vertigo, and epilepsy," are intimately associated with diseases of the nose (M'Bride).

Spirit of Mindererus (*liquor ammonii acetatis diluti*), in half ounce doses with plenty of water, every four hours, is a good diaphoretic. Salicylate of soda may be added in rheumatic cases (℞ 11). For the hoarseness or sore throat give inhalations of steam with compound tincture of benzoin (a teaspoonful to a pint of boiling water in a Maw's inhaler); also gargles of cold water with chlorate of potash and glycerine of tannic acid (a teaspoonful of each to a tumbler of water). Mustard may be applied to the front of the chest, and chlorodyne—five to fifteen drops in water—or other cough mixture (℞ 12) may be necessary. When the feverish symptoms have been allayed, a diuretic mixture (℞ 7) may be ordered, or small doses of calomel or blue pill may be given, with a saline aperient to follow in a few hours. For the prevention of colds the skin should be kept healthy in its action by the regular cold bath in the morning. Cod-liver oil—a dessert spoonful at bedtime—and tonics, as arsenic and iron, are good prophylactics in those who have a tendency to chronic catarrh. Local measures, as the inhalation of ammonium chloride vapour, and painting the mucous membranes with tannic acid and glycerine, or a solution of nitrate of silver—ten grains to the ounce—are of benefit; but if the attacks be frequent, and recovery slow, a change, either to sea air or to a mountainous district, is to be recommended.

Epistaxis, or bleeding at the nose, is described in surgical works, but it is mentioned here merely to classify the diseases in which it occurs as an important symptom. They are as follow, viz.:—Hypertrophy and valvular diseases of the heart; Bright's disease, especially the cirrhotic form. (In these, epistaxis indicates a tendency to, and it may be a warning of, cerebral hæmorrhage.) Tuberculosis and phthisis; Cirrhotic liver; Hæmorrhagic diseases—as purpura, scorbutus, leucocythæmia, pernicious anæmia, and hæmophilia, &c.; Intracranial growths, cerebral atheroma; Fevers—as typhoid, ague, small-pox.

Epistaxis has been observed in some cases to be vicarious with the catamenia (*vicarious menstruation*); and at puberty bleeding at the nose may readily be excited in some subjects. It occurs also from local causes, as acute nasal congestion and catarrh, ulceration of the mucous membranes, vascular tumours, &c.

Laryngoscopy.

The mirrors used in the examination of the larynx are too familiar to need any description. The interior of the larynx is usually illuminated by the reflected light of an ordinary lamp; but the electric and oxyhydrogen limelights are frequently used. Direct sunlight, although it has the advantage of showing the tissues in their natural colouring, is not often convenient. The lamp being placed at the side of the patient's head, and the reflecting mirror strapped to the operator's forehead, a beam of light is thrown into the pharynx. The patient keeps the mouth wide open and protrudes

the tongue, which he may hold down by a napkin. The laryngoscopic mirror, after being warmed over a spirit lamp, *and tested upon the back of the hand*, is gently introduced, and just touches the uvula, palate, and pharynx. It should be kept quite steady, as otherwise it is apt to excite retching. Gargling with iced water, and spraying or painting the throat with a 10 per cent. solution of cocaine, will lessen the sensibility, and allow of greater freedom in the examina-

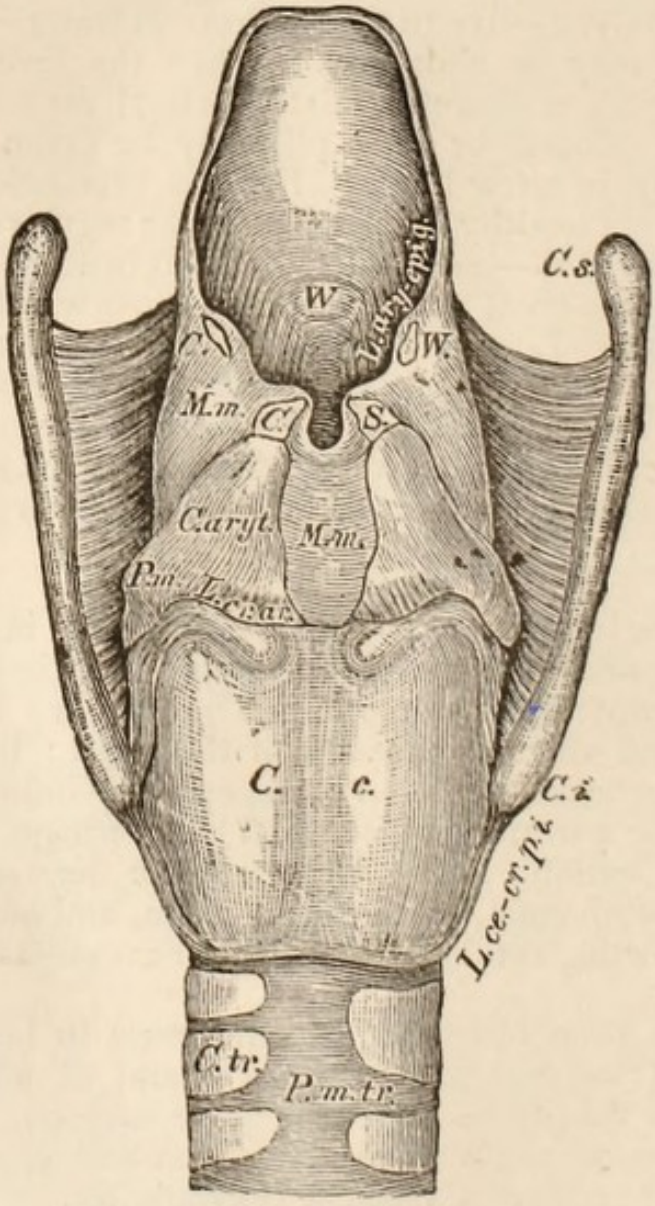


Fig. 8.—Larynx from behind after removal of the Muscles.—Epiglottis cushion (*W*); *L.ar.ep*, lig. ary.-epiglotticum; *M.m*, membrana mucosa; *C.W*, cart. Wrisbergii; *C.S*, cart. Santorini; *C.aryt*, cart. arytænoidea; *C.c*, cart. cricoidea; *P.m*, processus muscularis of cart. arytæn.; *L.cr.ar*, ligam. crico-arytæn.; *C.s*, cornu superius; *C.i*, cornu inferius of cart. thyreoidea; *L.ce cr. p.i*, lig. kerato-cricoideum post. inf.; *C.tr*, cart. tracheales; *P.m.tr*, pars membranacea tracheæ. (From *Landois and Stirling's Physiology.*)

tion of such cases. The mirror must be gently deflected until the image of the larynx is seen, the patient being instructed to take deep breaths, and to sound "ah," and high "e" (as in *feet*). Practice will generally overcome the early difficulties of getting a view of the vocal cords and the interior of the larynx; and it should be

remembered that the laryngeal structures are seen in the mirror *reversed*, the arytenoid cartilages, for instance, being seen at the lower part of the mirror, and the anterior angle of the glottis at the upper.

The Anatomy of the Larynx.—The accompanying diagrams (Figs. 8 and 9) will serve to review the general anatomy of the larynx. The cricoid cartilage, whose small narrow portion is

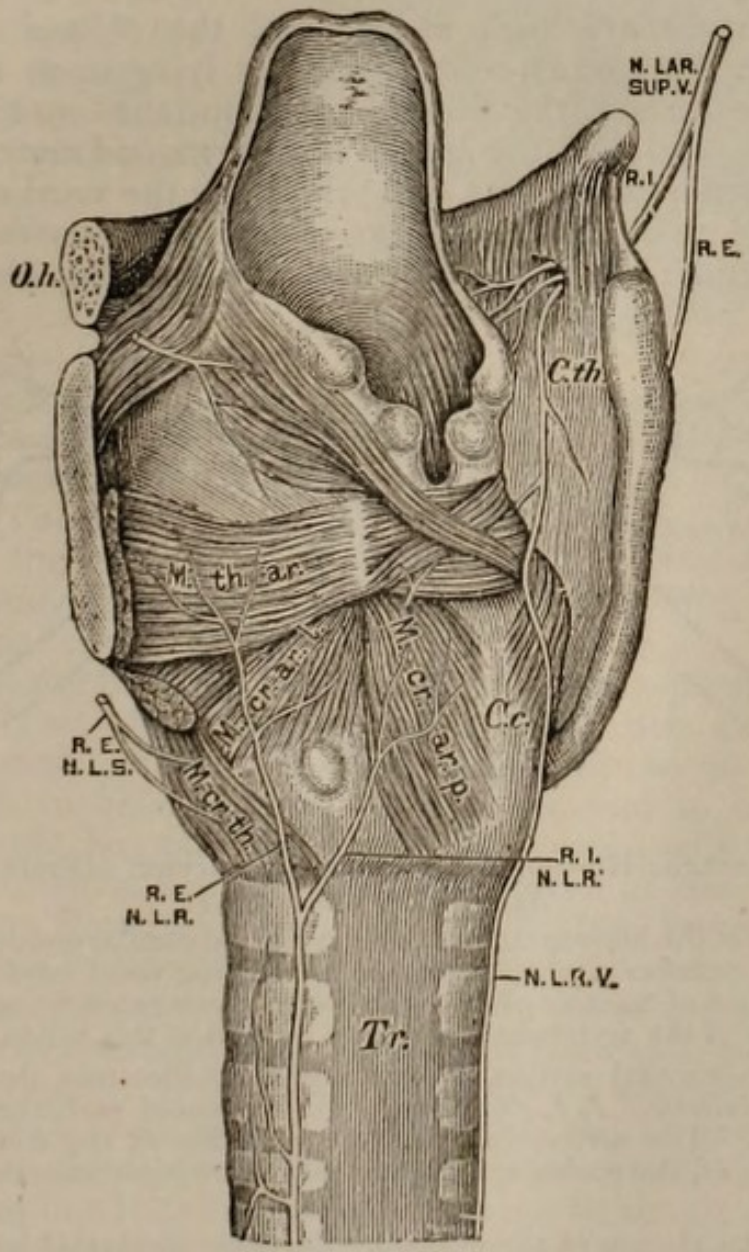


Fig. 9.—Nerves of the Larynx.—*O.h*, Os hyoideum; *C.th.* cart. thyreoidea; *C.c*, cart. cricoidea; *Tr*, trachea; *M.th.ar*, M. thyreo-arytænoideus; *M.cr.ar.p*, M. crico-arytænoideus posticus; *M.cr.ar.l*, M. crico-arytæn. lateralis; *M.cr.th*, M. crico-thyreoideus; *N.lar.sup.v*, N. laryngeus sup.; *R.I*, ramus internus; *R.E*, ramus ext.; *N.L.R.V*, N. laryngeus recurrens; *R.I.N.L.R*, ramus int.; *R.E.N L.R*, ramus ext. nervi laryngei recurrentis vagi. (From *Landois and Stirling's Physiology.*)

directed forwards, and the broad plate backwards, articulates with the inferior cornua of the thyroid. The triangular arytenoid cartilages articulate, at some distance from the middle line, with oval, saddle-

like, articular surfaces placed upon the upper margin of the plate of the cricoid cartilage. The true vocal cords arise close to each other, from near the middle of the inner angle of the thyroid cartilage, and they are inserted each into the anterior angle, or *processus vocalis*, of the arytenoid cartilages.

The *posterior crico-arytenoid* muscles pull the processus musculares of the arytenoid cartilages backwards, downwards, and towards the middle line, so that the cords are separated and the glottis widened as in inspiration.

The *transverse arytenoid muscle* and the *oblique* muscles (the latter seen in Fig. 9 as crossed bundles lying upon the posterior surface of the former, the fibres being continued up to the root of the epiglottis), and also the *lateral crico-arytenoid* muscles, approximate the arytenoid cartilages, and with them the vocal cords, and so *contract* the glottis. The *crico-thyroid* and *thyro-arytenoid* muscles, stretch and render *tense* the true vocal cords during phonation (see

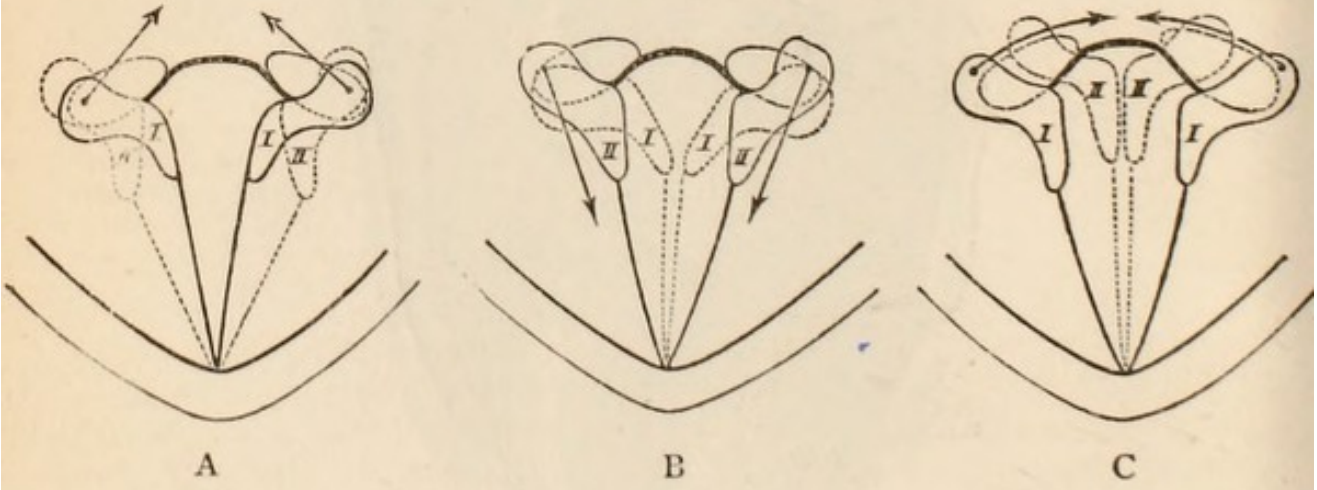


Fig. 10.—Schematic Horizontal Section of the Larynx. (From *Landois and Stirling's Physiology*.)

A. *I, I,* Position of the horizontally divided arytenoid cartilages during respiration; from their anterior processes run the converging vocal cords. The arrows show the line of traction of the *posterior crico-arytenoid* muscles; *II, II,* the position of the arytenoid muscles as a result of this action.

B. Schematic horizontal section of the larynx, to illustrate the action of the *arytenoid muscle*. *I, I,* Position of the arytenoid cartilages during quiet respiration. The arrows indicate the direction of the contraction of the muscle; *II, II,* the position of the arytenoid cartilages after the arytænoideus contracts.

C. Scheme of the closure of the glottis by the *thyro-arytenoid* muscles. *II, II,* Position of the arytenoid cartilages during quiet respiration. The arrows indicate the direction of the muscular traction; *I, I,* position of the arytænoid cartilages after the muscles contract.

Fig. 10). The *superior laryngeal nerve* is the sensory nerve to the mucous membrane of the larynx, and it also supplies the crico-thyroid muscle. All the other intrinsic muscles of the larynx are supplied by the *inferior* (*recurrent*) *laryngeal nerve*.

The following diagram (Fig. 11) shows the structures of the larynx as seen by the laryngoscope.

Acute Laryngitis.—In acute catarrh of the larynx there is

slight fever, with a dry sensation or feeling of tightness at the windpipe, sometimes also a tickling cough and pain upon swallowing. If the vocal cords be affected the voice becomes husky, the cough hoarse, and there may be complete aphonia. There is much tendency to "hawk," the expectoration being at first slight, if any; later, some mucus is ejected. The laryngoscopic examination shows the mucous membranes to be bright red and swollen, slightly coated with mucous secretions, and with sometimes small catarrhal ulcerations of the surface. The catarrh may affect the whole larynx or only parts. The epiglottis may be much swollen, and the mucous membrane over the arytenoid cartilages very frequently so, their mobility during inspiration and expiration being diminished. The false vocal cords may be so swollen and œdematous as to hide the true vocal cords, and to obliterate the ventricles of the larynx. When the true vocal cords are affected, they lose their white lustre and become more or less injected, the colour varying from pink to red; and if they be swollen, their margins become rounded and their mobility impaired.

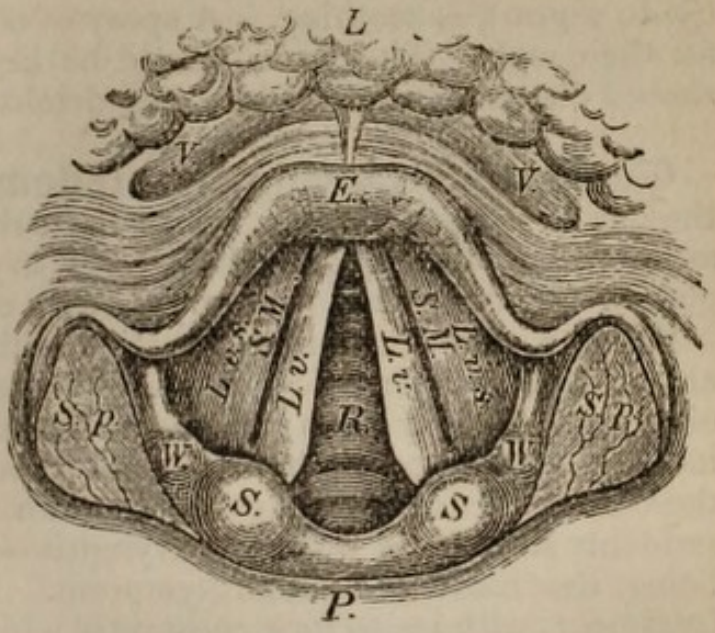


Fig. 11.—The Larynx as seen with the Laryngoscope.—*L*, Tongue; *E*, epiglottis; *V*, vallecula; *R*, glottis; *L.v*, true vocal cords; *S.M*, sinus Morgagni; *L.v.s*, false vocal cords; *P*, position of pharynx; *S*, cartilage of Santorini; *W*, of Wrisberg: *S.p*, sinus piriformes. (From *Landois and Stirling's Physiology.*)

The **causes** of acute laryngitis are direct exposure to cold or irritating vapours; extension of a cold from the nasal and pharyngeal mucous membranes; alcoholism; rheumatism; excessive use of the voice; and ulcerations and growths within the larynx. It occurs also in certain febrile conditions, as typhus, erysipelas, &c. Recovery is usual in adults; but laryngitis is apt to recur and to become chronic. In children it is more serious, and there is more liability to spasm (see *Croup*).

The **treatment** (of adults) in the early stages is the same as in acute coryza (p. 52). A cold compress over the throat may also be used. Aconite and ipecacuanha are useful drugs—the latter in small doses to increase the mucous secretions and render expectoration easy; and in teaspoonful doses as an *emetic*, if there be breathlessness and cough without relief, as frequently happens with children. The bronchitis kettle may be necessary, and hot sponges may be applied to the larynx. Ice may be sucked. The voice should be rested. Leeches are sometimes used over the upper part of the sternum.

The inhalation of a few drops of chloroform will relieve spasm. The inhalation of steam, with the compound tincture of benzoin (℥j to a pint), is soothing. A spray of cocaine (5 per cent.) is useful for the cough. The room should be kept very warm; and the diet should consist of milk and cooling drinks, beef tea and soups, &c.

Œdematous Laryngitis.—Œdema Glottidis.—Œdema of the larynx is most marked where the submucous tissue is most lax, as in the aryteno-epiglottidean folds, arytenoid cartilages, and epiglottis. The swelling is light yellow in colour.

Œdema may occur in connection with acute laryngitis, but it is also induced by chronic laryngeal diseases, and it occurs as a part of general dropsy from cardiac or renal disease. It may also be induced by swallowing hot liquids. It is said (very rarely) to follow the continued use of iodide of potassium. The symptoms may occur suddenly during an ordinary laryngitis—urgent inspiratory dyspnœa being the most prominent symptom. Prompt scarification is the treatment, with ice to suck constantly while there is danger of suffocation. An emetic may be given, and the general treatment of acute laryngitis should be carried out. Tracheotomy may be necessary. Hydragogue cathartics are indicated when the œdema is due to cardiac or renal disease.

Chronic Laryngitis.—In chronic catarrh of the larynx the mucous membranes and submucous tissues become thickened and hyperæmic, with more or less œdema. The surface is pale, dry, and shining, and coated with viscid mucus. Erosions and superficial ulcerations are common. In severe cases the calibre of the larynx may be narrowed, especially in the phthisical and syphilitic forms. The epiglottis and aryteno-epiglottidean folds or ligaments become much thickened. Several forms are described as *laryngorrhœa*, "*ozæna laryngis*," *pachydermia laryngis*, &c. Chronic laryngitis may follow repeated acute attacks, or be simply chronic from the first. It may be secondary to chronic laryngeal disease, or an indication of phthisis, alcoholism, or syphilis. Excessive smoking, overuse, and irritation of the recurrent laryngeal nerve, are also causes.

In **Phthisis of the larynx** the mucous membranes are paler, and thrown into folds. The inter-arytenoid and ary-epiglottic folds are infiltrated, the arytenoids appear as pink swellings, and small patches are seen on the mucous membrane. As the disease advances there is ulceration of the laryngeal structures, generally superficial, but sometimes extending to the deeper tissues. Most commonly the vocal cords suffer from infiltration, which gives them an irregular *nodular* appearance. If an ulcer affect the inner border, the cord has a notched, "eaten-out" appearance, and it is congested and swollen, the ulcer itself being of a dull grey-white colour. The false cords are frequently so swollen as to obscure the view of the true cords, and give the appearance of greater destruction than really exists. The epiglottis is also a frequent seat of ulceration in phthisis, and it generally is much thickened and hypertrophied.

Syphilis of the larynx presents the same appearances as phthisis, but the ulcers are said to be more circular in form and to have sharper edges, with lardaceous looking particles upon their surfaces. Ulceration is rapid, and the upper surface of the epiglottis is a favourite site. There may only be one ulcer present. Under treatment they cicatrise, and, if large enough, they may produce stenosis and deformities of the larynx and destruction of the cords. Gummata are occasionally found developed in the larynx, and they give rise to the same symptoms as tumours and morbid growths.

Perichondritis of the larynx may be primary, but generally it is a secondary affection, and often it is a result of phthisis or syphilis. It may also appear during the course of acute fevers, diphtheria, &c. The arytenoid cartilages are most commonly the seat of the disease, the cricoid sometimes, and the thyroid seldom being affected, and then it is secondary to disease elsewhere. The appearances at first, as the abscess forms, are the same as in ordinary inflammatory swellings, and these may project into the larynx and produce symptoms of stenosis. When the arytenoid cartilages suffer, their mobility is much impaired, and when limited to one, this impairment is very obvious with the laryngoscope. After pus has formed the abscess bursts, and it may leave excavations after expectoration of fragments of cartilage, &c.

Symptoms.—The foregoing chronic laryngeal diseases are attended with a feeling of irritation within the larynx and spasmodic cough; and the voice is either harsh or there is complete aphonia. The breathing may be embarrassed and noisy, and there may be much dyspnœa should the larynx be obstructed by thickening and œdema. Deglutition is painful, especially when the epiglottis is affected. In the severe cases, blood, pus, and laryngeal structures are expectorated.

The **diagnosis** between simple, phthisical, and syphilitic laryngitis rests far more with the history and other physical signs of these diseases than with the laryngoscopic appearances of each. In phthisis there may be advanced laryngeal symptoms before any of the physical signs of pulmonary disease can be made out; while, on the other hand, phthisical patients may have a simple chronic laryngitis which is in no way tubercular. The presence of laryngeal *anæmia* is strongly indicative of phthisis, and it should always suggest an examination of the lungs. M'Bride considers the presence of a swelling, irregular in outline, in the inter-arytenoid commissure, as strong presumptive evidence of tuberculosis. The lungs should always be examined in cases of chronic laryngeal disease.

The **prognosis** of chronic laryngitis, and syphilis of the larynx, is hopeful if seen early enough, and if proper treatment be carried out. When advanced, chronic laryngitis may continue for years, with occasional acute attacks, and without the general health suffering, if the patient be placed in favourable circumstances. In syphilis, a cure may be expected even if advanced, but cicatrisations

may permanently affect the larynx and the voice if the treatment should have been too long delayed. The phthisical form is, of course, the least hopeful, and it is very intractable to treatment.

Treatment.—In the treatment of chronic laryngitis, the principle of *rest* should be carried out as far as possible. Clergymen and others who suffer from excessive use of the voice, must avoid all unnecessary straining of the cords. Solutions of tannin, chloride of zinc, nitrate of silver, or of iodised glycerine (*see* R 13) should be applied to the vocal cords and larynx by means of a soft brush. Solutions of morphia or cocaine (5 per cent.) may be applied when there is much irritability. Iodine tincture, or the ointment of the red iodide of mercury, may be applied externally as a counter-irritant. Sudden changes of temperature are to be avoided, and change to a warm and dry climate may bring about a favourable result even in very chronic cases. The treatment of laryngeal phthisis is much the same; but the insufflation of powders as alum, tannin, iodoform, or iodol, and the inhalation of iodine (a teaspoonful of the tincture to a pint of boiling water) will be found to be useful. Oil of pine and creasote are also useful in steam inhalations. A solution of menthol in olive oil (20 per cent.) is sometimes injected into the larynx by means of a laryngeal syringe, in phthisis, and the application of a solution of lactic acid (20 to 80 per cent.) has been recently recommended by Krause (M'Bride). The solution of menthol and olive oil (5 to 20 per cent.) may be sprayed into the throat with De Vilbiss' spray. Menthol pastilles may also be used for painful throat. The general treatment of phthisis should be followed, cod-liver oil and the hypophosphites, &c., being indicated. Surgical procedures may become necessary. The value of Koch's treatment in laryngeal phthisis is not yet determined. In syphilis of the larynx, similar local remedies may be applied, while the general treatment by mercury and iodide of potassium is carried out. In perichondritis, abscesses should be opened if possible when suffocative attacks supervene, and laryngotomy should be performed if necessary.

Morbid Growths within the larynx.—These are papillomata and fibromata, or fibrous polypi, which are common; myomata, cysts or mucous polypi, which are not so frequent; and malignant growths.

The *fibroid* tumours are firm, smooth, white-yellow in colour, and they spring generally from the upper border of the vocal cords, and frequently at their anterior insertions. They may be the size of a pea or larger, and they are generally pedunculated.

The *papillomata* are wart-like, pedunculated or sessile, and of a grey colour. They may arise from the vocal cords, epiglottis, or aryteno-epiglottidean folds. Sometimes they are syphilitic in origin.

Cancer, generally epithelial, has a rough cauliflower appearance and grows rapidly. The vocal cord is the favourite site, and the disease extends upwards to the aryteno-epiglottidean folds. In the later stages the cervical lymphatics begin to enlarge and the can-

cerous cachexia is developed. The larynx enlarges and it is tender to pressure. Perichondritis and complete destruction of the larynx, with expectoration of blood and fragments of cartilage, &c., ultimately takes place. Two years is the usual duration of cancer of the larynx. It occurs in advanced life, while the other tumours mentioned may appear in the young.

The *symptoms* of morbid growths are at first much the same as in chronic laryngitis—the laryngoscopic examination revealing, however, the presence of the growth. There is a feeling of a foreign body within the larynx, and suffocative attacks (*inspiratory dyspnœa*) may supervene.

The *treatment* is entirely surgical; but in suspicious cases (warty looking growths), iodide of potassium should be first tried.

Laryngeal Paralysis.—The causes may be *central* or *peripheral*, the former being very rare, but it may occasionally occur with apoplexy and other brain diseases. The *peripheral* paralyses may be due to interference with the nerves, especially the pneumogastric and inferior laryngeal, by pressure of mediastinal tumours, enlarged glands, or aneurisms of the aorta; but in the majority of cases the paralysis is *myopathic*, and either due to paralysis of the muscles themselves or to mechanical conditions, as swellings and cicatrisations of the tissues. It is then generally incomplete (paresis). Paralysis may also be the result of diphtheria, lead and arsenic poisoning, or it may be due to hysteria.

Bilateral paralysis of the adductors.—If the whole group of muscles be paralysed, the voice is lost and the laryngoscope shows that the vocal cords are almost motionless during inspiration and expiration, and when phonation is attempted. Sometimes the paralysis is only partial and confined to certain muscles, and hence slight differences arise according as the *tensors* or *constrictors* are most affected (see p. 56). The commonest cause is hysteria, and hence bilateral paralysis of the adductors is sometimes spoken of as *hysterical aphonia*.

In *unilateral paralysis of the adductors* the voice is altered, and the laryngoscope shows that there is no movement of the cord forwards to meet its fellow—the rima glottidis being directed towards the paralysed side when phonation is attempted. Slight differences also occur when the *tensors* or *constrictors* are alone affected. It is generally the left cord that suffers, and this is due to some direct pressure upon the recurrent laryngeal nerve.

Bilateral paralysis of the abductors, or *dilators of the glottis*, is accompanied by noisy stridulous breathing, the voice being harsh and rough. The cords do not separate on taking a deep breath.

In *unilateral paralysis of the abductors* the dyspnœa is not so marked, and the affected cord remains near the median line when phonation is attempted.

Amongst the *neuroses* of the larynx, M'Bride mentions *mogiphonia*—an affection which is analogous to writer's cramp—*laryngeal vertigo*, and *nervous cough*.

In the **treatment** of paralysis the constant and induced currents

are both useful. In hysterical cases the electrodes are applied to the sides of the larynx with good results. In more difficult cases direct faradisation of the larynx may be used, the negative electrode being placed upon the neck, while an intra-laryngeal electrode is applied to the arytenoid cartilages. The treatment should be continued for a considerable time.

Laryngismus Stridulus.—Spasm of the glottis is a disease of early childhood, occurring chiefly in the scrofulous and rachitic; but it may also affect adults, and be either the result of hysteria or a consequence of pressure of aneurisms or tumours upon the recurrent laryngeal nerve. The chief symptom is the occurrence of sudden dyspnœa with stridulous and crowing inspiration—the attack often terminating in general convulsions. Recovery is usual; but recurrence of the attacks is frequent, and death may take place from suffocation.

The **causes** are generally reflex, as teething, worms, errors in diet, and exposure to cold draughts. It is common in hydrocephalus. The diagnosis is considered with true croup.

The **prognosis** is favourable if the cause be removable, and if the child is not weakly.

The **treatment** during an attack is to endeavour to relieve the spasm by hot sponges applied to the throat, by hot baths, and by dashing cold water upon the head and chest. The throat should be tickled or an emetic given, if possible, as vomiting tends to relieve the spasm. Inhalations of chloroform may be of use in severe cases, and artificial respiration should be tried if still unrelieved. An enema should be given to clear the bowels. Tracheotomy may sometimes be necessary. During the intervals of the attacks ammonium bromide may be given, and ultimately tonics, cod-liver oil, &c.—with attention to the diet and regulation of the bowels.

Croup or membranous laryngitis is a disease of childhood occurring between the second and seventh years. The mucous membrane of the air passages, especially of the larynx and trachea, become lined with a false membrane which is laminated, and usually white and tough. It is very frequently adherent to the epiglottis and vocal cords, and lying loose within the trachea, and sometimes it extends to the smaller bronchial tubes. It may, however, exist only in patches, with muco-purulent matter lining the larynx. The false membrane cannot be distinguished from diphtheritic deposit. In severe cases there may be bronchitis, pneumonia, or collapse of the lung; while congestions, œdema, and slight serous effusions are common. Much difference of opinion still exists as to the nature of membranous croup—some authors considering it to be a *laryngeal diphtheria*, while most others consider it to be a distinct disease (see *Diphtheria*).

Spurious croup is held by some authors to be simply laryngismus stridulus; but usually it is applied to cases of simple catarrhal laryn-

gitis in children, associated with sudden spasm and "crowing" inspiration, and *without* the development of a false membrane. It has a tendency to recur again and again in the same child, when exposed to cold or wet. The symptoms and treatment of the spasm have been considered with laryngismus stridulus.

The **causes** of true croup are exposure to cold and wet, or the inhalation of irritating vapours, swallowing hot water, &c. It sometimes occurs as a complication in measles, scarlatina, erysipelas, typhus and typhoid fevers.

The **symptoms** commence with slight fever and hoarseness, the cough being shrill, dry, and "brassy." There is at first no dyspnœa, but during the course of the disease this may arise suddenly. There is then much distress, the patient's muscles of forced inspiration come into play, the nostrils dilate, and all the symptoms of asphyxia are present. The inspiration is whistling and crowing, the voice is lost completely, and the cough is husky, while expectoration of fragments of membrane is attended with marked relief. The disease runs a remittent course, extending usually from two to six days, the child's breathing being noisy and difficult throughout, but with frequent exacerbations of suffocative distress. If the cough be loose, and the expectoration free, recovery may take place; but death may occur from suffocation, or from asthenia if the disease be severe and continued. The *prognosis* is, therefore, always grave.

Diagnosis.—In individual cases it is sometimes impossible to diagnose membranous croup and exclude the possibility of laryngeal diphtheria; but should the disease commence in the throat and a deposit be seen there, and if there be a history of contagion with enlargement of the glands, albuminuria, and much constitutional disturbance before the laryngeal symptoms—diphtheria will probably be the correct diagnosis. The absence of a history of contagion, the disease beginning in the larynx, and a history of exposure to damp or cold, point, in single cases, to croup. The age of the patient may also be suggestive.

The presence of the membrane differentiates the true croup from the false, but in the earliest stages it may not be possible to distinguish them; while spasmodic croup, beginning suddenly, without any previous history of illness, may either be laryngismus stridulus, or catarrhal laryngitis, with spasm superadded to the general symptoms—*spurious croup.* A few hours will generally settle the question. A *post-pharyngeal abscess* has been known to give rise to a "brassy cough" with dyspnœa; while *pertussis*, in the early stage, when the cough is dry and harsh, may resemble croup.

The **treatment** of true membranous croup is the same as in acute catarrhal laryngitis. The patient should be kept in a warm room, and hot sponges or poultices should be applied to the throat. The bronchitis steam kettle should be rigged up, and ipecacuanha should be given in small doses as an expectorant, or in teaspoonful doses as an emetic, when there is much obstruction to the breathing. For the solution of the membrane, a spray of lime water, or painting the throat with lactic acid, is recommended. Fragments of mem-

brane are to be removed with the finger, when possible. Ammonium bromide, chloral, and belladonna are useful for spasm; and quinine may be given to control the exudation (three grains every four hours for a child of five years). The strength should be supported by alcoholic stimulants and beef-tea. *Tracheotomy* may become necessary at any moment; but the practice of *intubation* has yielded some good results, and it is an operation to which the patient's friends sometimes more readily consent.

Percussion.

Although more fully discussed in special works upon physical diagnosis, it is necessary here to sketch the theory of percussion and its practical application to medicine, especially to diseases of the chest. The subject is a difficult one, and it is rendered more so by the different views of authors, and by the confusion in the use of terms which have, in practice, been generally applied without regard to the scientific nomenclature and to the properties of sound.

While endeavouring, so far as possible, to reconcile these different views, an attempt will be made only to briefly formulate the simple characteristics of sound, which are concerned in the production of percussion-tones, and to point out what is practically necessary for their proper interpretation.

Definition of Sound.—"All sounds originate in impulse, or in vibrations of the particles of elastic bodies, which generally reach the ear through the medium of undulations in the air."

If a small steel rod be fixed at one end within a vice, and if the free end be struck, sound will result, and the vibratory movement will be visible; while, at the same time, a fine wire suitably attached to the vibrating extremity, and allowed to touch the smoked paper of a quickly-revolving cylinder, may be made to record these vibrations graphically.

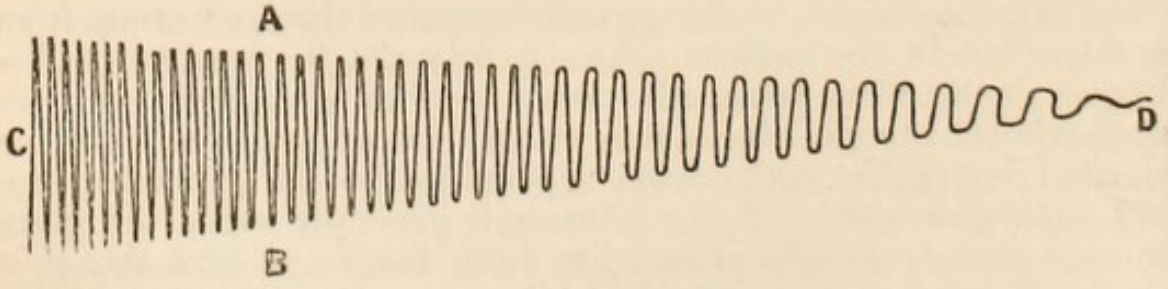


Fig. 12.—Sound-vibrations.

Tone depends upon the frequency of the vibrations. They must be rhythmical and sufficiently rapid; and the greater the frequency the higher the *pitch*. The *loudness* or *intensity* of the sound depends upon the *amplitude* of the vibrations—A to B. The *duration* of the sound is the time during which the vibrations last—C to D. The *undulations* in the air are set up by the alternate condensation and rarefaction of sheets or layers of air in contact with the vibrating body, and these layers propagate the impulse to adjacent layers.

The waves are, in free space, ultimately lost; but if the waves be confined they are reflected.

The undulations may be illustrated by observing what occurs in water when a pebble is dropped into it. That they are a phenomenon of translated motion and not of translated matter, may be shown by floating a cork upon the surface, when it will be seen to rise and fall with the waves, but is not carried on with them. If the water be confined in a trough, the waves are seen to be reflected from the sides.

No sound can be propagated in a vacuum, and the greater the density of a gas the stronger or more intense will the sound be. In water a sound travels four times faster than in air, and most solids conduct sound at a great velocity, and with greater loudness. Sonorous vibrations, however, are not readily communicated from gas to fluid, as will be considered when we come to treat of pleuritic effusion interfering with the conduction of breath-sounds.

Overtones — Harmonics. — When the string of a monochord is made to vibrate throughout its whole length, the sound produced is called the fundamental note. If a *node* be formed by touching the centre of the string, it will vibrate in two segments, and the note will be an octave higher because the two segments vibrate simultaneously at double the speed. In the same way *thirds*, *fourths*, &c., may be made to vibrate; and the corresponding higher tones of a fundamental note are called its overtones or harmonics. "Whenever a string vibrates throughout its whole length, it also vibrates in *halves*, *thirds*, *fourths*, &c.—the aggregate motion being exceedingly complex." Trained ears can detect the fundamental tone and its overtones; and upon the richness in number of these overtones the quality or *timbre* of a sound depends.* *Noise* is produced by a succession of sound-waves lacking periodicity, or by a number of tones which do not harmonise.

Vibrating Membranes.—Stretched membranes do not yield tone unless they are very tense and vibratile, or enclose an air-containing cavity. In the latter case the tone is produced by the conduction and rhythmical reflection of waves within the cavity—*i.e.*, *resonance.*

Resonance may be defined as "a sympathetic vibration resulting from the accumulation of small periodic impulses imparted by one sonorous body to another, *whose period or time of vibration is synchronous with it*"—*e.g.*, sounding-boards in stringed instruments. To procure a *clear* tone by rhythmical reflection or resonance, the size of the air cavity must be in relation to the flexibility of the vibrating membrane, or else they do not consonate, and the tone is more or less muffled or dull. This may be shown by holding a tuning-fork over a tall, empty jar, and pouring in water until the size of the resonant space or air column is such as to allow of the undulations of the air being in unison with the vibrations of the fork. The sound then becomes clearer and more intense.

The *intensity and pitch* vary with the size of the air column. A

* This will be well appreciated if the vowels be loudly sounded into an open pianoforte, with the *loud* pedal pressed down.

large cavity produces a tone strong or loud in intensity and low in pitch, while a small one yields a weak or soft tone which is higher in pitch. The pitch is also affected by the *tension*, *thickness*, and *density* of the vibrating membrane, so that if the membrane, say of a drum, be overstretched, the tone produced will be higher pitched, because of the quicker vibrations, and these will not consonate with the vibrations in the air-containing space within the drum, and, therefore, the tone will be less intense.

When a resonant space is partly open a smaller cavity will suffice to produce a resonant tone, and the size of the opening modifies the pitch—the wider it is, the higher the pitch, as may be demonstrated by filliping the cheeks and gradually opening the mouth.

In speaking of intensity, several terms have been used which call for explanation. The intensity may be said to be strong or loud, as opposed to weak or soft. Sometimes the word *dull* is used. The opposite of dull is *clear*, and hence clearness and dulness are terms often used in medicine in relation to the different degrees of intensity. These terms, however, are applied incorrectly, and they refer to the *quality* (timbre) rather than the intensity of a sound. A note struck forcibly upon the pianoforte will yield a sound strong in intensity or loud, and if struck gently, a soft or weak sound, which, however, may be perfectly clear. Long use has apparently sanctioned these terms in medical works, and hence in percussion, strong, loud, and clear are often applied to the intensity as opposed to weak, soft, and dull, the combination of the terms really including the *timbre*, along with the intensity. Physicians percussing the base of the lung often compare the *intensity;* and the same, when percussing the apices, will talk of high and low *pitch*.

Vibratile membranes enclosing air-containing spaces, which sometimes communicate with the external air, are the physical conditions concerned in the production of resonant percussion tones; and the two properties of sound which are the most important, are the *intensity* and the *pitch*. Irrespective of the force used, the relations of these conditions may be thus shown:—

		Intensity (and *timbre*).	Pitch.
Membrane, . . .	Flexible	Strong (clear)	Low.
	Tense	Weak (dull)	High.
Cavity, . . .	Large	Strong (clear)	Low.
	Small	Weak (dull)	High.
Opening, . . .	Narrow	Strong (clear)	Low.
	Wide	Weak (dull)	High.

It is possible to conceive of these relations of the membrane and cavity being disturbed as regards the intensity and pitch. A very tense membrane, for instance, which would otherwise give a high-pitched tone, enclosing a large cavity which would emit a tone clear

and strong. More forcible percussion, by increasing the amplitude, would then bring out the low-pitched tone by setting up vibrations of the contained-air, while light percussion would only give the higher-pitched note of the vibrating membrane.

Application.—In the application of the foregoing to percussion of the lungs, the vibrating membrane is not simple as in the case of the drum, but it is composed of layers of different density and tension, and must be held to include the component parts of the thoracic parietes, pleura, and lung tissues. The air-containing space also differs, inasmuch as its contained air is limited by multitudes of sacs, ranging in size and shape from the small air vesicles up to the large bronchial tubes.

With such a large number of resonant chambers, varying in size and tension, it is obvious that there can be no fundamental note, and the sound heard upon percussing a healthy chest is made up of a large number of tones which do not blend, and which have no definite or fixed relations. It is, in fact, a mixture of musical notes and noise.

The normal thoracic percussion-sound is produced by (1) the vibration of the thoracic parietes, pleuræ, and lung-tissues; and (2) by the vibration of the air contained in the pulmonary air vesicles and bronchial tubes.

Some authors consider the chest-wall the only part concerned in the production of the tone. Williams, adopting this theory, explains that the normal lung, filled with air, propagates the vibrations of the wall, and when the lung is consolidated it acts as a damper or *mute*, and hence the higher pitch, &c. This theory does not satisfactorily explain all the conditions. Other writers again deny that the chest-wall takes *any* part in the production of the sound. With regard, also, to the vibration of the air, Gee considers the vesicular elements too small to resonate, and that the percussion-tone is yielded by the larger bronchial tubes alone, the spongy lung-tissue interfering with its conduction. When, according to his view, consolidation takes place, the tone yielded is higher in pitch and clearer—the latter term being applied to the *quality* alone and not relating to the intensity.

The percussion-sounds possess *intensity* and *pitch*, and are *poor in timbre*, but in certain pathological conditions they may acquire this latter quality; and although the resonant chambers of the lung communicate with the external air, the *tissues* conduct the sounds.

Some writers consider the *intensity* of the percussion-tone as of little value, depending, as it does to a great extent, upon the force used and upon the state of the vibrating membranes. These authors consider the whole subject in relation to the *pitch*, the lung being normally a bad conductor unless consolidated, when it then yields quicker vibrations, and hence is higher in pitch.

According to Guttman "elevation of the pitch from *pathological* causes never occurs alone, unaccompanied by decrease in the intensity; the sound generally rises in pitch as it loses in clearness."

This dictum is practically, if not absolutely, correct, if we view the

percussion-tone as resulting certainly from the resonance of a great number of unequal chambers, but still striking the ear as a single sound, and all that is available for the comparison of the healthy with the consolidated lungs. The variations in the intensity and pitch in relation to this single percussion-sound would then be in harmony with the law already stated concerning resonant chambers, viz. :—that the smaller the air space the weaker is the intensity and the higher the pitch.

If, on the other hand, we attempt to regard the percussion-sound as a combination of sounds produced by *distinct and separate* resonance chambers all varying in size, *and each having its own intensity and pitch*, it is possible to conceive that the removal (by consolidation) of a number of the smaller and high-pitched elements (vesicles), while it diminishes the *intensity* of the percussion-tone as a whole, might *not* raise the pitch, as the larger elements (bronchi), if between the plexi-meter and the consolidation, would tend to keep the pitch low. As this can only apply to small consolidations—the larger ones diminishing the air spaces to such an extent as to diminish the intensity and raise the pitch, as a whole—Guttman's dictum may be accepted for practical purposes.

Although the membranes and resonant spaces are indissolubly associated in the production of percussion-sounds, they are here considered separately, in order to make clear the part each plays in the alterations of the intensity and pitch of the sounds.

I. **The Membranes.**—A. **The thoracic parietes**; B. **The lung.**

A. **The thoracic parietes** affect the intensity and pitch by the state of their tension and density. Increase of muscle on one side (labourers), deposit of fat, œdema, rigid and curved ribs, local thickening externally, or internally (pleura), and deep inspiration, will all diminish the intensity and raise the pitch, because the vibrations of the chest-wall are diminished in amplitude, and quicker in time. In these cases the percussion requires to be more forcible to bring out the resonant-tone, light percussion giving only a dull or flat note unless the air spaces beneath are caused to vibrate. On the other hand, the intensity is increased and the pitch lowered by muscular atrophy, emaciation, &c.

B. **The lung**, which may be regarded as the much thickened and altered internal layer of vibrating membrane, plays an important part in the production of the percussion-tone, as the state of tension of this organ is subject to variations which may affect the intensity and pitch. The lung in its normal state is slightly over-distended, and it has its peripheral surface to a great extent applied accurately to the external layers (thoracic parietes), and by virtue of its own elasticity it has a marked tendency to collapse. Should this occur from any pathological cause (as in early pleuritic effusion), the percussion-tone over it would become more intense and lower pitched because the lung-tissue has become more flexible. Should the collapse, however, become more complete, the lung then becomes more compact, ceases to yield flexible vibrations, and may now be regarded as a condensed body separated from the external layers of vibrating membrane by some pathological product, which may itself

(as in pneumothorax) form a resonant chamber; but where the lung remains in contact with the thoracic parietes, it now yields a note diminished in intensity and higher in pitch because of its increased density.* On the other hand, the tension of the lung may be increased (and with it the thoracic parietes) by full inspiration, and notwithstanding the increase in the volume of air within the lungs, the increased tension of the vibrating membranes diminishes the intensity and raises the pitch, unless more forcible percussion be used.

II. **The Resonant Spaces.**—As the object of percussion is to ascertain the amount of resonance, or state of the air-containing spaces, either in relation to lung disease itself or to diseases which affect the lung secondarily, it is obvious that this is the most important division of the subject.

A knowledge of the conditions which influence the state of the vibrating membranes is essential for the purpose of avoiding the sources of fallacy, and to indicate the necessity for more forcible percussion being used, when from changes in the thickness and tensity of the membranes they interfere with the production of the true resonant percussion-tone. Assuming for the moment that the tension of the membranes undergoes no change, or at least produces no antagonistic conditions, it may be stated that *the intensity of the percussion-tone is increased or diminished and the pitch lowered or raised, with the increase or diminution of the volume of air within the resonant spaces.*

In the practical percussion of the lungs, it will now be clear that for the proper interpretation of the percussion-sounds, the state of the three factors concerned in their production (parietes, lung, and resonant spaces) must always be kept in view. These may act *pari passu* to produce a tone diminished or increased in intensity and altered in pitch, but frequently they act in opposition, and the resulting tone is the balance or excess of the strongest element producing the tone. For example, the lung becoming relaxed in early pleuritic effusion tends to produce a tone low in pitch, even although there must now be *less* air within the air spaces—*i.e.*, the diminution in the tension of the lung-tissue more than compensates for the diminution in the volume of air, which would otherwise give a higher pitched note. Again, in deep, forced inspiration, the increased tension of the thoracic walls and lungs more than compensates the increased volume of air, and the tone becomes higher pitched. In *quiet* respiration, the air inspired tending to increase the intensity by its increased volume, is balanced by the increased tension of the lung and chest-walls, and hence the resonance remains practically the same.

The *pathological conditions* which alter the intensity and pitch of the resonant percussion-tones of the lung, are consolidations and

* The diminution in the volume of air which must necessarily result from collapse of the lung is not considered at present, although, of course, it also is the cause of diminished intensity; the object being, at present, to consider the different factors separately and independently.

exudations, collapse, œdema, excavations, &c.: while effusions of fluid or gas within the pleural cavity, enlarged organs, or the growth of tumours, affect it by their pressure. These conditions are described with the physical signs of their respective diseases.

It may be said of consolidations, &c., that they must be of sufficient *size* to alter the intensity and pitch of the percussion-tone, and that the more superficial they are the more readily are the differences noted. This latter fact may be due not only to a diminution of the size of the resonant spaces as a whole, but also sometimes to the consolidation, by its contiguity with the vibrating thoracic parietes, acting as a damper or *mute* to their vibration. Compensatory emphysema, also, may so surround a consolidation as to completely mask or neutralise the effect of percussion.

The tones produced by percussion are sometimes classified, as Tympanitic (stomach), Sub-tympanitic or Pulmonal (lung), Tracheal or Tubular, and Osteal. These are best learnt practically. Dr. John Wyllie's classification is as follows, viz.:—

Percussion.

(1) Hyper-Resonance.
- (*a*) Slight.
- (*b*) Marked.
- (*c*) Very marked (Tympanites). α High-, β Medium-, γ Low-pitched.

(2) Deficient Resonance.
- (*a*) Slight, comparative dulness.
- (*b*) Marked dulness.
- (*c*) Absolute dulness.

(3) Mixture of Dulness and Resonance, *i.e.*, a "Wooden or Boxy Note."

(4) Special Quality.
- (*a*) Cracked-pot sound.
- (*b*) Dulness with vibratile thrill to finger.
- (*c*) Bell sound, got with two coins and stethoscope.

Cavities.—These produce tones more or less resonant or tympanitic, but to do so the cavity must be at least the size of a pigeon's egg and superficial, with firm smooth walls surrounding the space.

If the cavity communicate with the external air, the pitch is higher when the mouth is open. Should it contain some fluid, changing the posture of the patient may modify the intensity and pitch, by the fluid altering the length of the vibrating column of air, thus—

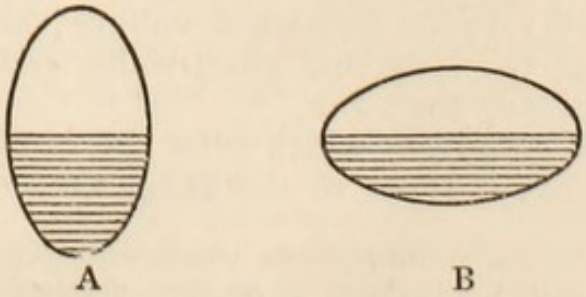


Fig. 13.—A, Cavity with fluid while in the erect position. B, Cavity while lying down.

A pyo-pneumothorax may affect the tone in the same way.

The *cracked-pot sound* (*bruit de pot fêlé*) is produced by strong percussion, during expiration, causing the sudden expulsion of air from a cavity or tube, the slight *hissing* noise attending the sound arising either at the outlet of the cavity or at the glottis. The sound may be imitated by loosely clasping the hands and knocking the back of one of them smartly on the knee.

The cracked-pot sound occurs in phthisical excavations, thoracic fistulæ, and in diseases associated with relaxed lung-tissue. It occurs also when forcible percussion is made upon the thorax of a screaming child.

Amphoric resonance (metallic ring, &c.) is a somewhat tympanitic note which is high in pitch. The membranes are tense and they probably produce over-tones of the fundamental or ground tone. It occurs chiefly in pneumothorax, but it may also be produced in smaller cavities with smooth walls. The combined auscultation and percussion with two coins renders the tone very distinct and metallic.

The *tracheal resonance of Williams* arises when the upper lobe of the left lung is collapsed or consolidated around the left primary bronchus. Opening the mouth raises the pitch.

Hydatid thrill is the tremulous sensation sometimes felt by the fingers on percussing a superficial tumour containing fluid.

Methods of Percussion.

The *immediate* method consists of tapping the chest directly with the tips of the fingers, but except as a preliminary to the mediate, or *to test the resonance behind the clavicles*, it is not much used.

The *mediate* percussion may be performed with a plexímeter and hammer, or with the finger acting as a plexímeter, and the stroke made (from the wrist) with a bent finger of the other hand. The latter is the usual and the best method, as the fingers adapt themselves to the irregularities of the chest, and are, at the same time, able to appreciate the *sense of resistance*, an important aid sometimes, in the diagnosis. The patient should keep the muscles of each side in the same state of tension, and should cross the arms well over when the back is percussed. The head should be held in the middle line when the apices are being examined. The percussion—with equal amount of force—should then be made symmetrically—*i.e.*, the corresponding parts should be compared as far as possible. As the stronger the force used the larger is the area over which the vibrations will be distributed, so will the tone produced vary in intensity. In percussion the force must be greater when it is desired to bring out the resonant tone through dense membranes, &c.; but it must be light when mapping out the edges of a solid which borders on a resonant space. The art of percussion is fully taught in the tutorial classes and clinical wards, but it can only be acquired by much practice.*

* The *lungs* extend about an inch above the level of the clavicles. The course of the anterior borders varies (*see* Medical Anatomy of the Heart, Fig. 2).

Auscultation.

The properties of sound, briefly sketched in relation to percussion, may all be applied to the sounds heard in auscultation of the lungs; but it is the *quality* (*timbre*) and the *duration*, which are generally of importance, the intensity and pitch being usually of secondary interest.

Origin of the Breath-Sounds.—A fluid—liquid or gaseous—passing along a narrow tube, does not produce sound unless an obstruction, constriction, or relaxation (by causing an increase in the calibre of the tube) set up vibration of the particles of the fluid vein.

In the respiratory tract such a constriction occurs at the glottis, and the vibration produced there during respiration is the cause of the breath-sound. These vibrations—like the vocal vibrations—are reflected down the windpipe, and they become weaker as they are conducted and diffused throughout an enormous number of bronchial tubes. According to Beau, Baas, and Gee, the glottis is the *only* source of the breath-sounds, and the loud *tracheal* murmur heard on placing the stethoscope upon the windpipe, the *bronchial* character of the respiration heard in the interscapular region, and the faint, breezy, *vesicular* murmur, heard best at the base of the lung posteriorly, all originate at the glottis, and are altered in character, according to the medium through which the vibrations are transmitted—the *vesicular* character of the murmur over lung being due to the bad conducting power of the spongy lung-tissue. This theory is supported by comparing the alterations in the character of the vocal vibrations, which are undoubtedly produced at the glottis, and which become muffled, or less clear, the further we proceed from the source of the sound in the respiratory tract.

The second theory in connection with the cause of the respiratory sounds applies only to the vesicular murmur. Chaveau, Niemeyer,

For practical purposes, it is sufficient to remember that in the mammary lines the lower borders are about the level of the *sixth* ribs; in the axillary lines, at the level of the *eighth* ribs; and in the back the lower borders are defined by lines sweeping round from the axillary points across the *tenth* ribs (about two and a half inches from the spine) to reach the spinous process of the tenth dorsal vertebra.

The lower border of the *liver*, in the mesial line, extends about two inches below the xiphoid cartilage; in the right mammary line it extends to the margin of the lower rib; and in the right axillary line it reaches the tenth intercostal space. The upper margin of the liver, *yielding absolute dulness*, is outlined by the lower border of the lung. The liver margins vary slightly with the position of the patient, and with deep respiration. The average area of hepatic dulness *in adults* is about *three* inches in the mesial line, *four* inches in the mammary line, and *five* inches in the axillary line.

The *spleen* lies parallel with the *ninth*, *tenth*, and *eleventh* ribs, of the left side, beginning one inch and a half from the spine, and extending as far as the left posterior axillary line. The upper part of the spleen is covered by the lower border of the left lung. Enlargements take place downwards and forwards, so that percussion from above, downwards, just in front of the left posterior axillary line (at the level indicated), is best calculated to detect an *early* increase in the size of the organ (see Fig. 14).

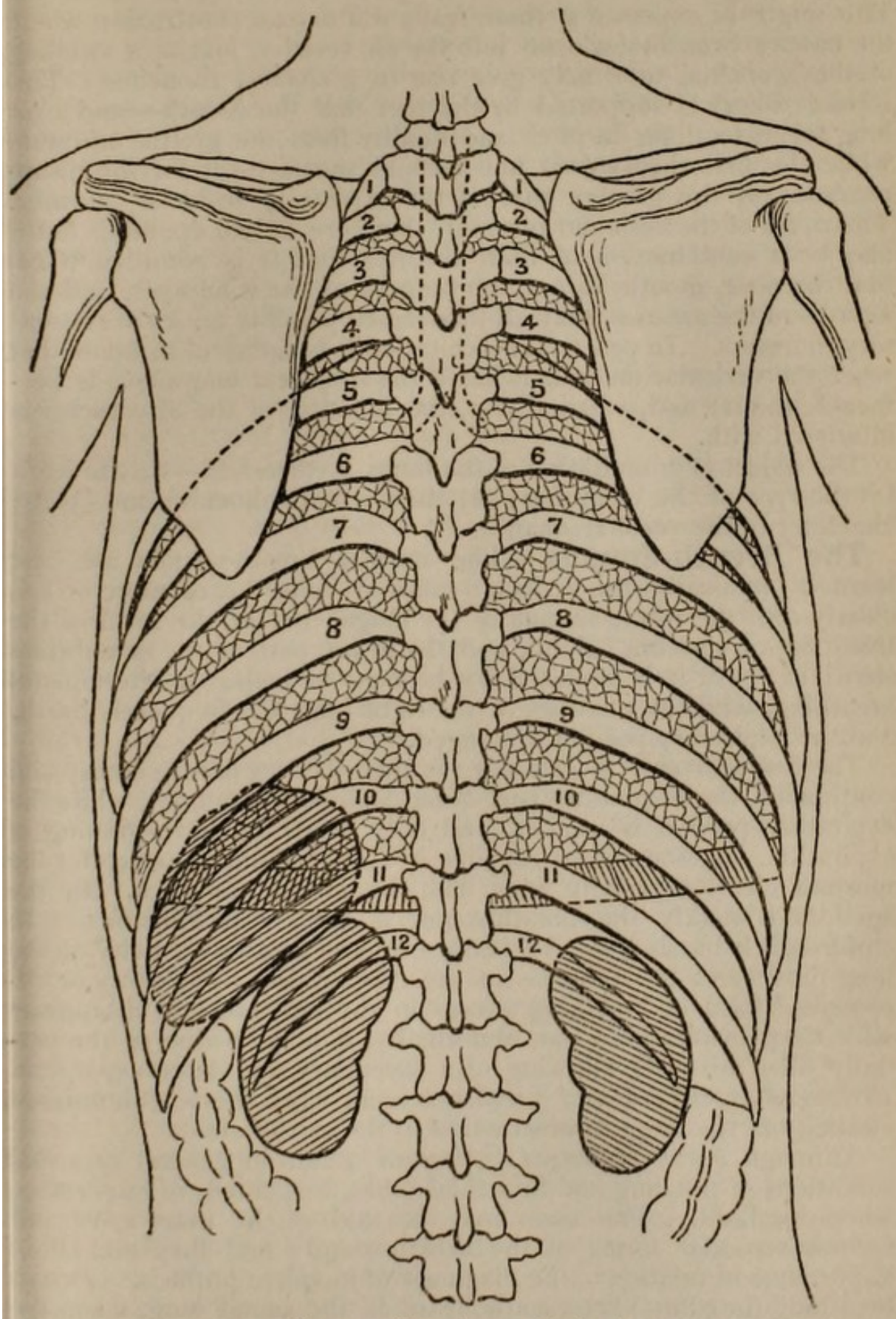


Fig. 14.—(Based upon Ferber.)

and others consider it to be produced by the vibration of the air particles entering the alveoli—*i.e.*, by numerous *stenosis murmurs*. This might be expected if there really did exist a constriction where the minute bronchioles open into the air vesicles, just as a swelling of the bronchial tube may give rise to a sibilant rhonchus. This second theory is supported by the fact that the breath-sound over lung seems to differ in pitch and quality from the glottic murmur; while the first theory does not account satisfactorily for the disappearance of the greater part of the expiratory vesicular murmur. The cause of the vesicular murmur, therefore, is still doubtful, but it may be a combination of both theories, and it is admitted (Gee) that the nose, mouth, throat, unevenness of the windpipes, and the mouths of the air-sacs, may all be deemed possible causes of respiratory murmurs. In practical auscultation it is sufficient to know that when the vesicular murmur is heard the subjacent lung-tissue is permeable to air; and, when absent, the function of the air vesicles is interfered with.

The object of auscultation of the lungs is threefold—viz., to learn (1) the type of the breathing; (2) the accompaniments; and (3) the character of the vocal resonance.

The Breath-Sounds.—The normal breath-sounds are best learned practically, the vesicular murmur being heard more or less clearly over the whole surface of the lungs; but at the level of the third dorsal vertebra behind, and the lower part of the manubrium sterni in front, it is replaced by broncho-vesicular (indeterminate) breathing, while at the apex of the right lung it is frequently harsh, and the expiratory portion prolonged.

The *vesicular type of breathing* is described as faintly breezy and continuous, the inspiratory part being audible throughout, while the expiratory portion is weaker, and only heard at the beginning of expiration. It sometimes requires a deep inspiration to render the murmur clear, especially when the chest-walls are thick. In the aged and weakly the breath-sound is *feeble* and indistinct. In children it is harsh (*puerile*), because of the greater elasticity of the lung-tissue and the thinness of the chest-walls. The *jerky* or *interrupted* form of breathing occurs in the nervous, and disappears with deep inspiration; but, abnormally, it may be due to the partially filled air-cells allowing of a secondary and later expansion. *Prolonged expiration and harshness* occur when there is diminished elasticity of the lung, or obstruction to the exit of air.

Although *harsh prolonged expiration* occurs in general catarrhal conditions of the lung and bronchial tubes, it is chiefly of importance when localised. The same may be said of the *interrupted* and *broncho-vesicular* forms of the breath-sound; and they are all of importance in relation to the diagnosis of incipient phthisis. *Puerile* breathing (in adults) is typically heard in the sound lung, when the other lung is collapsed by the pressure of a pleuritic effusion. The normal vesicular murmur—if there be no interference with its production by obstructions in the respiratory tract—may be rendered *feeble or indistinct* (distant) by enlarged organs and tumours, large

pericardial effusions, and pleuritic effusions, causing some collapse of the lung by pressure; and it may be *entirely abolished* by consolidations, large effusions, and in vesicular emphysema. The vesicular murmur, in some of these conditions, may be entirely replaced by harsh bronchial respiration, or it may be masked by accompaniments.

The *bronchial type of breath-sound* is not heard in the healthy chest, because the bronchial murmur being produced at the glottis, the spongy lung-tissue is a bad conductor of the sound. The character is harsh and guttural ("ch"), the inspiratory and expiratory portions being equal in duration, and with a short pause between them. Irrespective of the energy of the breathing, and assumiug that the bronchi are unobstructed, the intensity of the bronchial murmur depends upon the extent of the morbid condition (consolidation), and whether superficial or not.

Consolidations and compression of the lung, while obliterating the vesicular murmur, render the lung more homogeneous, and hence a better conductor of the glottis vibrations.

Excavations and cavities, with rigid and dense walls, give rise to bronchial breathing by the increased reflection of the vibrations. These conditions are produced by phthisis, pneumonia, collapse of the lung (effusions, pneumothorax, &c.), and new growths. Bronchial breathing is sometimes heard when a large pleuritic effusion exists, even although this tends to obliterate the bronchial tubes, and hence there is less sound to propagate. According to Guttman, when the fluid is abundant, bronchial breathing is only heard posteriorly where the lung is pressed close to the chest-wall. It may, however, be occasionally heard over the whole area of dulness, if the fluid is insufficient to obliterate the larger bronchial tubes. A tumour pressing upon a bronchus may cause stenosis, and originate a bronchial murmur; and a small solid in direct contact with a bronchus may conduct the sound with great distinctness.

Tubular breathing is a modified form of the bronchial breath-sound often heard in pneumonia. It is higher in pitch and less guttural in character ("hoo"). It has been suggested that when disease consolidates the air vesicles and bronchiola proceeding from a larger air tube, a current of air passing across the mouth of such a tube would produce the tubular tone—an effect concurrent with inspiration and expiration (Gee).

Cavernous breathing is the low pitched and reverberating breath-sound, not unlike the normal tracheal murmur. It generally indicates, but not always, the presence of a vomica.

Amphoric breathing may be low or high in pitch, and it resembles the sound made by blowing into an empty bottle. It is produced by air passing into a large cavity, and it occurs in large pulmonary excavations which have smooth walls, are superficial, and which communicate with a bronchus; and in pneumothorax it is well marked when there is a fistulous opening allowing the air to enter the cavity, and before there is complete collapse of the lung.

The *broncho-vesicular* or *indeterminate* breathing comes between the vesicular and the bronchial, and it partakes of the character of

each. It is heard normally in the right clavicular region of healthy men when breathing superficially, and at the level of the third dorsal vertebra behind, and at the lower part of the manubrium sterni in front, as already stated. It is (abnormally) due to insufficient expansion of the alveoli, or to obstruction by mucus; or it may result from the accompaniments masking the true respiratory murmur. When local, indeterminate breathing points to incipient phthisis; but it occurs in many other conditions.

Laennec's *souffle* or *puff*, Skoda's *veiled puff* (*souffle voilé*), and the *metamorphosing murmur of* Seitz are not of practical importance.

Accompaniments of Respiration—Râles.—In health there are no râles; but after a patient has been lying upon his back for some time a few crepitations may be heard at the base of the lung posteriorly. These, however, disappear with a few deep inspirations. At the apex, also, a clicking râle is often heard which a cough may remove. It is only when râles are persistent that they have pathological significance.

Râles occur in cavities and in the bronchi, and they are caused by the passage of air through fluid which, either by agitation or by originating bubbles, produce sounds appreciable by the stethoscope. Swollen mucous membranes may even produce them by vibration, but these sounds are generally *dry;* while sudden separation of the smallest bronchioles, when moist and cohering, is the accepted cause of the fine crepitant râles. The secretions may be serous, mucous, purulent, or sanguineous—the moist râles suggesting thin and watery secretions, while the *dry* sounds appear to be more tenacious and thick. Râles occur during inspiration chiefly, but also during expiration, and only rarely during the latter alone. A *post-expiratory* râle (Baas) is sometimes heard in large vomicæ. The intensity or clearness of the râles depends upon the energy of the respiration, and upon their size and seat (whether superficial or not). The ear soon learns to recognise whether they are transmitted or superficial, as in the former case they are more scanty and feeble (distant). Râles are not heard unless the bronchi are unobstructed.

Râles are frequently intensified by consonance in the cavity of the mouth, &c. They also become clear and high pitched when resonant, as occurs when the lung-tissue is sufficiently consolidated, or when associated with a cavity having smooth, dense walls, and which is superficial. The *metallic* character of a râle is due to consonance in a large cavity with smooth firm walls; but the râles may originate in the bronchi, and a pneumothorax or distended stomach may transmit them. Dry râles render the respiratory murmur harsh, and they lengthen the expiratory sound.

The râles may be thus classified:—

The *dry* (*rhonchi*)—

1. The high-pitched, *sibilant* or *cooing* rhonchus.
2. The medium-pitched; and
3. The low-pitched, *sonorous* rhonchi.

The *moist* râles—

1. Fine crepitations.
2. Medium sized, *sub-crepitant*, *fine mucus*, and *clicking râles;* and
3. Coarse, *bubbling*, *gurgling*, or *cavernous* râles.

The dry rhonchi, and the medium sized and coarse moist sounds, occur chiefly in bronchitis, in catarrhal conditions, and in phthisis. The fine crepitations are heard in pneumonia, in pulmonary œdema, and in collapse of the lung with slight catarrh. The fine crepitations are like the sounds produced by rubbing a few hairs between the fingers, close to the ear. The other sounds need no description, and they are best learned by experience. It is possible to find two or more different rales combined, as in œdema—the fine crepitations mixing with the coarser râles in the bronchial tubes. Hairs upon the chest may simulate fine crepitations; but moistening the chest with water will prevent these.

Pleuritic Friction.—The moist pleuritic surfaces, in health, glide over each other and produce no sound. When they become roughened by inflammatory deposit, and if no effusion separate the surfaces, a friction sound is produced. It may only be the slightest rub, or it may be well marked, and like the creaking of leather. The friction may often be felt (friction fremitus). The sound is best heard at the acme of inspiration, and it is not modified by coughing, and it is usually more localised than any of the intra-pulmonary sounds. Pressure with the stethoscope sometimes intensifies the friction sound. Peritoneal friction, and friction at the shoulder joint, &c., are sources of fallacy. Pericardial friction may be differentiated by instructing the patient to hold his breath during auscultation. Pleurisy, fracture of the ribs, &c., and inequalities of the pleural surfaces (as by tubercular and cancerous deposits) are the chief causes of pleuritic friction.

Hippocratic succussion is the splashing sound heard on shaking the patient during immediate auscultation of the chest. It frequently can be heard in pyo-pneumothorax, &c., and sometimes even in large cavities when the secretions are thin.

The Vocal Resonance.—The vibrations produced in speaking are conducted and diffused throughout the bronchial tubes, and the spongy lung-tissue, being a bad conductor, renders the sound indistinct. Practice alone can teach the normal resonance, and it varies in individuals, and depends upon the strength of the voice and the thickness of the chest walls. It is usually louder upon the right side, owing to the right bronchus being larger. The vocal resonance is sometimes better tested by *immediate* auscultation—the ear being applied directly to the chest, a soft handkerchief intervening.

The *vocal resonance* may be—

Impaired :	Normal.	Increased :
(*muffled*)	...	(*Bronchophonic*)
(*absent*)	...	(*Pectoriloquous*)
...	...	(*Ægophonic*)
...	...	(*Amphoric*)

Assuming that the bronchial tubes are not blocked and that there is no interference with the conduction of the vibrations, the vocal

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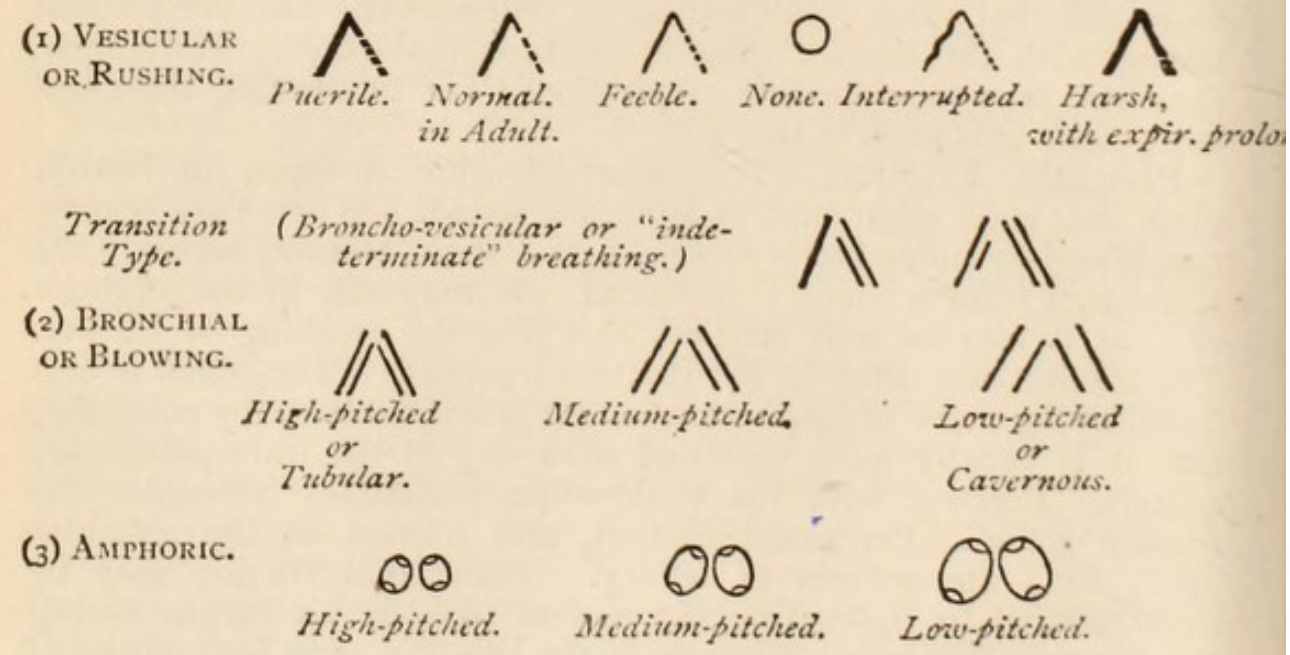
Types of Breathing.



Accompaniments.

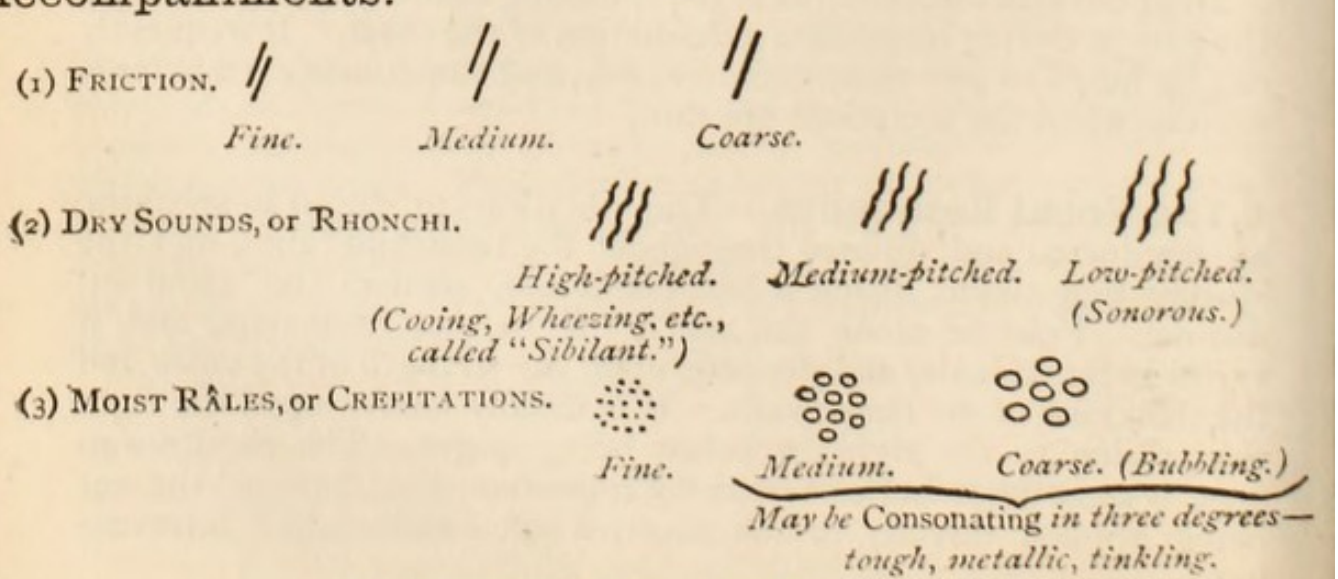


Fig. 15.—Breath-sounds and Accompaniments.

resonance is impaired (muffled) or absent, when the lung from any cause is separated from the chest-wall (pleuritic effusions—liquid or gaseous—tumours, &c.). This is not *always* the case in pleurisy with effusion, as occasionally even the whispered voice may be heard over the dull part. The vocal resonance is intensified by whatever makes the lung approach nearer to simple solid, or simple air, as in all *consolidations* (pneumonia, phthisis, &c.), and especially is it intensified over *cavities*. *Bronchophony* is that clear resonance which is generally compared with that normally heard in the inter-scapular region. It has sometimes a *sniffing* character. *Pectoriloquy* is very clear bronchophony, heard usually over a limited area, and it is reverberating in its quality; but it does not always indicate a cavity. *Ægophony* is the bleating (Punch) voice sometimes heard in pleuritic effusions. *Amphoric* resonance is metallic in character, and it resembles the blowing or speaking into a large empty bottle, the sounds having a distinct echo. It occurs chiefly as a symptom of pneumothorax, but it may occur in large cavities.

The character of the vocal resonance (clearness) is more important than its actual *loudness*, and it sometimes is necessary to test the whisper over cavities, &c., as "the ground-tone of the voice drowns the articulated over-tones." The thoracic resonance may also be tested by the cough and cry—the latter especially in children. Dr. Wyllie graphically represents the breath-sounds and accompaniments, as on the preceding page.

CHAPTER IV.

DISEASES OF THE RESPIRATORY SYSTEM—

Section II.

Contents.—Pertussis — Asthma — Hay asthma — Acute bronchitis—Fibrinous bronchitis—Pulmonary collapse (Atelectasis)—Catarrhal pneumonia — Congestion and œdema of the lungs — Croupous pneumonia — Hæmorrhagic infarction — Chronic bronchitis—Cirrhosis of the lungs—Emphysema—Fœtid bronchitis—Bronchiectasis—Gangrene of the lung—Phthisis, and miliary tuberculosis—Cancer of the lung—Hydatids—Pleurisy—Hydrothorax—Pneumothorax, hydro-pneumothorax, and pyo-pneumothorax — Pleurodynia — **The classification and diagnosis of the diseases of the pulmonary organs**—Causes of hæmoptysis.

Pertussis — Whooping-cough.* — The **pathology** is still doubtful. Some authors consider it to be of the nature of a neurosis affecting especially the vagus nerves; others suggest that it is due to pressure upon the nerves by swollen bronchial glands; and others

* Before reading the following diseases of the pulmonary organs, the student should revise the classification and the notes upon the same on p. 114.

again believe it to be a catarrh of the mucous membranes, and probably—like other infectious diseases—associated with a special organism. Rosenthal has shown that irritation of the superior laryngeal nerve (internal branch) produces relaxation of the diaphragm, spasm of the glottis, and convulsive expiration; and Beare believes that there is always inflammation of the windpipe just above the vocal cords. The age at which whooping-cough is common is from two to ten years; but it frequently occurs in adults. The period of incubation has been proved to extend to fourteen days.

The **symptoms** are at first those of ordinary catarrh, with a dry cough, and fever, and these symptoms may extend from a few days to three weeks or more before the characteristic spasmodic cough is developed. During this stage it may be impossible, in single cases, to make any positive diagnosis, but a dry incessant cough, which is worse at night, should always suggest the possibility of whooping-cough when it occurs in a child.

The *spasmodic stage* is reached when the cough becomes paroxysmal. It consists of a number of short expiratory puffs, succeeded by a long in-drawing crow or "whoop," the attack lasting about one minute, but sometimes even a quarter of an hour. It may be started by imitation of another child suffering from pertussis, by indigestible food, or inhalation of cold air, &c. There is often much distress. The expression is anxious and frightened, the face is swollen and often cyanotic, the eyeballs protrude, and the child usually clings to a chair or table during the violence of the shocks. The conjunctivæ are frequently ecchymosed, and hæmorrhage from the nose is common, while the attacks may terminate in expectoration of viscid mucus, in vomiting, or in general convulsions in very severe cases. Hernial protrusions are common. Small ulcerations at the frænum of the tongue sometimes appear at the third to the fifth week. Albumen and sugar are found in the urine frequently. The physical signs in the chest, in uncomplicated cases, are those of bronchial catarrh, viz.:—Sonorous and sibilant rhonchi, with coarse crepitations; prolonged harsh expiration; and no dulness upon percussion. The heart's action is very rapid during the paroxysm. There is little or no fever, and the child, during the spasmodic stage, appears fairly well between the attacks. This stage may continue from about four or six weeks to even two or three months. The whoop gradually disappears, but it may recur during the convalescence, with any fresh catarrh; and even, in some cases, it may occasionally be heard as long as a year after the attack. During the convalescence—sometimes described as a third stage—the symptoms and signs are those of ordinary catarrh, if no complications have supervened. The secretions are muco-purulent, the cough is frequent, but it is not spasmodic unless the patient relapse, which is very common.

The most frequent complications are bronchitis, catarrhal pneumonia, and collapse of the lung. Sometimes bronchiectasis is produced; while in exceptionally severe cases emphysema, and even extravasation of air into the pleura (pneumothorax), &c., may result

from whooping-cough. The cerebral complications (convulsions and coma) are the result of mechanical obstruction to the circulation. Cerebral hæmorrhage may result from rupture of a blood-vessel. In healthy children the *prognosis* is usually favourable if the disease be not severe; but it is serious in the very young, and death is very frequent—either the result of complications or of simple asthenia. Death is not common during the paroxysm, except in the very weakly and strumous; but in these it is always possible from syncope, or from intracranial hæmorrhage, &c. Whooping-cough seldom if ever recurs, and it is believed in some very mild cases it may run its course without any characteristic "whoop" or spasmodic stage.

The **treatment** of the first stage is that of ordinary catarrh. The child should be kept in a warm room and ℞ 14 or 15 should be prescribed, and the chest should be rubbed with Bowe's liniment, or a linseed poultice may be applied, if there be much bronchial catarrh. The diet should be simple and nourishing, and consist chiefly of milk. The bowels should be kept regular in action. During the spasmodic stage, bromides may be given, in a little syrup of Tolu, in doses according to the age. ℞ 16 and 17 are also useful remedies. Syrup of codeia is also a powerful and efficient drug in severe cases. Inhalations of chloroform for very severe spasm may sometimes be necessary. Quinine is said to be serviceable. Emetics are useful when there is much mucus obstructing the bronchial tubes. Very dilute carbolic sprays (1 per cent.) into the throat, are recommended. In lingering cases tonic treatment with a change to the seaside may complete the cure. The treatment of the complications is given under their respective diseases.

Asthma.—The term *asthma* is frequently applied to diseases associated with breathlessness. Hence we have *cardiac asthma* when the dyspnœa of heart disease is marked, and *gastric asthma* when flatulence in dyspeptic conditions produces embarrassed breathing. The ordinary dyspnœa which accompanies emphysema and bronchitis is sometimes loosely spoken of as an asthma. "Asthmatic bronchitis" is a convenient term, clinically, as true spasmodic asthma is invariably followed by irritation of the bronchial tubes; or on the other hand, and far more commonly, bronchitis is the primary disease and is the cause of the reflex spasm. *True spasmodic asthma*, however, might be classified with the nervous diseases, as pathologically it is "a *neurosis* of the breathing apparatus"; but practically it is best considered with the diseases of the respiratory system.

The symptoms are produced by sudden spasm of the bronchial muscles and diaphragm, and the laryngoscope has revealed that deep congestion of the upper part of the windpipe takes place during an attack. Leyden considers certain small crystals, found in the expectoration, to be the cause of the asthma by the irritation they produce upon the terminal filaments of the vagi. In subjects predisposed by inheritance, gout, &c., to attacks of asthma, certain odours, dust, pollen, and indigestion and flatulence, are all exciting causes; and nasal polypi are also known to be a cause of asthmatical

seizures. Long continued whooping-cough, chronic bronchitis, and severe cases of measles, by the depression produced in the respiratory organs, are believed to produce conditions favourable to the development of asthma.

Asthma frequently alternates in the same person, or in other members of the same family, with other nervous diseases and with skin eruptions, the most common being urticaria.

The **symptoms** come on very suddenly, and generally during the early morning hours; but in those subject to attacks there may be warning, as by ordinary coryza, bronchial irritation, general *malaise*, or indigestion and acidity, for a few hours previous to the seizure. There is a sudden feeling of constriction about the chest, and the patient struggles for air with the appearance of great anguish. Beads of sweat stand upon the forehead, the face is cyanosed, and the eyeballs are widely staring. This is accompanied by loud *expiratory* wheezing. The spasmodic contraction of the diaphragm keeps the chest in the position of forced inspiration, and percussion of the lungs yields a clear note (tympanitic or *band-box* sound) which often extends an inch or more *lower* than the normal area occupied by the lungs. The expiration is greatly prolonged, and the vesicular murmur is obscured by sibilant and sonorous râles, and, later, by moist crepitations. The spasmodic condition may last from a few minutes to several hours, when relief generally follows the free eructation of gas, or the expectoration of thin watery mucus; or the attack may terminate in the discharge of a large quantity of pale-coloured urine of a low specific gravity. Very often the bronchitis lasts for some days with frequent exacerbations of the spasmodic asthma.

Frequent attacks of asthma and bronchitis ultimately may lead to emphysema of the lungs, with dilatation of the right cavities of the heart, and dropsy; but the *immediate* prognosis is usually hopeful, death being rare, unless the attacks be associated with grave organic disease of the heart. The attacks are sometimes mild, and at other times serious; and they may leave the patient altogether, especially when occurring in the young. With older patients the prognosis should be more guarded, and it must be considered in relation to the history of the case, the number of the previous attacks and their effects. It must not be forgotten that cerebral hæmorrhage is possible during an attack.

The **diagnosis** is not usually difficult. Œdema glottidis, paralysis of the vocal cords, aneurisms pressing upon nerves, or stenosis of the trachea, all produce dyspnœa; but it should be noted that in asthma the dyspnœa is *expiratory*, and laryngoscopic examination may serve to differentiate the former diseases. Cardiac dyspnœa, emphysema, and bronchitis have other physical signs; but in the latter disease asthma may occur as a complication, or, again, the bronchitis may be the result of a previous asthmatical seizure. The history of the *onset* is most important, and especially is this the case in children, for, as Trousseau has pointed out, many cases of supposed broncho-pneumonia are really cases of true spasmodic asthma, as shown by their rapid recovery and their frequent recurrence.

The family history is also important as regards the diagnosis of asthma in children.

[**Hay asthma** or **hay fever** is sometimes described as a separate affection, the symptoms being caused by the irritation of the mucous membranes, by pollen, during the hay season. There is severe coryza with frontal headache and general *malaise*, and sometimes the irritation extends to the bronchial tubes, producing slighter forms of asthma. It is peculiar to certain individuals, and apt to resist treatment; but a short sea trip always results in recovery.]

In the **treatment** of true spasmodic asthma, *four* to *eight* drops of a 1 per cent. solution of nitroglycerine, in water, is the most effectual remedy for relief of the spasm in adults, care being taken to watch the heart's action. The dose may be repeated in three or four hours if there be no depression from its use, and it may be continued cautiously, at regular intervals, should the paroxysms render it necessary. Three to five drops of nitrite of amyl dropped upon blotting-paper and inhaled, may be tried in place of the above. Fifteen to thirty grains of chloral hydrate, with forty grains of bromide of potassium, may be given at the same time, or in place of the nitroglycerine, when it is deemed unadvisable to use it; or one-twelfth to one-fourth of a grain of morphia may be used hypodermically. Inhalations of ether or chloroform may be tried, and large doses (fifteen grains) of iodide of potassium are recommended. Stimulants are often necessary, and digitalis may be used along with these remedies when the heart threatens to fail.

Strong coffee to drink, or the smoking of cigarettes of stramonium, belladonna, &c., or the burning of nitre-paper, sometimes afford relief; and ten drops of either ipecacuanha wine, or of tincture of lobelia, in water, every five minutes, until some nausea is induced, may serve to lessen the feeling of oppression. The treatment after relief of the spasm is much the same as in bronchitis. The diet is important—light and easily digested animal food being ordered, while starchy foods, sweet stuffs, and even milk, should be avoided. Arsenic, quinine, and iodides are indicated in the treatment of chronic cases, between the attacks.

For *hay asthma*, antiseptic douches or sprays may be tried, if the sea-side or a short sea voyage is unattainable. Painting the mucous membrane of the nose with a solution of cocaine may be of service, and in obstinate cases, destruction of the Schneiderian membrane, by the electric cautery, may effectually cure the disease.

Acute Bronchitis. — **The pathological changes** in acute bronchitis begin with redness and swelling of the mucous membrane of the air-passages. The bronchial tubes soon become filled with mucus and muco-purulent secretions. In the *early* stages the expectoration is made up of ciliated columnar epithelium and of mucous corpuscles; but, later, it consists of mucus holding in suspension large numbers of small, round, or oval-shaped cells, shed from the lower epithelial layers, and undergoing fatty degeneration.

The basement membrane becomes swollen and œdematous, and the mucous glands are stimulated to increased secretion. There is accumulation of cellular structures and leucocytes within and around the inner fibrous coats of the bronchi, and the bronchial glands, in the immediate neighbourhood, become enlarged. Should recovery take place, the swelling of the basement membrane disappears, and the epithelial layers are replaced.

The causes of acute bronchitis are numerous. It is most common in damp and changeable climates, and in such, the feeble, aged, or very young are readily attacked. While exposure is the commonest cause, acute bronchitis arises as a secondary change in a

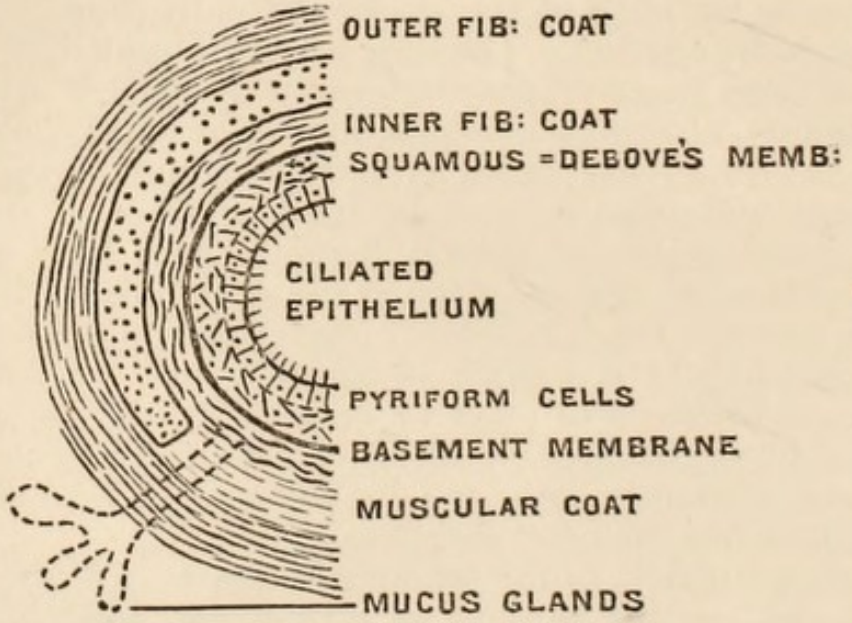


Fig. 16.—Diagram of bronchus.

great variety of diseases—*e.g.*, the exanthemata, rickets, chronic valvular disease of the heart, Bright's disease—all of which require to be remembered in the diagnosis. Irritating vapours in certain trades, dust, and pollen, may also set up the disease.

The **symptoms** vary according to the seat. If the trachea and larger tubes only be involved the symptoms are not so urgent as when the smaller tubes are affected, nor is the prognosis then so grave. In the first case—sometimes called *tracheo-bronchitis*—there is not much fever, and the chief symptom is a tickling cough with pain over the sternum. In the very mild cases, only the other symptoms associated with an ordinary coryza may be present; but if the bronchial tubes be still further affected there may be a considerable rise of temperature, greater frequency of the pulse, and some dyspnœa. The latter is chiefly *expiratory*. The accumulation of mucus in the tubes gives rise to râles which often may be heard at a distance from the patient. The face is flushed, and in extreme cases there is cyanosis; but the latter symptom is more marked in the form involving the smaller tubes—*capillary bronchitis*. In bronchitis of the larger tubes the cough is very frequent and loud, and the expectoration varies from simple glairy grey-white mucus

to muco-purulent secretion, and sometimes almost pure pus. It may be scanty at first, but it soon increases, while it may be so very tenacious as to allow of the spittoon being reversed without its escaping. It becomes more watery under treatment, and in the later stages, should recovery take place. The tenacious character of the sputum, when in quantity, gives rise to a feeling of suffocation, hence the old name of "suffocative catarrh." The cough and suffocative sensations are worst in the morning when the accumulation of mucus interferes with the respiration. The patient often requires to be propped up in bed to allow of easier breathing.

The *capillary* form of bronchitis is common in children, and it is generally ushered in by severe chills or rigors, frequently repeated, and the temperature rises as high as 104° Fahr. The head and upper part of the body may be covered with perspiration. The dyspnœa is the marked symptom, and the pulse is extremely rapid, but often full. The expectoration may be absent in the early stages of capillary bronchitis; but it should be noted that in children the secretions are swallowed, if any be coughed up.

Inspection of the chest, in the severe forms of bronchitis with dyspnœa, reveals considerable indrawing of the walls, particularly in the epigastric and hypochondriac regions; and in children all the lower ribs are seen to be drawn in with the difficult inspiration. There is no change in percussion over the lungs in uncomplicated cases, but if emphysema, collapse of the lungs, or broncho-pneumonia be present, the percussion tones may be impaired.

In auscultation, the breath sounds are altered. The vesicular murmur may be harsh, indistinct, or obscured by râles, or absent if the bronchial tubes be blocked with mucus. In the latter case coughing may develop the breath sound. It is prolonged in its *expiratory* part. The accompaniments are the coarser forms of râles, with sonorous and sibilant rhonchi, the vibrations of which may sometimes be felt through the chest-wall. Coughing may alter the character of the accompaniments. The râles are general, and are not localised, a point of some importance in relation to the diagnosis of phthisis. They do not consonate. (In acute general tubercular disease of the lungs, the râles are diffuse and consonating). The vocal resonance remains normal.

Simple acute bronchitis may run a course of fourteen days or more, and then terminate in recovery. It may, however, in extreme cases, be terminated by death in two or three days, especially in the aged and feeble, and the very young.

The **prognosis** will depend upon the severity of the attack and the presence or absence of complications. These are chiefly collapse of the lung, emphysema, broncho-pneumonia, and sometimes pleurisy. The disease frequently becomes chronic; but if it run to a fatal issue, the breathing becomes more and more shallow and convulsive seizures may terminate the case.

The **diagnosis** of acute bronchitis is not difficult; but it is sometimes not so clear whether the bronchitis be primary or secondary—or whether it be simple or complicated. Careful examination of each

case is necessary, keeping chiefly in view the exanthemata, rickets, chronic valvular disease of the heart, Bright's disease, tuberculosis, and scattered malignant nodules; while collapse of the lung, emphysema, catarrhal pneumonia, pleurisy, and croupous pneumonia are complications which should be noted and excluded.

Fibrinous Bronchitis or **Plastic Bronchitis** is a rare disease, consisting of the formation of a tough white-grey membrane within the bronchial tubes. It is not an extension of croupous membrane from the larynx, although similar in character. The symptoms are at first like acute bronchitis, but there is excessive dyspnœa, and it is not usually diagnosed until the expectoration of the casts reveals the nature of the disease. The character of the cough suggests stenosis of the trachea, or a foreign body. The prognosis is very grave.

Pulmonary Collapse (Atelectasis).—*Atelectasis* is the condition of uninflated lungs (or any part of them) in the new-born infant.

Pulmonary Collapse is a *secondary* condition, and it results from the primary disease so affecting the lobules that they are deprived of air. Gairdner's theory is, that pellets of mucus within the bronchial tubes may act as ball and socket valves, admitting no air during inspiration, but allowing of its escape during expiration. As this theory does not explain the entire absence of air within the collapsed lobules, and as Lichtheim has shown that complete absorption takes place after plugging the bronchial tube, Fagge thinks "that whenever even a small part of the organ fails to be acted upon by the forces which are concerned in inspiration, its elasticity brings about a total collapse of its substance, notwithstanding that the tubes which serve it may be patent." This theory serves to explain how acute bronchitis in a child produces collapse—the drawing in of the chest-wall and sternum causing collapse of the anterior borders of the lungs, and yet no plugging of the tubes is present.

Collapsed lung is very like red hepatisation. The lung is brown-red in colour. A section, however, is dry, smooth, and homogeneous, and the portions affected are depressed below the surface of the healthy lung. The collapsed portions are more dense and do not crepitate, and they may be inflated from the bronchial tubes. Sometimes the collapsed portions are œdematous and serum may be expressed. *Carnification* of portions of the lung is the result of continued pressure upon the collapsed lung, as in pleuritic effusions, tumours, &c.

Any obstruction of the bronchial tubes by tumour growths, enlarged glands, or aneurism, may produce collapse; while it is, of course, associated with all large pleural and pericardial effusions, and tumours which displace the lung. In acute bronchitis and in whooping-cough collapse is only common in children. It may only be temporary; and the important symptom is the increase of the dyspnœa superadded to the previous symptoms. Percussion will only reveal dulness when the collapsed portion is sufficiently large,

and then, also, the other physical signs of consolidation will be present. Collapse of the lung may terminate fatally, or in recovery, more or less complete. If recovery be imperfect, it may leave damaged lungs with emphysema, or it may lead to caseous pneumonia and phthisis in those of the strumous constitution. Inhalations of compressed air, and active expansion of the chest by gymnastic exercises, should be practised as early as possible.

Broncho-pneumonia: Catarrhal Pneumonia: Lobular Pneumonia.—When an acute bronchitis extends back to the air vesicles, a catarrhal pneumonia is produced, an event which is somewhat rare in adults, but is common in childhood, especially after measles, other fevers, and whooping-cough. The *sub-acute* form, however, or "cold settling down in the chest," is common enough in adults of delicate health, and in the aged or feeble. It may lead to phthisis by the subsequent caseation of the inflammatory products. The lung, on *post-mortem* examination—besides showing some lobules in a state of collapse, and others compensatorily dilated, with likewise the signs of bronchitis already described—is seen to have grey-white, semi-solid patches, slightly elevated and scattered throughout its substance. They are not nodular, and, when squeezed, they emit a yellow muco-purulent discharge. Microscopically, the alveoli are seen to be infiltrated with large round cells, which are very granular, and have one or more nuclei. They are derived from the epithelial lining of the air vesicles, and they are in various stages of proliferation. The process closely resembles that of acute bronchitis, Debove's membrane in the bronchial tube running continuously into the alveolus. These inflammatory products may become absorbed if recovery take place; but very frequently, in bad hygienic conditions, the disease becomes more or less chronic, and caseation of the nodules ultimately leads to the phthisical condition. There are often small patches of pleurisy present, especially if the patches of catarrhal pneumonia be superficial; but, according to Hamilton, there is no pleurisy in the simple acute catarrhal pneumonia, but almost invariably is it present in the second stage, viz., of *caseation*.

In children, rickets and bad ventilation, &c., predispose to the disease; while in adults probably secondary irritation is the cause, as from fœtid purulent matter from the bronchial or nasal mucous membranes being drawn along the bronchial tubes. In children the violent inspiratory efforts drawing into the alveoli the morbid inflammatory products of bronchitis, has been suggested by Hilton Fagge as a highly probable explanation of their greater liability to catarrhal pneumonia. The diagnosis is often uncertain; but if superadded to the symptoms and physical signs of acute bronchitis, there be a sudden rise of temperature—especially if a finer râle be now detected—it may be surmised that the alveoli have become affected by the general bronchial catarrh. If several contiguous lobules be involved, then the physical signs of consolidation—dulness on percussion, bronchial breathing, and bronchophonic resonance—may be made out. The pulse is increased in frequency, and in children it may be very rapid (150 to 200). The cough may cease, the child

may become very restless, the breathing shallow, and the case finally terminate in delirium, coma, and death with convulsions. The disease may simulate tubercular meningitis; but it should be noted that in children pneumonia is often ushered in by convulsions in the very earliest stages of the disease. The *prognosis* is less grave after measles, but even this depends upon the constitution and strength of the subject. Catarrhal pneumonia may terminate fatally within a few days, or it may run a course of from two to three weeks, and still ultimately recover. The sub-acute and chronic forms of catarrhal pneumonia are further considered with phthisis.

Congestion or Hyperæmia of the Lungs.—**Pathology.**—The lung is dark-red, heavier, and firmer, and it crepitates less than the normal lung. It looks like the spleen when severely congested. The bronchi are filled with bloody, frothy serum. The capillaries are swollen, and compress the alveoli. There are numerous extravasations throughout the lung-tissue.

Œdema of the lungs usually accompanies congestion. The exuded serum infiltrates the interstitial connective-tissue and the alveoli; and if this be in sufficient quantity, the lung may pit on pressure. The serum exuded is red in colour, but if œdema result from general dropsy, it has the usual pale-straw colour. Œdema occurs most readily at the bases of the lungs. In chronic cases of congestion with œdema, the lung may also show the appearances of *brown induration.*

The **causes** of congestion and œdema are the inhalation of cold air or irritating vapours; the ingestion of cold drinks when the body is warm and perspiring; diseases, as pneumonia, collapse, &c., of the lung, producing congestion of other parts; and the passive congestion due to valvular disease of the heart or to simple dilatation. Bright's disease, and increased force of the heart's action from any cause, may also produce a congestion of the lungs.

The **symptoms** are slight fever, with a feeling of heat and oppression in the chest, more or less dyspnœa and cyanosis, with a quick but full pulse. When œdema is superadded, these symptoms are much exaggerated—especially the dyspnœa. Cough, with frothy expectoration streaked with blood, will also be present. The physical signs consist of the presence of sub-crepitant and fine râles which obscure the vesicular breath-sound; and should there be œdema, there will be the signs of consolidation at the bases, as dulness upon percussion, bronchial breathing, and bronchophony. An acute congestion may terminate fatally in a few hours or days, death being due to carbonic acid poisoning. There is then gradually increasing somnolence, less and less effort made to expectorate, and lastly, coma. Recovery may take place by resolution. The passive forms of œdema, occurring in chronic valvular diseases of the heart and Bright's disease, develop more slowly. They may, however, prove *rapidly* fatal.

Croupous Pneumonia: Lobar Pneumonia.—**Pathology.**—The first stage—*congestion*—lasts for twenty-four or thirty-six hours, and the appearances are the same as described in con-

gestion of the lungs. The second stage—*red hepatisation*—consists of the consolidation of a lobe or two, and sometimes even the whole lung. It is usually the lowest lobe, and most frequently that of the right side, which becomes affected. The diseased part is brick-red in colour, and has the appearance of liver-tissue. It is tough, non-crepitant, and elastic, and it does not collapse. It sinks in water. The affected parts present some of the appearances of œdema of the lungs, but they exude no serum on pressure. The non-solidified parts of the lung, however, may exude serum, as they are deeply congested and frequently œdematous. The air vesicles are filled with fibrinous lymph, and there is almost always an exudation of lymph upon the pleural surface adjacent to the inflamed lobe. Microscopic examination reveals the air vesicles filled with fibrin, leucocytes, blood corpuscles, and desquamated epithelial cells. Blood-clots are frequently formed in the main stems of the pulmonary artery, and they seem, sometimes, to be the cause of death.

The third stage—*grey hepatisation*—succeeds the red in a few days, the colour of the affected lung being now *grey-granite*. The lobe is not so tough, and if squeezed a purulent secretion is forced out. The fibrin becomes granular, and the leucocytes undergo fatty degeneration. The colour is due to this, and also to anæmia. The exudation becomes more serous, and absorption takes place in those cases which terminate in recovery. Rarely, gangrene of the lung and abscess may follow acute croupous pneumonia. More frequently, in addition to pleurisy, there is effusion of lymph elsewhere, causing pericarditis, or peritonitis, and even sometimes meningitis. The bronchial glands also become enlarged. It is doubtful if a croupous pneumonia which goes on to the stage of grey hepatisation ever recovers, it being most commonly believed that resolution takes place, and recovery, when the disease does not progress beyond the second stage.

The **causes** of croupous pneumonia are not very clear. It appears to be frequently the result of exposure to cold and wet. A low state of health, or some wasting disease, such as diabetes, favours the occurrence of pneumonia; but it often attacks the robust. There is a growing opinion that croupous pneumonia is a constitutional disease of the nature of a fever, a theory strengthened by the fact that it is sometimes epidemic. Friedländer has lately discovered a micrococcus associated with croupous pneumonia. Croupous pneumonia is common in mid-winter and spring. Gout, rheumatism, diabetes, and especially chronic alcoholism, are well known to be constitutional states associated with, and sometimes actually concerned in, the production of croupous pneumonia.

The **symptoms** usually commence with a well-marked rigor, the temperature rising in a few hours to 104° or 105° Fahr. Sometimes there is no rigor, and only a feeling of chilliness; and frequently pneumonia begins very insidiously. If the onset be severe and sudden, there is sometimes vomiting—or perhaps a convulsion—with a marked flush upon the cheeks, headache, and general *malaise*. The pulse is quick, full, and bounding. The respiration is markedly

hurried, and the *alæ nasi* dilate with inspiration. The skin has a "pungent heat" at this stage when tested by the hand, although later there may be profuse perspiration. Pain is very frequently complained of in the side, and there is usually a short husky cough: later, the cough is accompanied by the expectoration of tenacious rusty-coloured sputum, which is characteristic. It is like Gregory's mixture in a very small quantity of water or albuminous fluid, and "badly mixed." Sometimes it is less viscid, and like "prune-juice." The spit-jar may be reversed without the matter escaping. Sometimes there is no expectoration throughout the whole course of the disease. The tongue becomes furred, dry, and brown, the bowels are generally constipated, and the urine is high coloured, scanty, and acid, and it deposits urates in abundance. The chlorides in the urine, as in all fevers, are diminished in quantity, and sometimes albumen is present. Jaundice is frequent. Herpes affect the lips. The temperature is subject to irregular variations, falling suddenly, and as suddenly rising. Delirium may be present early, and especially in the intemperate, and then, very frequently, the upper lobe of the lung is the part affected (see Influenza).

The **physical** signs in a typical case are very striking. The most important is the detection of fine crepitations, which are very characteristic. They are heard upon deep inspiration, and at the end of the act. They may occur in œdema, collapse of the lung, and even in capillary bronchitis, however, and they are, therefore, not quite pathognomonic of croupous pneumonia. They are often heard also at the bases of the lungs in patients suffering from some debilitating disease, and who have been in the recumbent position for some time; but a few deep inspirations cause these crepitations to disappear. The cause of the fine crepitant râles in pneumonia, and in the other conditions in which they are found, is the opening up of the very minute bronchioles and alveoli which have become slightly adherent, either from shallow and weak inspiration (debility), or from their being swollen (pneumonia, œdema, collapse, &c.). It may require careful examination for their discovery, and they are only heard at the *edge* of the consolidation; and sometimes, if the pneumonia be deep, they may not be heard at all, or only, after a few days, when the disease becomes more superficial. The vesicular murmur is obscured by these crepitations, or replaced by *bronchial* or characteristic *tubular* breathing—*i.e.*, if the bronchial tubes be not plugged by mucus. When so, the breath-sounds and crepitations are not heard unless coughing remove the obstruction. There is dulness upon percussion over the diseased lobe, and the vocal fremitus is increased. Bronchophony is present. When resolution takes place the fine crepitations appear again in a slightly coarser form—known as *redux crepitations*. The pleurisy which usually accompanies the pneumonia may give rise to a friction sound, and if there be effusion, the physical signs which characterise pneumonia may be very much modified.

Croupous pneumonia runs a definite course, and the fever usually terminates *by crisis* on the fifth to the tenth day. If prolonged

beyond the fourteenth day some complication is present, usually. The crisis is usually followed by profuse sweating, by sudden diarrhœa, or by the voiding of large quantities of urine.

Relapses are common, and the disease is apt to recur in individuals. The *prognosis* is always grave. Children recover more readily. The constitution and strength of the patient must always be considered in estimating the probable result. It is very grave when associated with alcoholism. All danger is not at an end although the *crisis* be past, as many cases die of sudden collapse, shortly, or two to three days thereafter. Sudden supervention of œdema, or failure of the heart, may cut off the patient at any time; or death may occur during the course of the disease by gradual exhaustion —the breathing becoming more and more shallow, the face livid, the skin covered with cold sweat, and the pulse weak, irregular, and finally lost.

Embolic Pneumonia. Hæmorrhagic Infarction.—Emboli may be simple or infective. The simple, when lodged in a terminal blood-vessel—so that no collateral circulation can nourish the parts supplied by the obstructed vessel—give rise to wedge-shaped infarctions, the base of the wedge being towards the periphery. The part affected is of a deep blood-red colour, sharply defined, and exuding blood upon pressure. When older they appear more granular, from fatty degeneration, and the tissue is more indurated. The parts adjacent are conjested and œdematous. Atrophy and cirrhotic contraction may ultimately obliterate the part, and a cure thus take place. A patch of the pleura is usually affected, as infarctions are superficial. Infective emboli are not wedge-shaped, and they give rise to suppurative inflammation and abscesses, and sometimes to gangrene. Infarctions are most common in the right lung. They are caused by clots breaking off in some part of the venous system, or right cavities of the heart, and these entering the circulation ultimately block the smaller arteries.

The **symptoms**, when the infarction is large enough, are sudden dyspnœa, followed by bloody expectoration, and the subsequent evidences of consolidation, as dulness upon percussion, bronchial breathing, and bronchophony. Infarctions most usually occur in heart disease; but they may arise from thrombosis of any part of the venous system. When small, they give rise to no symptoms, and even when large the co-existing disease may entirely mask the symptoms and signs. If the large branches of the pulmonary artery be blocked, the dyspnœa is very acute, and death takes place within a few minutes. In the infective form, there are repeated rigors, but not very high fever. The chills are marked in cases of pyæmia. There is sharp pain when the pleura is involved. The prognosis is always unfavourable. In the treatment, ammonia may be administered.

The Treatment of the foregoing diseases—the second group according to the classification on p. 114—may now be considered together, as they are all treated upon the same lines. In acute

bronchitis caused by cold, the initial symptoms, or the very mild cases affecting only the larger tubes, are treated in the same way as ordinary coryza. In the severe cases, in adults, a turpentine stupe should be placed for half an hour or longer around the chest, which should afterwards be covered with a layer of cotton wadding. This is preferable to poulticing, in many cases, as there is less liability to chill. Sometimes, however, it is too severe a measure, and then poultices must be used, with, or without mustard, care being taken to have the second poultice ready to place upon the chest the moment the first is removed. In children, poultices or liniments (Bowe's, or compound camphor liniment) may be used, the latter having the same advantage as the turpentine stupe in adults, as wadding may be kept around the chest continuously, and the nurse's hand passed underneath when the liniments are used. The temperature of the room should never be allowed to fall below 63° Fahr.; and in the severe cases with obstruction to the breathing, the atmosphere around the patient's head should be kept moist and warm by means of the bronchitis kettle. Great care should be taken when steam is used, that the lamp is never allowed to go out, as there is then danger to the patient from the cold damp hangings surrounding him. If the patient be robust enough, the early stages may be treated by depressants. Small doses of tartar emetic (one-sixteenth of a grain every three hours) or antimonial wine (twenty minims every three hours), or tincture of aconite (in drop doses every ten minutes for two hours) may be used during the first day. Carbonate of ammonia is the most useful remedy in bronchitis, and it may be prescribed upon the second day, or from the first, if it be undesirable to use depressants. ℞ 18 is a useful combination. Five to ten minims of solution of morphine hydrochlorate may be added to each dose when the cough is very troublesome. Sometimes a gargle of chlorate of potash may relieve the cough; and sprays are useful with children. Bartholow highly recommends a combination of carbonate and iodide of ammonia—five grains of each, every four hours—for bronchitis, catarrhal pneumonia, and croupous pneumonia. When the breathing is much obstructed by the tenacious mucus an emetic should be given. Half a teaspoonful or more of ipecacuanha wine (repeating in ten minutes if necessary) is the best for children. Apomorphia, hypodermically, may occasionally be required for adults when the mucus obstruction is very great. Quinine or antipyrin—fifteen grains of either—should be given when the temperature is high. The dose may be repeated three or four times at intervals of four or six hours. Chloral, fifteen grains, and bromide of potassium, forty grains, are the best remedies for sleeplessness. The dose may be repeated in two hours if necessary. Opiates should not be given for this purpose, as in the larger doses they tend to dry up the pulmonary secretions. Morphine *may* be given hypodermically for pain or sleeplessness. When the case is improving, and the fever has passed away, an expectorant mixture (℞ 19) may be used, or the carbonate and iodide of ammonia may be continued. Cod-liver oil and iron tonics are indicated during convalescence. Alcohol may be

necessary at times, or throughout the case, and the diet should be light and digestible, consisting chiefly of beef tea, milk, chicken broth, wine-whey, and egg flip, &c., given in small quantities, frequently repeated. The *causes* of bronchitis must be considered with the treatment; hence when due to valvular disease of the heart, digitalis will be indicated. In Bright's disease, also, the treatment will be modified. Fibrinous bronchitis may be benefited by a spray of lime water, which is a solvent for fibrinous matter.

In acute catarrhal pneumonia the treatment is the same as in acute bronchitis. In the sub-acute or chronic forms, turpentine, eucalyptus, and copaiba are useful remedies. Externally, iodine liniment or tincture is used—several coats being painted on the chest.

In active congestions of the lung, cupping or leeching at the bases, is useful in the earliest stage. Large mustard plasters may be placed upon the chest. Stimulating expectorants and digitalis are prescribed. In croupous pneumonia the treatment may commence with a large dose of quinine (twenty grains), or other antipyretic. Depressants may be used the first day if the patient be robust; but usually ℞ 20 is given from the beginning. The external agents used are the same as in bronchitis. Digitalis is considered by some authors to increase the embarrassment of the heart. If a heart tonic be necessary, five to eight minims of tincture of strophanthus would do away with this objection, as the strophanthus does not raise the blood pressure by acting upon the capillaries, like digitalis. Alcohol in large quantities is often very urgently needed in croupous pneumonia; and especially is it necessary with alcoholic patients. It should be continued for some days after the crisis, and the doses should be gradually diminished, if it has been necessary to order *large* quantities during the illness. During convalescence a tonic of carbonate of ammonia and iron should be given (℞ 28[A]); or iron and iodide of potassium (℞ 30) may be used when there is reason to believe that a deobstruent will be useful.

Chronic Bronchitis.—**Pathology.**—In chronic bronchitis the air-tubes are filled with muco-purulent matter, while the mucous membrane is seen to be much congested, thrown into folds, and of a dark red colour and glossy appearance. The smaller bronchi are often dilated. Minute depressions on the surface of the mucous membrane may be seen by the aid of a small lens, to be dilated mucous glands. Microscopically, the whole of the bronchial wall is seen to be much thickened, and infiltrated with cellular structures. The epithelial lining of the tubes is irregular, the cells being in parts cast off, and presenting the appearance of a germinating membrane of round and pyriform-shaped cells. The capillary blood-vessels are dilated, and the cartilages and muscular fibres atrophied. The cellular structures, confined by the elastic basement membrane, make their way along the lymphatics, and in very chronic cases they are the cause of chronic interstitial pneumonia (cirrhosis); and especially is this the case

when the cause of the bronchitis has been the inhalation of irritating dusts, as with coal miners, needle grinders, and stone masons. When the bronchitis is due to chronic valvular disease of the heart, the state of *brown induration* of the lung is induced. Punctiform hæmorrhages are then seen under the pleura. The whole lung is intensely congested, and throughout its substance there are indurated semi-solid patches, which, microscopically, are found to consist of blood effusions and epithelial cells containing brown pigment. The alveolar capillaries are dilated from mechanical causes. The congestions of the different organs, and the pathological conditions described in the section dealing with heart disease, will also be present. For the further pathological changes due to secondary complications, see *Emphysema*, *Interstitial Pneumonia*, and *Bronchiectasis*.

Chronic bronchitis may result from frequent acute attacks; but often it begins insidiously in those who are much exposed to cold and wet. It may be one of the results of chronic valvular disease of the heart; or it may arise in connection with the scrofulous diathesis, rickets, Bright's disease, and infectious fevers, &c.; or it may be due to the constant inhalation of irritating dust, as already mentioned.

The **symptoms** begin with a tickling cough and increased expectoration, as in the acute form. The cough and expectoration is worse in the morning. There is not much dyspnœa at first, but this gradually increases. There is no fever unless an acute attack supervene. There is emaciation in the later stages. The physical signs are the same as in the acute form, to which are added the symptoms and physical signs of the causal affections and secondary complications. These are discussed later as separate and distinct diseases. It should be noted that while chronic valvular disease of the heart may be the cause of bronchitis, bronchitis in its turn may increase the embarrassment of the heart; and it may—without valvular disease being present—so dilate and weaken that organ, that eventually all the secondary train of symptoms due to obstructed circulation (described under heart disease) may set in.

Recovery from chronic bronchitis may sometimes take place if not too far advanced; and children suffering from chronic bronchial catarrh frequently "outgrow" their attacks of "winter cough." If the patient be placed in favourable circumstances, his condition may be ameliorated, and life may be prolonged; but complete cure is a rare event. Death may result from an intercurrent acute attack, or from slowly developed emphysema, and dropsy, as in the valvular diseases of the heart.

Bronchorrhœa serosa is a form of chronic bronchitis accompanied by the expectoration of large quantities of thin, watery fluid. *Fœtid* bronchitis I have preferred to place in a separate group with bronchiectasis and gangrene of the lungs.

The diagnosis and differential diagnosis, are considered in a summary at the end of the section on diseases of the respiratory system.

In the **treatment** of chronic bronchitis the diet should be light, ut generous and nourishing. Over-loading the stomach predisposes o asthmatical attacks. The clothing should be suitable, and flannel hould always be worn next the skin. Damp atmospheres are to be voided. The medicines of most value are turpentine, eucalyptus, opaiba, benzoic acid, and cubebs; and these may be administered n capsules, in their proper doses. Easton's syrup combined with rsenic is a useful remedy. Cod-liver oil is often indicated. Some- mes the carbonate along with the iodide of ammonia, relieves; nd even, according to Bartholow, may *cure* a case of chronic ronchitis. An expectorant mixture is often required—℞ 19, or 18— hich, although apt to disorder the stomach when long continued, et often seems to give most relief to the patient. Codeia—in yrup or pill—is the best remedy for the cough. Mentone and an Remo are *stimulating* climates, while Torquay, Penzance, and Madeira are examples of the *sedative*. Bournemouth has the dvantage of a pine forest.

Subacute or chronic catarrhal pneumonia is frequently associ- ted with chronic bronchitis. It is mentioned in this place for the ake of the classification and the reasons given for the same, on . 114; but the disease is considered with acute catarrhal pneu- nonia, in part, and the remainder with phthisis.

Chronic Interstitial Pneumonia. Cirrhosis of the ungs. Fibroid Phthisis.—This disease is to be regarded nore as a secondary lesion rather than a primary one, as it lways follows some irritation in other parts. Some authors, owever, consider the pathology of cirrhosis as still unsatisfactory, nd it may be that it occasionally arises as a disease *per se*, and then is spoken of as a *chronic pneumonia*. In phthisis, cirrhosis is ften described as a part of the pathological condition. Chronic ronchitis, by extension of the cellular structures, along the peri- ronchial lymphatics; catarrhal pneumonia, for the same reason; ritating matter (coal, steel, and stone dust); and phthisis; are the rimary lesions which give rise to cirrhosis of the lungs. Thicken- g occurs in the fibrous tissue along the line of the lymphatic essels. The bronchi become dilated either from the expira- ry force of the cough, or from the contraction of the thickened terlobular septa (bronchiectasis). In very chronic cases, tubercles nd caseous material are found in the lung-tissue, while there is ore or less compensatory emphysema present. The pleura is ickened, and the cavity, in extreme cases, may be obliterated, the parietal and visceral layers may be firmly bound by numerous dhesions. The pulmonary vessels are dilated and thickened. Cir- osis is commonly bilateral, but often it is unilateral, especially hen the primary irritating cause (as in phthisis) is limited to ne lung. In *anthracosis*, the lung, besides being pigmented, has t-black nodules throughout its substance, which are hard, and ay be picked out. In *stone mason's* lung the nodules are grey, nd the lung is more markedly cirrhotic.

The **symptoms** of chronic interstitial pneumonia are mixed up

with the symptoms of the initial disease by which it is caused, and its presence may only, in many cases, be surmised. Inspection reveals the flattening, and in extreme cases, the indrawing of the chest-walls, due to the adhesions and their contraction. The heart is frequently drawn from its normal position. The measurement of the chest with the cyrtometer reveals great shrinking of the affected side as compared with the sound side—*i.e.*, in unilateral cases. The percussion-tone is impaired, and dulness is most frequent at the apices which are much shrunken. *Auscultation* gives the breath-sounds, accompaniments, and vocal resonance, according to the nature of the primary disease. In the phthisical—whether phthisis be the primary cause of the cirrhosis or *vice versâ*—the physical signs of phthisis are present. The history of the previous health, and the course of the disease, are, therefore, important points in the diagnosis.

Pleurisy with contraction may simulate cirrhosis; and so may malignant growths. When the cirrhotic contractions affect the heart, the case may terminate fatally, by ascites and dropsy.

The treatment is to remove the patient from the cause, if possible; but generally it can only be symptomatic. Cirrhosis, most frequently, can only be viewed as incidental to bronchitis or phthisis.

Emphysema of the Lungs.—There are two kinds of emphysema—viz., vesicular and interstitial. In the first there is an over distension of the pulmonary alveoli, while in the latter there is infiltration of air within the interstices of the lung-tissues. The two may co-exist when a rupture of the lung takes place. The air may, in extreme cases, make its way under the superficial fasciæ of the neck and chest, and it can there be felt to crepitate.

Vesicular emphysema is commonly a consequence of chronic bronchitis; but it may occur as a primary disease. The lungs are anæmic and much increased in volume. They overlap the heart in front, sometimes entirely. The lungs do not crepitate, and they feel like "thin bags loosely filled with feathers." The parts most affected are the anterior borders and apices. When the air vesicles rupture, large bullæ are often formed. The circulation through the air vesicles is hindered, and hence there is congestion towards the central parts, and the heart becomes dilated. This is followed by the train of symptoms leading to dropsy, described under the valvular diseases of the heart.

Microscopically, the alveoli are seen to be enormously dilated, while their walls and the interlobular septa are much thinned or wasted away. Fatty granules are seen amidst the remains of the alveolar epithelium. It has been suggested, in order to explain these cases which arise without any history of antecedent causal disease, that emphysema may be produced by changes within the pulmonary tissue itself. It appears sometimes to be hereditary.

The cause of vesicular emphysema has been shown by Jenner and Mendelssohn to be forced expiratory efforts, as in coughing; but Gairdner's *inspiratory* theory still accounts for the compensatory forms of the disease, such as occur in collapse, and in shrunken conditions of the lung.

The symptoms of emphysema are generally superadded to those of chronic bronchitis. The dyspnœa gradually increases, and at last it becomes very great. The patient complains of a "feeling of inflation" about the chest, and any intercurrent attack of bronchitis or asthma is always dangerous, and may prove fatal. There is only a cough when bronchitis is present. Very rarely is there hæmoptysis in emphysema. Inspection reveals the "barrel-shaped" chest in the advanced cases. The sternum and upper ribs are arched, and the arching of the dorsal vertebræ is increased. The circular shape of the chest is well seen when the cyrtometer is used, and a tracing is made. When the patient is made to cough, there is marked bulging at the root of the neck and in the upper intercostal spaces. There is distension of the veins at the root of the neck. Sometimes a transverse groove is formed when the thorax is markedly arched in its whole extent. In marked emphysema, with a barrel-shaped chest, there is little or no movement of the ribs in respiration, the diaphragm being the chief muscle used in the act.

The *percussion* yields important information, especially over the region of the heart. In the vertical diameter the dulness may not commence until the fifth or even the sixth rib is reached. In extreme cases the lungs cover the heart entirely, and there is no dulness on percussion, while palpation cannot detect the apex beat. There may then be pulsation in the epigastric region. The liver, in advanced cases, is also depressed – dulness (in the mammary line) beginning only when the seventh or eighth rib is reached. Percussion of the bases of the lungs reveals a clear note an inch or two lower than the normal, the character of the tone being almost tympanitic. *Auscultation* in an uncomplicated case of vesicular emphysema, reveals an absence or enfeeblement of the vesicular murmur. The normal bronchial breathing heard under the right clavicle and at the level of the fourth dorsal vertebra appears distant or greatly enfeebled. These physical signs are, however, in most cases masked altogether by the râles and rhonchi of the associated bronchitis, and *prolonged expiration*, rather than an absence of the vesicular murmur, is apparently present. Jenner has pointed out that sometimes the pulse after coughing becomes full and tense, and afterwards it is lost for a little time. A tricuspid murmur may be detected. Death may result, like heart disease, by the obstruction of the circulation leading to dilatation of the heart, stasis, and dropsy.

The prognosis is grave in advanced emphysema. If there be much bronchitis, or if the patient be subject to severe attacks of asthma, there is great danger. A certain amount of emphysema is not incompatible with long life if the patient be placed in favourable circumstances.

The treatment of vesicular emphysema can only be palliative, and it consists in the treatment of the asthma and bronchitis, or in assisting the heart as in cardiac dilatation (*see* p. 34).

Fœtid or Putrid Bronchitis.—This condition occurs in cases of chronic bronchitis with dilatation of the bronchi. It is more

apt to attack those in middle life. The mucus accumulates within the dilated tubes and becomes putrid. The expectorated matter is highly offensive and of a dirty grey-yellow colour. Microscopically, numerous organisms, crystals, and fat globules are found. The offensive odour is due to the presence of volatile fatty acids. When the sputum is allowed to settle in the jar, three layers are formed—the upper opaque green-yellow and frothy, the middle an albuminous liquid like serum, and the lower stratum opaque dirty-yellow, and made up of pus cells and detritus.

The symptoms in severe cases consist of the sudden development of the *gangrenous odour*, with increased dyspnœa, followed soon by typhoid symptoms leading to collapse, coma, and death. The *post-mortem* examination reveals the sloughing and inflamed bronchial tubes full of the offensive matter; while here and there are patches of gangrene in the lung-tissue, with œdema. Slight cases sometimes recover; and some others run on for months.

Bronchiectasis.—This affection is closely allied, *clinically*, to the preceding. There is an offensive expectoration, and a dilatation of the bronchial tube, but as the latter is more defined than in the simple dilatation associated with putrid bronchitis, it gives rise to different physical signs, and to a different *manner* of expectorating the offensive liquid.

There are three forms—the cylindrical or fusiform, the saccular, and cavernous. The cavities contain muco-purulent matter, and the walls are smooth. Sometimes there are fibrous bands crossing the cavities. The walls are never granular. The disease is often associated with cirrhosis of the lung, and it has been thought that contraction of the fibrous tissue produced the dilatation of the bronchial tube. The theory most accepted, however, is that the cavity is produced, like emphysema, by continual forced expiratory efforts—*i.e.*, coughing.

The **symptoms** inseparable from bronchitis—as cough, dyspnœa, lividity, &c.—are present; but the one symptom peculiar to the disease is the absence of all expectoration for a time, and then the sudden coughing up of a large quantity of offensive liquid. The physical signs are only marked when the cavity or cavities are large, and repeated examinations discover variations in the breath-sounds and percussion-tones, depending on the size of the cavity and whether it be full or empty. When large and full, there may be found dulness on percussion, and an absence of the breath-sounds over the part; when empty there is no dulness, but bronchial breathing and bronchophony may be made out. The râles too are coarser than those usually heard in the normal-sized tubes.

Gangrene of the Lung.—Gangrene is always the result of severe injury to, or intense inflammation of, a tissue. In the case of a lung being the seat of gangrene, the characteristic appearances of pneumonia are generally seen, but the gangrenous part is very soft and of a dirty green or black colour. The tissues emit a horrible odour, unless the inflammation has been so severe as to destroy life early, without giving time for putrefactive changes.

The **causes** are numerous. Croupous pneumonia may lead to gangrene in drunkards, and in the old and feeble, or in those suffering from diabetes or other chronic wasting disease. Gangrene seldom follows croupous pneumonia, in the otherwise healthy and robust individual. More commonly septic matter, by direct extension, as in ulcerating cancer of the œsophagus, suppurating hydatids or abscess of the liver, ulcers of the stomach, and even purulent empyema—sets up gangrene of the lung. Septic emboli, derived from a cerebral sinus (as in ear disease), or from an ulcerative endocarditis, may also produce gangrene. From the bronchial tubes, foreign bodies or food—especially when vomiting has occurred while using anæsthetics—and putrid matter derived from putrid bronchitis and bronchiectasis, may light up gangrene of the lungs.

The characteristic symptom is the horrible fœtor, only absent in the rare and very rapid forms. The sputum is like that which occurs in cases of fœtid bronchitis and bronchiectasis, but it differs in not separating into layers in the jar. It is dirty-grey or green in colour, sometimes black. Hæmoptysis is not common. If the gangrene be superficial, it may affect the pleura and a septic pleurisy may result. The pulse is feeble, small, and rapid. There is pyrexia, and the patient soon passes into the typhoid state if the disease be severe and about to terminate fatally. The stethoscope does not reveal any characteristic sign peculiar to gangrene. Sometimes fine râles and the signs of a cavity—as cavernous breathing and loud bronchophony—are made out, and percussion dulness may be observed. If phthisis be present these signs prove nothing, unless they have developed at the same time, or after the fœtor and expectoration.

The **treatment** of the members of the fœtid group is the same, inasmuch as they all require inhalations of turpentine, carbolic acid, iodine, creasote, or eucalyptus. Internally, carbonate of ammonia, camphor, quinine, and especially *iron*, should be administered. The diet should be very generous, and alcohol should be allowed. *Fœtid* bronchitis requires also the same stimulating treatment as the ordinary forms. Bronchiectasis—as regards further treatment—should be viewed in the same light as a case of chronic phthisis. Surgical treatment may sometimes be possible, and relief follows the free drainage of the cavity. Gangrene of the lungs does not allow of much time for treatment, if acute; but turpentine and eucalyptus—five drops every two hours—are recommended.

Phthisis Pulmonalis, and Miliary Tuberculosis.—The special works on pathology must be consulted upon this subject, especially as the views of authors differ considerably regarding the origin and the variety of forms of tubercular disease. Koch's important researches, although widely accepted, are still meeting with some opposition, it being maintained that the bacilli are absent in some cases of undoubted phthisis, and that they have been found present in two cases, at least, of bronchiectasis. The parasitic theory of the origin of phthisis is, therefore, still unsettled. For

clinical purposes a mere outline of the pathology of phthisis is all that can be attempted in a work of this kind.

Subacute or chronic catarrhal pneumonia very often leads to phthisis (*see* p. 87). The continued infiltration of the cells into the surrounding tissues causes hyperplasia of the connective tissue. The catarrhal products form dense homogeneous masses, which interfere with the circulation of the blood, and so affect the nourishment of the tissues that a condition favourable to caseous or cheesy transformation is produced. There is absorption of the fluids, fatty degeneration of the cell elements, and disintegration of fibrinous matter—the result being the formation of a soft cheesy solid. Softening of these masses begins in the centre, and cavities (vomicæ) are thus formed, which may coalesce by the breaking down of the surrounding tissues. These cavities are at first irregular, but ultimately they become smooth. Fibrous bands are sometimes seen traversing the cavities. When putrefactive changes take place, the affected parts become foul and brown-green in colour. The sputum is then offensive. The examination of the expectoration with the microscope reveals elastic fibres, with mucus and purulent cells and *débris*. Hæmorrhage may result from erosion of blood vessels. The apex of the lung is the usual seat of the disease, but a whole lung may be affected (phthisis florida). In the *later* stages of the forms of phthisis which commence with catarrhal pneumonia, tubercles are superadded to these conditions. The form just described is often called *caseous phthisis*.

Tubercular phthisis is the form believed by some authors to begin by the deposit of tubercles in the lung, chiefly at the apex, and these leading to desquamative pneumonia produce the same changes as in the caseous form. Hamilton doubts this mode of origin. The tubercles are grey-white, translucent, semi-solid nodules about the size of a millet seed. Microscopically, they are seen to be composed of a reticulum enclosing numerous cells within its meshes. In or about the centre, *giant cells* with numerous nuclei are found. The tubercles become caseous, and then they soften and break down along with the surrounding caseous pneumonic patches. There is, in short, a necrosis of the pulmonary elements. The mucous membranes are ulcerated by the tubercles.

In the form sometimes known as *fibroid phthisis*, there is slow development of cirrhotic tissue, causing atrophy and degeneration of the true lung-tissues. Tubercles ultimately develop. (*See* Chronic Interstitial Pneumonia, p. 95).

The pleura (in all forms) is usually affected with chronic inflammation, and ultimately the tubercles invade the inflamed parts. A cavity often ruptures into the pleural sac, and pyo-pneumothorax is the result. If pleural adhesions have formed it may discharge through the thoracic wall (fistula). The bronchial glands enlarge and undergo caseation, and they may sometimes suppurate and burst into the trachea, bronchi, or œsophagus. The enlarged glands may also give rise to suffocative attacks, especially in children, by pressure upon the trachea or vagi. The peritoneum, bowel, urinary tract,

and the larynx are often affected by tubercular disease. In advanced and chronic cases the liver is often fatty; but perhaps this is greatly due to the cod-liver oil given in the treatment. The kidneys suffer from waxy disease, and hence there is albuminuria. Secondary pyæmic abscesses sometimes affect other parts, as the brain, liver, kidneys, &c. In the cases of phthisis which recover, there is either extrusion or absorption of the caseous matter, with formation of connective tissue and subsequent contraction; or the caseous matter may become calcified.

Miliary tuberculosis is the sudden and general infiltration of the pulmonary, as well as other tissues, with an enormous number of tubercles brought by the blood current. The infective source may be at a distance, or may itself be in the lung. A case of phthisis may terminate by the acute dissemination of tubercles. Owing to the greater susceptibility of the apices to tubercular infiltration, a case beginning as an acute miliary tuberculosis may, on the other hand, terminate as a case of ordinary phthisis.

While there is great difference of opinion as to the *pathological* classification of tubercular diseases, yet, clinically, it is possible to classify them thus, viz.:—the *caseous* form; *tubercular phthisis; fibroid phthisis;* and *miliary tuberculosis*. The forms are often *mixed* more or less; but the antecedent disease, history, mode of origin, and frequently the symptoms often enable such a differentiation to be made.

The caseous form occurs in the weakly or strumous. The predisposition may be acquired by bad hygienic conditions and surroundings. Measles and whooping-cough are often associated with catarrhal pneumonia, and many cases terminate in caseous phthisis. A severe neglected cold is the commonest cause of all. The tubercular form is hereditary, and it is closely associated with the strumous diathesis. A bad shape of chest favours the development of tubercular phthisis. Damp climates and bad hygiene are also unfavourable. Phthisis is believed to be communicable to those predisposed to the disease; but the exposure requires to be somewhat prolonged.

The symptoms of phthisis vary according to the form which attacks the patient. In the early stages of the sub-acute or chronic cases (*caseous phthisis*), the usual complaint is that of a severe cold, with cough and expectoration of mucus, and later, of muco-purulent matter. The "cold" does not get better, or there is apparently improvement; but he soon fancies he has caught another cold, and these being repeated, he ultimately complains of continuous pains in the chest, feverish attacks, debility, and general *malaise*. The appetite fails, and the patient feels unable for his work. The cough begins to be very troublesome, and perhaps the expectoration may be tinged with blood. A smart attack of hæmoptysis frequently occurs at this stage. The patient becomes anæmic and he loses flesh rapidly. The emaciation may proceed so far as a fourth, to even, ultimately, a half of his original body-weight.

In the early stages frequent chills, not unlike ague, with hectic

flush upon the cheeks and hot hands, give place to undoubted pyrexia, accompanied by severe perspirations. The temperature varies from 100° in the morning to 102° or 103° Fahr. in the evening, but it is reduced after sweating. The pulse is feeble and quick, and after exertion of any kind it becomes still more so. The pain in the side is not acute unless pleurisy, at the lower part of the chest, is present. The co-incident pleurisy at the apex does not cause acute pain, as the expansion of the chest is not great in this region. The hæmoptysis may be the first symptom calling attention to the case; and by some authors it is held to be itself sometimes a cause of phthisis. Others believe it to be always secondary to an incipient lung disease, and, therefore, a symptom. Sometimes, however, a patient may have an attack of hæmoptysis, and no further symptom of phthisis has developed.

When a patient begins to recover from an attack of hæmoptysis, the expectoration becomes less and less sanguineous, and its brown-red colour becomes clearer, until only the ordinary muco-purulent matter is coughed up. The expectorated matter is frequently "nummular." Microscopically, elastic fibres may be seen on examination of the sputum. (Some of the expectorated matter should be boiled for three minutes in an equal part of a solution of caustic soda (15 grs. to ℥i); next the resulting liquid is put into a conical glass with pure water. Some of the deposit which follows is then put upon the microscopic slide and covered with a cover-glass.) For the detection of the bacillus tuberculosis, the following preparation is necessary (*Heneage Gibbes' method*):—

"Take of rosaniline hydrochloride two grammes, methyl-blue one gramme; rub them up in a glass mortar. Then dissolve aniline oil, 3 cc., in rectified spirit, 15 cc.; add the spirit slowly to the stains until all is dissolved, then slowly add distilled water, 15 cc.; keep in a stoppered bottle. The sputum having been dried on the cover-glass in the usual manner, a few drops of the stain are poured into a test-tube and warmed; as soon as steam rises pour into a watch-glass, and place the cover-glass upon the stain. Allow it to remain four or five minutes, then wash in methylated spirit until no more colour comes away: drain thoroughly and dry. Mount in Canada balsam. The bacilli are stained *red*."

Before proceeding to the physical signs, the symptoms of the other forms—so far as they differ from the "caseous form" or acquired phthisis—may first be described. There are two types of constitution associated with tubercular disease—the delicate strumous, and the lymphatic strumous. In the first, the figure is tall and thin, with long neck and prominent larynx; the shoulders slope, and the thorax is narrow or flat, and it has little expansile mobility; the hair is fine and generally fair, sometimes "lanky"; the eyes are large and bright, and the skin is delicate and flushes easily; the fingers are long and often "clubbed" at the extremities. They are bright and nervous individuals, fond of active work, but easily fatigued. Those of the *lymphatic* type are heavier in make. They are slow in their movements; and their skins are coarser and

have a "bad colour." The hair is more frequently dark. Epistaxis—and, in women, disorders of the menstrual functions—are common in tubercular subjects. In rare cases the menstruation is *vicarious*.

The chronic tubercular form of phthisis usually begins very insidiously. The appetite is variable, and there is loss of weight without apparent cause. The patient is easily fatigued, feverish attacks begin (*hectic fever*), and a short dry cough, pains in the chest, and nocturnal perspirations may be among the earliest symptoms. Hæmoptysis may occur now, or it may be the first symptom. The pulse is quick and feeble, and there is breathlessness especially upon making any exertion. The respiratory movements—always shallow in tubercular patients—become very rapid ; but actual dyspnœa can only be said to exist in the very advanced cases.

Fibroid phthisis is the most chronic of all. The early symptoms are those of a chronic bronchial catarrh. The cough is at first dry and the expectoration consists of simple mucus. In course of time his condition becomes worse ; but there is little emaciation in the early stages. After a few years, feverish attacks—in the evening generally—nocturnal sweats, hæmoptysis, and the other symptoms as in the tubercular form, begin to manifest themselves. Ultimately, the case is indistinguishable from the tubercular form in its further progress, except that the history of its origin, and the results of contraction of the fibroid tissue enable a diagnosis of this variety of phthisis to be made. There is increase of the connective tissue and contraction in *all* the forms of advanced phthisis, so that the name "fibroid" expresses the most striking pathological factor in the disease rather than a special variety. Fibroid phthisis, clinically, is therefore chronic interstitial pneumonia (cirrhosis of the lung) *plus* tubercles and formation of cavities ; or slowly developed tubercular disease *plus* great increase of fibrous tissue.

Miliary tuberculosis is not usually described as a form of phthisis, as it is a general disease affecting many organs. As there is always an infective source (sometimes in the lung itself), and from the liability of the apices of the lung to be affected and the case ultimately to become one of tubercular phthisis—it is convenient to describe it here.

The most prominent symptoms are the pyrexia and the dyspnœa. The onset is generally sudden. The temperature ranges from 102° to 105° Fahr., and often it is peculiar in being higher in the morning than in the evening. The skin is covered with sweat during the remissions of the temperature. The breathing is very hurried, and often there is a lilac or purple appearance of the lips, cheeks, fingers, and nails—the latter appearance suggesting tuberculosis, if no heart disease or emphysema be present to account for the venous obstruction. The cough is short, dry, and troublesome, and the expectoration is sometimes streaked with blood. The physical signs are generally vague ; but sometimes slight dulness is made out at the apices. A soft friction rub has been described, which is supposed to arise when the tubercles are superficial. "Consonating" râles are sometimes heard. The physical signs of bronchitis are often present,

and these obscure the finer râles. A further means of diagnosis lies in the examination of the choroid which may be affected by the tubercles. The disease may terminate in the development of typhoid symptoms and death, in two, to four, or six weeks; or the case may become chronic, and the patient phthisical. The presence of tubercles in other organs—as in the brain, with the symptoms of meningitis; or the presence of jaundice when the liver is affected; or pains in the joints, &c.—may help to a correct diagnosis.

Physical Signs of Phthisis.—In the early stages the signs may be very doubtful, and repeated examinations, at intervals, may be necessary before a positive diagnosis can be made. Amongst the earliest signs noticeable are diminished mobility and slight flattening at the apex of the affected side. The vesicular murmur may be deficient or absent, but the plugging of the bronchial tubes with mucus may be the cause of this, and the patient should be instructed to cough during the examination. Instead of deficiency, there may be harshness with prolonged expiration, limited to the apex. This should not be mistaken for the *puerile* breathing found on the sound side, and compensatory to a consolidation of the opposite side. A "jerky" or "interrupted" form of breathing, limited to the apex, is also suggestive of early phthisis, the irregular opening up of the small bronchioles and air vesicles, from adhesion of their surfaces, being the cause of this sign. It should be noted that the heart's impulse may give rise to this jerky breath-sound, and in very nervous people it may occur without organic disease being present. A few moist râles limited to the apex, or even one little "clinking" râle—brought out by coughing during the examination—is highly suggestive of incipient phthisis, when associated with one or more of the symptoms. The vocal resonance is increased if there be sufficient consolidation, and percussion reveals dulness if the disease be sufficiently advanced. The normal breath-sounds heard under the right clavicle, and at the level of the fourth dorsal vertebra behind, are not to be mistaken for exaggerated breath-sounds due to consolidation.

As the disease advances the signs become more marked. The mobility of the chest becomes still more diminished, and the flattening and hollowing above the clavicles are more obvious. *Palpation* also reveals the diminished mobility, and if, at the same time, the *vocal fremitus* be tested, it will be found to be increased. The cyrtometer, in very advanced cases with shrinking of the lung from contraction of fibrous tissue, reveals diminution of the affected side; but the same thing happens in old cases of pleurisy. *Percussion* reveals dulness over the consolidated parts. *Auscultation* may discover the form of breath-sound to be bronchial, tubular, amphoric, or cavernous; and the vocal resonance to be increased (bronchophony). The "metamorphosing murmur of Seitz" is a murmur, very harsh during one-third of the inspiratory part and then replaced by bronchial breathing in the remaining two-thirds. It is sometimes heard when there is a cavity. In large cavities whispering pectoriloquy is often heard, although it is not absolutely diagnostic of a cavity. It is sometimes *not* heard when a cavity is

present. The *accompaniments* vary from simple crepitations (fine and coarse, or consonating) to the large mucous *gurgling* râles heard in cavities.

The heart-sounds are sometimes very distinctly heard, owing to the consolidated lung being a better conductor; and a systolic pulmonary murmur is often present, due to compression of the pulmonary vessels by the consolidated tissue around them. The percussion dulness is never so absolutely toneless as in pleuritic effusions. The "cracked-pot" sound (*bruit de pôt fêlé*) is frequently elicited in percussing over a cavity. It should be noted, however, that it is sometimes heard on percussion over effusions and in pneumonia; or on percussion over the chest of a screaming child. Comparison of the state of the two lungs is more difficult when both lungs are affected, the normal standard of comparison being lost. In the very chronic fibroid forms of phthisis there is great dulness and shrinking; and the heart, stomach, or liver may be drawn up by contraction of the fibrous tissue.

The **prognosis** in phthisis is always grave; but as regards the duration of life it is almost always doubtful, and it should be very cautiously given. A severe acute phthisis may become chronic; and the prognosis in the third stage may be more safely given than in the early stages, as the disease is more likely now to remain chronic. They are carried, as it were, past the period when "acute galloping consumption" is probable. The temperature and its course, and the pulse, are important guides. Ordinary cases have intervals of comparative health; but the duration is very variable, and the end is often sudden. Syncope frequently terminates a case. Œdema of the ankles, in advancing disease, may be due to cardiac debility or to Bright's disease. It is an unfavourable symptom. Waxy disease, ulcers, or simple catarrh of the bowels, producing frequent attacks of diarrhœa, may terminate a case by exhaustion.

The rupture of a cavity into the pleura gives rise to pneumothorax, and if old adhesions have formed there, the air may make its way into the subcutaneous tissues—*subcutaneous emphysema*—if both layers are ulcerated through. Pulmonary embolism, resulting from thrombosis of the femoral veins, is a common accident. The tubercular disease may ultimately affect the meninges, peritoneum, kidney, or spinal cord. *Fistula in ano* is very commonly associated with phthisis.

The Treatment of Phthisis.—The prophylactic treatment is highly important. All children inheriting the tubercular *diathesis* should be carefully trained physically. They should have plenty of fresh air and light, and good nourishing diet; and they should be warmly clothed. Gymnastic exercises in moderation, singing, early hours, the avoidance of damp atmospheres and cold winds, are all highly important. Any tendency to catarrh must be promptly treated; while measles and whooping-cough must have careful management, especially during the later stages, and during convalescence. Children suffer much, in these times, from over-

pressure of the brain in schools, and especially is this the case with strumous and delicate children, who are often naturally bright, intelligent, and eager to learn. These should never be allowed to become fatigued with their lessons. Growing youths of the delicate type are often threatened with phthisis. The same general and hygienic considerations apply with even greater force to them. Tonics should be prescribed (as ℞ 21, 22, 23 or 2), and cod-liver oil should be taken whenever they show signs of failure of appetite, lassitude, or of anæmic debility. The cod-liver oil emulsions—especially those combined with maltine—are highly useful. Colds should never be neglected. As a preventive, cold bathing in the morning is to be recommended, but carried out through the winter only when the patient is sufficiently robust. Sub-acute or chronic catarrhal pneumonia is most frequently the beginning of phthisis—a cold "settling down upon the chest," as the patients so frequently express it. The treatment of this stage should be active. Up to this time, if the patient have received any treatment at all, it will have been for bronchial catarrh; and it may be necessary to continue the expectorant mixtures, &c., if the cough and expectoration be troublesome. As a rule, however, the sooner they are regarded as threatened with phthisis the better. The chest should be painted with iodine, or a fly-blister may be used over the affected part—generally the apex of the lung. Turpentine, eucalyptol, and copaiba, in capsules, are useful in sub-acute catarrhal pneumonia, as these drugs are eliminated by the lungs, and thus they have a local action upon them. A mixture of carbonate and iodide of ammonia—five grains of each thrice daily—is highly recommended by Bartholow. ℞ 24 is useful for the cough, and is at the same time a stomachic sedative and tonic. Cod-liver oil and iron should be prescribed. Niemeyer's pill is useful in the tubercular forms, and for the pyrexia of all forms (℞ 25). When the acute symptoms disappear the patient should be sent to the sea-side, or to some dry and healthy place inland. A long sea voyage to the Cape of Good Hope, or to Australia or New Zealand, is to be recommended (see Dr. Wilson's little book, *The Ocean as a Health Resort*, and Otter's *Winters Abroad*). No patient should be sent abroad who has marked fever, or who is subject to repeated attacks of alarming hæmoptysis. Davos, in Switzerland, has been highly extolled of late as a winter residence for consumptive patients.

During the course of phthisis various important symptoms call for special treatment. Hæmoptysis is treated with absolute rest, cold food, ice, &c. A mustard plaster over the consolidation is often useful; but, oftener, ice (in an ice bag) is more efficacious. A mixture of sulphuric acid and sol. of morphia (℞ 26) should be prescribed. If it fail, acetate of lead (℞ 9) should be tried. Mixtures containing opium, digitalis, and ergot, are sometimes ordered. In severe cases ergotine should be used hypodermically. No stimulants should be given while there is hæmoptysis. The patient should not be allowed to rise for a fortnight or longer. For the severe sweating, a pill containing three grains of the oxide of zinc is recommended.

Sponging the limbs and body with vinegar and water is useful. In severe cases atropia may be injected (℞ 27). The cough is best treated with codeia—a grain for a dose every four or six hours, in the form of a pill. Begbie's mixture is much used (℞ 24) for cough; and sometimes an occasional dose of an ordinary expectorant mixture (as ℞ 19) gives relief, by clearing the bronchial tubes. Chlorodyne — ten minims, in water—may be allowed for severe cough; but when too often given it tends to dry up the secretions. It should not be allowed when the kidneys are much affected. Gargles of bromide of potassium, or sprays, often relieve the cough. Fever is best treated by large doses of quinine (ten to twenty grains); or by antipyrin (fifteen grains every four hours). Other antipyretics may be tried. Niemeyer's pill is indicated in chronic pyrexia. Diarrhœa is sometimes very intractable, and it often resists all treatment. Mixtures containing astringents—as bismuth, opium, kino, catechu, and chalk—are often prescribed. A powder containing twenty grains of bismuth and two grains of Dover's powder is useful—every four hours. Copper, in pill (℞ 28), is often prescribed. Simple laudanum (fifteen minims), or solution of morphia (twenty minims), or one-third grain of morphia as a suppository, may all be used, if not contra-indicated by the state of the kidneys, and when simpler drugs, as bismuth and chalk, fail. The treatment of the laryngeal symptoms has been given with the laryngeal diseases. The results of injection of Koch's "tuberculine," or some modification of it, remain yet to be proved. It is possibly the beginning of great changes in the treatment of consumption.

Cancer of the Lung.—Cancer rarely occurs before the age of forty. It is common in the lung as a secondary disease to cancer of the breast. It is usually the soft variety; and it may be diffused throughout the lung or form a distinct mass. The appearance of the diseased part is like hardened brain tissue, and it is yellow-white, homogeneous, and rather firm—hence the name *encephaloid*.

The symptoms consist of the development of the cancerous cachexia, associated with progressive emaciation. Should the cancer form a tumour, there will be the usual signs of consolidation of the lung—dulness over the part, bronchial breathing and increased vocal resonance. The breath-sound may be cavernous if the mass surround a bronchus. There is more or less dyspnœa and pain, while the pressure symptoms vary, and, like aneurism, they depend upon the seat of the disease. There is no fever. *Cough* is always present, and it is hard and dry. Sometimes it is due to deposits within the lung tissue, and often it results from pressure upon the bronchi or nerves by a solid mass. Sometimes there is expectoration of rusty-coloured gelatinous matter. Enlarged and indurated glands may be found in the axilla or neck.

The disease is always fatal, and the treatment can only be palliative.

Hydatids of the Lungs. Echinococci.—The cyst contains

the embryo—the scolex with its four suckers and row of hooklets. Echinococci migrate from the intestines to the lungs. They affect usually the lower lobe. They set up, when large enough, severe paroxysmal pain in the side and back. Dyspnœa is almost always present, and at times it amounts to a feeling of suffocation. The cough is dry; but if adhesions have formed and the cyst rupture into a bronchus, there is expectoration of a large quantity of serous liquid and masses of hydatid *débris*. This may be fœtid, and it may only occur at long intervals.

Inspection may reveal a great enlargement of the affected side. There is dulness on percussion over the tumour, and increased resistance on palpation. The heart and liver may be displaced. Vocal fremitus is diminished, and the vesicular murmur is absent or replaced by bronchial breathing. The signs of a cavity may be made out if the cyst should have ruptured.

Recovery may take place if the cyst rupture and get free exit, as the cavity then contracts and cicatrises; but death may follow from exhaustion, secondary inflammation, tuberculosis, hæmorrhage, gangrene, or other intercurrent disease.

Puncture and free evacuation of the sac is the treatment. Often it is only after using the aspirator that the disease can be diagnosed—especially from pleuritic effusion. A microscopic examination, discovering the hooklets, at once clears up the matter.

Pleurisy.—In *acute* pleurisy there is first congestion of the subserous connective tissue, followed by dryness of the pleural membrane, and slight effusion of fibrinous lymph. This gives a dull lustre to the pleura. The effusion of lymph begins at the base, and rises in curved lines. It may be higher posteriorly, but this depends upon whether the patient took to his bed early or not. When the lymph is scraped off, the pleura appears unaltered, but microscopically the epithelium is seen to be in a state of cloudy swelling, and desquamating. The cells may undergo fatty degeneration, and complete absorption may take place; or small adhesions may result, the disease terminating without effusion in quantity, and is then known as *dry* pleurisy. Pleurisy *with effusion* is the inflammation of the pleura associated with the out-pouring of serous fluid, so as more or less to fill the cavity. The fluid is straw-coloured, sometimes tinged with blood (hæmorrhagic form); and it may be purulent from the first, or may soon become so (empyema). The hæmorrhagic form is usually tubercular in its origin.

The lung at first floats, but ultimately it becomes flattened against the spine, about its roots. The sound lung is often congested and œdematous. If the right pleural cavity be affected, the effusion pushes the heart to the left, and the diaphragm and liver down; if the left side, then the heart is pushed to the right, and the spleen dulness may be made out to be lower than normally. The intercostal spaces bulge, and the thorax has a globular appearance on the affected side. The effusion may become emulsionised and absorbed; but more usually the disease becomes chronic, and absorption takes

place only in part, while adhesions form and enclose the fluid. These chronic encapsulated effusions may remain in this condition for years, with occasional exacerbations. The adhesions may ultimately produce depression of the ribs and distortion of the spine. The purulent form (empyema) may ulcerate through the pleura and thoracic wall (fistula), or it may burrow into other regions and light up fatal inflammation. It sometimes makes its way into a bronchus and is expectorated, with relief of the symptoms. In very favourable cases, the chronic form may end in organisation, and a permanent fibrous union of a much thickened pleural membrane terminates the disease by complete obliteration of the sac.

The chief **cause** of primary pleurisy is exposure to cold, especially when the body is perspiring. It is common in early life, and a predisposition, or constitutional condition, seems, sometimes, to favour an attack. Secondary pleurisy is far more common, and it often occurs along with bronchitis, pneumonia, catarrhal pneumonia, pericarditis, embolic pneumonia, or pyæmia, &c. Caries of the ribs, cysts of the liver, &c., may light up a pleurisy. It is also very frequently associated with Bright's disease, gout, rheumatism, diabetes, cancer, and with eruptive fevers.

The **symptoms** may begin with a feeling of chilliness followed by fever, with sharp lancinating pain in the side; but sometimes there is only sudden sharp pain without warning or fever. The pain is generally at the side and base of the thorax. *Friction*—from a slight rub to coarse creaking—is heard over the seat of the pain, but in the very early stage it may be absent at the first examination. If the friction-sound be coarse its vibration may be felt (*Friction fremitus*). Dyspnœa is present, and the breathing may be characteristic. The respiration is shallow and rapid, owing to deep inspiration causing sharp pain. In drawing a long breath there is a sudden "check" in the breathing, accompanied often by a jerking in of the thorax. The attack may terminate by resolution or by the formation of adhesions; but often it becomes a case of **pleurisy with effusion**. This form, however, does not *always* begin with pain in the side. There are often feverish attacks and irregular chills without any local signs or symptoms. The fever continues with evening exacerbations—the temperature ranging from 101° to 104° Fahr. Should there be severe rigors, the effusion is probably purulent, or the disease may be tubercular in its origin. The pain is felt below, and to the outside of the mammary region, sometimes at the back, and it is occasionally referred to a point much lower, as the lumbar or iliac regions. (In "diaphragmatic pleurisy" the pain is often referred to the pit of the stomach, and the other symptoms are often obscure.) The pain is much increased by coughing, but it is generally absent after the effusion has taken place. The cough is *suppressed*, and there is little expectoration unless bronchitis be also present. The cause of the dyspnœa is three-fold, viz.—the shallowness of the respiration hindering the proper aëration of the blood; fever increasing the necessity for oxygen; and the effusion interfering mechanically with the lung.

The *decubitus* in cases of pleuritic effusion is suggestive. The patient lies on the affected side to allow more freedom to the sound lung. According to Traube, the patient in the early stage of pleurisy lies on the sound side, as the blood gravitates from the diseased side and relieves the nerves from pressure; but just as often the patient prefers to lie "on his pain," which helps to check the full expansion of the chest. The expression of the face is anxious and weary, pale or cyanosed. The digestion is impaired; and the urine is scanty and loaded with urates.

The physical signs of pleuritic effusion are very definite. *Inspection* reveals non-expansion of the affected side, and if the effusion be great in quantity, there is bulging of the thorax and intercostal spaces. *Palpation* not only confirms what is seen, but may also yield a sense of increased resistance or fluctuation. The vocal fremitus is also diminished or absent—a very important sign in the differential diagnosis. The *percussion* is dull, and when there is great effusion it is absolutely flat and toneless. In the early stages, when the effusion is limited, the percussion-tone is tympanitic and high pitched (see **Percussion**), particularly in the infraclavicular region of the affected side. Sometimes the "cracked-pot" note can be elicited in cases of pleuritic effusion. The various displacements of the heart, liver, spleen, &c., can also be made out by careful percussion. On *auscultation* the vesicular breath-sound is absent, or replaced by bronchial or tubular breathing, and if the lung be completely collapsed there is absolute silence over the effusion. The vocal resonance is also diminished, and it is often ægophonic, or has a peculiar whiffing character. In the latter case when the patient is asked to say *one*, *one*, *one*, it sounds like *one-f*, *one-f*, *one-f*. Bronchophony is present when the effusion is moderate in quantity, and it is often heard at the root of the lung even when there is a large effusion.

When absorption takes place the physical signs return in reversed order, and, if complete, friction is heard again, as in the early *dry* stage. Fine crepitations and râles are also heard when the lung begins to expand.

The *course* of a pleuritic effusion is slow. Beginning in one or other of the methods described, there are three or four days of gradually increasing fever, and then *continued* fever for about ten days. When effusion takes place rapidly the temperature falls. Absorption may be rapid at first; but slow absorption is the rule. The urine increases in quantity, and sometimes it contains albumen. The effusion may take months to completely absorb, but during this time additional attacks of inflammation often serve to again increase it, and the case becomes very chronic. Rigors indicate that the fluid has become purulent, and an empyema is then established. Œdema of the sound lung is highly dangerous in cases of pleuritic effusion, and it may terminate the case very rapidly. The chronic forms, by leading to purulent infiltration or tuberculosis, cause chronic ill health, and death by gradual exhaustion. The inflammation may extend to the pericardium. Pneumonia may co-exist with the pleurisy—*pleuro-pneumonia*.

The Treatment of Pleurisy.—When the attack manifests itself by severe stabbing pain in the side, a fly blister is the best treatment; and twenty grains of quinine should be at once administered. Leeching, or cupping, is sometimes good practice. A hypodermic injection of half a grain of morphia should be given before applying the blister. Smaller doses of quinine should be given thereafter, and a mixture of iodide of potassium (℞ 29) prescribed. A sharp saline purge is useful. A few drops of chlorodyne should be allowed for the cough. If the pleurisy be still *dry*, but affecting several points over the chest, the strong liniment of iodine or turpentine stupes, should be used externally. Diuretics, as acetate of potash, nitrate of potash, and digitalis, are sometimes ordered; and pilocarpine is sometimes given to produce diaphoresis—one-eighth of a grain hypodermically. In the more chronic forms of dry pleurisy, mercurial ointments may be rubbed into the chest with benefit. The diet should be light; and *dry food* suggests the possibility of limiting the effusion.

The *medical* treatment, when there is pleurisy with effusion, is the same; but the question of *paracentesis thoracis* remains to be considered. It is chiefly with regard to the time most suitable for tapping, that there is a difference of opinion. An effusion should not be tapped too early, as complete absorption may take place without interference. If the fluid be purulent, it should at once be removed, as absorption cannot take place. After a reasonable time (say a fortnight) without any signs of absorption, and if the fluid be considerable in quantity, *paracentesis thoracis* should be performed, before there is any chance of the lung being bound down by adhesions. It should also be performed—at any period during the course of the effusion, early or late—when there is embarrassment of the heart and dyspnœa, and also when one side of the chest is full, even without urgent symptoms, as the patient is in danger of his life from sudden syncope. The needle is usually inserted just below the inferior angle of the scapula, care being taken to keep close to the upper border of a rib, to avoid wounding the intercostal artery immediately above. A preliminary exploration with a hypodermic needle may be made. The fluid should be drawn off slowly, but no attempt need be made to withdraw the whole of it as absorption frequently takes place after the removal of a small quantity. The operation may be repeated again and again, if necessary. When the fluid is purulent, attempts may be made to remove it by frequent tappings; but generally, opening between the ribs, washing out with antiseptic lotions, and the establishing of free drainage, is the best treatment. Draining away, by means of a long elastic tube to a jar of antiseptic fluid placed under the bed, is sometimes very successful in chronic empyemata occurring in children. The negative pressure expands the lung. In many cases of empyema, further surgical treatment becomes ultimately necessary.

If paroxysmal cough be excited during the operation of tapping, a hypodermic injection of morphia will relieve it. The subsequent treatment of the case is *as before*—frequent paintings of iodine, or

friction with mercurial ointments. The syrup of iodide of iron (℞ 30) and cod-liver oil should be ordered. Inhalations of compressed air may be tried. Residence for a time in a dry mountainous district may complete the cure.

Hydrothorax. Pneumothorax. Hydro-pneumothorax. Pyo-pneumothorax.—*Hydrothorax* is produced by the effusion of serum into the pleural cavities, and it arises from purely mechanical causes. It is usually bilateral, and is due to obstruction or interference with the circulatory apparatus—*e.g.*, emphysema, cirrhosis, tumours pressing upon the large veins; and especially is hydrothorax liable to occur in the course of *cardiac* and *renal disease*. It may arise also as part of a general dropsy, whatever be the cause.

When the effusion is rapid, sudden dyspnœa is the most striking symptom, and if large in quantity death may be sudden. The physical signs are the same as in pleurisy with effusion; but being usually bilateral, there is no displacement of the organs.

Pneumothorax arises when air or gas accumulates within the thoracic cavities. *Hydro-pneumothorax* is the accumulation of serum and gas; and in *pyo-pneumothorax* pus and gas are present. The air may enter through the wall of the thorax after an injury; but most frequently pneumothorax results from rupture of the lung, a common event in phthisis after the formation of cavities. An empyema—or an abscess of the liver, kidney, or of a bronchial gland—burrowing into a bronchus, may produce a pyo-pneumothorax. The amount of gas retained within the thoracic cavity depends upon the compressibility of the lung. If there be much consolidation, or if old adhesions be present, the amount of gas may be small. As the lung collapses—in cases of rupture of the lung—the orifice through which the air has entered is closed, and a valvular arrangement is established. If the inlet be easy and the exit difficult, there is a "packing" of air within the cavity, and this gives rise to characteristic physical signs. The pleura may become inflamed and a purulent exudation is then poured out, the contained air or gas acting as an irritant to the pleural membranes.

Pneumothorax often develops insidiously when associated with phthisis, but sometimes it occurs suddenly, and there is then severe pain, great dyspnœa, and symptoms of collapse. The skin is covered with cold sweat, the pulse and respirations are rapid, and cyanosis ushers in a fatal termination. Death may occur more slowly by venous stasis and general œdema. *Palpation* reveals increased tension and diminished resistance where there is gas. The *vocal fremitus* is diminished or absent, only in those cases in which there is more or less complete collapse of the lung. When old adhesions and consolidation are present the vocal fremitus may be as strong as before. When fluid is present along with gas, *succussion* is a characteristic sign, and it is best heard by shaking the patient suddenly, while the examiner's ear is applied to the chest. The percussion-tone over gas is tympanitic and resonant. The liver

dulness may be extinguished in extreme cases of pneumothorax of the right side. When fluid is present—*hydro-pneumothorax*—there is dulness, and signs similar to pleurisy with effusion, over the lower part of the thorax. In pneumothorax, percussion with two coins, while using the stethoscope, produces a well marked *metallic echo. Auscultation* reveals *amphoric* breathing; while the heart-sounds, râles and cough, all have a metallic quality. The *vocal resonance* is increased and echoes through the cavity. The dropping of fluids produces *metallic tinkling.*

The prognosis (in all) is generally unfavourable; but in phthisis, pneumothorax, by giving *rest* to the lung, may improve matters for a time. In other cases, death generally results from secondary pleurisy within a few weeks.

Hydrothorax, pneumothorax, and hydro-pneumothorax may require to be relieved by aspiration, if the dyspnœa be very great. Inhalations of chloroform, or a hypodermic injection of morphia, may be given. Pyo-pneumothorax is treated by a free opening and drainage, as in chronic empyemata.

Pleurodynia, or pain in the side, is frequently the result of a strain. It is sometimes rheumatic in its origin. It requires to be differentiated from acute pleurisy by careful auscultation; and likewise from intercostal neuralgia, early herpes zoster, and surgical affections of the ribs. The pain is in the muscles in true pleurodynia, and it is relieved by firm pressure.

Belladonna liniments or plaster, mustard poultices or a blister, generally relieve. A tight binder is useful, and iodide of potassium with salicylate of soda (10 grs. of each) may be given in obstinate cases, twice or thrice daily, for a few days.

The Classification and Diagnosis of the Diseases of the Pulmonary Organs.—The diseases of the bronchial tubes, lungs, and pleuræ are closely associated, and it is rather the exception than the rule to find one single and uncomplicated affection.

The continuity of the respiratory tract makes it obvious that an inflammation of any part, by simple extension, may affect the other tissues, and hence a bronchitis, pneumonia, and pleurisy, with the symptoms and physical signs of each, may all co-exist. The diagnosis may then be that of the initial, or most prominent disease, while the other pathological conditions may be viewed as secondary, if unimportant in their relation to the primary or chief disease. More frequently, however, a double diagnosis must be made and stated. Such combinations as bronchitis and catarrhal pneumonia; asthma, bronchitis and emphysema; croupous pneumonia and pleurisy; phthisis, pleurisy, and pneumothorax, hydro-pneumothorax or pyo-pneumothorax; cirrhosis of the lung and bronchiectasis; and many other, and even more complicated conditions, are very commonly met with in practice.

Without repeating the symptoms and signs of the diseases, already given systematically and in a synoptical form—a few words only need be said upon the important points of difference between those

affections having some resemblance to each other in their symptoms, and which may possibly lead to errors in diagnosis.

The classification given here is purely a clinical one, and it has been framed for clinical purposes alone. An attempt has been made to preserve order in the mind, by arranging the diseases with regard to the commonest sequence of events. Sometimes it is the resemblance of the symptoms—especially in the early stages of disease—that has induced me to place them together; or a prominent symptom has served, as in the *fœtid* group, as a basis for classification. The whole arrangement has been made with a view of assisting the diagnosis, by bringing allied affections closer to each other, and thus to suggest a comparison and exclusion of different diseases.

The diseases of the pulmonary organs may be tabulated in the order in which they have been discussed throughout this work, viz. :—

I. **Neurotic.**—Pertussis. Asthma (and hay asthma).
II. **Acute.**—A. Acute bronchitis. Fibrinous bronchitis. Pulmonary collapse (Atelectasis). Acute catarrhal pneumonia.
B. Congestion of the lungs. Œdema of the lungs. Croupous pneumonia. Embolic pneumonia (Hæmorrhagic infarction).
III. **Chronic.**—Chronic bronchitis.
Sub-acute and chronic catarrhal pneumonia.
Chronic interstitial pneumonia (Cirrhosis).
Emphysema.
IV. **Fœtid.**—Fœtid bronchitis.
Bronchiectasis.
Gangrene of the lung.
V. **Phthisis** (including Miliary Tuberculosis).
VI. **Special.**—Cancer; and Hydatids of the lung.
VII. **Pleuritic.**—A. Pleurisy—Acute; with effusion; Empyema.
B. Hydrothorax; Pneumothorax; Hydro-pneumothorax; Pyo-pneumothorax.
C. **Pleurodynia.**

In the early stages of *whooping-cough*, a positive diagnosis may be impossible as the case may prove to be a simple catarrh, or it may be the beginning of a graver disease, as bronchitis and catarrhal pneumonia. A dry incessant cough, worse at night, is suggestive. In *asthma*, the cardiac and gastric forms require to be differentiated, and œdema glottidis, paralysis of the vocal cords, aneurisms pressing upon nerves, and stenosis of the trachea, all produce dyspnœa. In true asthma, however, the dyspnœa is *expiratory*. Cardiac dyspnœa, emphysema, and bronchitis have other physical signs; and the latter disease is invariably associated with spasmodic asthma, and may itself be the cause of the seizure. The history of the *onset* is important. Nasal polypi should be noted as a possible cause of asthma. *Hay asthma* requires to be distinguished from a *simple* coryza (*vide*).

Acute bronchitis may begin with tickling cough and coryza, and the serious symptoms may develop later. *Fibrinous bronchitis* is

only diagnosed from the expectoration; but the character of the dyspnœa may be suggestive. *Collapse* of the lung superadded to bronchitis and pertussis is generally temporary, and increased dyspnœa, with sometimes signs of consolidation—in children especially—may be observed. *Catarrhal pneumonia* existing with bronchitis raises the temperature and increases the dyspnœa. Sometimes patches of consolidation can be made out. The râle in catarrhal pneumonia is *sub-crepitant*, and not so coarse as in simple bronchitis of the larger tubes. The capillary form of bronchitis is common in children, and produces greater dyspnœa than when the larger tubes are affected. It is sometimes difficult or impossible to differentiate a capillary bronchitis from a catarrhal pneumonia. They may co-exist. In the early stages of croupous pneumonia, especially if the disease begin deep in the lung, the case may resemble acute bronchitis; but the mode of onset, the sudden rigor, dull pain in the side, the fine crepitations, with subsequent development of the physical signs of consolidation, soon clear up the case, if a typical one. But it may prove to be simple acute congestion of the lung, if there have been no marked rigor, and if the physical signs of croupous pneumonia do not subsequently develop. In bronchitis the râles are *general*, and not localised. Phthisical crepitations and râles *are* localised—most frequently at the apex, but sometimes at the base of the lung. An intercurrent attack of bronchitis may mask a case of phthisis, but the signs of consolidation may be discovered; or after a week of treatment, the general bronchitis may clear up and leave the localised râles. There is no percussion dulness in simple bronchitis. Acute tuberculosis is sometimes very difficult to differentiate from bronchitis; and so also is diffuse cancer of the lung. In acute tuberculosis the course and severity of the fever, the sweats, the colour of the lips and nails, or the consonating character of the râle, may suggest a correct diagnosis; in cancer (diffuse) the cachexia and emaciation are important signs. Bronchitis is more frequently a secondary rather than a primary disease, and hence the exanthemata, rickets, chronic valvular disease of the heart, and especially Bright's disease, must always be kept in view.

Collapse of the lung has a definite cause, and it may be permanent after long-continued whooping-cough. In after years, signs of slight consolidation at the apex, with weak breath-sounds in that region, should not be mistaken for phthisical consolidation. There are no crepitations in such a case. A large collapse, during an acute disease, might be mistaken for pneumonia by presenting the signs of consolidation, but the history and onset will serve to distinguish them. The dyspnœa in collapse is *suddenly* increased.

A *catarrhal pneumonia* may require differentiation from a deep croupous pneumonia, and from acute tuberculosis. The distinguishing signs have been alluded to when speaking of their relations to bronchitis. Pneumonia in children is often ushered in with convulsions, and the case may look like one of tubercular meningitis.

There is practically no difference in the initial symptoms of *acute*

congestion or *hyperæmia of the lungs*, from the first stage of croupous pneumonia. There is no marked rigor in congestion, and it is only the subsequent course that reveals the true nature of the disease—*i.e.*, in croupous pneumonia the development of the physical signs. *Œdema* of the lungs is generally bilateral, and it affects both bases. The dyspnœa is increased, and the expectoration is frothy.

Croupous pneumonia may resemble a pleuritic effusion, especially if there be slight effusion of lymph associated with the pneumonia; but in croupous pneumonia the rigor, fine crepitations, rusty sputum, bronchial or tubular breathing, and subsequent course, with no displacement of the organs—serve to distinguish the two diseases. In pleuritic effusions bronchial breathing may be heard when the fluid is moderate in quantity; but later, it disappears when the effusion is greater. The two diseases—pleuro-pneumonia—may exist together. The presence or absence of vocal fremitus is important in relation to consolidation *v.* effusion. Sometimes an acute onset in croupous pneumonia, with head symptoms, suggests a meningitis. The chest should be examined. In aged persons and in drunkards there may be no cough to draw attention to the pneumonia. In the early stage, acute bronchitis and congestion of the lungs have to be remembered in relation to the diagnosis. *Embolic pneumonia* (hæmorrhagic infarction) is differentiated by the history and onset.

Chronic bronchitis requires the same differentiations to be made as in acute bronchitis. As bronchitis is frequently associated with collapsed lobules of the lung, and patches of catarrhal pneumonia—which may at any time undergo caseation—the differential diagnosis of these conditions from phthisis is a very frequent necessity in practice. It is only the very early cases of phthisis which present any difficulty, when dulness upon percussion, and the other physical signs of consolidation, are doubtful. In these cases only the discovery of lung fibres in the sputum, or the presence of the bacilli, can put the matter beyond doubt. The detection of a finer râle at the apex is suggestive, but it only means that there is a small patch of catarrhal pneumonia in that region (which may or may not become caseous), or that the finer bronchial tubes are affected. Very frequently indeed, in cases of chronic bronchitis, auscultation reveals these sub-crepitant râles, not only at the apex, but at the base, and in small areas over the back. Often, too, there is a little friction rub indicating that there is a small patch of pleurisy as well. In such cases the diagnosis is chronic bronchitis, with either patches of catarrhal pneumonia or catarrh of the small tubes, with pleurisy. When pleurisy is made out it is difficult to believe that the lobules have escaped entirely. Without the pleurisy the sub-crepitant râle may mean either capillary bronchitis or catarrhal pneumonia. *Chronic pneumonia* with bronchitis is understood to be chronic interstitial pneumonia or cirrhosis of the lung, when signs of the development of fibrous tissue are present. It may also be taken to mean secondary catarrhal pneumonia, especially when small pleurisies are present, and before the symptoms of actual caseation develop,

when it becomes a phthisis—*basic* or *apical*. It is often difficult to diagnose saccular dilatations of the bronchi from phthisical cavities. Fibres of lung-tissue found in the sputum decide the matter; and the history of the course of the two diseases may be entirely different.

Chronic interstitial pneumonia is known by the slowness of its development, the history of antecedent chronic bronchitis, or long exposure to irritating dusts, or alcoholism; later, by the signs of phthisis with the displacements due to contraction. In the early stages it can only be surmised, and not until the consolidation diminishes the sonority of the percussion-tones, can it be positively diagnosed. Dilatations of the bronchi are commonly present with cirrhosis.

Emphysema is distinguished from asthma by the spasmodic character of the dyspnœa in the latter disease, while there are no alterations in the shape of the chest. Bronchitis is very often present with emphysema. The physical signs of emphysema are usually clear enough to distinguish it from all other diseases. Pneumothorax may resemble it in having a resonant note on percussion, but it is unilateral, while emphysema is bilateral, and the other physical signs are very different. Cardiac dyspnœa will have the physical signs of valvular disease. There is a cycle of diseases very commonly met with in practice, viz.:—bronchitis, asthma, emphysema, cardiac dilatation, Bright's disease, and dropsy—which requires careful examination, and inquiry into the mode of origin and history, before making a diagnosis. It may be difficult to decide what is the initial disease, in a chronic case presenting itself to the physician for the first time.

The members of the **fœtid group** can hardly be mistaken for other diseases, except phthisis, in which sometimes the expectoration is also fœtid. The *manner* of expectorating in bronchiectasis, with the changeable character of the physical signs, distinguishes that disease from the others of the group. In gangrene of the lungs the severity of the symptoms, associated with the strong odour, will usually make the diagnosis easy. Phthisis may resemble it, but the symptoms are never so severe, and the odour in gangrene is horribly intense. Simple fœtid bronchitis has the expectoration separating into three layers. The symptoms are not so urgent as in gangrene of the lung, and there are no signs of pulmonary disease.

Phthisis sometimes resembles typhoid or intermittent fever when the physical signs are obscure. The course and subsequent developments clear up the case, and there is a better appetite and less thirst in phthisis than in fevers. Incipient phthisis may be mistaken for atonic dyspepsia—the latter condition being common in early phthisis. In the physical examination of such cases the natural differences found on comparison of the two sides of the chest—especially the right and left infra-clavicular regions—should be carefully noted. When both lungs are affected with phthisis, the physical signs are

sometimes not so clear, owing to the normal standard of comparison being absent. The dulness upon percussion may then not be so obvious, and the diagnosis must rest upon the presence of other physical signs, and the symptoms. The differential diagnosis of phthisis has been also alluded to in the paragraphs immediately preceding (*Bronchitis*, &c.) The differences between caseous and tubercular phthisis may be tabulated thus, viz. :—

Caseous.	Tubercular.
Acquired by the strumous, . .	Hereditary.
Begins as bronchitis and catarrhal pneumonia, ...	Begins insidiously, but sometimes as acute tuberculosis.
Occurs in youth or middle age, .	Occurs any age.
Hæmoptysis not so common, .	Very common.
More frequently unilateral, . .	Bilateral.
Physical signs marked, . .	Sometimes not.
Larynx not affected, . . .	Laryngeal phthisis often.
Progress slow, ...	Often rapid.

It is convenient to tabulate here also the most important causes of Hæmoptysis, viz. :—

1. Inflammations and ulcerations of the nasal, laryngeal, tracheal, or bronchial mucous membranes.
2. Cardiac disease, with congestion or hæmorrhagic infarctions of the lungs.
3. Phthisis.
4. Cancer of the lung, or of adjacent structures, ulcerating into the bronchial tube.
5. Gangrene of the lung.
6. Rupture of the lung by violence.
7. Aneurisms (rupture of).
8. Certain blood diseases, as scorbutus, purpura, leucocythæmia, &c.
9. Degenerative changes within the pulmonary blood-vessels themselves.

The blood may come from the mouth in cases of caries of the teeth, or in ulcerations of the gums in strumous children. Blood from the stomach (hæmatemesis) is black, acid, and contains food and no air; while from the respiratory passages it is red, alkaline, and frothy. It is *coughed* up when from the lungs, and there is no nausea. It is sometimes impossible to say whence the blood comes, and it is only the subsequent developments that reveal the cause. In practice phthisis is by far the commonest cause, and next to it, cardiac disease.

Pleurisy with effusion requires to be distinguished from consolidation of the lung (see *the differential diagnosis of croupous pneumonia*), and from tumour, abscess, or cyst. Abscess of the liver, hydatid cysts, and enlargements of the kidney, differ in their history. Tumours cause irregular bulging of the thorax, and the vocal fre-

mitus is increased; while in pleuritic effusion it is absent. The effused lymph in pleurisy takes a definite course, rising in circles from the base of the thorax. The "diaphragmatic effusions" may give rise to pain in the pit of the stomach, and in the absence of all other symptoms, may very readily escape diagnosis. The temperature should be noted, and a careful physical examination made of the heart and lungs. The progress should be watched. Aspiration may be necessary before a positive diagnosis of pleuritic effusion can be made. Repeated rigors in a case of pleurisy indicate the formation of pus (empyema). If sudden pallor and syncope should occur, with first a fall of the temperature, followed soon by a rise, during the course of pleurisy—hæmorrhage has probably occurred. The disease then is probably tubercular in its origin. *Hydatid cysts* may give rise to signs like pleuritic effusion. There is no fever, and they develop slowly. Aspiration may remove all doubts.

Pneumothorax may be mistaken for very large vomicæ; but the latter are almost invariably at the apex. Cavities form slowly, the chest-wall is retracted, and the vocal fremitus is marked. The opposite conditions are present in pneumothorax. If fluid be present in the chest (hydro-pneumothorax), succussion is often heard; and this is rare in vomicæ, however large.

CHAPTER V.

THE URINE.

Contents.—General remarks—Quantity, specific gravity, colour and transparency, odour and re-action—**Albuminuria**—**Peptonuria**—**Urea**, and its estimation—**Sugar**, and its estimation—Tests for bile, blood, pus, urobilin, indican, and acetone—Tests for chlorides, phosphates, and uric acid—**Examination of deposits**—*Naked-eye appearances*: mucus, pus, uric and oxalic acids, urates, oxalate of lime, triple phosphates, amorphous phosphates—Urinary concretions—*Microscopic examination of deposits*—*Inorganic deposits:* uric acid crystals, urates, oxalate of lime, triple phosphates, neutral phosphates of lime, cystin, leucin, and tyrosin—*Organic deposits:* tube casts, blood corpuscles, pus, epithelium, spermatozoa—Micro-organisms, and parasites.

As so large a number of diseases during some part of their course are associated with renal changes, and as so many apparently primary affections are really secondary to kidney disease—the examination of the urine should be more or less a matter of routine. Even when negative results are obtained, as is the case in a large proportion of the cases, the information gained is often of importance in relation

to the *prognosis* as well as the diagnosis. No case can, therefore, be said to be complete without an investigation of the urinary secretion. In Bright's disease, it is, of course, of special importance; and as the symptoms in all forms may begin very insidiously, it is often only by the examination of the urine that the disease is detected in the early stages. For these reasons, therefore, it will be well for the junior practitioner to continue to a great extent the routine practice of the hospital, until experience teach him how far such an examination may be unnecessary.

When the urine is to be examined the patient should be directed to empty the bladder at a certain time, and the quantity should be collected and measured for the next twenty-four hours. He should also be directed to micturate before going to stool; and in some cases of incontinence it may be necessary to use a catheter frequently. The whole of the urine need not be kept, but a small quantity, at each micturition, may be placed aside in a cylindrical glass. By this means a fair average sample is obtained—the urine varying in acidity and specific gravity in relation to the food and liquids taken during the day. A preliminary inquiry as to the act of micturition may elicit the fact that there is *pain* or *increased frequency*. Sugar in the urine, or great acidity, may give rise to the former—and an increased volume (as in diabetes mellitus and insipidus, and in chronic Bright's disease) to the latter symptom. There are many *surgical* affections in which micturition is painful and frequent. (See Caird and Cathcart's *Surgical Handbook*.)

The examination of the urine should first include the consideration of the *quantity*, *specific gravity*, *the colour and transparency*, *the odour and re-action*.

1. **The Quantity.**—In adults the normal amount of urine passed within the twenty-four hours should average about fifty ounces. Ten or fifteen ounces, *less* or *more*, may be allowed for the variations, which depend chiefly upon the character of the diet, the quantity of fluid imbibed, and the occasional withdrawal of the fluid through other channels.

The urine is *diminished* in heart disease; in acute Bright's disease, and in the latest stages of the chronic forms; in renal colic; in diarrhœic affections, and after effusions and hæmorrhages; in irritating nervous reflexes and shock; in severe anæmic conditions; in fevers; and from mechanical and surgical causes. Complete suppression may also result from the action of drugs (cantharides, turpentine, &c.). The urine is *increased* in diabetes mellitus and insipidus; in the chronic forms of Bright's disease, and in cystic disease of the kidneys; and by diuretics.

2. **The Specific Gravity.**—This varies in health and it should always be considered with the quantity of urine passed. Normally, the larger the quantity of fluid, the lower is the specific gravity. Taking fifty ounces as the average quantity passed in the twenty-four hours, the specific gravity should be about 1,020. It is estimated by the *urinometer*—care being taken that it do not touch the sides

of the glass vessel. All froth should be previously removed from the surface of the urine to allow of a clear reading. Any departure from these normal relations indicates an increase or a diminution of the solids excreted by the kidneys. When the specific gravity is *low* (in relation to the quantity of urine), it indicates either diminished tissue change throughout the body, or the failure of the kidneys to excrete the urea and uric acid products. A low specific gravity is often a precursor of uræmia (v. Jaksch). The specific gravity is *high* in diabetes (sugar), and in fevers (urea). It is *low* in chronic kidney affections.

3. **The Colour and Transparency.**—The colour of normal urine is characteristic, and is due to the pigments. The urine is pale in diabetes; in chronic Bright's disease; anæmia; and after "neurotic" seizures. It is dark when concentrated, as in fevers, diarrhœa, and dyspepsia, &c. In grave organic disease the deposits are sometimes much darker from adherent pigments (purpurine, melanin, &c.). Rhubarb and senna, when taken medicinally, colour the urine a light brown; santonin colours it yellow. When carbolic acid is absorbed, the urine is olive-green in colour. *Blood* in small quantity, imparts a "smoky" tinge to the urine; when in large quantity, the urine is bright red. *Bile pigments* in the urine give it a yellow-green appearance, well seen when the vessel is slightly agitated. The urine is normally *transparent*, but the presence of pus, mucus, or oil globules may render it opalescent; and after cooling, the cloud may be due simply to urates. Alkaline urines are generally clouded with pus, &c. Albuminous urines, and urine containing bile, urobilin or indican, are often *frothy*.

4. **The Odour.**—Normal urine has a characteristic odour. Decomposed urine smells of ammonia. Bloody urine is very offensive. Turpentine, when taken medicinally, imparts an odour of sweet violets to the urine; and copaiba, cubebs, sandal-wood oil, &c., may be detected by the smell, in the urine of patients taking these drugs.

5. **The Re-action.**—Healthy urine is slightly acid. It is tested by red and blue litmus papers. The urine normally becomes less acid after a meal, and its re-action is subject to variations during the day. It becomes alkaline on standing for some time. In fevers the acidity is increased; and in severe anæmic conditions the urine is often alkaline. The latter condition has been pointed out by von Jaksch, as suggesting a ready means of determining whether the morbid process in chlorosis is continuing or not. Ammoniacal urine is frequently induced by the use of a dirty catheter.

Albuminuria.—The presence of albumen in the urine must always be regarded as a serious morbid symptom, although it does not always indicate structural changes within the kidneys. It is believed by some to be occasionally present in healthy urine, but only in small quantity. In the latter case, it may only be temporary, and it is supposed to be due to disturbances of the circulation affecting particularly the renal organs. In estimating the value

of albuminuria as a symptom in medical cases, due regard must be given to these so-called *accidental* causes; and the surgical affections (inflammations of the urinary tract—*e.g.*, pyelitis, cystitis, urethritis, vaginal discharges, &c.), must be excluded. In such cases the albumen present is small in quantity, and the microscopic examination, with the absence of tube casts and the discovery of pus cells, &c., will enable the physician to discriminate these conditions.

Serum-albumen and *peptone* are the two varieties which are important—especially the former. Other albumens have been separated, but they need not be considered here as they do not as yet come within the range of practical medicine.

The most common cause of albuminuria is kidney disease. The amount of albumen present, however, is no guide to the severity of the lesion—at least in chronic cases. Albuminuria occurs also in acute fevers, and then it is due to secondary structural changes within the kidneys; in severe anæmic conditions—allowing of *exudation*; in heart disease, emphysema, &c.—from mechanical causes; and it is often present in epilepsy and other paroxysms. It may sometimes be present temporarily from some error in diet—a highly albuminous food (pastry, &c.) frequently producing it. A turbid urine in the morning with albumen in small quantity—*then*, but at no other time—is due generally to inflammation of the prostatic or other portion of the urethra. Sometimes in kidney disease—even in *acute* nephritis—the albumen may be absent in some of the urine passed.

Tests for Albumen.—(A) *Nitric acid.*—Take about an *inch* of urine in the test-tube and allow the nitric acid to run slowly down the inside of the tube. It will form a layer at the bottom, and if albumen be present a white ring forms, more or less dense according to the quantity present, and extending gradually *upwards* through the urine. To add too small a quantity of nitric acid, or to allow the acid to mix with the urine, may spoil the test. A *brown ring* (urohæmatine) between the nitric acid and the urine is seen to form when only a trace of albumen is present, or when the urine contains none. In applying this test, very turbid urines require to be filtered. When the albumen is present in very small quantity, allow half an hour to elapse for the formation of a ring. There are three sources of fallacy in this test:—(1) Concentrated urine may precipitate urates, but *heat* clears these up, and, moreover, the precipitation begins at the surface of the urine and extends *downwards* (Roberts); (2) nitrate of urea may form, but this precipitate also clears up with gentle heat; and (3) resinous matters in the urine precipitate with nitric acid. (The *quantity* of albumen present is usually estimated by the careful application of this test.)

(B) *Boiling Test.*—The urine in this test must be slightly acid. It is well to add a drop of acetic acid in every case, and more must be used should the urine be alkaline. The upper part of the column of urine may be heated and the opalescence (when albumen is present) contrasted with the urine in the lower part of the tube. Should precipitation occur on boiling, acetic or nitric acid should be cautiously added to the boiled urine. If the precipitate be phos-

phates, they dissolve—if albumen, the coagulation remains. The chief source of fallacy arises in connection with the relative amount of acid used. If the quantity of albumen present be small in amount, the acid forms a soluble nitrate; and if phosphates be present as well as albumen, and the acid added be relatively small in amount, soluble albuminates may result. *Resins* are also a source of fallacy. These are soluble in alcohol—albumen is not.

(C) *Acetic Acid and Ferrocyanide of Potassium.*—After filtering the urine, if necessary, add a considerable quantity of acetic acid, and then a few drops of a 10 per cent. solution of ferrocyanide of potassium. If albumen be present in quantity a white precipitate forms—if only a trace, the urine becomes opalescent. This test may be applied by first preparing a fresh solution of the acid and ferrocyanide, and then adding it slowly (as in the cold nitric acid test). A ring forms between the urine and re-agent.

(D) *Biuret Test.*—Treat the urine with caustic potash, and then add cautiously drops of a solution of copper sulphate. The green precipitate which forms is dissolved if albumen be present, and the fluid assumes a red-violet colour.

It is unnecessary to allude to the other and numerous tests for albumen, some of which are, however, very delicate. The above will serve all practical purposes. The elaborate *quantitative* analysis of albumen is a matter for the laboratory. The cold nitric acid test gives it approximately, or the *picric acid* test with Esbach's albuminimeter may be used—directions for using being purchased with the tube.

Peptonuria.—The discovery of a simple test for peptone in the urine, by Hofmeister, and the subsequent investigations by von Jaksch, have brought this subject before the practical physician. According to von Jaksch—from whose work the following is taken—"the causes of peptonuria are quite different from those to which the other forms of albuminuria are due." It appears in the urine commonly, but not invariably, when suppurative processes (pneumonia, phthisis, purulent pleuritic effusion, purulent meningitis, &c.) are present in some part of the system, and when the products of such suppurations (peptone) pass into the blood, and are eliminated by the kidneys (*Pyogenic peptonuria*). Its detection is an indication of such suppurations, and, therefore, *peptonuria* has a high degree of significance in clinical medicine. Peptonuria, however, occurs also in severe cases of scurvy, and it may be present in ulceration of the intestine, in phosphorus poisoning, and in puerperal states. Excluding these, the discovery of peptonuria will indicate the stage of softening in pneumonia, or that purulent changes have occurred in pleuritic effusions, or in abdominal tumours, &c.

Tests for Peptone.—The urine should be tested for albumen by tests B, C, and D. If B and C give no results, add acetic acid alone to a fresh sample of the urine, and, if no precipitate form, apply the biuret test (D). The latter test will detect peptone if present in sufficient quantity, but the fluid becomes red instead of violet. A second test is to add first a concentrated solution of acetic acid, and then a mixture of acetic and phospho-tungstic acids. If peptone

be present, a clouding takes place either directly or shortly afterwards. Small quantities of peptone may also escape this test.

When serum albumen is present, it requires to be removed by precipitation with a metallic oxide, and then filtering.

Hofmeister's test is more accurate, but it is very elaborate (see v. Jaksch).

Urea.—The significance of an increase or diminution of this body in the urine has been mentioned with the diseases in which such changes occur. The normal amount of urea excreted by healthy adults varies from about 300 to 500 grains, or more, in the 24 hours. An albuminous diet increases the quantity. It is estimated as follows:—

Russell and West's Method.—Take 25 c.c. of the hypobromite of soda solution in the conical flask; also 5 c.c. of urine into the small test-tube, taking care that they do not mix; attach to the graduated glass cylinder in the usual way; then allow urine and hypobromite sol. to mix, and in fifteen minutes read off the quantity of gas evolved within the graduated cylinder.

Calculation—

(1) 37·5 c.c. N. : c.c. in burette : : 1·55 gr. : grs. urea in 5 c.c.
(2) 5 c.c. : 28·4 (*i.e.*, ℥i) : : (grs. urea in 5 c.c.) : grs. urea in ℥i.

Instruments are now graduated so as to record the amount of urea in grains *per ounce* without the necessity of making this calculation.

Sugar.—A small proportion of sugar is present in healthy urine, but the usual tests do not discover it. Glycosuria is often a symptom in the gouty. It sometimes occurs in fevers; in diseases of the heart, lungs, and liver; and in diseases of the brain—especially when the lesion affects the region of the fourth ventricle. The latter conditions are somewhat rare. Persistent glycosuria is the prominent symptom of diabetes mellitus. Other sugars besides grape-sugar are found in the urine, but the latter is the only important one clinically.

Tests.—*Fehling's Solution.*—Take about an inch in depth of the test solution in a test-tube, and heat until it begins to boil; then add two drops of the urine. If sugar be abundant, a thick *yellow* ring of copper suboxide is formed which may quickly become *red*. If no change occur on the application of this test, go on adding urine and boiling until an equal bulk of urine has been added to the test solution. Boil, and if no clouding appears on cooling, the urine is free from sugar.

Trommer's Test.—Caustic potash solution is first added to the urine, and then a strong solution of copper sulphate is added drop by drop, until the cupric oxide formed is no longer dissolved. Heat the mixture in a test-tube. If sugar be present the yellow-red oxide is formed before the boiling point is reached, and the solution loses colour. This test is not reliable for small quantities of sugar; and, besides, the re-action occurs with other bodies—*e.g.*, uric acid, bile pigments, mucin, &c.

Phenyl-hydrazin Test.—This test is believed by v. Jaksch to be free from all fallacies. He takes two parts of hydrochlorate of phenyl-hydrazin, and three of acetate of soda, and places them together in a test-tube containing about two drachms of urine. A little water may be added if the salts do not dissolve on gently warming the solution. The test tube is now placed in boiling water for twenty to thirty minutes. Afterwards it is placed standing in cold water. A yellow crystalline deposit occurs when sugar is present; and this sediment when examined under the microscope is seen to be composed of long *yellow acicular crystals*—detached or in clusters (phenyl-glucosazon). The test is very delicate.

Quantitative Estimation of Sugar.—*Pavy's Method.*—Take 10 c.c. urine and dilute to 100 c.c. in the burette. Place 20 c.c. Pavy's sol. into the flask, cork, and attach to burette; boil one minute and keep simmering to expel air. Drop from burette, and shake flask, till Pavy's sol. = colourless. Read quantity of urine used.

Calculation.—(1) 20 c.c. Pavy's sol. = 0·01 gram: or 0·154 grains glucose .·. $\frac{1}{10}$ of the quantity of urine used from the burette : 28·4 c.c. (*i.e.*, ℥j) : : 0·154 grains : grains glucose *per ounce*. Multiply by the total amount of urine passed in twenty-four hours.

Fehling's Method.—10 c.c. Fehling's sol. diluted, and placed in a porcelain dish. Urine placed in the burette, as in Pavy's method, and dropped into Fehling's sol. until colourless.

Calculation.—10 c.c. Fehling's sol. = ·05 gram: or ·77 grains glucose .·. $\frac{1}{10}$ of quantity of urine used from the burette : 28·4 c.c. (*i.e.*, ℥j) : : ·77 grains : grains glucose *per ounce*. Multiply by the total amount of urine passed in the twenty-four hours.

Tests for *bile*, *blood*, and *pus*; *urobilin*, *indican*, and *acetone*; and for *chlorides*, *phosphates*, and *uric acid*.

Bile.—This re-action is really due to the pigments in the bile. A few drops of urine and of fuming nitric acid are placed *separately* upon a white porcelain slab, and then allowed to run into each other. If bile be present, there is a play of colours—violet, green, red—gradually fading away. The tests for the *bile-acids* are tedious, and only suitable for the laboratory.

Blood.—(1) A drop of tincture of guaiacum is added to a small quantity of urine in a test-tube, and about half an inch of ozonic ether is poured in. A *blue* colour forms. (2) The urine may be treated with caustic potash and boiled. The resulting precipitate of basic phosphates with hæmatin is coloured a bright-red. (3) The spectroscopic test may be applied.

Pus.—Add a solution of potash to the sediment, and if pus, it becomes *ropy*—*mucus* becomes thin and flocculent.

Urobilin.—The urine is dark and often frothy. The clinical interest of urobilinuria is associated with hæmorrhages and extravasations of blood into the tissues. The blood-colouring matter is re-absorbed and eliminated by the kidneys. Gerhardt suggests "that a chloroform extract of the urine should be treated with solution of iodine, and caustic potash added, when a beautiful green fluorescence develops."

Indican.—The urine has a rich dark-brown colour, and it is generally frothy. Indicanuria, clinically, is a symptom of *albuminous putrefactive changes* within the system; but it occurs, sometimes, in simple constipation.

Jaffe's Test.—About a drachm of urine is mixed with an equal quantity of hydrochloric acid, and then a solution of a hypochlorite is added, drop by drop, and shaken up with the urine. An indigo-blue results if the hypochlorite is not used in excess.

Acetone.—Acetonuria occurs in fevers, diabetes, cancer, cerebral irritations, &c. A large nitrogenous diet tends to produce it. Acetonuria may sometimes render the prognosis of a case more grave (diabetes and cerebral affections). The most ready *test* consists of treating two drachms of the urine with freshly-made concentrated sodium nitro-prusside, and strong solution of caustic potash. The solution becomes red, but this soon disappears, and if acetone be present, it is replaced by a purple or violet colour on adding acetic acid. The latter re-action does not take place in the absence of acetone.

Chlorides.—Add nitrate of silver to the urine, and the result is a white precipitate which is insoluble in nitric acid.

Phosphates.—The same test as above, but the white precipitate formed *is* soluble in nitric acid.

Uric Acid.—Boil some suspected urine with a few drops of nitric acid in an evaporating dish. When dry, add a few drops of dilute ammonia. The result is a red-purple of *murexid*. To add solution of potash, further, produces a *blue-purple* colour.

Examination of Deposits.—The urine should be allowed to stand for several hours in a cylindrical, or if the deposit be small in amount, in a conical glass. A pipette is then used to remove some of the deposit, and place upon a microscopic slide under a cover-glass. Magnifying powers of 50 and 350 are necessary.

Naked-eye Appearances of Deposits.—*Mucus.*—A small quantity is present in health, which may be seen as a delicate cloud throughout the urine. In catarrhal conditions the quantity is increased, and after a few hours it forms a denser gelatinous-looking layer at the bottom of the glass.

Pus.—This deposit somewhat resembles mucus, but it is thicker and not so transparent. The potash test may be used to differentiate mucus from pus. Both mucus and pus may appear in the urine *before* it cools.

Uric acid and *oxalic acid* crystals may appear in the urine entangled in the mucus, shortly after the urine is passed. Uric acid crystals resemble grains of cayenne pepper. When in greater quantity they form a dense red deposit, which is soluble in alkaline solutions. They occur in gouty conditions. Oxalic acid crystals indicate imperfect oxidation, and they are found often in certain forms of dyspepsia.

Urates.—These deposits are by far the commonest. They consist of amorphous urates of ammonia, soda, lime, or magnesia, and they

are often associated with uric acid crystals and oxalate of lime. In urines which become quickly alkaline, the urates may be present along with phosphates of lime. The deposit of urates appears after the urine cools, and it varies in colour from a pink or pink-white to brick-red. It occurs in all feverish conditions; in renal congestion; in dyspepsia and liver complaints; and after active exercise with perspiration. Urates are entirely soluble on heating the urine.

Oxalate of Lime.—This deposit generally forms two layers—the upper being white in colour, and the lower grey and more gelatinous-looking. Urates are frequently present along with the oxalates. The latter are soluble in a strong solution of hydrochloric acid, but not in acetic acid. The presence of oxalate of lime is not significant unless in large quantity. Oxaluria is a form of dyspepsia associated with much depression; but oxalates in the urine may occur after the ingestion of rhubarb, tomatoes, beetroot, &c.

In alkaline urines, triple phosphates (ammonio-magnesium phosphates) form *white* deposits when seen alone. *Amorphous phosphates* form light flocculent deposits. Phosphates are generally present along with pus in ammoniacal urines—the result of inflammation of the bladder, or of the urinary passages. Phosphaturia occurs also in chronic dyspepsia. Phosphatic deposits are soluble in acetic acid.

Urinary concretions (chiefly urates and uric acid, and more rarely phosphates) are frequently found, and are of interest in relation to renal colic. Foreign bodies and impurities are discussed in surgical works.

Microscopic Examination of Deposits—**Inorganic Deposits.**—*Uric acid crystals* vary much in shape and size, but they are always brown-yellow in colour. The lozenge shape, rhombic tables, and spiculate forms are the commonest (see Fig. 17).

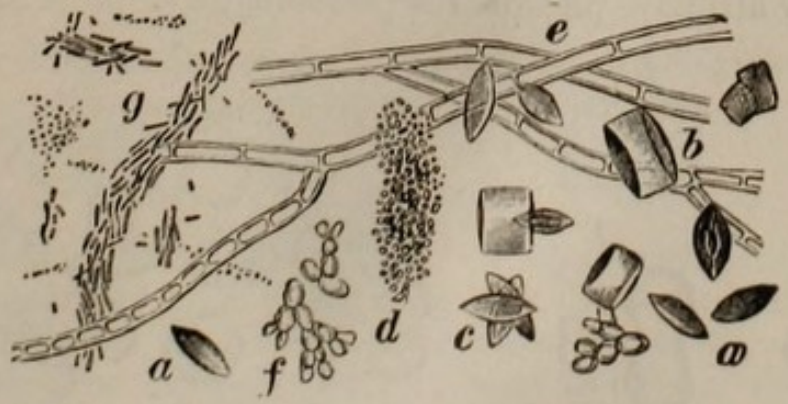


Fig. 17.—*a, b, c,* Uric acid; *d, g,* Micrococci and bacilli; *f,* Yeast-fungi; *g, e,* Mould-fungi. (From *Landois and Stirling's Physiology.*)

Urates.—Amorphous urates appear as fine granules. Ammonium urates appear as round globules (see Fig. 18).

Oxalate of lime crystals appear as octahedra, dumb-bells, and in compound forms (Fig. 19).

Triple phosphates have chiefly the "knife-rest" shape (see Fig. 18).

Neutral phosphates of lime appear as wedge-shaped prisms, often forming rosettes; while *basic phosphates of magnesia* form elongated rhomboids.

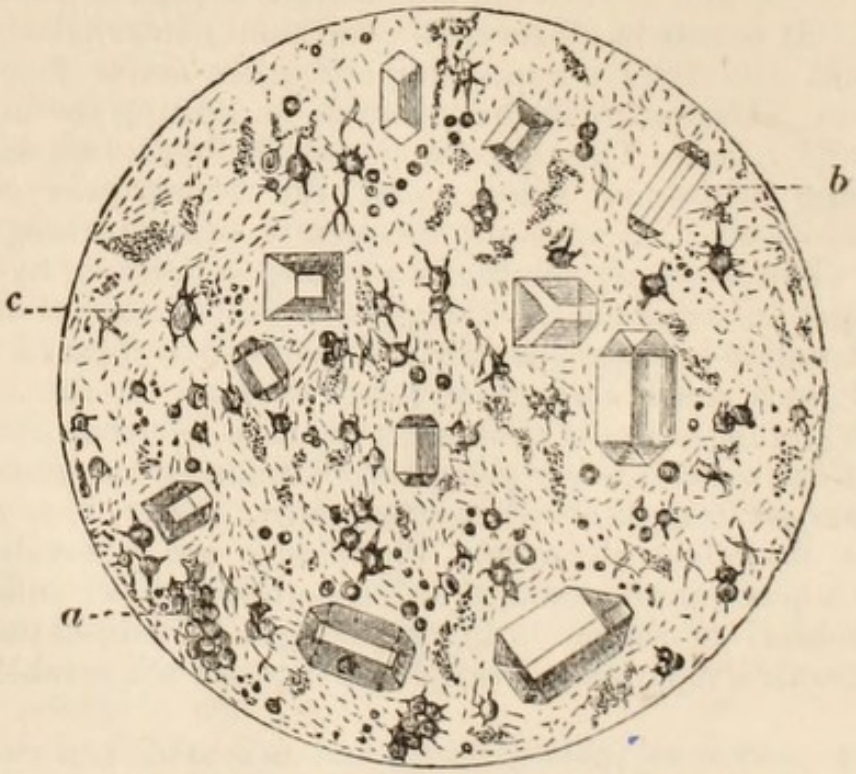


Fig. 18.—Deposit in Ammoniacal Urine (alkaline fermentation).—*a*, Acid Ammonium urate; *b*, Ammonio-magnesium phosphate; *c*, Bacterium ureæ. (From *Landois and Stirling's Physiology.*)

Cystin, *Leucin*, and *Tyrosin.*—Cystin forms symmetrical hexagonal plates. Leucin and tyrosin are generally found together. The former are spherical in shape, and the latter are circular and in bundles (see Fig. 20). They are found in the urine in cases of acute yellow atrophy, phosphorus poisoning, &c.

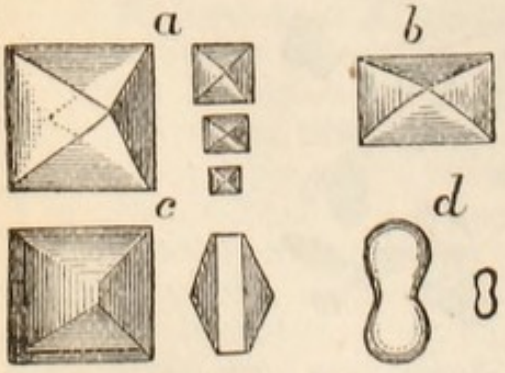


Fig. 19.—Oxalate of Lime.—*a*, *b*, Octahedra; *c*, Compound forms; *d*, Dumb-bells. (From *Landois and Stirling's Physiology.*)

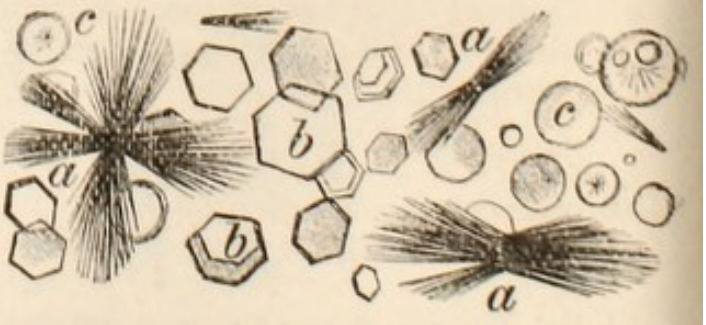


Fig. 20.—*a*, Tyrosin; *b*, Cystin; *c*, Leucin. (From *v. Jaksch and Cagney's Clinical Diagnosis.*)

Organic Deposits—Tube Casts.—As these have been found in non-albuminous urine, and in cases where no renal affection could be suspected, their presence (without other symptoms) does not always imply disease. The chief forms are the *epithelial*, *granular*, *fatty*,

waxy, *hyaline*, and *blood* casts. These forms are often *mixed*. When the casts are present in number, they are highly significant. The presence of epithelial and blood casts in albuminous urine always indicates acute renal disease. They are figured below. Granular casts vary in size. Sometimes they are coloured (pale-yellow to brown). They also indicate inflammation of the kidneys—generally the more chronic forms. Fatty casts are found in chronic forms of Bright's disease with fatty degeneration. The fat globules may be on the surface of granular casts, or they may form cylinders alone. Waxy and hyaline casts are somewhat longer than the others. They are

Fig. 21.

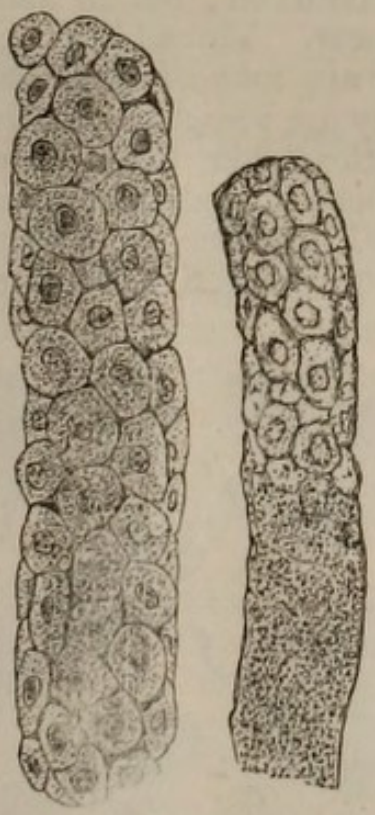


Fig. 22.

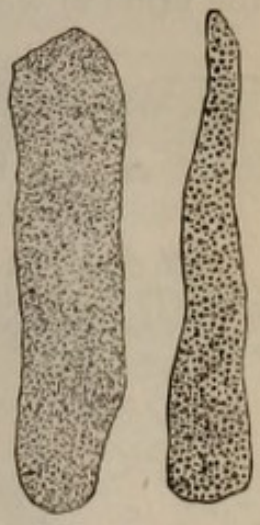


Fig. 23.

Fig. 21.—Blood cast; altered corpuscles lying near it.
Fig. 22.—Epithelial casts.
Fig. 23.—Finely granular casts.

(From *Landois and Stirling's Physiology.*)

homogeneous, and more difficult to find under the microscope, without staining. The waxy casts are found in acute and chronic Bright's disease, and in waxy degeneration of the kidneys. The hyaline casts are seldom found in urine free from albumen (Leube), but their actual significance is not understood. They are often present in disease of the kidneys, but it is the coating upon these casts (epithelial, fat, leucocytes or blood corpuscles, &c.), which is of clinical importance.

Cylindroids are the long ribbon-like casts found by Thomas in the urine of scarlet-fever patients. They have been observed in renal diseases and in healthy urine. The hyaline casts and cylindroids are believed to be products of epithelial secretion, probably connected with the exudation of albumen within the tubules. A

dilute solution of iodine and iodide of potassium may be used to detect the hyaline casts.

Red blood corpuscles in the urine may retain their proper form, or may appear as attenuated and shrunken yellow rings—the biconcave character being often lost. The latter is generally the case when the blood is intimately mixed with the urine and does not form a sediment. This occurs when the hæmorrhage is from the kidneys, or from the pelvis or ureters. When the blood is not diffused the origin is probably the bladder.

Pus.—The cells are readily detected under the microscope. Sometimes they are unaltered; but in alkaline urines they swell up and the nuclei disappear. The addition of acetic acid renders the nuclei visible. They are found in inflammations of any part of the urinary tract, and they are sometimes derived from abscesses in the neighbourhood. In the latter cases they are usually fatty. The addition of the iodo-potassic iodide solution stains them mahogany-brown, while epithelial cells become a light-yellow colour. Pus, in the urine of women, may be derived from the vaginal secretions.

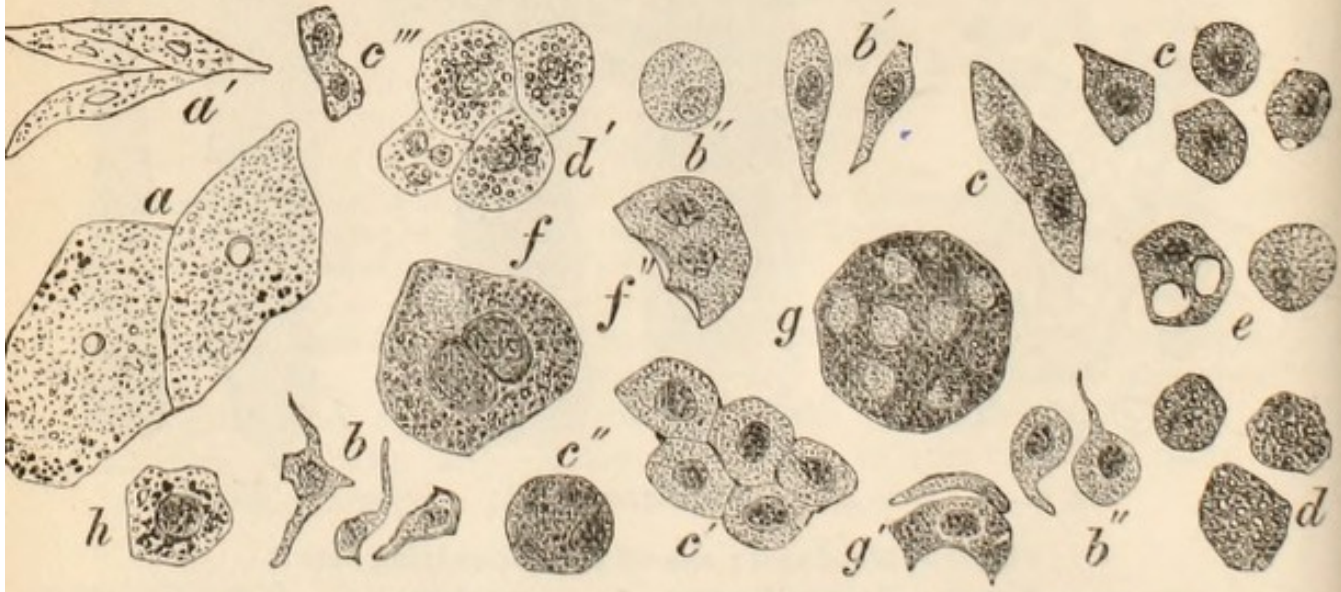


Fig. 24.—Epithelium from the Urinary Sediment.—*a*, *a'*, Squamous epithelium from the urinary sediment: *b*, *b'*, *b''*, Epithelium from the bladder; *c*, *c'*, *c''*, *c'''*, Epithelium from the kidneys; *d*, *d'*, Fatty epithelium from the kidneys; *e-h*, Epithelium from the bladder. (From *v. Jaksch and Cagney's Clinical Diagnosis.*)

Epithelium.—It is only when epithelial cells are present in quantity in the urine, that they are of diagnostic importance. They indicate a catarrh of some part of the urinary tract. They differ in form, and sometimes it is possible to locate the seat of origin. (See Fig. 24).

Cancer cells cannot be clinically differentiated from the normal cells with any certainty. Cells from pigmented tumours may sometimes be detected.

Spermatozoa are readily recognised by their tadpole appearance.

Micro-organisms and Parasites (see Fig. 17).—Moulds, yeasts, and fission-fungi are often found in the urine. Moulds are common

in diabetes, while fission-fungi and yeasts appear in ammoniacal urines. Bacteriuria is observed after the use of unclean catheters. The tubercle bacilli may be found in phthisical cases with renal complications. The presence of infusoria has no pathological significance. The parasites which affect man and which may be found in the urine, are as follow, viz., *Distoma hæmatobium*, *Filaria sanguinis hominis*, *Echinococci*, *Eustrongylus gigas*, and sometimes *Ascarides*.

CHAPTER VI.

DISEASES OF THE URINARY SYSTEM.

Contents.—Acute and chronic parenchymatous nephritis—Cirrhotic Bright's disease—Waxy disease of the kidneys—*Differential diagnosis of the three forms of Bright's disease*—Diabetes mellitus—Diabetes insipidus—Renal calculi—Hydronephrosis—Pyelitis, and suppurative nephritis—Perinephritis, and perinephritic abscess—Malignant disease of the kidney—Tubercle, cystic disease, and hydatids of the kidneys—Floating kidney—Paroxysmal hæmaturia—Chyluria—Active and passive congestion of the kidneys—Uræmia, *and the diagnosis of kidney affections.*

Bright's disease is a group of acute and chronic diseases of the kidneys, having *albuminuria* as a characteristic symptom. Albuminuria alone, however, is by no means pathognomonic. The classification is as follows, viz. :—

I. Acute and chronic parenchymatous nephritis, or acute and chronic Bright's disease.

II. Cirrhosis of the kidneys, or cirrhotic Bright's disease.

III. Waxy disease of the kidneys.

Acute Bright's disease may be subdivided into *glomerular nephritis* and *tubular nephritis*, according as the glomeruli or the tubules are most affected—the former being the case in scarlet fever. Glomerulo-nephritis is known as the scarlatina kidney.

Professor Grainger Stewart tabulates the different forms of Bright's disease thus, viz. :—

I. Inflammatory—
  (A) Glomerular nephritis.
  (B) Tubular nephritis, { Simple. / Fatty kidney. / Atrophied kidney. } As stages.
  (C) Both forms together.

II. Cirrhotic Bright's disease.

III. Waxy disease of the kidneys, { Simple. / Enlarged. / Atrophied. } As stages.

IV. Combined forms.

Acute Parenchymatous Nephritis—Tubular Nephritis—Acute Desquamative Nephritis—Acute Bright's Disease.—**Pathology.**—The kidney is increased in size and weight. The capsule strips off easily and reveals a smooth surface beneath it. The cortex is pale, with small extravasations throughout its textures; and the pyramids are deeply congested. The microscopic examination reveals changes in the convoluted tubules, almost exclusively. Fine deposits—cloudy granulations—are seen within the epithelial cells, and these, producing distention, almost occlude the tubules which frequently become dilated and tortuous.* In the later stages, fatty degeneration of these products of inflammation give the organ a yellow mottled appearance (large fatty kidney). In the extremely chronic stage, the organ becomes atrophied. According to Grainger Stewart, *all* forms of Bright's disease end in atrophy, if the patient live long enough, and the disease do not terminate in recovery.

The **causes** of acute Bright's disease are, sudden exposure to cold when the skin is warm and perspiring; scarlatina, diphtheria, typhoid fever, and erysipelas; pregnancy; substances which irritate the kidneys, as cantharides, turpentine, copaiba, &c.; and hereditary influences. Acute Bright's disease is commoner in youth than in the aged; and a constitutional type is described—pale, light-haired, and flabby individuals—who are predisposed to this form of the disease.

Most frequently the early **symptoms** of acute Bright's disease are obscure, and there is little constitutional disturbance. Œdema of the ankles, or a puffiness of the eyelids, is often the first symptom which attracts attention, and calls for examination of the urine. At other times, there is a history of a chill followed by fever and lumbar pains, with painful, and frequent, attempts at micturition, resulting in the passage of a few drops of urine, often bloody.

The urine is diminished in quantity, and sometimes there may be entire suppression, which may lead to very urgent symptoms, and death within a few days (*uræmia*). The specific gravity is increased to 1,025 or 1,030 in the early stages, and this is due to the diminished amount of water secreted; but soon, if the disease proceed, it is as low as 1,010 or 1,005. The urine is acid in re-action, and it is often loaded with urates; and if it contain only a small proportion of blood, it has a smoky appearance. When the quantity of blood in the urine is greater, the colour is from a red hue to a dark-brown, according to the amount. Albumen is always present in this form of Bright's disease, and it may be roughly estimated by the quantity which appears with an inch of urine in the test-tube, when the cold nitric acid test is applied. In severe cases, the amount of albumen may show from a half to three-fourths of the bulk of urine within the test-tube. In mild cases, a mere hazy ring may be all that is present.

* In the scarlet fever kidney, these changes affecting the glomeruli more than the tubules, the Malpighian bodies are more prominent on section of the kidney. The microscopic changes are also observed in this neighbourhood (glomerulo-nephritis).

The tube casts found in acute Bright's disease are epithelial and hyaline, granular and blood casts, along with numerous free blood corpuscles, granules, and *débris* (see Figs. 21, 22, 23). The quantity of urea excreted—as estimated with the total amount of urine passed in the twenty-four hours—is much diminished, and so are the other solid constituents of the urine. The œdema of the ankles and eyelids appears early, and it soon extends to the other parts of the body, and to the serous cavities if the disease advance. In the later stages, as the amount of water in the urine increases, the acidity diminishes, and there is an increase in the quantity of the solid constituents excreted; but the quantity of urine voided is still below the normal, and of a low specific gravity. The tube casts are then seen to be fatty, as in the chronic form, while the dropsy is much increased. There is loss of appetite, nausea and vomiting, and often diarrhœa; and the patient soon becomes anæmic and emaciated. The vision may become impaired from simple anæmia, or it may be the result of *albuminuric retinitis*.

The *history and course* of acute Bright's disease are modified by the cause, as in scarlatina, diphtheria, &c. Pregnancy can only be viewed as an exciting cause, apt to produce congestion of the kidneys in patients already predisposed to Bright's disease. In scarlatina, the glomeruli being more affected than the tubules, there is a greater diminution in the quantity of urine passed, and there is more chance of suppression. In acute Bright's disease, arising spontaneously, the fever (if any) lasts only for a few days. Recovery from this form is quite common; but death may ensue in the course of a week from uræmia. The case generally progresses slowly, and the dropsy appears gradually, unless the urine be very scanty, and then dropsy may occur within a few days. Death may result later from exhaustion, or by gastro-intestinal disturbance and anæmia. In the cases which recover, it may be three months before the health is restored.

The **chronic form of Bright's disease** may follow the acute; but more commonly it develops insidiously, the result of chronic alcoholism, syphilis, malaria, chronic suppuration, or metal poisoning. It may arise from any cause which depresses the vitality of the tissues in those predisposed, constitutionally, to Bright's disease.

The pathological condition is that of the *large white kidney* already mentioned. The cortical part of the kidney is much enlarged, and there is hyperplasia of the connective tissue, which, by contraction, subsequently leads to atrophy of the organ. Instead of the epithelium of the tubules being affected with cloudy swelling—as in the acute form—there is fatty degeneration within the cells, and the tubules are filled with the products of the epithelial cells destroyed.

The **symptoms** develop slowly, when not preceded by the acute form. For some time, there may be only loss of appetite and strength noticeable, but gradually the patient's face acquires a pale and characteristic earthy colour. The œdema of the ankles and face now appears, and quickly spreads over the whole body. The

scrotum may be immensely distended, and there is more dropsy, ultimately, in this form of Bright's disease than in any other. The serous cavities become affected, and hence pleuritic and pericardial effusions are frequent. Œdema of the lungs and glottis are common complications, the latter being highly dangerous, as it may cause death by suffocation. The changes in the urine are the same as in the acute form ; but there is often less albumen, and the *hyaline and fatty* tube casts predominate. They become more and more granular, fatty, and broader in shape as the disease advances. The skin becomes sodden. The mucous membranes are dropsical, and hence vomiting and diarrhœa are common symptoms. At first the heart-sounds are accentuated ; but later they are only feebly heard, while the pulse—at first increased in tension—becomes quick, small, and compressible. *Albuminuric retinitis* is common, and the impairment of vision may be one of the first symptoms calling attention to the state of the kidneys. Dyspnœa results from œdema of the lungs, or from pericardial or pleuritic effusions. Sometimes convulsions and other symptoms of uræmia develop. A fall in the specific gravity of the urine indicates a diminution of the solids excreted, and this symptom may precede, for a few days, the onset of uræmia.

Complete recovery from the chronic form of Bright's disease is always possible, but it is somewhat rare. Partial recovery to moderate health is frequent, although albumen may be detected in the urine a year or two after the dropsy has disappeared. Recurrence is the rule, and the prognosis will depend upon the amount of the dropsy, and the state of activity of the kidneys. Death may ultimately be due to pneumonia or pleurisy, to phthisis, exhaustion, or uræmic coma.

Cirrhotic Bright's Disease. Interstitial Nephritis. Granular Contracted Kidneys. Gouty Kidneys.—Pathology.—The kidneys are much reduced in size and weight. The capsule is thickened and adherent, and when removed reveals an irregular surface. The tissue of the kidney is tough and resistant on section, and cysts of various sizes are present throughout its substance. The cortical portion is much atrophied. The organ is red-brown usually, but often it is of a yellow or fawn colour. Microscopically, the capsule is seen to be much thickened, and there is increase of the connective tissue throughout the organ, especially beneath the capsule and around the Malpighian bodies. The tubules are seen to be compressed. The organ may be affected unequally. The secondary changes are hypertrophy of the heart and arterioles, and these alterations in the heart and blood-vessels produce a condition favourable to cerebral hæmorrhage. *Albuminuric retinitis* is common in this form of Bright's disease.

The **causes** of cirrhotic Bright's disease are chiefly gout, chronic alcoholism, syphilis, and lead poisoning. Prolonged use of irritating drugs may also produce cirrhosis. It is a disease of middle life, and it affects males much more than females.

The **symptoms** develop very insidiously, and it is not until the

disease is well advanced, as a rule, that any diagnosis is made. Thirst, and the secretion of large quantities of urine (making it necessary to rise frequently during the night) are early indications of cirrhotic Bright. Sometimes it is amblyopia, hemianopsia, diplopia, or other impairment of vision which first suggests the possibility of the disease being present. Frequently, a secondary disease, or a group of symptoms apparently referable to quite another system than the renal, prove to be due to cirrhosis of the kidneys. Examples of these conditions are cerebral hæmorrhage; asthma or dyspnœa; cough and palpitation; convulsive seizures; vertigo and headaches, acidity, indigestion, and flatulence; epistaxis; or failure of vision, &c. As the disease advances there is failure of the strength, and loss of body weight. The skin becomes dry and yellowish, and the hair "spiky." The urine is pale in colour, feebly acid, of low specific gravity (1,003 to 1,008), and greatly increased in quantity (80 to 300 ozs. or more). There is usually albumen present, but only in small quantities, and often it is absent for a time, so that repeated examinations are necessary in suspected cases. The solid constituents of the urine, especially urea, are reduced. Pale, transparent hyaline casts are present, but they are not numerous, and they must be carefully searched for. The sediment should be collected from a large quantity of urine. There is great breathlessness upon exertion. There is little dropsy in the cirrhotic form of Bright's disease, so long as the urine voided is great in quantity; but any sudden diminution in the quantity increases the œdema, and usually, then, uræmic symptoms supervene. The œdema of the ankles may be due to the debility, or to failure of the heart in the later stages of the disease. During the earlier course of cirrhotic Bright's disease, the left side of the heart becomes much hypertrophied, and the second sound is heard to be much accentuated. These changes, according to most authors, are held to be due to the increased tension in the arterioles. The pulse is full and slow, incompressible and tense. After fatty degeneration of the hypertrophied muscular fibres—in the very latest stage of the disease—the heart-sounds are feeble, and the pulse is small, more frequent, irregular, and weak. Pericarditis, endocarditis, hydrothorax, and œdema of the lungs are common complications towards the end. Erysipelas and eczema are common in all chronic forms of Bright's disease. Uræmia is the usual termination—the symptoms being ushered in by obstinate vomiting and diarrhœa, headache, and vertigo. The patient becomes more and more stupid and ultimately comatose, and the case often ends in convulsions.

The ophthalmoscopic examination during the early course of cirrhotic Bright, reveals the optic discs swollen; veins enlarged and tortuous; arteries small; hæmorrhagic extravasations; and white spots upon the retina. Ultimately there is optic atrophy. The impairment of the vision may be due, however, to anæmia, or to chronic uræmia.

Waxy Disease of the Kidneys. Lardaceous or Amyloid Disease.—**Pathology.**—There is a waxy or amyloid degeneration affecting the renal vessels, and the vascular tufts of the

glomeruli. The afferent and efferent vessels, vasa recta, and renal epithelium are also affected. The pathological process is of the nature of an exudation, or deposition, of amyloid or waxy material, which reacts to *iodine solution*—the affected parts becoming red-brown in colour when it is applied. The kidneys are increased in size and weight. The capsule strips easily, and reveals the surface of the kidney pale and glistening. On section, it is found to be firm in texture, and the cortical part is enlarged and pale, while the cones are deeply congested. The supra-renal capsules, liver, spleen, intestines, &c., are also waxy. In the later stages the kidneys become atrophied. Frequently, parenchymatous inflammation co-exists.

The **causes** of waxy disease are chronic suppurations and ulcerations, phthisis, diseases of bones and joints, syphilis, cancer, and scrofula.

The **symptoms** of waxy disease of the kidneys arise in connection with the above-mentioned causes, and hence they are often obscured, or regarded as secondary, to the prominent symptoms of the primary wasting disease. There is anæmia, and more or less œdema of the lower extremities. There may be general ascites, especially if the liver be also involved. The urine is pale and increased in quantity, and is of a low specific gravity—especially if cirrhotic Bright's disease be also present. It may be diminished in quantity, and high in specific gravity, if the waxy disease be associated with parenchymatous nephritis. The secretion of urea is not diminished. Albumen is present when the disease is fully established. Transparent hyaline casts are found in the urine, and they are highly refracting when composed of waxy material. There is gradual weakness and exhaustion as the disease advances, and uncontrollable diarrhœa, sometimes vomiting, may terminate the case. Cases recover when the cause can be removed. Uræmia is only common in the combined forms.

The Diagnosis of the Different Forms of Bright's Disease.—The symptoms and physical signs may conveniently be summarised in columns, thus :—

Acute and Chronic Bright.	**Cirrhotic B.**	**Waxy B.**
Develop insidiously ; or there is chill, fever, and lumbar pain. Sometimes presence of an obvious cause.	Develops slowly, without chill; and cause not so obvious.	Develops slowly, and a cause very obvious ; and evidences of waxy disease elsewhere.
Œdema; and in chronic form, great dropsy.	Little dropsy.	Little dropsy usually.
No hypertrophy of heart in the acute; heart-sounds accentuated in the chronic, and further evidences of increased tension in the blood-vessels, later.	Great hypertrophy of the heart, and great tension in the blood-vessels.	No hypertrophy, or tension.

Acute and Chronic Bright,	**Cirrhotic B,**	**Waxy B.**
Uræmic symptoms common.	Uræmic symptoms common.	Uræmic symptoms rare.
Urine diminished.	Urine increased.	Urine increased.
Urea diminished.	Urea diminished.	Urea normal.
Urine contains blood.	Urine contains no blood.	Urine contains no blood.
Specific gravity of urine is at first high, but it becomes low; in the chronic form it is low.	Specific gravity low.	Specific gravity low.
Urine dark and deposits urates.	Urine pale, and no sediment.	Urine pale, and no sediment.
Albumen in large quantities; but less in the chronic form.	Albumen small in amount.	Albumen small in amount.
Epithelial, granular, and blood casts predominate; hyaline and fatty casts in the chronic form. There are numerous tube casts.	Hyaline casts predominate; no blood casts. The tube casts are few in number.	Waxy casts predominate; and no blood casts. The tube casts are few in number.

The Treatment of Bright's Disease.—Acute Bright's Disease.—In adults, when the symptoms are urgent, one-tenth to an eighth of a grain of pilocarpine nitrate should be injected hypodermically. If called to a case of uræmic convulsions, chloroform inhalations may be used; but the injection of half a grain of morphia is still better treatment, although the state of the kidneys would seem to contra-indicate the use of it. Chloral and bromide of potassium are useful in less urgent cases. Vapour baths should also be used in all cases, and these may very readily be given by means of Allan's spirit kettle. Filling bottles with hot water, and pulling damp stockings over them, is a method that will always be available. Half a dozen of such bottles may be laid around the patient as he lies between blankets. Purgatives are also indicated, and those are best to use which produce watery stools. The compound powder of jalap is the usual one (sixty grains or more for an adult), and one-sixteenth to an eighth of a grain of elaterium may be combined with it. Croton oil may be used. Diluents, as milk, or milk and lime-water if the stomach be irritable, and cream of tartar freely diluted, should be allowed. Stimulants are to be avoided. A mustard leaf over the kidneys, or dry cupping, is also of value during the early stages of acute Bright's disease. Digitalis is sometimes used as a diuretic, but it is of doubtful service in the early stages, when the tubules are blocked rather from congestion and inflamed membranes, than from inflammatory débris. Later, it is of the highest value. The diet

should be entirely non-nitrogenous. Iron tonics are valuable when the acute symptoms have subsided.

Chronic Bright's disease often requires the same treatment as the acute during the course of the disease. Digitalis as a diuretic is now indicated. Paracentesis abdominis is often necessary to relieve the dyspnœa, or to help absorption of the fluid. The skin may require to be punctured when very tense with the œdema. Southey's tubes are not recommended, on account of the erysipelas which almost invariably follows. Simple pricks with a tenotomy knife are better. Iron in the form of steel drops—ten to twenty minims in water thrice daily, after food—is the best remedy for the chronic cases. The constipation and headaches which often attend its use, should be counteracted by the use of mineral waters, &c. Warm clothing, limited supply of nitrogenous food, exercise, and a warm, dry, unchangeable climate are all advisable.

Cirrhotic Bright's Disease.—In cases with a syphilitic history, give iodide of potassium with minute doses of perchloride of mercury; in lead poisoning, give iodide of potassium alone. Arsenic and chloride of gold have been recommended in other cases. Iron is also useful. The treatment can only, as a rule, be symptomatic, as, *e.g.*, the treatment of dyspepsia by mineral acids and a bitter, &c. In acute attacks with uræmia, the treatment is the same as in acute Bright's disease. The diet should be simple, and should consist of milk, eggs, and fruit (if no diarrhœa). Stimulants should be avoided. Flannel should be worn next the skin, and a warm, dry climate is to be recommended.

Waxy Disease of the Kidneys.—The treatment is to check chronic suppurations, if possible; and to keep up the patient's strength with iron, cod-liver oil, and a generous diet. For the diarrhœa, Fowler's solution with laudanum—in small doses, frequently repeated—is an efficient remedy. In cases associated with syphilis as a cause, iodide of potassium should be prescribed.

Diabetes Mellitus.—**Pathology.**—There is a perversion of the glycogenic function of the liver, producing an excessive flow of sugar from the liver into the blood. The organ seems to be too active. The morbid anatomy is obscure. Irritations at the roots of the vagi have been found, and also small tumours. Dickinson has described degenerative changes of the nervous tissue in the pons Varolii. Atheroma of the blood-vessels, especially at the base of the brain, is frequently found in cases of diabetes mellitus. The liver is often enlarged and hyperæmic. The pancreas is sometimes hypertrophied, and sometimes atrophied. The mucous membranes are hyperæmic, and the viscera emit a sweet smell at the *post-mortem* examination. The kidneys are often enlarged. The blood serum contains more fat, and it is sometimes almost milky in appearance.

The **causes** are as follow, viz.:—Blows or falls; shock; strong emotions; cerebral disease; tumours of the fourth ventricle; long-continued errors in diet, especially in the obese; exposure to cold and fatigue; and hereditary influences. Diabetes is commoner in males than in females, and it affects the young and the old.

The **symptoms** begin with muscular weakness and general debility. In the obese, there may be for a considerable time symptoms of indigestion, with occasionally glycosuria. Great thirst is soon complained of, and the patient perhaps notices that he is passing large quantities of pale urine, and that he requires to rise frequently during the night to relieve the bladder. There is dryness of the mouth and fauces, and often a sweet taste is noticed. The tongue is red and clean, but dry and clammy, and there is often *dry smacking of the tongue and lips* when the patient is speaking. The appetite is often voracious. The gums often swell and bleed. As a rule—except in the obese, and when indulgence in the pleasures of the table is the supposed cause—digestion is good; but sometimes a feeling of *emptiness* at the pit of the stomach is complained of, and occasionally there is vomiting. The bowels are usually constipated. The skin is dry, and the furrows of the hand are often white and scaly. There is great emaciation when the disease is fully established; and there is loss of virile power in man, and suppression of the catamenia in the female. The breath of the patient may sometimes be noticed to be sweet, or it may have the odour of new-mown hay. The temperature is often sub-normal. There is œdema of the ankles, and the dropsy may soon become more general. Albumen may be present, as Bright's disease is often associated with the diabetes.

The urine is acid, and is much increased in quantity; but this will vary with the amount of water drunk. It varies from 70 or 100 ounces, to gallons, per diem. The specific gravity is high—ranging from 1,020, to as high as 1,040 or 1,050. The colour is *pale-greenish*, and there is no sediment. The sugar present may amount to from 1 or 2 per cent. to 10 or 15 per cent. Fifty ounces of sugar have been known, in one case, to be passed in the twenty-four hours. The urea is normal in amount, but sometimes it may be very much increased when the patient is confined to nitrogenous food.

There are numerous *secondary diseases* known to be associated with diabetes mellitus which should be noted carefully, as sometimes the symptoms are obscure, and the patient often seeks relief from what he believes to be a primary affection. Impairment of the vision (by cataract, and atrophy of the optic discs, &c.) may be an early sign. Carbuncles and boils; pruritus vulvæ and eczema; gangrene of the toes, &c.; a low form of pneumonia (phthisis), and sometimes a croupous pneumonia; diabetic coma, convulsions or delirium; are all examples of secondary conditions which may occur during the course of diabetes. In *diabetic coma* there is at first great restlessness, agitation, and præcordial uneasiness, sometimes delirium or convulsions, and then gradually increasing somnolence, complete coma, shallow breathing, and death. It is supposed to be of the nature of an *acetonæmia*. It is only in very exceptional cases that sugar is found *permanently* in the urine, and no other symptom of diabetes present.

The **prognosis** varies much, but it is always grave. It is more favourable, as regards amelioration, in those who are advanced in

years, when the diet is rigidly adhered to, and is found to relieve the symptoms. In the young, diabetes is very fatal, the average duration being one or two years; but very acute cases, in children especially, may not last more than a few weeks. Cures have been reported, and certainly life has been prolonged beyond the expectation of both physician and patient; but, as a rule, sooner or later, the disease gains and ultimately produces a fatal result. The prognosis, therefore, will depend upon the success of the treatment, the state of the digestive powers under the special diet, and the amount of control gained over the excretion of the sugar.

Diabetes insipidus is characterised by the passage of enormous quantities of pale watery urine, free from sugar and albumen.

The **pathology** is still obscure; but similar changes to those found in diabetes mellitus have been described. The disease appears to be sometimes hereditary. At other times, a shock, injuries, cold and fatigue, and particularly syphilitic tumours of the brain, are reported to be causes of diabetes insipidus.

The **symptoms** consist of the gradual development of *polyuria* (one to five gallons of urine daily), great thirst, and the distress produced by the necessity of having so frequently to relieve the bladder. These symptoms are frequently superadded to the symptoms of a causal affection—as syphilis or brain disease. The urine, besides being greatly increased in quantity, is pale, specific gravity 1,002 to 1,006, faintly acid, and it is free from sediment. The solid constituents (in relation to the total quantity of urine passed in the twenty-four hours) are increased, except uric acid. There is gradual loss of body-weight.

The **prognosis** is not dangerous as regards life, although death may result from a causal or intercurrent affection. The cases resulting from syphilis are very favourable.

The **treatment of diabetes mellitus** consists in arranging the diet. Ordinary bread, potatoes, beans, peas, rice, carrots, turnips, and parsnips, and all articles containing flour, sugar, or starch, must be excluded. Greens, lettuce, tomatoes, and spinach are allowed. *Diabetic bread* must be resorted to, and milk may be allowed—especially butter milk and skimmed milk. Meat (fresh or salt), fish, oysters, eggs, fats, and nuts (except chestnuts) are all allowable. Light acid wines and *diabetic whisky* may be necessary, when stimulants are required. A few drops of sulphuric acid in water will satisfy thirst. Walking exercise is to be encouraged. Of the medicines used, opium and codeia have been found the most useful. Arsenic (three minims of Fowler's solution) with opium (ten minims of the tincture), is also a useful combination. Carlsbad waters may be tried.

In **diabetes insipidus**, galvanism (one electrode applied to the neck below the occiput, and the other applied to the hypochondriac regions) should be tried; and a course of iodide of potassium should be given in cases of syphilitic origin. Pilocarpin and ergotin have been sometimes apparently successful.

Renal Calculi.—The size of these range from mere grains up to very considerable "stones." The most common are the uric acid calculi, and those composed of triple phosphates and carbonate of lime.

The uric acid calculi are arranged in concentric layers, composed sometimes of alternate layers of uric acid and oxalate of lime. The uric acid calculi are hard, and of a red-brown colour. The pure oxalate of lime calculi are very rare, and they are hard and rough. Calculi of cystine are still rarer, and they are of an amber colour. Phosphatic calculi are the next common to those of uric acid, and they are light, soft, and of a dull white colour—rough, but sometimes smooth. They are composed of lime and ammonio-magnesium phosphates, and they may form around a uric acid calculus; but this is only likely to happen when the urine becomes alkaline, and with a uric acid calculus which has long been confined in the pelvis of the kidney.

Calculi may affect both kidneys, but more usually only one. They may light up inflammation of the kidney; but if in the pelvis, a *pyelitis* results, which may extend down the ureter. The ureter becomes much thickened. The passage of a calculus down the ureter produces *renal colic*, and if it become impacted, then the ureter and pelvis of the kidney become dilated, and the pressure backwards causes, ultimately, an atrophy of the kidney. If ulceration take place, then an abscess may form which may reach the surface in the lumbar region, or point below Poupart's ligament, or ulcerate into the important surrounding organs.

The **causes** are the gouty *diathesis*, sedentary habits, indulgence in highly nitrogenised food, and hereditary influences.

The **symptoms** vary with the size of the calculus. If very small, a dull pain in the lumbar region (felt when the patient is at rest, and, therefore, not muscular) is the only symptom. Even a large calculus retained in the pelvis of the kidney frequently gives rise to no disturbance. When sufficiently large, and when passing into the ureter, renal colic is the result. The pain comes on almost always with great suddenness, and it is agonising. It shoots up to the shoulder blade, and down into the groin, thigh, testicle, and often to the glans penis. The testicle of one side is drawn up. The patient may, in extreme cases, roll about in agony, beads of cold sweat collect upon the brow, the features are pale and pinched, and the expression pained and anxious; vomiting is usual. Frequent attempts at micturition are made, resulting in the passage of small quantities of urine which is often bloody, and may contain pus. The patient may faint, and he is sometimes convulsed. Should there be complete anuria, then convulsions and coma may terminate the case. The paroxysm usually terminates suddenly by the calculus passing into the bladder, when there is complete relief. If a calculus ulcerate through the ureter, a fatal peritonitis is produced. The attack varies in length according to the size of the stone passing along the ureter, and the pain is always worst just as it is about to pass into the bladder. Sometimes the renal colic lasts one or two days, but more usually only a few hours. Pyelitis is present when the

calculus is retained in the pelvis, and then the urine may appear "milky" from the presence of mucus and pus. The passage of mucus and pus may give rise to milder attacks of renal colic.

Hydronephrosis may result from obstruction of the ureter, and then a tumour, usually globular, may project downward from the hypochondrium; and the sac may ultimately ulcerate into the stomach or bowel, or establish a fistulous opening externally.

Although the first attack of renal colic may appear suddenly, and terminate favourably, it is more usual for the disease to run a chronic course. Repeated attacks—often milder as regards pain—is the rule; while the secondary pathological conditions, already mentioned, slowly develop, except in those cases which ulcerate into the peritoneum, &c. The latter cases are quickly fatal. Death may take place from waxy disease, exhaustion, or pyæmia. Recovery has resulted from the discharge of the calculus through a fistulous opening externally.

The **treatment** of urgent renal colic may require inhalations of chloroform, followed by morphia hypodermically (one-half of a grain). A warm bath is highly serviceable. These may require to be frequently repeated during the passage of a stone along the ureter. Potash and lithia salts are indicated when uric acid is present in the urine. Thirty grains of acetate of potash, or forty grains of the citrate, may be given thrice daily, freely diluted; the effervescing lithia water is given in doses of five to ten ounces, freely diluted. Benzoate of ammonium (ten to twenty grains) is useful for phosphatic calculi. Butter milk and skimmed milk may be ordered *ad libitum*. A calculus lodging in the pelvis, giving rise to severe pain or urgent symptoms, should be treated surgically—by removal and free drainage.

Hydronephrosis (Dropsy of the Kidney).—This results from obstruction of the ureter, either by a calculus, or by adhesion of the mucous surfaces after inflammation; or from pressure outside by tumours, or uterine displacements, &c.

There is dilatation and thickening of the ureter, which may be of the size of the small intestine. If the obstruction be great, the kidney structure may be completely atrophied, and the kidney is represented by a large saccular dilatation. The membraneous sac may be so large as to half-fill the abdominal cavity, in extreme cases. The urine within the sac is generally pale, alkaline, and of low specific gravity. It may be tinged with blood, and sometimes it contains pus. It is rare for both kidneys to be affected, although the other kidney is usually enlarged from the increased work thrown upon it.

The **symptoms** arise slowly, and the diagnosis is made when a fluctuating tumour can be felt. Pains are complained of when adhesions are formed with the adjoining organs. Pressure upon the colon produces constipation; upon the diaphragm it causes dyspnœa; and upon the stomach it causes nausea and vomiting.

The **treatment** is not to interfere, unless life be endangered, and

then the aspirator may be used. Manipulation of the tumour has sometimes emptied the sac, the obstruction being overcome.

Pyelitis, and Suppurative Nephritis.—The two diseases are almost always associated. The mucous membranes are much thickened, and the epithelium is altered. The kidney is enlarged and congested, and it has yellow spots throughout its substance. These suppurating points run together and form abscesses, and the kidney structure is broken up and disintegrated. With a high power of the microscope, colonies of bacteria are found within the tubules.

The **causes** of pyelitis and suppurative nephritis are extension of inflammation from the bladder, whether simple or gonorrhœal; decomposed urine in cases of paraplegia; and the use of catheters which have not been made thoroughly aseptic. Stricture of the urethra, enlarged prostate, and an enlarged and displaced uterus or a pelvic tumour, may so obstruct the discharge of urine as to lead to pyelitis. A calculus lodged in the pelvis of the kidney produces direct pyelitis, and the inflammation soon extends to the kidney. Pyelitis may occur also in the course of various fevers—especially puerperal fever and pyæmia. Copaiba, turpentine, and cantharides are direct irritants to the mucous membranes of the urinary tract.

The **symptoms** are usually associated with irritability of the bladder (cystitis). In mild cases of pyelitis from cold, or from the use of irritants, there is pain in the region of the kidneys and ureters, with some fever, and the deposit of urates, pus, and epithelium is slight. In the severe cases the urine is alkaline, thick, and yellow, and soon throws down deposits in two layers. The lower layer is heavier and firmer, and when stirred it is tenacious and ropy—the upper layer is whiter, is not tenacious, and it can be decanted with the urine easily, while the lower stratum sticks to the jar. Microscopically, mucus and pus corpuscles, with crystals of triple phosphates, and sometimes epithelial cells from the pelvis of the kidney, are found; and also large numbers of bacteria. As the case advances there is great debility and emaciation, and frequent rigors or "chills." The fever is sometimes very like typhoid, and the case often terminates with delirium, stupor (from uræmia), diarrhœa, and exhaustion. The simple cases of pyelitis are curable in a few weeks; but the marked cases become chronic, and may extend to one, two, or three years. Suppurative nephritis shortens the duration.

The **treatment** of the mild cases is to dilute the urine. Acetate of potash should be prescribed, and barley water. When the urine is alkaline, give ammonium benzoate (ten grains, four times a day). Gallic acid is useful; and quinine should be administered to control the fever and inflammation. Iron tonics are also indicated. Small doses of eucalyptol are useful to subdue the irritation; and in pyelitis, associated with calculi, ℞ 31, is useful, especially when there is cystitis, &c.

Perinephritis. Perinephritic Abscess.—The connective tissue around the kidney becomes congested and inflamed, and suppuration soon follows. The pus may rupture into the peritoneum or bowel; or it may discharge in the lumbar region; or burrow along the psoas muscle, and open under Poupart's ligament.

The **causes** of perinephritis are direct contusions and injuries; extension of a pelvic cellulitis; and chronic pyelitis.

The **symptoms** are *pain* in the lumbar region, especially when movement is attempted, or when the part is pressed. There is fever and general *malaise*. Rigors indicate that suppuration has occurred. A swelling may now be discovered in the flank, and fluctuation may be made out. The case may terminate in discharge of the pus, as indicated.

The **treatment** is to apply cold in the very early stages, and to give smart purgatives. Leeches are also applied to the lumbar region; and quinine and morphia should be given. After suppuration, a free incision should be made, and the abscess well drained.

Malignant Disease of the Kidney.—Sarcomata occur in children. In adults, cancer may be primary or secondary, and the tumour attains to a large size in a very short time.

Pain and hæmaturia are the chief symptoms, and soon the development of a tumour in the right or left hypochondrium—extending into the iliac regions ultimately—indicates the nature of the disease. There is marked cancerous cachexia as it develops.

Tubercle of the Kidney. Phthisis of the Kidney.—The tubercles may be disseminated or localised. The disease may begin in the bladder, pelvis, or testicles, and then spread to the kidneys.

The urine soon becomes ammoniacal, and it is thick with pus and detritus. Blood and albumen are present. Pain in the lumbar region is usual, and it is often paroxysmal. A tumour may sometimes be made out. If the bladder be also the seat of the disease, there is frequent micturition and symptoms of cystitis. Uræmic symptoms may supervene, or death may occur from slow exhaustion.

Cystic disease of the kidneys is generally associated with the cirrhotic condition. The symptoms are similar to cirrhotic Bright; and sometimes a tumour may be made out (see diagnosis).

Hydatids of the kidney are rare. There are no symptoms during the early development. Pain, and the development of a smooth, elastic tumour, are the first indications. The sac may rupture into the pelvis of the kidney, and give rise to severe renal colic. When the vesicles reach the bladder, the pain ceases; but, again, there is pain in the urethra in attempting to pass these bodies. Hooklets found in the urine are diagnostic, and their presence at once clears up a doubtful case. The *treatment* is the same as in renal colic; but when the case is clear, injection of iodine or bile should be practised.

Floating or movable kidney may cause a dragging pain in the back, or it may give rise to secondary symptoms, chiefly affecting the digestive organs—as nausea and vomiting. Secondary peritonitis,

or perinephritis, may supervene. The kidney itself can be felt, freely movable, and eluding the grasp. It may descend as low as the iliac region, but it is usually felt about midway between the inferior border of the ribs and umbilicus. It descends upon full inspiration, and ascends upon full expiration. The *treatment* consists of endeavouring to fix the kidney by means of a pad and spring bandages.

Paroxysmal hæmatinuria is a rare disease, characterised by the occasional discharge of dark, port-wine coloured urine, and associated with general *malaise* and pain in the loins. Malarial poisoning is sometimes a cause, and sometimes it appears to be due to a chill, in certain individuals. It sometimes follows extensive burns, and sunstroke. Anti-malarial remedies are usually prescribed.

Chyluria is a condition caused by the parasite *Filaria sanguinis hominis;* or sometimes by dilatation of the lymphatic vessels. The urine contains a milky coagulable deposit, pink-white in colour; and, as this frequently clots in the bladder, there is often pain in the attempt to pass these clots through the urethra. Turpentine may be prescribed.

Simple active congestion of the kidneys arises from the use of irritating drugs, as turpentine, cantharides, copaiba, &c. Pain, and sometimes hæmaturia, with frequent micturition, are the chief symptoms. The treatment is to remove the cause, and to give diluents—as Vichy water, barley water, &c.

Passive congestion of the kidneys is due to obstructive circulatory changes—as heart disease, emphysema, and thrombosis of the veins, &c. The urine is scanty, dark in colour, acid, and it deposits urates in abundance. The specific gravity is high, and the urea and other solid constituents are increased in quantity. Albumen is present, and dropsy supervenes. The treatment is that of the central cause—digitalis and cathartics usually being indicated; and the management of the case, for a time, is the same as in acute Bright's disease.

The Diagnosis of Diseases of the Urinary System.—The differentiation of the several forms of *Bright's disease* has already been made (p. 136). The *passive congestion* of the kidneys, which results from obstructive circulatory diseases, has also been mentioned. The amount of albumen in such cases is generally small, but in severe cases it may be large. The history of the disease—especially of the early symptoms—is important; but it is often difficult to say which is the initial disease when a cycle of chronic diseases exists, as bronchitis, &c., with dilated heart, and albuminuria. It may be a case of chronic bronchitis, with emphysema and dilated heart, with passive congestion of the kidneys; or a case of chronic Bright's disease with secondary bronchitis, &c., &c. As in the lung affections, the *diagnosis*, then, is that of a "complication of diseases."

Albuminuria occurs in some individuals very readily, and it may only be transient. It sometimes is paroxysmal, with complete disappearance during the intervals. Pastry, and other articles of

diet, in excess, often produce albuminuria. It occurs also in fevers, erysipelas, diphtheria, and in chronic affections of the skin. Also, in simple congestion of the kidneys with a removable cause, albuminuria is but temporary. It should be noted that albumen is present —generally in smaller quantities, however—in all diseases of the urinary system associated with blood and pus in the urine—*e.g.*, calculi, pyelitis, cystitis, gonorrhœa, &c. It is sometimes present also in persons who have none of the other symptoms of Bright's disease, and who appear to enjoy good health, although the albuminuria is permanent. Such cases, however, must always be regarded with suspicion—especially in relation to life assurance—as, even with no other sign, it is, as it were, a *leakage* from the system which must call upon the reserve strength.

Diabetes insipidus may resemble a case of cirrhotic Bright's disease in which albuminuria is absent for a time. The history, and the presence of other physical signs of cirrhotic disease, will usually clear this up. Glycosuria must be *permanent* to constitute a case of *diabetes mellitus*. Minute quantities of sugar in the urine is a symptom of gout. It occurs frequently after chloroform and ether inhalations; and, also, sometimes in asthma, whooping-cough, epilepsy, and croup—especially after the seizure. Glycosuria is also often present after the ingestion of a large quantity of saccharine food. It should be noted, however, that transient attacks of glycosuria may precede for a time the fully developed form of it (diabetes mellitus).

Renal colic may be mistaken for biliary colic. The situation of the pain, and the subsequent development of jaundice in the one case, and of irritable bladder with hæmaturia in the other—will soon distinguish the diseases. Uric acid is the commonest calculus; but a microscopic examination of the urine is important in determining the nature of a calculus. Phosphatic crystals found in the urine may proceed from a uric acid calculus embedded within the pelvis of the kidney, and which has become encrusted with phosphates.

In the simple forms of *pyelitis* the characteristic epithelium of the pelvis of the kidney may be found in the microscopic examination of the urine. When not found, the diagnosis must frequently be conjectural.

Suppurative nephritis may be mistaken for typhoid fever in the later stages, and when the history of the case has not been made clear.

Hydronephrosis is frequently mistaken for ovarian tumour, or *vice versâ*. The development from above downwards should suggest a proper diagnosis; but sometimes aspiration is necessary before an opinion can be expressed. The fluid withdrawn from the sac in hydronephrosis contains urea, uric acid, and epithelium.

Perinephritis, *hydronephrosis*, *hydatid cysts*, *cystic disease*, and *cancer* are all characterised by the presence of a tumour, but in the first there is a history of suppuration; in hydronephrosis, cystic disease, and hydatid cysts there is an enlarging tumour without much pain. The tumour, when large enough, is felt in front, and

percussion reveals the colon above and in front of the swelling, separating it from the liver or spleen dulness. In cancer there is pain and hæmaturia. The examination of the urine may reveal the vesicles of a hydatid cyst, but otherwise—without an exploratory puncture—it cannot be differentiated. *Cancer* of the right kidney resembles a tumour of the liver; but the fingers may be pushed between the liver and the tumour. In the left kidney, it has to be distinguished from splenic enlargements; but the shape, and the position of the colon—in front of the renal, and behind the splenic tumour—and the history of the case, and presence of hæmaturia, are the points to be noted. Ovarian tumours and aneurisms may be mistaken for cancer of the kidney. The mode of development, position, and form, and the presence or absence of hæmaturia are the points of importance in the differentiation. Hæmaturia, without other symptoms, should suggest paroxysmal hæmatinuria. A spectroscopic examination should be made in doubtful cases.

New growths of the bladder, &c., cystitis, retention and incontinence are considered in surgical works, although often coming under the physician's care. *Suppression* of urine (anuria) is often *not* surgical in its origin—as in the case of fevers, shock, and other causes not understood. Cupping and diaphoresis is the treatment of such cases. A small piece of camphor may be eaten, and is found, clinically, to be of service.

Uræmia is produced by the retention in the blood of the toxic urinary constituents. *Acute* uræmia sets up violent headache, vertigo, disordered vision, twitchings of the muscles (especially of the face), and, finally, convulsions. Between the convulsive seizures the patient lies, more or less, in a comatose condition, but sometimes he is conscious throughout. At other times there are no convulsions, and the patient becomes comatose early. The pupils are dilated, and the breathing is shallow and irregular, but sometimes it is stertorous. The patient may seem to improve occasionally, and then relapse. Sometimes there is marked and even wild delirium. If the cause cannot be removed, the case terminates in fatal coma. *Chronic* uræmia consists of the development of these symptoms more slowly. Constant drowsiness, nausea, and vomiting are early symptoms; and *albuminuric retinitis* is generally present. Intense headaches are complained of, and the acute symptoms ultimately supervene and terminate the case.

In acute Bright's disease, uræmic symptoms may last three or four days—sometimes rapid cases terminate in a few hours—and the chronic cases may extend for weeks or months, with more or less improvement during short intervals, while under proper treatment. The *prognosis* will depend upon the cause. Very acute uræmia, in scarlatinal Bright's disease, or in pregnancy, is often completely relieved; whereas in chronic Bright's disease, it generally indicates that a fatal termination is at hand. In the *diagnosis of uræmia*, alcohol, opium, belladonna and strychnine poisoning; cerebral disease; and epileptiform convulsions—should all be remembered. An examination of the urine should be made as soon as possible—

drawing some off with a catheter, if necessary. In the *treatment* of uræmia, chloroform inhalations, and even a hypodermic injection of half a grain of morphia—for severe convulsions—may be given in the emergency. (See also the treatment of acute Bright's disease).

Such surgical diseases as stricture of the urethra, prostatic disease and abscess, should be remembered in connection with the secondary diseases of the kidneys, and their more remote symptoms in the later stages, which often cause patients to seek the advice of the physician.

CHAPTER VII.

DISEASES OF THE DIGESTIVE SYSTEM.—Section I.

Contents.—Stomatitis (*various forms*)—Inflammations of the throat—Tonsillitis—Diseases of the œsophagus—**Dyspepsia,** including the forms: *simple acute, atonic,* and *nervous; acute gastric catarrh; chronic gastric catarrh* (*irritable, oxaluric,* and *hepatic*)—Gastric ulcer—Cancer of the stomach—Dilatation—Acute (toxic) gastritis—Gastralgia—*Differential diagnosis of diseases of the stomach*—Hæmatemesis.

Stomatitis.—There are various forms of inflammation of the mouth. *Simple stomatitis* may be caused by local irritants, or it may be due to extension of catarrhal inflammation from the adjacent mucous membranes. The *follicular* or *aphthous* form is very common in children, and it consists of a fibrinous exudation of a grey-yellow colour. These may, at first, be only the size of a pin-head, but often they run together and form patches which ulcerate and afterwards cicatrise. This form is associated with frequent and acid stools, and with derangement of the digestive organs. The *ulcerative form* is common in the strumous child, especially when the hygienic surroundings are bad. The parasitic form—*muguet* or *thrush*—is due to the presence of the vegetative growth—*oidium albicans.* It gives rise to large white and curdy-looking patches upon the mucous membrane of the mouth, tongue, palate, and lips. In this form there is vomiting and diarrhœa. The patient becomes anæmic and emaciated; and cerebral symptoms, in severe cases, may supervene (spurious hydrocephalus). The *gangrenous form* or *cancrum oris* is a very severe type of inflammation which may destroy the whole cheek, and the necrosis may extend to the jaws. Mercury, in continued overdoses, produces an inflammation of the mouth and gums.

In all forms of stomatitis there is at first heat and dryness of the mouth, with pain. The pain is increased when acid or sweet substances come in contact with the erosions or ulcers. The secretions soon become increased, and the breath becomes fœtid. In the

severe ulcerative and parasitic forms, and especially in the gangrenous stomatitis, the symptoms are often intense and of an adynamic type.

In the **diagnosis** syphilis and diphtheria require to be remembered (*vide*).

The **treatment** depends upon the form. Simple stomatitis is best cured by regulating the diet—avoiding acids and sweets for a time. Milk with lime water, or with a pinch of bicarbonate of soda, is best for infants. The same line of treatment is indicated in the ulcerative forms. Chlorate of potash—a teaspoonful to a tumbler of warm water, to which a teaspoonful of glycerine may be added—is a useful gargle or mouth wash. A little may be swallowed. Borax and honey, as a local application, is useful for infants. The ulcers may be touched with caustic or blue stone. In the parasitic form ℞ 32 should be used. The gastric derangement may best be treated with bismuth and soda, ℞ 33. In the mercurial stomatitis iodide of potassium is indicated.

Glossitis, or inflammation of the tongue, is treated in surgical works. Secondary swelling, due to obstruction of the salivary duct by a calculus; gummata; cancer; and hypertrophy of the tongue—should be noted in the differential diagnosis.

Inflammations of the Throat.—*Simple acute sore throat* is generally the result of cold; but it may be caused by excessive smoking, alcoholic excesses, or other irritations. The pharyngeal mucous membrane becomes red and swollen, and the follicles often project and are enlarged (*follicular sore throat*). The tonsils are swollen and red, and the orifices of the Eustachian tubes may be affected. Glairy mucus lies upon the mucous membranes, and hæmorrhagic extravasations are often present. Ulceration often occurs during the acute stage.

When the sore throat is *chronic*, there is less redness and swelling, but the veins are seen to be tortuous and the follicles prominent. Superficial ulcerations and erosions form, and the mucous secretion contains pus, and blood occasionally. The palate is relaxed, often on one side, and the acute attack often leaves the *relaxed throat*. A relaxed sore throat may, however, arise without any acute symptoms. The *herpetic* form of sore throat is characterised by a vesicular eruption, usually affecting the tonsils. A sub-acute ulcerated throat, when the ulcerations are dirty and wide spread, is sometimes described as a "drain throat." The chronic ulcerated throat may follow the acute—syphilis and scrofula being often the constitutional diseases producing these.

The **symptoms** of sore throat are too familiar to need much description. They are all associated with general *malaise*, headache, and symptoms of "cold in the head," with the appearances just described. Pain in the ears is commonly present when the Eustachian tubes are affected. In the chronic forms, "hawking" becomes a disagreeable habit.

In **tonsillitis**, or **quinsy**, the inflammation may be superficial, and may be followed quickly by ulceration; or it may affect the connective tissue (peritonsillitis) or the deeper structures (interstitial tonsillitis)—the two latter forms commonly ending in suppuration.

There is intense hyperæmia of the tonsils, extending to the palate and uvula. The tonsils may be so swollen as almost to meet in the middle line. The secretion of mucus is increased, and it soon becomes muco-purulent. When pus forms, it tends to burrow down, and into the pillars of the fauces.

The **symptoms** are at first those of an ordinary "cold or chill," followed soon by heat, constriction at the throat, and pain on swallowing. The temperature rises to 102° or 103° F. The breath is fœtid, and the tongue heavily coated. Pain shooting up to the ear is common, and it is often associated with noises in the ear and temporary deafness, due to the pressure upon the Eustachian tubes. The voice has a muffled tone, and the breathing is affected, especially if there should be much œdema of the surrounding parts. In inflammation of the deeper structures, the tonsillitis is usually unilateral, and the parts have a hard, brawny feel. Rigors indicate suppuration, and there is danger of the pus burrowing down beneath the aryteno-epiglottic folds, and the possibility of œdema glottidis supervening. *Fluctuation* may be made out with the finger. In severe tonsillitis, the glands of the neck enlarge and are very tender.

The *chronic* form of tonsillitis is generally a manifestation of the strumous diathesis, and less frequently the result of acute attacks. Cold is generally the cause of acute tonsillitis. The tonsils have been supposed to be in some way connected with the sexual organs, but these relations are not very well defined. Chronic hypertrophy of the tonsils, with frequent subacute attacks of inflammation, is a very common condition met with in practice. Tonsillar affections are more common in youth. The *duration* of acute tonsillitis is about a week or ten days. Rapid recovery is the rule after free exit of the pus.

[*Retro-pharyngeal abscess* may be the result of inflammation of the loose connective tissue, but more usually it is caused by disease of the cervical vertebræ, or by suppuration of the deep lymphatic glands, extending to the pharynx. There is fever in the first case, and then a hard swelling may be made out, with pharyngeal obstruction. The latter symptom, with stiffness of the head, may be the first symptom calling attention to the abscess, when it is secondary. There is danger to life if not evacuated.]

In relation to the **diagnosis** of sore throats, generally, the scarlatinal pharyngitis and syphilitic ulcerations have to be remembered. *Simple sore throats* are frequently accompanied by red rashes, especially in children. Diphtheria requires to be noted, and the superficial form of tonsillitis with "diphtheritic" looking ulceration, requires to be carefully differentiated. The *ulceration* in the superficial tonsillitis is more limited to the tonsils (see *Diphtheria*). Ordinary sore throats often accompany fevers, and inflammations of the air-passages.

and lungs; but as the symptoms of the latter are sometimes latent, a sore throat and cough should always suggest an examination for them.

The Treatment of Inflammations and Ulcerations of the Throat.—The simple acute forms have been considered with the treatment of general catarrh, and need not, therefore, be repeated (see *Coryza*). *Quinine* should be given, in five grain doses, every fourth hour, in acute tonsillitis, with much fever. Warm gargles of hot milk and water may be ordered; but sometimes ice, and cold gargles, give most relief. After suppuration the tonsils should be incised.

The chronic sore throats are best treated by astringent and alkaline gargles or douches. An alkaline lotion—*strength*, five grains of carbonate of sodium to an ounce of water—is useful to remove mucus. Sulphate of zinc, sulphate of copper, or nitrate of silver—*strength*, one grain to the ounce of water—are all used as astringent gargles. Glycerine with tannin is also much used, and it may be painted on with a brush. The ulcerated sore throats require gargles of chlorate of potash, or of Condy's fluid. ℞ 32 may be used as a local application, or boroglyceride—the latter especially when the patient is too young to gargle. Iron should be prescribed during convalescence, and in the chronic forms of sore, relaxed, or ulcerated throat it should be continued for a considerable time. Burroughs and Welcome's atomizer is a useful instrument for the application of ointments and vapours to the throat and nose. *Paroleine*, used in this way, is said to be efficacious in the chronic relaxed forms of sore throat with much hawking of mucus.

The **œsophageal diseases** may conveniently be grouped, as pain and difficulty in swallowing (dysphagia) are common to them all. An *œsophagitis* may result from extension of inflammation from the mouth or stomach; but more frequently it is caused by the swallowing of irritant and corrosive substances. *Stenosis* of the œsophagus may arise, as a secondary condition, the result of cicatrisation of the mucous membrane. More frequently, stenosis is caused by compression, as by aneurisms and tumours, enlarged thyroid, or deep lymphatic glands. Oftenest of all, it is the result of pressure by cancerous developments. Fibroid polypi, parasites, and foreign bodies may also cause stenosis; and it may also be purely *spasmodic*—without any organic lesion. Dilatation of the œsophagus from a weakening of the walls (fatty degeneration), and diverticula, usually produced by the lodging of foreign bodies within the tube—are frequently met with.

An intelligent and cautious use of the œsophageal bougie will serve to locate obstructions and differentiate stenosis from dilatation. The obstructions are usually at the lower third of the œsophagus. Regurgitation of the food is a common symptom of obstructive disease of the œsophagus, especially when the lesion is severe and localised. *Auscultation* reveals the normal œsophageal sound as suddenly arrested, and gurgling and sputtering sounds with the

commencement of regurgitation. Hunger and thirst, with gradual emaciation, are symptoms common to all obstructions of the œsophagus. Cough and dyspnœa are often caused in the attempts to take food.

The **diagnosis** of stenosis is not difficult, but to trace its cause is sometimes not so easy. Hysterical and hypochondriacal dysphagia have to be remembered, and usually the symptoms in such cases are not so urgent, and there is not such emaciation. Dysphagia may also be the result of paralysis of the palate (see *Diphtheria*). The history of the case (scalds, burns, &c.), and the presence or absence of the cancerous cachexia are important points. The signs of aneurism, mediastinal or cervical tumours, enlarged thyroid or lymphatic glands, have all to be looked for, confirmed, or excluded.

The **prognosis** in organic obstruction is usually very grave; but simple cicatrisations are sometimes relieved by systematic dilatation.

The **treatment** of œsophagitis is to give bland food. In the obstructions the treatment can only be palliative, or surgical interference may benefit the case. Neural sedatives may be given in spasmodic cases. Dilatation with bougies is practised in the simple forms.

Dyspepsia.—Dyspepsia consists of a disturbance of the normal chemical and vital changes concerned in the act of digestion. It is a functional disorder, and it may exist as an independent affection, or it may be the result of acute or catarrhal inflammation of the stomach. It is often present as a secondary consequence of disease affecting other organs of the body; and it may be due to debility after fevers and exhaustive diseases.

The **pathology** of dyspepsia is often obscure. It appears to be sometimes the acid and sometimes the ferment of the gastric juice, which is at fault; but the proper mastication of the food, the nature of the food itself, and the action of the salivary glands, liver, and pancreas, have all to be considered in the diagnosis and treatment of a case of dyspepsia. These can only be understood by a thorough knowledge of the act of digestion (see Landois and Stirling's *Physiology*).

There are various forms of dyspepsia described, and these will be taken in order, although in practice they are not so sharply defined, and frequently the forms are mixed.

Simple acute dyspepsia is generally sudden, and the result of error in diet. The ingestion of large quantities of fruit, such as grapes, figs, &c., will in most individuals produce a sensation of discomfort, which is soon followed by sickness and pain, and which generally ends in vomiting, with immediate relief. It is not always caused by fruit, but, indeed, the individual idiosyncrasies are too numerous to mention in detail, and "what is one man's food is often another man's poison." Experience, only, can teach one what he is to avoid. Similar symptoms may arise, and yet, apparently, there has been no self-indulgence, and the food has been wholesome enough. Persons of weak digestion naturally, unpleasant sights,

eating in haste, and active exercise immediately after a meal, may all excite an attack of acute dyspepsia with pain and vomiting. The more *chronic* state of dyspepsia generally results from long continued abuse of certain articles of diet, of which *tea* is a common example. *Gout* is also a very common cause.

Atonic dyspepsia may be subacute or chronic. It is the form associated with general debility or loss of tone, and it often arises from co-existing chronic disease. It is most common during convalescence from typhoid fever, and from other exhaustive diseases. The appetite is poor, and there is discomfort or pain shortly after eating. There is no thirst, as in the catarrhal type, and no vomiting, as a rule; but acidity, and sometimes nausea, with eructation and flatulence, are the prominent symptoms. Constipation sometimes exists; but diarrhœa is more frequent, especially when the ordinary diet is adhered to.

Nervous dyspepsia is a chronic form associated with the nervous diathesis. The appetite is often good, but frequently vitiated. Vomiting is common, and the pain and acidity are often great, and accompanied by the usual flatulence, water-brash, and nausea, common to the whole group. Gastralgia is common, and pain is often present before a meal, and is relieved by eating.

In the three preceding forms of dyspepsia, there are no symptoms of a catarrhal state of the mucous membrane. Thirst is absent, and generally there is no headache. The patient does not complain of a bad taste in the mouth. The tongue is pale and flabby, and it is not coated with fur, as in the following forms.

Acute catarrhal dyspepsia, or more commonly, **acute gastric catarrh,** is a very common disease. It is readily produced by over-indulgence in the pleasures of the table, by errors in diet, and by the excessive use of condiments and alcoholic stimulants.

Certain atmospherical conditions, especially during the summer and autumn, seem to favour its development; while feverish colds and other more serious disorders, often are accompanied or followed by gastric catarrh. The congestion of the stomach, which is associated with valvular disease of the heart, is a very common cause of this form of dyspepsia. The gouty and rheumatic *diatheses* are also favourable to its development.

The **pathology** is that of a simple congestion, and subacute inflammation of the mucous membrane of the stomach. This may affect the entire organ, or it may exist in patches—the cardiac end of the stomach being generally the most affected by the hyperæmia. The mucous glands are prominent, and in severe cases, there is much tenacious mucus lining the coats of the stomach. Superficial erosions and ulcers may also be present.

The **symptoms** begin with slight nausea, loss of appetite, and vertigo, with pain or discomfort in the epigastric region. There is often heartburn, acidity, flatulence, regurgitation, and vomiting—the latter giving temporary relief. The tongue becomes coated with white or brown fur, and it is dry and clammy. The patient com-

plains of a bad taste in the mouth, especially in the morning. The breath is offensive. There is almost always great thirst, and severe frontal headache. The temperature rises one or two degrees. The bowels are confined; or diarrhœa may be present. The patient is much depressed and easily fatigued during the day, and at night he is often sleepless. In the aged the vertigo is often so great, and sometimes so sudden, as to simulate an apoplectic seizure. With proper treatment, recovery is the rule in about a week or ten days.

Chronic Gastric Catarrh.—The **pathological conditions** in the chronic form are more marked. The mucous membrane of the stomach may be indurated and much thickened; but sometimes it is much softened. The glands are either shrunken or have become cystic. Hæmorrhages and erosions are more marked, and the mucus is great in quantity and very tenaceous, and the membrane is of a *dirty slate-grey colour*.

The **symptoms** are similar to those of acute catarrh—only more variable and more chronic in character. The tongue is not so coated with fur, but it is red and cracked, and often flabby. The appetite is capricious. Various forms are described, viz.:—

The Irritable.—The tongue is red, cracked, and painful; the appetite often keen, but easily satisfied, or the patient eats voraciously and overloads the stomach; thirst is always present. There is pain an hour or two after a meal, and great irritability of temper, with eructation, pyrosis, vomiting, and other symptoms common to indigestion. The bowels are generally loose.

The Oxaluric.—The patient has a sallow, worried, or hungry look; the tongue is pale and indented; the appetite is poor, and there is not much pain after eating. There is general *malaise* and great depression; the urine is acid, and there is excess of lime crystals deposited. There is not so much thirst in this form; and it is frequently associated with the nervous diathesis.

The Hepatic.—This is the "bilious" form or "sick headache." The tongue is furred—usually more so at the back; and the circumvallate papillæ are enlarged. There is frontal headache, and often there is slight jaundice and vertigo, with "black specks floating before the eyes." There is great thirst. The urine is loaded with urates, and there is diarrhœa usually—the stools being often hot and irritating—along with the other symptoms common to the whole group. More or less congestion of the liver co-exists with this form of gastric catarrh. When the liver is only slightly affected, the case is one of *hepatic dyspepsia;* but when there is marked jaundice, pain, and enlargement of the organ, with tenderness upon pressure, along with the foregoing symptoms, the two conditions may be diagnosed—*i.e.*, gastric catarrh, with congestion (and enlargement) of the liver. (See congestion of the liver.)

In the **diagnosis** of this class of diseases (dyspepsia), acute gastric catarrh and typhoid fever; stomachal vertigo in the aged; and in the chronic forms, cancer and gastric ulcer—have to be noted. (See diagnosis at the end of the section, p. 161).

The **treatment** of the different forms of dyspepsia and gastric

catarrh are here taken in order, but it should be noted that as the forms met with in practice are frequently mixed, so the treatment requires to be modified accordingly.

Acute dyspepsia, arising from error in diet, requires a prompt emetic. Mustard, or salt and water may be used. A dose of castor oil may be necessary, and twenty drops of laudanum may be given, to an adult, along with it—if there be griping pains. Draughts of hot water are useful, and the diet should be light and consist chiefly of milk for a few days.

Atonic dyspepsia requires a careful regulation of the diet from the first. The "milk cure" is useful to commence with in extreme cases, the diet being gradually strengthened as the digestive powers recover. The milk may be peptonised if necessary (℞ 34). Benger's food is a highly useful preparation. Beef tea or Valentine's beef juice may be added to the dietary; and in some cases, when milk does not agree, beef tea must be substituted. Chicken soup, chicken cream, and chicken follow later. A well cooked chop should be the first meat attempted; and gradually, as digestion improves, the patient resumes his ordinary diet. A little claret may be allowed at dinner, or very small doses of whisky and water may suit better, in the debilitated cases which seem to require a stimulant. The rule is to eat in small quantities at a time. A change of air is to be recommended, and gentle exercise. The following prescriptions may be used during the course of atonic dyspepsia. (℞ 35, 36, 37, 38.) The Byno-hypophosphites are very useful in feeble digestion.

Nervous dyspepsia is treated sometimes like the atonic form, and sometimes it requires the treatment of the catarrhal types. Morphia or opium may be necessary for the pain; but it should be carefully prescribed, in case of the patient contracting the opium habit. It is better to seek to improve the general condition by arsenic and iron preparations. It is needless, however, to prescribe these when gastric catarrh co-exists. First, therefore, treat the catarrh (see below), then prescribe one or other of the prescriptions given in the atonic form (℞ 36, 37). The saccharated carbonate of iron is a mild and useful chalybeate. Liquor arsenicalis may be given in three minim doses, thrice daily in water, immediately after meals. The latter is useful in gastralgia. Treat any reflex irritations, as ovarian disease, decayed teeth, worms, &c. The diet should be carefully regulated on the lines already given under atonic dyspepsia. Strong tea is to be avoided in this, and in all forms of dyspepsia.

Acute Gastric Catarrh.—If the stomach be overloaded, an emetic may be given, and draughts of hot water should be given immediately thereafter. A saline purgative is useful, or ℞ 39 in acute cases, and no further treatment, beyond attention to the diet, may be necessary.

In those cases in which the onset is gradual, and the symptoms more lasting, bismuth, rhubarb, and soda should be given in small doses before food, as directed in ℞ 40. If there should be excessive diarrhœa, the rhubarb should be diminished to one grain. An opium pill may be necessary.

Sometimes the hepatic symptoms predominate, and then the prescriptions given under *hepatic dyspepsia* are indicated. The diet should be light and digestible, consisting chiefly of milk, light milk puddings without eggs, rice and milk, and toast, beef tea, chicken, &c. ℞ 36 or ℞ 43 is useful later.

Chronic Gastric Catarrh.—The regulation of the diet is here again by far the most important consideration in the treatment. In severe cases, the "milk cure" should be tried for a lengthened period. Skim-milk—about eight ounces every three hours, day and night (when awake)—may be given and continued for a month, or longer, without any other article of diet. The stomach gets a complete rest. Washing out the stomach with the syphon tube is also good practice, if not continued too long (see *Dilatation of the Stomach*).

In cases of less severity, the diet may be regulated as indicated in atonic dyspepsia, all starchy, saccharine, and fatty foods being avoided. Lettuce, celery, spinach, cauliflower, and tomatoes may sometimes be allowed for these. In a month or two the diet may be strengthened, but during a course of such treatment, no medicine need be given, except an occasional aperient. In the milder forms of chronic catarrh, or during an exacerbation, ℞ 40, containing bismuth, rhubarb, and soda may be ordered. The symptoms are very variable, and sometimes a chronic case will require at times very different treatment. Small doses of arsenic or silver (with the usual precautions) may be tried. Bismuth and charcoal is a useful combination for flatulence and acid fermentation. Bartholow recommends a drop of the tincture of iodine, with a drop of carbolic acid (*well diluted*) every few hours, as a remedy for abnormal fermentation and vomiting. When fairly well, a tonic (℞ 36 or ℞ 43) may be prescribed. In the *irritable* form, bismuth and pepsine preparations are indicated for a time. Only bland food (milk chiefly, and barley water) should be allowed. ℞ 41 is useful. An after-dinner pill (℞ 42) may be given in less acute cases. The *oxaluric* form requires the use of mineral acids, ℞ 43 being the best. It is frequently associated with the *nervous diathesis*, and hence the treatment of that form of dyspepsia may also be indicated.

The *hepatic* form of dyspepsia, with sometimes considerable congestion and enlargement of the liver, is a very common condition. It is met with in varying states of mildness from the patient who complains of feeling "livery," or who has an acute gastric catarrh, with some symptoms referable to the liver—up to the severe type of the disease associated with chronic gastric catarrh.

The rapid, but mild form, may be relieved by a blue pill at night, followed by a Seidlitz powder in the morning. If associated with acute gastric catarrh, the treatment will be the same as indicated in that disease. If the liver symptoms be more prominent, calomel, iridin, euonymin, and podophyllin are proper remedies. Calomel is highly useful when the stomach is irritable. ℞ 44—nitric acid and taraxacum—is very useful; and also the pill of mercury and rhubarb (℞ 45). Nitrohydrochloric acid is highly useful, and is much used (℞ 43). Draughts of weak alkaline waters are useful for thirst, and

to flush the tubules of the kidneys, and dilute the urine when clouded with urates. If the pain in the hepatic region be great, in consequence of the liver congestion and enlargement, turpentine stupes may be used, or mustard may be applied. It is important in these cases to relieve the portal system—and possibly the hæmorrhoids, which are so frequently present in liver affections—by gentle but efficient cathartics.

Gastric Ulcer.—In by far the greater proportion of cases the ulcer is solitary; but sometimes they are multiple, and they may coalesce. They are usually found on the posterior wall, the lesser curvature, and near the pylorus. The acute ulcers are round or oval in shape and about the size of a shilling piece. They have smooth edges at first; but when chronic, the edges become indurated in shelving layers, the point of the excavation reaching the muscular or even the peritoneal layer. Perforation may take place; and it is often only prevented by the thickened connective and granulation tissue. Cicatrisation of large ulcers near the pylorus may narrow the orifice and lead to dilatation of the stomach. Secondary adhesions to neighbouring organs, from local inflammation, are frequent; and sometimes communications are established between the stomach and colon; or an external fistulous opening may result. When perforation takes place, the contents of the stomach pass into the peritoneal cavity and set up general peritonitis.

The actual **cause** of a gastric ulcer is the blocking of an arterial terminal twig by a clot (embolus) cutting off the blood supply. Atheroma and endarteritis, by inducing thrombosis, are important factors in their production. Gastric ulcers are well known to be associated with the anæmic and puerperal states; while exhaustive diseases—as tuberculosis, &c.—favour their development. The disease is very common in young servant maids. Extensive burns of the chest and abdomen are known to be associated with ulceration of the duodenum, so that a *nervous* element must be considered a factor in the causation of ulcers.

The **symptoms** are generally of a chronic character, and for a time simple indigestion may be all that is suspected. Sometimes a sudden perforation takes place without there having been any previous symptoms to suggest the presence of an ulcer. Chronic dyspepsia with severe pains, sometimes referred to the side and front, and often shooting through to the back, are suggestive; and if there should be also vomiting of blood, then a gastric ulcer may fairly be assumed, when the other conditions mentioned under the diagnosis, later, can be excluded. The vomited matter is well described, sometimes, as like hare soup or "coffee grounds," but at the end of the act pure blood is frequently brought up. The pain is often very violent, and burning or gnawing in character, and the patient can often indicate with a finger the exact spot of maximum intensity. During a paroxysm there may be great prostration or syncope, and even in some cases convulsions. The pain is almost always relieved by vomiting. There is much tenderness on pressure over the seat

of the pain; and the pain is excited by food, especially when hot. At other times it is the presence of undigested food which excites the pain, about three hours after a meal, and then probably the seat of the ulcer is the pylorus.

The presence of anæmia is also an element in suspected cases, when the symptoms are not so manifest, as it is chiefly in chlorotic patients that gastric ulcers are formed. When perforation occurs there is sudden and violent abdominal pain, with great prostration, and death usually within a few hours. In the chronic cases there is loss of body weight and strength—the patient soon learning that to eat brings on a paroxysm of pain. The *course* is generally lengthy, with improvement and relapses from time to time; but a large proportion of cases terminate in recovery, especially under treatment. In the fatal cases—in addition to the sudden ending of a case by perforation and fatal peritonitis—death may result from severe hæmorrhage; or from actual exhaustion from loss of blood, or from want of nourishment. The pathology, history, course, and treatment of an ulcer of the duodenum is much the same as a gastric ulcer. They are often the result of extensive burns. Blood may be vomited, but more frequently it is found in the stools. The pain comes later, a few hours after eating. (**Diagnosis**, see p. 161.)

The **treatment** of gastric ulcer consists of giving the stomach as complete rest as possible. The avoidance of all irritating articles of diet—or, in short, an exclusively milk diet for several weeks—is the proper treatment. The milk may be given with lime water, and the nourishment may be supplemented by Valentine's beef juice, or beef tea, well made and free from fat. The patient may be supported by injections *per* rectum. Alkaline mineral waters are recommended to be used occasionally—to wash the coats of the stomach. Arsenic, silver, or bismuth are the usual drugs administered—Fowler's solution of arsenic being considered the best remedy. For hæmorrhage give ice to suck; and it may also be applied to the epigastrium with good results. Ergotin should be injected, if necessary. The pernitrate of iron may be given by the mouth. If perforation take place, absolute rest, with hypodermic injections of morphia, is all that can be attempted in the way of treatment.

Cancer of the Stomach.—The stomach may be affected by any of the three forms—the scirrhus, medullary, or colloid. The scirrhus is cartilaginous and forms dense masses; the medullary is softer and more rapid in growth; while the colloid differs from them both in appearance, and derives its name from the gelatinous degeneration of the cancer cells. The latter form is slow in growth, and less common. A cancerous mass tends to drag the stomach down, and should it affect the pylorus—which is by far the commonest site—the stomach suffers consequent dilatation from the stenosis. When the cardiac end of the stomach is the seat of the disease, the organ becomes contracted. Chronic catarrh of the stomach is present with the cancer, which is usually primary. Fatty heart and tuberculosis are often complications.

The **etiology** is still obscure, but heredity is established as a cause; and it rarely affects individuals under forty years of age.

The early **symptoms** are not distinctive; and until a tumour can be felt or the cachexia be developed, the symptoms are merely those of dyspepsia, in which *pain* is an almost constant feature. The pain, however, is not marked if the body of the stomach be the seat of the disease. It is invariably present when the pylorus, or cardiac portion, is affected. In some rare cases, cancer—especially the colloid form—may run its course without any characteristic sign, and only the *post-mortem* examination reveals the true nature of the disease. As the disease advances the dyspepsia becomes more aggravated. Pain, loss of appetite, discomfort and distress after food—as acidity, pyrosis, flatulence, and vomiting—are the symptoms common to all forms of cancer. Should the disease affect the cardiac end of the stomach, there is obstruction of the food, and pain—usually referred to the œsophagus. The emaciation and loss of strength is quicker in this form, and the feeling of hunger is sometimes difficult to satisfy. Vomiting of blood (hæmatemesis) is present in most of the cases, although the quantity rejected is not so great as in gastric ulcer. The vomited matter may be only tinged with blood, or small masses of brown-black decomposed blood may be present when the case is advanced. In many cases of cancer there is no hæmatemesis. Diarrhœa, in the later stages, is the rule—due to irritating matter passing into the bowels. Blood may also be present in the stools. The cancerous cachexia is that pallid, earthly, or "fawn" colour of the skin, associated with the debility and the emaciated appearance which always accompany the disease. A weak action of the heart, feeble pulse, and œdema of the ankles are secondary consequences, which generally usher in a fatal termination.

The tumour cannot be felt at all should it affect the cardiac end of the stomach, or should the disease be the colloid form. In the latter case, a diffused sense of resistance over the stomach may be appreciable. Tumours of the posterior wall may also escape detection. When the pyloric end of the stomach is the seat of the disease, hard nodular masses may be felt, especially if the patient be much emaciated. The tumour in such cases is generally much lower down than one would expect. Distention of the stomach may prevent the palpation of a tumour (see *Diagnosis*, p. 161, for the sources of error, &c.). It should be noted that a tumour of the stomach does not descend with full inspiration. Cancer may result in perforation and peritonitis; or in fistulous communications. The liver is often affected along with the stomach; and masses of adhesions to the neighbouring organs and structures are commonly found. The mesenteric glands are enlarged, and sometimes the cervical glands. The average duration is about one year.

The **treatment** can only be palliative. Milk and beef tea may be the only food which can be borne. Washing out the stomach gives relief. Arsenic sometimes relieves, and Bartholow recommends equal parts of carbolic acid and tincture of iodine—one drop of each, well diluted—for vomiting. In most cases morphia must be used, either by mouth, or hypodermically.

Dilatation of the stomach may arise as a disease *per se*, and then it is the result of over-eating or drinking. Excessive beer-drinking is a common cause. More usually, dilatation is the result of stenosis of the pylorus, and this may occur from simple inflammation and subsequent contraction of the submucous connective tissue; or it may be the result of cicatrisation of a gastric ulcer. Most frequently, it arises from cancerous disease. Tumours of other organs may also press upon the pylorus, and give rise to dilatation. The stomach walls are much thinned, and if there be no stenosis, the muscular layers are much atrophied. The organ is sometimes enormously dilated.

The **symptoms** are those of chronic dyspepsia; but the vomiting of large quantities of sour and semi-liquid food—putrid and fermenting—is the characteristic symptom. *Sarcinæ* are present in great numbers. Cramping pains in the limbs are often present. The urine is diminished in quantity, and the fæces are dry—the bowels being constipated. There is loss of body weight and strength, and in severe cases, actual emaciation—even when cancer is not the cause.

The **physical signs** vary with the state of the stomach in relation to the food. *When full*, there is dulness at the lower borders (when the patient is in the erect position), and the stomach is seen to be bulging, and to reach below the umbilicus; *when empty*, a tympanitic and metallic note can be elicited over the same area. *Splashing* of the stomach contents can readily be made out when the abdomen is shaken suddenly with the palm of the hand. A more exact diagnosis may be made by passing a stomach syphon tube, and feeling the point of it through the abdominal wall, in an abnormally low position.

Good results, in simple uncomplicated cases, may be expected by Kussmaul's treatment of washing out the stomach with warm water and very dilute Condy, once a day. Hyposulphite of soda may also be used for the destruction of the sarcinæ. Washing out the stomach, however, is apt to keep up the distention, when too long continued. At the beginning, however, it is useful to purify the stomach, and thus to prepare it for other treatment by getting rid of the putrid matter, which is constantly affecting the wholesome food newly introduced. A *dry diet* is important; and the avoidance of all starchy and saccharine substances. Emetics may be given when the stomach tube cannot be used. Tonic doses of strychnine and hydrochloric acid should be prescribed. Galvanism may be tried in obstinate cases.

Acute gastritis, or inflammation of the stomach, is usually caused by irritating and corrosive poisons (toxic gastritis). The ingestion of large quantities of cold water or iced food, when the body is heated, may produce a rapid congestion with acute pain in the epigastrium. **Gastritis suppurativa** is a rare form of inflammation, in which the submucous tissues become much thickened, and an abscess may form.

The **symptoms** are pain and vomiting; fever and depression being marked in the suppurative form.

The **treatment** consists of morphia—by the mouth, or injected hypodermically; bland food—chiefly milk and peptonised gruels; and the application of hot fomentations or poultices.

Gastralgia, in its true form, is a chronic neurosis—the sensory nerves of the stomach being in some peculiar state. It is associated with the neurotic temperament, or with the hysterical, and frequently it is an accompaniment of uterine disease. The abuse of tea is supposed to be, in some cases, a cause of the affection. The pain occurs in paroxysms, and it is felt in the epigastrium, radiating over the chest and abdomen, and through to the back. It is *diminished* by pressure. The attacks are usually short and recurring, and they are accompanied by palpitation of the heart, weak pulse, and often great prostration. There is usually great flatulence and, ultimately, vomiting.

The **treatment** requires morphia or opium in the first place; and afterwards, quinine, arsenic, iron, or silver, should be given for a lengthened period. Care must be taken not to continue the silver pill too long, in case of producing *argyria*. The *milk cure* may be tried; and in all cases the food should be light, and only taken in small quantities at a time, rather than in regular meals.

The **differential diagnosis** of the foregoing diseases of the stomach—viz., dyspepsia and gastric catarrh; ulcer, cancer, gastralgia, and dilatation.

The various forms of *dyspepsia* are not always sharply defined, but more frequently the symptoms are mixed, and the diagnosis is made according to the prominent symptoms and physical signs. Dyspepsia is often an independent functional affection; but it is frequently a result of antecedent disease—especially the atonic form.

The state of the heart should always be examined, as cardiac patients very frequently refer their symptoms to the stomach—the dyspepsia being a secondary consequence of heart disease. The reverse is also frequently the case. Dyspepsia is also, sometimes, a symptom of incipient phthisis; or it may be associated with Bright's disease. These are familiar instances, but any grave organic disease in other organs may produce a dyspeptic state. *Gout* must also be remembered as a constitutional cause of dyspepsia.

An *acute gastric catarrh* may turn out to be typhoid or remittent fever. Almost all feverish conditions produce more or less gastric catarrh; but the history, the amount of fever and depression, and the future course of the disease, are the chief points in the diagnosis. The *vertigo a stomacho læso* (Trousseau)—occurring especially in aged persons—must be differentiated from cerebral disease.

Chronic gastric catarrh is often associated with mental diseases, especially melancholia and hypochondria; but a more common differentiation required in practice is *chronic gastric catarrh* from

ulcer of the stomach, *cancer*, and *gastralgia*. These require also to be distinguished from one another, and from a few diseases affecting other organs.

The most characteristic symptoms of *ulcer* of the stomach are the shooting pains localised in front and posteriorly, coming at any time, aggravated by pressure, and generally worse after eating, or after drinking hot fluids; the "coffee-ground" character of the vomit; and the anæmic appearance of the patient. It is often not easy to be sure, in cases of chlorosis with chronic gastric catarrh, whether an ulcer may, or may not, be present, especially if the characteristic vomit be absent. The treatment may serve to distinguish them, as an exclusive milk diet improves the ulcer but not the chlorotic condition.

In *cancer of the stomach* the pain is more fixed and continuous; the vomiting *at first* does not contain blood, and it is not a constant symptom; and the emaciation, cachexia, and age are important points. If palpation reveal the presence of a tumour, and possibly enlargement of the sub-clavicular glands, along with these symptoms, then the case is clear; but a tumour of the liver or pancreas, a movable kidney, or aneurism of the hepatic artery, require to be distinguished from cancer of the stomach, when the other symptoms which characterise that disease are obscure or absent. It is important to notice in this relation that a tumour of the stomach does not descend with full inspiration. Distention of the stomach, or the presence of fluid in the peritoneal cavity, may entirely conceal a tumour growth.

In *gastralgia* the pain is paroxysmal, and there is relief for prolonged intervals, and it is not increased by pressure. Eating may not increase the pain; and there is no vomiting of blood, nor emaciation with cachexia. The neurotic temperament is generally obvious. *Neuralgia of the solar plexus*, *hepatic colic*, *myalgia*, and *intercostal neuralgia* require to be differentiated from gastralgia. In the first there is great systemic depression with inhibition of the heart's action; in hepatic colic there is jaundice, pain in the right hypochondrium shooting to the umbilicus, and fever; in myalgia the pain is not so lancinating, and it is relieved by pressure; in intercostal neuralgia the pain is localised to the nerve, and there are tender points along its course, and at the spine.

In *chronic gastric catarrh* alone, the pain is not so intense, is more diffused, and it is often relieved by eating. Pressure does not aggravate the pain. Vomiting is not always present, but if so, there is no blood.

In *dilatation of the stomach* the physical manifestations of the disease serve to differentiate the condition from all others. It must not be forgotten, however, that it may be produced by cancerous strictures, and by pressure of tumours of other organs.

Hæmatemesis, or vomiting of blood, is a symptom of many diseases. There may be a sensation of warmth and distention in the stomach, nausea, and faintness, followed by vomiting. The blood is acid in re-action, black-brown in colour, and is not aërated. It may

be mixed with food. If the blood be vomited at once, it is red and coagulated. Vomited fruit may sometimes resemble blood.

As blood may be swallowed, then vomited, a careful examination of the nares and palate should be made. When from the lungs, the blood is *coughed* up, and not vomited, and the previous history or the physical examination of the chest, may reveal the source. Blood from the lung is alkaline, bright red, and frothy.

Rupture of a blood-vessel in the stomach is the essential cause of hæmatemesis, and this may be due to disease of the vessels; but hæmatemesis may occur in cirrhosis of the liver, acute yellow atrophy, yellow fever, aneurisms of the hepatic artery, and calculi or tumours in the neighbourhood of the portal vein. More common causes are obstructive diseases of the heart and lungs, the hæmorrhagic diathesis, alcoholism, cancer and ulcer of the stomach—the latter being much the commonest cause. The hæmorrhage is sometimes vicarious; and sometimes it arises from the arrest of a hæmorrhoidal discharge.

The *immediate treatment* should consist of absolute rest, ice to suck, and ice applied to the epigastrium. A ligature may be tied round the thighs—or the legs may be suffered to dangle over the bed, while the shoulders are slightly raised. Morphia, with dilute sulphuric acid, is a useful remedy (℞ 26). Tincture of perchloride of iron is also useful. Milk *only* should be allowed for a few days. The later treatment depends upon the cause.

CHAPTER VIII.

DISEASES OF THE DIGESTIVE SYSTEM.—Section II.

Contents.—Enteralgia—Constipation—Acute and chronic diarrhœa (various forms): Intestinal catarrh, and enteritis, &c.—Dysentery—Ileus, or obstruction of the bowels: Intussusception—Ulcers of the intestine, and cancer—Intestinal parasites—Acute and chronic peritonitis—Typhlitis, perityphlitis, and inflammation of the appendix vermiformis (Appendicitis)—Tabes mesenterica—*Differential diagnosis of intestinal diseases*—Intestinal hæmorrhage—Ascites.

Enteralgia, or colic, is a very common functional disorder, and it is of the nature of a neurosis like gastralgia. It is caused by improper food (indigestible); impaction of hardened fæces; flatulent distention of the bowels; and exposure to cold. Hysterical and neurotic persons are liable to have attacks of colic; and the disease is a symptom associated with various constitutional states—as the malarial and syphilitic—and with metallic poisoning, especially *lead*.

The twisting pains about the umbilicus may begin suddenly and violently, producing symptoms of collapse; but it is more usual to have warnings in the shape of small griping pains in the abdomen, gradually increasing in intensity until the patient feels compelled to

"double up." Pressure diminishes the pain, although after a time the abdomen may be tender. The colic is usually relieved by passage of the bowels. There is frequent desire to micturate, and constipation usually co-exists with enteralgia—especially in lead poisoning. There is no fever. The abdomen is distended when the cause is flatulence.

The *prognosis* is favourable in the simple forms of colic.

The **treatment** depends upon the cause. Morphia is indicated in the first place; and a laxative as soon as possible. Castor oil is soothing to the bowel and safest. It may be given with twenty drops of laudanum in cases of less urgency. In children, a few drops of paregoric, with one drop of essence of peppermint, in sugar and water, will be found serviceable. Quinine—in five grain doses, repeated every two hours till fifteen or twenty grains have been given—is useful treatment in neuralgic forms. Iodide of potassium must be given in large doses for lead poisoning, and continued in smaller doses for a lengthened period. Enemata of asafœtida may be tried; and arsenic is useful for the chronic cases. It is well to use hot poultices, or turpentine stupes, in cases of acute colic. They seem to relieve, and cannot do harm, while often a case of apparent enteralgia proves ultimately to be a much more serious affection (see *Diagnosis*, p. 178).

Constipation—when simple—is a state of defective alvine evacuation. In most adults the bowels are moved once daily; in some others, twice; and often again, only on alternate days (and even longer), and yet there is no interference apparently with the ordinary health. The *causes* of constipation are deficient secretion of the intestinal tract, liver, and pancreas; a failure of the peristaltic movement of the bowel from loss of tone in the muscular layer, as occurs in people of sedentary habits, and in aged persons; and certain states of the nervous system—as melancholia, spinal disease, &c., and notably by the action of lead. Abnormal conditions of the mucous membrane of the intestinal canal, diseases of the liver and pancreas, anæmia, and improper food, are common causes of constipation.

The **symptoms** associated with constipation are those which are common to dyspepsia—especially the *hepatic* form. Dulness may be made out, in extreme cases, in the line of the descending colon, and at the sigmoid flexure. Defæcation is often extremely painful, and blood, mucus, &c., may be discharged with the fæces. The *sphincter ani* may suffer from the propulsive efforts, and the mucous membrane may become cracked and fissured. Hæmorrhoids are very common; and in severe cases of constipation, varicosity of the lower limbs, pelvic congestions with uterine disorders, and sciatica, are frequently induced. The fæcal matter is often scybalous, and sometimes there is an alternate condition of constipation and diarrhœa. The latter condition may exist along with a considerable accumulation of fæcal matter in the bowel, a central canal being sometimes established through the mass. In chronic cases nutrition

is impaired, and the skin becomes dry and subject to eczema, erythema, psoriasis, &c. Headache and vertigo are common consequences.

The **treatment** will depend upon the cause. If the stools show a deficient secretion of bile, liver stimulants should be prescribed—calomel, iridin, euonymin, podophyllin, or ℞ 45. Phosphate of soda is also recommended. Paresis of the bowel is best treated by strychnine or nux vomica, with belladonna and iron (℞ 46, 47). Electricity is useful—one electrode being placed in the rectum, and the other being passed over the abdomen. Kneading the abdomen should be tried first. Two or more dessert-spoonfuls of the liquid extract of cascara sagrada with glycerine (equal parts) is a very useful remedy, especially with aged persons. Cascara sagrada is one of the rare purgative medicines, the use of which diminishes the need for it—the doses, consequently, may be gradually reduced. It may be given in small doses as a bowel tonic. Constipation is a symptom of many diseases, and it requires attention along with the treatment of these—*e.g.*, lead poisoning, cirrhosis of the liver, certain forms of dyspepsia, gastric catarrh, &c., &c.

The regulation of the diet in simple functional constipation is highly important. Wheaten or brown bread, oatmeal, fresh vegetables, and fruit are all indicated. Regular evacuation is to be inculcated, and exercise is to be taken. Plethoric individuals may use the alkaline and aperient mineral waters in the morning; but the regular practice of taking strong purgative medicines should be condemned.

Diarrhœa.—*Acute and Chronic.* **Intestinal Catarrh and Enteritis.**—The *acute* form of diarrhœa may arise from a morbid state of the mucous membrane of the intestine (catarrh), or it may be the result of increased secretion, or peristaltic action, of the bowels. Acute diarrhœa may be excited by exposure to cold, errors in diet, and by impure drinking-water; or it may arise from mental causes. It occurs as an epidemic in the late summer and autumn from decayed vegetable matter impregnating the air.

The *chronic* form of diarrhœa may be established after repeated attacks of the acute, when there has been prolonged exposure to one or other of the causes above-mentioned. It is a common disease in hot countries, and especially in China, where it is known as the "white flux." Diarrhœa occurs as a concomitant symptom of severe organic disease, as in typhoid and puerperal fevers, dysentery, &c.; and in the tubercular ulceration and waxy disease associated with phthisis. Sometimes ulcerating malignant disease of the intestines sets up diarrhœa, but this is a somewhat rare condition. Diarrhœa may also be an effect of organic disease elsewhere. It is common in heart disease, and in Bright's disease, in which, if not excessive, attempts should not be too readily made to check it, as it is often nature's method of relieving the system of *materies morbi*.

The *catarrhal condition of the intestines* may especially affect certain

parts, and hence the names, *duodenitis*, *ileitis*, *colitis*, *typhlitis*, and *proctitis* (*catarrh of the rectum*). The *pathological* changes consist of hyperæmia, followed by over-growth and desquamation of the epithelium, in the acute forms; and in the chronic forms, the changes are similar, but the mucous membranes are more thickened, ulcerated, and of a slaty colour, and they are covered with tenacious mucus and purulent matter. The epithelial cells are cloudy, and are advanced in fatty degeneration. The solitary follicles are enlarged. In *typhlitis* the catarrhal changes are similar; but the cause is usually the presence of a foreign body (hardened fæcal matter generally). The importance of this condition lies almost wholly with the localised inflammations (perityphlitis and appendicitis) which may follow. (See p. 176.) In the chronic forms, thickening and stenosis at the ileo-cæcal valve may occur. In *enteritis* the inflammatory changes are associated with the deposit upon the mucous membrane of a thick, dense, grey-white deposit or membrane, sometimes firmly adherent, and afterwards thrown off in casts or shreds.

The Symptoms.—Simple uncomplicated cases of diarrhœa are too familiar to need description. In infants, the stools are often green and acid, and the buttocks are frequently red and excoriated. In children and adults "English cholera" is a common complaint during the fruit seasons. In some cases the stools approach to the "rice-water" appearance which is characteristic of true cholera. "Choleraic diarrhœa" is sometimes described as a special form. In the very young, simple diarrhœa is a dangerous complaint, and when there are signs of depression, coldness of the body, sinking in of the eyes, and fontanelles, a fatal result may shortly be expected.

[In *duodenitis*, diarrhœa is *not* the rule, and often there is constipation. Jaundice is a prominent symptom, owing to obstruction of the bile duct with catarrhal products. Pain and soreness in the right hypochondriac and umbilical regions—about three hours after food—is always complained of. The other symptoms are as described under hepatic dyspepsia.]

Ileitis and *colitis* have diarrhœa as the prominent symptom, with pain and tenderness over the affected parts. The stools have a yellow or green colour, and they are very frequent. In severe cases they may be like the "rice-water" discharges of cholera. Flatulence, borborygmi, and colicky pains are always present in the chronic forms of intestinal catarrh. There is great emaciation, which in children may be very rapid and the case may terminate fatally after only a few hours' illness.

In *acute proctitis*, or catarrh of the rectum, there is uneasiness or burning pain in the rectum, with *tenesmus*, the pain radiating to the hips and back. The mucous membrane is often prolapsed, and the bladder sympathises with the rectum in its efforts, creating a constant desire to micturate. The colon above the sigmoid flexure is often impacted with fæces, and hard scybala from time to time are passed. The surrounding tissues often become inflamed (*periproctitis*), and this may lead to the formation of an abscess. The

condition is, therefore, analogous to that which takes place upon the other side (typhlitis and perityphlitis). The *chronic* form of proctitis has similar symptoms, but there is more *débris* and mucus in the stools, and less pain.

Enteritis is characterised by distention of the abdomen and great tenderness at the outset. There is no fever; but colicky pains are very frequent and they recur from time to time. Soon a state of *tenesmus* is induced, and a diarrhœa begins, with severe straining, which ultimately results in the passage of casts of membrane, when great relief is experienced. There is generally much emaciation and debility, and the digestive functions are much disordered. Acidity, ulcers of the mouth, and a red coated *irritable* tongue are usually present. The course of enteritis is irregular. A case may last three or four weeks and recover; or it may become more or less chronic.

The **treatment** of a symptomatic affection like diarrhœa, requires a careful consideration of the cause. In all cases of diarrhœa, whatever the cause, bland and non-irritating food is necessary. Milk with lime water, or milk boiled with rice and then strained, barley water, weak tea and toast, light milk puddings, and later, fish and chicken – is the diet indicated. When diarrhœa is the result of a cold, ten or fifteen minim doses of laudanum may be given every three or four hours, and the patient should at the same time keep his bed. If it should be the result of irritating materials in the bowel, and accompanied by griping pains, castor oil with twenty drops of laudanum is the most efficient remedy. Poultices may be applied to the abdomen if the pains be acute. When diarrhœa is the result of impure drinking-water, or when it arises from mental causes, astringent and sedative mixtures may be given (℞ 48), and the cause removed if possible. The form which accompanies organic disease, as tuberculosis, waxy degeneration, dysentery, typhoid fever, and malignant disease, is considered elsewhere. It should be noted, however, that opiates must be given very guardedly—if at all—in cases of diarrhœa occurring in the course of Bright's disease. The chronic forms of diarrhœa (simple) require astringent treatment —bismuth, opium, and iron being the usual remedies given.

With infants, ℞ 49 is useful when urgent astringent action is necessary. A powder may be given of bismuth, chalk, and mercury (℞ 50) when the cause is gastro-intestinal catarrh. In such cases, if the child be bottle fed, absolute cleanliness of the feeding apparatus must be inculcated. A change of milk is beneficial; and a pinch of bicarbonate of soda in each bottle is of value. Attention must be paid to the proper dilution of the milk according to the age. Nestlé's or Mellin's food may be substituted for cow's milk, if the child does not appear to thrive or gain weight. All the forms of intestinal catarrh—except *duodenitis*, which is considered with gastric catarrh and hepatic dyspepsia—are treated upon the same lines. Ileitis, colitis, simple uncomplicated typhlitis, and enteritis, all require astringent remedies,

the diarrhœa being the prominent symptom. Dilute sulphuric acid, opium, lead and opium or copper pills, bismuth and chalk mixtures, and sometimes alkalies, are all much used (℞ 51, 52, 48). Regulation of the diet, as before mentioned, is highly important. Bismuth is best for children. Arsenic and opium—both in minute doses, and frequently repeated—is a useful combination in the more chronic states (in adults). The more serious form of typhlitis, with perityphlitis, is considered as a separate disease. In catarrh of the rectum (proctitis) the bowels should be moved freely either by the use of enemata or by a dose of Epsom salts—it being highly important to clear the colon. Afterwards, the astringent treatment is commenced, and emollient enemata (warm chamomile and opium infusions, starch or oil) or morphia suppositories, must be used. Morphia may also be given hypodermically. Leeches to the margin of the anus will relieve the very acute cases. In the chronic forms, tannin, or nitrate of silver (ten grains to the ounce) may be applied or injected *per rectum*. Enemata of hot water give great relief. The bowels should be regulated, and as the patient is usually run down, cod-liver oil and a nutritious diet are necessary.

Dysentery.—The **pathological changes** may be of a *catarrhal* or *fibrinous* character. In the former there is first an intense but superficial hyperæmia of the mucous membrane of the bowel, limited, it may be, to the rectum, sigmoid flexure, or cæcum, but often involving the whole of the large intestine. The congestion extends to the deeper submucous connective tissue, which becomes greatly thickened. There is much mucus thrown out but it is not very adherent. The follicles are enlarged and the mucous membrane softens and disintegrates, throwing off shreds and leaving rugged portions attached. It is a rapid necrosis produced by the burrowing pus cells within the intestine. Should the parts recover, cicatrices are formed. In the *fibrinous* or diphtheritic form, the initial hyperæmia is universal and deep, and there are extravasations of blood, which infiltrate the whole of the tissues. A fibrinous exudation is poured out upon the surface of the mucous membrane, which becomes dense and unyielding. Gangrene is the ultimate result, if the case proceed further. The membrane then becomes detached in patches and casts, leaving a raw-looking ulcer to heal by cicatrisation; but this can only be when the gangrene has been very limited.

The subsequent contractions of the cicatrices may lead to narrowing of the bowel, and severe ulcerations may penetrate to the peritoneum and end in perforation. The mesenteric glands are enlarged, and they may become purulent. The liver is often affected in the same way, and large abscesses may form in its substance. The two forms, catarrhal and fibrinous, may be present together in one case.

The **causes** appear to be chiefly climatic, as dysentery is a disease of hot countries. In malarial districts, and especially in the late summer and autumn, dysentery is particularly liable to attack individuals. It occurs as an epidemic, especially when there is

over-crowding and bad hygienic conditions, and it is probably connected with a specific germ.

The **symptoms** may begin with mild *tormina*, abdominal tenderness over the affected part, and diarrhœa, followed soon by feverishness and general *malaise*. In the severe epidemic form, however, the onset is usually sudden and violent. The characteristic symptom is the burning pain in the rectum, with violent straining and purging. At first the stools contain fæcal matter, but soon they are composed only of blood and mucus, and later, shreds of mucous membrane and *débris*. In a few days the blood increases in quantity and gangrenous sloughs may be passed, and sometimes entire casts of the part of the bowel affected. The fluid portions of the stools are puriform and highly offensive, and they are sometimes full of granules which have been described as like "sago grains." The fever is often high, and is of the intermittent type. There is usually nausea and vomiting. The bladder often sympathises with the bowel, and painful attempts at micturition are made without result. The urine is scanty and acid. The patient soon becomes exhausted, the features are pinched, the expression is anxious, and stupor and collapse may end the case. The severity of a case of dysentery is estimated by the straining of the bowels. In severe cases the attacks may be every few minutes, and may extend over a few days. In milder cases, the *tenesmus* may only occur ten or twenty times in the day.

The complications are phlebitis, peritonitis, and abscess of the liver—the latter being the most common. The mild cases may terminate in convalescence in about a week; and the severe cases gradually improve, and are free of characteristic symptoms in about three weeks—should the strength of the patient have enabled him to combat the disease. Sometimes a *chronic* dysentery is induced and recovery is only partial, with frequent relapses. There is then long continued ill-health, unless the patient be placed in very favourable circumstances. The case may terminate in waxy degenerations of the organs (liver, spleen, kidney, or bowel) leading to dropsy and death. The narrowing of cicatricial tissue may also lead ultimately to interference with nutrition, and death by exhaustion.

The **prognosis** is very grave in the severe forms.

The **treatment** of dysentery requires the administration of large doses of ipecacuanha—twenty to forty grains every four or six hours. The first doses are vomited, but subsequently a tolerance is established. It should be given in milk, and the diet should consist chiefly of milk, eggs, beef juice, custard, and chicken-broth. Brandy is necessary to sustain the strength.

Instead of ipecacuanha, laxative doses of Epsom salts, with dilute sulphuric acid, are believed by many authorities to be quite as efficient. In the later stages of dysentery, arsenic and copper, combined with opium, are the usual remedies. Bismuth is sometimes used. The bowels should be washed out from time to time with warm water; and enemata of starch and laudanum are very

grateful during the course of the disease. Injections of nitrate of silver (twenty grains to the ounce) are highly recommended. They may be given after a hypodermic injection of morphia. Suppositories may also be used. Poultices, or turpentine stupes applied to the abdomen, afford relief. The discharges should be disinfected with strong solution of sulphate of iron, and immediately removed from the room.

Ileus, or Obstruction of the Bowels.—The causes of obstruction may be usefully classified into three groups—viz: (1) extrinsic causes, (2) changes in the walls of the intestine, and (3) changes within the canal.

(1) The extrinsic causes are tumours and displaced organs which press upon the bowel. Ovarian and mesenteric tumours, cysts of the peritoneum and cancerous growths, floating kidney and displaced spleen, are examples of such obstructions. Constrictions by old bands of adhesion are common, especially in the neighbourhood of the appendix vermiformis; but bands often arise elsewhere, as between the mesentery, bowel, and pelvic organs. Slits in the mesentery or peritoneum, or weak parts in the diaphragm, are also possible causes of constriction of the bowel. Strangulated hernia is discussed in surgical works.

(2) The changes which occur in the intestinal walls themselves, producing obstruction, are intussusception; cicatrisation and shrinking after dysentery, typhoid fever, or syphilis; tumours, polypi, hydatid cysts, and cancer; and twisting or torsion of the bowel (volvulus). In the latter disease, the sigmoid flexure is the commonest situation in the *young;* while the cæcum is the more usual seat in adults.

Intussusception is by far the commonest cause of obstruction and it occurs chiefly in early life. It is always the upper part of the bowel which slips into the lower part, the cause being distention or paresis of the lower part, with spasm or contraction of the upper part. There are three layers of the bowel concerned with an intussusception, *viz.*, the *entering* (C), *returning* (B), and *receiving* (A) layers. The bend of the entering layer remains fixed (E), while the bend of the receiving (upper) layer is constantly folding in (D). The most common part involved is the ileum, which thus passes into the cæcum. The invaginated bowel may sometimes be felt in the rectum, and it is even sometimes projected through the anus. The circulation is impeded in cases of intussusception—the mesentery being drawn in with the bowel. The parts become hyperæmic, and serum is exuded. The peritoneum is also congested, and in the later stages peritonitis is always a consequence. The bowel may become gangrenous, and large portions may slough and be passed *per anum*.

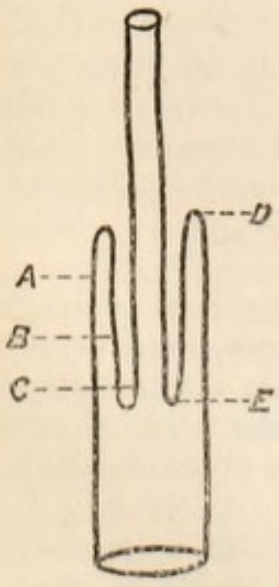


Fig. 25.
Intussusception.

A fatal peritonitis may result, when a slough has separated before there is complete union of the contiguous layers of the bowel. With these conditions, there is not necessarily *complete* obstruction of the canal.

(3) Changes within the canal causing obstruction are, the accumulation of fæcal matter—chiefly in the cæcum, ascending, or descending colon; biliary calculi and foreign bodies serving as nuclei for such accumulations; and chalk and magnesia deposits—when such have been taken in large quantities, medicinally. Habitual constipation tends to produce paresis of the bowel, and thus to favour the later development of obstructive conditions.*

The **symptoms** common to all forms of obstruction are pain, distention, and partial or complete stoppage of the action of the bowels, with vomiting. The pain may not be very severe, and is rather, at first, a tenderness over the seat of obstruction. Sometimes, however, it is very acute, while in all cases there is, in addition to the abdominal tenderness, occasional attacks of colic pain. It is usually referred to the umbilical or iliac regions. The abdomen soon becomes distended with gas—usually in twenty-four to forty-eight hours, in cases of complete obstruction. The muscles become rigid. A tympanitic note can be elicited over the whole abdomen, except at those parts where accumulation of fæces, or other solid obstruction, gives a dull note. If the abdomen be not too tense, the intestines may be felt as dilated cylinders above the obstruction. The breathing is embarrassed by the abdominal distention, but both the dyspnœa and distention are relieved, for a time, when there is much vomiting—or when the obstruction is incomplete and allows of occasional evacuations of the bowel. This last symptom is very deceptive, and is apt to raise the hope that the obstruction is overcome, and all danger is passed. In intussusception, diarrhœa may be present. The vomiting, when the obstruction is acute, is an early symptom. It first consists of the food, then of mucus and bile, and later, it approaches to the fæcal character. There is no fever during the initial stages; but the temperature rises with the continuation of the acute symptoms. The patient looks very anxious, the features become pinched and drawn, the eyes sunken, the voice husky, and then follow, usually, the signs of collapse, with stupor and death.

The physical signs of obstruction differ with the cause and the seat of the obstruction, and they must be considered in close relation to the symptoms and the history. A tumour or displaced organ may be felt, and the history of its development, or the presence of cachexia, may throw light upon the case. *Constrictions* are likely after attacks of peritonitis and perityphlitis—the symptoms then being sudden and very acute. If the symptoms be acute and attack the patient very suddenly—without a history of previous inflammation, and especially after some violent exercise—it is probably a twist in the bowel (volvulus). A slow development of the obstruc-

* In connection with obstruction of the bowels, the reader should turn to articles on Typhlitis, Perityphlitis, and Appendicitis (p. 176).

tive symptoms points to a gradual occlusion—as by tumour growths, and cicatrisation after dysentery, typhoid fever, or syphilis. Fæcal accumulations may be felt *per rectum*, or by abdominal palpation over the line of the colon and cæcum. A previous attack of gall-stones may suggest that a biliary calculus has served as a nucleus for fæcal masses. In obstruction of the upper part of the bowel the symptoms are more rapid, and the abdomen is not distended. The urine is diminished in such cases, while it is increased with *low* obstructions.

Bloody stools are strongly suggestive of *intussusception*, and the bowel, sometimes, may be felt *per rectum*. There is often violent *tenesmus*, and it should be noted that this is the only form of occlusion which is so frequently accompanied by diarrhœa. The tumour caused by intussusception is peculiar. The shape and position changes under manipulation. It feels like a tense, cylindrical air ball. The ileum may descend into the rectum in about thirty-six hours. Some cases may terminate favourably in the sloughing of the bowel; but these are rare. Some again run a chronic course and ultimately recover; but these cases are also very exceptional. Death from gangrene or perforation is a common termination. Peritonitis is common to all forms of obstruction, and in the later stages, it may result from perforations, after acute inflammation set up by foreign bodies lodging in the bowel, or by gangrene, or simply by extension of the hyperæmia to the peritoneum. At the beginning of the peritonitis there is increased pain; and if the abdomen be not too distended with gas, diminished movement and flattening may be made out. This may suggest, or confirm, the site of the primary inflammation.

The **prognosis** is always grave. Those cases arising from impaction of fæces are more favourable than any other. The *duration* of a case varies. A child may die of shock in a few hours; while other cases linger from two to three weeks.

The **treatment** of obstruction of the bowels varies with the cause. Until this be ascertained, no attempt should be made to produce evacuation by the use of purgatives or enemata. Complete rest is indicated, and morphia should be injected until the patient lies in a state of semi-somnolence from which he can just be aroused, if necessary. In cases of *volvulus* no further medical treatment is required; but later, in this and in other forms of obstruction, surgical procedures have to be considered. The bowel may be punctured with a fine needle in all cases of *extreme distention*, whatever be the cause.

In intussusception, the bowel may be distended with water, air, or gas, in an attempt to force back the intruding bowel. Sometimes a drachm of bicarbonate of soda injected into the rectum, and followed immediately by a drachm of tartaric acid, is the method used for distending the bowel. The anus must be held tight with a compress, to prevent the escape of gas. Distention with an ordinary bellows is just as effectual, and safer. Inflation should only be carried out in the early stages, whatever the means used, as in

the late stages adhesions may have formed, and the tissues are more liable to rupture. Warm applications are grateful, and an ice bag should be used over any local tenderness. The diet should be of the lightest description. Ice may be given to suck, and stimulants may become necessary to counteract the tendency to collapse.

Obstruction, due to impaction of fæces, is treated differently, according to the seat. When rectal, or in the descending colon, efforts should be made to soften the fæces by injections of warm water and soap, or castor oil. When impacted in the cæcum care must be taken not to interfere too actively or too soon (see *Typhlitis*). Saline laxatives are best—especially Epsom salts. Electricity should be tried when these remedies fail. The *surgical* treatment of obstruction is a highly important question to decide—an operation offering the only hope of relief in many cases.

Ulcers of the Intestines.—The duodenal ulcer has already been mentioned with gastric ulcer. In addition to the tubercular ulcers which are associated with phthisis, the cæcum, appendix vermiformis, and rectum, are often attacked with ulceration. The gastric and duodenal ulcers are generally the result of thrombosis and embolism, and in the case of the duodenal ulcers they are sometimes the result of burns of the skin; but in ulcers of the cæcum, appendix vermiformis, and rectum, *direct irritation*, by solid fæces or foreign bodies, is the commonest cause. Perforation and peritonitis may result from these (see *Typhlitis*). Rectal ulcers are treated locally, and they belong to the domain of surgery. The chief reason for noting these ulcers here, is to keep before the mind of the practical physician the fact that an obscure ulcer of the bowel may be a source of blood-poisoning (septicæmia).

Cancer of the Intestines.—The cæcum, flexures of the colon, and, most commonly, the rectum—are the parts liable to be affected. The *symptoms* are fixed dull pains, with frequently sharp attacks of darting paroxysmal pain. The cachexia gradually develops, emaciation follows, and the presence of a tumour completes the diagnosis. In the rectum cancer excites severe burning pain, with *tenesmus*. The stools may indicate a narrowing of the bowel, and later they contain mucus and blood. The hæmorrhage is often severe. The *prognosis* is entirely unfavourable. The average duration is about a year; but secondary complications—as perforation—may terminate a case earlier. The *treatment* can only be palliative—consisting of easily digested food, and morphia for the pain. Large doses of charcoal, by the mouth, are useful to control the intense fœtor of these cases.

Intestinal Parasites. Cestoda (Tape Worms).—*Tænia solium; T. saginata. Bothriocephalus latus.*—The *tænia solium* is derived from embryos (cysticercus cellulosus) contained in pork, while the *tænia saginata* is derived from embryos in beef. The proglottides in the former are thinner, softer, and more transparent, and the lateral branches of the uterus are fewer in number. The head or scolex of the tænia has a rostellum, a crown of hooks, and suckers. They infect the small intestine and are usually solitary. The Bothriocephalus latus is larger than the T. solium, and its segments are not detached at maturity.

The **symptoms** connected with tape worms vary much. Colicky pains are very frequent. A large appetite associated with emaciation, palpitation, itching at the nose and anus, and often nervous phenomena—as choreic movements, epileptiform attacks, impaired vision, &c.—are more or less common. Sometimes, however, there are no symptoms, and the general health of the patient seems good. Most frequently, the passage of segments, *per anum*, is the first and only indication of the presence of a tape worm.

The **treatment** consists of a preparatory course of light diet and semi-starvation for a day, with a large dose of castor-oil administered at night. In the morning the extract of male fern, or a decoction of pomegranate, is given. If the bowels do not act, a second dose of castor-oil can be given. The stools should be kept and a search made for the head. If not passed, the segments develop again within three months.

Nematoda. Ascarides lumbricoides (Round Worms).—These infest the small intestine. They may be solitary, but usually there are two or three, and sometimes they exist in large numbers. They are propagated by drinking water, the ova having strong envelopes which resist extreme temperatures both of heat and cold. The worm is cylindrical in shape, tapering at both ends, the cephalic end being slightly larger. The female (which is more commonly met with) is fifteen inches long; the male about ten inches. They are brown-yellow in colour.

The general **symptoms** are the same as in the preceding group—as colicky pains, capricious appetite, nausea, and vomiting, itching of the nose and anus, choreic and hysterical seizures, and grinding of the teeth at night, &c.

The **diagnosis** can only be conjectural, unless a worm be passed—or vomited, as sometimes (rarely) is the case. Obstruction of the bowels may arise from a bundle of worms.

The **treatment** is to prepare the patient as in the treatment of tape worms; then to give *santonin*, in doses according to the age. The santonin should be administered in castor oil; and it should be noted that its administration often affects the vision—everything appearing yellow to the patient.

Oxyurides vermiculares (Thread worms).—These are small, the female being about half an inch in length, and the male less. They are white, and resemble fragments of sewing-cotton. They infest the rectum chiefly, and sometimes the vagina, and they are always in great numbers when present at all. Children are most liable. The most characteristic symptom is the itching at the anus, and sometimes excitation of the genital organs. An offensive vaginal discharge is sometimes produced by thread worms.

The **treatment** consists of the administration of *santonin*, and the rectal (and vaginal) injection of strong infusions of quassia. The parts should be sponged with weak carbolic lotion. Iron (Syrupus Ferri Iodidi) should be prescribed to improve the general condition, which is generally low.

Peritonitis.—*Acute, chronic, and tubercular.*—In *acute* peritonitis there is first hyperæmia, with minute extravasations, and dryness of the membrane. The inflammation may be general or local. Soon, a thin adhesive exudation is poured out which agglutinates the neighbouring surfaces. This becomes firmly adhesive if the endothelium proliferate, and connective tissue is formed from the new cellular elements. If the fibrin deposit be much increased, there is a thick coating formed, and a serous fluid is poured out. The swelling and hyperæmia extend to the sub-peritoneal connective tissue and muscular layer. The quantity of fluid poured out may range from a few ounces to gallons, and in the latter case the diaphragm is pushed up and the thoracic organs displaced. The effusion may be *hæmorrhagic*. Sometimes it is purulent, and this is always the case when peritonitis is secondary—the result of a perforation. Subsequently the adhesions contract, producing deformities, and often these dangerously interfere with the functions of the abdominal organs.

In the *chronic* form of peritonitis the changes are similar, but there is often little or no fluid exudation. The intestines are matted together, and the peritoneal cavity may be subdivided into smaller cavities. *Tubercular* deposit is common, and it is often a secondary consequence of tubercular ulceration of the bowel, coincident with phthisis. Abscess formation is common in the encysted chronic peritoneal inflammations, those above the umbilicus tending to dissect upwards, and those below taking a course along the femoral vessels. Fistulæ may be established. The hepatic duct or portal vein may be compressed.

The **causes** of peritonitis are intense cold, and blows; more commonly, it arises from extension of inflammation from the stomach, intestines, bladder, pelvic organs, &c.; or it is a result of perforation. It occurs also in the course of fevers, pyæmia, albuminuria, &c.

The **symptoms** of primary acute peritonitis begin with severe and continuous pain in the abdomen, accompanied by fever. There is generally a history of a chill. When peritonitis is caused by extension of inflammation from neighbouring organs, it is usually announced by the marked increase of pain and fever. When it is the result of perforation of the stomach or bowels, the onset of pain is sudden and very violent. Fever is not an early symptom in the latter case. In all cases, peritonitis is soon associated with flatulent distention of the abdomen. The pain is so intense that the patient cannot bear to be touched, and he lies with his knees drawn up, to relieve the abdominal tension. Paresis of the bowel causes constipation. Sometimes diarrhœa is present, however—the bowel suffering from secondary irritation. The abdomen is tympanitic, especially in the lines of the colon, but there is dulness in the flanks when there is sufficient effusion of fluid. The liver dulness may disappear. The tongue becomes quickly coated with thick, white fur, and there is much thirst. *Vomiting* is a specially marked symptom in all forms of peritonitis, and very often there is hiccough. There is rapid failure of the strength. The pulse is small and wiry

—the frequency often being 120 to 140 beats in the minute. The fever is remittent in type, 103° to 104° Fahr. being common. When collapse occurs, the temperature falls and the pulse may rise to 150 or 200 beats in the minute.

In the *chronic* form of peritonitis the symptoms begin insidiously. Colic pains, indigestion, and sometimes vomiting, with loss of flesh and strength—are early indications. The abdomen gradually enlarges, by the accumulation of gas and serous fluid. In very chronic cases the abdomen is often retracted, and there is matting of the intestines, and pressure upon the iliac veins which may produce œdema of the lower limbs. Well-marked ascites is seldom made out. *Tubercular* peritonitis may arise along with tubercular disease elsewhere, or it may follow a simple chronic peritonitis.

The *course* of a severe *acute* peritonitis is rapid. When it is the result of perforation, death may take place within a few hours. When a simple primary disease, death may not take place for a week. The more favourable forms are those which arise from extension of inflammation from the neighbouring organs. Local inflammations are less serious. An acute attack may become chronic.

The **prognosis** should always be guarded; and in acute general peritonitis it is most unfavourable.

The **treatment** of acute peritonitis consists of the free use of morphia, either by the mouth or hypodermically. When the inflammation is local, leeches may be used. An ice bag is best at the very first, but hot turpentine stupes, or poultices, are soon required, and they are more grateful to the patient. If there be much fever, quinine (10 grains, and repeat in two hours), or antipyrin (fifteen grains every three or four hours)—should be given. The diet should consist of milk and beef tea chiefly. The chronic forms require local applications—as fly-blisters, mustard, and tincture of iodine. Iodide of potassium with syrup of iodide of iron, may be prescribed during convalescence (℞ 30).

Typhlitis: Perityphlitis: Inflammation of the Appendix vermiformis (Appendicitis).—*Typhlitis* may exist alone as a catarrhal affection; but it is more commonly associated with *perityphlitis* (inflammation of the neighbouring connective tissue), and both are generally the result of localised inflammation produced by hardened fæcal matter, &c. Fæcal pellets may lodge in the appendix vermiformis, and ulcerating through to the peritoneum may light up a general peritonitis. Grape seeds, orange pips, &c., lodging in the appendix vermiformis, are said to be, sometimes, causes of inflammation and ulceration. More commonly, however, the swelling of the mucous membrane at the mouth of the appendix (the result of irritation), or the blocking of the appendix by fæcal accumulations in the cæcum, causes retention of the secretions within the appendix, and a catarrhal appendicitis is induced which leads to secondary inflammation (perityphlitis) or, sometimes, to ulceration and perforation.

The **symptoms** of typhlitis may be acute or chronic. In the former, pain is felt in the right iliac region. There is tenderness upon pressure, and the patient generally lies upon his right side with the right lower limb flexed. Constipation is usual, but often there is diarrhœa. There is general *malaise*, fever, nausea, and sometimes vomiting. Palpation may reveal a prominence over the cæcum, if there be any impaction of fæces. When perityphlitis is present—as is almost invariably the case—a hard brawny swelling may readily be made out just above the *crista iliі*. It specially tends to suppuration, and this is indicated by rigors, and the usual constitutional symptoms. When the inflammation involves the appendix vermiformis, the pain and tenderness is much lower down. M'Burney states that in cases of appendicitis the tenderness is most marked at a point lying two inches from the right anterior superior iliac spine in a line drawn from that point to the umbilicus. The significance of this sign is that usually this point corresponds with the position of the base of the appendix. Appendicitis may or may not be associated with inflammation of the cæcum. There is usually a history of chronic constipation. When the appendix is affected the pain is very acute, and there is no impaction or interference with the functions of the bowel—but general peritonitis is a more common sequel. As the case advances, the face becomes pinched and the eyes sunken. The pulse is small and rapid, and the heart is weak.

In the simple and uncomplicated chronic form of typhlitis, the symptoms are those of an intestinal catarrh. Palpation may reveal tenderness and a "doughy feel" over the cæcum, with gurgling upon pressure. Flatulent movements, and pain, are frequently referred by the patient to the cæcal region, and the pain is aggravated by constipation. The mild forms of typhlitis are readily cured in about a week. The severe forms associated with perityphlitis take longer; and the *prognosis* as to recovery must be guarded. The great tendency to suppuration should be noted, and also the possibility of general peritonitis. The presence of foreign bodies in the appendix vermiformis, with ulceration and perforation, is a very fatal condition; but cases of appendicitis sometimes recover, although recurrence is the rule. The chronic forms of typhlitis may recover; but if there be much thickening around, or any stenosis of the ileo-cæcal valve, the prognosis is unfavourable as regards a cure.

The **treatment** of typhlitis, &c. All active purgatives must be avoided, but if the fever is not great and the tenderness is slight, mild and repeated doses of sulphate of magnesia is the best treatment. Opium should also be prescribed. When there is obstruction of the bowel by impaction of fæces, along with high fever, and tenderness over the cæcum—the case requires careful handling. The morphia should then be pushed, as in other forms of obstruction—no purgative being given. The morphia may be given with minute doses of atropia (about $\frac{1}{240}$ gr.), and given until the patient is well under its influence. This treatment is to be

continued until the bowels move naturally, or until the tenderness becomes less marked, when the Epsom salts may then be cautiously administered. Milk and lime water, ice, and champagne are useful when there is vomiting. Hydrocyanic acid may also be prescribed. Turpentine stupes or poultices should be applied to the abdomen, and leeches are useful, if applied early. Enemata of soap-suds should be injected when the time arrives for the removal of the impaction without risk. The treatment of peri-typhlitis is the same as typhlitis, and the aspirator is used when pus forms. Further surgical procedures may be necessary with impacted fæces and appendicitis, &c.; and, indeed, the whole question is of importance in relation to *early* surgical interference, as the risk of removal of the appendix is not very great, while perforation is always fatal.

Tabes Mesenterica.—This is almost exclusively a disease of childhood, and it consists of the chronic strumous enlargement and suppuration or calcification of the glands of the mesentery. The abdomen gradually enlarges, and it is tender to pressure and has a diffuse "doughy" feeling. Colicky pains are frequent, and they are accompanied generally by attacks of diarrhœa—the stools being often very offensive. The appetite is fitful and often it is very voracious; while notwithstanding the food consumed there is great emaciation. The skin hangs in loose folds, and the face of the child is very pinched and small. Recovery sometimes takes place under treatment; but death by exhaustion or complications is the general rule.

The **treatment** is considered with the general treatment of strumous diseases.

The differential diagnosis of the foregoing diseases of the intestines, peritoneum, &c.—*Enteralgia or colic* is distinguished from other pains by its seat, and termination; and by the absence of fever and of tenderness upon pressure. If due to malarial causes the attack is periodic; when syphilis is the supposed cause the attacks occur at night—like the syphilitic headaches. *Lead poisoning* and its symptoms must be noted. *Hepatic* and *nephritic colic*, *appendicitis*, and *gastralgia*, are distinguished by the seat of the pain and the presence of other symptoms associated with these affections. *Flatulence*, and *fæcal accumulations* are the commonest causes of colic. *Perforation* gives rise to sudden and most acute pain; but the previous history as regards the likelihood of there having been an ulcer, may suggest the diagnosis, and the future course will confirm it. *Peritonitis* is associated with fever and great tenderness on pressure. *Hysterical* tenderness of the abdomen is accompanied by the other manifestations of hysteria, and pressure upon the abdomen while the patient's attention is occupied with some other subject, does not excite pain. *Rheumatism in the abdominal muscles*, when present, is probably associated with rheumatic pains elsewhere, and there is not much tenderness on pressure.

Constipation is either a symptomatic affection, or a simple functional disorder. All patients suffering from constipation should be thoroughly examined, as the diagnosis of a *simple* case can only be made by exclusion. Mental states, lead poisoning, and spinal disease; dyspepsia, and hepatic congestion with catarrh, liver disease, &c., are all associated with constipation of the bowels. Concerning the opposite condition of *diarrhœa*, the same may be said, and the reader is referred to the causes of this affection (see *ante*). The various forms of intestinal catarrh, with diarrhœa, may not be distinguishable. A *gastric catarrh* frequently co-exists with a *duodenitis;* but the pain in the right hypochondrium, and the presence of more or less jaundice, indicates that the duodenum is affected. Catarrhal jaundice would probably be the more correct diagnosis. *Ileitis*, *colitis*, and *typhlitis* (when not associated with perityphlitis, as is usually the case) are sometimes distinguishable in a case of diarrhœa, by the tenderness and distention, or by the fæcal accumulations in the ileum, colon, or cæcum. Catarrh of the rectum is known by the tenesmus, &c.; but uterine displacements, rectal polypi, and hæmorrhoids must be excluded. *Dysentery* and membraneous *enteritis* must also be remembered. Tape-worm segments sometimes resemble the casts of a membraneous enteritis. To differentiate cancer of the intestines from catarrh or ulcerations, will be impossible if no tumour can be felt, and no cachexia be present. With regard to a tumour, in this relation, fæcal accumulations, aneurism, floating kidney, cancer of the cæcum, peritoneal growths, cysts, ovarian tumours, and perityphlitis, &c., have all to be noted. The age, and the presence of cachexia are important as regards the diagnosis of malignant growths. *Fæcal accumulations* will move, or a purgative may clear up the case. *Aneurisms* are pulsating and expansile. The patient should be turned over on his face to examine such a tumour, as the aorta may communicate a pulsation to the hand through the tumour. *Floating kidneys* are freely movable. The seat, history, mobility, mode of development, &c., are considerations of high importance in the diagnosis of abdominal tumours.

The actual cause of *obstruction of the bowels* is seldom easy to determine, and in most cases it can only be inferred. Diarrhœa previous to obstruction—especially in a child—followed by bloody stools and tenesmus, suggests intussusception. Sudden violent pain with obstruction, following active exercise, suggests a twist in the bowel; but if there be a history of previous peritonitis, it is probably caused by tight strangulation within an old adhesion. Cases occurring in women who have borne many children, and in patients suffering from external herniæ, are probably due to old peritonitic adhesions. A floating kidney or tumour may compress the bowel; and palpation and inspection may reveal an impaction of fæces. The latter may be felt per rectum, when low enough. As biliary calculi sometimes serve as nuclei for fæcal accumulations, a history of previous hepatic colic would suggest this form of obstruction.

Inspection and palpation of the abdomen may reveal important

signs. When the obstruction is high up—in the duodenum or jejunum—the vomiting and hiccough is marked, but the vomiting is not fæcal. The urine in such cases is much diminished; when the obstruction is low down, it is increased. The abdomen is not distended when the obstruction is high up. If seen sufficiently early—before the distention becomes general—the examination of the abdomen may reveal that the whole of the colon is empty, and that the swelling is mostly round the umbilicus (obstruction at the ileo-cæcal valve); or that the colon is distended (rectal obstruction, or accumulation at the sigmoid flexure). Below the obstruction the colon (or rectum) is empty. Typhlitis and perityphlitis may arise from the irritation of some foreign body lodged in the cæcum, and it is frequently associated with impaction of fæces. Primary appendicitis does not produce obstruction; but the secondary perityphlitis may interfere with the action of the bowel, The subjects of appendicitis have usually a history of antecedent constipation.

Intestinal hæmorrhage is a symptom of many conditions. When occurring high up, the stools are tar-like, and the blood is mixed with the fæces; when from the descending colon or rectum, the blood appears unchanged. The causes are rupture of a blood-vessel in ulcerations of the bowel from the duodenum downwards—or even from a gastric ulcer when the blood has not been vomited; obstructive diseases of the heart, lungs, and liver—the latter causing increase of the blood pressure within the portal system; rectal ulcers; cancer; hæmorrhoids; typhoid fever; purpura, scorbutus, and other blood diseases.

When the rupture is sudden, and the blood effused is great in quantity, there is sudden paleness, noises in the ears, faintness, and loss of consciousness. The pulse is weak and rapid, and it may become imperceptible at the wrist. In typhoid, the temperature falls.

Ascites is also a symptom of many diseases. The beginning is usually obscure, and it is often only detected when a causal affection suggests an examination of the abdomen. There is increased fulness of the abdomen, and the fluid extends the iliac and hypogastric regions when the patient is in the erect, and the flanks when in the recumbent position. In extreme cases, the umbilicus is forced outwards. Tapping the abdomen with the hand excites a characteristic wave motion; and percussion reveals a tympanitic note over the distended bowel, and dulness at the flanks. Fluctuation is marked when the fluid is in sufficient quantity. The breathing may be much embarrassed when the abdomen is full. The urine is diminished in quantity. The lower extremities and scrotum become much swollen. The differential diagnosis of ascites from ovarian tumours and pregnancy is discussed in gynæcological works; but chronic peritonitis, a distended bladder, enlarged spleen, tumours, and cysts are also possible sources of error. In ascites there is no tenderness, and the abdominal walls become thin, while the contrary is the case in peritonitis. The other conditions require careful palpation and percussion—with attention to the history and mode of development—to distinguish them.

CHAPTER IX.

DISEASES OF THE DIGESTIVE SYSTEM.—Section III.

Contents.—Jaundice—Catarrhal jaundice—Biliary calculi (gall-stones)—Cirrhosis of the liver—Cancer of the liver—Hydatids—Inflammation of the liver and abscess—Acute yellow atrophy—Congestion of the liver, cyanotic atrophy, pigment liver, fatty liver, syphilitic diseases of the liver, waxy disease, and perihepatitis—Thrombosis of the portal vein—Suppurative pylephlebitis—Diseases of the pancreas—*Differential diagnosis of the more important diseases of the liver—Murchison's table of the causes of jaundice.*

Jaundice, or **Icterus,** is a symptom common to many diseases of the liver, &c. (See *Diagnosis*, p. 189.)

Catarrhal Jaundice.—*Catarrh of the Bile Ducts, &c.*—The common duct is the part most affected, the initial change being hyperæmia of the mucous membrane. The duodenal end of the duct suffers most, and the mucous membrane soon becomes swollen and covered with tenacious mucus. The endothelium proliferates, and the *débris*, with the mucus and swelled membrane, all serve to occlude the duct and to obstruct the flow of the bile. These changes generally end in subsequent liquefaction, and recovery within a few weeks; but continued or repeated attacks may lead to fibroid degeneration of the liver and atrophy of the cells.

Catarrhal jaundice may arise from malarial influences, but more frequently it is produced by the extension of catarrh from the duodenum. The latter arises in connection with gastric catarrh, and over-eating and drinking; and the slighter forms of it are associated with the hepatic dyspepsia already described.

The **symptoms,** in the malarial form, may begin suddenly with pain or discomfort in the right hypochondriac region. There is usually constipation. The tongue becomes coated, the appetite is lost, and there is fever and general *malaise*. In two or three days the conjunctivæ become yellow, and soon afterwards the whole skin becomes tinged. The pulse is slow, and the heart weak.

The most common form, however, is the jaundice which follows gastro-duodenal catarrh. For some time previously the patient complains of the state of his stomach. A furred tongue, bad taste in the mouth, flatulence, nausea, and vomiting, with depression of spirits—the latter being very marked in cases of jaundice—is the usual history and course of a case. The jaundice gradually appears. The urine is high coloured, and exhibits a re-action to nitric acid when a few drops are placed upon a white porcelain slab and allowed to come in contact with the acid. A play of colours—green, blue, violet, red—is then observed. The stools are clay-coloured and offensive. As digestion is imperfect—especially of the fatty constituents of the food—there is much flatulence. The skin is often itching; and the

vision may be affected so that objects appear yellow. The liver increases in size, and it may be tender. The case may run a course of six weeks, and then recover; but chronic cases sometimes lead to organic changes in the liver, as already mentioned.

The **treatment** consists of limiting the patient in his diet to skim-milk and beef-juice for a time. Fresh meat and fish may be allowed later. Minute doses of calomel ($\frac{1}{12}$ to $\frac{1}{6}$ of a grain) thrice daily, are useful to allay irritation of the mucous membrane. Quinine, in anti-pyretic doses (ten grains), may be used in the early stages of the malarial forms. Effervescing saline aperients, occasionally, or sixty grains of phosphate of soda, thrice daily, well diluted—should be ordered. Euonymin (two grains) or iridin (four grains) may be given at night, followed by the aperient in the morning. Sulphate of magnesia is a useful saline to use in gouty patients. Bitartrate of potassium (as a lemonade) is a useful diuretic in the later stages. Sometimes a dose of jalap powder acts well in relieving obstruction of the ducts.

Biliary Calculi.—*Gall-stones* (*Cholelithiasis*) —Gall-stones are composed of mucus, bile pigments, carbonate of lime, and phosphates, with cholesterine—the latter being the chief substance. Mucus generally forms the nucleus around which the other materials aggregate in concentric layers. They vary from a small pea to a large size, so as, occasionally, almost to fill the gall-bladder. They are yellow-brown or dark-coloured, very light in weight, and they may be found in any part of the biliary passages, sometimes in great numbers. Ulcerations of the mucous membrane, hypertrophy of the walls of the gall-bladder, and peritoneal inflammation are common results of the irritation by gall-stones. Sometimes they become impacted in the cystic, hepatic, or common duct, and the ulceration which ensues may end in perforation.

The **cause** of the formation of gall-stones is some interference with the normal composition of the bile by which cholesterine is precipitated. The mucus which is poured out in catarrh of the biliary passages, is believed to re-act upon the glycocholate of soda, which keeps the cholesterine soluble in the bile. Gall-stones are more common after middle life, and in the obese, and they occur most frequently in those who lead sedentary lives. Females seem to be more liable to this affection than males.

The **symptoms** vary somewhat with the position of the calculi. They may be present in large numbers in the gall-bladder without symptoms; but generally there is slight and intermittent pain, in the right hypochondrium, in such cases. Nausea and feverish chills may also be present. Small stones in the ducts may only irritate them without producing the urgent symptoms of occlusion. When there is impaction, the pain is intense and commanding—so much so, as sometimes to culminate in epileptiform convulsions. The pain is described as burning or boring, and it radiates to the shoulders and back. It is most intense in the region of the gall-bladder and it shoots towards the umbilicus. The vomiting, which

is invariably present, does not relieve the symptoms. When the hepatic duct is occluded there is jaundice and enlargement of the liver. When the stone passes from the gall-bladder into the cystic duct the pain is intense, and it is only relieved when it either falls back again, or passes into the common duct, when a fresh paroxysm soon takes place, especially when the duodenal orifice is reached. Jaundice is not usual in impaction of the cystic duct, but it is present in occlusion of the hepatic or common duct. The pain suddenly ceases when the calculus passes into the duodenum. Peritonitis may be excited around the seat of impaction. In chronic cases of impaction, the liver undergoes degenerative changes (fibroid conditions, with atrophy of the cells).

The fæces should be diluted with water, and a careful search made for the calculi. After the passage of the stones, the health, as a rule, is restored, and the tenderness over the right hypochondrium, and the jaundice, gradually disappear. An acute attack may last two to five days, but if the stones be numerous the period is indefinite. Peritonitis and perforation are possibilities; and sometimes a cure results from discharge of the calculus externally, or into the stomach or intestine—a fistulous communication being established. A calculus sometimes forms a nucleus in the bowel for aggregations which may produce obstruction of the bowel (ileus).

The **prognosis** is generally favourable; but the possibility of a grave termination must always be remembered.

The **treatment** of hepatic colic, consists of the use of morphia hypodermically—as much as half a grain being sometimes necessary. Smaller doses may suffice. In extreme cases, chloroform inhalations must be given to tide the patient over the agonising pain, until the morphia take effect. Hot fomentations or turpentine stupes should be applied to the hepatic region; or a hot bath may do much to relieve. Prophylactic treatment should be advised. This consists of the avoidance of all starchy, saccharine, and fatty foods—lean meat, eggs, fish, fruit, and succulent vegetables being allowed. Wine may be allowed, but malt liquors and spirits are forbidden. Regular exercise should be inculcated. For medicine, nothing equals the phosphate of soda, in drachm doses, thrice daily, well diluted. The phosphate may be continued for several months.

Cirrhosis of the Liver.—The organ is at first hyperæmic and increased in size. It is brown-red in colour, denser, and has a granulated appearance. These changes are due to the increased formation of connective tissue. This subsequently contracts, and the liver becomes harder and assumes the "hob-nailed" appearance—the surface being unequal, nodulated, and traversed by bands of connective tissue. These compress the cells, which undergo fatty and amyloid degeneration. The blood-vessels of the liver are also compressed, and hence there is stasis of the blood in the whole portal system, leading ultimately to ascites. The bile-forming and glycogenic functions of the liver are interfered with. The peritoneum is opaque and much thickened locally, and often there are

adhesions to the neighbouring organs. The liver is frequently bile-stained and hence the name, (κιρρος = yellow).

The chief **cause** of cirrhosis of the liver is the abuse of alcohol. Syphilis is also a probable cause; and chronic impaction of gall-stones may lead to fibroid degeneration.

The **symptoms** in the early stages are generally referred to other organs—gastric and gastro-intestinal disorders being the fore-runners of cirrhosis of the liver. Slight pain and tenderness over the liver, with perhaps some appreciable enlargement, may be present. The skin may have an "icterode hue," but rarely is there actual jaundice. The appetite is poor, vomiting and pyrosis are common, while flatulence and attacks of diarrhœa are frequent. These symptoms, however, may all be due, or they may be referred to alcoholic excesses or catarrh, and ascites often is the first symptom pointing definitely to liver disease. The area of liver dulness is diminished; but sometimes when the development of connective tissue is great, it remains enlarged. As a result of obstruction of the portal circulation, the spleen may be enlarged, and the superficial veins of the abdomen stand out clearly. This latter symptom is due to the attempt of a collateral circulation to compensate for the internal venous obstruction. As a result of the stasis, hæmorrhages are common — from the stomach (hæmatemesis) and intestines. Ulcerations of the stomach are frequent. The stools are often black and offensive. Hæmorrhoids are invariably present, and bleeding from them relieves the portal system. Hæmorrhages are also frequent into internal organs, and from the lungs, and particularly from the nose (Epistaxis). The digestion of fat, owing to the diminished secretion of bile, is very imperfect, and hence the fœtid eructations and offensive stools. There is much emaciation, the face being pinched and looking out of all relation to the size of the abdomen. The nose is very frequently red—the small blood-vessels being very prominent. The urine is diminished in quantity, and deficient in urea; but urates are abundantly deposited. Œdema of the ankles and genital organs occurs with the development of the ascites.

The *course* of cirrhotic liver is a very chronic one. The early symptoms often escape detection. The fibroid changes may affect other organs, as the heart, lung, and especially the kidneys. Brain symptoms, as delirium and coma, may supervene; or intercurrent maladies—pleurisy, pericarditis, or pneumonia—may terminate a case. Gradual failure by exhaustion from hæmorrhages, is another termination.

The **prognosis** is always unfavourable, except when detected early, and the patient is amenable to proper treatment.

The **treatment** can only be palliative. Bartholow recommends the use of sodium phosphate. Chloride of sodium, chloride of gold, perchloride of mercury, arsenic, and phosphorus are drugs which are sometimes prescribed with apparent benefit. Purified ox's bile, in pills, may sometimes be of value (℞ 53). If there be a syphilitic taint, iodide of potassium, with perchloride of mercury, should be

given. The diet should consist largely of milk. If the ascites be great, the abdomen may require to be tapped.

Cancer of the Liver.—The liver may be affected by cancerous nodules, or by one large isolated mass. When the nodules are scattered and small, their size varies from a pea upwards; when few in number, the size is that of a pigeon's egg. Fatty changes take place in the centre, and in the larger nodules there is a central umbilication which can often be appreciated, on palpation, through the abdominal walls. The nodules may be soft (medullary cancer), or hard and cartilaginous (encephaloid). The liver is much enlarged and hyperæmic, and if the cancer be sufficiently advanced, the portal veins, lymph spaces, and bile ducts, may be compressed. Hæmorrhages sometimes occur into the peritoneum when the nodules reach the periphery. There is often local peritonitis. The majority of cases of cancer of the liver are *secondary* to cancer elsewhere—chiefly from the stomach.

Heredity is the chief factor known in the causation; and it occurs between the ages of forty and sixty, as a rule.

The **symptoms** are often not characteristic, and there is only uneasiness in the right hypochondrium, indigestion, and gradual development of the cachexia, with emaciation. If the stomach be the primary seat of the disease, the more prominent symptoms will be referred to that organ. When the liver is affected, pain is usually present, and on palpating the hepatic regions, there is tenderness, and the nodules—with sometimes their umbilication—may be detected. The organ is enlarged, irregular, and indurated, and the dulness may extend to near the umbilicus, in a case well advanced. Jaundice is not usually present unless the hepatic or common duct be compressed. Ascites is present in about half the cases; and often it is the result of secondary peritonitis, and not always caused by compression of the portal vein, nor by portal thrombosis. Gastro-intestinal catarrh, with diarrhœa, piles, and intestinal hæmorrhages, are common. The skin is dry and harsh, and the complexion "fawn," or earthy, in colour. The emaciation is rapid, and the debility great. The progress of a case is not uniform. Death may take place in a few weeks, and sometimes a case extends to a year or longer.

The **treatment** can only be palliative, consisting chiefly of morphia for the pain. Tapping may be necessary in ascites with extreme abdominal distention.

Hydatid Disease of the Liver.—Echinococci frequently affect the liver, and chiefly the right lobe. When they reach the liver, they become embedded and surrounded by a firm, tough capsule, composed of the neighbouring connective tissue. The membrane of the embryo is a sac composed of concentric layers, and containing numerous daughter vesicles, varying in size from a pea to a large egg. The fluid of the sac is milky in appearance, and is slightly alkaline. The scolices, or immature tæniæ, spring from the inner

lining membrane of the daughter vesicles, and they are possessed of a head, four suckers, and a row of hooklets. Usually there is but one cyst present in the liver; but there may be two or three. The hepatic tissue becomes atrophied by the pressure.

The embryos reach the liver from the intestines, by the portal vein or bile duct. The ova are discharged with the excrement of the dog, and the drinking water or vegetables consumed by man may be contaminated, should no precautions be taken to guard against such a possibility. Hydatid disease is common in Ireland and Australia.

The **symptoms** manifest themselves only when the cystic tumour becomes large enough to disturb the functions of the liver by pressure. It often, however, simply causes an enlargement of the liver, and sometimes it forms a projection from that organ. A feeling of weight, uneasiness, or slight dragging pain in the right hypochondrium, accompanied by disorders of digestion, may be complained of. If the portal vein or bile ducts be compressed, ascites or jaundice will be present. The diaphragm and heart may be displaced, causing dyspnœa and exciting cough. If the tumour be large, the intercostal spaces and the right side may bulge considerably. The tumour may often be felt as globular and elastic, with fluctuation; and a feeling of tremor ("hydatid purring") may be communicated to the fingers. Percussion reveals the liver or tumour enlargement. If the vena cava be compressed, the feet and ankles become œdematous. Should the cyst burst into the stomach, there is pain, and vomiting of the parasites; if the intestine be perforated, they are passed in the stools. They may also burst into the pleura, and cause an acute pleurisy; and they may perforate into the pericardium or peritoneum with fatal results. A cure sometimes takes place when they ulcerate into a bronchus, and are expectorated. Another termination is by *Calcification*.

The usual duration is one or two years—sometimes even longer. They sometimes cure spontaneously; or if they ulcerate into the bile duct they are destroyed by the action of the bile. Death may occur in one or other of the methods mentioned above; or from gradual failure of the strength, if they are not diagnosed and relieved by aspiration.

The **prognosis** is always grave when they are large, or when aspiration is impossible. They often end favourably when discharged by the stomach, bowel, or bronchi.

The **treatment** is to aspirate the cyst—the result being almost invariably a cure, unless a number of cysts be present. The milky fluid should be examined by the microscope for the hydatid hooklets. Electrolysis is also a successful method of treating hydatids. The injection of bile or iodine is often recommended.

Inflammation of the Liver and Abscess.—**Pathology.**—There is first hyperæmia of the hepatic cells of the part affected, and the cells become cloudy and granular from albuminous matter deposited within them. The patch has a red-yellow appearance, and

it becomes more yellow in the centre after suppuration. The patches ultimately coalesce, and form an abscess which varies in size from a pea to a hen's egg, or larger. The walls are at first rough, but ultimately they become smooth, and lined by connective tissue. Sometimes they absorb *entirely;* but oftener they remain encysted. The abscess tends to perforate the abdominal wall, or to burrow along the spine to the inguinal region; but sometimes it perforates the stomach or bowel, and sometimes even the diaphragm into a bronchus, when it becomes expectorated.

The **causes** of abscess of the liver are blows—especially in hot climates—and miasmatic influences; and particularly is abscess of the liver associated with dysentery and ulceration of the bowels. Stimulants and condiments in excess, and impaction of calculi, are also causes. The form occurring with dysentery and ulceration of the intestine is believed to be produced by infective emboli lodging in the hepatic veinules.

The **symptoms** are ushered in by a feeling of chilliness, headache, and fever. In some cases, even of large abscesses, the symptoms are very obscure, or there may be none, and it is often difficult or impossible to recognise their development during the course of a dysentery which masks the symptoms. In a typical case, pain in the back and right shoulder, a coated tongue and bilious vomiting, with increased frequency of the pulse—soon follow. Pain and tenderness in the hepatic region may be made out on palpation.

The liver may be enlarged, and in large abscesses it may extend as high as the fourth rib; and downwards, for a few inches below the level of the false rib. Jaundice is not invariably present, unless the cause be impacted calculi, when this symptom occurs early. The rigors are usually severe, with pus-formation, especially in the pyæmic forms; and vomiting also becomes more constant. If the abscess become encysted, the liver gradually diminishes in size. Fluctuation can rarely be made out, and only when the abscess is large, or superficial. The pain and tenderness diminish, and the urgent symptoms disappear with the absorption or discharge of the pus; but if the abscess continue to enlarge, there is increased pain with movement and coughing, &c., and the breathing becomes embarrassed. The heart may be displaced. A right pleurisy and pneumonia frequently co-exists; and ultimately adhesions form, and the abscess burrows through to a bronchus and the pus is discharged. Death may take place by discharge of pus into the pericardium or peritoneum, &c.

An abscess of the liver may form in about a week; and the case may run on for three months and then terminate favourably by absorption or discharge of the pus. Convalescence is very slow, when fistulous openings are established. The cases associated with dysentery are very fatal.

The **prognosis** must always be guarded. Pyæmic forms are always fatal. Discharge through the lungs is the most favourable result next to natural absorption and cicatrisation. The effects of treatment and early aspiration must also be observed, in giving an opinion.

The **treatment** consists of quinine in large doses (twenty grains) in the early stages of the disease. Morphia may be given with it when there is much pain. Warm fomentations and turpentine stupes should be applied to the hepatic region. As soon as pus can be made out the aspirator should be used; and even in those cases where no pus has been reached, the puncture never does harm, and sometimes seems to relieve the patient. The diet should be generous, and alcoholic stimulants should be given freely. The treatment of the dysentery (when present as the causal affection) is mentioned elsewhere.

Acute Yellow Atrophy of the Liver is an acute diffuse parenchymatous inflammation produced by causes as yet unknown. It occurs most commonly in women during the state of pregnancy. The organ is very much diminished in size. It is soft and tears easily, and it is of a uniform yellow colour. Microscopically, the cells are seen to be replaced by fatty granular matter, fat globules, pigment, bacteria, and acicular crystals of leucin and tyrosin. The kidneys are also yellow, and the endothelial cells are infiltrated with fatty granular matter. Ecchymoses are found in the peritoneum and elsewhere.

The **symptoms** begin insidiously as a simple gastric catarrh, with vomiting and slight jaundice. An extreme state of wakefulness, with intense headache, is a common condition. The jaundice gradually deepens, and the liver percussion is much diminished. The fever is of the remittent type. Convulsions and coma usually terminate the case. The urine is normal in quantity and in specific gravity, and acid in re-action. The urea is much diminished, and leucin and tyrosin are present in quantity. Death takes place in about a week. There is no rational treatment known. Phosphorus may be tried. Purgatives and mineral acids may be given.

The following affections of the liver need only a brief mention, as either they are already considered with diseases in which they arise as secondary conditions, or they are of minor importance from a clinical point of view:—

Congestion of the liver may be *active*, and due to excesses in eating or drinking; or it may arise from malarial causes. The liver is enlarged and tender, and the other symptoms are the same as already described under the *hepatic* form of gastric catarrh. The *passive* form (nutmeg liver) is that which is due to mechanical obstruction of the circulation—as in valvular disease of the heart. Congestion, with jaundice, may arise, also, from violent emotions. Chronic congestion leads to *chronic* (*cyanotic*) *atrophy*, with degeneration of the cells and increase of the connective tissue. This condition resembles *cirrhosis* in its pathology, and, like that disease, ultimately leads to ascites, &c.

Pigment liver is a malarial disease, usually running a chronic course. There is anæmia, and pigment granules from the spleen are found in the blood. Ascites and hæmorrhages, and ultimately brain disturb-

ance and death, is the usual course. *Fatty liver* occurs in tubercular diseases, and as a result of chronic alcoholism. The organ is enlarged, and the edges are rounded. The long continued use of cod-liver oil in phthisical conditions accounts for many of the cases. *Waxy disease of the liver* arises also in tubercular affections; but also as a result of syphilis and chronic wasting diseases and suppurations. The liver is usually enlarged, and the sharp edge is characteristic. *Syphilis* may also cause *perihepatitis*, *interstitial hepatitis*, and *gummatous hepatitis*. The liver in very chronic cases is shrunken and nodular. Cicatrices are common. *Perihepatitis*, or chronic inflammation of the liver capsule, is not always due to syphilis; but it may arise with any inflammatory condition of the liver, and from unknown causes. The liver loses its sharp edge and becomes rounded, by the contraction of the capsule. There is pain over the hepatic area, and if there be much contraction, ascites follows. The kidneys are usually diseased (cirrhotic).

Thrombosis of the portal vein is caused by cirrhosis, chronic atrophy, cancer, and tumours. The *symptoms* are apt to come on suddenly during the course of these chronic diseases. Sudden ascites, diarrhœa, scanty urine, vomiting sometimes, and gradual enlargement of the superficial abdominal veins,—are the important indications, if death do not take place, for a time. No treatment.

Suppurative pylephlebitis, or suppurative inflammation of the portal vein, is always a secondary disease. Ulcerations, suppurations, and abscesses in the immediate neighbourhood of the portal vein, may spread to it. There is abdominal pain, rigors, and sweats. The attacks are paroxysmal, and the temperature may rise to 105° Fahr. The liver and spleen enlarge, and there is usually jaundice. The abdomen is tender and it soon enlarges. Vomiting and diarrhœa are present; and soon delirium and coma terminate the case. There is no treatment possible. Morphia may be injected for the pain. The average duration is about two to three weeks.

The diseases affecting the pancreas are chiefly of pathological interest, and the treatment of them can only be symptomatic. They are *inflammation*, *cysts*, *calculi*, and *cancer*. The latter is the commonest. In all there is radiating pain in the epigastrium and back. In cancer, the pain is worse in the erect posture. The tumour is not readily felt. Fatty stools are suggestive. Jaundice commonly follows.

The differential diagnosis of the more important diseases of the liver may now be considered shortly.

Jaundice is a symptom common to liver disease, and to affections of the bile ducts. The most common causes are catarrh of the bile ducts and duodenum; gall-stones; congestion of the liver; and sometimes cirrhosis and cancer. In the two latter diseases, the jaundice is slight, and it is only slowly developed along with the symptoms of liver disease. The following table is useful:—

MURCHISON'S TABLE OF THE CAUSES OF JAUNDICE.

A. Jaundice from Mechanical Obstructions of the Bile Duct.

I. *Obstruction of foreign bodies within the duct.*
   1. Gall-stones and inspissated bile.
   2. Hydatids and Distomata.
   3. Foreign bodies from the intestines. } *rarer.*

II. Obstructions by inflammatory tumefaction of the duodenum, or of the lining membrane of the duct, with exudation into its interior.

III. *Obstruction by stricture or obliteration of the duct.*
   1. Congenital deficiency or obstruction of the duct.
   2. Stricture from perihepatitis.
   3. Closure of orifice of the duct in consequence of an ulcer of the duodenum.
   4. Stricture from cicatrisation of ulcer in bile ducts.
   5. Spasmodic stricture (?).

IV. Obstructions by tumours closing the orifice of the duct, or growing in its interior.

V. *Obstructions by pressing on the duct from without*, by
   1. Tumours projecting from the liver itself.
   2. Enlarged glands in the fissure of the liver.
   3. Tumours of the stomach, pancreas, kidney, ovary, or uterus; and post-peritoneal or omental tumours.
   4. An abdominal aneurism.
   5. Accumulations of fæces in the bowel.
   6. A pregnant uterus.

B. Jaundice Independent of Mechanical Obstruction of the Bile Duct.

I. *Poisons of the blood interfering with the normal metamorphosis of bile.*
   1. The poisons of the various specific fevers:—Yellow, remittent and intermittent fevers; relapsing, typhus, enteric, and scarlet fevers; and "epidemic jaundice."
   2. Animal poisons:—Pyæmia, and snake poisons.
   3. Mineral poisons:—Phosphorus, mercury, copper, antimony, &c.
   4. Chloroform and ether.
   5. Acute atrophy of the liver (?).
   6. Cirrhosis, and other forms of chronic atrophy of the liver.

II. *Impaired or deranged innervation interfering with the normal metamorphosis of bile.*
   1. Severe mental emotions, fright, anxiety, &c.
   2. Concussion of the brain.

III. Deficient oxygenation of the blood interfering with the normal metamorphosis of bile.

IV. *Excessive secretion of bile, more of which is absorbed than can undergo the normal metamorphosis.*
   Congestion of the liver—(1) Mechanical, (2) active, (3) passive.

V. Undue absorption of bile into the blood from habitual or protracted constipation.

Hepatic colic has already been referred to, and *enteralgia*, *hepatalgia*, and *gastralgia*, *nephritic colic*, *lead poisoning*, &c., have been considered under the diagnosis of enteralgia (p. 178).

Cirrhosis, cancer, hydatids, abscess, acute yellow atrophy, and congestion, are diseases of the liver—the more important, or more striking diagnostic symptoms of which may usefully be contrasted. *Cirrhosis of the liver.*—There is a history of alcoholism, and frequent attacks of gastric catarrh, with much flatulence. The jaundice is very slight and is slowly developed. The liver is almost always diminished in size, and no globular tumour or nodules can be felt. The early symptoms are generally attributed to congestion, and later, the development of ascites—with the other symptoms associated with cirrhosis—confirms the diagnosis. The spleen is enlarged, and the urine is deficient in urea, but may contain leucin and tyrosin. In *cancer of the liver* the age, cachexia and emaciation, with enlargement of the liver and development of the cancerous nodules, are the most important differential points. *Hydatid disease of the liver* produces enlargement, and sometimes a globular tumour can be felt. The development is slow, without fever, and it is generally painless. Palpation of the tumour reveals the characteristic, elastic fluid feeling, and possibly "thrill." It is quite different from the hard nodules of cancer. It is more like an abscess; but in the latter case there will have been great fever and rigors. An enlargement of the gall-bladder may simulate a hydatid closely; but the seat, and the history, should suggest the diagnosis. An aneurism of the hepatic artery gives rise to a heaving expansile pulsation. A hydatid cyst pushing up the diaphragm simulates, very closely, a pleuritic effusion. In such cases the use of the aspirator can at once clear up all doubt.

Inflammation and abscess of the liver is distinguished by the history of rigors, tenderness on pressure, and the presence of some causal affection—as dysentery or malaria. It may be confounded with *hydatid disease*, *enlargement of the gall-bladder*, *cancer*, *abscess of the abdominal wall*, *and purulent pleuritic effusions*. The first three have been already considered. An abscess of the abdominal wall does not disturb the liver functions. In empyema the history at the commencement of the disease is important. The use of the aspirator will clear up many doubtful cases. *In acute yellow atrophy* of the liver, the extreme wakefulness and headache, the sudden rise of the temperature, jaundice, and diminution of the size of the liver—are the striking features of the case. It occurs most frequently in pregnant women.

Congestion of the liver may be mistaken for catarrh of the bile ducts; but in the former disease the jaundice follows the liver symptoms, while in the latter it is usually preceded by the symptoms of gastro-duodenal catarrh. The passive forms of congestion due to heart and lung disease, have also to be noted.

CHAPTER X.

DISEASES OF THE NERVOUS SYSTEM.—Section. I.

Contents.—Neuralgia—*Tic-douloureux, Sciatica, Herpes zoster, Neuritis*—Peripheral paralysis—*Causalgia, &c.*—**Electricity**—*Ziemssen's motor points*—Paralysis of the cranial nerves—Facial paralysis—**Scheme of the brain and spinal cord**—Paraplegia, hemiparaplegia and hemianæsthesia—**Acute and chronic myelitis, and softening of the cord**—*Compression paraplegia—Secondary degenerations of the spinal cord—Spastic spinal paralysis—Alcoholic, syphilitic, hysterical, and reflex paraplegia—Spinal weakness, irritation, anæmia, and congestion*—Landry's acute ascending paralysis—Locomotor ataxia—Acute and chronic spinal meningitis—Poliomyelitis anterior acuta (infantile paralysis)—Poliomyelitis anterior sub-acuta vel chronica—Progressive muscular atrophy—(*Sclérose latérale amyotrophique* and *pachyméningite cervicale hypertrophique*)—Pseudo-hypertrophic paralysis—Bulbar paralysis—Multiple sclerosis—Paralysis agitans.

Neuralgia.—The changes which take place in the nerve trunks or within the nuclei of the cerebro-spinal centres, in cases of neuralgia, are still unknown. Pains of all kinds—as pleuritic and cardiac pains, renal and hepatic colic, peritonitis, &c.—are conveyed to the cerebral centres by the sensory nerves; but these are not considered as neuralgic pains, clinically, although some of them are really of that nature. Neuralgia is a pain, usually periodic, connected with a nerve, and which may arise from obscure changes within the spinal or cerebral centres, and be associated with a nerve trunk; but it is not easy to define it accurately, although a very common clinical entity. The pain may also arise from a peripheral irritation, and it is then a "reflex neuralgia"—*e.g.*, caries of the teeth. Pressure upon a nerve trunk by growths, tumours, or bony excrescences, gives rise to severe pain—the pain being often referred to the peripheral extremities of the nerve. Neuralgia usually affects one side only. It is very rarely bilateral; and a characteristic symptom is the presence of "painful points" in the course of a nerve, aggravated by pressure, and generally found where the nerve issues from a bony canal, or pierces the fascia. A certain amount of anæsthesia of the skin supplied by the nerve affected, is almost invariably present in cases of neuralgia. The constitutional condition is an important factor in the causation of neuralgia, and hence syphilis, rheumatism, lead poisoning, and malarial diseases, &c., must be considered in the treatment.

Neuralgia of the Fifth Nerve.—*Trifacial neuralgia, Tic-douloureux.*—The severe form of this affection is somewhat rare, but the milder forms are common. The pain is paroxysmal, and attacks the patient with great suddenness, and it is extremely violent.

The whole of the nerve is not always affected. The muscles of one side of the face are contorted, and the expression indicates the intense suffering. The paroxysms may only last a few seconds or a minute, but they recur from time to time. The attack may extend over a few hours, with remissions extending over a few days, or longer. In the chronic cases, the whiskers or beard, on one side, may be worn off by the constant friction to which the face is subjected during the painful seizures; and even the configuration of the bones may become flattened, and the expression altered, in the very protracted cases. The "tender points" are well marked in these cases, and pressure upon a tooth, or a cold draught, may excite an attack. The severe type of trifacial neuralgia is often associated with mental disease, and with migraine. It is believed never to arise from any *reflex* causes: although pressure upon a decayed tooth may excite an attack in those subject to the disorder. The milder forms of neuralgia of the fifth nerve are frequently reflex. Chronic inflammation of a tooth pulp is the commonest cause; but other forms of dental disease give rise to it. The dentist should be consulted, and a thorough examination of the teeth should be made in all cases of facial neuralgia. The possibility of an early morbid growth pressing upon the nerve trunk has also to be remembered.

Neuralgia may affect other nerves; but these need not be described in detail. "Cervico-occipital" and "cervico-brachial" neuralgia may be noted. Neuralgic pains in the arm may be due to reflex causes; and dental disease is frequently a cause of these pains. Disease of the articular processes of the cervical vertebræ is also a possible cause, and so is aneurism of the subclavian or axillary arteries.

Sciatica is a very common neuralgia, differing from other forms inasmuch as the pain is more or less constant, with severe paroxysms of "lightning" pain. In severe cases the pain sometimes radiates into the sciatic nerve of the opposite side, and it is much increased by pressure. The "tender points" may be readily discovered, and the whole length of the nerve is unduly sensitive. In the chronic and severe cases there is wasting of the muscles, partial anæsthesia of the skin, and the knee and hip-joints become permanently flexed. The chief *causes* of sciatica are exposure to cold, and over-fatigue—besides the cases which may arise from pressure on the nerve by sacral and other growths. Fæcal accumulations, when excessive, may give rise to the milder forms, and certainly constipation aggravates the pain in all cases of sciatica, whatever the original cause. In the *diagnosis* of sciatica, when the pain is in the calf, an examination for femoral thrombosis should be made. Hip-joint disease can be excluded by testing the joint by pressure. Very advanced cases of sciatica may be mistaken for spinal disease. The possibility of sacral growths and tumours pressing on the sacral plexus must be noted.

The *duration* of neuralgia is variable, and it depends upon the amenability to treatment. *Tic-douloureux* is most obstinate, and may be incurable. The disease may last for years. In sciatica the

improvement is often very slow. Some cases extend over a year with more or less pain at intervals. The mild cases may cure in a week or two; but some of the severe cases extend over a long period of time, and render the patient totally unfit for any employment. Neuralgia—however wearing out it may be—takes long to produce death by exhaustion.

Herpes Zoster. *Zona. Shingles.*—This affection is often included among the skin diseases, owing to its being associated with a vesicular eruption; but it is believed to be due to morbid changes within the ganglia of the posterior nerve roots, and in adults, at least, it is almost always accompanied by severe neuralgic pain. The pain generally precedes the eruption, which consists of groups of papules, which very soon become vesicles, drying up in about a week. Sometimes the vesicles become purulent, and the dried-up pustules may leave small cicatrices. The early groups are those nearest the origin of the nerve, and these may often be seen healing up while the later groups, following the distribution of the nerve, are in the stage of maturation. The vesicles sometimes coalesce and leave large purulent and eczematous looking patches. The groups vary in number from one or two to eight or ten. Three clusters are a very common number, when an intercostal nerve is the seat of the disease. This is perhaps the commonest site; but the nerves of the arm or leg, and the cervical or facial nerves often suffer. Herpes zoster is almost invariably unilateral. The pain often continues for a long time after the disappearance of the eruption. The *causes* are unknown; but it has been observed to follow the use of arsenic, medicinally, in a few cases. In the *diagnosis* of herpes zoster the most important differentiation is that of erysipelas, when the nerve affected is the first division of the fifth. When seen early there is no difficulty, but when the upper eyelid is swollen and the redness is diffused, a mistake may be made. The history of a unilateral lesion, and the absence of a high temperature and constitutional disturbance, are the points to be noted. Ulceration of the cornea and iritis frequently attend this form of shingles, and the eyesight may be permanently damaged. In other cases—when seen late—the occurrence of purulent-looking patches, instead of groups of vesicles, may appear puzzling; but not when there are several of them in the course of a nerve. When an intercostal nerve is affected, simple pleurodynia, intercostal neuralgia, caries of the ribs, and pleurisy, have to be excluded—in the early stages, before the appearance of the eruption.

Neuritis, or inflammation of a nerve trunk, may result from wounds or injuries, and sometimes from direct exposure to cold. Often it is caused by syphilitic deposits—especially in the cranial nerves—pressing directly upon the nerve fibres. The latter form is generally chronic in character and it leads to atrophy and degeneration of the nerve trunks; but the acute forms begin with hyperæmia and pass on to suppuration and softening. Recovery may take place even after extensive inflammation.

The *symptoms* begin with chilliness and headache, with severe pain in

the line of the nerve shooting to the peripheral distribution. There is often cramp of the muscles supplied by the nerve; anæsthetic areas arise from destructive changes within the nerve trunk, in the later stages. When chronic changes ensue, the muscles waste, and certain "trophic" changes appear—as loss of hair or nails, herpes, or "glossy skin," &c. The electrical re-actions become abnormal.

The *duration* is short in the acute form, and recovery may be complete or partial. In the chronic forms, the electrical re-actions are a guide to the course and prognosis. The *treatment* consists of leeches along the course of the nerve, when possible—in the acute forms. Morphia should be injected; and quinine may be given in large doses. In the chronic forms, galvanism should be used; and large doses of iodide of potassium should be administered.

The Treatment of Neuralgia.—All reflex causes, as caries of the teeth, should be removed. Five grain doses of quinine—repeated twice within four hours, at the time when the attack is expected—will often ward off the paroxysm. The quinine has also a curative effect. Fifteen grains of antipyrin may be used in the same way. If a constitutional cause be suspected, iodide of potassium, in five or ten grain doses, should be prescribed. Gelsemium and aconite are also recommended. Exalgine has of late gained a great reputation in the treatment of neuralgia (℞ 54). The hypodermic injection of morphia (half a grain, if necessary) or of pure chloroform (five to ten minims) are highly beneficial—not only as palliative, but, ultimately, as curative measures. The hypodermic injections are often imperative, especially in cases of sciatica. Galvanism may also be tried in persistent neuralgia. Arsenic, iron, and cod-liver oil are often strongly indicated. In herpes zoster, the patches of vesicles should be treated with zinc ointment, and covered with cotton wool and collodion. In sciatica, constipation should be actively treated; and surgical measures may be necessary (blistering, Corrigan's button, nerve stretching, sciatica needles, &c.)

Peripheral Paralysis.—Paralyses of the cranial nerves are of most interest to the physician; but any nerve, or group of nerves, may be affected, and the symptoms will vary according to the distribution of their branches to the muscles. A few examples of these may conveniently be introduced here.

Injuries to the brachial plexus, or simple pressure upon the nerves as in "crutch paralysis," may be followed by more or less loss of power and sensation in the arm. Stretching the nerves—as frequently happens in the young, from active gymnastic exercise—may produce a partial loss of power (paresis) with tingling sensations in the limb. Sleeping with the arm over a chair is another familiar instance, with similar symptoms. In more obscure cases, aneurism, bony growths, or tumours must be carefully searched for. Caries in a wisdom tooth has been known to cause severe pain in the arm, with partial paralysis. This is of the nature of a "reflex" paralysis, and it disappears with the removal of the cause.

Paralysis of the *serratus magnus* supplied by the *posterior* or *long*

thoracic nerve, may be produced in certain trades by over-exertion, or by a blow upon the shoulder. The "wing-like" projection of the scapula, and the inability to raise the arm above the shoulder, are the prominent symptoms.

Paralysis of the muscles supplied by the *circumflex nerve*, is also common. The deltoid is the chief muscle affected, and the patient cannot lift his arm from the side. Disease of the shoulder joint should be noted in the diagnosis of this form.

Other examples might be given, but it will be obvious that only anatomical knowledge of the nervous distribution is necessary, to suggest the seat of the lesion and to properly interpret the symptoms. It should be noted that the unopposed contraction of the sound (antagonistic) muscles produces contractions and deformities; and that a lesion of one nerve may soon spread to other nerves. The electrical re-actions of the nerves and muscles are modified in paralysis (see p. 200).

Anæsthesia (loss of common sensation), **analgesia** (loss of *painful* sensation), **hyperæsthesia** (increased sensation), and **causalgia** (burning sensation) are morbid conditions, affecting the sensory fibres of the nerves, and they are often associated with paresis and paralysis of the nerve trunks. They are also, however, just as frequently symptoms of diseases of the brain and spinal cord. The anæsthesia may be measured by means of the æsthesiometer, with due regard to the degrees of sensibility of the different parts of the body. These abnormal sensations in affections of mixed sensory and motor nerves, arise from the same causes which produce the paralysis; but they are sometimes absent, and they often, when present, differ in degree—the paralysis being often great while the sensory fibres are only slightly affected.

Causalgia is believed to be a morbid condition of the peripheral end organs, and it is frequently associated with a "glossy" state of the skin. Cold applications relieve the pain. It is a functional disorder which, in many cases, is soon cured, or gradually passes away; but sometimes a severe type is met with, in which there is ulceration of the skin, painless whitlows, and shedding of the nails. Affections of the nerve trunks are sometimes—as in spinal disease—followed by painful swellings of the joints. In some cases they may end in anchylosis and deformity of the limbs.

The **diagnosis** of affections of the nerve trunks is generally obvious; but *progressive muscular atrophy* must be noted. All possible causes of compression of the nerve must be looked for—as tumours, &c. *Reflex* causes must also be excluded. In the lower limbs, a pelvic tumour may compress the sacral plexus and simulate a case of spinal disease.

The **prognosis** is favourable in the majority of the simple affections of the nerve trunks. The paralysis generally passes off in a few weeks provided the cause can be removed.

The **treatment** of the peripheral paralyses is suggested by the cause. Electricity, rest, counter irritation, and the removal of reflex causes are necessary. The electrical re-actions are important in estimating the probable rate of recovery.

The following figures (26, 27, 28, 29, 30) show the chief motor points and indicate the muscles supplied by them :—

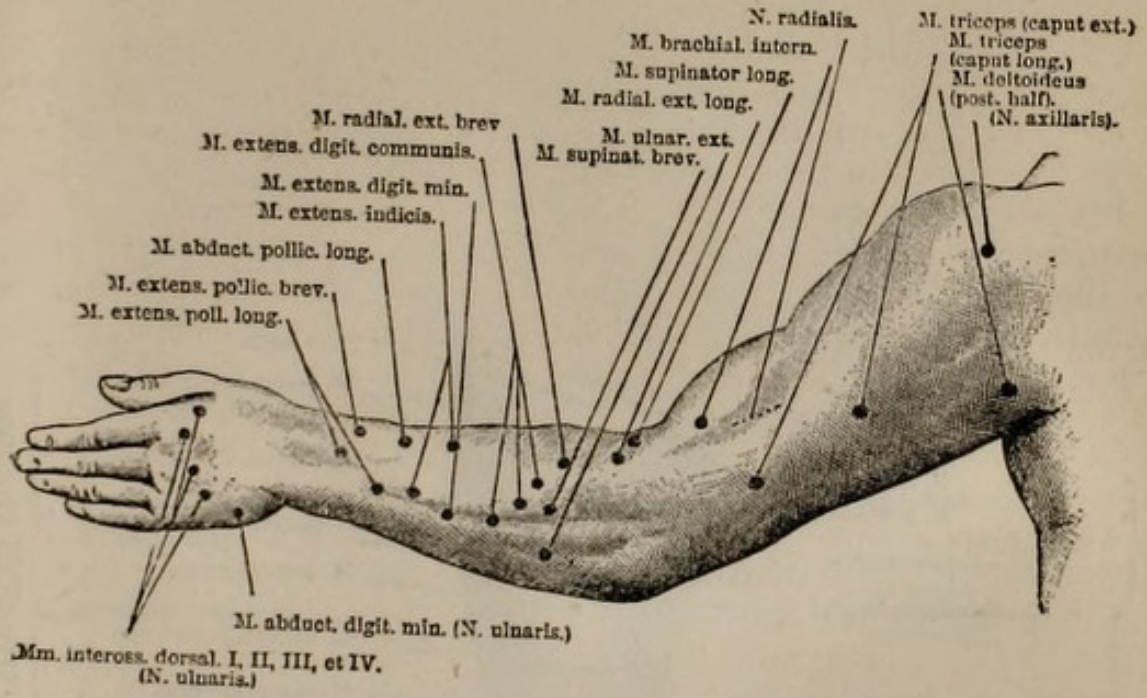


Fig. 26.—Motor points of the radial nerve and the muscles supplied by it; dorsal surface (from Landois and Stirling's *Physiology*).

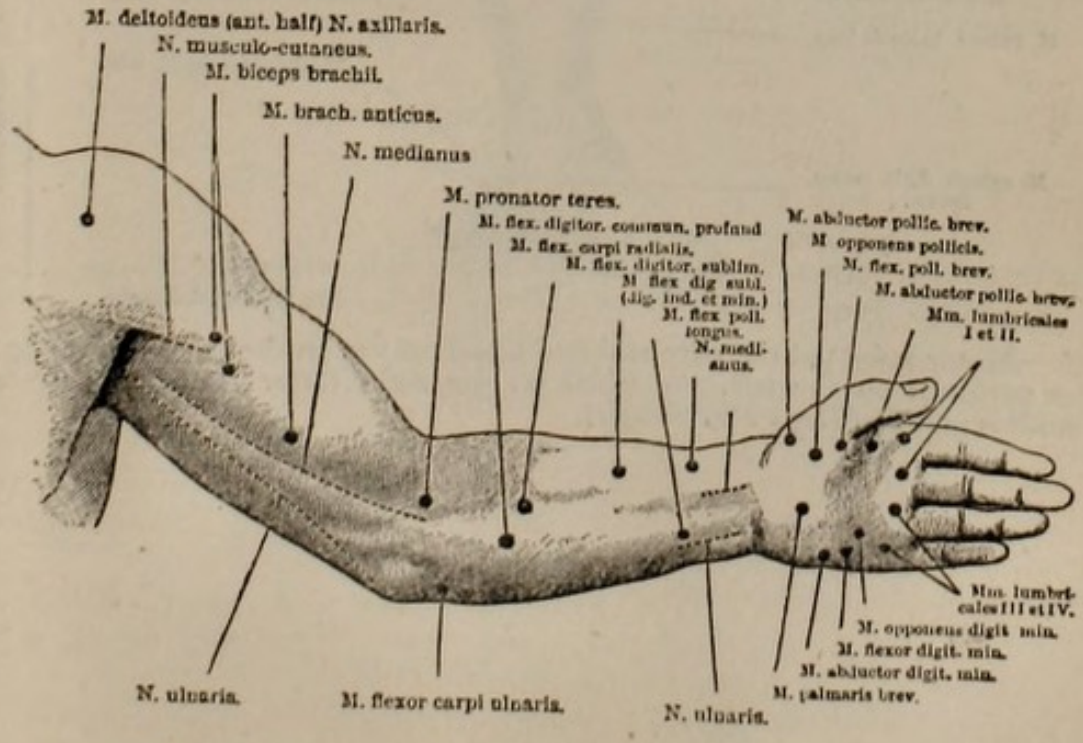


Fig. 27.—Motor points of the median and ulnar nerves, with the muscles supplied by them (from Landois and Stirling's *Physiology*).

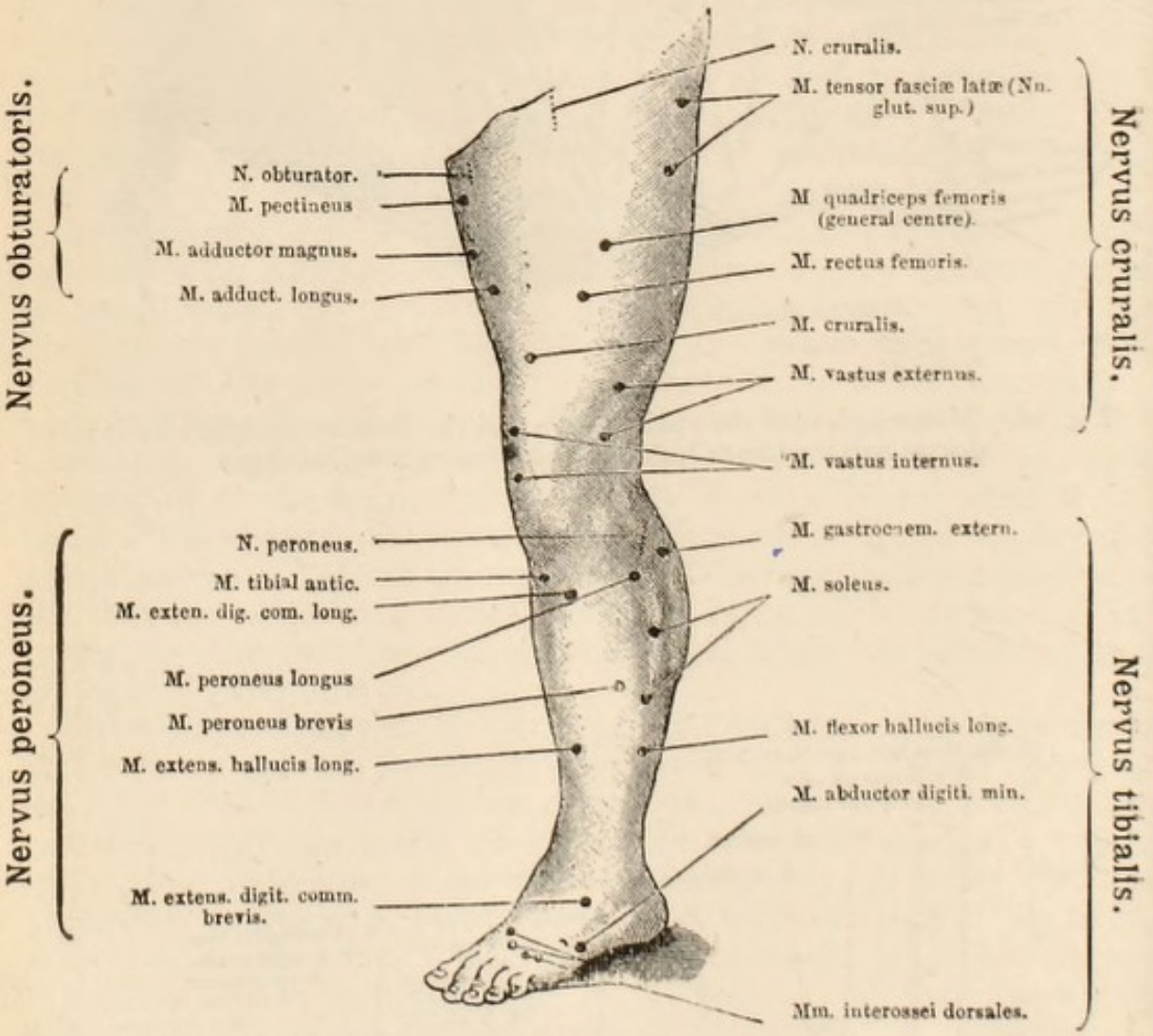


Fig. 28.—Motor points of the peroneal and tibial nerves on the front of the leg; the peroneal on the left, the tibial on the right (after *Eichhorst*) (from Landois and Stirling's *Physiology*).

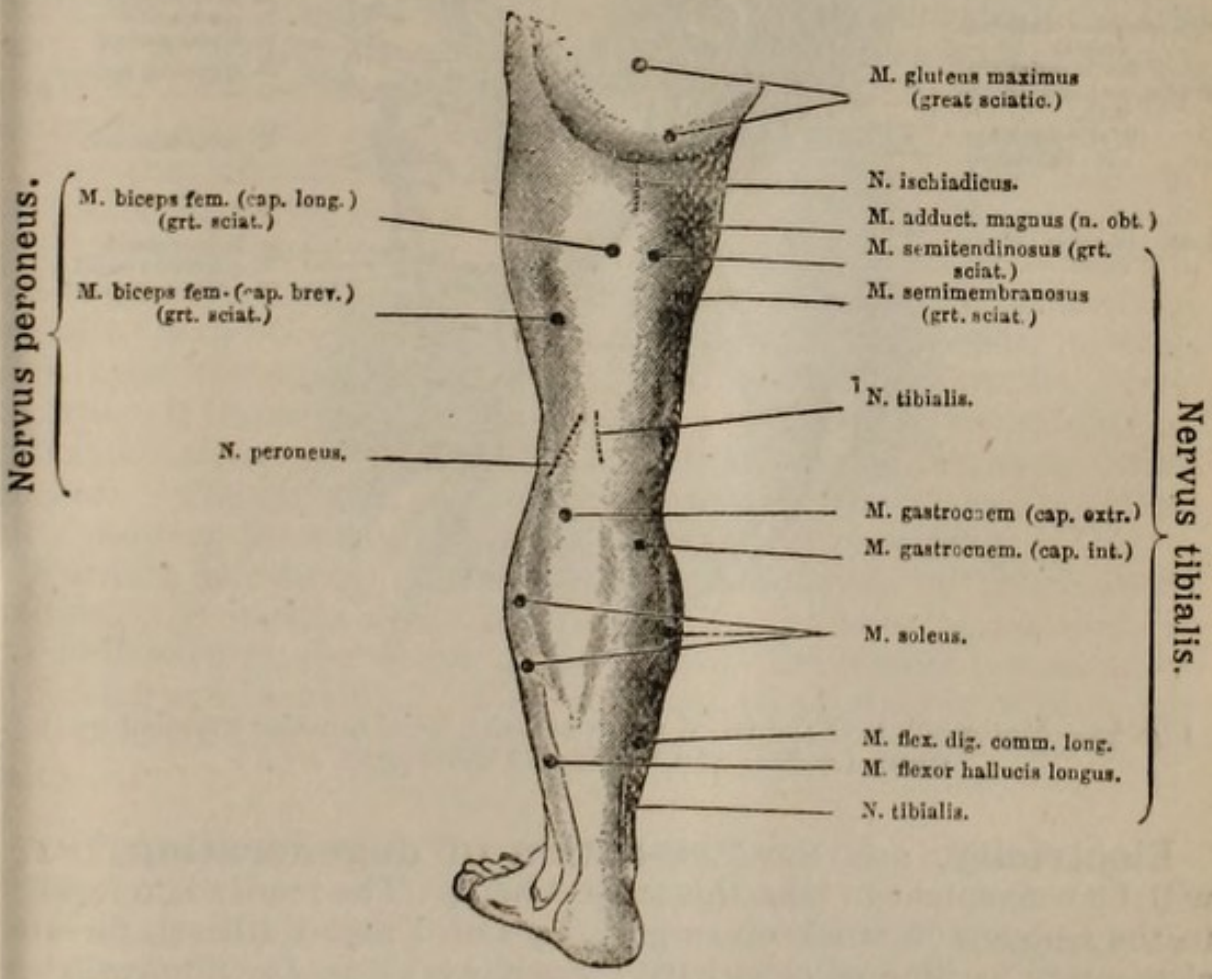


Fig. 29.—Motor points of the sciatic nerve and its branches; the peroneal and tibial nerves (from Landois and Stirling's *Physiology*).

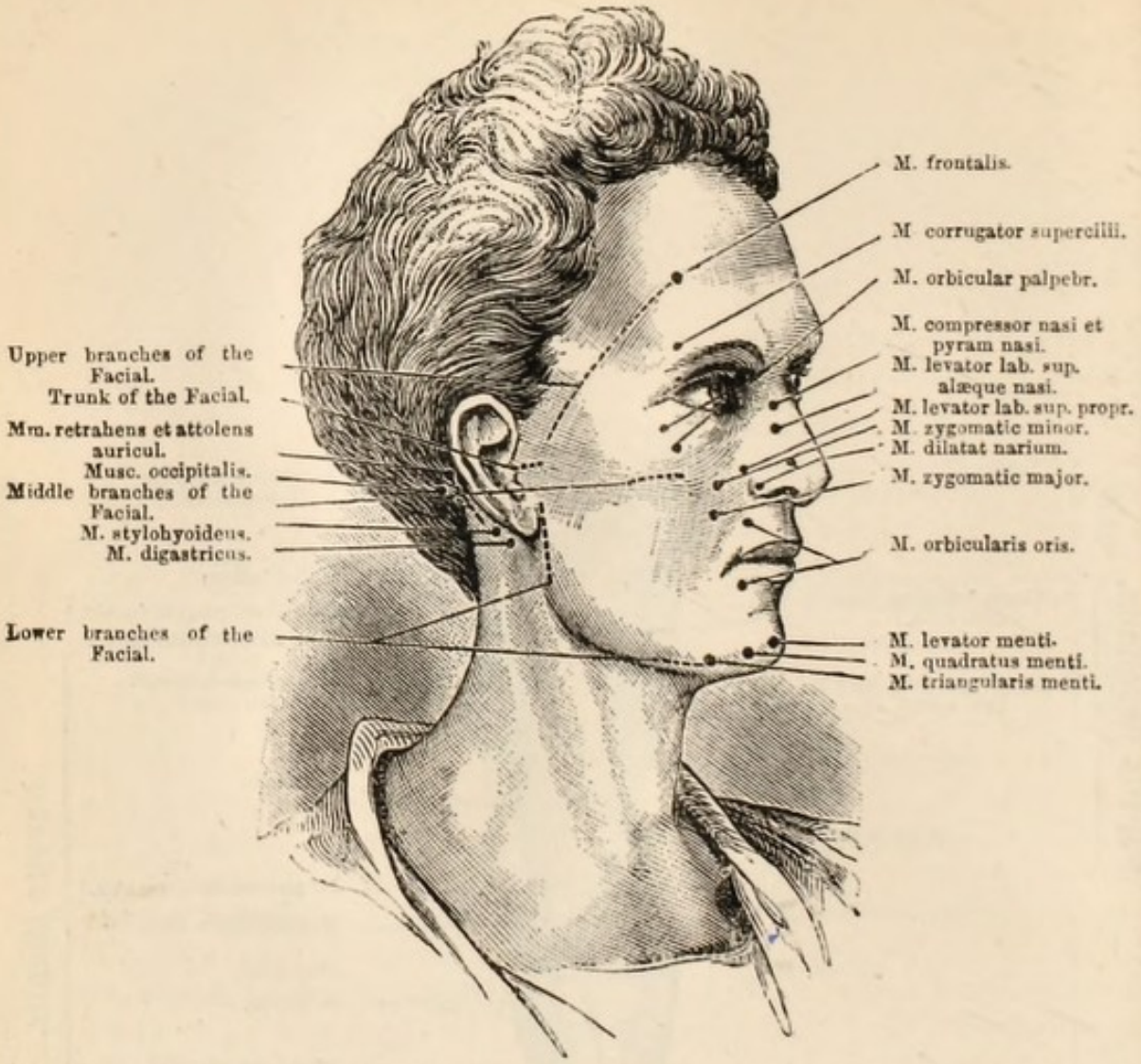


Fig. 30.—Motor points of the facial nerve and the facial muscles supplied by it (from Landois and Stirling's *Physiology*).

Electricity, and the "**re-action of degeneration.**" It will be convenient to take this subject here. The reader is referred to the companion work on surgery, by Caird and Cathcart, for an elementary outline of electricity. Such works as *De Watteville's*, and *Steavenson & Lewis's*, may also be consulted.

Galvanism consists of the use of the "*primary, continuous, constant,* or *galvanic*" current. When using it the skin should be made moist with salt and water. The one pole is placed upon the sternum or spine, while the other is pressed upon the motor point of the muscle it is desired to test.

At the beginning a very weak current should be used, and this should be gradually strengthened. The normal re-actions will be found to be as follow, viz. :—

A *weak* current produces a slight contraction at the *cathodal closure*, when *no* contraction occurs at the anodal opening or closing, or at the cathodal opening. A *strong* current produces a very strong contraction with the *cathodal closure*, and weaker contractions at the anodal opening and closing, and still *no contraction* with the cathodal

opening. *A very strong current* produces tetanus with the cathodal closing, strong contractions at the anodal opening and closing, and *now* a weak contraction at the cathodal opening.

[The terms "make" and "break" are sometimes used for "closing" and "opening." The current is *made or closed*, the moment the sponge is placed upon the muscle (the other pole being always held firmly against the spine or sternum); and it is *broken or opened*, the moment it is lifted off.]

Erb's "*re-action of degeneration*" consists of a "qualitative" change in the galvanic re-actions. *Instead* of the cathodal closing contraction being the first to appear, it is the *anodal closing* contraction, which may be as strong as, or even greater than, the cathodal closing; while the *cathodal opening* contractions (which are the last to appear normally) *now* may appear *before* the anodal opening contractions.

The contractions themselves are also *slower* and more *tonic* in character than the normal, and sometimes only *half the number of cells* are required to produce them. Powerful *faradic currents* often fail to produce contraction when the abnormal galvanic re-actions are present. Erb believes that the abnormal re-action depends, in some way, upon the separation of the affected structures from the nerve centres. If the nerve should not become restored, the excitability to galvanic currents is diminished, and ultimately it becomes extinguished. The muscles, in many cases, regain their normal re-action after showing for many weeks the degenerative re-action.

Faradic electricity is the "*secondary*, *induced*, or *interrupted*" current. It excites clonic contractions in the muscles, but there is no difference in the action of the poles as the current is constantly "making and breaking." The usual method of using is to place the two poles, a few inches apart, over the nerves or muscles to be tested.

Paralysis of the Cranial Nerves.—**The olfactory (1st) nerve.** Paralysis of the first nerve results in a loss of the sense of smell (anosmia). It is sometimes unilateral, and generally unnoticed then by the patient. This condition is of frequent occurrence in cases of hemiplegia. When both sides are affected the loss of smell is complete. Perfumes or bad-smelling odours are the same to him; but *pungent* or *irritating* vapours stimulate the terminal filaments of the fifth nerve, and the impressions thus reach the brain, and often cause him to sneeze. The gustatory sense is also, at the same time, impaired. He can still distinguish bitter from sweet, sour and salt tastes, but his appreciation of delicate flavours is entirely lost. Blows upon the head, either injuring the olfactory bulbs, or the temporo-sphenoidal lobes—in which, according to Ferrier, are the centres of smell—are causes of *anosmia*. Closure of the posterior nares, or chronic catarrh affecting the Schneiderian membrane, are also causes of loss of smell, independent of lesions of the olfactory nerves.

Paralysis of the trifacial or fifth nerve, causes anæsthesia of

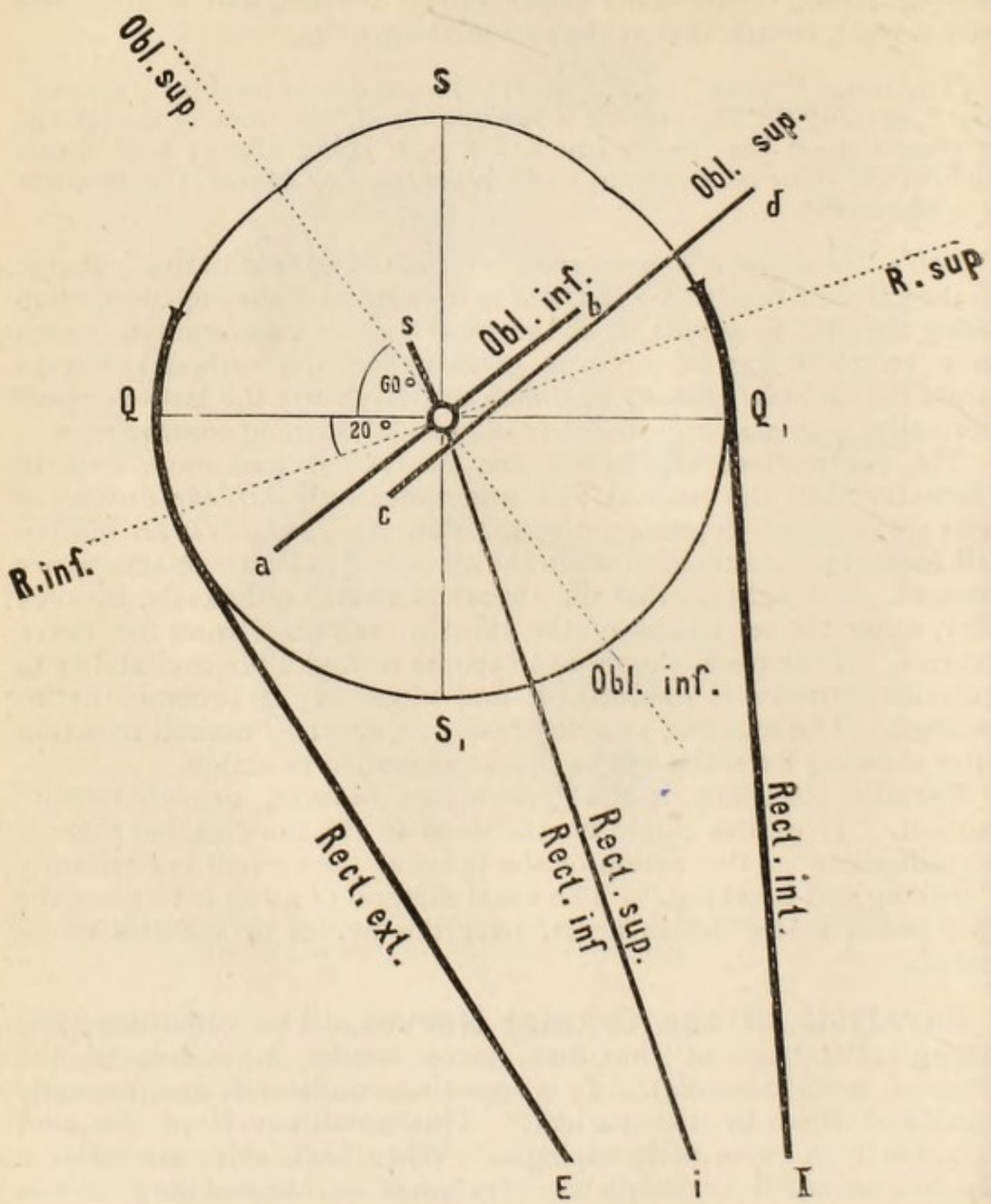


Fig. 31.—Scheme of the action of the Ocular Muscles —S S_1, Visual axis ; Q, Q_1, horizontal axis. 1. The rectus internus (I) and externus (E) rotate the eye almost exactly inwards and outwards. The plane of traction lies in the plane of the paper ; Q, E, is the direction of the traction of the external rectus ; Q_1, I, that of the internal. The axis of rotation is in the point of rotation, ○ at right angles to the plane of the paper, so that it coincides with the vertical axis of the eyeball. 2. The axis of rotation of the R, superior and inferior (the dotted line, R. sup., R. inf.) lies in the horizontal line of separation of the eye, but it forms an angle of about 20° with the transverse axis Q, Q_1 ; the direction of the traction for both muscles is, indicated by the line, *si*. By the action of these muscles, the cornea is turned upwards and slightly inwards, or downwards and slightly inwards. 3. The axis of rotation of both oblique muscles (the dotted lines, Obl. sup., and Obl. inf.) also lies in the horizontal plane of separation of the eye-

the face, and the nasal mucous membrane is insensible to pungent vapours—as ammonia and acetic acid; but the sense of smell is not impaired. The sense of taste in the anterior part of the tongue is lost, as in facial paralysis—the explanation being that the chorda tympani is the true gustatory nerve, and it is closely connected with both the fifth and the seventh nerves. Subjective sensations (numbness and

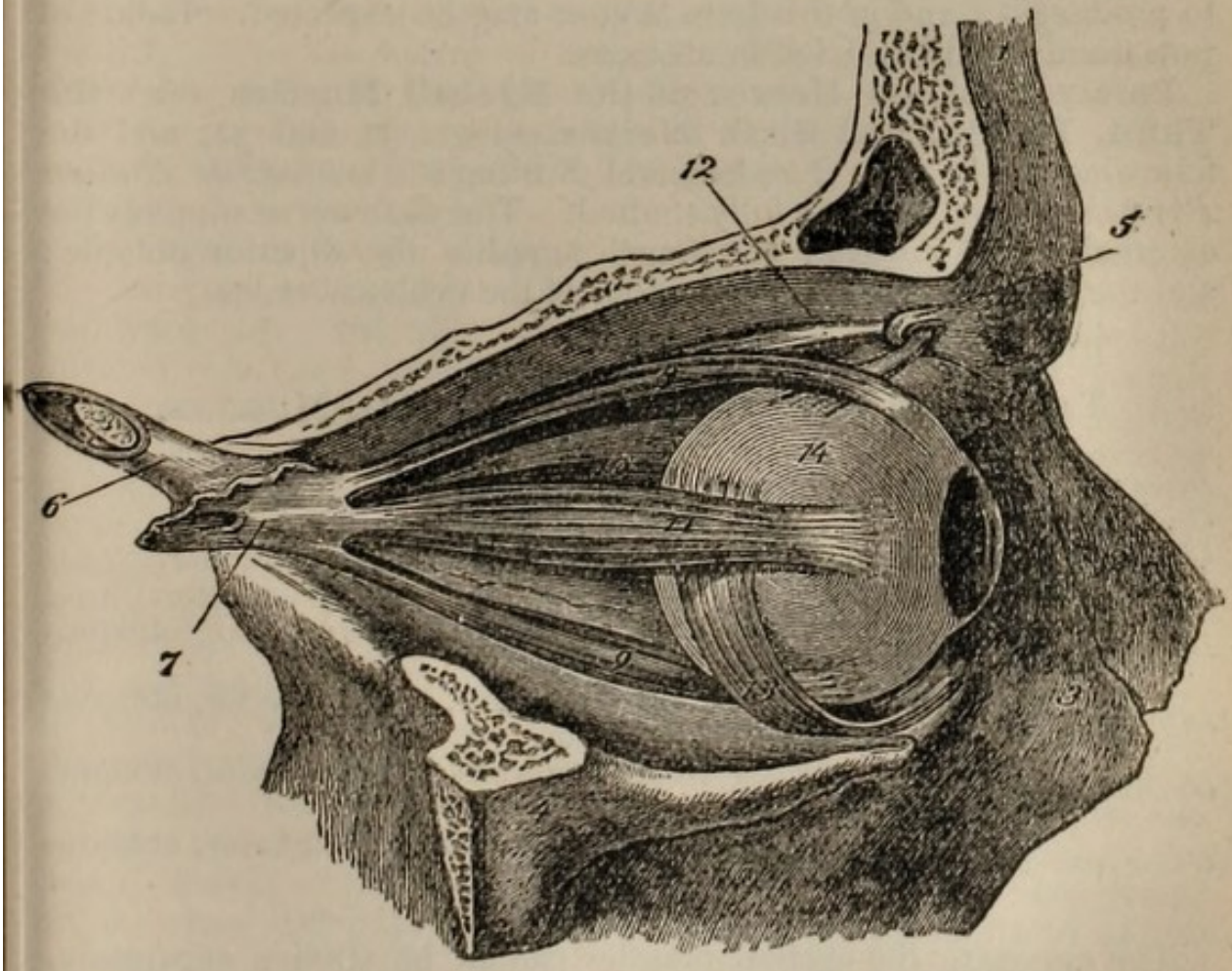


Fig. 32.—Lateral view of the muscles of the eyeball.—5, Trochlea or pulley of the superior oblique muscle, 12; 6, optic nerve; 8, superior, 9, inferior, and 22, external rectus; 13, inferior oblique. (Landois and Stirling's *Physiology*).

tingling, &c.) are complained of, and a characteristic sensation is the feeling only half of a cup or glass when it is put to the lips. It appears to him to be broken. The conjunctiva is insensible. Mastication is imperfect. In some cases the gums become spongy and bleed, the circulation is interfered with and the affected cheek becomes livid, and often ulceration and sloughing of the cornea follow. These changes are due to the trophic centres being cut off,

ball, and it forms an angle of 60° with the transverse axis. The direction of the traction of the *inferior* oblique gives the line *ab*; that of the *superior*, the line *cd*. The action of these muscles, therefore, is in the one case to rotate the cornea outwards and upwards, and in the other outwards and downwards. These actions, of course, only obtain when the eyes are in the primary position—in every other position the axis of rotation of each muscle changes. (Landois and Stirling's *Human Physiology*, 4th Ed., p 965).

it being now established that the fibres concerned in these changes lie in the inner portion of the trunk of the fifth nerve.

The *causes* of paralysis of the fifth nerve are generally serious organic affections—as caries and necrosis of the bones; syphilitic disease; cancerous or sarcomatous growths; aneurism of the internal carotid; and morbid changes within the Gasserian ganglion. Prolonged exposure of one side of the face to cold, has been known to produce it; and in this form a cure may be expected. Iodide of potassium should be tried in all cases.

Paralysis of the Nerves of the Eyeball Muscles, *viz.*: the Third, Fourth, and Sixth Nerves.—Figs. 31 and 32, and the following table from Landois and Stirling's *Text-book of Human Physiology* should be carefully studied. The sixth nerve supplies the external rectus muscle; the fourth supplies the superior oblique; and the third supplies the remainder of the ocular muscles.

TABLE OF THE ACTION OF THE OCULAR MUSCLES.

Inwards,	Rectus internus.
Outwards,	Rectus externus.
Upwards,	Rectus superior, obliquus inferior.
Downwards,	Rectus inferior, obliquus superior.
Inwards and upwards,	Rectus internus, rectus superior, obliquus inferior.
Inwards and downwards,	Rectus internus, rectus inferior, obliquus superior.
Outwards and upwards,	Rectus externus, rectus superior, obliquus inferior.
Outwards and downwards,	Rectus externus, rectus inferior, obliquus superior.

The nerves to the eyeball muscles cannot be studied separately, because the two eyes being moved simultaneously, different nerves are concerned in the production of a given movement; *e.g.*, the *external rectus*, supplied by the sixth, moves with the *internal rectus* of the other eye, and this muscle is supplied by the third nerve; also, paralysis of a muscle of one eyeball, besides producing its own symptoms, gives rise to a *deviation* from the normal movement in the opposite eyeball.

For convenience in description the *left* eye will be supposed, all through, to be the one affected, with the understanding that the *converse* is true when the paralysis affects the right one.

In paralysis of the external rectus, supplied by the sixth nerve, the eye cannot be moved *outwards* when its fellow moves *inwards*. If the patient be directed to look at an object which is gradually moved, the further the object is carried to the left the greater is the difference between the direction of the two eyes. This squint is called the *primary deviation*, and it is very obvious when the paralysis is complete. When the paralysis is only partial, the affected eye may only lag behind its fellow, and there is no squint; but by covering the

sound (right) eye, and then removing the cover after he has fixed the object with the affected eye, the patient finds that he has not his right eye fixed upon the object, and so he is observed to move it quickly to the left.

When the paralysis is more or less complete, force is used to bring the eye round, but in doing so, some part of the force is thrown upon the associated muscle of the other eye—*viz.*: the *internal rectus*, and hence the sound (right) eye is pulled too far to the left. This movement of the sound eye is called the *secondary deviation;* and it is important in slight cases of paralysis, because it serves to distinguish a paralysis of the external rectus from an ordinary strabismus produced by a contraction or shortening of one, or both, of the internal recti.

Diplopia, or double vision, is commonly present in paralysis of the external rectus, but only when the eyes are moved to the paralysed side; and the *false image* (*i.e.* the image seen by the affected eye, and which may be distinguished by the patient covering that eye with a piece of violet-coloured glass) is *outside* the true image, and the two images get wider apart as the object is moved further to the paralysed (left) side. This latter symptom distinguishes paralysis from "concomitant strabismus." Each eye should be tested separately, as sometimes double images are formed upon the retina of a single eye. It should be noted also that a *sudden* development of diplopia does not exclude the possibility of it being the result of concomitant strabismus; as should there have existed a tendency previously, and the eyes (or general health) should have become weakened, fatigued, or impaired, this symptom may occur abruptly. In paralysis of the external rectus there is not always double vision, and sometimes the image is only "blurred." Giddiness, and sometimes staggering, arises, when the patient uses the affected eye *alone;* and as the object appears to him to be farther out than it really is, if asked to strike at it, his hand goes to the outside (erroneous projection).

In paralysis of the (left) superior oblique, supplied by the fourth nerve—the inferior rectus having now (in part) lost its antagonistic muscle—the patient squints to the right and slightly upwards, when he attempts to look at his feet. The *secondary deviation* of the sound eye will be downwards and to the left. The double images are seen when the patient looks downwards, and the *false* image lies *below* and to the left of the true one, and it appears *tilted*. It also appears to be on a plane nearer to the eye than the true image. To prevent a sensation of giddiness the patient often carries his head down and to the right.

In paralysis of the third nerve, the four remaining muscles of the eyeball are affected, when the whole nerve is involved. As, however, a branch only may be paralysed, the muscles must first be considered separately:—

The (left) *internal rectus:* the *primary deviation* occurs when the eyes are moved to the right; the affected eye cannot be moved inwards, and a *divergent* squint is produced. The *secondary deviation*

of the sound eye is towards the right. The double vision, and other symptoms, are the same as in paralysis of the external rectus, only *converse*, as regards the direction.

The (left) *inferior rectus:* the squint is a little upwards when the patient looks downwards, and the rotation of the eyeball is to the left. Diplopia is present, and the false image is below and to the right of the true one, and it has its upper end "tilted" to the left.

The (left) *inferior oblique:* the affected eye squints a little downwards when the patient looks upwards, and the eyeball is rotated to the right. The false image lies above and to the left of the true one, and it has its upper end tilted to the left.

The (left) *superior rectus:* the affected eye squints a little downwards when the patient looks upwards, and the eyeball is rotated to the left. The false image lies above and to the right of the true one, and it has its upper end tilted to the right.

When the *whole* of the third nerve is paralysed, the eyeball on the affected side *cannot be moved inwards or upwards.* It can be lowered slightly if, at the same time, it be carried a little outwards. Giddiness is generally marked. The primary and secondary deviations vary with the position of the object upon which he is directed to look. In addition to these symptoms, the *levator palpebræ* is paralysed, and the upper eyelid droops (ptosis); but this symptom may occur without paralysis of the ocular muscles. There is also paralysis of the sphincter of the pupil, and consequently *dilatation* (*mydriasis*); and paralysis of the ciliary muscles causing *loss of accommodation.*

The **causes** of paralysis of the third, fourth, and sixth nerves are often obscure. When a single nerve, or a branch of the third is affected only, recovery appears to be the rule. Aneurism of the internal carotid is a possible cause; and syphilitic and malignant growths are possibly the commonest causes when more than one nerve, or branch, is involved. Syphilis may sometimes be the cause of ptosis, without there being any evidence of a gummatous formation. Ptosis is common in the aged, and it is probably due to dilated and tortuous arteries compressing the fibres (in part) of the third nerve. Paralysis of the nerves to the eyeball muscles may be due to disease at the base of the brain, and it is frequently an early symptom of locomotor ataxia.

The **treatment** should consist of large doses of iodide of potassium whether the suspected cause be syphilis or not. Galvanism is also good treatment—a current of from six to fourteen cells being used for two or three minutes, the anode being placed on the back of the neck and the cathode upon the closed eyelids.

Facial paralysis, or paralysis of the seventh cranial nerve; **Bell's paralysis.** A *peripheral* facial paralysis may be caused by an inflammation of the nerve itself, and the swelling extending to the part which issues from the narrow stylo-mastoid foramen, a compression results. Prolonged exposure of one side of the face to cold, or even general exposure, may give rise to this condition. At

other times inflammation of the parotid or cervical glands, tumours, or wounds of the face, may produce facial paralysis.

Within the aqueductus Fallopii, the nerve may be affected by disease of the petrous portion of the temporal bone. Chronic disease of the tympanum very often leads to caries and necrosis. Facial paralysis has been known to follow a severe blow upon the side of the head. Within the cranium, disease of the base of the skull, tumours, and syphilitic growths, may be the cause; but in this latter group *other* nerves are likely to be involved, and the symptoms of brain disease are superadded. A minute point of softening in the pons has been found to cause facial paralysis; and facial paralysis has been known to precede tubercular meningitis as an early symptom. Some cases appear to be "reflex." The "central" causes of facial paralysis are considered with cerebral hæmorrhage and hemiplegia.

The **symptoms** are generally very obvious. The mouth is drawn to the sound side when the paralysis is complete, and the cheek of the paralysed side is smooth and devoid of wrinkles. The eye remains wide open and cannot be closed, although during sleep the eyelids approximate far more closely than it is possible for the patient to produce voluntarily. When the attempt is made to forcibly close the eyelids, the cornea is turned up under the lid—this being due to the association of the nerves of the ocular muscles with the act of forcibly closing the eyelids. The conjunctivæ may become inflamed by dust, &c., entering the unprotected eye. The reflex movement of the eyelids is abolished. The cheek of the affected side is not held close to the teeth, and it bulges out with the breathing. Mastication is imperfect, and food accumulates between the cheek and the teeth. Attempts at laughing, smiling, and whistling fail to produce any alteration in the expression of the affected side. The pronunciation of the labial consonants is imperfect. In some cases, the mouth in repose is not drawn to the opposite side, and this is frequently the case in facial paralysis the result of exposure to cold, and in the so-called "rheumatic" type. In the severe forms resulting from caries of the bones, the muscles seem to lose their tone, and the antagonistic muscles of the opposite side produce extreme distortion, and the eye stares very fixedly.

Smell and *taste* are impaired, the former being due to a dry state of the nostril resulting from the tears escaping over the cheek in extreme cases, and the latter due to the implication of the chorda tympani. The sense of *hearing* is often rendered very acute, and this occurs when the stapedius muscle (supplied by a branch of the seventh) is paralysed, and allows its antagonist, the tensor tympani, to keep the tympanum overstretched. The soft palate is often affected, and the uvula is frequently turned to the paralysed side.

A double facial paralysis may be produced by a syphilitic gumma involving both nerves at the base of the brain; or by disease of the petrous portion of the temporal bones on both sides. The face then is absolutely expressionless, when any of the movements, as smiling, whistling, weeping, &c., are attempted.

In the **diagnosis** of facial paralysis, it is only the very mild forms that may escape notice. A careful examination will generally reveal the symptoms, as for instance, the *incomplete* closure of the affected eye. *Peripheral* facial paralysis is differentiated from *central* paralysis by the fact that in the latter the patient is quite able to close the eye.

During the **course** of a facial paralysis, when the muscles begin to show signs of recovery, the face is frequently distorted by spasmodic contractions, due to the efforts of the patient to use the muscles of the affected side. These generally disappear as the patient gets well; but sometimes a permanent *tonic contraction* is induced, and this is invariably the case when the paralysis is incurable. The cases of facial paralysis which result from cold generally get well within four or six weeks; but sometimes they are prolonged to six months. In the *mild* cases, the muscles and nerves re-act to both galvanic and faradic currents; in the severe cases the "re-action of degeneration" (see p. 200) is present.

The **prognosis** is estimated by the electrical re-action; for should the re-action of degeneration be present, no improvement can be expected for two or three months, and perhaps, for six or eight months—and even then there may be permanent stiffness or weakness of the muscles for years, or for life.

The **treatment** of facial paralysis consists of blistering behind the ear; and the administration of small doses of mercury, and large doses of iodide of potassium. Both kinds of electricity are used, but neither will shorten an attack. Electricity seems to be most useful when the case is beginning to recover. In using *galvanism* the anode should be placed behind the ear, and the cathode moved over the paralysed half of the face.

Paralysis of the ninth or hypoglossal nerve is a rare affection, characterised by an extreme wasting of one-half of the tongue. A cancerous nodule pressing upon the nerve, and disease of the atlas and neighbouring bones, have been known to produce it.

The Brain and Spinal Cord.—In attempting to prepare a semi-diagrammatic scheme which may be useful in understanding the diseases of the brain and spinal cord, I have avoided those details which seem, at present, to be of less practical importance, as they only serve to complicate the diagram. The beautiful drawings of the spinal cord in Dr. Bramwell's work, have served as a model for the spinal portion of the scheme.

The brain is represented as a coronal section, with the posterior third removed, so that the reader looks into the brain from behind. The first, second, and third frontal convolutions (1, 2, 3), with part of the superior temporal lobes (Te), are seen in the section. The corpus callosum (Co-Ca), corpora striata (Cs), optic thalami (O.T), lenticular nuclei (L.T), and internal capsules (I.C) terminating in the crura cerebri (Cr), which are cut, and show the divisions into crusta (C) and tegmentum (T)—are the anatomical points, figured

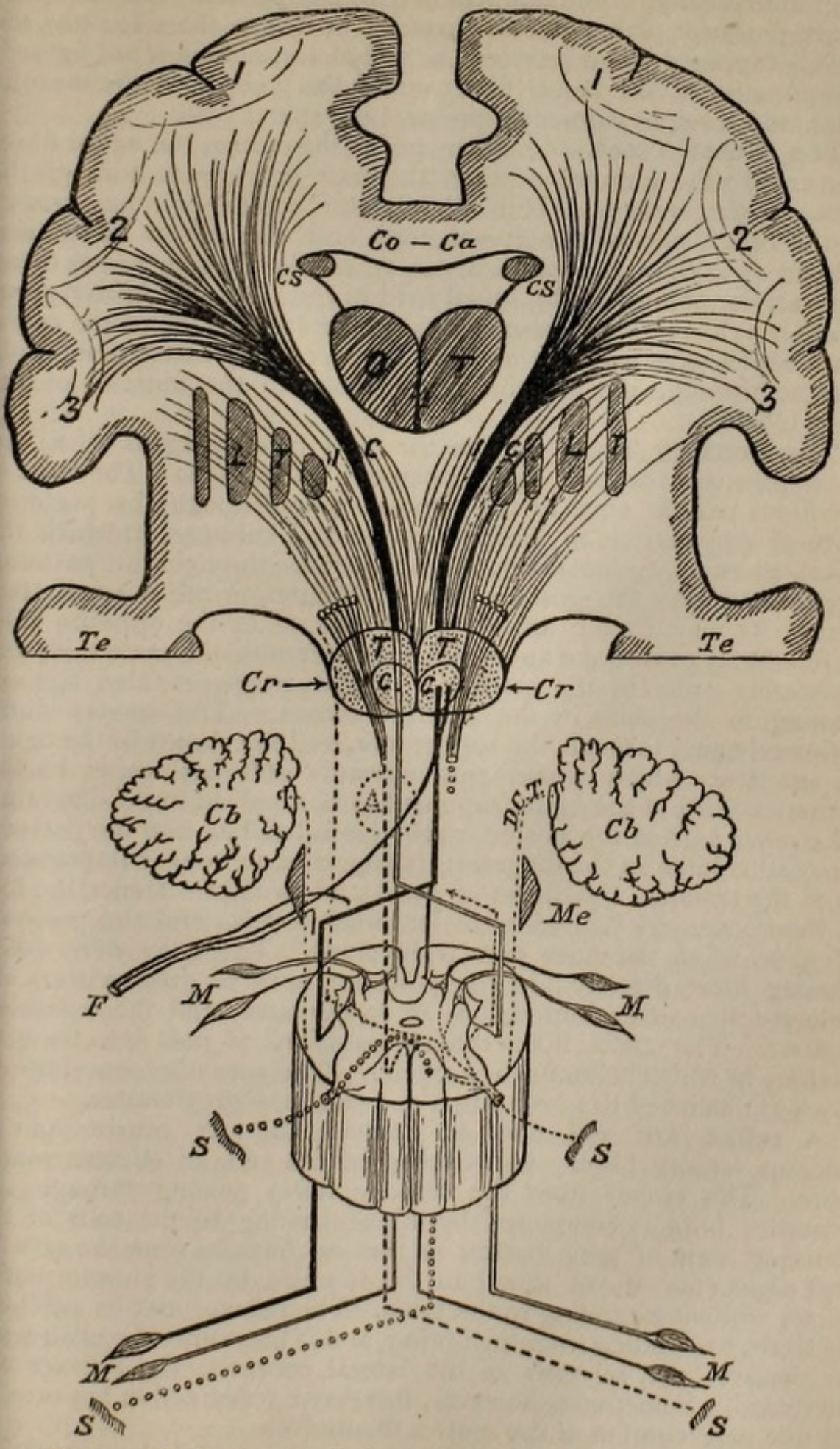


Fig. 33.—The brain and spinal cord with sensory and motor tracts.

diagrammatically. The cerebellum (Cb) is supposed to be cut in the antero-posterior plane, and the halves reflected to the side; and the pons is supposed to lie between the peduncles, although not figured. The crossing of the motor fibres marks the position of the medulla (Me), and a single segment represents the spinal cord.

The Motor Tract.—Commencing at the cortex, the motor fibres converge to the internal capsule, and pass downwards through the crusta to the medulla. Here, the largest bundle of fibres crosses to the other side of the spinal cord, and descends in the lateral column (*crossed pyramidal tract*). A smaller bundle passes down the anterior column of the spinal cord of the same side (*direct pyramidal tract*). From these columns, in their course down the spinal cord, fibres pass into the anterior horns of grey matter, and from thence, through the anterior nerve roots, to the muscles of the body (M.M).

The Sensory Tract.—From the sensory end organs (S.S) the fibres pass into the cord by the posterior nerve roots. The most of the fibres pass at once to the opposite side, through the posterior horn of grey matter, to the postero-internal columns, in which the fibres ascend. Some fibres, in their course through the posterior horn, bend into the postero-external column, while a small tract makes its way to the antero-lateral column of the opposite side. A few fibres pass to the antero-lateral column of the same side, and ultimately cross in the medulla. Small twigs are also figured, passing to the cells in the anterior cornua. The sensory fibres ultimately pass through the tegmentum, and are shown in the figure as *cut fibres* which would pass upwards and backwards to the posterior third of the brain (supposed to be removed in the diagram). The *inner part* of the posterior root (seen bending into the postero-external column in the diagrams) is supposed to convey impressions from the tendons, and those of *touch* and *locality*. Hence the loss of the "knee-jerk" in cases of locomotor ataxia, and the presence of *pain*, when the inner part is involved. The *outer fibres* (seen passing more directly into the posterior horn of grey matter) are believed to convey impressions of *temperature*, and the *cutaneous reflexes*. The *central* fibres convey sensations of pain into the grey matter, in which columns they ascend. The *muscular sense* impressions are believed to pass up by the columns of grey matter.

A reflex arc will now be understood. It consists of an afferent sensory fibre, a transferring centre, and an efferent motor fibre. The circuit from the sensory fibres passing through the posterior horn is completed by fibres passing to the cells of the anterior horn of grey matter, so that an impulse from the sensory end organs may travel round to the muscles, by the anterior nerve roots, without ascending to the brain. The reflexes may be *inhibited* by fibres descending from the brain; and if these fibres be obstructed by disease, *e.g.*, sclerosis of the lateral columns, the reflexes are increased. Sometimes, however, increased reflex action appears to be due to excitation of the centres themselves.

The reflexes are classified into three groups—(1) the superficial,

(2) the deep or tendon reflexes, and (3) the organic reflexes. The *superficial* reflexes may be arranged in an ascending series, beginning with the lowest, viz., the plantar (sacral), gluteal, cremasteric (lumbar), abdominal, epigastric (dorsal), and interscapular reflexes (lowest cervical). They are excited by scratching or tickling the cutaneous surfaces in these regions. The conjunctival and pupillary reflexes are also important. The *cilio-spinal centre* lies in the lower cervical part of the cord, and it is connected with the dilatation of the pupil. The motor fibres pass from this region by the anterior roots into the cervical sympathetic, and they are antagonised by the fibres, to the pupil, of the third cranial nerve. The *deep* reflexes are the "knee jerk" and ankle clonus. According to Gowers, the centre for the former is in the second, third, and fourth lumbar—and for the latter, in the first, second, and third sacral segments. The *organic centres* are numerous, but in the sacral region of the cord those concerned with micturition, defæcation, and the sexual functions are highly important in relation to paraplegia and other diseases of the spinal cord.

Trophic cells exist in the anterior horns of the grey matter of the cord, and any interference with them by disease causes wasting of the muscles, and there is an increased liability to the formation of bed sores.

The facial nerve (F) has been introduced to explain *crossed* paralysis. Three fibres only have been figured—one descending from the cortex with the motor fibres, and crossing in the pons; a second, supposed to spring from the floor of the fourth ventricle; and a third, arising from the lower part of the medulla. These join, and emerge from the side of the medulla, as the seventh or facial nerve. A lesion affecting the fibres in the right internal capsule will cause hemiplegia and a facial paralysis of the left side; a lesion in the left half of the pons (at A) will produce a hemiplegia of the *right* side and a facial paralysis of the *left* side (*crossed paralysis*). The different origins of the seventh nerve fibres may also account for some of them (in central lesions) escaping destruction and producing only partial paralysis. In *Bell's paralysis* the whole nerve is involved, and the paralysis is complete.

The direct (ascending) cerebellar tract (D.C.T.) is of less importance clinically; but it may ultimately be proved to be concerned with co-ordination of the muscular movements. The **commissural** fibres and "association" fibres connect corresponding parts of the brain. They exist also in the spinal cord, connecting the different segments; but they have not been figured.

The various **conducting paths** of the spinal cord are known by the effects of injuries and disease. The trophic or nutritive centres for the *descending* fibres are situated in the cerebrum; and for the *ascending* fibres they are believed to be situated in the spinal ganglia of the posterior nerve roots. When the conducting paths are separated from their trophic centres (by section, experimentally, or by disease), degeneration results, and this serves to map out the columns into tracts.

The accompanying diagram of a transverse section of the spinal cord (from Landois and Stirling's *Physiology*) shows the parts affected :—

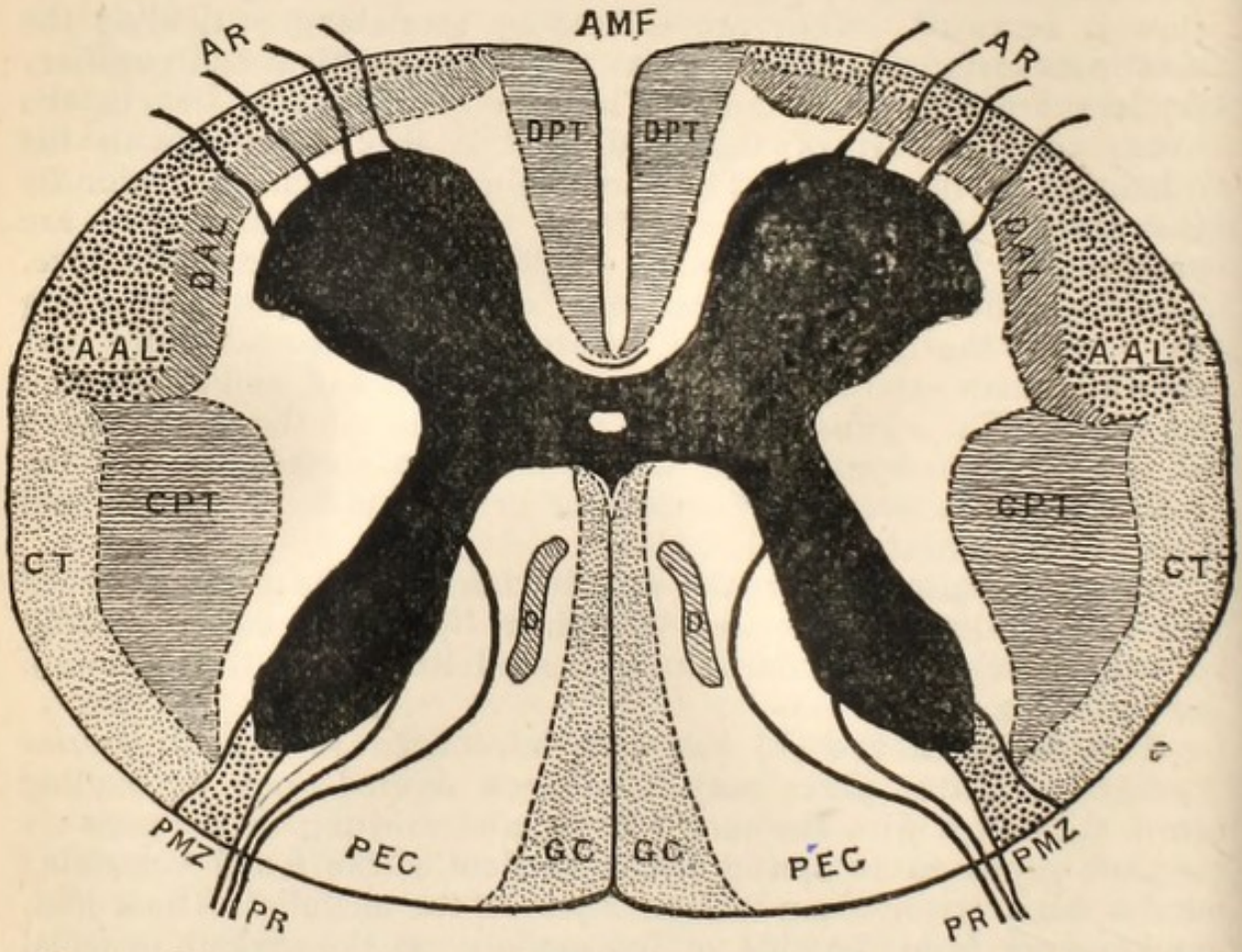


Fig. 34.—Scheme showing the degeneration tracts and the paths that do not undergo degeneration in the cord. A.M.F., anterior median fissure; D.P.T. and C.P.T., direct and crossed pyramidal tracts; A.R. and P.R, anterior and posterior roots; A.A.L. and D.A.L., ascending and descending antero-lateral tracts; C.T., cerebellar tract; D., comma-shaped tract; P.M.Z., posterior marginal zone; P.E.C., postero-external column; G.C, Goll's column postero-internal column).

Of these tracts, the important ones, clinically, are the direct and crossed pyramidal tracts; the anterior and posterior horns of grey matter; and the postero-external columns, and postero-internal or columns of Goll.

Paraplegia is a form of paralysis, variable in amount, which affects both sides of the body up to a certain level. The lesion may only attack a single segment of the spinal cord (and secondary descending degeneration of the motor tracts then follows); or the disease may implicate the cord for a considerable part of its whole length. If the disease be situated in the lumbar enlargement, the lower limbs are paralysed. If situated higher, the abdomen and chest are also involved; and if it extend to the cervical regions the upper limbs will be paralysed as well. A transverse lesion of the cord, beginning at the periphery, may gradually extend across, and as the motor fibres for the more distant parts appear to occupy the

outermost layers of the lateral tracts, this may account for the feet and legs often being paralysed before the thighs and hips. Complete destruction of a segment is always associated with *anæsthesia*, in all the parts below the lesion; but this does not often occur, as the lesion is seldom so complete and the sensory impulses seem to be more easily transmitted than the motor. *Paræsthesia*, however ("pins and needles," tingling, formication, &c.), is often complained of.

The *gait* of the paraplegic patient varies with the degree of loss of power. When sufficiently developed, the limbs are lifted with difficulty and generally too high, and the foot hangs down and is placed on the ground very clumsily. The legs seem stiff. The paralysis is often complete, and walking is impossible. The inability to stand with the feet close together and the eyes closed, and also the sensation of walking upon soft carpets, are symptoms which point to sclerosis of the posterior columns. In such cases, the disease is not necessarily locomotor ataxia. The lower limit of a lesion is sometimes ascertained by the condition and height of the reflex actions already mentioned. The superficial reflexes may be present when the deep reflexes are absent, or *vice versâ*—in the same patient. When the paraplegia is due to a limited lesion, so that the reflexes are present, the muscles as a rule do not waste. The bladder and rectum are often affected in cases of paraplegia. In exceptional cases the mechanism of micturition is normal, but in most there is either retention or incontinence.

Fig. 35 shows the sensory fibres passing up to the brain, and to the two micturition centres in the cord. Normally, when the bladder is full, it excites the mucous membrane and the impulse travels up these fibres. From the brain, an impulse may travel down (probably in the lateral columns) *inhibiting* the centre for the sphincter muscle which is constantly kept contracted by tonic nerve force, and at the same time *exciting* the detrusor centre to empty the bladder by an impulse to the motor nerve. If it should be inconvenient that micturition should take place, then the impulse travels down the other nerve *exciting* the sphincter to keep up its contraction, while *inhibiting* the detrusor.

In paraplegia, with the sacral regions unimpaired—the lesion being higher in the cord—micturition may be carried on *reflexly*, no stimulus reaching the sensorium, and of course no possibility of controlling the act. When the reflex centres are destroyed (myelitis, &c.) there is complete paralysis of the bladder (and rectum) with the paraplegia. The bladder is capable of retaining a certain amount of urine, but when the pressure increases it escapes, and *incontinence* is the result. In paralysis of the detrusor muscle, there is *retention*, until the pressure of the urine in the bladder is sufficient to overcome the sphincter muscle; or the patient requires to strain with the abdominal muscles in order to empty the bladder. With paralysis of the sphincter, there is *incontinence*. Spasmodic contractions of these muscles, leading to incontinence (detrusor) and retention (sphincter), are common. The former condition is a frequent cause

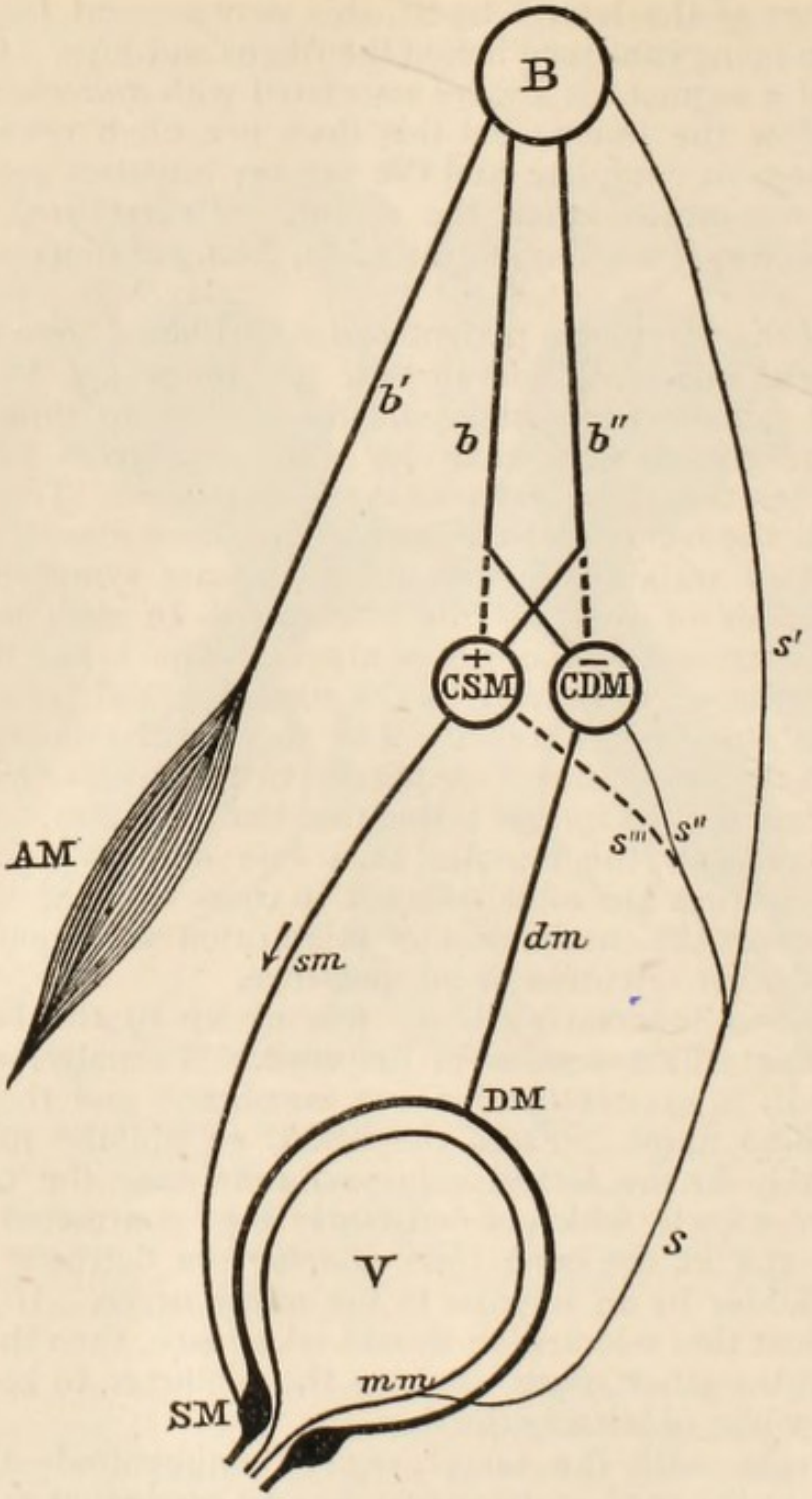


Fig. 35.—Diagrammatic representation of the parts concerned in the mechanism of micturition while at rest. (After *Gowers*, but considerably modified by Bramwell.)—The sphincter muscle (S M) is in a state of contraction, the result of nerve force continually sent to it from its tonic centre (C.S.M.) in the spinal cord, through *sm*, as indicated by the arrow. V = the bladder, which is represented as empty. S.M, Sphincter muscle. D.M, Detrusor muscle. A.M, Abdominal muscles. *mm*, Mucous membrane of bladder. B, The brain. C.D.M, Spinal centre for the detrusor muscle. C.S.M, Spinal centre for the sphincter muscle. S, S′, S″, S‴, Sensory fibres proceeding from the mucous membrane of bladder up to the spinal cord and brain. *dm*, Motor nerve, from the spinal centre for the detrusor muscle. *sm*, Motor nerve from the spinal centre for the sphincter muscle. *b*, Nerve filament proceeding from the brain to the spinal centres of the detrusor and sphincter muscles. An impulse from the brain through *b* inhibits the sphincter centre (dotted line) and excites the detrusor centre. *b″*, Nerve filament proceeding

of incontinence in children, and it may be due to reflex causes (worms, &c.). Spasm of the sphincter is common in hysteria. When the micturition centres are deprived of their functions by disease or injury, the bladder becomes distended and the urine dribbles away. (As this, sometimes, gives rise to the belief that the patient is passing his urine freely, the abdomen should always be carefully palpated).

In paraplegia the urine is often turbid, alkaline, and ammoniacal. Cystitis and nephritis are common, and the latter may be the ultimate cause of death. Bed-sores are also common. The *sphincter ani* is often paralysed along with the vesical changes; and priapism is often present, in cases of paraplegia.

Hemiparaplegia, and **hemianæsthesia**, are conditions which may be produced by injuries, tumours, or sclerosis, of one half of the cord. The symptoms are characterised by paralysis of the same side, below the lesion; with anæsthesia of the *opposite* side. A sclerosis may sometimes, at the beginning, affect only one half of the spinal segment, and then ultimately extend across.

Myelitis—*Acute and Chronic*—**Softening of the Cord.**—In acute inflammation of the spinal cord the central grey matter is often the chief seat of the disease, but it extends to all parts of the cord; hence varieties are described as *acute*, *general*, *central*, *transverse*, *unilateral*, and *disseminated myelitis* (Bramwell). There is first hyperæmia, with extravasations and serous transudations which give a moist and softened appearance to the parts affected. The colour ultimately changes to yellow-white, the nerve elements becoming fatty. The adjacent meningeal membrane becomes thickened and adherent. Microscopically, the cells are seen to be swollen and the reticulum thickened. There is exudation of colloid material around the minute vessels, the capillaries and veins being much dilated. There is granular disintegration of the nerve fibres and ganglion cells. *Softening* of the cord may follow the acute inflammation; but it is a pathological condition which sometimes arises without any previous myelitis. The *chronic* form of myelitis is characterised by the greater development of the neuroglia with thickening of the vessels, and a deposit of numbers of amylaceous bodies with atrophy of the nerve elements. The cord is *firmer* than the normal in its affected parts.

The **causes of myelitis.**—Myelitis may be produced by injuries, and fractures of the vertebræ; and neighbouring inflammations may extend to the cord—as in spinal meningitis, with which myelitis is almost always, more or less, associated. Cancerous, tubercular,

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from the brain to the spinal centres of the sphincter and detrusor muscles. An impulse along *b''* strengthens the sphincter and inhibits the detrusor centre. *b'*, Nerve filament from the brain to the abdominal muscles.

Note.—When the bladder is empty its walls are collapsed. The condition represented in the figure is purely diagrammatic.

and syphilitic inflammations may also extend and affect the cord. Syphilis is believed by Erb to be an important and undoubted cause of myelitis. It is, then, generally the *disseminated* form. Myelitis is sometimes a complication of fevers, and of acute rheumatism. It may also be excited by prolonged functional activity of the cord, as in standing for long periods, muscular fatigue, sexual excesses and abuse; or by exposure to cold and damp—especially the lying for a time upon damp ground. The age at which the greatest number of cases occur, is between ten and thirty-five years; but cases are frequently met with later in life. The *chronic* forms, and *softening* of the cord, are due to the same causes. Defective nutrition may be an additional cause of the latter, when it does not follow an acute inflammation. *Softening* is more apt to follow the acute myelitis; while *sclerosis* results from the chronic forms.

The **symptoms** of a typical case of acute myelitis—when it occurs as a *primary* disease—are ushered in by fever and general *malaise*. Sometimes, however, the spinal symptoms attack the patient at once, and, again, a premonitory stage is sometimes described in which there are derangements of sensation and slight pains in the limbs or back, without much fever. When fully developed there is "girdle sensation," and pain in the back; but the latter is not intense, nor aggravated by movements, unless the associated meningitis be severe, and, then, there is much more soreness developed by percussion of the spine. Pain, or sensations of intense fatigue in the lower limbs, with tingling or formication, sensations of heat or cold, partial anæsthesia, and sometimes hyperæsthesia, are complained of; and also sometimes "bearing down pain" in the bladder and rectum. Priapism is a frequent symptom. Tremors or spasms may precede the paralysis of the lower limbs which soon supervenes and becomes more or less complete, with loss of electro-contractility. The bladder and bowels are often affected by the paralysis, and the urine becomes alkaline and ammoniacal. Cystitis and nephritis may follow. Sensation becomes more or less completely lost up to the middle of the body, where a zone of hyperæsthesia may sometimes be made out.

The reflex activity is, generally, at first increased, but later it is lost. The differences depend upon the site and variety of the lesion within the segment of the cord. The lesion may spread up, or across the cord. When the anterior cornua are affected, there is wasting of the muscles, which present the "re-action of degeneration," and there is a marked tendency to the formation of bed-sores. If the myelitis ascend unusually high the pupil is affected. It is *dilated*, if the sympathetic centres be irritated; and *contracted*, if they be destroyed. The muscles of respiration may become paralysed if the cervical regions be affected, and severe dyspnœa then precedes a fatal termination. Fever may be present, absent, or irregular, throughout. The pulse is quick and often irregular, especially when the cervical region is the seat of the disease. Acute myelitis very rarely extends to the brain. The *course*, obviously, must vary. Complete recovery is only possible when the disease runs a short

course and has not been severe. This is somewhat rare, and more frequently the acute passes into the chronic form, with secondary degenerative changes. *Central* myelitis affects the grey matter and includes the anterior horn. This form runs a rapid course, and death may result from asphyxia—the muscles of respiration being more apt to be affected by the rapidly ascending lesion. Rapid wasting of the muscles, bed-sores, and early loss of reflex action, characterise the central form of myelitis. The *traumatic* form of myelitis is a transverse lesion, usually situated above the dorso-lumbar enlargement. The muscles do not rapidly waste, and the reflexes are exaggerated. In many cases permanent deformities result from contraction and wasting of the paralysed muscles. Death may be due to cystitis and nephritis, or to secondary developments, as pneumonia, bronchitis, &c.

The **differential diagnosis** of acute myelitis and spinal meningitis is tabulated on p. 227 (spinal meningitis); while that of myelitis from other conditions will be considered further on.

The **prognosis** in acute myelitis is generally unfavourable. In mild cases, recovery is possible. Syphilitic cases are more hopeful; but the majority, if they live through the acute stage, end in becoming chronic.

The **symptoms of chronic myelitis** vary with the form. These are the *chronic transverse*, *disseminated*, *peripheral* or *annular*, *focal* (a single patch), and *general* varieties (Bramwell). In most cases the onset is gradual. Disorders of sensation precede the motor disturbances. Numbness, tingling, a feeling of softness under the feet as if walking upon cushions, pain in the back, and "girdle sensation," are some of the first symptoms. A feeling of fatigue or weight in the lower limbs, with slight paresis, constipation, and sometimes a difficulty in emptying the bladder—are also among the early symptoms. As the case progresses there is more or less complete loss of sensibility in the sensory nerves which pass into the affected area of the cord. The impressions of tickling disappear first, and then touch, pressure, temperature, and finally pain (Rosenthal). The sensations in the anæsthetic regions are often strangely altered—as a hot test-tube being supposed to be a cold one, and *vice versâ*. There is often great delay in the transmission of sensations. The sexual functions are lost.

The paresis, or paralysis, almost always extends from below upwards; and its amount, along with the variations in the symptoms, depends upon the seat and extent of the myelitis. Paraplegia is the usual condition, and the paralysed muscles lose their electrical re-actions. The general health is at first good; but ultimately there is complete paralysis, and the development of bed-sores, cystitis, and nephritis—and death may result from these conditions or from intercurrent maladies. There is never true recovery from chronic myelitis, although the morbid processes are often arrested for long periods of time. The progress is very slow.

The **treatment** consists of absolute rest in the *acute* cases of myelitis. The patient should lie upon the side or face. Sinapisms,

ice, or hot douches to the spine, are useful. Leeches may be highly beneficial in the early stages. Quinine may be prescribed, and iodide of potassium is often indicated. In *chronic* cases, Erb recommends the hydropathic treatment, and galvanism. The treatment should also include a consideration of the cause—when possible to remove or relieve it.

From the short sketch given of the pathology of myelitis, it will be seen to be a disease which may affect any part, or parts, of the cord, and the signs will vary according to the seat of the inflammation. According to Hilton Fagge, it is also the commonest disease of the cord, and several authors believe with him, that many of the so-called functional disorders—as reflex paraplegia—are really due to myelitis. For these, and other reasons, it is convenient to regard myelitis as the *type* of spinal cord disease, and a reference to the diagram of the motor and sensory tracts, &c., will assist one to understand the physical signs produced by myelitis, and all other obstructive lesions of the cord. *Paraplegia* (or paresis) is the prominent symptom of this group, and although it occurs as a symptom in other diseases not included here, the description already given applies to all of them. The further advantage of a "paraplegic" (or myelitic) group is the fact that in obscure cases—and there are many—it is difficult or impossible to diagnose the etiological factor. In most cases of spinal cord disease, the diagnosis is made by inference, or by the process of exclusion, and it is often so doubtful that the mind is left with a certain number of possibilities. The diseases excluded from this group are best considered as "special;" or another prominent symptom serves—as *rapid wasting of the muscles*—to classify them more usefully. The adoption of myelitis as the type of this group, is also convenient, as the other members of the group may have their symptoms contrasted, and the differential diagnosis made at the same time. The diseases included in the "paraplegic" group are *compression paraplegia, secondary degenerations, spastic spinal paralysis, and alcoholic, syphilitic, hysterical, and reflex paraplegia.* The neurasthenic group—*spinal weakness, irritation, anæmia, and congestion*—is closely allied to the paraplegic group, clinically; and it may conveniently be considered as part of it.

Compression-Paraplegia.—The most frequent cause is *caries of the spine* (Pott's disease), often producing "angular curvature." It may be simple or scrofulous. *Malignant disease* of the spine is the next in frequency. It often is secondary to malignant disease of other organs. Erosion of the vertebræ by *aneurisms*, and very rarely by *hydatids; hæmorrhages* in and around the cord; *meningeal tumours*, or *gummata*—are also causes of compression-paraplegia. The pathological condition of the cord is that of a transverse myelitis. In old cases there is secondary degeneration of the descending lateral columns.

The *symptoms* frequently begin with indefinite pains, due to irritation of the nerves coming off from the cord about the level of the

lesion. These pains are referred to the parts which the nerves supply, and hence pleurodynia, intercostal or brachial neuralgia, colic, or sciatica. There is usually anæsthesia of these parts, or impaired sensibility, and often hyperæsthesia. Sometimes the muscles are thrown into clonic, or even tonic spasms. These symptoms are more common in malignant disease, and the pain is described as shooting and burning in character. The early symptoms sometimes precede the paraplegia for months, and even longer. The paralysis may occur rapidly, but oftener it develops slowly. It has no tendency to extend up the cord. The reflex contractions are exaggerated. The bladder is often unaffected—except in the late stages when the paraplegia is complete. The sensory impressions are often much slowed in their transmission to the brain. A painful sensation (dysæsthesia) is often complained of, when the leg is only slightly pinched; and it is sometimes referred to the opposite limb. In the *diagnosis* of the different causes of compression-paraplegia, Pott's disease may be observed; but caries of the cervical or lumbar regions may not present any external deformity. The patient should be made to stoop, that the *flexibility* of the spine may be noticed. Hæmorrhages are characterised by *sudden* pain and onset of the symptoms. Malignant disease is more common in the lower, and caries in the upper half of the spinal column. Pain in the back points to a growth; but *rachialgia* and *hysteria* have to be remembered. Meningeal tumours and gummata are often difficult, and to diagnose them from caries, without deformity, and from a simple primary transverse myelitis, must sometimes be impossible. Meningeal tumours are said to be slower in growth, and a case may extend to five years or longer. The average duration of cases of compression-paraplegia is about a year.

The *treatment* consists of mercury and iodide of potassium in suitable cases. The actual cautery, extension, and plaster of Paris jackets in the cases of caries of the spine—are surgical considerations.

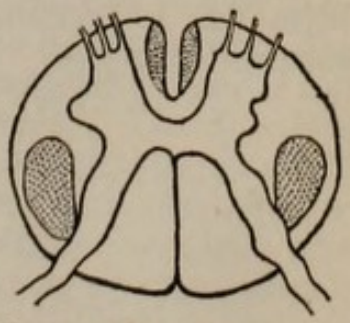

Fig. 36.*— Transverse spinal cord, secondary *descending* degeneration.

Secondary degenerations of the cord are grey degenerations found in certain strands, and which arise from loss of function by disease or injury parting the fibres from their trophic centres. The ascending fibres in the postero-internal and antero-lateral columns may be affected, but these degenerations give rise to no symptoms; but degeneration of the descending fibres in the anterior and lateral columns causes motor weakness and rigidity in the lower limbs, and ultimately contractions and deformity. The rigidity occurs late, and after the paraplegia has existed for some time. *Early* rigidity can only be explained by supposing the spinal (or in the case of cerebral disease, the brain) centres to be in a state of irritation. The knee jerk and reflexes are exaggerated, the reflex arc not being obstructed.

Secondary degeneration may follow transverse myelitis or compression-paraplegia; or it may be due to central disease, and the symptoms proper to those affections will also be present. Secondary degenerations are believed to be permanently incurable.

Spastic Spinal Paralysis—Spastic Paraplegia—Simple Primary Sclerosis of the Lateral Columns.—The sclerosis affects the posterior part of the lateral columns of the cord symmetrically. It is a *rare* primary disease of the cord, affecting adults between the ages of thirty and fifty. It occurs also as a congenital condition, and it is believed to be sometimes due to injuries at birth. The *causes* of primary lateral sclerosis occurring in adults are quite unknown. It affects males more than females. The *symptoms* are very like those of the secondary degenerations, which affect the same tract of fibres in the cord, although in primary lateral sclerosis the disease does not extend in the lateral columns quite so far posteriorly. There are spasms, rigidity, and contraction, with motor weakness, and subsequently paralysis of the lower limbs. The deep reflexes are exaggerated, the knee-jerk and *ankle-clonus* being marked; and the gait is characteristic. The patient is said to walk like a "Highland piper." The legs get soon wearied, and the back may be stiffly arched when taking exercise. The bladder and bowels are unaffected; and there is no pain nor impairment of sensibility. The disease may run a course of ten or fifteen years. The patient may die of phthisis or some intercurrent malady. The *treatment* of primary lateral sclerosis is not satisfactory. Galvanism seems to be the most suitable treatment. Nitrate of silver is often prescribed, and Fagge advocates the use of the extract of calabar bean.

The *differential diagnosis* of primary lateral sclerosis and paraplegia due to a chronic transverse lesion, has been tabulated by Bramwell thus:—

	Primary Lateral Sclerosis.	Secondary Lateral Sclerosis after a Chronic Transverse Lesion of the Cord.
Condition of Muscles.	Motor weakness and rigidity are developed together. The rigidity is usually more marked than the paralysis.	Motor weakness is the first symptom. The rigidity follows the paraplegia. In the earlier stages the paralysis is more marked than the rigidity.
	There is no muscular atrophy at the upper level of the paralysis.	There may be muscular atrophy at the upper level of the lesion.
Onset.	Very slow and gradual.	Usually more rapid.

SENSORY FUNCTIONS.	No sensory disturbances.	More or less or even complete anæsthesia. In cases of slow compression, subjective sensations (shooting pains, &c.) due to pressure on the posterior nerve roots, are present.
THE CONDITION OF THE BLADDER AND RECTUM.	Normal.	The bladder and rectum are usually affected.
TROPHIC CONDITION OF THE SKIN.	No trophic disturbances of the skin.	Trophic disturbances of the skin sometimes occur.

Alcoholic, Syphilitic, Hysterical, and Reflex Paraplegia.—These functional conditions may be considered together. No organic lesions have been found in such affections, although in syphilitic cases the cord has sometimes appeared to be softened. It has been suggested that syphilis affecting the blood vessels of the *pia mater*, by diminishing the lumen, may produce an *anæmia* of the spinal cord, which may be the cause of the paraplegia. Many of the so-called cases of *reflex paraplegia* have been shown to be really cases of myelitis; and some authors consider this form, occurring in cases of chronic visceral disease, to be caused by *extension* of inflammation to the cord rather than by a reflex *inhibitory* action. The *symptoms* in all forms consist of more or less complete paralysis of the lower limbs, with impairment of sensibility. In the *alcoholic* form it is more frequently incomplete (paresis). Constitutional *syphilis* does not affect the spinal cord so frequently as the brain. The functional condition alluded to here does not include compression-paraplegia from syphilitic tumours or growths. It improves with anti-syphilitic remedies. The *hysterical paraplegia* is generally complete when attempts are made at walking. The patient, however, can generally raise the leg when in the recumbent position. There is impaired sensibility, and anæsthesia and hyperæsthesia are often marked. *Reflex paraplegia* may be caused by tight stricture of the urethra; disease of the urethra and bladder; chronic kidney disease; dysentery; intestinal worms; or a displaced uterus, &c. Many other causes are conceivable. When due, reflexly or otherwise, to severe organic disease which cannot be cured or alleviated, this form of paraplegia is not so favourable as the other three members of the group—in all of which a complete cure is possible. The *diagnosis* is chiefly inferred from the history and surroundings, and by exclusion of the other, and more serious members of the paraplegic group. There is no tendency to the formation of bed-sores, no rapid muscle waste, and the bladder and bowels are not paralysed. The *treatment* is considered elsewhere.

Spinal Weakness and Irritation. Spinal Anæmia, and Congestion (?).—There is still a difference of opinion with regard to this group—some authors describing all four conditions as separate and distinct affections, while others are inclined to attribute the symptoms of spinal weakness or irritation to anæmia or congestion of the cord. They all seem to be closely associated—especially the first three—and many authors doubt if such a disease as congestion of the cord really exists.

Spinal Weakness is characterised by muscular fatigue with pains in the legs. The patient complains of feeling easily tired, and occasionally he has feelings of numbness or formication in the feet. "Irritable weakness" of the generative organs is often present. The *causes* are over-fatigue of body and mind, and sexual excesses. The *diagnosis* can only be made after exclusion of all grave organic disease of the cord. The condition is often associated with the hysterical; and the same may be said of spinal irritation.

Spinal irritation or "*rachialgia.*" This affection is often superadded to the preceding one, if it may not be considered a more violent form of it. The additional characteristic symptom is the great pain and tenderness on pressure upon certain regions of the spine. The cervical and dorsal regions are the usual seats of the disorder. It is believed to be of the nature of a neuralgia affecting the posterior spinal roots, and neuralgic affections are often present along with the spinal irritation. The pain is always increased by movement, and a hot sponge passed down the spine may produce intense burning sensations. Some cases are related in which pressure upon the vertebræ has set up such agony as to render the patient insensible. The older writers believed the cause to be congestion; Hammond and others believe it to be associated with *anæmia* of the cord. Uterine disease is a common cause; and certainly females are affected with rachialgia more than males. Railway accidents, and shocks to the spine of any kind, are also causes of this disease.

Anæmia of the Cord.—This affection is believed by many to be the true pathological condition giving rise to the symptoms of the two preceding disorders. It is still doubtful. Paralysis in animals may be produced experimentally by cutting off the blood supply to the cord. Embolism may produce a like result, theoretically; and severe hæmorrhage might give rise to anæmia of the cord. The *symptoms* consist of numbness and formication in the feet, paresis, and sometimes paralysis, with the signs of spinal irritation and weakness.

Congestion of the cord is said sometimes to produce similar symptoms to *anæmia*; and a paralysis due to one or other of these conditions is said to be differentiated by the fact, that in congestion the patient is benefited or relieved by the prone or upright positions, and is much worse if allowed to lie upon his back; while in *anæmia* of the cord the reverse is true, and the patient should be kept as much as possible in the recumbent position. *Concussion* of the spine may produce a congestion of the cord and paraplegia; but this subject is more properly considered in surgical works.

In the *treatment* of the neurasthenic group the severe cases may require absolute rest. The prone position may be adopted in the *congestive* cases, if such be found beneficial. In milder cases the patient is allowed to go about, unless movement excites irritation. A generous diet should be allowed, and wine may be taken in moderation. Moderate exercise and a change of air are to be recommended. Quinine, iron, strychnine, and cod-liver oil are all useful. During the early stages, leeches, or cupping over the spine, are highly beneficial; or blisters may be applied. Morphia may be injected subcutaneously in cases with severe pain. Galvanism of the spinal cord is often useful.

Landry's Acute Ascending Paralysis.—This is a rare disease of the spinal cord, the pathology of which is unknown. Abnormal sensations may at first be complained of, as numbness, formication, and great muscular weakness; but the characteristic symptom is the paresis, then paralysis, of the feet and lower limbs, gradually extending to the body and arms. Swallowing becomes difficult, and death may result in a few hours or days, from asphyxia—the medulla being invaded. A case may extend to ten or twelve days or even longer. Some few cases recover. The bladder and bowels are not paralysed, and bed-sores do not form. There are no symptoms of sensory disturbance, the disease being confined to the motor tracts. There are no spasms in the muscles, and the limbs lie flaccid, although the electrical contractility is always present. The reflexes are present at first, but after a few days they are lost. There is no treatment known to be of any benefit.

Locomotor Ataxia.—The histological changes are similar to those which occur in myelitis, but the disease is limited to the posterior columns of the cord, and extends longitudinally. It is a *sclerosis* of the posterior columns, and it affects chiefly the dorsal and upper lumbar regions of the cord, but occasionally it extends as high as the restiform bodies.

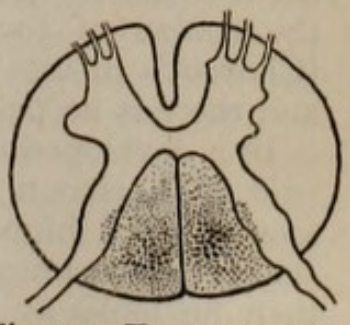

Fig. 37.—Transverse section of cord in locomotor ataxia.

In the lumbar region the *external* divisions of the posterior column are most affected; while higher, and especially if found in the cervical region, the *inner* divisions, or columns of Goll, suffer most. The posterior roots are often, also, diseased; and the sclerosis may extend to the lateral columns. The spinal ganglia and anterior nerve roots escape. Grey degeneration sometimes affects the optic nerves, and sometimes the third and sixth nerves. The joints are sometimes disorganised. The meninges are sometimes (rarely) affected, although the pia mater is frequently found congested, and sometimes thickened and adherent to the cord.

The **causes** of locomotor ataxia are obscure. Severe chills,

sleeping on damp ground, sexual excesses, syphilis, over-fatigue, injuries and concussion, and, especially, hereditary influences of a neurotic character—are all mentioned in the etiology of the disease. It is said, sometimes, to follow acute fevers, diphtheria, acute rheumatism, and pneumonia. It is more frequent in males, and the cases are most numerous between the ages of thirty-five and fifty years. Most authors agree in believing "an inherited tendency to nervous disorders" to be the chief cause of locomotor ataxia; while the conditions mentioned above are, more or less, exciting causes.

The **Symptoms.**—It is important to keep the *early* symptoms of locomotor ataxia well in view, as mistakes are very readily made during the slow and gradual development of the disease. (1) Pains in the lower limbs—frequently ascribed to rheumatism—are very common, and they usher in the more characteristic "lightning" or "toothache-like" pains so often present later. They occur in paroxysms and the patient may be free of them for days, or weeks, at a time. Changes in the weather, over-fatigue, and strong mental emotions, are said to excite them, and they are not always confined to the legs, but sometimes they spread to the trunk and more rarely to the arms. (2) "Gastric crises" are common, consisting of periodic attacks of stomach disorder, *i.e.*, flatulence, vomiting, and lightning pains in the abdomen, with giddiness, palpitation and general *malaise*. The attacks last for a few days at a time. (3) Diplopia, strabismus, nystagmus, inequality of the pupils, impairment of vision, or colour-blindness—are all early symptoms. The ophthalmoscope often reveals atrophy of the disc. The paralysis producing diplopia and strabismus, &c., is often transitory. "Spinal myosis" (extreme contraction of the pupils) is a very common condition, both early and late. The "Argyle-Robertson symptom" consists of insensibility of the pupils to *light*, the contraction with accommodation for near objects, still being present. It may occur as an early symptom and then it generally persists throughout the course of the disease. (4) *Joint affections* may occur early (Charcot's disease). The knee is commonly the first joint affected, and there is no pain, redness, or heat, and no history of an injury.

In a developed case of locomotor ataxia the gait is very characteristic. There is a more or less complete loss of co-ordination. The foot is lifted high, thrown out, and brought to the ground with a stamp. It is most marked when the patient *begins* to walk, and then he looks as if balancing himself, and he requires to keep his eyes open. The gait is made more obvious when the patient is directed to walk along a line—the course taken being very irregular and "shaky."

Sensation is impaired and numbness, formication, anæsthesia, and paræsthesia, are often present. "Girdle sensation" is complained of, not only round the waist, but sometimes felt as a band round a limb. Sometimes it is present as an early symptom. The patient feels the ground soft under his feet or like "walking upon clouds," &c. *Painful* sensation is often absent when simple *tactile* impressions are readily noticed; and sometimes a gentle touch appears

to give rise to severe pain, when the limb is quite analgesic. The impressions are often retarded in their course to the brain. Cold and hot test tubes are generally discriminated, even after common sensation is lost. Sometimes this sense is also impaired. A very striking symptom, and often an early one, is the swaying and inability to balance the body steadily, whenever the eyes are closed. Although this symptom is common in locomotor ataxia it is not pathognomonic, as it occurs in other spinal diseases. The condition is usually present when there is defective sensation in the lower limbs, whether due to locomotor ataxia or to other spinal disease. There is a loss of the muscular sense, and this may be demonstrable in other ways, as when the patient is asked to point to his toe after the leg has been moved—when he often indicates a point far from it. The muscular sense may also be tested by weights. The sense of resistance is reduced or abolished. The contractile power of the muscles is generally normal. Incontinence of urine, and sometimes unconscious passage of the bowels, especially in cases where anæsthesia is marked; and impairment of the sexual functions with nocturnal emissions—are late symptoms. During the early stages the sexual functions are excited—the genital organs being irritable. The knee-jerk (tendon reflex) is almost invariably absent; and this may also be an early symptom. The muscles do not rapidly atrophy, and their electrical re-actions are normal. The subsequent end of locomotor ataxia is often a state of complete paralysis with atrophy of the muscles, bed-sores, cystitis and paralysis of the bladder. Sometimes the disease spreads to the upper extremities, and then after premonitory symptoms of numbness and pain in the fingers, there is loss of co-ordination and inability to use a knife and fork, or to button the clothes, &c. In rare cases the upper extremities are attacked first. Cerebral disease may ultimately develop; but many cases seem to become quiescent for years. Periods of six, twelve, and even twenty or thirty years are quoted as the *duration* of locomotor ataxia. Phthisis is a common intercurrent disease.

The **treatment** recommended by Erb is galvanism of the spine. Absolute *rest* is indicated. Nitrate of silver is believed to be a useful remedy, given with the usual precautions against *argyria*. Iodide of potassium should be given in suitable cases. *Cold water* douches and packs are also recommended by Erb. The application of mustard or the use of blisters, sometimes relieves. Morphia is often required for pain. The general condition may be improved by cod-liver oil and phosphates. Nerve stretching (sciatics) is sometimes tried for relief of pain. Wonderfully good effects have been recorded as to the benefits of *suspension* of the patient by the chin and occiput. The clothing should be warm, flannel being always worn. The diet should be generous, but coffee, tea and alcohol should be given up. Tobacco is hurtful.

The **diagnosis** of locomotor ataxia is often very obscure during the early stages, and care must be taken that *neuralgic* or *rheumatic* pains in the limbs are ascribed to their true cause. The eye symptoms, loss of the knee-jerk, and the inability to stand with

the eyes closed and the feet together, should suggest a thorough examination. *Joint affections* may be mistaken for surgical diseases. In a developed case the diagnosis is usually easy. *Disease of the cerebellum* gives rise to no spinal symptoms, and the gait is staggering, with giddiness, vomiting, and other head symptoms. A *chronic spinal sclerosis*—especially when the posterior columns of the cord are much affected—may simulate a case of locomotor ataxia; but the absence of lightning pains, eye symptoms, &c., and the presence of the knee-jerk—which is usually exaggerated in sclerosis—the gait, and the presence (in multiple sclerosis) of headache, giddiness, tremors, and defective speech, &c., may serve to differentiate these diseases. A very advanced case of locomotor ataxia, with paralysis and muscle atrophy, might lead one to suppose a disease of the motor tracts to be present. *General paralysis* of the insane is excluded by the prominent cerebral symptoms, and the gait.

Spinal Meningitis—*Acute and Chronic.*—The membranes, in the *acute* form, become hyperæmic. There are minute extravasations, and the surrounding tissues are infiltrated with serum. The exudation becomes fibrinous and partly purulent. The roots of the spinal nerves become thickly coated, and they are swollen and softened. The cord participates more or less in the inflammation. In the *chronic* form the membranes become thickened and adherent; and the cord sclerotic, with atrophy of the nerve elements. If the posterior roots be affected, degeneration of the posterior columns may follow (Rosenthal).

The **causes** are:—exposure to cold and damp; injuries and diseases of the vertebræ; bad hygiene in scrofulous subjects; and extension of disease from the brain, or from neighbouring tissues. Spinal meningitis affects males more than females; and it is a disease which occurs most frequently in youth.

The **symptoms** begin with a rise of temperature, headache and nausea, with general *malaise*. The most characteristic symptom is the intense pain in the back shooting up the neck, and into the loins and lower limbs. The body is kept *rigid*, as the slightest movement aggravates the pain. The lower limbs are in a state of spasmodic contraction, and there is often retention of urine. These muscular contractions are increased by attempts at movement; but not by irritating the skin. This symptom helps to differentiate meningitis from tetanus (Jaccoud). There is hyperæsthesia and hyperalgesia in the parts of the skin supplied by the nerves which pass into the affected spinal area. "Girdle sensation" is often present. Later, partial paralysis supervenes, and anæsthesia of the parts which were at first hyperæsthetic. Reflex movements are not abolished. The electro-contractility may be weakened, or it may be normal. The urine is acid throughout. If the meningitis affect the cervical regions, there is dyspnœa, dysphagia, and slowing of the pulse, with weak action of the heart—the respiratory and cardiac functions being disturbed. Death may result within a few hours or days from asphyxia. If such a case affect the medulla there will

also be delirium, vomiting, and ocular derangements, &c. In other cases there is extension of the paralysis, and death from exhaustion in two to four weeks. Some cases recover, and many pass into the chronic form.

In *chronic* spinal meningitis the symptoms are somewhat similar, but the development is slow. The pain is not so acute, and the sensory derangements, rigidity, and spasms, occur in slighter forms and at irregular intervals. Ultimately, there is weakness and fatigue of the muscles; numbness, tingling, and anæsthesia, become more constant; and lastly there is paraplegia. Partial recovery is possible, but, most frequently, permanent lameness results.

The **prognosis** is almost always unfavourable.

The **differential diagnosis** of myelitis and spinal meningitis may be tabulated thus (Bramwell):—

Myelitis.	**Meningitis.**
Pain in the back not prominent.	Pain in the back, increased by movement, is generally very marked.
Shooting pains and hyperæsthesia are seldom prominent. Anæsthesia quickly appears, and is generally well marked.	Shooting pains in the limbs or trunk, and hyperæsthesia are generally prominent features. Anæsthesia occurs later, and is relatively slight.
Paralysis appears early, and is much more prominent than cramps and spasms.	Muscular cramps, spasms, and rigidity of the limbs, and stiffness of the back are more marked than motor paralysis, which is late in appearing.
The sphincters are often paralysed; the urine is often ammoniacal.	The sphincters are not paralysed, the urine is not ammoniacal.
Trophic disturbances of the skin are common.	Trophic disturbances of the skin are rare.
Fever is sometimes considerable, but may be absent.	Fever generally well marked.

The **treatment** of spinal meningitis. In acute cases the local treatment is the same as in acute myelitis. Hypodermic injections of morphia may be necessary for the pain. In the less severe cases, opium may be prescribed, or opium, aconite, and ergot, may be used, as recommended by Bartholow (two minims of the tincture of aconite, five to ten minims of the tincture of opium, with fifteen to thirty minims of the fluid extract of ergot—every two hours during the early stages). Quinine is also useful, in large doses; galvanism and massage are necessary for the paralysed muscles during the stage of convalescence. Iodide of potassium is useful

in the late stages; and it is also indicated in the chronic forms of meningitis.

Poliomyelitis Anterior Acuta—Infantile Paralysis. Acute Atrophic Paralysis (Fagge). (1) *In infants;* (2) *In adults.* This disease has long been known as *infantile paralysis*; but as it is found to occur (very rarely) in adults, the first term is more appropriate, and is the one most generally adopted. [Poliomyelitis: from πολιος, *grey*, and μυελος, *marrow*].

The **pathological condition** found in acute inflammation of the anterior horn of grey matter is of the nature of a "degenerative atrophy." An inch or more in length of this part of the cord may be affected. The *post-mortem* examination reveals no naked eye appearances which can be deemed characteristic, unless the disease have existed for many years. In such cases a shrinking of the anterior part of the cord, on one side, or both, may be obvious when a section is made. Microscopically, delicate connective tissue, corpora amylacea, and a few shrunken cells are found in the affected areas. In the more recent cases, there is *inflammatory softening*, and the multipolar cells have almost entirely disappeared, while free nuclei and granules are present in large numbers. The nerve fibres, in the affected parts, are entirely destroyed.

The **causes** are not well known. It occurs most commonly in children between one and four years of age; but cases are recorded earlier, and later. Teething and chills are sometimes blamed. It often arises during convalescence from fevers, or severe illness; and this is the case, also, in the form occurring in adults.

The **symptoms**, in the child, begin with fever and *malaise*, as a rule, although spasmodic twitchings or convulsions may be the earliest indications. The fever may only last a day or two, and the convulsions may be repeated during the same time. The child is then apparently well, and the attack attributed to teething or other infantile disorder. It is noticed shortly afterwards that the child does not use a limb, or that perhaps even more than one limb is powerless. The limb affected is relaxed and the reflex movements are lost. The faradic re-action is diminished very soon, and it may be absent in a fortnight; and the galvanic current soon yields the "re-action of degeneration." The limbs are often attacked in succession; but the paralysis is always *complete* within a few hours, and there is no progressive advance, nor tendency to bladder complications or formation of bed-sores. The sensory functions are normal, and the paralysed limbs are pale and cold. Recovery may be complete; but this is rare. Partial recovery is the rule, and for some weeks the case looks hopeful. Eventually, however, the improvement ceases, and the affected arm or leg—for in the majority of cases only one limb is paralysed—remains shortened, and undeveloped either in bone or muscle. Contractions and deformities often result, and "club-foot" is a common sequel to infantile paralysis.

In *adults*, the history of a case of poliomyelitis anterior acuta, is

very similar, only it is never ushered in by convulsions, and the limbs are not shortened, although a certain amount of contraction and deformity may follow. In the **diagnosis**, *peripheral paralysis from pressure*, need only be mentioned. The **prognosis** must always be guarded.

The **treatment** consists of the use of electricity—both faradic and galvanic. Great improvement sometimes follows the *long-continued* use of galvanism. A generous diet, with cod-liver oil and iron tonics is indicated; and the deformities may be, in a measure, prevented by the use of bandages and splints.

Poliomyelitis Anterior Subacuta vel Chronica.—Subacute or chronic inflammation of the anterior horns of the grey matter in the spinal cord produces a thickening of the connective tissue, destruction of the multipolar cells, and degenerative atrophy of the cell elements, as in the acute disease. The **causes** are unknown. It occurs most frequently between the ages of thirty and fifty.

The **symptoms** begin with weakness in the lower limbs—both being generally affected. Actual paralysis develops within a few days or weeks. The reflexes are absent. The muscles are relaxed and they waste very rapidly. The electrical re-action is abnormal—the anodal closing contraction being more marked than the cathodal closing contraction, even at the beginning. The faradic re-action is lost. There is little or no anæsthesia, and no bladder complications, nor tendency to the formation of bed-sores. The disease may extend to the arms (and in some exceptional cases it begins with the arms), and a fatal termination may occasionally result from the still further extension of the disease upwards. There is then interference with respiration and deglutition. It is far more frequent that the case ends in complete, or almost complete recovery. The improvement does not commence, however, for about six weeks, and it may be months before the patient regains the perfect use of his limbs. The gradual return of the normal galvanic re-action serves to estimate the rate of recovery.

The **prognosis** should be guarded; but it is hopeful, and recovery is the rule.

In the **diagnosis**, *lead paralysis* sometimes resembles it, and should be noted (see Lead Paralysis).

The **treatment** consists of galvanism. Iodide of potassium is useful sometimes.

Progressive Muscular Atrophy. — The chief **pathological change** consists of atrophy of the multipolar cells in the anterior horns of grey matter in the spinal cord. The neuroglia becomes thickened, and the nerve elements are destroyed. The muscles, supplied by the nerves from the affected area within the cord, undergo rapid atrophy, and the interstices between the atrophied muscles become filled with adipose and connective tissue. The muscles lose their normal red colour and be-

come much paler. In extreme cases they are little more than "bands of white fibrous tissue."

The causes are very uncertain. Over-use of the muscles and extreme fatigue are sometimes blamed. It occurs more frequently in males; and the average age is between thirty and forty years.

The characteristic symptom is the weakness in certain muscles, with generally obvious muscular atrophy. Certain groups of muscles are affected, especially the muscles of the ball of the thumb; and the atrophy gradually extends to the other muscles of the hand and arm. Soon afterwards the disease appears in the other hand, but it does not always progress in a symmetrical manner. The lower limbs are the last, as a rule, to be affected. There is great weakness in the affected muscles, increased by fatigue and cold, and, in the case of the hand, the prehensile power is much diminished. The hand may present the "griffin" or "bird-claw" like deformity. The loss of muscle is sometimes obvious, but not always so, as the development of fat may conceal it. The testing the muscles with the faradic current will prove the absence or presence of muscular tissue; or a small piece of the muscle may be removed with a "harpoon" for microscopical examination. When the lumbar or abdominal muscles are involved there is much hollowing of the back when the patient attempts to stand. Transient "fibrillary twitchings" of the muscles are generally present in progressive muscular atrophy. The sensory functions are normal, and there is no pain or numbness. The rate of progression varies from two years to ten or even twenty. The disease may remain stationary for two or three years. The cases which progress rapidly are more dangerous, as the atrophy frequently extends to the trunk and destroys life by interfering with the respiratory movements. Bronchitis, pneumonia, and phthisis are common intercurrent diseases, which are very fatal when the intercostal muscles or diaphragm are involved. Some cases end in "bulbar paralysis." *Syringomelus*—a hollow space occupying the centre of the cord—is a pathological condition which sometimes gives rise to symptoms identical with progressive muscular atrophy. Extension of the sclerosis to the anterior horns of grey matter in locomotor ataxia, or multiple sclerosis, &c., produces muscular wasting, in addition to the already existing symptoms proper to these diseases.

Charcot has described two additional forms of progressive muscular atrophy. The one he calls *sclérose latérale amyotrophique* and the other *pachyméningite cervicale hypertrophique*. The former consists of a sclerosis of the lateral columns which spreads to the anterior horns of grey matter chiefly in the cervical region. There is combined paralysis and rigidity of the lower limbs, with atrophy of the muscles of the upper limbs. Byrom Bramwell agrees with Charcot in describing amyotrophic lateral sclerosis as a distinct affection; and as it appears to be more rapid in its progress, and invariably fatal by extension to the medulla (bulbar paralysis), it is important to discriminate this form from the simple progressive, and

from the second form mentioned below. The *treatment* is unsatisfactory.

The second form described by Charcot consists of a chronic thickening of the dura mater, which adheres firmly to the cord, and presses upon the nerve roots coming off in the cervical region. There is progressive wasting of the muscles of the upper limbs, and rigidity of the lower limbs. The ulnar and median nerves are especially involved, and the wrist is *extended* instead of flexed.

In this form there is sensory disturbance; and anæsthesia, numbness, and tingling, with pain and rigidity of the neck, generally precede, and continue along with the atrophy. Sometimes "bullous eruptions" arise upon the arms. Charcot considers this form to be sometimes curable; but in the later stages, there is often paralysis of the bladder and rectum, and the formation of bed-sores. This form does not extend to the medulla.

In the **diagnosis** of progressive muscular atrophy—besides the differentiation of Charcot's two forms, and the exclusion of the indiscriminate (transverse) or secondary lesions—the *peripheral paralyses*, *lead paralysis*, and *chronic rheumatism* have to be noted. A knowledge of the anatomical distribution of the nerves will serve to distinguish the first, and the use of electricity will confirm the diagnosis. In peripheral paralysis the re-actions are markedly abnormal. The same tests, *plus* the symptoms of lead poisoning, will exclude lead paralysis.

The *treatment* of progressive muscular atrophy is often unsatisfactory. Faradic and galvanic electricity are both recommended; and in some cases, favourable results have been recorded.

Pseudo-hypertrophic Paralysis.—In this disease the muscles become atrophied, and their substance is replaced by large quantities of adipose and fibrous tissue—sometimes only the latter. The abnormal muscles are red-yellow in appearance, and the fibres may still be recognised. The changes within the anterior horns of grey matter in the spinal cord, which one would expect to find, have not as yet been satisfactorily made out, although a few cases have been recorded in which the nerve cells were found atrophied, and the grey horns disorganised. Many authors believe it to be "primarily an affection of the muscles themselves."

The **causes** are obscure. Heredity seems to play a part in its production, as cases are reported in which several members of a family were affected. It is a disease of childhood, and it affects males more than females.

The **symptoms** begin with motor weakness. After a few months, or even a year, of gradually increasing paralysis, the change in the size of the muscles becomes apparent. The calves generally suffer first, and then the *erector spinæ* in the loins, and the *glutei* muscles. Sometimes the whole of the trunk, and all parts of the limbs are affected. The muscles seem to be enormously increased in size, and the child has an excessively over-developed appearance. When

standing he widens the legs, and projects the abdomen, and the hollow of the back is increased. He frequently falls, and he cannot rise without using his hands, and with apparent effort he "climbs up his knees." The electro-contractility is diminished. There are no tremors, as a rule, in the muscles, and the sensory functions are normal. The mental condition is sometimes defective. The bladder and rectum are not affected. Sometimes the knees are kept flexed, and the *tendo-Achillis* may be contracted (club-foot). The disease sometimes does not advance for a few years; but at last the arms become involved, and complete paralysis is the ultimate result. Death may take place from exhaustion; or from phthisis, which is a common intercurrent disease.

The **prognosis** is always unfavourable.

The **treatment** is not satisfactory. Faradic electricity may be tried; and mechanical appliances may be used to lend support to the weak limbs.

Glosso-labio-laryngeal Paralysis—Progressive Bulbar Paralysis.—In this disease the medulla does not appear much altered, to the naked eye. Sometimes it appears unsymmetrical, or shrunken. There is extreme atrophy of the nerve roots, especially those of the hypoglossal and facial nerves; but sometimes the eighth, sixth, and part of the fifth (motor) are affected. In specially prepared sections, the multipolar cells of the nuclei are found to have undergone degenerative changes, and the nuclei present the appearance of a myelitis in the late stages (sclerosis).

The **causes** are unknown. It is a disease of advanced life, as a rule; and it may be the result of extension of spinal disease upwards, as in progressive muscular atrophy, with which it is occasionally associated.

The **symptoms** begin very insidiously. Headache and giddiness are complained of, and the patient notices that he is apt to "choke" upon his food. Difficulty in utterance soon follows, and the voice acquires a nasal tone from the paralysis of the palate. The *labial* consonants present the greatest difficulty to him. The tongue gradually wastes, and the saliva dribbles from the mouth, which trembles visibly whenever speech is attempted. The mental faculties are, however, quite clear. The voice becomes husky, and it is ultimately lost when the disease extends to the nucleus of the vagus; and there is also, then, occasional suffocative attacks, dyspnœa, irregular action of the heart, and great oppression. The sensibility is not impaired, except in the palate. Taste may be impaired, or absent. The muscles affected present the "re-action of degeneration," and, if far advanced, electro-contractility may be lost. The disease may run a course of from one to five years. Death may result from sudden syncope, starvation, or intercurrent disease, especially pneumonia. The symptoms of bulbar paralysis sometimes resemble those associated with effusions of blood into the pons, acute inflammation, or a tumour. The two former diseases

occur suddenly. The latter develops slowly, but it sets up irritative symptoms from the first, while, in bulbar paralysis, the onset is slow, gradual, and without irritation. Tumours also give rise to other symptoms, as swelling of the eyelids, &c. ; and to ophthalmoscopic appearances (choked disc, &c.). General paralysis of the insane is easy to exclude. In the early stage of bulbar paralysis, the trembling of the lips resembles that which occurs in general paralysis, but the mind is not affected in the former disease.

The **treatment** is unsatisfactory. Faradic and galvanic electricity may be tried. Nitrate of silver is sometimes prescribed. Feeding by means of the stomach tube, or by nutrient enemata, may become necessary.

An *acute form of bulbar paralysis* is described, which is generally fatal within a few days. It is caused by inflammation of the medulla, and changes take place similar to myelitis, within, and limited to the bulb. Cases have recovered. The treatment is the same as in myelitis.

Multiple or Disseminated Sclerosis.—This is a disease which affects the brain as well as the spinal cord. It consists of the formation of irregular or rounded patches of sclerosed tissue, which feel hard, and are of a yellow-grey colour. Microscopically, the patches present an appearance similar to those changes found in chronic myelitis. In the brain they are found scattered through the white matter of the hemispheres, while in the cord any part may be affected.

The **causes** are not clear. It occurs with most frequency between the ages of twenty and thirty years.

The **symptoms** are very variable, and depend upon the seat of the sclerosis. The characteristic symptoms of a case in which it is general are *tremors* ; *slow, accented and "scanning" speech ;* and *nystagmus.* The tremors are only present when movements are attempted, and they are especially marked when the patient is asked to lift a glass of water to his lips. The hand trembles violently, and the water is generally spilled. The nystagmus is only marked when the patient is directed to look at different objects in rotation. It is not ceaseless, and it may not be observed when the eyes are at rest. The mental faculties are not bright, and subjects suffering from this disease are liable to attacks of *stupor*—sometimes followed by transitory hemiplegia. The patient is inclined to be emotional, and this aggravates the trembling movements. Constipation is the rule, and headaches and vertigo are almost invariably complained of. The other symptoms depend upon the seat of the sclerosis in the spinal cord. Subjective sensations (numbness, tingling, &c.) are often present. Anæsthesia, or impairment of tactile sensation, is not usually present. The reflexes are increased when the lateral columns alone are affected, and the limbs then become rigid and the gait *spastic.* The symptoms peculiar to locomotor ataxia will be present when the posterior columns are sclerosed, but there is no loss of sexual power. The eye symptoms, described as occurring in locomotor ataxia, may be present. The muscles, in exceptional cases,

may become atrophied. The *course* is slow and irregular—five to ten years being the usual duration, and death generally results from some intercurrent affection, as pneumonia or phthisis.

The **treatment** is not satisfactory. Electricity may be tried. Strychnine, arsenic, or nitrate of silver may be prescribed.

In the **diagnosis**, *mercurial poisoning* may give rise to tremors not unlike those which occur in multiple sclerosis; but the history will exclude this disease. Locomotor ataxia must be noted (see Diagnosis, page 225). The tremors in *paralysis agitans* can sometimes be diminished or arrested at will—the movement being otherwise *continuous*. *Hysterical* affections may sometimes simulate a case of multiple sclerosis in some of its symptoms. When the tremors and cephalic symptoms are absent, the other diseases of the spinal cord must be differentiated by careful examination of the motor and sensory tracts, &c. In the early stages it sometimes is impossible to make a confident diagnosis.

Paralysis agitans, or shaking palsy, is a nervous disease which consists of oscillatory movements affecting chiefly the limbs, but sometimes also the neck and tongue. It generally begins in the arm, and gradually spreads to the leg, and then to the limbs of the other side. The head often shakes, but there is no continuous movement of the eyeballs (nystagmus). At first it occurs in paroxysms, and the movements may be controlled. The limbs are quiet during sleep, but later the palsy is constant. The movements are much aggravated by emotion or excitement of any kind. The electro-contractility is unimpaired, except that the muscles get quickly exhausted. The speech is *jerky* and slow. There is often pain in the limbs. The head is carried forward, and the gait is hurried as if to prevent a fall. Sometimes the patient when starting to walk finds himself impelled to run backwards. In the later stages there is rigidity of the muscles, with paralysis. The duration is very chronic, and paralysis agitans is essentially a disease of the old, but sometimes (rarely) it affects the young. It is a *functional* disorder; but, sometimes, patches of sclerosis are found in the brain.

The **treatment** is not satisfactory. Prolonged rest of the limbs during the early stages should be tried. Chloride of barium (in one grain doses) is recommended; and arsenic sometimes produces improvement.*

* It may be of use to the reader, and an aid to the memory in the examination of a case of spinal cord disease, to note the order and classification of the foregoing diseases. The "paraplegic" group comprises twelve diseases; then follow three *special* diseases, viz.:—*Landry's acute ascending paralysis*, *locomotor ataxia*, and *meningitis*; the group of four diseases associated with *muscular atrophy*; and lastly, three diseases associated with *trembling* of the muscles.

CHAPTER XI.

DISEASES OF THE NERVOUS SYSTEM.—Section II.

Contents. — Cerebral hæmorrhage; Apoplexy; Occlusion of the cerebral vessels—*i.e.*, *thrombosis*, *embolism*, and *syphilitic disease;* Hemiplegia; Aphasia—Intra-cranial tumours—Cerebral abscess—Meningitis; acute, chronic, and tubercular; Pachymeningitis—Cerebral congestion and anæmia—Sunstroke—Chronic hydrocephalus—Epilepsy—Hysteria; Catalepsy; and Hypochondriasis—Chorea—Writer's cramp; Athetosis; Singultus or hiccough—Meniere's disease—Migraine—Tetanus—Hydrophobia—**The differential diagnosis of brain diseases.**

Cerebral Hæmorrhage; Apoplexy; Occlusion of the Cerebral Vessels—Thrombosis, Embolism, and Syphilitic Disease. — These diseases may with clinical advantage be considered together, as they form a group in which the symptoms are very similar, and the points of difference may afterwards be stated. In *cerebral hæmorrhage* there is rupture of a blood-vessel in the brain—the most frequent site being the internal capsule, corpus striatum, lenticular nucleus, or optic thalamus. More rarely, it occurs in the cerebellum, pons, or medulla. The symptoms arise from the damage done to these, and the neighbouring parts. The clot may vary in size, from a pea to a large walnut, but in extensive ruptures the fluid blood often diffuses into the ventricles. The clot undergoes changes, and there may be secondary inflammation of the surrounding cerebral tissue. Ultimately a spongy connective tissue is developed, which encloses the clot—in those cases which survive. Small clots may become absorbed, and large clots—instead of becoming enclosed and thus promoting a favourable termination—often light up general inflammation. In chronic cases when the tissues have been much broken up, "atrophic degenerative" changes take place in the descending fibres of the nerve tissue, through the *crus* and *pons* to the spinal cord (*secondary degeneration*).

The commonest *cause* of cerebral hæmorrhage is disease of the cerebral blood-vessels—*atheroma* and *miliary aneurisms;* and any sudden increase of the blood pressure may produce the rupture. The latter may arise in hypertrophy of the heart and gout; cirrhosis of the kidney; muscular effort; the use of stimulants; hot and cold baths; and violent emotions. The tendency to *atheroma* is often inherited; and it rarely occurs before the age of forty years.

In *thrombosis* and *embolism*, there is occlusion of a blood-vessel by the formation of a clot, in the former disease—while in the latter, a small clot, concretion, or particle of fibrin (embolus) is carried up in the circulation and becomes impacted. Emboli may be derived

from thrombi developed elsewhere in the circulation, from atheromatous disease, generally; and most commonly they are derived from diseased valves of the heart. The left carotid, from its position is more liable than the right to receive these emboli. The embolus generally passes up to the left Sylvian artery, in which it becomes

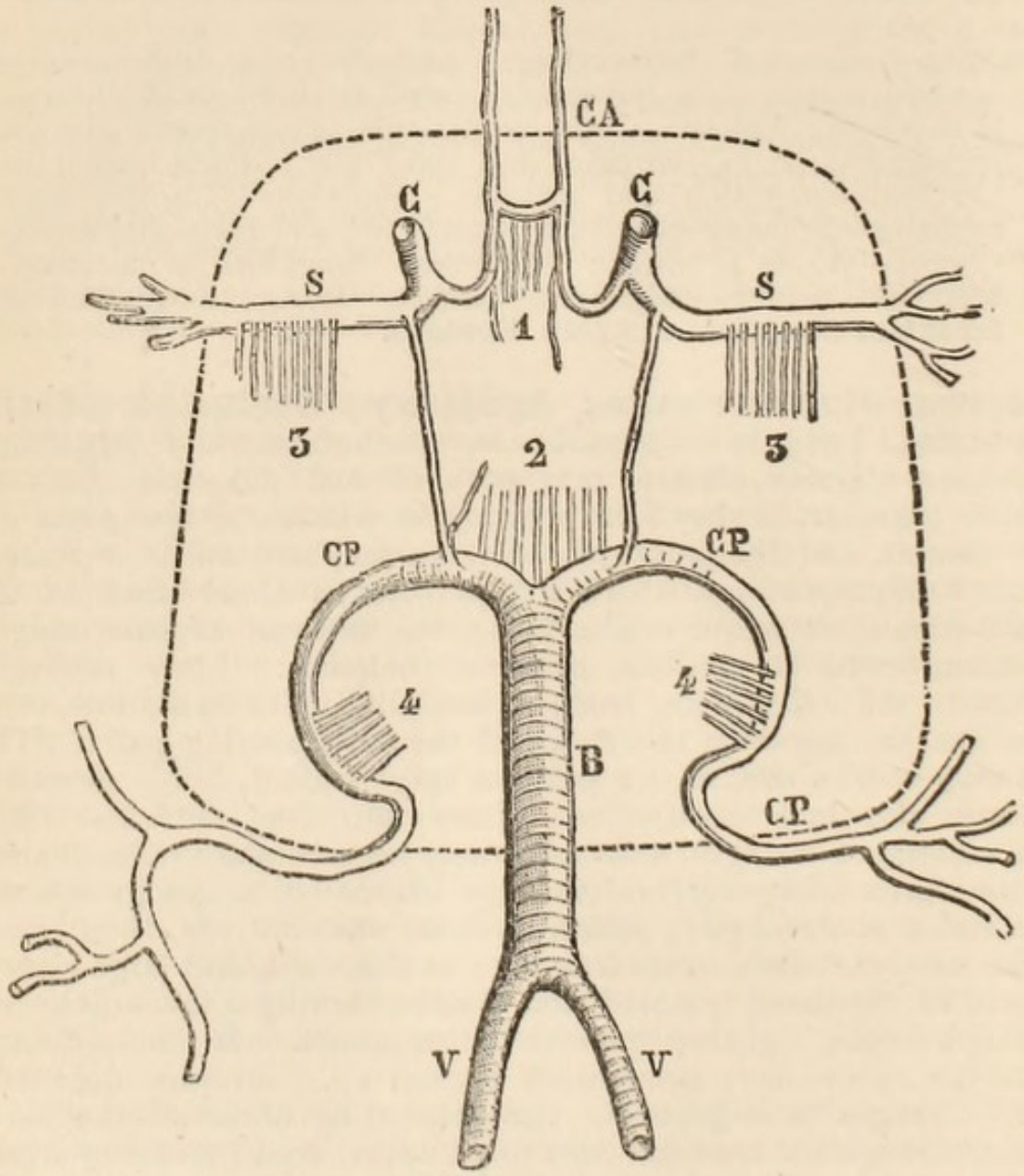

Fig. 38.—Arteries of the base of the brain, or circle of Willis.—C, C, Internal carotids; CA, anterior cerebral; S, S, Sylvian arteries; V, V, vertebrals; B, basilar; CP, posterior cerebrals; 1, 2, 3, 3, 4, 4, groups of nutrient arteries. The dotted line shows the limit of the ganglionic area. (From *Charcot.*)

impacted and hence the greater frequency of right hemiplegia. The vertebral arteries are rarely entered by emboli. The effect of the clots is to cut off the blood supply to the brain in the regions corresponding to the distribution of the blood-vessels. It is important to note that the brain is supplied by *two sets*, viz. :—(1) the blood-vessels to the *cortex* which have a free anastomosis; and (2) the

blood-vessels to the *centres* which are terminal. The plugging of the latter produces a complete anæmia, while in the former the circulation may be soon restored by the collateral branches.

The *antero-median* group (1) supplies the anterior part of the head of the caudate nucleus. The *postero-median* group (2) supplies the internal surfaces of the optic thalami and the walls of the third ventricle. The *antero-lateral* groups (3, 3) supply the corpora striata, the anterior parts of the optic thalami, and the internal capsules. (One of these nutrient arteries is larger than the others, and is the "lenticulo-striate artery" or "artery of cerebral hæmorrhage." It terminates in the anterior part of the caudate nucleus after crossing the upper part of the internal capsule). The *postero-lateral* (4, 4) supply large parts of the optic thalami.

The *anterior cerebral* curves round the corpus callosum, and supplies the gyrus rectus and the supraorbital lobe, the first and second frontal convolutions, the upper part of the ascending frontal, and the inner surface of the hemisphere as far as the quadrate lobe. The *posterior cerebral* goes to the region of the occipital lobe and the inferior aspect of the temporal lobe. The *middle cerebral*, or *Sylvian* artery, divides into four branches, which go to the posterior parts of the frontal lobes, the ascending frontal, and to all the parietal lobes,—*i.e.*, chiefly to the motor areas, the angular gyrus, and to the first temporo-sphenoidal lobule. Thus the *anterior cerebral* supplies the prefrontal area, and a small part of the motor area—viz., that for the leg centre in the paracentral lobule and upper end of the ascending frontal (and perhaps that for the trunk). The *posterior cerebral* supplies the centre for vision, and that connected with the course of the posterior part of the optic expansion, and also the sensory part of the internal capsule. The *middle cerebral* supplies the motor areas of the cortex (except part of the leg centre), and the basal ganglia (in part), the auditory centre, and that for speech.

Occlusion of a *terminal* blood-vessel results in *softening* of the brain tissue supplied by the vessel. "White or yellow softening" results from a complete block of the vessel by coagulation of the blood beyond the embolus, and the consequent prevention of a backward flow from the capillaries. The tissues affected become yellow from fatty deposition in the nerve cells. "Red softening" occurs when there is no such coagulation. A hyperæmia is then produced, and the tissues are coloured by the "diapedesis of the red blood corpuscles." Minute extravasations often occur from rupture of the capillaries. In a few weeks the "red softening" becomes "yellow softening," from the transformation of the hæmoglobin, and fatty degeneration of the nerve elements. When death occurs rapidly the brain tissue may appear quite normal and there is no softening.

The *causes* of thrombosis are chronic endarteritis; pressure by tumours, &c.; and weakening of the blood current, such as occurs in the aged, and in those suffering from chronic wasting disease. The most common source of emboli is the heart. Endocarditis; clots within the auricle, from wasting disease; fatty heart; and aortic

aneurisms—are the conditions which may give rise to them. *Syphilitic* disease of the cerebral arteries produces its symptoms from occlusion of the blood-vessels by the gradual thickening of their coats.

The **symptoms** common to this group of cerebral diseases are the symptoms which are associated with **apoplexy, hemiplegia,** and **aphasia.** Any one or all of these conditions may occur as a result of cerebral hæmorrhage, thrombosis, embolism, or syphilitic disease of the cerebral arteries ; but it is better to describe them separately, as they often require (clinically) to be regarded as substantive affections, the actual causes of which can only be inferred.

Apoplexy is not always sudden ; but the "warnings" are often very indefinite. Epistaxis, and hæmorrhages into the conjunctivæ, sometimes occur several months before a "shock." The temper becomes irritable, and the speech and memory are frequently defective for some time previously, and many complain of slight paresis, numbness, or tingling in an arm or leg. Giddiness, diplopia, and headaches come somewhat nearer to the actual shock, and often are the immediate forerunners; while the other prodromata may be supposed to be due to diseased blood-vessels or to very small ruptures. In a typical apoplectic seizure there is generally violent pain in the head, and in a few minutes the patient becomes comatose. Sometimes the headache is complained of for a longer period, but the insensibility is rarely delayed beyond a few hours, if it be about to supervene at all. Sometimes, again, there is no warning, and the patient falls with a loud cry ; or there may only be nausea and vomiting, and the unconsciousness comes on very slowly and after a few hours have elapsed. The face may be flushed or pale. The breathing is at first stertorous, with puffing out of the cheeks, and soon frothy saliva appears at the lips. The conjunctivæ are insensible, and the pupils may be contracted, dilated, or unequal. When the lesion is situated in the pons, the pupils are often extremely contracted (pin-points), and the respirations are very slow. The eyes and head are often turned to the side on which the lesion is situated in the brain (*conjugate deviation*). This symptom is probably due to the lesion exciting "rotatory movements," such as occur in animals when one side of the brain is injured. The pulse is often full and tense, and sometimes very slow. The forehead and body are generally bathed in perspiration. Sometimes apoplexy begins with convulsions, and the amount of insensibility varies much. If incomplete, pinching the limbs may reveal that only one is drawn away ; or, on raising the arms, one may fall helplessly back, while the other remains rigid. Sometimes, however, there is no hemiplegia. In apoplexy, the temperature at first falls, and it remains low in rapidly fatal cases ; but in a few hours it rises two or three degrees, and keeps up when inflammatory changes supervene. It may reach 107° Fahr. shortly before death. If recovery be about to take place the temperature falls again within three days. A pulse below sixty is a bad sign, and so also is a very rapid or irregular pulse. The breathing may be of the Cheyne-

Stokes character, and just before death it becomes extremely slow, with long intervals. The *duration* of a case varies from a few minutes to two, eight, and even fourteen days or longer. Consciousness may return partially, and may be followed by drowsiness or delirium; but these cases may still terminate fatally. Sometimes consciousness is completely regained; and if this should not occur within forty-eight hours, and be accompanied by other favourable symptoms—death may be expected within the periods above-mentioned. Death may result from pneumonia or œdema of the lungs.

Hemiplegia is the result of unilateral disease of the brain. The leg, side of body, arm, and face, are more or less paralysed upon the opposite side from the lesion in the brain. The face often escapes, and there is never the complete paralysis which occurs in peripheral lesions of the seventh nerve. The eye can be closed voluntarily—and this cannot be done in a case of Bell's paralysis. Sometimes, however, he cannot wink with the affected eye (paralysis of the *orbicularis palpebrarum*). The mouth may be only drawn slightly to the sound side. The tongue sometimes cannot be protruded beyond the teeth; but when this is possible, the tip generally points to the paralysed side. The speech is defective, even when the intelligence is not much impaired. Almost always both leg and arm are paralysed; but sometimes the arm alone may be affected. The chest muscles of the damaged side do not act so well. Some amount of anæsthesia and hyperalgesia is believed to be present in the early stages of hemiplegia, but, if so, it quickly disappears. As a rule, the mental condition during the onset is not favourable to the testing of the sensory nerves. In a recent hemiplegia the limbs are usually redder, and they may be raised in temperature (1° Fahr.). The damaged limbs are liable to sweat and to become œdematous. Sometimes bed-sores form. The electrical re-actions vary, and they are often normal. "Early rigidity" may be present, with flexion of the fingers, hand, and elbow. This condition is believed to be due to laceration and irritation of the brain tissues, or to *inhibition*. In the later stages, the muscles contract, and there is much deformity. The hand and arm is flexed, and the nails may even pierce the skin of the palm. It often requires much force, and it gives much pain, to straighten them. This "late rigidity" is due to the secondary degenerations in the lateral tracts of the spinal cord irritating the sound nerve fibres which are connected with the sclerosed columns. The reflexes are increased. Spasms and tremors are common in the paralysed limbs; and "post-hemiplegic chorea" is a less frequent after-effect. The facial nerve of the opposite (sound) side is sometimes paralysed (crossed hemiplegia), and this occurs when the pons is the seat of the lesion. If the upper part of one lateral half of the pons be the seat of a lesion destroying the facial nucleus, there is also a crossed paralysis, and probably, then, the adjacent nucleus of the sixth nerve also suffers, and there will be a paralysis of the external rectus as well. Extreme disease of the pons may cause a paralysis of both facial nerves. If there be paralysis of the third nerve along with the facial, the lesion is in the *crus*.

Charcot states that when a lesion is confined to the grey nuclei of the corpus striatum the paralysis is transitory and incomplete ; when it involves the internal capsule, it is complete and permanent. The anterior two-thirds of the internal capsule contain the motor fibres, and those to the leg lie nearer the median line. Probably, this is why the leg recovers first, as these fibres are more likely to escape complete destruction. The posterior third of the internal capsule contains the sensory fibres, and lesions affecting this part give rise to anæsthesia, hyperalgesia, and loss of tactile sensibility. Sight, hearing, and sense of smell, may also be affected, and Jackson says there is often lateral hemianopsia.

Hemiplegia usually follows apoplexy, although not always. In some cases there is no antecedent apoplexy, but merely a confusion of mind followed by paralysis. Transitory attacks sometimes usher in the severe form, and lesions of the cortex sometimes give rise to temporary paralysis, which disappears with the establishment of the collateral circulation in the part affected. This cannot occur when terminal vessels to the basal ganglia are plugged. An embolus may plug the central artery to the retina and cause blindness ; and coarse lesions may light up *double optic neuritis*. The mental functions in chronic cases of hemplegia are much impaired, as a rule.

Aphasia.—Under this head, are grouped several affections concerned with the loss of speech and memory. *Aphasia* means the inability to use spoken language, and it is "the outgoing language and motor processes that are interfered with." If it be due to a *loss of memory* for words, it is said to be *amnesic aphasia*. *Alalia* means inability to articulate—as in bulbar paralysis, &c. *Agraphia* —another form, in which there is inability to recognise, or form the written characters—may co-exist with the aphasia, and sometimes it is present alone. The power of expression by signs may also be lost, sometimes. The memory for written language is often present, and aphasic patients read the papers, sometimes over and over again. A good test of his understanding is to read a book aloud with him, and ask him to turn over at the proper place. The *third left frontal*, or *Broca's convolution*, including a part of the *second left frontal convolution*—is the seat of the memory and language centres. This area is supplied by the middle cerebral artery, which is so very often the seat of obstruction by an embolus, and hence the frequent association of right hemiplegia with aphasia. Aphasia may be the result of embolism, thrombosis, hæmorrhage, inflammation and abscess, and tumour ; or it may be due to mental conditions. *Embolism* is the commonest cause. To explain the absence of aphasia when the internal capsule and descending pathways are much injured, Dr. Broadbent thinks there must be two routes for language, and suggests that the commissural fibres probably connect Broca's convolution with the corresponding parts on the other side, and hence the impulses travel to the pons and medulla by this route. In left-handed persons, aphasia has been known to occur from a lesion of the *right* third frontal convolution.

The intelligence in aphasic patients may be unaffected, although, most frequently, there is mental weakness, especially when the aphasia is associated with hemiplegia. The sense of smell is sometimes lost. The patient is often unconscious of his mistakes; but sometimes the half-amused and half-annoyed expression which follows the use of a wrong word, shows that the intelligence is not impaired. Aphasia sometimes disappears, or is cured, before the associated hemiplegia; or, again, it remains after the patient has recovered the use of his limbs. When due to syphilis a cure may be expected; and in young patients, *training* and education may produce good results. The longer aphasia exists, the less is the prospect of recovery.

The **differential diagnosis** of each member of this group may now be considered—leaving the general diagnosis of apoplexy, &c., from other diseases, until the end of the section. *Thrombosis* is somewhat rare; and it occurs in the aged and very feeble, and the onset is *gradual*. To decide between cerebral hæmorrhage as a cause of *apoplexy*, and embolism and syphilis—the age is an important point. Apoplexy, occurring in a patient over fifty years, is probably due to hæmorrhage; while the younger the patient the greater is the probability of its being due to embolism or syphilis. To differentiate the latter two, a careful examination must be made for evidences of syphilis; and the heart must be examined for endocarditis, and the liver, spleen, and kidneys for enlargements (infarctions). Heart disease is the commonest source of emboli. It should be noted that embolism and cerebral hæmorrhage may co-exist.

In a case of *hemiplegia* without loss of consciousness, the probability is that it is due to obstruction of blood-vessels—embolism, syphilis, or atheromatous disease. The attack may be gradual or sudden, generally the latter. The association of hemiplegia with aphasia points to obstruction of the Sylvian artery, and it may be due to any one of the three causes just stated. The same careful examination as before, is here necessary. In syphilis the symptoms are apt to be irregular, but atheroma of the blood-vessels may also be general. In syphilis a peculiar somnolent condition, and a "sulky disposition" are described. He resents examination. The cranial nerves are often affected, and the paralysis often leaves one limb and attacks another. Such cases may have paralysis at one time and not at another. Sometimes, there is only a drowsy condition, or headache, loss of memory, vomiting, &c., with no hemiplegia. Hemianæsthesia—when the posterior third of the internal capsule is involved—is generally due to hæmorrhage; but it may be due to extensive atheromatous disease. In the history of a case of hemiplegia it is important to know if it began with apoplexy. The more marked the coma, and the longer the duration and severity of the initial state, the more likely is it to have been due to rupture of a blood-vessel—especially if occurring after fifty years of age.

The **treatment** of cerebral apoplexy—whether due to hæmorrhage or to obstruction of the blood-vessels—is quite the same. Should there be any warnings of a seizure, venesection may be

practised, or leeches may be applied to the mastoid processes. An active purge may be administered. When the shock has occurred the head should be raised, and an ice-cap worn; while mustard should be applied to the stomach or lower extremities. The room should be darkened, and the utmost quiet maintained. Two drops of croton oil may be given, and it is the most convenient purgative for such cases, as it may be dropped upon a small piece of sugar, and placed far back upon the tongue and swallowed unconsciously. Tincture of aconite may be given in one minim doses, every ten minutes, to reduce arterial tension. Bromide of ammonium or potassium, should be prescribed in ten or twenty grain doses, when the patient is very restless. Should the patient recover consciousness, the bromides should be continued for some time, and then ℞ 55 may be ordered. In the later stages, galvanism and faradic electricity with massage, &c., should be commenced. The diet should be very light and digestible. Tonics may be given. Stimulants must never be used in the early stages.

Intra-Cranial Tumours.—These include aneurisms, sarcomata and gliomata, &c., hydatids, and cancerous, tubercular, and syphilitic tumours.

The **symptoms.** Headache is a prominent symptom, and the seat of the pain may sometimes indicate the situation of the tumour. The pain is often paroxysmal, and it is worse upon coughing, and upon taking a deep inspiration. The scalp may be tender at certain points. The headache may at times be so agonising as to cause delirium. Vertigo is very commonly present, and especially when the cerebellum is the seat of the tumour. Vomiting and constipation are present in most cases, and if the vomiting be persistent there is loss of flesh and emaciation in extreme cases. Epileptiform convulsions are common to all tumours of the brain; but Reynolds believes convulsions to be more frequently present in tumours affecting the posterior lobes, or the cerebellum. The pulse is often slow. The sight is often affected, and as the vision may be good at the centre, yet defective at the periphery, the visual field should be tested in all directions. It varies in amount from a mere mistiness to actual blindness; but the blindness often comes on suddenly, even although the ophthalmoscope has for some time revealed the changes mentioned below. These changes consist of the conditions known as "choked disc," optic neuritis, and atrophy of the disc. The "choked disc" probably arises when "the fluid, which is always present in small quantity in the arachnoid cavity, is driven into the optic nerve . . . . whenever the intra-cranial pressure is from any cause increased." Optic neuritis may be secondary to a choked disc, or it may arise from extension of inflammation downwards. Atrophy of the disc follows these pathological changes, but sometimes it occurs as a chronic change without antecedent neuritis or choked disc. (See the diagrams of these conditions in Gower's or Liebreich's atlas, and compare with the normal). The mental state often becomes changed, and there is loss

of memory, irritability, depression, and stupor. Many cases become maniacal, and require to be removed to an asylum.

The foregoing symptoms are common to all intra-cranial tumours; but they are often accompanied by other signs which enable them to be more exactly localised.

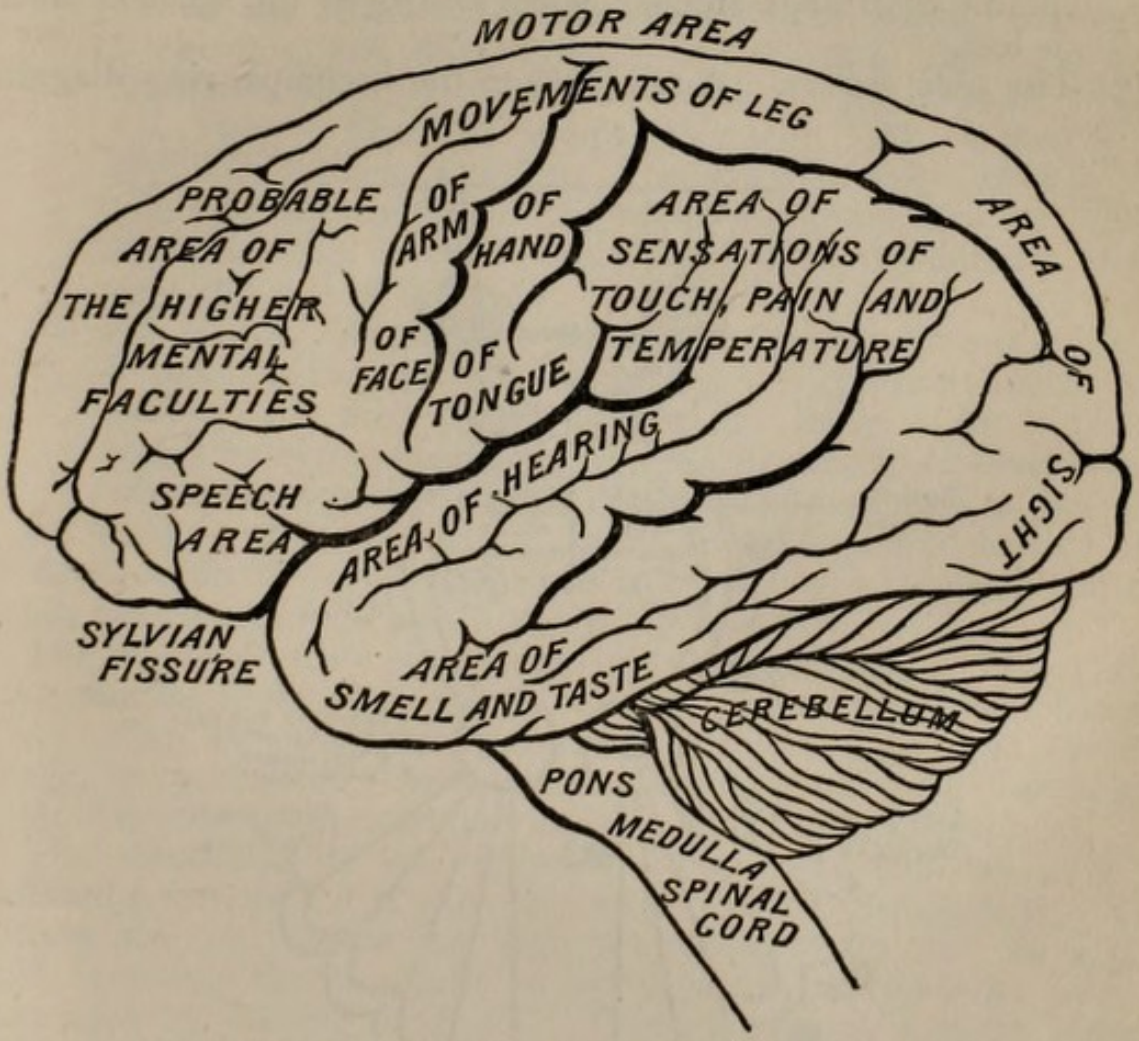

Fig. 39.—The brain areas. (From *Ranney*).

(1) Tumours affecting the cortex may produce spasms and convulsive movements (Jacksonian epilepsy) in the hand or arm of the opposite side, and if situated in the posterior lobes there may be disorders of sensation. One would expect unilateral blindness and deafness in disease of the angular gyrus, or superior sphenoidal convolution; and loss of taste and smell when the under surface of the temporo-sphenoidal lobe was affected.

(2) Tumours affecting the motor-tract, corpus striatum, optic thalamus, &c., will produce spasms and contractions in the limbs of the opposite side when the lesion is *irritative*, but paralysis when the lesion destroys the parts without irritation.

(3) Aphasia may result from a tumour affecting Broca's convolution and the immediate neighbourhood.

(4) Tumours of the medulla affect the speech, deglutition, and respiration, &c.

(5) Tumours of the cerebellum cause loss of co-ordination, and there is a reeling, staggering gait. The vertigo is generally marked, and convulsions, optic neuritis, and blindness are common symptoms.

(6) The *olfactory nerve.* When irritated, there is often a complaint of strange smells; when destroyed the sense of smell is lost.

(7) The *optic nerves.* A reference to the accompanying diagram

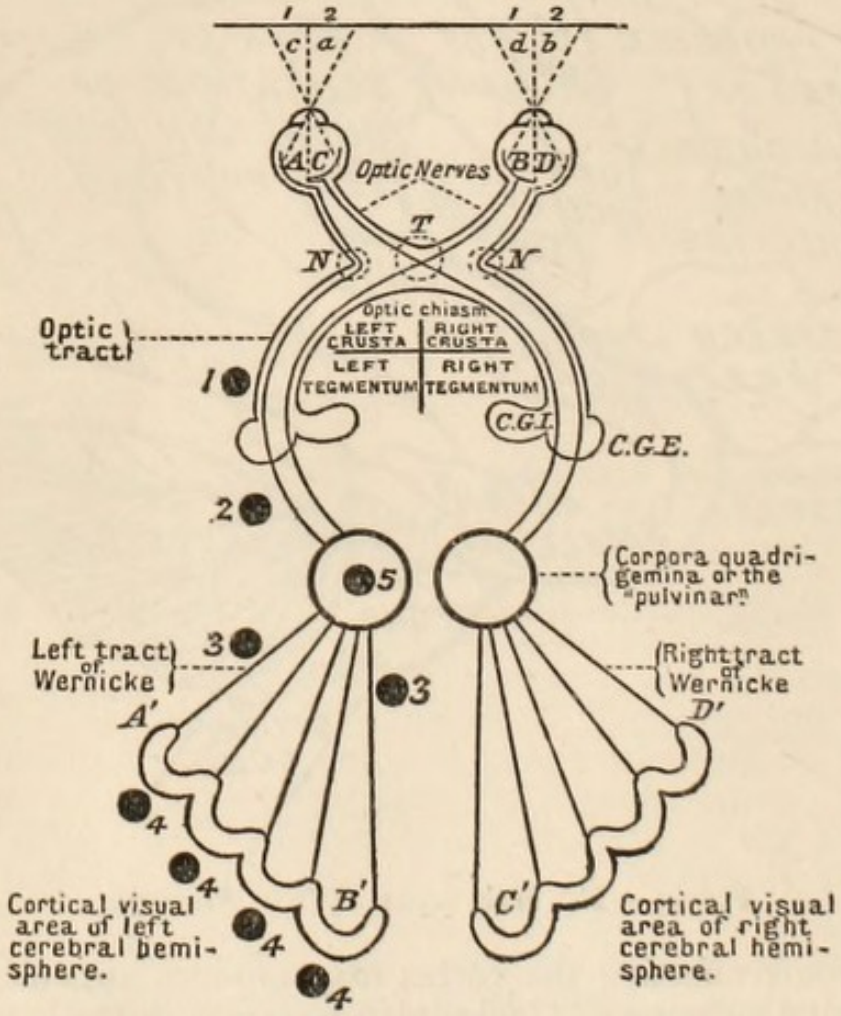

Fig. 40.—A diagram of the optic tract, to explain hemianopsia. The rays, *a b* and *c d*, from the objects in front of the eyes (1, 2), fall upon the temporal and nasal halves of the retina, as indicated, at the capital letters A B and C D. A and D to A′ and D′ indicate the course of the fibres from the temporal halves of the retina; while B and C to B′ and C′ show the course of the nasal fibres—the important difference being the decussation of the latter at the optic chiasma. From this point backwards the fibres (both temporal and nasal) pursue the same course, passing through the optic tract, external geniculate body, corpora quadrigemina or "pulvinar" of the optic thalamus, and the internal capsule to the occipital lobes. A and B to A′ and B′ indicate the fibres associated with the left cerebral hemisphere; while C and D to C′ and D′ indicate the fibres of the right. A lesion, involving the tract at 1, 2, 3, 4, or 5, produces blindness of the temporal half of the left eye (A), and the nasal half of the right (B)—*i.e.*, *right lateral hemianopsia (homonymous).* A lesion at T produces blindness of the nasal halves of both eyes—*i.e.*, *bi-temporal hemianopsia*; while two lesions at N N (very rare) will produce blindness of the temporal halves—*i.e.*, *double nasal hemianopsia.* The lesions at T and N N are *heteronymous.* [From *Ranney* (modified).]

will explain the various defects of vision possible by pressure upon the chiasma, or part of the optic tract.

(8) The *third nerve*. A tumour of the *crus*, when *irritative*, gives rise to nystagmus in the eyeball of the same side, and spasm or rigidity in the muscles of the opposite side of the body. When the lesion is *destructive* there is ptosis, convergent strabismus, and dilated pupil, in the eye of the same side, and paralysis or paresis of the muscles of the opposite side of the body. The eye symptoms occur very frequently in syphilitic lesions. Lesions of the corpora quadrigemina also affect the movements of the eyes; and there may be double optic neuritis, and sometimes paralysis of the muscles of the opposite side of the body.

(9) The *fourth, fifth, and sixth nerves*, when implicated, point to a lesion of the *pons*, and there may be disorders—motor and sensory—of the opposite side of the body. An *irritative* lesion involving the *fifth* nerve will give rise to *tic-douloureux*, while a *destructive* lesion will produce anæsthesia of the face.

In tumours at the base of the brain, *retinitis* occurs earlier; and pressure upon the cavernous sinus will produce a fulness about the orbit, swelling of the eyelids, and often epistaxis.

The *course* of intra-cranial tumours is very indefinite. They may take months to develop, and the early symptoms are often obscure. They often terminate in convulsions and coma, cerebral hæmorrhage, or meningitis. An aneurismal tumour may be relieved, and syphilitic tumours are certainly curable under proper treatment.

The **diagnosis** of the different forms of tumours can only be inferred; and often it is quite impossible to know their nature. The history and constitution may suggest a correct diagnosis. In syphilitic tumours, the headache is worse at night, and the pain is increased by tapping the head. There is often a state of somnolence present, and the paretic or paralytic symptoms which may be present are liable to disappear and return, affecting often different limbs. The **treatment** should consist of the administration of large doses of iodide of potassium (and bromides) in *all* cases.

Cerebral Abscess.—This disease is seldom, if ever, primary; but it follows injuries, diseases of the ear (especially chronic suppuration of the tympanum, &c.), chronic disease of the nose, and general pyæmia. In pyæmia the abscesses are generally multiple and small. When secondary to disease of the ear and nose, or the result of an injury, they are generally single and larger. They vary in size from a pea to a hen's egg.

The **symptoms** vary much. In pyæmia the symptoms of blood poisoning generally mask the symptoms of suppuration in the brain. In other forms, severe pain in the head, noises in the ears, nausea, vomiting, and epileptiform seizures, are present. The temperature rises to 103° Fahr. The pulse is sometimes exceedingly slow. The pupils sometimes act sluggishly. Rigors are not always present; and the fever is intermittent in type. When the abscess is

developed, the symptoms of compression, and the means of localisation, are the same as in intra-cranial tumours. Death may be ushered in by delirium, epileptiform fits, stupor and coma. There may be a prodromal stage lasting a few weeks or months. When acute inflammation begins the case terminates in about a week. The **diagnosis** generally rests upon the association of one or other of the causes with the above symptoms; and the **treatment** is the same as in meningitis. Surgical procedures may save life in some cases.

Meningitis.—Tubercular: Acute and Chronic Meningitis.—Pachymeningitis.—Tubercular meningitis is an inflammation of the membranes of the brain produced by the development of tubercular granulations. The tubercles vary in size, from very minute points to a pin's head, and they are grey-white in colour. They are more numerous around the arteries at the base, but they are also distributed along the course of the arteries of the convexity of the brain. A sero-purulent effusion accompanies the tubercles. The membranes themselves are thickened and opaque, and there is increased effusion into the ventricles, and often œdema of the cortex.

The **causes** of tubercular meningitis are favoured by bad hygienic conditions. The disease occurs in those who have inherited the tubercular *diathesis;* and it is common between the ages of two and six years, and between twenty and thirty. The *type* of child apt to be affected is described as "the pale, thin-skinned, blue-eyed child of pale, flabby, and delicate parents."

The **symptoms** are sometimes divided into stages. In the *prodromal* stage, which may last for three months, there is irritability, loss of appetite and sleep, and emaciation. The child grinds his teeth, or cries out in the night. There is often a frown upon the brows. Headache and vertigo is complained of, and there is often vomiting without apparent cause. The bowels are irregular, and the abdomen is apt to be swollen. *Double vision* may be an early sign.

The stage of *excitation* is characterised by fever (102° or 103° F.), which is remittent in type. The pulse is irregular and subject to great variations in frequency. It often becomes very slow. The headache, vomiting, and constipation is now marked. The headache is subject to exacerbations, and it is increased by exposure to strong light. There is increased fretfulness, and all movements cause pain. The head and neck are generally held rigid, and other muscles are in a state of spasm. Convulsions are common. The *hydrocephalic cry*, or shriek, is often heard at this stage. The period of excitation lasts one or two weeks. *Tache cérébrale* is the name given by Trousseau to a symptom of tubercular meningitis. It consists of a bright red line produced by gently scratching the skin. It appears quicker, remains longer, and is more marked than a similar line produced in a healthy child. Before the actual development of the stage of depression, there is a period in which excitation and depression exist alternately. This stage may last

from one to three weeks. There are paroxysms of pain and convulsions, alternating with periods of somnolence and torpor. Strabismus, double vision, inequality of the pupils, and retinal changes —are prominent symptoms at this period. The ophthalmoscopic examination reveals the *choked disc* and *atrophy*. Tubercles are often detected in the choroid.

Sometimes there is now an improvement; but it is almost invariably followed by the stage of *depression*. In this, there is delirium, convulsions, and gradually increasing unconsciousness. The pupils dilate, and there is often nystagmus. The coma deepens, and the respirations become more and more shallow. The breathing may be of the Cheyne-Stokes type. The pulse—which has been irregular and slow—becomes rapid and feeble before death. The stage of depression may last one or two weeks. A tubercular meningitis, arising as a secondary disease (as in pulmonary tuberculosis) has no prodromal symptoms. There is intense headache and delirium, but no convulsions (in adults).

In **simple acute meningitis** there is intense hyperæmia, followed by a purulent and fibrinous exudation, over the base and convexity of the brain. The ventricles may be distended with fluid. Primary meningitis is somewhat rare. It may arise from excessive mental efforts, sunstroke, or alcoholism. The secondary forms are caused by extension of disease from the bones, as in ear disease; or by injuries; or it may be secondary to erysipelas, or pyæmia. It sometimes occurs during the course of Bright's disease, acute rheumatism, or puerperal fever. The **symptoms** begin with a chill, the temperature rising to 103° or 104° Fahr. The face is flushed, and the eyes are injected. Headache and vertigo, nausea and vomiting, are prominent symptoms. There is delirium, with illusions and hallucinations. In the earlier stages there is hyperæsthesia of the skin, and spasms of the muscles of the neck and the extremities. Irritation of the cranial nerves accounts for the eye disturbances. Later, the symptoms of depression supervene, and there is somnolence, coma, and paralysis. The ophthalmoscopic changes are the same as in the tubercular form; but no tubercles are found in the choroid. The duration is about one to eight weeks—the condition during that period being very variable. It usually terminates fatally, but sometimes there is recovery. The **diagnosis** in relation to other diseases is considered later. The differentiation from tubercular meningitis requires the consideration of the history of the onset, the presence or absence of tubercles in the choroid or other organs, the age, the causes, and the *diathesis*.

In **chronic meningitis** the membranes are much thickened, and adherent to the brain, and the cranial nerves are often involved. These conditions are frequent in old cases of mania and dementia. Injuries to the head and chronic alcoholism are supposed causes. The **symptoms** are often associated with mental disorders. Headache, vertigo, double vision, ringing in the ears, rigidity of the neck, and neuralgia of the face, or spasms of the ocular or facial muscles, are common. In the later stages, there is mental weakness; and

paresis or paralysis of groups of muscles, with irregular breathing, and a weak pulse, may ultimately follow.

Pachymeningitis—Hæmatoma of the dura mater.—In this disease there is first hyperæmia of the meninges, with an exudation which develops into a new membraneous formation. Hæmorrhages take place, from the rupture of the thin walls of the blood-vessels in the new membrane, and ultimately the new formation has the appearance of a cyst containing fibrinous matter and broken down blood-clots. The **causes** are blows and injuries to the head; and chronic alcoholism, Bright's disease, cirrhosis of the liver, and heart disease, are said to be predisposing conditions. The **symptoms** are not characteristic, and the diagnosis from cerebral hæmorrhage and embolism, &c., is the chief consideration and difficulty. There is headache, vertigo, and *tinnitus aurium*, and the pupils are often much contracted. These symptoms are often followed by apoplexy, from which the patient may not recover, and the condition may be (clinically) indistinguishable from cerebral hæmorrhage, &c. Should the patient recover from the comatose state, there is still headache and somnolence, and if the lesion be on one side there is paresis or convulsive movements in the limbs of the opposite side of the body, and possibly contraction of one pupil only. Hemiplegia may ultimately result. Sometimes there is fever; and the pulse is usually weak and rapid. The first stage may last a few days, or longer, and if death do not take place with the apoplexy, the secondary changes may run a course of a few weeks—rarely a few months. There is always mental weakness left. The **treatment** is the same as in cerebral congestion and hæmorrhage.

The Treatment of Meningitis.—The head should be raised, and an ice-cap should be used after the head has been shaved. Leeches may be applied to the mastoids, in the severe acute forms. A quick purgative should be administered. Tincture of aconite in small doses (one minim, well diluted, frequently repeated) may be useful in robust patients with increased arterial tension. Quinine is indicated, in large doses, if an antipyretic be required. A cold pack-sheet may be used when the temperature is high. Bromide of potassium, in doses according to the age, is the drug most generally used. Some authors prescribe small doses of mercury; but others condemn the practice. Small doses of opium are recommended when the bromide fails to soothe. Iodide of potassium may be combined with the bromide in the tubercular forms. The bromide and iodide should be continued for a time. Mustard may he applied to the neck. The room should be kept cool, quiet, and dark. The nourishment can only be fluid, and should consist largely of milk and beef-tea.

Cerebral Congestion and Anæmia.—Cerebral congestion may be caused by sunstroke; long continued mental work with sleeplessness; alcoholic excesses; and by the use of certain drugs—as belladonna, opium, &c. A *passive* form of congestion

may be the result of heart disease, or of obstruction to the blood-vessels by tumours—intrathoracic or cervical.

The **symptoms** in mild cases, consist of headache aggravated by light and noise; singing in the ears; sleeplessness; irritability of temper; and often there is stomach disorder with nausea and vomiting. The pulse is quick and irregular.

The severer forms have these symptoms intensified, and they are accompanied by hallucinations and illusions, and sometimes by mild delirium. Neuralgia is common, and tingling and numbness in the lower limbs may be complained of. The head feels hot, and the eyes are suffused. An *apoplectic* form is described, in which the patient loses consciousness and presents all the appearances of apoplexy. Sometimes there is only a temporary confusion of mind, without unconsciousness. The symptoms of the *passive* form are not so pronounced. An ophthalmoscopic examination reveals the retinal veins enlarged, aud swelling of the optic disc. The membrana tympani is congested. Surface thermometers reveal a slight rise in the temperature of the scalp.

The *duration* of the mild cases is very short; and under treatment both the mild and severe types are generally cured in from three days to a fortnight. Sometimes, however, the severe type is apt to recur, and a radical change in the occupation of the patient, or a prolonged rest from all mental work becomes necessary. The *apoplectic* form may usher in cerebral hæmorrhage; and it is only known from this disease by the fact that recovery takes place very soon without hemiplegia, &c. The *passive* forms vary in their duration and course with their causes.

Cerebral anæmia may result from severe hæmorrhages, chronic wasting diseases, and heart disease affecting the circulation.

The **symptoms** of acute anæmia are such as characterise an attack of *syncope*. Convulsions may be superadded. In the slow *chronic* form the symptoms are similar to those of congestion; but the history of the causes, the presence of anæmia, the low temperature of the scalp, and the ophthalmoscopic and otoscopic examinations revealing the absence of hyperæmia—will serve to differentiate the two conditions. The course and duration depend upon the cause. Simple anæmic cases recover under treatment.

The **treatment** of cerebral congestion consists of raising the head high, and applying ice. The feet should be kept warm. Leeches may be applied to the mastoid processes. A brisk purgative should be given. Tincture of aconite, and bromide of potassium are the best remedies. The diet should be light, and all stimulants, including tea and coffee, should be forbidden. The *cause* should be removed. The substitution of light manual labour for the mental work, is highly beneficial. In *cerebral anæmia* the treatment of the syncope has already been indicated. In the chronic forms, iron, arsenic, and strychnine are useful. Morphia may be required for sleeplessness and delirium.

Sunstroke or **Heatstroke.**—The only pathological change

found is general congestion of the viscera. *Meningitis* may sometimes follow sunstroke. The *symptoms* vary in different cases. A "cardiac" form is described, in which sudden syncope, dimness of vision, vertigo, and continued prostration, are the prominent symptoms. The face is pale, and nausea and vomiting are frequent. The pulse is feeble and often slow. Death may occur as a sudden result of this form of sunstroke; but many cases recover.

The attack may begin in another way—*i.e.*, giddiness, headache and general *malaise*—then delirium and convulsions, and gradually coma, supervene. The temperature is often very high (106° or 107° F.), and the pulse rapid. These cases are generally fatal—death taking place within a few hours. In the cases which recover, there is, generally, a great liability to headache; and sometimes mental weakness, and epilepsy, are direct results of sunstroke.

The **treatment** consists of the application of ice to the head and cold douches to the neck and chest. Quinine should be prescribed. In failure of the circulation brandy must be allowed. Bromides are indicated for the headaches.

Chronic Hydrocephalus.—The **causes** of chronic hydrocephalus are obscure. The rachitic constitution is an important factor in its development; and a chronic inflammation of the ependyma may lead to the effusion. A mechanical compression of the straight sinus, or vein of Galen, by a tumour, &c., may also cause effusion. The fluid is straw-coloured and may amount to as much as sixteen ounces, or more in extreme cases. The ventricles of the brain are much distended, and the motor centres are depressed and flattened. The condition is often *congenital*.

The **symptoms** are generally marked. The immense size of the head—which is held up only with difficulty—the large forehead with the drooping eyes, and the apparent lack of intelligence, at once make the case obvious. The fontanelles are large, and often pulsating. The appetite is often voracious. There is usually paralysis with rigidity of the lower limbs. Epileptiform fits are common. The *duration* is variable; but many cases die in one year, while others, again, survive for fifteen and twenty years, or even longer. Convulsions and coma usually terminate the case ultimately. The **treatment** consists of supporting the strength. Iodide of potassium may be tried; and *tapping* sometimes gives good results.

Epilepsy.—The **pathology** of epilepsy is still obscure. Tumours of the brain, thickening of the membranes, and changes in the shape and contour of the cranial bones, are frequently found; but these appear to be accidental alterations. The arteries of the brain are sometimes diseased, and it has been suggested that epilepsy may be due to sudden spasm of the cerebral blood-vessels. Nothing is known of the **causes** of epilepsy beyond the fact that it is hereditary, and that it occurs in families in which the neurotic diathesis is strongly marked. Irritation of peripheral nerves, dentition, alcoholism, sexual excesses, and injuries to the head—are exciting

causes. The convulsions may also be excited by strong emotions. The greatest number of cases develop at puberty.

The **symptoms** depend upon the form. Two forms are described: *epilepsia gravior*, the severe epilepsy with convulsions (*le grand mal*); and *epilepsia mitior*, the mild epilepsy (*le petit mal*). The first form is the common epilepsy, and the convulsions may occur with, or without warning. In many cases there is a marked *aura*, such as a sensation of heat or cold stealing up the arm or leg, and sometimes the attack may be warded off by grasping the limb tightly. At other times the *aura* consists of strange smells, illusions, and hallucinations, or of spasms in a limb. Headache, or giddiness, or indefinite pains referred to the stomach, with despondency and irritability, are common a few hours before the seizure. When the attack occurs, there is loss of consciousness, sometimes a sudden cry, and the patient falls in the convulsion. There is at first great pallor, and the muscles are in a state of *tetanic rigidity*. The reflex functions are abolished. Respiration is suspended, and the head and neck are held stiffly; the jaws are closed and the limbs are extended. The face becomes blue, in consequence of the venous stasis. In a minute or so, the *clonic spasms* begin. The face, lips, larynx, &c., twitch, and the eyes roll about. Sometimes the tongue is severely bitten, and the froth at the mouth may be bloody from this cause. Hæmorrhages may occur into the conjunctivæ and eyelids, and sometimes severe bruises, and even fractures may result from the violent convulsions. The presence of these conditions in an early case, without other symptoms, should suggest *nocturnal* attacks, sometimes. The clonic stage lasts for a few minutes, and then generally the patient passes into a state of somnolence, for a few hours, from which he can only be partially aroused. The pupils, formerly dilated, are now contracted. Sometimes consciousness returns at once, and the patient looks around with a dazed look—quite unaware of what has happened. Sometimes the epileptic fit is followed by a state of excitement, in which the patient is violent and quarrelsome, and during which he may even commit homicidal acts. One attack may be succeeded by another, or by several; but more usually there is only one seizure. An *apoplectic* form is described by Jaccoud, which is characterised by the greater length of the comatose stage, and by temporary—sometimes more permanent—paralysis.

The mild form of epilepsy—*le petit mal*—is not associated with convulsions, but it is characterised by very short attacks of *absence*, or complete forgetfulness. The attack comes on suddenly with pallor of the face and dilatation of the pupils. It may be so slight as to attract little attention, and the patient is quite unconscious of any defect. Sometimes, instead of *absence*, there is a sharp attack of vertigo, with loss of consciousness for a second or two, and generally some mild convulsive movements. Jackson does not consider *unconsciousness* to be necessary, to constitute a case of epilepsy. Local convulsions may take place without unconsciousness. "Masked" epilepsies are described, in which attacks of

neuralgia, or transient delirium, with "insane impulse," take the place of, or alternate with the convulsions.

The *course* of epilepsy is very chronic, and it may terminate in a state of complete dementia. The attacks may be prevented, in many cases, by avoiding the exciting causes. *Le petit mal* is said to affect the mind more quickly than the common epilepsy—loss of memory occurring early in the mild form. The removal of a reflex cause does not always cure the epilepsy even when due primarily to reflex irritation. A *status epilepticus* may be induced.

Hystero-epilepsy is a combination which is frequently met with in practice. The patient is generally an epileptic, primarily, and the hysterical condition has developed later.

In the **treatment** of epilepsy, nothing can be done for the seizure beyond loosening the clothes at the neck, introducing a soft roll of cloth between the teeth to prevent biting of the tongue, and seeing that the patient is in a position to prevent his injuring himself. In some cases, when the *aura* starts in the arm the patient may prevent a fit by tightly grasping the limb. Bromide of potassium is the best remedy for epilepsy. It may be given in large doses (from thirty to sixty grains or more in the day). Anæsthesia of the fauces should be produced, and the dose should be graduated so as to reach this point. The drug should be continued with a gradually diminishing dose, for a long period—perhaps one or even two years. A few drops of liquor arsenicalis may be given daily to prevent *bromism*. Inhalation of nitrite of amyl may sometimes avert an attack. A dose of chloral at night may likewise be useful in averting nocturnal seizures. The diet should be regulated, and tea, coffee, alcohol, and tobacco should be forbidden. In all cases of epilepsy a careful search should be made for cicatrices, or marks of injury to the head. In the latter case trephining may be of the utmost service to the patient. Cicatrices involving a nerve, and the source of an *aura*, should be dissected out. Reflex causes, as worms, &c., should be searched for, and treated. Nitrate of silver in small doses, and with due regard to its staining powers, is another remedy sometimes prescribed; but the continued use of bromide of potassium has given by far the best results.

Hysteria. Catalepsy. Hypochondriasis.—This group, in a work of this kind, may be dismissed with a few words, although, in practice, the first and last of these affections are very common. They are all functional disorders, and hysteria is characterised by motor and sensory disturbances of the most varied and irregular description.

Hysteria occurs most frequently in females; but sometimes in males. The age at which it appears is commonly about puberty, and it may exist, more or less, up to the climacteric period. It is not necessarily associated with disorders of the sexual organs. It should be regarded as a *neurosis*, in which there is an unstable condition of the nervous matter. An hysterical attack consists of noisy and vehement excitement in which tears and laughter alternate.

The patient is at one moment sad and at another joyful. All sorts of sensory derangements are complained of. Sensations of cold and heat, numbness and tingling, palpitations and "globus hystericus," with flatulence and other stomachic symptoms, are only a few of the many possible subjective sensations. A severe form of headache—*clavus hystericus*—is a common complaint, and is described as like a nail being driven into the head. Flashes of light, ringing in the ears, and strange smells; spots of anæsthesia, hyperæsthesia and analgesia; hysterical joints, coccydynia, &c.—are common hysterical affections. All sorts of paralyses are simulated, and dysphagia, aphonia, hemiplegia, and especially paraplegia, are often very perplexing. The urine is pale and watery, and paralysis of the bladder with retention is a very common hysterical disorder. The hysterical fit consists of a very brief stage of tonic rigidity followed by clonic spasms. There is screaming and no loss of consciousness; and the reflexes are not abolished. The patient may pass into a state of *ecstasy* without fits. The important diagnostic points in hysteria are considered later, with other nervous diseases, at the end of the section.

In **catalepsy** there is a paralysis of the cerebral functions apparently, but no actual unconsciousness. It occurs in young hysterical subjects, and consists of rigidity of the limbs, for hours or days, in all sorts of odd positions. The mind is in abeyance and the muscular system—the whole or in part—is in a state of spasm. The face is pale and the eyes open and staring. The breathing is shallow, and the pulse small and weak. Sometimes tactile sensation, pain, and reflex action are diminished or abolished. The attack generally passes away, but relapses are common.

In **hypochondriasis** the patient believes, without cause, that he is suffering from bodily disease. The pains which arise in such an individual always seem to be of the most agonising description. Cancer, heart disease, phthisis, and especially diseases of the genital organs, are a few examples of the many disorders which hypochondriacal patients appear continually to dread. Some slight pain or symptom arises, and at once his mind is concentrated upon himself, and he fears the worst. A careful examination should be made in all such cases, as organic disease may be present. The mental condition is one of great misery and depression, leading ultimately to permanent melancholia, and insanity in extreme cases, if the patient should not succeed in throwing out of his mind the habit of watching his health.

The **treatment** of hysteria is often very difficult. In the young, prophylactic measures should be carried out by the parents whenever the neurotic temperament is manifest. Regular hours, exercise, and feeding, should be inculcated, and the habit of self-control exercised from the beginning. The moral sense must be trained, and all improper literature must be carefully withheld. In the treatment of hysterical fits, a calm and apparently unsympathetic manner, may do much to restore the patient's self-possession. A large dose of bromide of potassium may be useful, and it should be

continued, in smaller doses, should the patient be markedly hysterical. Guarana, coca, as well as bromides, are useful for hysterical headaches. In anæmic girls, iron, arsenic, and the phosphates, or cod-liver oil—are indicated. In some women asafœtida or valerian may be used when there is much flatulence, or when attacked with "globus hystericus." An inhalation of chloroform may be used in severe cases, and morphia may be injected hypodermically. Electricity is of value for the various forms of paralysis; and combined with Mitchell's massage treatment, good and permanent effects are produced, in suitable cases. The *cataleptic* condition may, if protracted, require forced feeding. *Hypochondriasis* must be treated as a mental disorder.

Chorea, or St. Vitus' dance, is a functional nervous affection. Some authors believe it to be due to emboli irritating the nerve centres. Others consider it to be an excitement or irritation within the corpus striatum. It is often associated with acute rheumatism. The age at which it is common is during the period of the second dentition, and at puberty. In mild cases, only one group of muscles may be affected, and sometimes the jerking or twitching is not obvious until the patient is directed to make some movement. When asked to show the tongue it is projected suddenly, and as suddenly withdrawn. At other times there is continual "fidgets." Chorea may begin gradually, and it may arise from imitation of some peculiar movement observed in other children. Sometimes, and especially when caused by a sudden fright, it begins suddenly. In severe cases, all the voluntary muscles are involved. The limbs are thrown about continually, the face is contorted, the eyes roll and squint, and the fingers are twisted into every conceivable shape. The skin becomes excoriated, and the limbs are often bruised; the breathing is sighing and spasmodic; the heart's action is tumultuous, and a mitral murmur is usually present. The pulse is exceedingly rapid, irregular, and weak. There is often tenderness of the spine, and hyperæsthesia of the skin. The reflexes are increased. When the spasmodic movements are limited to one side, it is known as *hemichorea.* The *course* of the mild cases is very chronic, but ultimately there is recovery. The same cannot be said of the acute and severe forms, as death generally takes place from exhaustion in about a fortnight.

The exciting **causes** are worms, sexual abuses, frights and strong emotions, and pregnancy; while anæmia and amenorrhœa, &c., are favourable conditions for the development of chorea. The cases which occur during pregnancy are more dangerous, and the chorea—if recovered from—is apt to recur in subsequent conceptions.

The **treatment** consists of the removal of all exciting causes and reflexes, when possible. Absolute rest and quiet, with a well-regulated light diet—afterwards increased to a full generous diet—will often cure a mild case of chorea without other remedies. Arsenic, however, is of the highest value in chorea. The neural

sedatives may be required, and bromide of potassium, succus conii, chloral and opium are all used. In the severe cases, inhalations of chloroform often become necessary. Large doses of morphia are sometimes given. Trousseau advocates the use of large doses of strychnine. The anæmic patients should be prescribed iron, phosphates, and cod-liver oil.

Writer's cramp, and other allied affections, need only be shortly noticed. These disorders are brought about by over-use of the muscles. There is no paralysis, and the fingers may be used for all other work. Fatigue and pain are felt in the fingers and forearm. Sometimes the attempt to write produces a tonic spasm. Again, there is loss of power or weakness, with fibrillary trembling. *Pianist's cramp* may involve both hands. The *treatment* in these affections is to order prolonged rest. Galvanism and massage are the best remedies. Easton's syrup, hypophosphites, or cod-liver oil should be prescribed, if the system have run down.

Athetosis is a chronic condition sometimes associated with old standing paralysis. There is constant movement of the fingers or toes. The patient is unable to keep a finger still in any position in which it may be placed.

Singultus, or hiccough, is generally caused by indigestion. Sometimes it is a very formidable affection to treat, and it ushers in a fatal termination in many cases of severe organic disease. Bartholow recommends a sudden, strong faradic shock through the diaphragm, just as the spasm is about to occur. Inhalation of ether or chloroform, and the injection of morphia, hypodermically, should be tried.

Meniere's disease is an auditory vertigo, with deafness, produced by a sudden exudation, or hæmorrhage, into the semi-circular canals. The attacks may at first be short, but ultimately they become more or less constant, with exacerbations. The giddiness is often extreme, and it consists of a sensation of whirling to the *right or left—or vertically*—and sometimes it is likened to sea-sickness, and accompanied by nausea and vomiting. Noises in the ear are almost always complained of. Some cases seem to improve, and are free for long intervals. Quinine seems to be the best treatment.

Hemicrania, migraine, or sick headache, is believed to be due to changes in the circulation. There is paroxysmal unilateral headache, which seems to be sometimes periodic, and accompanied by nausea and vomiting. The supraorbital region is the part most affected. The sight is disordered in a peculiar way. It may only be dim; but frequently a zig-zagged outline or patch appears to obstruct the vision. The face is sometimes pale and sometimes flushed during an attack. Strong light and noise aggravate the pain. There is often tenderness upon pressure over the cervical ganglia. The paroxysms may last for a few hours or a day or two. The disease is more common in women than in men. The *treatment*

consists in regulating the diet. Arsenic, caffeine, and bromide of potassium are useful remedies. Galvanism is sometimes used, with good results.

Tetanus consists of violent paroxysmal and tonic contractions of the muscles of the body, and it is produced by an increased excitability of the reflex function of the spinal cord.

The *causes* are usually lacerated wounds or injuries, especially of the extremities and when a nerve is involved. *Cold* appears, sometimes, to be an exciting cause. *Strychnine* poisoning produces symptoms similar to tetanus.

[**Tetany** is another form, sometimes described as a separate affection. The tonic spasms are generally confined to the distal parts of the extremities.]

The *symptoms* of tetanus consist of sudden and paroxysmal contraction of the muscles, the jaws being first affected (*trismus*). The lips are retracted (*risus sardonicus*). *Opisthotonos*, *emprosthotonos* or *pleurosthotonos*, are conditions arising from the muscular contraction. The first is the commonest, and it occurs when the spinal muscles are firmly contracted so as to arch the back, and the body rests upon the head and the heels. Emprosthotonos is the opposite condition —*i.e.*, the body is bent forward; and pleurosthotonos is lateral inclination. The two latter conditions are rare. The paroxysms are excited by movements and by noise, and a slight touch may produce a seizure. There is often difficulty in swallowing. Feverish symptoms develop later; and at the beginning the wound looks irritable.

If very acute, death may result from interference with the respiratory muscles. In milder forms, rest and nourishment may be taken between the attacks. The *prognosis* may be favourable if the spasms be not severe, and if the intervals between them get gradually longer. The *treatment* consists of removing any cause of reflex irritation. Wounds and cicatrices must be searched or opened for splinters and foreign bodies. A lacerated nerve should be divided. Bromide of potassium should be given in large doses. Curare may be injected (one-fortieth of a grain is recommended to begin with). Warm baths are soothing; and a spinal ice-bag is beneficial.

Hydrophobia is due to the inoculation of animal poison—especially that contained in the saliva of the dog. The pathological changes found are merely those of general congestion of the viscera. Special importance has been attached to hyperæmia and signs of irritation at the roots of the vagus, phrenic, and sympathetic nerves.

The *symptoms*, after a stage of incubation which averages from one to two months, begin with irritation in the wound or cicatrix caused by the bite of the dog. The wound becomes livid in appearance and very painful, and the lymphatics may be sometimes seen to be inflamed. At this stage there is great apprehension and depression. Feverish symptoms soon develop, and after this stage has lasted twenty-four to forty-eight hours, the violent symptoms supervene. The breathing is at first sighing and jerking, but soon it is interfered

with by the tetanic spasms. The throat feels constricted, and there is intense thirst, but any attempt to swallow brings on a severe suffocative seizure. Violent hawking—not unlike the barking of a dog—is often present. The mind generally wanders. The slightest touch excites the spasms, which are very similar to those seen in tetanus. The whole acute stage may only last one or two days; death being the usual result from exhaustion, asphyxia, or general convulsions. The *treatment* should consist of cauterising every wound, early if possible, with nitrate of silver. Pasteur's inoculative treatment is the only treatment which can offer any hope to the patient. All other therapeutic agents have failed.

The Diagnosis of Brain Diseases.—In the first group, it has already been pointed out how far it is possible to differentiate the conditions which give rise to apoplexy, hemiplegia, and aphasia. There remains, however, the consideration of apoplexy in relation to other diseases, a subject of the greatest importance practically. Frequently such cases come before the physician without any history; and it is often difficult, and sometimes impossible, to give any positive opinion—at least for a few hours. Great caution must be exercised in the diagnosis of cerebral hæmorrhage (and its allied affections) from *injuries to the brain*, and from *narcotic* and *alcoholic poisoning*. The apoplectic symptoms are not distinguishable, and it is only by the *history and surroundings* that the diagnosis may sometimes be inferred. The rule is never to give a confident opinion in such cases as are found in the street, &c.; but to order the patient to be put to bed and to be carefully watched. Mistakes are frequently made by forgetting the possibility of there being two conditions present. An alcoholic case may readily have a cerebral hæmorrhage; or the odour of brandy may be the result of kindly, but ignorant interference in the way of treatment. A fracture of the cranium may also be a result of a fall, *after* the shock; and any suspicion of foul play must be carefully noted at the time.

Other conditions which must be differentiated are *uræmia* (serous apoplexy), *organic diseases* of the brain and membranes, as *tumour*, *abscess*, or *meningitis;* and the apoplectic form of *epilepsy*. The presence of albumen in the urine, with the history of kidney disease, will suggest uræmia, and the subsequent course of the case may confirm the diagnosis; but it should be remembered that kidney disease and cerebral hæmorrhage are often associated. The organic diseases of the brain should have a history of early symptoms, and some of the conditions mentioned under the localisation of tumours and abscesses may be present. Epilepsy is known by the comparatively rapid recovery from the apoplectic state, and, possibly, a history of a previous seizure and recovery. Pachymeningitis may be the cause of the comatose state, but this is not so important from a clinical point of view. In simple *congestion of the brain* (apoplectic form) the reflexes are normal, and there is rapid recovery without hemiplegia.

In all cases of apoplexy, it is important to know if the attack was

sudden or *gradual*. The presence of hemiplegia or rigidity, convulsions, inequality of the pupils, or the "conjugate deviation," is important, as these symptoms point to cerebral hæmorrhage.

With regard to *intra-cranial tumour* and *abscess*, the diagnosis in most cases rests upon the balance of the probabilities. In relation to the preceding group it may be said that tumour appears at any age, while *thrombosis* is a disease of the aged. The headache in tumour is very intense, and epileptiform convulsions are more common than in apoplexy. The cranial nerves are affected by tumours, and the symptoms develop slowly. From tumour, a cerebral abscess is distinguished by quicker development, fever, and the presence of a causal affection. The different forms of tumour can only be inferred. Tubercular growths occur in the young; aneurisms occur in adults and in older people, who otherwise appear to be in good health. Tubercular growths are generally deeply situated, and they do not, as a rule, affect the cranial nerves. Hydatids generally produce numerous epileptiform convulsions. Syphilis—the most important to diagnose—has already been specially mentioned. (See pp. 241 and 245.)

The differentiation of *acute* from *tubercular meningitis* has already been discussed. *Meningitis* may sometimes require to be distinguished from fevers, tumour and abscess of the brain, cerebral congestion, disease of the inner ear, and from uræmia. Fevers are excluded by the presence of convulsions, paralysis, or retinal changes; and by the non-development of diarrhœa, or rashes, &c. Meningitis is quicker in development, and the symptoms are diffused, and there is fever; an abscess can only—in the absence of causal disease—be distinguished from meningitis by having a period of latency after symptoms of acute inflammation, and by the presence of "localising symptoms," already discussed under intra-cranial tumours. From *cerebral congestion* meningitis differs in being accompanied by high fever, in being longer in duration, and followed by greater depression. In ear disease, the early history is important. In uræmia, the examination of the urine, and the history of dropsy, &c., will generally clear up the case.

Cerebral congestion and anæmia have already been compared. The symptoms come on without fever, and the attacks are generally short. Sometimes the congestive seizures may resemble *delirium tremens*, *epilepsy*, *stomachal vertigo*, or any disease in which there is mild *delirium*.

Convulsive seizures or "fits" occur as a symptom in a vast number of diseases. To enumerate these would be but to repeat what has already been stated in its proper place throughout this work. No opinion should be expressed as to the nature of an "epileptiform" attack until the whole of the systems have been carefully examined. *In children*, teething, worms, solid particles of undigested food, pneumonia, tubercular meningitis, hydrocephalus, and, indeed, the onset of almost any acute disease with fever, are only a few of the common conditions in which convulsions may be present. *In adults*, cardiac and renal disease (uræmia), many diseases

of the brain and spinal cord (and injuries), puerperal eclampsia, very acute painful diseases (as gall-stones and renal colic), anæmia (emboli), poisons, hysteria, *alcoholism*, syphilis, caries of the teeth (rarely), and other reflex causes, have all to be excluded before *epilepsy* can be stated with confidence to be the substantive disease. The differentiation of epilepsy from simple hysteria and malingering is not usually difficult. In epilepsy the reflexes are absent, especially the conjunctival reflex; the eyes are open and staring, the face is pallid, the skin is cold, and the spasms are often at first *tonic*, then followed by the *clonic*. The pupils are dilated, the tongue is often bitten, and there is insensibility to pain. The thumbs may be doubled in, and when forcibly withdrawn, they *remain* extended. In malingering, or in hysteria, these conditions may be absent or reversed. As hystero-epilepsy is a combination of the two states it is sometimes more difficult to be sure of the prevailing one. The patient may have learned to imitate her epileptic fit very well. *Le petit mal* requires to be differentiated from syncopal attacks and vertigo from other causes. The paralytic affections which are so frequently met with in the hysterical, are differentiated by the history, the non-development of trophic changes, and the preservation of electro-contractility, &c. There is never facial paralysis in hysterical hemiplegia, nor any history of antecedent apoplexy.

The diseases of the nervous system in which *tremors* occur as a prominent symptom, are paralysis agitans, chorea, multiple or disseminated sclerosis, and mercurial poisoning, &c. In *paralysis agitans* the movements are continuous, although they may sometimes be controlled by an effort of the will. In simple "senile trembling" the head is chiefly affected, and there are no other symptoms, such as paresis or paralysis, or backward propulsion, as occur in the true paralysis agitans. In *chorea*, the well developed cases have no resemblance to the others. The extremities are "flung" about, and the grimaces are characteristic. In the mild cases, which are only called forth by directing a muscular effort to be made, it is more an irregular "jactitation" than a tremor. In *multiple sclerosis*, the tremors are only present when muscular efforts are made, while the speech is affected, and there is nystagmus. In *mercurial poisoning* the tremors are seen only when movements are attempted; and there will be defects of vision, a grey-blue line on the gums, ptyalism, fœtid breath, and loss of co-ordination, besides a history of exposure to the vapour of mercury.

Vertigo is a symptom of many affections, requiring—like convulsions—an exhaustive examination before regarding it as a substantive morbid condition. It is present in Meniere's disease, and in ear diseases in general. Cardiac, cerebral, and stomachal vertigo should be noted. Stomachal vertigo may not be associated with any very pronounced symptoms of gastric disorder, and it is, therefore, apt to be overlooked and mistaken for cerebral disease—especially as it occurs most frequently in the aged.

With reference to the various forms of paralysis, there are still three diseases—described elsewhere—which have to be kept in

view in relation to the diagnosis—viz., chronic plumbism, or lead poisoning; diphtheria (post-diphtheritic paralysis); and general paralysis of the insane. The latter disease has not been treated in this work, as it belongs to the domain of Insanity. In practice, however, it is met with very frequently, especially in the early stages, and as often the delusions of grandeur, &c., are absent, or concealed at the beginning of the disease, mistakes are frequently made. If the mental condition do not at once strike one, then the trembling of the lips, the slow and difficult speech, and the staggering gait—especially when asked to turn suddenly—should suggest the disease; and the testing of the memory, intelligence, and the moral state, will usually clear up the matter. Cerebellar tumours, bulbar paralysis, cerebral syphilis, and alcoholism, may sometimes have symptoms resembling general paralysis in some respects; but a careful consideration of the other symptoms which characterise these diseases will generally admit of a correct diagnosis being made by exclusion.

CHAPTER XII.

DISEASES OF THE HÆMOPOIETIC SYSTEM.

Contents.—Diseases of the spleen—Diseases of the lymphatic glands—Hodgkin's disease — Myxœdema — Leucocythæmia — **Anæmia**; *chlorosis; progressive pernicious anæmia; parasitic anæmia—Method of examining the blood with Gower's hæmacytometer*—Addison's disease—Scorbutus—Purpura—Hæmophilia.

The Spleen.—This organ may be attacked by acute inflammation which may ultimately lead to the development of an abscess. Blows or injuries may excite such inflammations; but more commonly they arise from infarctions, caused by emboli blocking the vessels. To the natives of India a blow in the region of the spleen is highly dangerous, and often it is fatal. The spleen is often enlarged and congested in acute infectious fevers, and in leucocythæmia. It is often displaced by pleuritic effusions, &c., and it is sometimes *movable*, like a "floating kidney." The spleen is often affected by waxy degeneration, along with waxy disease of other organs. Hydatid cysts sometimes lodge in the spleen.

Some of these affections are only of pathological interest. Others, as enlargement, are important in relation to other diseases. The *symptoms* of inflammation are often very obscure; but the seat of the pain, rigors, and, in the later stages, the evidence yielded by palpation and percussion, may enable a correct diagnosis to be made.

A sudden pain in the region of the spleen in a case of heart valvular disease points to embolism. The *prognosis* in abscess is unfavourable. The *treatment* should consist of turpentine stupes locally, or hot poultices. Quinine, in large doses, is indicated. The aspirator may be used when suppuration has occurred.

The Lymphatic Glands.—Simple inflammation and tubercular disease of the glands (when they are external) are more properly considered in surgical works. A *progressive diffused* form of tubercular disease is described by Fagge, in which nearly all the glands of the body are affected. To the physician, diseases of the internal glands—as the bronchial and mesenteric—are of importance, and they have already been alluded to. Large masses may form tumours (Lymphomata), which, in the thorax, may give rise to the signs of consolidation, and be accompanied by *pressure* symptoms. The glands may also be affected by cancer or syphilis, the differential diagnosis resting chiefly upon the age and the differences between the tubercular diathesis, the cancerous cachexia, and the history of syphilis, &c.

Adenia, Lymphadenoma, or Hodgkin's disease, is a special disease affecting the glands, spleen, and the blood. The causes are not known. The glands enlarge, and may be firm or soft. They are not painful to the touch. The disease affects the whole body, beginning generally with the cervical, and extending to the axillary, inguinal, bronchial, mediastinal, and mesenteric glands. In most cases there is fever. The white blood corpuscles are not increased in number. The red corpuscles are diminished—hence *anæmia* is a prominent symptom. The spleen is enlarged. The glandular enlargements excite pressure symptoms. In the thorax, there may be the usual signs of pressure—as dysphagia, dyspnœa, and the effects of pressure upon the nerves, as in other intrathoracic tumours. The *treatment* consists of the administration of arsenic, iron tonics, and cod-liver oil. Iodide of potassium may be tried. The *duration* is apt to be very chronic; and death may ultimately result from exhaustion, or from intercurrent diseases, as pneumonia, phthisis, &c. Some cases recover.

Disease of the Thyroid Gland.—Myxœdema.—In this disease, there is general deep-seated œdema, and great increase of the connective tissue throughout the body; and in the skin, mucous membranes, glands, nervous matter, &c., there is excess of mucin deposited or formed. The thyroid gland atrophies, and may finally disappear, and this seems to be the primary cause of the affection. The etiology, however, is still obscure. It is far more frequent in females—especially about middle life—although cases are met with in children. Prolonged lactation, worry, &c., are blamed as exciting causes. The disease seems to be related to adult *cretinism*, and myxœdema has followed the removal of the thyroid gland, both in man and in animals (Horsley).

The symptoms.—The face is pale, puffed up, and heavy looking.

The expression is stupid. The lips are thick and protruding, and the eyelids appear dropsical, as in kidney disease. Sometimes the distention of the lower eyelids is such as to present the appearance of little bladders; but acupuncture of these does not yield serum. The nose is enlarged. The skin is dry and scaly, and there is absence of perspiration. The hands are thickened, broad, and "spade-like," the fingers being somewhat clubbed. There is thinning of the hair of the scalp and eyebrows. The speech is slow, and hesitating or drawling. The memory is impaired, and the mind weak. The movements are sluggishly or stiffly carried out; and the patient becomes very easily fatigued, and soon breathless. The pulse is slow. The appetite and digestive functions are poor. The urine is often increased in quantity, and in the later stages *sometimes* contains albumen. The temperature is often slightly subnormal, and the patient complains of feeling cold. The thyroid gland is much smaller, and may have entirely disappeared. The progress of the disease is very slow—uncomplicated cases lasting about six years. Death may occur from exhaustion, uræmic poisoning, or from cerebral coma. In the **treatment**, arsenic, iron, massage, galvanism, and faradism, are usually tried, but all have given place, recently, to the remarkable effects of injection, or feeding, with the juice of the thyroid gland of the sheep. An extract may be used, but the glands themselves may be eaten—the dose being about a half to a whole thyroid daily. Improvement follows within a few days; and as yet no failures have been recorded. Cardiac tonics may be prescribed; and excessive exertion should be avoided while taking the thyroids.*

The Blood.—Leucocythæmia.—This disease affects the spleen, lymphatic glands, and the blood. Although believed to be due to some functional irritation of the glands connected with the formation of the blood, nothing further is known regarding the causes of leucocythæmia. The white blood corpuscles are enormously increased in number, both absolutely and relatively, while the red blood corpuscles are diminished. The relations may be from one (white) to six (red), or even fewer red corpuscles. The white corpuscles are larger than the normal, and they may contain several nuclei. The colour of the blood is pale, the specific gravity reduced from 1,055 to 1,040 or less, and it is alkaline in re-action. The spleen and lymphatic glands become much enlarged, and the marrow of the long bones undergo pathological changes. The other organs, as the liver and kidneys, &c., often suffer as well from "lymphoid" enlargement. Different varieties of leucocythæmia are described according as the spleen, lymphatic glands, or bones are most affected by the disease.

The **symptoms** begin with gradually increasing weakness and anæmia. Vertigo, *tinnitus aurium*, palpitation, and breathlessness are early symptoms, which gradually get more and more marked.

* *Acromegaly*—a rare disease which has recently been described—seems to be allied to myxœdema and cretinism. There is general enlargement of the bones, &c.

The vision is often affected, and bleeding from the nose is very common. As the anæmia progresses, there occur œdema of the ankles, swelling of the eyelids, and other dropsical conditions. An anæmic murmur is heard at the base of the heart. The pale appearance of the blood when compared with a drop of normal blood upon a handkerchief, is very striking. The hæmacytometer will reveal the abnormal changes. The lymphatic glands become enlarged, and the spleen is found by palpation and percussion to be increased in size and density. In extreme cases of splenic enlargement the abdomen is prominent, and the mesenteric glands may be felt to be enlarged, and firmer. Constipation and diarrhœa alternate. The urine has a specific gravity of 1,020 to 1,030. The urea is diminished, but uric acid is increased, and hypoxanthin is present.

The *course* of leucocythæmia is chronic, the average duration being probably two years. The case may terminate by exhaustion, or by some intercurrent disease, as pneumonia, œdema of the lungs, phthisis, &c.

The *diagnosis* is not difficult; but in the early stages leucocythæmia cannot be differentiated from simple anæmia or chlorosis. The *treatment* consists of the administration of iron tonics and quinine. Transfusion of blood, and extirpation of the spleen, have been tried. No specific treatment is known. The diet should consist of fresh meat, fish, eggs, and milk.

Anæmia; Chlorosis; Progressive Pernicious Anæmia; *Parasitic Anæmia.*—In all the forms of anæmia there is, more or less, a diminution in the number of the red blood corpuscles. The blood is thinner in quality, and does not so readily coagulate. The organs are paler and dryer, and fatty degeneration of the heart, and other organs, is very frequently found. In *chlorosis* the aorta is sometimes found to be narrow in its calibre. In the *pernicious* form, small bodies, or immature blood corpuscles, have been described by Eichorst as being often present. Minute hæmorrhages occur in the skin, mucous membranes, and other organs. An ophthalmoscopic examination often reveals these hæmorrhages in the retina.

The **causes** of anæmia are numerous. The female sex suffers most. There appears to be a special type of constitution—pale, weak, and delicately formed individuals—in which the tendency to anæmia remains permanent through life. The iron treatment in such cases does not seem, even although long continued, to produce any impression upon the quality of the blood. In other cases it may be said that any cause which interferes with the natural physiological and hygienic laws of health—such as insufficient food, air, light, and exercise—will tend to produce anæmia. Disorders of digestion, prolonged lactation, over-fatigue, menorrhagia and other hæmorrhages, sexual excesses, malaria, syphilis, and malignant or exhaustive diseases, are primary affections which are soon followed by anæmia and debility. Chronic albuminuria (Bright's disease) is always associated with a peculiar pallor and a "pasty-looking" com-

plexion. In *chlorosis*, sexual derangements seem always to be associated with the anæmia. The causes of the pernicious form are still unknown. It occurs oftenest in women who have been repeatedly pregnant.

The **symptoms** of simple anæmia begin with languor and weakness, and gradually increasing pallor. The face, lips, gums, and conjunctivæ lose their natural colour and become paler and unhealthy looking. The conjunctival membrane covering the lower eyelid, appears sodden or hydræmic in advanced cases. The heart sounds are feeble, and *systolic* murmurs may readily be heard in all the cardiac areas. The "venous hum" is heard at the root of the neck, and in other places, when the stethoscope is placed over the large veins. The pulse is weak, rapid, and often irregular. The breathing is embarrassed, and palpitation is complained of upon making the slightest exertion. Syncopal attacks are frequent, and a hæmorrhagic tendency is sometimes developed. Œdema of the ankles or eyelids is very commonly present, and it is due to the diminished quantity of albumen, and the increased quantity of water in the blood (*hypalbuminosis* and *hydræmia*). The appetite is poor, and constipation is the rule, until the later stages, when diarrhœa often becomes rather troublesome. The urine is neutral or alkaline, pale in colour, and the specific gravity is low. The mental faculties are weakened, inasmuch as mental effort soon exhausts the brain. Hysterical seizures are frequent. Strong light and noises are disagreeable to the patient. Hyperæsthesia and hyperalgesia are often present; and neuralgia is a very common affection in the anæmic. In extreme cases of anæmia affecting the brain, the head symptoms become more marked, and mild delirium is present, or acute mania may supervene (see Anæmia of the Brain.) Epileptiform convulsions frequently occur. There is amenorrhœa in the female, and loss of sexual power in the male. The muscles become flabby and weak; but in some cases the body appears well nourished, and the amount of fat deposited in the tissues may even be increased.

The *course* of anæmia depends much upon the cause. Œdema of the lungs, or pneumonia, may supervene and may prove fatal. A perforating ulcer of the stomach is an occasional complication. If associated with a curable disorder the blood may be improved in the course of six weeks, and recovery may take place within three months if the case be a simple and uncomplicated one. The *chlorotic* form is the "green-sickness" which so frequently occurs in girls at the age of puberty. It is invariably associated with amenorrhœa, and there is no œdema of the ankles as a rule. The body appears fairly well nourished, and the other symptoms are the same as in simple anæmia. Hysteria is common. Recovery, or partial recovery, may be expected under treatment; but some cases may take an unfavourable course and terminate in pericarditis, endocarditis, perforating ulcer of the stomach, or other intercurrent malady.

The *pernicious* form is only known from the simple anæmia in the later stages, when the rapidly increasing debility and pallor, epistaxis, or hæmorrhages into the skin and retina, feverish attacks,

and the utter inefficacy of the iron tonics prescribed at the beginning of the disease, indicate the hopeless character of the case. The œdema is often general and is always present. The fatal termination occurs within two to four months.

[*Parasitic Anæmia* is described by Fagge as a form of anæmia caused by nematode worms in the small intestine (*Anchylostomum duodenale*). They feed upon the blood. The disease is common in Egypt and in other hot countries, where the drinking water is impure. It is rapidly fatal if not treated. The *treatment* consists of santonin or oil of male fern.]

Method of Examining the Blood with Gower's Hæmacytometer. — A solution of sulphate of soda containing 104 grains to four ounces of distilled water, to which 60 minims of strong acetic acid have been added, is first prepared. The thicker pipette is now used to draw some of this solution up to the mark (995 c.mm.), and it is then blown gently into the mixing jar. The finger of the patient is now pricked, and a drop of blood exudes. *The finger must not be squeezed.* The smaller pipette is now used to draw blood from the wound up to the mark (5 c mm.), and after carefully wiping the pipette, this is also transferred to the jar, and mixed thoroughly. A drop is then placed upon the centre of the hollowed slide, and a cover-glass placed upon it. The slide is placed upon the stage of the microscope, and after a few minutes the counting of the corpuscles may begin. The number of red corpuscles in ten squares, multiplied by ten thousand = the number in a cubic millimetre. The normal number is five millions. The average number of white corpuscles, as compared with the red, is *one* white to about five hundred red, but it varies very much, the white corpuscles being increased after meals.

Addison's Disease is a peculiar affection, consisting of a low form of inflammation (tubercular?) of the supra-renal capsules. The connected nerves and the solar plexus are involved. Pigment is deposited in the skin and mucous membranes, and there is great anæmia. The *causes* are unknown. The *symptoms* begin very gradually. There is first great weakness, and the digestion is much impaired. Later, the gastric symptoms become prominent, and along with the extreme pallor which now develops, there are pains in the stomach, nausea, vomiting, and diarrhœa. The symptoms peculiar to the anæmic state are now marked, and then the characteristic deposits in the skin and mucous membranes begin to appear. These consist of patches of dusky pigment, which become ultimately bronze or copper-coloured. The mucous membrane of the mouth becomes affected, and then the skin at the armpits, and later the chest. It spreads over the whole body; but the skin of the palms and the soles of the feet remain free. There is febrile disturbance in the acute cases; but the course is usually chronic, and a fatal termination takes place in about a year or eighteen months.

In the *diagnosis*, *pityriasis versicolor* should be noted; and soap and water may require to be used in some cases, before expressing an opinion as to the nature of the disorder. Before pigmentation occurs, the extreme *asthenia* and pallor may suggest the disease, but at this stage the diagnosis can only be conjectural.

Scorbutus.—Scurvy is produced by abstinence from fresh meat and vegetables. Bad hygienic conditions favour its development, and individuals weakened by syphilis or other constitutional disease, are more liable to suffer, when placed in circumstances which compel them to live on salt meat and fish.

The **symptoms** begin with anæmia, and then gradually all the symptoms associated with that affection make their appearance. Muscular rheumatism is often present. The characteristic symptoms of scurvy appear later, and consist of inflammation and putrid ulceration of the gums, which bleed easily; extravasation of blood into the muscles, or under the skin; ulcerations of the skin itself, especially of the lower extremities; and often hæmorrhages into the intestine, or from the nose, stomach, or kidneys. The breath is fœtid, and the mouth is very painful as the decomposing sloughs separate and leave raw surfaces. The extravasations into the muscles give rise to indurated lumps which can be felt. The spleen is enlarged; and the urine is diminished in quantity, and contains albumen. In very bad cases, the bones, periosteum, and joints are affected. Deformities may result from the extravasations, and death may be due to endocarditis, pleurisy, peritonitis, pneumonia, or exhaustion after hæmorrhage.

Purpura.—*Purpura simplex* is the form characterised by small petechial extravasations under the skin without hæmorrhages elsewhere. *Purpura hæmorrhagica* is a much more serious form, in which occur not only petechial extravasations, but severe hæmorrhages under the skin, and into other organs.

The simple form begins with languor and anæmic debility, or with sudden epistaxis. Blue-red spots soon appear, especially affecting the lower extremities and the body. They vary in size from a pin's head to a pea, and they gradually change in colour—green, brown, and yellow. They occur in crops, and slight injuries produce ecchymoses readily. Hæmorrhage from the mucous surfaces is common; but there is no sloughing of the gums as appears in scurvy. The hæmorrhagic form is characterised by extensive extravasations and bleedings, which increase the anæmia and produce its train of symptoms. Syncope may be the result of a large internal hæmorrhage. When true articular rheumatism is present along with the purpuric spots, it is sometimes described as a special form—*purpura rheumatica*; but rheumatic pains are common to all forms. Fever is not present as a rule.

An ordinary case lasts three weeks or more, and it may extend to several months. Most cases recover; but the severe hæmorrhagic form may prove fatal. A cautious prognosis should always be given as simple forms sometimes become hæmorrhagic.

In the **diagnosis**, *scorbutus*, *hæmophilia*, *progressive pernicious anæmia*, and *leucocythæmia* should be noted and excluded.

Hæmophilia is a congenital, inherited, constitutional condition, in which there is great liability to hæmorrhage. The victims of this affection are known as *bleeders* (see Surgical Works). In some cases the

bleeding arises spontaneously. It may be internal, and may give rise to urgent symptoms; and if slow and chronic, anæmia results from these repeated attacks of hæmorrhage.

As the diseases of the blood-forming organs are all managed upon the same lines, it has been found convenient to defer the **treatment** until the diseases were themselves considered. The blood-tonics —iron, arsenic, quinine, manganese, and cod-liver oil—are indicated. Before commencing a course, attention must first be given to the digestive functions. If gastric catarrh be present, ℞ 40 may be given for a short time. If digestion be weak, a mineral acid and bitter, or pepsin may be prescribed (see the treatment of stomachal affections). The diet should be carefully regulated, beginning with light milk food, fish, chicken, and beef juice, if necessary; and the sooner that a good generous diet of fresh meat, eggs, vegetables and fruit can be borne, the better. Fresh air, light, and gentle regular exercise are highly important. Stimulants, as wine or whisky, may in some cases be necessary, but these will be ordered with due care in relation to the possibility of developing a taste for alcoholic beverages. The saccharated carbonate of iron is usually well borne. Freshly-made Blaud's pills (℞ 56)—two or three, thrice daily, after food—is a favourite form of prescribing iron. The tincture of perchloride of iron is highly recommended, care being taken to instruct the patient to watch its action upon the teeth. It should be taken through a glass tube, and the mouth and teeth should be carefully cleansed after each dose. Chemical food is useful, and easily taken. Fellows' and Churchill's syrups of the hypophosphites, and Easton's syrup, are much prescribed. The syrups of the phosphates, and lacto-phosphates of iron and of lime—the latter especially when iron does not agree—are much used. "Ferro-Maleski" is a preparation of iron with a vegetable compound, which may prove to be highly serviceable. Denaeyer's peptonate of iron is useful when the digestion is weak. The combination of iron and arsenic sometimes produces wonderful results (℞ 1). A purgative at frequent intervals is often required during a course of iron treatment. ℞ 57 may be ordered. In extreme cases of anæmia, Weir Mitchell's massage treatment, with forced feeding, may be necessary. *Transfusion* of blood may be tried, as this treatment has been lately revived, and good results are recorded by Brackenridge and others. In *purpura* and *scurvy*, turpentine is indicated for the hæmorrhages. The treatment of scorbutic conditions consists of the ingestion of fresh meat and vegetables, and the administration of lime juice. Arsenic may be tried in progressive pernicious anæmia and in Addison's disease. Since the discovery of the good effects of thyroid-feeding in myxœdema, it has been suggested (Affleck) that perhaps feeding on supra-renal capsules might be of benefit in Addison's disease. For pernicious forms of anæmia, an *antiseptic* treatment has been suggested lately by Hunter. Two grains of β-naphthol are given (in a pill) thrice daily. Good results of this treatment are recorded by Gibson.

CHAPTER XIII.

CONSTITUTIONAL AND GENERAL DISEASES.

Contents.—Gout; *Lithæmia*—Arthritis deformans—Acute, chronic, and gonorrhœal rheumatism—Rachitis—Osteo-malacia—Syphilis—Chronic alcoholism; *Delirium tremens*—Trichinosis—Anthrax—Lead and mercurial poisoning.

Gout.—Lithæmia.—Gout may be *acute* or *chronic*. The chief pathological condition is the enlargement of the joints—especially the smaller joints—and the deposition therein of urate of soda. The bursæ are often affected. A section of a joint shows the articular surface to be covered with an incrustation of white acicular crystals. Ordinary inflammation, thickening, and deformity are the results of this deposit, and chalk-like accretions (tophi) of urate of soda, are mixed with the inflammatory products. They are often found in the external ear, and generally in or near the helix. Suppuration may take place around the tophi, but never in the joints themselves. The kidneys are frequently cirrhotic (gouty kidney), and the heart hypertrophied.

Lithæmia is the name proposed by Murchison—and approved of by Garrod and Fagge—for that condition of the blood which is present in gouty individuals. The urea is not fully oxidised, and uric acid is found in the blood serum as a result of the imperfect oxidation. It is excreted by the kidneys. The state of *lithæmia* is that which gives rise to the so-called "*irregular, latent, or suppressed*" gout.

The presence of excess of uric acid in the blood is believed to be the cause of the actual attack of gout. Garrod believes that the deposit of urate of soda in the joints is an antecedent condition, the result of lithæmia, and probably these deposits excite an attack of gout. Any sudden failure of the kidneys to excrete the uric acid, or the ingestion of food rich in nitrogenous matter and sugar, may excite an attack of gout in lithæmic subjects. After an attack the uric acid seems to be removed from the blood, and the patient feels much better.*

Gout is markedly hereditary, and it chiefly affects the male descendants, but females suffer more from gout than is generally stated in medical works. Long-continued rich living may produce a gouty state; and certainly in those who have inherited the gouty diathesis, a rich dietary is a well-known cause of attacks. Chronic lead poisoning is also a cause of gout. Exposure to cold, and excessive fatigue or mental worry, may also excite attacks in the lithæmic.

* *Garrod's Method of Detecting Uric Acid in the Blood.*—Two drachms of blood serum are put in a large watch-glass and acidified with acetic acid. A linen fibre is placed in the fluid until the evaporation leaves a gelatinous mass. The fibre is then examined with a pocket-lens for the characteristic uric acid crystals.

Symptoms.—The *onset* of acute gout is very sudden, although sometimes there are premonitory symptoms—as indigestion and flatulence, or sleeplessness and irritability, &c. During the early morning hours the patient is awakened by pain in the metatarso-phalangeal joint or "ball" of the great toe. Sometimes the pain is in the ankle or instep, or in the knee—especially if it have been previously injured in any way. The pain becomes very intense, and it is described as grinding, boring, and intolerable. He cannot bear the slightest touch, and it is impossible to gain rest for the foot in any position. Soon the parts become dusky red, and the skin becomes shining, swollen, and tense. Slight chills and rigors usher in the feverish state. The temperature rises according to the amount of local disturbance. The parts become œdematous, and ultimately, in the later stages, the skin desquamates.

The pain may moderate in intensity within a few hours; but for the next two or three days the attack is renewed towards evening. The other foot is generally attacked as well. The urine is acid, and deposits copious urates. The case gradually improves during the next eight or ten days if the patient be not a chronic sufferer, in which case the attack may extend over a few months, with frequent exacerbations and improvement. The chronic cases always feel much improved in health after a seizure. After a few attacks the other joints may be the seat of pain, either during an acute seizure or during the chronic course of gout. The small joints of the fingers are especially liable to suffer in this way. They remain red and thickened, and the patient, in severe cases, may hardly ever be entirely free from pain.

Gout is never immediately fatal, but it tends to shorten life. There is liability to cirrhosis of the kidneys and hypertrophy of the heart, with its consequent risk of cerebral hæmorrhage. Deformities are common, and the patient is often much crippled. A single attack of gout may serve as a warning, and with strict regulation of the diet, &c., it may never recur—if the patient be not markedly gouty by inheritance. Patients may remain free from attacks for a year or longer. During the intervals, *lithæmia* may be present; or this condition may exist without the patient ever having an attack of acute gout. Dyspepsia is the common result of this condition. The symptoms of lithæmia consist more or less of those indefinite pains known as "gout in the head," "gout in the stomach," &c., with aching pains in the limbs, attacks of gravel, giddiness, "bilious" headaches, and noises in the ears, with sleeplessness and depression. Sometimes lithæmia gives rise to epileptiform convulsions.

Arthritis Deformans.—Rheumatoid Arthritis.—The causes of this disease are still unknown. It occurs most frequently in women, especially when exhausted from frequent child-bearing, or from prolonged lactation. Males may be affected, when debilitated from any cause. The disease is not gout, and neither is it rheumatism, as the old name, "rheumatic gout," would appear to indicate. It may be proved to be due to a nervous disease, and to be a condition analogous to the enlarged joints which occur in locomotor ataxia.

The synovial membranes become thickened, and fluid is effused into the joints during the early stages. Later, the interarticular cartilages become ulcerated, and then absorbed, leaving the bare bones in apposition. The bones become thickened and enlarged (eburnation). There is pain and stiffness, with crackling of the joints when movement is attempted or the limb manipulated. Ultimately, the lower limbs are flexed, and the patient is permanently crippled. The wrists are extended, and the fingers are bent to the ulnar side. An acute form is described in which there is fever and effusion into the joints with symptoms like acute rheumatism. The heart, however, is not liable to be attacked. The chronic forms are more common.

The *prognosis* is not always unfavourable, as some cases recover under treatment.

Rheumatism.—Acute; Chronic; and Gonorrhœal.—In acute articular rheumatism or rheumatic fever, the symptoms are ushered in by indefinite pains in the limbs (*muscular rheumatism*) with general *malaise*. The diagnosis of "rheumatic cold" is generally made at this stage, and for a couple of days it may be uncertain whether this is to be the sole extent of the rheumatic affection, or whether the acute rheumatic fever is about to develop. Should the latter be the case, the temperature gradually rises, and there may be a feeling of chilliness, or rigors occur from time to time. The pain and swelling are, at first, located to one joint—generally one of the larger ones—and the peculiarity of rheumatism is its liability to assail, suddenly and rapidly, one joint after another. Several joints may be affected at one time, and should the smaller joints suffer—as the knuckles—it is generally *along with* one or more of the larger ones. *Gout*, on the other hand, generally attacks the smaller joints.

In rheumatism the joints are very tender to the touch, and in some cases—*e.g.*, the knee—the effusion of fluid into the joint can very readily be made out. The surrounding veins are not dilated, and generally there is little or no redness. There is often severe sweating, the perspiration having a characteristic sour smell. *Sudamina* may appear as the result of severe sweating. *Erythema multiforme* is a skin affection which may appear during the course of acute rheumatism.

The temperature is generally in relation to the pain and extent of the articular inflammation, but the pulse is often more rapid than one would expect from the amount of fever. 103° F. is a common temperature. The heart is almost invariably affected in acute rheumatism, and an examination of the chest should be made daily, during the course of the disease. Pericarditis and endocarditis are the commonest affections. Pleurisy, bronchitis, and sometimes catarrhal pneumonia and œdema of the lungs, are common complications. In some cases—especially in children—the heart may be affected, and there is no pain in the joints. The appetite is generally good. The urine is scanty, dense, acid, and high coloured, and it deposits urates plentifully. A small quantity of albumen is often present.

Hyperpyrexia may supervene in a case of acute rheumatism.

The temperature may rise as high as 107° to 110° F. within a few hours. The symptoms associated with such a condition are the cessation of pain and sweating, followed soon by cerebral symptoms. Drowsiness passing into unconsciousness, or acute maniacal excitement with convulsions, may usher in the fatal termination of a case.

The *causes* of acute rheumatism—in addition to hereditary predisposition—are exposure to cold and damp, and severe muscular exertion. It often follows scarlet fever, during the desquamative stage. Sleeping in a damp bed is a very common cause.

The *course and duration* of acute rheumatism varies with the effects of treatment. It is not uncommon for cases to run on for a month or longer. They are very seldom fatal. Many cases are cut short in their course. Acute rheumatism is apt to recur within a few years. Some few cases become chronic and a joint may remain permanently injured (anchylosis). There is never *purulent* disorganisation of a joint.

The **chronic** form of rheumatism begins very slowly, with less pain, and no fever. It is a disease of middle life, occurring in those who have inherited the rheumatic *diathesis*, and it is often a natural consequence of advancing years, especially in those who have been exposed much to cold and wet, through life. In the former class the deformities from contraction of the tendons and enlargement of the joints, are often very severe.

Gonorrhœal rheumatism is really of the nature of a pyæmia. An important symptom is the *fixed* character of the pain, limited to one joint—generally the knee.

The **differential diagnosis** of gout from rheumatism, arthritis deformans, and pyæmia is important. In gout the small joints are affected; the attack is sudden; the skin desquamates; and the fever is in relation to the local inflammation. The opposite conditions are present in acute rheumatism. *Arthritis deformans* occurs amongst the poor; there are no paroxysms, and the changes are very gradual and symmetrical, without deposition of urate of soda. In *pyæmia* the severe constitutional disturbance, rigors and dry skin; the joints showing a deeper blue-red; the presence of rashes and abscesses, and the history, will indicate the true nature of the case. In *acute* rheumatism, effusion should be made out in at least one joint, before expressing a positive opinion. In the *chronic* forms, locomotor ataxia, spinal disease, and such diseases as are associated with pains in the limbs—as scorbutus, syphilis, hysteria, sciatica, &c.—should be noted and excluded. The pains in such cases are not always limited to the joints.

The Treatment of Gout, Rheumatism, and Arthritis Deformans.—*Gout.*—For the acute seizure morphia may be used hypodermically—as much as half a grain being injected into the arm. The sooner that colchicum is given the better. ℞ 58 should be at once prescribed. Salicylate of soda is sometimes used. The inflamed parts may be covered with lint soaked in a lotion composed of atropia and morphia (℞ 59), and covered over with gutta-percha

tissue. *Cold* applications must never be used. The limb should be raised and supported upon a pillow. However red and inflamed the toe may appear, leeches ought never to be used. The diet should be of the lightest description, consisting of milk and arrowroot, tapioca, &c. Should the case be seen early, and the attack be the result of indulgence in the pleasures of the table, a prompt emetic may be of great value. Should this be contra-indicated, a purgative may be used—salts, or "black draught" being the best. When the acute symptoms have subsided the treatment should still be continued. The diet must be carefully regulated, and it should consist of chicken, fish, beef tea, and soups. Milk, and light milk-puddings may be allowed. All sweet meats and wines must be cut off; but, if necessary, a small quantity of whisky with potash water may be taken. For the lithæmic state, the same precautions as to diet are necessary. Potash or lithia water may be freely drunk. The chronic cases require similar care and treatment. Guaiacum and iodide of potassium are additional remedies. The clothing should be warm, and flannel should be worn next the skin. Walking exercise is to be encouraged. The chief watering places, and baths, to which gouty patients resort are Vichy and Vals, Carlsbad and Aix-la-Chapelle, Bath and Buxton. The mineral waters should never be taken during an acute attack, or if there should be kidney disease present.

Rheumatism.—In the treatment of *acute* rheumatism, all remedies have given place to salicylate of soda (salicylic acid or salicin). The only bad effect likely to follow its use is the depression which results from the long-continued doses. This should be watched and counteracted by stimulants, or the remedy changed for a time. Nausea and vomiting is sometimes induced; and deafness, giddiness, and ringing in the ears are very common, with the use of the salicylates. The bicarbonate of potash may then be used instead. The dose of salicylate of soda should be twenty grains every two hours during the first day, and then it should be reduced to *ten* grains (℞ 60). The smallest quantity which will subdue the pains is the point one should desire to maintain; and if this can be done without producing depression, no other remedy need be used. The diet should be light and digestible, consisting largely of beef tea and milk. Alkaline drinks, milk and potash, &c., should be allowed. The joints should be enveloped in flannel or cotton wadding, and the patient should be kept between blankets. The *chronic* forms of rheumatism may sometimes be relieved by salicylate of soda when the attack appears to be somewhat acute in character. Iodide of potassium is highly useful, whether the rheumatism be due to syphilis or not. Bicarbonate of potash is also good. In many cases arsenic, iron, and cod-liver oil are the best remedies. Arthritis deformans is similarly treated—*i.e.*, with arsenic, iron, and cod-liver oil; and a full generous diet is allowed. Malt liquors—especially porter—are indicated. The baths and watering places for chronic rheumatic cases are Strathpeffer and Harrogate, Buxton, Bath, Wiesbaden, Aix-les-Bains, Pyrmont, &c., &c. The baths should not be taken by those rheumatic patients whose hearts are affected. Galvanism is sometimes used; and a

course of *massage* treatment may be highly useful for chronic rheumatism.

Hyperpyrexia in rheumatism is treated by cold. The patient should first be placed in a bath comfortably tepid (90° to 110° Fahr.), and cold water is added gradually. Ice-cold water may be applied to the head and spine in severe cases. The bath is continued or repeated until the fever is reduced. The patient should be removed from the bath when his temperature falls to 102° Fahr.

Rachitis.—Rickets is a disease of childhood, the symptoms of which begin to appear in the first or second year. Sometimes it develops about the period of the second dentition, but these cases are generally mild. It occurs in the children of the poor, especially when the surroundings have not been good; but it occurs also in the children of debilitated parents, whatever the class. The rickety constitution may be inherited. In rachitis the long bones become thickened at their epiphysial extremities. The flat bones are thickened. The cartilaginous parts increase without the deposition of the earthy principles which render the bones hard and firm. The growth is arrested, and deformities result from the weight of the body, or from traction of the muscles. The legs are bent, the spine is curved, and the thorax projects (pigeon breast, &c.). Lesions of other organs are usually associated with rickets, and hence tabes mesenterica, chronic diarrhœa, enlarged spleen, &c.

The **symptoms** usually begin about the fourth to the eighth month, but the early course is not characteristic of rickets. There is diarrhœa and vomiting, with great emaciation and fever. The stools are light in colour, acid, and offensive. The appetite is fitful, and it is often voracious; sometimes the thirst is intense. The child is listless and peevish, and prefers to sit or lie quietly all day. There is tenderness of the whole body, so that moving the child excites an outcry. At a later stage, profuse perspiration of the head is common. The limbs feel hot, and they are evidently painful. The urine deposits abundant lime salts. The child's face looks older and pinched, and the anterior fontanelle remains open and does not diminish in size.

The swelling at the end of the long bones may now be distinguished, and the legs are seen to be bending. The spine begins to curve, and the sternum is in process of being projected forwards. The pelvis is also deformed. Dentition is absent, or much delayed, or the teeth may be decayed and imperfect. The symptoms of the gastro-intestinal disorders increase, the abdomen enlarges, and the emaciation and anæmia become more marked.

The *course* and *duration* of rickets vary much. It extends from a few months to a year or much longer. The cases which occur early are more severe, and if recovery take place, the deformities are more marked. Those cases which occur during the period of the second dentition usually recover without any marked permanent changes in the bones.

The common complications of rachitis are bronchial catarrh and

pneumonia, capillary bronchitis, and pleurisy with effusion. *Laryngismus stridulus* is common in rachitic children. Protracted diarrhœa and waxy disease of the intestines, chronic hydrocephalus—sometimes with convulsions—and enlarged lymphatic glands, are all common conditions associated with the rachitis.

Osteo-malacia, or **Mollities ossium**, is a rare chronic disease of the bones which occurs in adults. It affects females more than males. The earthy elements are absorbed, and the bones become softened. Deformities of the spine and pelvis, &c., result. The *symptoms* consist of burning pains in the bones followed by deformities. The patient "shrinks" together in consequence of the bony framework becoming absorbed. Fractures are easy and common, and the bones do not unite. The prognosis is always unfavourable. Death usually takes place from some intercurrent affection. The causes are unknown.

The **treatment** of rickets should consist of general and hygienic remedies. Good air, exercise, bathing, and a generous diet, are all necessary. The lime preparations and cod-liver oil are indicated. Pepsin and bismuth are useful for diarrhœa and vomiting. It may be necessary to use ℞ 49. The saccharated carbonate of iron is useful. If infants require to be removed from the breast, the best substitute is cow's milk diluted to one-third or one-fourth with lime water. Mellin's or Nestlé's food may be used.

Syphilis.—The *primary* stage of syphilis comes under the notice of the surgeon. The *secondary* and *tertiary* symptoms come under the care of the physician. Some time after the primary disease—a period varying from six weeks to six months or a year—the secondary symptoms appear. There is generally, for a few days, fever of an intermittent type, the temperature sometimes rising as high as 104° F., and falling with the appearance of a rash. The patient generally complains of pains in the limbs and joints (rheumatism), and in the back. Headaches are common at night. Sometimes there is no fever, and the syphilitic rashes appear, without any apparent disturbance of the health, and without attracting much attention. The syphilitic ulceration of the fauces may also be so slight, that the patient may be unaware of the condition. In most cases, however, a sore throat is complained of, and the rash is copious enough to excite both attention and alarm.

The syphilides which appear are "copper-coloured," and very varied in character (polymorphic). They do not set up "itching," and they assume a semi-circular shape generally. These eruptions are described in works on the skin, and are figured in all skin atlases. They are *macular*, *follicular*, *papular*, *pustular*, and *squamous*, &c. There is no absolute differences between the syphilides of the secondary and tertiary stages; but it may be said that the *early* syphilitic rashes are bilaterally symmetrical, superficial in character, more numerous, and they have little tendency to ulcerate or leave cicatrices. The tertiary syphilides are non-symmetrical, tend to run together, and they are fewer, and often leave cicatrices. They also affect the deeper structures.

The syphilitic sore throat shows congestive changes involving the tonsils and pharynx. The mucous surfaces are unhealthy looking, and yellow ulcers with sharply defined red borders are commonly present. Milk-white spots—often compared with the effects of nitrate of silver applied to the mucous membrane—are often found on the inside of the cheeks, lips, and soft palate. They may be round or oval in shape, or indefinite. *Iritis* is a common effect of syphilis. According to Hutchison, it does not appear after the sixth month. The patient may complain of dimness of vision, and this should suggest an ophthalmoscopic examination for *retinitis*. The ophthalmoscope may reveal a hazy appearance about the fundus, and the disc red and swollen, with an indistinct margin. The hair falls out, not so much in patches, but generally. The lymphatic glands enlarge. The glands at the elbow and in the occipital regions are frequently found to be enlarged, upon palpation. The duration of the secondary stage is about three or four months.

The *tertiary* symptoms are numerous; but to enter into details would be but to repeat what has already been mentioned throughout this work. The syphilitic cachexia may be more or less marked. Eruptions appear on the head and elsewhere. The palms of the hands and the soles of the feet are often affected by squamous syphilides. The nails may be rough and thick. Gummatous formations and nodes may appear on the bones of the skull, sternum, ribs, tibia, &c.; and in the organs of the body. The testicles should always be examined when searching for gummatous formations. Sometimes there is albuminuria. Neuralgia is often due to syphilis, and it is characterised by being worse at night. Ptosis may be a symptom of syphilis. Ulcers, periostitis, and suppurations are common; and so are—cerebral and meningeal tumours; syphilis of the cerebral arteries; aortic valvular disease; syphilis of the spinal cord; chronic syphilitic laryngitis and stenosis of the bronchi or œsophagus; phthisis; hepatic gummata and cirrhosis; ulcerations of mucous membranes, &c. In the female there is often chronic uterine disease and ulceration of the womb. Miscarriages and abortions are very common. The ulcerations of the throat may extend or involve the bones of the hard palate. The red blood corpuscles are diminished in number, and the white corpuscles are increased, in syphilis. Tertiary symptoms may not succeed the secondary, especially when the patient has placed himself under treatment.

Congenital syphilis is characterised by the symptoms of catarrh of the nasal mucous membrane ("snuffles") about six weeks after birth. At the same time, eruptions appear, chiefly about the nates at first, and often there are condylomata about the mouth and anus. Stomatitis is common, and small ulcers are frequent about the mouth and tongue. The child appears pale, asthenic, and emaciated. The bowels are apt to be loose, and the stools foul-smelling. The face is pinched and old-looking.

Should the child recover under treatment, symptoms of the syphilitic constitution are apt to manifest themselves about the

period of the second dentition. The bridge of the nose sinks in, and the teeth may be peg-shaped and irregular, with one or more of the evidences of syphilis described above. The hair is often scanty, and the nails are rough and split easily. The bones may have nodular growths upon them, and the forehead is often prominent looking, and suggestive of hydrocephalus.

The **cause** of syphilis, apart from its b ing a hereditary disease, is the direct inoculation of the syphilitic virus. Impure sexual intercourse is almost invariably the manner in which the poison is conveyed; but it must be remembered that *sometimes* syphilis may arise in other ways. Any abraded surface—mucous membrane or skin—may absorb the poison; hence unclean catheters, discharges, blowing tubes used by workmen in turn, and tobacco pipes used by different persons—are examples of methods by which persons may be innocently inoculated. A kiss may transmit the poison to another. It is sometimes conveyed by vaccine lymph. During the primary and secondary stages, the secretions from ulcers and syphilitic sores are capable of inoculating the healthy; but those of the tertiary period are not directly contagious.

The **prognosis** of syphilis must be guarded. Most cases under treatment recover; but many cases end in permanent ill-health or disablement. Sometimes severe cases end fatally, and this is often the case with infants. With weakly adults—especially when markedly strumous—the consequences of acquiring syphilis are often disastrous, as the ulcerations and degenerative changes are in them very serious, and they often lead to a fatal result.

The Treatment of congenital syphilis consists of giving the child very minute doses of grey powder (℞ 61). The eruption may be treated with zinc, bismuth, starch, and sometimes calomel dusting powders. Ordinary hygienic care and "hand nursing" are required. Cow's milk—diluted one-third or one-fourth—and Mellin's or Nestlé's food may be used.

For adults, iodide of potassium is prescribed, and should improvement not begin very shortly, small doses of mercury should be added (℞ 62). Mercury should not be given to the weakly or strumous. Iron and cod-liver oil are useful adjuncts, and should always be given to the strumous instead of mercury. *Inunction* and *fumigation* of mercurials are severer methods. Never produce salivation if possible. Opiates and bromides may be given for pain and sleeplessness. The local treatment of ulcers, &c., consists in the use of boric lotion and ointment, mercurial applications and "black wash." In *iritis* a little calomel may be dusted into the eye. Chlorate of potash is the best gargle for syphilitic sore throats. Marriage should not be allowed until two years after the disappearance of all the symptoms.

Chronic Alcoholism—**Delirium Tremens.**—**The pathological changes** in chronic alcoholism are those of congestion and catarrh of the mucous membranes generally. The heart and liver are often fatty; and the latter, with the kidneys, may be cirrhotic. The brain neuroglia undergoes thickening, and the vessels tend to

become atheromatous. The brain membranes are often thickened, and pachymeningitis has been found in some cases.

The symptoms of chronic alcoholism consist of impairment of the intellectual powers and perversion of the moral sense, with certain physical changes which are characteristic. The former state is manifested by loss of memory and judgment. Ordinary duties are neglected, and in conversation there is incoherence and rambling. A peculiarly suave and unctuous manner seems to be common. In others a morbid irritability is often present. The spirits are often dejected. There is indifference to all the family ties, and the man has no care for the welfare of wife or child. He is frequently attacked with gastric symptoms. The appetite is lost, and the craving for stimulants increases. A state approaching imbecility is the ultimate result, and general paralysis is a common termination of chronic alcoholism. Two types are described; the pale, flabby, and fat—and the purple-faced, "pimpled," and bloated conditions. In the fully developed cases there is often numbness and tingling in the limbs, with tremors affecting the whole body. Disorders of digestion increase, hallucinations occur at night, and epileptiform fits are frequent. Often there is hæmatemesis, and hæmorrhoids which bleed freely. Later still, with the development of kidney disease or cirrhosis of the liver, there is dropsical effusion into the limbs or abdomen (ascites). Heart disease may be present from calcareous and atheromatous changes in the valves.

In **delirium tremens** there is hyperæmia of the brain, with increased effusion into the ventricles. The brain substance appears œdematous. Delirium tremens is more apt to occur in those who have indulged in alcoholic stimulants without taking food.

The *symptoms* consist chiefly of continuous trembling, sleeplessness, and noisy delirium. Characteristic hallucinations and illusions are present. The patient sees all sorts of animals, spectres, demons, and monsters, and he appears to be in a state of "horror," or fright. Under a hallucination, he may be dangerous to himself and to others. He is often noisy and furious. There is fever and sweating, with the usual accompaniments of such a state. The *course* is usually acute; and recovery is the rule under treatment, provided no dangerous complication supervene. Pneumonia, cerebral hæmorrhage, and meningitis are common. The pneumonia—chiefly affecting the upper lobe of the lung—may be present from the beginning, and it may be the actual cause of the delirium. A case may extend from two or three days to ten days. Death may take place from failure of the heart's action.

The *treatment* of chronic alcoholism is not very encouraging. The gastric symptoms are treated as in chronic gastric catarrh. Arsenic may be tried, and general tonics, as iron and cod-liver oil. Total abstinence from all alcohol must be enjoined, and, if possible, *forced*. A suitable diet must be arranged. In *delirium tremens*, bromide of potassium is given in large doses, and plenty of beef tea. The diet is important, and it should be strengthened very gradually. The patient must, of course, be protected during the acute stage.

Trichinosis.—This parasitic disease is due to the presence of the Trichina spiralis, so frequently found in pork. There are three stages described. The first or *intestinal* stage follows the ingestion of the sexually mature worm. A microscopic examination of the fæces may reveal the presence of the worms. The symptoms are vomiting and diarrhœa. The second, or stage of *migration*, begins within a week, when breeding may be said to be fairly started. The immature trichinæ bore their way through the tissues of the body until they reach the muscles, where they become encapsuled—which constitutes the third stage. During the second stage, the symptoms consist of rheumatic-like soreness of the muscles—often with great tenderness to the touch—œdema of the eyelids, rigors, fever, and perspirations. Bronchitis, pleurisy, or pneumonia may complicate a case. The third stage is the stage of *encapsulation*, in which the symptoms occurring in the second stage gradually subside. The "harpoon" may be used in doubtful cases. Typhoid fever, inflammation of the bowels, and rheumatism have to be excluded in the diagnosis.

The **treatment** is to give large quantities of glycerine and water—one part of glycerine to two parts of water—in the early stages. Carbolic acid and tincture of iodine are also useful. As the disease arises from eating raw meat (ham and sausages), this practice should be discontinued. Cooking destroys the trichinæ.

Anthrax, or *Wool-sorter's Disease.*—There are several forms of this disease; but they are all due to a single microzyme—the Bacillus anthracis. The most common form is *Malignant Pustule* or *Charbon.* It consists of the development of a vesicle upon a hard base. This may occur on any exposed part, and it is produced by the inoculation of a slight sore or scratch. After a short period of incubation, there is burning pain in the sore, and a papule develops which soon becomes vesicular, ruptures, and dries up, leaving a scab. The base becomes indurated, and œdema soon spreads up the arm. The lymphatics and glands are affected. After a few hours there follow severe pyrexia, with delirium and prostration, diarrhœa, sweating, acute pains in the limbs, and sometimes convulsions. Death results from the pyæmia. The other forms affect the gastro-intestinal mucous membrane, and the thoracic viscera, respectively.

The early **treatment** of the first and commonest form consists of destroying the pustule with caustics. The other forms and the later stage of the malignant pustule require antipyretics, and the maintenance of the strength by a generous diet and stimulants. Some cases recover.

Two forms of chronic poisoning often come before the physician in practice—viz., poisoning by **lead** and **mercury.** The former occurs in painters, plumbers, glassmakers, shotmakers, typefounders, &c. It may, in others, be the result of contamination of the drinking water by lead. "Aërated waters" are also a source of

the poison. Sometimes lead is found in coarse sugar, and in snuff. The symptoms are, viz.:—colic pains, often mild in character, but persisting over long periods; constipation; a blue line on the gums; and paralysis of the limbs. The line on the gums consists at first of a row of black dots, and treatment by iodide of potassium increases the blue colour after a few days. The blue line is caused by the deposit of sulphuret of lead, and it only seems to arise in those who do not pay particular attention to the teeth. It is the presence of tartar that sets up the decomposition. The paralysis may affect the whole body; but more generally it is the upper limbs which are paralysed, and generally one limb more than another. The extensors and supinators of the hand are powerless and wasted, and hence the "dropped wrist" of lead paralysis. It is still doubtful whether the disease is central or local. In some cases a degeneration of the anterior horns of grey matter has been found. Alterations in the muscles suggest some pathological change in the nerve endings. Gout is very often associated with lead poisoning; and the patient becomes anæmic and cachectic. Sometimes *tremors* are present.

In the diagnosis, *progressive muscular atrophy* has to be noted. The most striking difference is the absence of a re-action to faradic electricity, in lead paralysis, and the abnormal sensitiveness to galvanism which is present.

The *treatment* consists of iodide of potassium in large doses. Sulphate of magnesia is good for the constipation. Castor oil and laudanum may be used. Galvanism is useful for the paralysis. Workmen exposed to the poison should be instructed to drink sulphuric acid lemonade, and to be particularly careful in washing their hands, &c., before eating.

In poisoning by **mercury** the characteristic symptom is the *tremors*. They are general, and are worse under the influence of excitement, or when voluntary motion is attempted. Salivation, ulceration of the gums, and anæmia may all be present. The speech is slow and jerky, and the memory is impaired. Cerebral symptoms, or epilepsy may supervene. Poisoning by mercury occurs in water-gilders and workers in quicksilver. The *treatment* consists of iodide of potassium. Bromides, and hyoscyamine, &c., may be used to control the trembling.

CHAPTER XIV.

FEVERS AND MIASMATIC DISEASES.*

Contents. — Typhoid fever — Typhus — Relapsing (Famine) fever—Measles—Scarlet fever—German measles—Smallpox—Chickenpox—Dengue and Plague—Glanders and Farcy—Intermittent and remittent fevers—Yellow fever—Mumps—Influenza — Epidemic cerebro-spinal meningitis—Cholera—Diphtheria.

Enteric, or Typhoid Fever.—The pathological conditions found in typhoid fever may be divided into the special and general. The *special* morbid changes consist of hyperæmia and swelling of the mucous membrane of the lower part of the ileum, particularly around Peyer's patches and glands. The congestion often extends to the peritoneum, &c. Later, the glands become infiltrated, and the solitary follicles may enlarge to the size of a pea; while the patches are raised, oval in shape, and may ultimately coalesce. Necrotic softening and sloughing may follow, and the excavations left extend to the muscular coat of the bowel, and sometimes even to the peritoneum. These typhoid ulcers are described as "elliptical in form, their margins thick and sharply defined, and their long diameter parallel with the intestine." Restoration may take place by granulation and cicatrisation. Many cases do not go on to ulceration. The mesenteric glands become enlarged—secondary to the changes in the intestine.

Amongst the *general* morbid changes, congestion and enlargement of the spleen is important, sometimes, in relation to the diagnosis. The organ is at first firmer, but later it becomes exceedingly soft. The remaining general changes are those which are associated with all continued fevers, viz.:—softening and fatty degeneration of the heart, liver, &c.; granular infiltration of the kidney epithelium, with albuminuria; anæmia and œdema of the brain; catarrh of the bronchial mucous membranes—often with collapse or œdema of the lungs, and sometimes (rarely) pneumonia; and general congestive and catarrhal changes.

The **cause** of typhoid fever is believed to be the presence within the system of a germ, which arises and develops in connection with decomposed animal matter. The activity of the germ develops a special poison which produces the typhoid state. The usual vehicles by which the disease is introduced are drinking-water, milk and other foods, and air. The excrements of patients suffering from typhoid fever contain the germs; but the disease is not markedly

* Some of the miasmatic affections—as pertussis, hay asthma, &c.—have been more conveniently considered elsewhere.

contagious. A certain "susceptibility" to the poison seems to be necessary for its development; and one attack seems, but not always, to protect from a second seizure. The most common period of life in which typhoid occurs is between the ages of fifteen and thirty years. The period of incubation is doubtful. It appears to range from a few days to as long as four weeks.

The **symptoms** begin very insidiously, and during the early stages it is often difficult or impossible to be confident of the nature of the disease. In the *mild* cases, the fever is often well advanced before it is suspected. In typical cases, however, the diagnosis may safely be made, usually within a few days after the real onset. A prodromic stage, lasting a week or ten days, sometimes occurs, in which the patient feels "out of sorts." He is easily wearied, and he complains of loss of appetite, headache, and languor. Sometimes there is diarrhœa early, and at other times constipation. A feeling of chilliness may also be present. A few cases begin without warning, and again a very common mode of onset is that of a slowly developing *gastric catarrh.*

When the disease is fairly begun and the patient takes to his bed, a chart of the temperature may afford the earliest indication of the true nature of the affection—the "step by step" rise of the temperature during the early stages being very characteristic of typhoid. The fever has an evening exacerbation and a morning remission, but the course is gradual, as indicated in the chart on next page.

The subsequent course is also shown in the chart—the morning remissions becoming gradually greater. Two relapses are figured. The maximum temperature is generally reached by the fourteenth day. During the first week, the symptoms are those which characterise a gastric catarrh, in which there is diarrhœa, but often constipation. In the so-called "gastric type" the constipation may be present throughout the whole course. About the seventh or tenth day, rose-coloured spots may appear upon the abdomen. They continue to come out in crops—the original ones disappearing, and a fresh crop developing in other regions. They have been compared to flea-bites, and they are not raised above the level of the skin. They disappear upon pressure. The spleen enlargement may be made out at the end of a week, but it is more distinct later.

The stools—in the typical forms—are frequent, and of the "pea-soup" character.* As the disease progresses there is pain upon pressure, and gurgling, in the right iliac fossa. The abdomen is often distended. The patient often complains of aching pains in the limbs and back. Gradually a somnolent condition, and low muttering delirium supervene. In some cases the delirium is violent and furious, while in others it culminates in complete stupor and coma. Picking the bed-clothes, and subsultus tendinum are common, and the urine and fæces are often passed involuntarily. The pupils are generally dilated. The urine is at first diminished, but after-

* A microscopic examination would reveal numerous *micrococci*, and Klebs and others claim to have discovered a special organism.

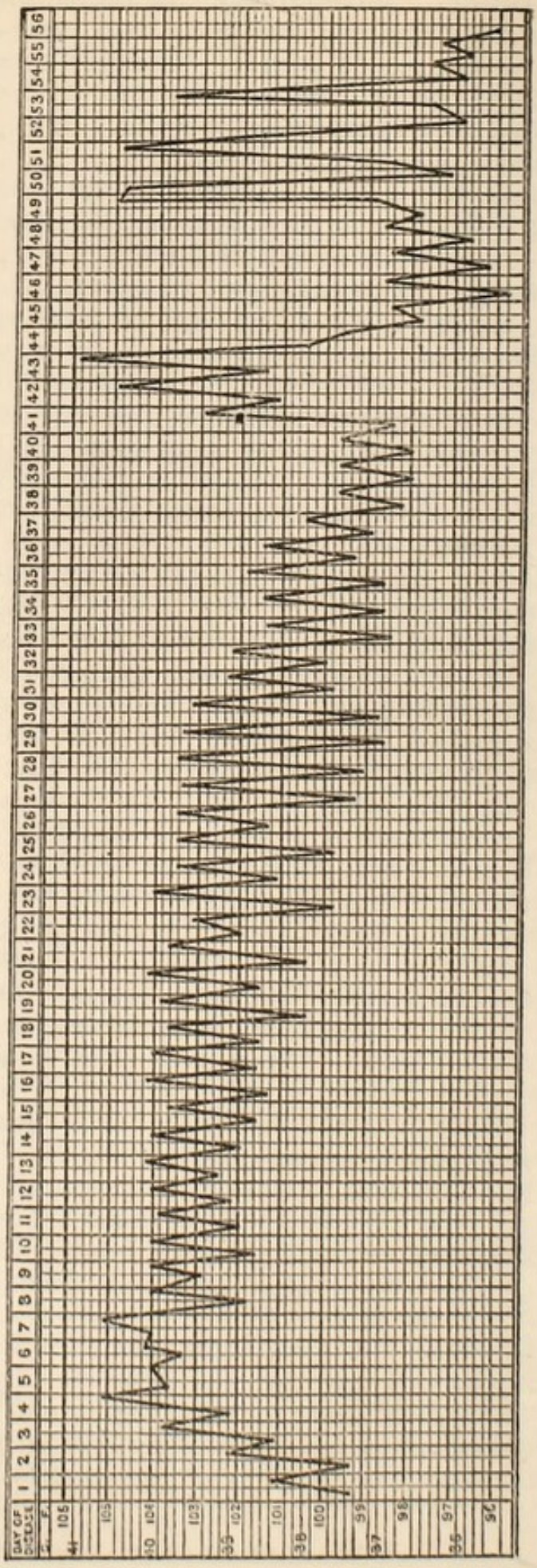

Fig. 41.—Course of the temperature in Typhoid Fever (Wunderlich).

wards it may become increased in quantity. The urea is increased, the chlorides diminished, and albumen is often present. The teeth become covered with sordes, the face thin and pinched, and the lips dry. The tongue in typhoid is red at the edges, and often has the fur in two parallel lines from before backwards. In *putrid* cases the tongue is like a hard, dry, glazed ball. The heart-sounds are indistinct, and the pulse is very frequent and compressible, and the tension very low.

The *course* and *duration* vary. Should recovery be about to take place—generally about the fourth or fifth week—the patient becomes conscious, and the delirium and diarrhœa cease. The temperature gradually becomes normal, the termination being by *lysis*, during which the gastric symptoms improve, and the spleen diminishes, &c. Very mild cases may terminate within three weeks; but usually typhoid runs a course of five or six weeks, and even may be continued much longer as there is a great tendency to relapse. Death may take place from the exhaustion and emaciation which results from a continued high temperature.

During the course of typhoid, *hæmorrhage from the intestines* is a very frequent complication. Sudden collapse and pallor, with a fall of the temperature and a sudden increase in the frequency of the pulse, are the indications that a hæmorrhage has taken place. It may occur at any period. *Perforation* of the bowel is also another possible complication. It occurs in the later stages—from the third to the fifth week—and it is due to extensive ulceration reaching the peritoneum, and opening into the peritoneal cavity. Sometimes it is the spleen or a gland which ruptures into the peritoneum. Sudden pain and distention of the abdomen, a feeble pulse, and symptoms of *collapse* may usher in a fatal termination; or the escape of the contents of the bowel into the peritoneal cavity may produce acute peritonitis—if the patient recover the shock. Death within a few days is the usual result of perforation in such cases. If the perforation take place when the patient is comatose, there may be no apparent evidences of it. The other possible complications described are, thrombosis from cardiac weakness; epistaxis; erysipelas; bronchitis; œdema glottidis; diphtheritic ulcerations; pneumonia and pleurisy; Bright's disease; tuberculosis; bed-sores; mental derangements, &c. In the cases which recover, dyspepsia is commonly present for many months after typhoid fever.

Various *types* are described according to the prominence of certain symptoms, viz.:—the *abdominal*, *gastric*, *nervous*, *putrid typhoid*, and *hæmorrhagic* forms.

The **prognosis** must always be guarded. Should the temperature never rise above 103° F., and should the patient be fairly robust, a favourable issue may be hoped for. Frequent remissions of the temperature make the case still more hopeful, as they spare the patient's strength. The effects of treatment, the age, and temperament, have all to be reckoned in the prognosis. Alcoholic patients do badly. The possibility of complications makes it very hazardous to give any opinion as to the ultimate result.

In the **diagnosis** of typhoid fever the "step by step" rise of the temperature in the early stage, is a highly important symptom. *Gastric catarrh* has no such rise, and the history, subsequent course under treatment, without enlargement of the spleen, or appearance of any of the other symptoms—as the rose-coloured spots, the stools, and pain in the right iliac fossa, &c., will serve to distinguish the simple catarrh. In some works, a **simple continued fever** is described, which seems to be related, or may be regarded, as a separate form of typhoid. The temperature rises slowly and remains about 101° to 103° F., for a few days (or as long as ten days) with gastric symptoms—such as coated tongue, slight delirium, constipation usually, and all the other signs of pyrexia. It terminates without any further developments. It is commoner in hot climates: and it occurs in older people, while the true typhoid fever is more apt to attack the young.

The chief diseases—besides those just mentioned—from which typhoid fever may require to be distinguished, are as follow—viz., typhus fever; general miliary tuberculosis, or acute phthisis in which the physical signs are obscure; ulcerative endocarditis; tubercular meningitis; epidemic cerebro-spinal meningitis; pyæmia; and abdominal affections (peritonitis, typhlitis, &c.). Some of these affections only require to be mentioned in order to be readily excluded, in most cases. Typhus fever occurs rather abruptly, the rash is different and earlier, constipation is the rule, and there are no abdominal signs. The pupils are generally contracted. In tuberculosis, the repeated examination of the chest, and the examination of the choroid for tubercles, may yield important information. The presence of dyspnœa or sweating, and the behaviour of the temperature, or the appearance of the patient, &c., may suggest a clue, in the absence of symptoms peculiar to typhoid. In ulcerative endocarditis the cardiac murmur is marked. In meningitis there is great intolerance of light and noise, whereas in fever, the senses are blunted. The headache increases during the course of meningitis. In pyæmia, the temperature is subject to great variations, and the joints are usually inflamed. The abdominal affections have their physical signs, but none of the symptoms proper to typhoid, and the patient—except sometimes in the later stages—has not the "felled look," so common in fever.

The **treatment** of mild cases of typhoid fever requires little more than the regulation of the diet, and absolute rest in bed, with ordinary nurse's care. No specific remedy is known; but *calomel* in large doses has its advocates. For the reduction of the temperature, quinine, or antipyrin—in twenty grain doses, if necessary—should be used. The cold bath, or wet pack, is another effectual remedy for pyrexia. The bath should be comfortable when the patient is first placed in it. It is then rapidly cooled down. It should never be used when the heart's action is weak, or if there be any tendency to hæmorrhage. A bath should not be continued longer than a quarter of an hour at a time.

Digitalis is sometimes used as an antipyretic; but it cannot be

given when the stomach is irritable. Morphia may be given for restlessness. Astringents, as opium and bismuth, &c., may be prescribed for excessive diarrhœa. Ergotin should be injected, hypodermically, when there is hæmorrhage from the intestines; and morphia, when there is perforation. Ice may be applied to the abdomen when there is hæmorrhage. A tendency to bed-sores is counteracted by the application of alcohol and white of egg. Copaiba with castor oil, is a good dressing for bed-sores. Stimulants are not as a rule required, but any failure of the heart's action should be met by the administration of small doses of whisky, or brandy, in water. They are contra-indicated when there is hæmorrhage.

The milk diet should be continued throughout, and many cases are fed upon *milk* alone. It should be given in small doses (two to four ounces) every three hours. Weak chicken tea may be allowed. Beef tea, sometimes, increases the diarrhœa, and should not then be given. The milk may be diluted with lime water, or barley water, if the stomach be irritable The white of an egg with milk, may be allowed. During convalescence, light milk puddings, chicken, and fish are gradually added; but great caution is required in giving solid food, as relapses are very commonly due to this cause.

The room should be well ventilated; and the stools should be disinfected in the bed-pan before removal. This can be done with sulphate of iron or carbolic acid, &c.

Typhus Fever.—The **pathological conditions** found in typhus consist merely of *congestive* changes, and all the organs are more or less affected. Some slight ulcerations of the intestines may be present. The blood is darker, and not firmly coagulable; and thrombi are common in the veins.

The **causes** of typhus are overcrowding, bad food and air, &c. The nature of the germ is unknown. The disease is very contagious, and it is often epidemic and more virulent during times of famine. One attack exempts from a second. The incubation period extends from a few hours to nine or twelve days—some say even to twenty-one days.

The **symptoms** may begin with a short prodromic stage, in which there is headache, giddiness, sleeplessness, and general *malaise*. Often the patient is suddenly seized, and in a few hours he lies in a state of profound adynamia. The fever has not the gradual "step by step" rise of typhoid, but reaches 103° or 104° Fahr. in two or three days, and is continued with an exacerbation in the evenings. The patient —when the case is a typical one—lies with a completely "felled look." The eyes are half closed, fixed, and dull, and the pupils are contracted; the mouth is open, and the tongue is dry, black, and glazed-looking; the teeth are covered with black sordes; the face and skin are dusky, and the conjunctivæ are injected. Often there are pains in the back and limbs. Typhus cases emit a peculiar odour, and many nurses are able to recognise a case from this symptom. About the fourth to the seventh day, a dark, mulberry-coloured rash appears,

somewhat resembling measles. It generally appears first on the back, and between the shoulders. The spots may be few or numerous, and are slightly raised, and in the early stages they disappear for a moment on pressure. *Crops* of them disappear, and appear, during the second week. It is supposed, by some authors, that the darker the eruption the more unfavourable is the case. At the end of the first week there is active delirium and sleeplessness, or the "low muttering" form with hallucinations and illusions. The state of *coma vigil* is common. The pulse is very frequent and compressible, and the heart-sounds rapid, feeble, and indistinct. The stools are dark and offensive, but there is no diarrhœa. The abdomen is not distended. The urine is scanty—and usually contains albumen. The urea is increased, and the chlorides diminished. The urine and fæces are discharged involuntarily when the patient is comatose. The spleen is enlarged. Bed-sores are common.

The patient may die during the second week from failure of the heart, or from hypostatic congestion of the lungs, &c., or from coma. Many cases recover about the fourteenth day. The return to consciousness is sudden (crisis), and the patient falls into a quiet sleep. The tongue begins to clean, and the skin bursts into a perspiration. The patient is extremely feeble, but gradually the pulse and heart recover strength, and the case passes into convalescence. The *duration* of a case of typhus fever varies from three to four weeks, or longer if there be complications. Some cases run a much milder course than the typical one just described. Instead of coma, there is only confusion of mind and slight fever. The common complications during the course of typhus are the following, viz.:—bronchitis, pneumonia, congestion of the lungs, gangrene of the lungs, pleurisy; erysipelas; thrombosis of the femoral vein; hæmorrhages from mucous tracts and from the kidneys; sometimes paralysis; gangrene of the skin (bed-sores) or of the extremities; &c. Mental derangements often occur as sequelæ to typhus fever.

In the **diagnosis** of typhus fever the following diseases must be noted and excluded, viz.:—typhoid fever; meningitis; uræmia; delirium tremens; pyæmia; and measles. The mistake should not be made of taking some complication—as bronchitis or pneumonia—for the substantive disease. Often, however, it is impossible to diagnose typhus until it has run part of its course.

For the differentiation of the above affections see also the diagnosis of typhoid fever. In uræmia, there is possibly a history of kidney disease, with violent headache and delirium, and often convulsions. Some urine should be drawn off with a catheter (if necessary), and examined for albumen. In delirium tremens, the excitement is different in character, and the nature of the delusions, and the previous history—are generally sufficient to differentiate this affection in the absence of other symptoms peculiar to typhus. In a case of typhus, occurring in a child, measles must be noted. In measles, there is catarrh of the respiratory passages, and the rash is brighter, and appears first upon the face. The typhus rash is mulberry-coloured, and generally appears first upon the back. Some-

times, in pneumonia, there is a rash which strongly resembles that of typhus fever (Grainger Stewart).

The **treatment** of typhus fever is upon the same lines as the treatment of typhoid. As there is greater adynamia and stupor, the bladder should be carefully watched, and the catheter used when necessary. There should be complete isolation, as typhus is very contagious. The excretions should be thoroughly disinfected and removed.

Relapsing (Famine) Fever.—The **pathological changes** are indefinite. The organs are congested, and occasionally wedge-shaped infarctions are found in the spleen, &c. The spiral-shaped organism—*Spirochæte Obermeierii*—is found in the blood during a paroxysm, and it is believed to be the cause of the fever. It develops amongst the filthy, crowded, and unhealthy or weakened population; and it occurs only as an epidemic, and is markedly contagious. The drinking-water is believed to be the chief vehicle by which the disease is carried to the system. The incubation period extends from four or five to seven days.

The **symptoms** begin with general *malaise*, rigors, and a rather sudden development of fever. The temperature ranges rrom 102° to 103° Fahr., with an evening exacerbation, accompanied by all the usual phenomena of pyrexia—as headache, coated tongue, gastric disturbance, increased frequency of the pulse, &c., and pains in the back and limbs. The bowels are confined. The liver and spleen enlarge, and there is pain in the hypochondria. There is weakness and emaciation, but no delirium. The characteristic symptom, which gives the fever its name, occurs about the end of the week, and consists of the sudden fall of the temperature and a cessation of the other symptoms. In three or four days, however, the attack occurs as before, but—if a favourable issue be about to take place—the second seizure is not so pronounced as the first. Several relapses may take place in succession, all of which tend to reduce the patient. The *duration* of an ordinary case (without continued relapses) is about three weeks. Bronchitis and pneumonia are the commonest complications. The **prognosis** is generally favourable, except in very weak and starved people.

The **treatment** is not very satisfactory. Salicylate of soda, in large doses, is recommended. Iron tonics should be given during convalescence. The management, and diet are the same as in fevers generally.

Measles—Rubeola.—Measles is a contagious eruptive fever, associated with a catarrhal condition of the respiratory mucous membranes. One attack usually, but not always, protects from a second. Although a disease of childhood, infants at the breast generally escape. The incubation period extends to nine or eleven days, and some authors say to *fourteen* days.

The **symptoms** begin with chills, headache, and weariness, the temperature rising two or three degrees, rather abruptly. Sometimes

in young children the attack begins with a convulsion. On the first or second day, the hyperæmia of the pharynx, larynx, nares, and conjunctivæ develops. The catarrhal symptoms then appear—as sneezing, watering at the eyes, and cough, with sibilant râles heard on auscultation, &c. Sometimes there is epistaxis. The temperature generally falls on the second day, to rise again, however, about the *fourth*, when the characteristic eruption makes its appearance. The rash comes out first upon the face and cheeks, and it is dark-red, sometimes sharply defined, and at other times accompanied by a diffuse redness. The spots disappear for a moment on pressure. They often run together in crescentic groups. The skin feels uneven, but the elevated papules are smooth and soft. The rash quickly spreads to the trunk, and then, in a less degree, to the limbs generally. It remains *acute* for about twenty-four hours, and about the seventh or ninth day it begins to fade, and the temperature returns to the normal (crisis). During convalescence the skin over the papules is shed in "branny" scales. The cough and catarrhal symptoms continue throughout, and there is abundant mucous secretion from the affected membranes. The case terminates in about a fortnight if no complications supervene. The common complications are catarrhal pneumonia and bronchitis, or croup; diarrhœa; chronic conjunctivitis, iritis or ulcer of the cornea; and inflammation of the middle ear, &c. Phthisis (after catarrhal pneumonia), diphtheria, and Bright's disease are common sequelæ of severe measles. The severity of the attack varies with the strength of the poison apparently, as epidemics may be mild or severe. Sometimes measles has no accompanying catarrh; and sometimes there are cases in which there is no eruption. The eruption is sometimes *suppressed*, and it may appear as late as the tenth or twelfth day—the little patient, during that time, being supposed to be struggling with some bronchial affection only. A *hæmorrhagic* form is described, in which there are hæmorrhages into the skin, and from the mucous membranes, with great weakness and prostration.

The **prognosis** of simple cases is always good; but an excessively high temperature, the hæmorrhagic form, and cases in which the eruption is slight or "suppressed," are unfavourable, and death may result from these conditions, or from severe complications.

The **treatment**, in mild cases, requires only rest in bed, warmth, and, if necessary, a simple diaphoretic and cough mixture (℞ 63). A simple laxative should be given. The skin may be rubbed with vaseline. Quinine may be used, if antipyretic treatment be indicated; and cold baths and packs are sometimes necessary, when the temperature rises and continues above 104° Fahr. The room should be warm, and should be kept darkened. The patient should be isolated. For the bronchial complications, the treatment is the same as in simple bronchitis and catarrhal pneumonia (*vide* p. 91). The diet should consist almost exclusively of milk.

Scarlatina. Scarlet Fever.—In addition to the morbid changes common to all fevers, there are special features in the patho-

logy; but these are described along with the physical signs and symptoms. Scarlet fever occurs as an epidemic, sometimes mild and sometimes severe, and this depends upon the strength of the poison. A germ has been found, but further evidence of it being the cause of the fever is still required. The disease is common in children between the second and fifth years, but it may attack persons of any age. Infants are not liable. A susceptibility to the poison seems to be necessary, as many escape who have been exposed to the contagion, and it is admitted that no fever is more contagious than scarlatina. The breath, skin, and epithelium all contain the poison, and it may retain its power, lurking in the clothes, &c., for many months. Milk is very frequently the vehicle by which the poison is conveyed to the system. Sore throats—without the other symptoms of scarlet fever—are common in those who nurse scarlatinal cases. One attack of scarlatina generally protects from a second seizure, but not invariably. The incubation averages about a week; but the period extends from a few hours to fourteen days (?).

The **symptoms** begin suddenly with chills, headache, and vomiting; and sometimes, in the young child, with a convulsion. The throat symptoms generally develop at the same time, with high fever. The rash appears in about twenty-four or thirty-six hours. It appears first upon the chest and inner parts of the thighs, and after affecting the trunk it spreads to the limbs. In some very mild cases the rash is evanescent, and may escape notice; but, as a rule, it is well developed in twenty-four hours, and gradually fades away in about three or four days. Sometimes the rash is delayed, and appears so late as the fifth day (Scarlatina latens). It is bright red in colour, and diffuse usually, with dark punctiform spots throughout, due to the enlargement of the papillæ. The skin itself is œdematous. The throat is at first intensely congested, the tonsils are enlarged, and ulceration generally follows. There is swelling of the submaxillary glands, and often of the cellular tissue of the neck. The tongue at first is coated with fur, but it assumes a bright red "strawberry" appearance about the fourth day. The urine is scanty, dark red, or smoky looking, and it deposits urates on cooling. It generally contains albumen. The fever runs a continuous course, the temperature reaching 103° or 105° Fahr. almost at once, remaining at about 103° or 104° Fahr. for the next three or four days, when it gradually falls (lysis), and terminates in about ten or twelve days. During the height of the fever there is headache and often delirium. The pulse is increased in frequency in relation to the temperature. The skin becomes moist with the cessation of the fever, and desquamation then begins—scaly or furfaraceous in the mild cases, and large flakes peeling off in the severe. The desquamation continues for six, eight, and sometimes ten weeks, according to the severity of the dermatitis.

During the *course* of scarlatina, there is a tendency to certain complications which must be kept well in mind. A case may begin very mildly, and end very seriously. The cervical glands may suppurate, or a diffuse cellulitis may dissect down the neck. The

throat symptoms may be very severe, and swallowing may be difficult. Deep suppuration may supervene. Sometimes a diphtheritic slough forms and extends up and down the respiratory passages. *Inflammation of the middle ear* is very common. Laryngitis and œdema of the larynx, bronchitis and pneumonia, pericarditis and endocarditis, pleurisy, or peritonitis—are frequent complications. Hyperpyrexia is common in the earlier stages, and it requires vigorous treatment. The commonest complication, however, is *acute glomerulo-nephritis*, and as a consequence of this there is dropsy, and uræmic symptoms may develop at any time during its course. It occurs usually about the *third* week. Acute rheumatism is very common.

A few *types* of scarlet fever are described, according to the prominence of the symptoms; hence, S. simplex, when there is no sore throat; S. anginosa, when the throat symptoms are marked; S. maligna, when the type is so virulent as to threaten life by producing great prostration and nervous symptoms. Death may occur from syncope or from gastro-intestinal irritation; or the fever may produce such adynamia as to be well described as the "typhoid" type.

The **prognosis** of the simpler forms is favourable; but the possibility of even the mild forms assuming a severe type—or terminating in dangerous complications—makes it necessary to be guarded in expressing an opinion of the future course. The malignant forms are highly dangerous to life.

In the **diagnosis** care should be taken to express no confident opinion about a sore throat (especially in children) until the skin has been carefully examined. The scarlatinal rash may not appear until the next day, or it may have been so slight as to have escaped notice, and subsequent desquamation, or Bright's disease, reveals the true nature of the disease. Diphtheria, syphilis, and the ordinary sore throats have to be noted.

The **treatment** of scarlatina is purely symptomatic as no specific is known. At the beginning, drop doses of the tincture of aconite may be given for the fever. Quinine may also be used, and the cold bath or pack may be necessary. If the child be old enough, a gargle of chlorate of potash and warm water, or Condy's fluid (diluted), should be ordered. When not old enough to gargle, the throat may be painted with boroglyceride. Ice may be sucked. For the swelling of the glands, gentle rubbing with warm olive oil is soothing. A wet compress may be rolled around the neck in the early stages when the throat is acutely inflamed. Drop doses, according to the age, of the tincture of belladonna, are useful when the rash is delayed. The skin should be rubbed with carbolised vaseline, and especially should this be carried out regularly during the long stage of desquamation. The diet should consist almost exclusively of *milk*. Complete isolation is necessary; and sheets saturated with carbolic acid should be hung over the doorway of the bedroom in cases treated at home. The treatment of the various complications will suggest itself; and it does not differ from the management of simple con-

ditions of the same nature. The ear must have careful attention. The state of the kidneys must be watched. Suppuration of the glands must be relieved, &c.

Roetheln—German Measles.—This affection partakes of the character of both scarlatina and measles, inasmuch as there is a sore throat, like the former, and a rash and catarrh, like the latter. It is, however, quite a different disease from either, and an attack of one does not protect from either of the others. It is a contagious disease, with a period of incubation of about ten or fourteen days. The **symptoms** begin suddenly with slight *malaise* and fever. The invasion period lasts about four days. The eruption appears upon the breast, arms, and face, and then becomes general. At first, it is quite like measles, but afterwards it becomes more diffuse, and more like the scarlatinal rash. It fades away in about a week or ten days, and is followed by "branny" desquamation. These symptoms are accompanied by more or less catarrh of the respiratory mucous membranes, with sneezing, cough, and mucous expectoration. One attack exempts from another. Adults are often affected. The **prognosis** is almost always favourable. The **treatment** consists of keeping the patient warm, and perhaps the administration of a diaphoretic or cough mixture.

Variola—Smallpox.—The nature of the smallpox virus is unknown. Smallpox occurs at all ages—even affecting the *fœtus in utero*. The negro races are highly susceptible. One attack generally protects from a second, but not always. It occurs as an epidemic, and the pustules—moist or dry—contain the poison, which may preserve its power for many months when adherent to articles of clothing, &c., which have not been exposed to free ventilation. During the course of a case of smallpox, it is contagious from beginning to end. The incubation period extends to ten or fourteen days.

The **symptoms** of a typical case begin with a chill, or a series of them, during which the temperature rises to 103° or 104° Fahr., and it remains high until the stage of eruption, with a slight morning remission. The pulse is full and bounding, and increased in frequency according to the degree of fever. At this stage the pains in the back and limbs are present, and in some cases they are described as agonising. There is nausea and vomiting, headache, sleeplessness, and sometimes delirium and convulsions. The appetite is lost, and the thirst is generally intense. The face is flushed and the conjunctivæ are injected. At this period—the first, second, or even as late as the third day—the *initial* rashes appear. Sometimes they are absent. They are either of the erythematous or hæmorrhagic type, and they appear first on the lower part of the abdomen, genitals, and thighs, and next upon the chest. The erythematous form resembles either the rash of scarlatina or measles—the hæmorrhagic consists of minute extravasations (petechiæ). They fade in about twenty-four hours.

The *stage of eruption* occurs about the fourth day, when the papules appear upon the forehead, face, and scalp, and then they extend to the body. They soon feel hard and "shotty." The temperature falls, but rises again when the papules begin to suppurate. In the ordinary typical cases the eruption is complete in about a day, or a day and a half. On the third day of the eruption the papules become *vesicular;* and about the fifth day, *pustular.* Umbilication of the vesicle is noticed when the vesicle is fully matured. The *stage of suppuration* is reached about the *ninth* day of the disease. The pustules enlarge, the skin around them becomes swollen, and when numerous the œdema is general. The maturation proceeds in the order of the appearance of the papules, and it is accompanied by more or less fever, according to the form of the disease. The distress of the early stages recurs. The itching is often intense, and the delirium often maniacal. The pustules begin to dry up about the eleventh day, and a clear yellow exudation covers the surface of the pustules. At this stage the *odour* is peculiar. As the pustules dry up, the swelling falls, and cicatrices begin to form, leaving the well-known disfigurements of smallpox.

The course of a case varies somewhat with the form. The *discrete* form has the pustules separate and distinct; the *corymbic* has *clusters* of pustules; in the *coherent*, they are in contact; and in the *confluent*, they unite or flow together. *Hæmorrhagic variola* is another form characterised by hæmorrhage into the pustules and extravasation of blood into adjacent parts. There are also bleedings from the nose, gums, kidneys, or uterus, and great prostration.

The *discrete* form is the mildest, and is that form which is most likely to occur in the vaccinated. When the stage of eruption is reached the temperature suddenly falls, and the patient becomes fairly comfortable. In severe cases of the discrete, or in the coherent form, the decline is more gradual. In the *confluent* form there is scarcely any remission of the fever. The mucous surfaces, conjunctivæ, bronchi, vagina, &c., become covered with the eruption, and symptoms will arise according to the seat and irritation. Ulceration of the cornea is common. Stupor and delirium with nervous symptoms are generally marked in the confluent form. There is generally a more acute stage of invasion in this form, and diphtheritic exudations (pharynx, larynx, &c.) are very common. The urine generally contains albumen.

Varioloid is the name given to that modified form of smallpox which occurs in those who have been previously attacked, or who have been vaccinated. It generally resembles a mild attack of the discrete form.

The *course* of smallpox varies with the form. The discrete, corymbic, and coherent may be mild or severe, but they run a more definite course, and the severity is gauged by the amount of the pustular eruption. The *confluent* and *hæmorrhagic* forms are more dangerous. Common complications, or sequelæ, are pneumonia, pleurisy, pericarditis, and pyæmia; ulcers of the cornea, blindness, &c.; chronic ear disease and deafness; abortion in women; suppura-

tions of the joints, abscesses; and diphtheritic affections of the throat or larynx. An ordinary case of discrete smallpox runs a course of about six weeks. The serious forms may extend to several months if complications supervene. Death may result from the severity of the fever, or from intercurrent affections. The **prognosis** must always be guarded; but after a few days when the form is defined, it may be more favourable, in relation to what has been said above.

The **treatment** of smallpox is purely symptomatic, and need hardly be detailed. It consists of the treatment of a *fever*. Quinine is used for high temperatures. Bromide of potassium for headache and delirium. Morphia may be required. Ice in the mouth is very grateful. For the treatment of the face, to prevent pitting, many expedients have been recommended. Cold compresses, painting with tincture of iodine during the papular stage, glyceride of starch, &c., are a few of these remedies. According to Professor Stewart, the prescription of a Hungarian physician of carbolic acid, olive oil, and triturated chalk promises good results (℞ 64). The diet should consist of milk, beef-tea, eggs, &c. A stimulant may be required. Complete isolation and disinfection are necessary. The patient's friends who come in contact with him, or who have been in any way exposed to the infection, should be re-vaccinated.

Varicella or **Chicken Pox** is a mildly contagious disease of childhood. It is characterised by an eruption with slight fever. The papules appear on the first day upon the trunk, and slightly upon the forehead, the number being usually very few (thirty or forty), but sometimes numerous. They become vesicular in a few hours. The fever declines upon the second day, and the vesicles dry up, leaving in some cases a slight cicatrix about the fifth day. It usually occurs as an epidemic. There is still doubt about the period of incubation. It may extend up to the twenty-seventh day (Trousseau). A fortnight is believed to be the usual incubation; and eighteen days is the longest period usually allowed. The *treatment* consists of the administration of a febrifuge if necessary. Generally, nothing is required. The *differential diagnosis*, in severe cases, requires only that smallpox be excluded.

Dengue, Breakbone Fever, is a contagious disease, occurring as an epidemic in tropical countries. It begins with violent rigors, pains, and swellings in the joints, with the appearance of a rash like scarlatina. The temperature falls with the development of the rash; but in a few hours (it may be within three days) a second paroxysm occurs with great prostration and headache, &c., with the development of another eruption, usually beginning upon the palms of the hands and accompanied by intense itching. The termination, after about three days' fever, is by lysis. Desquamation follows. The joints may remain stiff for a considerable time. The incubation period appears to be very short, cases being recorded of almost immediate seizure after exposure to the poison. In the *treatment*, salicylate of soda should be tried. Iron should be given to counteract the prolonged debility which usually follows an attack. The *prognosis* is almost always favourable.

Plague is an epidemic contagious disease characterised by enlargement of the lymphatic glands, with the formation of bubœs, boils, and carbuncles, with hæmatemesis and hæmorrhages from the bowel, &c.; generally ending in death.

Glanders and Farcy are diseases due to one cause. The former is an affection characterised by a foul discharge from the nasal mucous membrane with fever and general *malaise*. The latter occurs when the lymphatics and glands are affected alone, or it may exist along with the discharge from the nostrils. In both forms there is often, also, a pustular eruption on the skin. The face is often swelled, and abscesses may form near the joints. Glanders and farcy are caused by the inoculation of the poison from the horse, ass, and a few other animals that may be suffering from glanders. Grooms, &c., are occasionally attacked. The disease may be acute, and terminate fatally within three weeks. Some cases run a chronic course. There is no *special* treatment known.

Malarial Fevers. Intermittent (*Ague*) and **Remittent Fevers.**—The **pathological changes** consist chiefly of alterations in the spleen, liver, and blood. The spleen becomes greatly enlarged (ague-cake), and often infarctions are present. In the pernicious form of malarial fever, abscesses and gangrene of the spleen have occasionally been found. The liver is also enlarged, and stained with pigment if jaundice should have been present. The white blood corpuscles are increased, and the red blood corpuscles are much diminished in number, during and after an attack of fever. Pigment granules are thrown off which form capillary embolisms (melanæmia). The brain and other organs may be affected by these embolisms, or the organs may be hyperæmic.

The **cause** of the fever is malaria, and it is conceded that a particular germ is connected with the poison which develops in marshy and low-lying localities, and in special regions abroad. The incubation period extends from a few hours to several weeks, but the average is about a fortnight.

The **symptoms** in both the intermittent and remittent fevers generally begin with prodromes. There are weariness and an inclination to yawn; pains in the back and limbs; stomachic symptoms and headaches. Sometimes there is only a tendency to gastric catarrh, with slight jaundice. Other cases, again, begin without warnings.

In **intermittent** fever the onset consists of the development of the *cold stage*, followed soon by the *hot* and the *sweating stages*. In the first stage—which lasts usually a quarter to half an hour—there is a feeling of chilliness, which gradually increases, and is accompanied by shivering. The cold feeling becomes intensified, and the patient hangs over the fire or vainly endeavours to get warmth by heaping up the bed-clothes, &c., upon him. The pains in the back and limbs are generally increased, and often there is nausea and vomiting. The pulse is small, and increased in frequency, and in

tension. The temperature begins to rise at the onset of the chill. At the conclusion of the cold stage, the *hot stage* begins. At first there is a feeling of comfort. The pains are relieved, and the other symptoms are modified. Soon, however, the headache is worse and there is throbbing in the temples. The skin, instead of being blue, and the face pinched, now becomes flushed and warm looking. The pulse becomes full and strong. Vertigo and nausea are present, and there may be transient delirium. After an hour (or sometimes longer) the *sweating stage* is reached. When this occurs the fever declines, and the symptoms are relieved. The sweat is acid, and has an "animal" odour, due to the organic solids and fatty acids present. The urine contains large quantities of uric acid and urates, which are deposited on cooling. The urea is increased. The sweating stage may last twelve hours, at the end of which the patient appears exhausted. There are great variations in the character of the stages, some cases being much milder than others. After an interval, there is a repetition of the three stages. The paroxysm may be daily (*quotidian*), or on alternate days (*tertian*), and so on. Hence there are also *quartan*, *double quotidian*, *double tertian*, *triple tertian*, *duplicated tertian*, &c., &c. The duration of the paroxysm seems to be longer in the *quotidian* than in any of the other types; but it runs a shorter course—the average duration being a month to six weeks.

Sometimes in malarial subjects, instead of an attack of fever there is some other manifestation of malaria. This may be *neuralgia*, *intermittent hæmaturia*, *pulmonary hæmorrhage*, *diarrhœa*, *jaundice*, or *a skin eruption*, &c. Neuralgia is by far the commonest, and it chiefly affects the fifth nerve. The periodic nature of the attack is suggestive, and it may recur like the fever. It is usually *quotidian*. *Sciatica* and *angina pectoris* are also sometimes malarial in origin.

A *pernicious* form of intermittent fever is described. The attack may take this form when the ague follows some other disease which has reduced the vital powers of the patient. The first attacks are not generally fatal; but this result may follow succeeding attacks. The severity and nature of the symptoms have allowed of a classification into several forms, the names of which will suggest the condition. They are as follow, viz.:—the *algid* (in which there is much depression of the heart); the *choleriform;* the *diaphoretic* or *sweating; pernicious icteric* (jaundice and "biliousness"); *nephritic* (hæmaturia and albuminuria); pneumonic; and cerebro-spinal (Jaccoud).

Relapses are very common during the course of intermittent fever, and they are apt to occur at the period—according to the type—at which the paroxysm would have occurred had no treatment been attempted. The result of repeated attacks is to produce in the patient the state and appearances of the *malarial constitution*. There is anæmia from loss of the red blood corpuscles; and the liver and spleen are enlarged and tender, with symptoms of functional disorder very frequently (jaundice, &c.). The ankles may be œdematous; the

skin has a yellow or earthy hue; the individual is thin; palpitation is common; the urine may be albuminous. Waxy disease may affect the organs; or pigment granulations may cause infarctions, &c., by blocking the blood-vessels (emboli). Other sequelæ mentioned are cirrhosis of the liver, dropsy, tuberculosis, mania, &c.

In **Remittent Fever** there are also the three stages, but the fever, although *abating*, does not wholly *depart* for a time. There are, however, distinct remissions, and the "bilious" symptoms are marked—jaundice being usually present. The *remissions* are either *quotidian* or *tertian*. A mild attack lasts four or five days, with all the usual conditions associated with pyrexia—coated tongue, foul breath, nausea, vomiting, headache and throbbing of the temples, &c. The severe forms have fewer remissions, and the very grave forms have delirium and stupor—and the "typhoid" state, with hæmorrhages and sometimes choleraic symptoms, may supervene. The disease—even in its grave form—is not usually fatal in robust subjects. Yellow fever may be mistaken for remittent; but the "black vomit" distinguishes it from the latter disease.

The **prognosis** in malarial fever—intermittent or remittent—is not usually unfavourable. The first seizure is generally the worst; and with prophylactic treatment, succeeding attacks are modified.

In the **treatment** of intermittent fever, *quinine* has displaced all other remedies. It should be given in large doses (twenty grains), about three hours before the paroxysm, when it is possible. It should be continued, in smaller doses, throughout the seizure. The hypodermic injection of pilocarpine (one-twelfth to one-sixth of a grain of the nitrate) is also recommended for the chills. If the gastric symptoms be severe, a grain of calomel, followed in a few hours by a saline purge (Seidlitz powder), is very useful. Phosphate of soda may be given occasionally. Eucalyptus is a good anti-periodic. For the chronic malarial constitution, no remedy is better than *arsenic*.

Quinine is useful as a prophylactic. Five grains in the morning is the usual dose; and exposure to fatigue, to intense heat, and to the early morning and night air of malarial districts, is to be avoided. For the enlarged spleen, the ointment of the red iodide of mercury is highly beneficial. A piece the size of a bean should be rubbed into the skin, in the splenic region, for as long as the skin will permit. In the treatment of the remittent form of malarial fever, quinine is also the best remedy. It should be given at once—a large dose—and continued in smaller doses.

Yellow Fever.—This fever is believed to be of malarial origin, but differs from the preceding two in being infectious. It occurs only as an epidemic in climates where the temperature averages above 70° F.: and it is often endemic on the sea-coast in warm countries. Cold destroys the action of the germ. Filth, and hygienic evils—as bad drainage, &c.—favour its development. It is common on board ships when there has been overcrowding.

The pathological changes are of a general description and consist chiefly of congestions and extravasations of blood into organs and serous cavities. The stomach is inflamed, the liver is yellow in colour, and fatty degeneration is present. The skin is of a deep mahogany colour. The incubation period is one to three days.

The **symptoms** generally begin abruptly with intense pain in the back and high fever. Sometimes there is a prodromic period in which there is general *malaise*. The temperature is at its highest in about twenty-four hours, and it begins then to fall (lysis), and becomes normal at the fourth day. During this stage the stomach is irritable, the tongue coated, the bowels generally constipated, and vomiting is easily excited. Delirium may be present, and jaundice begins to appear. Often at this stage there is remission or even a complete intermission of the fever with favourable symptoms. This may last from one to four days, and sometimes it is entirely absent. Then the temperature rises again, 104° F. being reached on the second day, and this is accompanied by cramping pains. The jaundice becomes marked and the "black vomit" appears. Hæmorrhages are present; and there is usually complete suppression of urine with uræmic symptoms. Should the case take a favourable turn, the temperature falls abruptly, and the above symptoms gradually disappear. Several forms as "the algid, sthenic, hæmorrhagic, and typhous" are described.

In the **treatment** of yellow fever, isolation is important. There is no specific, hence the management only consists of the treatment of the symptoms as they arise, as indicated in fevers generally. Careful disinfection, and quarantine arrangements, must be enforced.

Cynanche Parotidea—Mumps.—This affection occurs as an epidemic usually, and it consists of a specific inflammation of the parotid or other salivary glands. Sometimes the inflammation attacks the breasts or testicles, and especially is this likely to occur during subsidence in the parotid (*metastasis*). There is fever and pain with the swelling, and the face soon assumes a broad and peculiar appearance. The parts are tender to the touch. The stomach is disordered, and the breath foul. Swallowing is difficult. The glands very seldom suppurate, and the subsidence is generally complete within a fortnight. The incubation period extends from five to twenty-four days. The *treatment* is to give mild laxatives, to apply hot fomentations, and to keep the body warm. A diaphoretic mixture may be useful.

Influenza.—This disease occurs as an epidemic, which from time to time spreads over large areas of the habitable globe. It appears to be the result of a germ, which resists all efforts at extinction, and when once established it remains active—at least such has been the experience of the late and the past epidemics—for two to four years, disappearing for months and reappearing in the autumn, and even continuing through the winter if the latter should be a mild

or "open" one. It is doubtful if the disease be actually communicable from one person to another. The period of incubation is short, and one attack does not protect from a second.

The **symptoms** begin suddenly with chill and fever, intense frontal headache, nausea and vomiting, and severe pains in the back and limbs. The temperature rises as high as 103° or 104° Fahr., during the first or second day, and remains, with a remission in the morning, for two or three days, when it returns to the normal (crisis)—if there should be no complications. Often there is acute delirium. The pulse is full, bounding, and increased in frequency. The attack may terminate in a burst of perspiration; or in the discharge of a large quantity of pale urine; or in diarrhœa. The patient is left in a very exhausted state, and it may be three or four weeks before the feeling of excessive weakness passes away. Relapses are very common, especially if the patient leave his bed too soon. Sometimes the disease has not this abrupt course, but begins gradually with slight fever and aching in the limbs and back, described often as "a feeling of having been beaten all over." This form is usually accompanied by catarrhal symptoms, either of the gastric organs or of the respiratory tract. If the latter, there is sneezing, a cough with expectoration, and the physical signs of bronchial catarrh. The patient fancies he has caught a severe "cold;" and, indeed, in many cases, it is impossible to say at the time that such is not merely the case. It is only the long continued nervous debility and lassitude, along with the fact that influenza is prevalent, which give rise to the suspicion that he suffers from the latter affection. The common complications are gastro-intestinal catarrh; bronchitis, pleurisy, congestion of the lungs, and pneumonia; cardiac irritability; inflammation of the external auditory meatus, or of the middle ear; and inflammations of the eye. Pneumonia is very common, and it is remarkable how often (as in alcoholism) it affects the upper lobe of the lung. The chest should be carefully examined, and often the consolidation, fine crepitations, and tubular breathing will be found at the apex. Sometimes, however, no physical signs can be detected, and then—if there be no obvious signs of other complications to account for the continued high temperature—a *deep* pneumonia should be suspected. Phthisis may follow an attack of influenza, in those predisposed to tubercular disease; and mental derangements or weakness sometimes follow an attack.

The **prognosis** is favourable in the robust, but the complications may produce chronic ill health even in them. In the weakly and aged, the sequelæ are sometimes very dangerous, and many deaths occur in this class after prolonged illness from gastric or other complications.

The **treatment** consists of controlling the fever by quinine or antipyrin preparations; the maintaining of the strength by stimulants and beef-tea, and later by a more generous diet; and also the treatment of the various complications as they arise. Pilocarpine may be found useful. Bromides, gelsemium, and even morphia are given for the severe headaches.

Epidemic Cerebro-spinal Meningitis. — Cerebro-spinal fever, as this disease is sometimes called, occurs as an epidemic from time to time in America, Germany, and elsewhere, but it is not often met with in the British Islands. It appears to be due to some miasmatic poison, but as yet the nature of the germ is unknown. It is not believed to be *directly* contagious. It occurs chiefly in young men, and young recruits are specially mentioned as being liable. The pathological changes are those of an inflammation of the meninges, and sometimes also of inflammation of the brain and spinal cord.

The **symptoms** begin abruptly with chills, fever, violent headache, and vomiting, pain in the back, and great prostration. The muscles of the head, neck, and back, are held stiffly to prevent movement, and the surface of the body is painfully sensitive. There is great intolerance of light. The headache is extreme, and there is usually wild delirium and excitement, followed, later, by somnolence or stupor. The limbs are kept flexed. The face is pale, pinched, and frowning. The tongue becomes coated with fur, and the teeth covered with sordes. The pulse is never very frequent in the early stages, but it is often irregular and subject to changes—being at one time fast and at another slow, with variations in the tension. Towards the end of a case about to terminate fatally, the pulse becomes very rapid. In some epidemics *eruptions* on the face and body are present. These may be herpetic, petechial, or like the rash of measles or typhus. Hæmorrhages sometimes occur, and purpuric patches are frequent. A case generally reaches its height in three to six days when either the symptoms become gradually modified, or fatal coma supervenes. Many cases end in suppression of urine with uræmic symptoms. Some cases *abort* early; others quickly produce death by collapse.

In the cases which recover, there is a protracted convalescence of two to three months. Relapses are common. Many cases have only a partial recovery; and paralysis of the cranial nerves, hemiplegia, aphasia, deafness, and mental weakness, &c., are common sequelæ.

In the **diagnosis**, tubercular meningitis and typhoid fever should be noted.

The **treatment** of epidemic cerebro-spinal meningitis consists of the free administration of opium. The hypodermic injection of morphia is the best method. Iodide of potassium may be prescribed; and fly blisters to the spine, and galvanism, are used when the acute symptoms have disappeared.

Cholera (*Asiatic or Epidemic Cholera*). — The **pathological changes** are general in character, except for the presence of the characteristic "rice-water-like" material in the stomach and intestines. The mucous membrane of the gastro-intestinal tract is congested, and marked by extravasations of blood. The basement membrane of the bowel is bared, and the various glands in the mucous membrane are enlarged and prominent. The liver is

fatty; the spleen is smaller and firmer, unless secondary fever have occurred, when it is enlarged and congested; the kidneys are like the *large white kidney*, and the epithelium is granular and often seen cast off into the lumen of the tubules. The serous coverings (peritoneum, pleura, pericardium, &c.) are dry, and have lost their glistening appearance. The lungs are congested. The blood is dark in colour, thick, and feebly coagulable.

The **cause** of cholera is the development of a poison associated with a germ. The latter has been described by Koch as the "comma-shaped" germ, and it is believed to be an important factor in the causation. The poison is conveyed to different countries by clothes and rags, by the air, and by ships, &c., and it then spreads in all directions. Malarial and low-lying regions appear to be more favourable to its development. Cholera is *not directly* contagious; but the stools and fomites contain the germ, and after a short time the poison is developed and is spread through the air, or by the drinking water. The latter is believed to be the most usual vehicle by which the poison is conveyed to the system. *Heat*, and a moist, stagnant atmosphere favour the epidemic. A certain *susceptibility* appears to be necessary; and irregular habits, alcoholic excesses, bad air, fear of the disease, and depression, &c., predispose to attacks. The period of incubation is irregular—one day to a week, and even two, is stated; two to four days is the average.

The **symptoms** are divided into stages. *First, or prodromic stage.* The attack may begin with *diarrhœa* or *cholerine*. The former does not differ from the diarrhœa which results from cold or from errors in diet. There are some colic pains, and the first evacuations are copious, but coloured. In *cholerine*, there is vomiting and purging, and the patient soon passes into the cholera state. It may, however, be controlled, and, like diarrhœa, it may be regarded as an independent affection. Whichever way the attack begins, there is soon continued purging, and the stools soon become like rice-water, or "whey-like." The strength fails very rapidly and it is out of all proportion to the apparent loss. In a day, or two days, there are severe cramping pains in the limbs. The tongue and breath are cold, and the voice is husky and weak.

The *Second, or Algid Stage.*—The rice-water evacuations are increased in frequency, and vomiting begins, if not already present (cholerine). The patient is weak, giddy, and cold. The temperature falls rapidly (about 90° Fahr.). The pulse is rapid, weak, and small, and the heart-sounds become feeble. Cramps are felt, especially in the calves. There is thirst, and the tongue is white and pasty, and the breath cold. The face is pinched and blue, and the eyes sunken and staring, with dark circles around them. The skin appears wrinkled. The urine is diminished in quantity, and may be suppressed altogether. It is often albuminous. The depression is excessive, and death may result at this stage, after only two or three days' illness. If re-action take place, then the *third stage* begins. This simply consists of cessation of the purging, and

the gradual disappearance of the other symptoms. The temperature rises, the pulse regains its strength, and so on. It may, however, be incomplete, and the stomach may remain irritable for some time. Ultimately, there is recovery, but many cases relapse into a "typhoid" state—*cholera typhoid.* This consists of reactionary fever, combined with uræmia. The urine is albuminous, and there are headaches and stupor. The tongue becomes coated and the teeth covered with sordes. The patient has the "felled look" of a severe fever. There is "low muttering delirium," the abdomen is distended, diarrhœa continues, cramps and convulsions are common, and eruptions appear on the hands and spread to the body. Death may take place from coma; but sometimes there is recovery, after a long convalescence.

The average duration of a fatal case is two to three days; and *nine* days is the average duration of cases which recover. The "typhoid" stage may last two to nine days. The mortality is great, and the prognosis is always grave.

The **treatment** of cholera requires that the initial cholerine, or diarrhœa, should be checked by astringents. Opium and dilute sulphuric acid are the best for this purpose. Chlorodyne is useful, and morphia may be used hypodermically. The latter is also required for the severe cramping pains. The diet during this stage is important, and should consist chiefly of boiled milk, milk foods, chicken broth, soft boiled eggs, &c. If the symptoms be threatening all solid foods should be avoided.

Many authors maintain that the use of astringents, after cholera is fairly established, is useless, and some say even injurious. Probably a prescription of carbolic acid and bismuth (℞ 65) is the best to use for both the diarrhœa and the vomiting, Mustard may be applied to the epigastrium to check the latter symptom. Stimulants must only be used in very moderate quantity, and with caution. In the stage of collapse they appear very necessary. Warm baths are sometimes very beneficial. Ice to suck is very grateful. For the uræmic symptoms, give spirit of Mindererus and infusion of digitalis (both well diluted). When the collapse is very great, the injection into the veins of a warm saline solution sometimes produces wonderful results. At the stage of re-action the diet must be very cautiously strengthened, beef tea, chicken broth, gruel, or arrowroot being allowed in small quantities. The patient should, of course, be isolated, and all discharges should be thoroughly disinfected. The linen should be burned. During the prevalence of an epidemic, preventive measures are necessary. Great care should be taken to avoid such food (fruit, &c.) as is likely to set up diarrhœa. Regular hours should be inculcated, and all manner of excesses avoided. Ordinary hygienic precautions—including the use of disinfectants—should be actively carried out. Milk, and the drinking water should be *boiled.*

Diphtheria—**Pathology.**—At first, there is intense hyperæmia of the throat, followed in about twenty-four hours by the appearance

of grey-white points upon the tonsils, uvula, and around the pillars of the fauces. These soon coalesce to form a membrane, which, when examined microscopically, is seen to be composed of pus cells, cellular elements, &c., in which lie embedded numerous colonies of micrococci. Later, this membraneous patch becomes raised and is cast off. In the croupous form of the disease there is an additional exudation of fibrin into the epithelium, and while the micrococci infect the deeper layers causing still further exudation, the first layer separates and is cast off in patches. Should the patch be removed, a raw bleeding surface will be found beneath it. The disease may extend into the nasal passages, or down into the larynx. In septic and gangrenous forms, the membrane becomes decomposed, and numerous bacteria are present. The glands in the neck, and the submaxillary glands enlarge, and the cellular tissue in the neighbourhood is affected by secondary inflammation. Micrococci are present in the lymphatics. Should the membrane extend into the bronchi, there is mechanical obstruction to the breathing, and emphysema, atelectasis, and œdema may be produced. If the lung be affected, there are extravasations and infarctions within its substance, and the micrococci are found there also. In the septic forms, the heart may become fatty; and ulcerative endocarditis is common. The blood is black and non-coagulable. The kidneys are congested, and micrococci are found within and around the Malpighian tufts, and the epithelium is cloudy and granular. The brain is hyperæmic, and the spinal nerve sheaths are often thickened with inflammatory products and extravasations within the sheaths. Swellings and extravasations often occur within the muscle tissues.

Diphtheria is an acute specific and contagious disease, which may arise sporadically, and it often occurs as an epidemic. It is not yet settled whether the micrococci are the cause, or their presence an effect, of the disease; but some sort of organism is believed to originate the poison. The disease is "inoculable and communicable," the poison being carried by the air, clothes, &c. The discharges and membraneous flakes, contain it. The young are more frequently affected—the most common period being between the second and seventh years. Bad hygienic conditions favour its development. Diphtheria often follows scarlet fever and measles, smallpox and typhus, &c. The incubation extends from three to ten or twelve days.

The **symptoms** begin, in the *catarrhal form*, like an ordinary sore throat. There is chilliness and headache, with pains in the back and limbs. The throat feels hot and burning, and there is pain on swallowing. The fever may be mild or severe. When the seizure is violent there may also be nausea, and vomiting with ringing in the ears, &c. The appearances of the throat are as described above; and within a day or two the patches of dirty-grey slough may be considerable in size (but not so large as in the croupous form). These patches become detached about the third day, and the mucous membrane beneath appears red and swollen. The glands, &c., swell; the tongue is heavily coated with white fur;

and the breath is fœtid. The symptoms subside within a few days; but it is to be noted that *diphtheritic paralysis* may follow even the mild attacks.

In the *croupous form* of diphtheria the symptoms may begin like the catarrhal, and about the fourth or fifth day, the membrane assumes the croupous character, with high fever, and swelling of the glands to a greater extent than in the catarrhal form. Sometimes, however, the onset of the croupous form is sudden and violent, with headache, severe pains in the back and limbs, vomiting, and high fever followed often by delirium. The membrane in this form is "yellow-grey," and is "thick, tenacious and leather-like." The appetite is lost, the stomach is catarrhal, and the bowels may be confined, or otherwise. The urine is scanty and contains albumen. The symptoms may decline in about a week, in favourable cases; but while there is apparent improvement the membrane may be extending to the nasal passages or larynx, &c. Croupous diphtheria may, however, begin in the larynx.

Subsequently there is embarrassment of the breathing; the odour of the breath is very offensive; the glands enlarge still more; and a state of adynamia or collapse may supervene. The symptoms become very marked when there is blood-poisoning (septic form). The face becomes earthy and sallow looking, the pulse irregular and often slow, and there is great prostration. The *gangrenous* form is very severe and fatal. When the membrane extends to the nose there is stuffiness and sometimes epistaxis, and a nasal tone is imparted to the voice; when it extends to the larynx there is "croupy" cough, dyspnœa, &c. It may extend up the Eustachian tubes, and set up noises in the ears and deafness; or if the lachrymal duct be affected, there is blocking of the tears, &c. Such severe cases, with complications (if not quickly fatal) may extend to four or six weeks, before convalescence is established.

During the course of diphtheria, and generally about the second week, symptoms of paralysis may begin to be manifest. The palate suffers first, and there is difficulty in swallowing, regurgitation of the food into the posterior nares, and the voice acquires the nasal tone. About the fourth week these symptoms are marked, and deglutition may be very difficult or impossible. The palate hangs limp, and is anæsthetic. The heart is often feeble and very slow, and it may become suddenly paralysed. The respiratory nerves may also be affected.

The **post-diphtheritic** paralyses include the palate, the ocular muscles, the laryngeal muscles, and the muscles of the upper and lower limbs, and of the neck. Any group may be affected; and generally speaking, the paralysis occurs in relative frequency, in the order mentioned. The paralysis often varies in character from day to day. It may follow mild attacks. The muscles first lose their faradic, and later their galvanic irritability. Recovery is the rule, but sometimes there are fatal cases from asphyxia, pneumonia, or intercurrent affections. The paralysis may exist for several weeks only, but often it remains for several months.

The **prognosis** in diphtheria must always be guarded. The septic cases are unfavourable; and extension of the membrane to the larynx is very dangerous, and usually fatal. A low temperature, with a slow, irregular pulse in septic cases, is very unfavourable. Sudden syncope during the course of diphtheria is very common.

In the **diagnosis**, the ordinary sore throats, and the syphilitic and scarlatinal throats must be noted. The dirty grey slough in diphtheria, does not appear until after twenty-four hours. It may be very difficult, or impossible, to differentiate laryngeal diphtheria from membraneous croup; but this question has already been discussed. (*See* Membraneous Croup.)

In the **treatment** of diphtheria, attention should be directed to the local source of infection. The parts should be kept as clean as possible, and for this purpose chlorate of potash, Condy's fluid, solutions of borax, and other antiseptics are used, either as gargles or as local applications. The painting the throat with nitrate of silver solution (10 p. cent.), or with the perchloride of iron mixed with glycerine, is also useful. Caustics should never be employed. The nares may be syringed frequently with weak solutions of Condy's fluid. "Steaming" the nostrils and throat is soothing, and freshly burned lime put into hot water and inhaled is often of great benefit. Spray douches are useful, and disinfectants may be used in this way. Lime water, lactic acid, and quinine are highly recommended for local application by means of a spray (atomizer). Quinine may be used as an antipyretic, and Bartholow strongly recommends a prescription containing the iodide and bromide of ammonium, to be given during the course of the disease. The diet should be generous, and alcohol should be allowed in fairly large doses, frequently repeated. The case should be thoroughly isolated, and great care should be taken to disinfect the discharges. The linen used by the patient should be burned, and the room should be thoroughly disinfected. For the paralytic affections iron and quinine, strychnine and phosphates, should be ordered. Galvanism of the muscles is useful. Tracheotomy may be necessary to relieve urgent dyspnœa; but as a remedy it has not been very satisfactory, as regards the reduction of the mortality.

CHAPTER XV.

GENERAL DATA, USEFUL FOR REFERENCE.

Contents.—Classification of skin diseases—Rules for the management of infants—Incubation periods, and the duration of infection; isolation and disinfection—Certifying the insane—Classification of the poisons according to the treatment—Signs of approaching death.

1. CLASSIFICATION OF THE SKIN DISEASES.

The author has not entered into the diseases of the integumentary system in this work, as he believes that a good skin atlas is preferable to short sketches of the skin affections, and more in keeping with the general objects of this handbook, as mentioned in the preface. The following classification, by Dr. Bulkley, and believed by Dr. Allan Jamieson to be the best, is here appended as a guide.

CLASSIFICATION OF DISEASES OF THE SKIN.

CLASS I. **Morbi cutis parasitici.** Parasitic Affections.
,, II. **Morbi glandularum cutis.** Glandular Affections.
,, III. **Neuroses.** Neurotic Affections.
,, IV. **Hyperæmiæ.** Hyperæmic Affections.
,, V. **Exsudationes.** Exudative or Inflammatory Affections.
,, VI. **Hæmorrhagiæ.** Hæmorrhagic Affections.
,, VII. **Hypertrophiæ.** Hypertrophic Affections.
,, VIII. **Atrophiæ.** Atrophic Affections.
,, IX. **Neoplasmata.** New Formations.

Class I.—Morbi cutis parasitici. Parasitic Affections.

A. VEGETABLE.
- 1. Tinea trichophytina (trichophytosis) (*parasite — Trichophyton tonsurans*)
  - corporis (tinea circinata).
  - capitis (tinea tonsurans).
  - barbæ (sycosis parasitica).
  - cruris (eczema marginatum).
- 2. Tinea favosa (favus) (*parasite—Achorion Schœnleinii*).
- 3. Tinea versicolor (chromophytosis) (*parasite—Microsporon furfur*).

B. ANIMAL.
- 1. Phthiriasis (pediculosis)
  - corporis
  - capitis
  - pubis

  (*parasite—Pediculus*).
- 2. Scabies (*parasite—Acarus scabiei*).

Class II.—Morbi glandularum cutis. Glandular Affections.

A. DISEASES OF THE SEBACEOUS GLANDS.
- I. Due to faulty secretion or excretion of sebaceous matter.
  - 1. Acne sebacea
    - oleosa
    - cerea
    - cornea

    (seborrhœa).
    - exsiccata (xeroderma).
  - 2. Acne punctata
    - nigra (comedo).
    - albida (milium).
  - 3. Acne molluscum (molluscum sebaceum).
- II. Due to inflammation of sebaceous glands with surrounding tissue.
  - 4. Acne simplex (A. vulgaris).
  - 5. Acne indurata.
  - 6. Acne rosacea.

B. DISEASES OF THE SWEAT GLANDS.
- I. As to quantity of secretion.
  - 1. Hyperidrosis.
  - 2. Anidrosis.
- II. As to quality of secretion.
  - 3. Bromidrosis.
  - 4. Chromidrosis.
- III. With retention of secretion.
  - 5. Dysidrosis.
  - 6. Sudamina.

Class III.—Neuroses. Neurotic Affections.

1. Zoster (herpes zoster, zona).
2. Pruritus.
3. Dermatalgia.
4. Hyperæsthesia cutis.
5. Anæsthesia cutis.
6. Dystrophia cutis (trophic disturbances).

Class IV.—Hyperæmiæ. Hyperæmic Affections.

A. ACTIVE.
- 1. Erythema simplex
  - idiopathicum.
  - traumaticum.
- 2. Roseola.

B. PASSIVE.
- 1. Livedo mechanica.
- 2. Livedo calorica.

Class V.—Exsudationes. Exudative or Inflammatory Affections.

A. Induced by Infection or Contagion.
1. Rubeola (morbilli, measles).
2. Rubella (rötheln).
3. Scarlatina.
4. Variola.
5. Varicella.
6. Vaccinia.
7. Pustula maligna.
8. Equinia (glanders).
9. Diphtheritis cutis.
10. Erysipelas.

B. Of Internal or Local Origin.
- I. Erythematous.
  - 1. Erythema. (multiforme. nodosum.)
  - 2. Urticaria.
- II. Papular.
  - 3. Lichen. (simplex. planus. ruber. scrofulosus.)
  - 4. Prurigo.
- III. Vesicular.
  - 5. Herpes. (febrilis. iris. progenitalis. gestationis.)
- IV. Bullous.
  - 6. Hydroa.
  - 7. Pemphigus. (vulgaris. foliaceus.)
  - 8. Pompholyx (cheiro-pompholyx).
- V. Pustular.
  - 9. Folliculitis barbæ (sycosis).
  - 10. Impetigo.
  - 11. Impetigo contagiosa.
  - 12. Ecthyma.
- VI. Multiform, *i.e.*, erythematous, papular, vesicular, pustular, &c.
  - 13. Eczema.
  - 14. Dermatitis. (calorica. venenata. traumatica.)
- VII. Squamous.
  - 15. Dermatitis exfoliativa (pityriasis rubra).
  - 16. Psoriasis.
  - 17. Pityriasis capitis.
- VIII. Phlegmonous.
  - 18. Furunculus (furunculosis).
  - 19. Anthrax (carbuncle).
- IX. Ulcerative.
  - 20. Ulcus (simplex. venereum.)
  - 21. Onychia.

Class VI.—Hæmorrhagiæ. Hæmorrhagic Affections.

1. Purpura (simplex. papulosa. rheumatica (peliosis rheumatica). hæmorrhagica.)
2. Hæmatidrosis (bloody sweat).
3. Scorbutus.

Class VII.—Hypertrophiæ. Hypertrophic Affections.

A. Of Pigment.
1. Lentigo.
2. Chloasma.
3. Melanoderma.
4. Nævus pigmentosus.
5. Morbus Addisonii.

B. Of Epidermis and Papillæ.
1. Keratosis pilaris (lichen pilaris).
2. Ichthyosis.
3. Cornu cutaneum.
4. Clavus.
5. Tylosis (callositas).
6. Verruca: vulgaris. senilis. acuminata. necrogenica.

C. Of Connective Tissue.
1. Scleroderma.
2. Sclerema neonatorum.
3. Morphœa.
4. Elephantiasis Arabum.
5. Dermatolysis.
6. Frambœsia (or yaws).

D. Of Hair.
1. Hirsuties.
2. Nævus pilosus.

E. Of Nail.
1. Onychogryphosis.
2. Onychauxis.

Class VIII.—Atrophiæ. Atrophic Affections.

A. Of Pigment.
1. Albinismus.
2. Leucoderma (vitiligo).
3. Canities.

B. Of Corium.
1. Atrophia cutis: maculosa (maculæ atrophicæ). propria. linearis (striæ atrophicæ).
2. Atrophia senilis.

C. Of Hair.
1. Alopecia.
2. Alopecia areata.
3. Trichorexis nodosa (atrophia pilorum propria, or fragilitas crinium).

D. Of Nail.
Atrophia unguis.

Class IX.—Neoplasmata. New Formations.

I. Benign New Formations.

A. Of Connective Tissue.
1. Keloid.
2. Fibroma (molluscum fibrosum).
3. Xanthoma (xanthelasma, or vitiligoidea).

B. Of Granulation Tissue.
1. Lupus: vulgaris. erythematosus.
2. Rhinoscleroma.
3. Scrofuloderma.
4. Syphiloderma.

C. Of Blood-Vessels.
1. Nævus vasculosus.
2. Angioma (telangiectasis).

D. Of Lymphatics.
1. Lymphadenoma cutis.
2. Lymphangioma cutis.

E. Of Nerves.
Neuroma cutis.

II. MALIGNANT NEW FORMATIONS.

1. Lepra	tuberosa / maculosa (elephantiasis Græcorum).
2. Carcinoma	epitheliomatosum (epithelioma and rodent ulcer). / papillomatosum (papilloma).
3. Sarcoma.	idiopathicum. / pigmentosum (melanosis).

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2. RULES FOR THE MANAGEMENT OF INFANTS.*

1. Warmth, Cleanliness, Fresh Air.—Keep them warm: let the clothing be warm, but not tight. Wash them all over with warm water daily, wiping them thoroughly dry afterwards. Give them plenty of fresh air: send them out, at least for a short time, every day that the weather is fine; and, while they are out, air the room, by freely opening the window.

2. Nourishment while the Child is under Seven Months old.—The mother's milk is the most natural, and accordingly the proper food for infants. Therefore, if the mother has plenty of milk, let her suckle her child and give it *nothing else* till it is seven months old. If the mother has too little milk, still let the child have what there is; and, in addition, cow's-milk and water, as directed in Rule 3. Till the child is seven months old, milk must be its *only* food.

3. How to bring up "by hand."—If the child *must* be brought up by hand, it should be fed with milk and water out of a bottle. At first, there should be nearly as much water as milk; but when the child is a month old, two parts of milk should be mixed with one of water: after this, the proportion of milk should gradually still further be increased, till, at four or five months, it is given plain. If, at any time, the milk seems to disagree, a tablespoonful of lime water should be added to each bottleful. *Give the child no other nourishment whatever.* A very large number of the children that are brought up by hand die in childhood; and this mortality is for the most part due to the practice of beginning too soon with gruel, corn-flour, &c. These are not proper nourishment for children under seven months old, and should never be given to them. While the child is under a month old, do not give it more than half a teacupful of milk and water at a time. The bottle should draw easily. It should be very carefully washed out after every time it is used. Then bottle, cork and tube should be kept separately in a bowl of clean water till next time they are needed. If the bottle is not quite clean, the milk may sour, and may thus make the child ill.

4. Importance of Regular Feeding.—The child should be put to the breast *regularly:* for the first six weeks, during the day, in general not oftener than every two hours; afterwards about every three hours. During the night, it does not need to be fed so often. A child soon

* Used at the New Town Dispensary, Edinburgh.

learns regular habits as to feeding. It is a very great mistake, to give the breast to the child whenever it cries, or to let it be always sucking, particularly at night: this is bad for both mother and child. If the child is brought up by hand, it should be fed with the same regularity: never give it the bottle *merely* to keep it quiet. If the child is weakly, the intervals between the feedings must be somewhat shortened, both during the day and during the night.

5. Nourishment when the Child is over Seven Months old.—If at seven months, the child is strong and healthy and has cut a few teeth, it may now have one or two meals a day of milk slightly thickened with good well-baked bread or well-boiled porridge. *It should still have*, besides this, *plenty of plain breast or cow's milk.* At ten months, it may once a day have a little meat-broth made with barley or rice, without vegetables. At twelve months, it should be taken from the breast. Till the child is two years old, no solid animal food should be given. *Even at two years, milk should still be the chief food.*

If at seven months, the child is weakly or sickly or is backward in teething, milk must remain the only food for some time longer.

6. Avoidance of Stimulants, &c.—Tea, beer, whisky, and other stimulants, cheese, fruit, and pastry, as also "soothing-medicines," "sleeping-draughts," "cordials," "teething-powders," &c., *should never be given;* and even ordinary medicines should, if possible, be given only after proper medical examination and advice.

General Rules for Infant Feeding.

From Keating's *Diseases of Children* (Rotch).

Age.	Intervals of Feeding.	Number of Feedings in 24 Hours.	Average Amount at each Feeding.	Average Amount in 24 Hours.
1st week	2 hours	10	1 oz.	10 oz.
1 to 6 weeks	2½ hours	8	1½ to 2 oz.	12 to 16 oz.
6 to 12 weeks, and possibly to 5th or 6th month	3 hours	6	3 to 4 oz.	18 to 24 oz.
At 6 months	3 hours	6	6 oz.	36 oz.
At 10 months	3 hours	5	8 oz.	40 oz.

3. INCUBATION PERIODS AND THE DURATION OF INFECTION. ISOLATION AND DISINFECTION.

Incubation periods (full extent).

Erysipelas,	7 days.
Diphtheria,	12 days.
Pertussis, Measles, Scarlatina, Roetheln, Small-pox	14 days.
Chicken-pox,	18 days.
Typhus fever,	21 days.
Mumps,	24 days.
Typhoid fever,	?
Influenza	?

A boy who has been exposed to infection, should be carefully disinfected and isolated, and should not be allowed to return to school until the full extent of the incubation period has elapsed. All books, &c., which have been exposed to infection, should be destroyed. In cases where the boy has been exposed to infection, but has previously had the disease, he must still be quarantined—except in the case of pertussis, roetheln, chicken-pox, or mumps, when one day's detention (with disinfection) will be sufficient.

Duration of infection; when disinfection, &c., is carried out. A boy may return to school as stated below, viz. :—

Erysipelas.—After desquamation has entirely ceased. Generally three to four weeks.

Diphtheria.—After recovery; and *not less* than three weeks from the beginning of the illness.

Pertussis.—When the spasmodic cough has gone—generally six weeks from its first appearance.

Measles.—When desquamation has entirely ceased—two to four weeks.

Scarlatina.—When desquamation has entirely ceased—six, eight, or even ten weeks.

Roetheln.—In fourteen days from disappearance of rash.

Small-pox.—In six weeks after the crusts have disappeared.

Chicken-pox.—In a week after the last crust has disappeared—generally three weeks from the appearance of the vesicles.

Typhus fever.—When convalescence allows of it—never less than fourteen days, in the very mild cases.

Mumps.—In fourteen days after all swelling has disappeared.

Typhoid fever.—When convalescence allows of it—sometimes two or three months.

Influenza.—When convalescence allows of it—very variable.

Ringworm.—A week after effective treatment has been carried out; but the case should still be watched.

In the treatment of infectious disease, *isolation* of the patient, and *disinfection* of the sick-room, clothes, and person, &c., are of the highest importance. When the case cannot be removed to an hospital for infectious diseases, a room must be selected as far from the other members of the family as possible. All curtains, rugs, carpets, and hangings, should be removed; and old sheets, saturated with carbolic lotion, should be hung over the doorway both inside and outside. When possible, a room communicating with a bath-room and dressing-room should be used, and the whole suite completely isolated. The nurse should observe great care, when passing from the sick-room, that her dress does not convey infection outside. Her dress ought to be changed in the ante-room (bath-room or dressing-room) before passing through the passage or hall. If practicable, a separate entrance should be used.

In certain cases, the stools from the patient require disinfection before being cast into the closet. Sulphate of iron, or strong carbolic acid, may be used for this purpose. All crockery, &c., used at meal times, and required outside the sick-room again, must be washed and rinsed out with dilute carbolic lotion before being returned to the general household.

For the disinfection of the patient, the skin in many cases (as in measles and scarlet fever) requires to be daily anointed with carbolised vaseline. The linen used by the patient should be either burnt or placed in boiling water, to which some Condy's fluid has been added. In some cases, as typhus, small-pox, cholera, &c., the *burning* of the linen is imperative. When the patient is considered free from infection, there should be a final bathing with carbolic soap—special attention being given to the hair—and finally he emerges from the sick-room in a state of nudity to receive fresh linen and clothes in a different bedroom. It is well to arrange—in the case of children after scarlatina, for example—for the patients being sent direct to some place for convalescence, without coming in contact with other children for another week or two. For the final disinfection of the sick-room there is nothing like plenty of fresh air. The walls should be stripped of the paper, and after removing the irons, sulphur pastilles may be lighted and the doors tightly closed. The pastilles should be placed on spars of wood laid across tubs of water, as the sparking from them may be dangerous if not surrounded in this way. After a few hours, the doors and windows may be opened. The rooms are thoroughly washed and scrubbed with strong carbolic soap and water. All mattresses, bedding, and clothing which may not be destroyed, should be sent away to be subjected to a high dry heat, which is the most effectual means of destroying germs. In the severe infectious diseases it is safer to burn the bedding, &c. The room, after being thoroughly disinfected, should remain untenanted and freely ventilated for some time afterwards. The drains should be inspected, and thoroughly flushed with water and crude carbolic acid.

4. CERTIFYING THE INSANE.

Having first got the history of the case, proceed with the examination as if the man were sane. In extreme cases where it may be necessary to conceal the fact that you are a physician called in to examine the mental state, the conversation can only be general; but if known to him, then it is convenient to suggest that his friends consider his health to be impaired, and that he requires medical advice. No mention need be made that it is insanity that is suspected. The information supplied by the friends will often suggest the line of examination and save much time. When the patient, however, is suspicious, another visit may be necessary before a certificate can be conscientiously filled up.

During the conversation which follows, note the *expression of his face and eyes, attitudes, and manner*, &c. The expression may be *weak, silly, vacant, stupid, or imbecile.* Often there is extreme *restlessness and excitability*, and such cases are generally easy to certify, as the delusions very soon are manifest. The angry, morose, or suspicious subject is more difficult to manage; but *taciturnity, and refusal to answer simple questions* (put in a mild and soothing way) are accepted as evidence, and worthy of noting in the certificate. The best evidence to note in the "*facts indicating insanity observed by myself*"—is some *obvious delusion.* The *memory* should be tested in some simple and general way—as asking the day and date, his age, how long he has been in town, &c. The *speech* may be rambling and incoherent. A question may be answered absurdly, and the reasoning power may be quite lost. Many cases are *noisy*, the patient *shouting or outrageous and threatening* in his manner. Any *suicidal or homicidal* tendency should be noted. When a ridiculous statement is made be sure to add "which is a delusion," if it be not obvious. The *self-control* may be tested by contradiction, or otherwise producing some irritation in the patient. The temperature should be taken in all suspicious cases, as the delirium of fever may simulate a case of insanity.

The facts recorded by others should always have the name of the informant included in the certificate, and it is well to read over the whole certificate carefully after filling it up, as the omission of certain parts may be important from a legal point of view. Clouston suggests that the following conditions should be noted and excluded in the examination of the insane, viz.:—"Drunkenness, drugging by opium or other narcotics, meningitis, cerebritis, brain syphilis, the fevers, sunstroke, traumatic injury to the head, hysteria, the cerebral effects of gross brain diseases, simple *delirium tremens*, the temporary cerebral effect of moral shock, or the delirium which precedes death in many diseases and in old age." He adds, however, that "many of these conditions and diseases may lead to, or be associated with, real mental disease, and require treatment as such."

5. CLASSIFICATION OF THE POISONS ACCORDING TO THE TREATMENT.

(From the author's *Synopsis of Therapeutics*, published by Y. J. Pentland.)

POISONS.

All cases of poisoning require *general*, and most cases require also *special*, treatment.

General Treatment.

This depends upon the condition and symptoms manifested by the patient, and consists of the proper use of the following, viz. :—

1. **Stimulants.** — Brandy. Ammonia. Camphor. Coffee (an enema if necessary). Rousing. Cold. Battery.
2. **Sedatives.**—Morphia. Chloroform (for spasms, &c.). Demulcents (gruel, arrowroot, barley-water, milk, raw eggs, linseed tea, or oil). Warmth. Poultices. Recumbent position.
3. **Artificial respiration**—may be necessary.
4. **A purgative**—may be required.

Special Treatment.

The poisons may be classified thus :—

DIVISION I.—POISONING NOT TREATED WITH EMETICS, NOR WITH THE STOMACH TUBE.

Group 1.

Acetic Acid. **Sulphuric Acid.** **Oxalic Acid.** (Oil of Lemons.) **Tartaric Acid.** *** Hydrochloric Acid.** *** Nitric Acid.**	Give quantities of *Soap* and *Water*, *Chalk*, *White-wash*, or *Magnesia*.

Group 2.

Caustic Potash. **Caustic Soda.** **Ammonia.**	Give *Vinegar*, *Oil*, and *Demulcents*.

Group 3.

Carbonic Acid (Carbonic Oxide). **Chlorine Gas.** **Coal Gas.** **Sewer Gas.**	*General Treatment.*

*The bicarbonates of potash or soda, and ammonia (well diluted), are even better antidotes for hydrochloric and nitric acids, but *not* for oxalic acid.

Division II.—Poisoning treated with Emetics and Stomach Tube.*

Poisons with Special Antidotes.

Group 1.

Arsenic.—Hydrated Peroxide of Iron (freshly prepared, by adding Carbonate of Soda to Steel drops and filtering).
Barium.—Epsom Salts or dilute Sulphuric Acid.
Belladonna (Atropia, Henbane, &c.).—Pilocarpine (hypodermically, in ½ gr. doses, and repeated if necessary).
Carbolic Acid (and Creasote).—Epsom or Glauber's Salts well diluted, or Saccharated Lime. White of egg (raw).
Chloroform (swallowed).—Carbonate of Soda (well diluted). Nitrite of Amyl (inhaled).
Chromic Acid (and Bichromate of Potash).—Chalk. Carbonate of Magnesia.
Copper.—Eggs (raw). Milk.
Curara.—Strychnia.
Cyanide of Potassium.—Sulphate of iron (well diluted). Atropia.
Mercury. { Corrosive Sublimate, / Red and White Precipitate, } White of egg (raw).
Iodine.—Starch.
Lead.—Dilute Sulphuric Acid. Epsom Salts. Glauber's Salts.
Phosphorus.—Sulphate of Copper. French Oil of Turpentine. *N.B.*—Do not give oils or fat. Use Epsom Salts as the purgative.
Picrotoxine (Cocculus Indicus).—Chloral. Bromides.
Silver.—Salt. White of egg (raw).
Stramonium.—Pilocarpine.
Zinc (Burnett's Fluid).—Carbonate of Soda or Potash (well diluted). Tannic Acid. Eggs.

Poisons with Atropia as the chief antidote. (Use $\frac{1}{60}$ gr. hypodermically, and repeat the dose within 20 minutes, if necessary.)

Group 2.

Aconite.
Benzoine.
Caffeine.
Calabar Bean.
Gelsemium.
Jaborandi.
Muscarine (Poisonous Mushrooms).
Mussel.
Nitre.
Nitrite of Soda.
Nitrobenzol.
Nitroglycerine.
Opium and Morphia.
Prussic Acid (Oil of Bitter Almonds).
Resorcin.

*Sometimes these cannot be used, and often they are not required,—*e.g.*, in strychnia, jaborandi, curare, gelsemium, nitroglycerine, and zinc poisoning.
In poisoning by carbolic acid and cyanide of potassium give the antidote before the emetic.

N.B.—Additional antidotes are, Digitalis for Aconite; Morphia for Caffeine; Chloral and Strychnia (in bad cases, $\frac{1}{60}$ gr. of the sulphate, hypodermically), for Calabar Bean; and Ergot for Nitro-glycerine.

Poisons with Tannic Acid as the chief antidote. (Use 30 grs. for a dose.)

Group 3.

- **Antimony.**
- **Colchicum.**
- **Conium.**
- **Digitalis** (and Convallaria).
- **Ergot.**
- **Lobelia.**
- **Strychnia** (and Nux Vomica).
- **Tobacco** (and Nicotine).
- **Veratria.**

N.B.—Additional antidotes are, Aconite for Digitalis; Strychnia ($\frac{1}{60}$ gr. of the sulphate, hypodermically), for Lobelia; Bromides and Chloroform for Strychnia.

Poisoning treated on general principles (*see ante*).

Group 4.

- **Alcohol.**
- **Arum Maculatum** (Lords & Ladies, &c.).
- **Bryony.**
- **Camphor.**
- **Cantharides.**
- **Chloral** (and Paraldehyde).
- **Colocynth.**
- **Croton Oil.**
- **Laburnum.**
- **Nightshade.**
- **Nitrite of Amyl.**
- **Paraffin Oil.**
- **Privet.**
- **Savin.**
- **Turpentine.**
- **Yew.**

Vermin Killers—generally contain Strychnia.
Crayons—contain Lead, Arsenic, Chromic Acid, &c.
Fly Papers—contain Arsenic (generally).
Hair Dyes—contain Lead (generally).
Rat Pastes—contain Phosphorus and Arsenic.
Neuraline—contains Aconite.

N.B.—**Liniments** are often compound, and complicate the treatment; as Liniment of Aconite, Belladonna, and Chloroform; Liniment of Belladonna and Morphia, &c.

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6. SIGNS OF APPROACHING DEATH.

In the progress of a case, the recognition of unfavourable symptoms is of great importance. In "sudden" or "unexpected" deaths, there are no warnings; although there may have been some premonitory symptoms, or a short illness which has not excited any alarm or anxiety. It is, however, during the *course* of a disease—especially of a chronic case—that one should be on the alert for a "change,"

or for the development of signs which indicate the approach of a fatal termination. Towards the end of a case the symptoms shade into one another, no matter what system of the body is originally affected; but they differ in the early stages.

In diseases of the *circulatory organs*, any alteration in the character of the breathing, or gradually increasing dyspnœa, is significant. The expression becomes intensely anxious, and the patient requires to be propped up in bed. The breath becomes cold. The heart-sounds, and murmurs, may be distinct enough, owing to the excited action of the heart; but they become feeble towards the end. The pulse becomes irregular, rapid, and feeble. The veins are distended and pulsating. Ultimately, the breathing becomes more and more shallow, and if some bronchitis be present —as is usually the case—the mucus collects in the bronchial tubes and trachea, and the râles are loud and coarse. The extremities become cold and pale. Cold sweat often breaks upon the skin and forehead. Sometimes there are slight convulsions, and there may be angina pectoris. Gradually the patient becomes unconscious, the eyelids are half-closed, and the eyes fixed and glassy. Sometimes the termination is more sudden (syncope); and when clots obstruct the pulmonary vessels the dyspnœa (or convulsions) becomes urgent, and the end quickly takes place. When any of the secondary complications of heart disease are present, there will be additional symptoms according to their character. Sometimes a heart case terminates by *collapse*. In such cases, the features become pinched, shrunken, and livid. Cold perspiration breaks out upon the body; the breathing becomes shallow, irregular, and sighing; the pulse, quick, irregular, feeble, or imperceptible; and often there is hiccough or vomiting. Transient delirium or convulsions may supervene.

In diseases of the *respiratory organs*, the heart is often secondarily affected, and the train of symptoms ushering in death, may be similar to those above described. In many cases death takes place from carbonic acid poisoning, the accumulation of mucus or inflammatory products interfering with the aëration of the blood. The râles in the trachea become distinct; and they become less and less removed by coughing. The breathing becomes more and more difficult, and the patient gradually becomes unconscious, and dies from asphyxia.

In *renal disease* the manner of death may depend upon the nature of the complications. These may be relieved during the course of the case, or they may prove directly fatal. In chronic cases, when the complications do not immediately threaten life, death is due to the non-elimination of effete matter, so that the gradual onset of *uræmia* with its train of symptoms, must be carefully watched, and delirium, eclampsia, and coma may terminate the case.

In diseases of the *digestive system*, the acute cases may terminate in collapse. In the chronic cases which prove fatal the changes are very gradual. Leaving out of consideration the special symptoms peculiar to each disease, death generally results from emaciation and failure of nutrition. Such affections as are associated with

vomiting and diarrhœa, or cases of stricture of the œsophagus and stomach, &c., often terminate by starvation. The patient gets weaker. There is languor, a feeble circulation, and coldness of the extremities. The hands, feet, nose, and ears get dusky. The breathing gets more and more shallow. The pulse at last becomes almost imperceptible, and any attempt to sit up or move produces faintness. Such symptoms are common to all chronic diseases in which there is slow wasting away.

In fevers generally, the *typhoid state* is the commonest mode of termination.

In diseases of the *nervous system*, the chronic spinal affections either terminate ultimately in extension to the vital parts, or by some intercurrent affection. In brain disease, the termination of acute cases is generally by coma, and often by convulsions. The patient becomes gradually unconscious, in many cases; the breathing becomes irregular and stertorous; secretions accumulate in the bronchial tubes; and death appears to end by asphyxia.

In simple *senile decay*—beyond the obvious appearances of gradual failure—it is often impossible to detect any physical sign which would indicate that death was near at hand. There is often mild delirium; and in many cases there is wild and noisy excitement for a few days before the unconsciousness which supervenes and ushers in the fatal termination. Many cases, certainly, run a course similar to the chronic wasting diseases already described; but others, again, when much debilitated, seem to die of sudden failure of the nervous apparatus of the heart. The previous visit may not have enabled the physician to detect any change in the circulatory organs.

CHAPTER XVI.

POST-MORTEM EXAMINATION.

(Summarised, by permission, from Woodhead's *Practical Pathology*.)

In some cases it is advisable to get a history of the case, and especially when the death has been a suspicious one, or due to some accident, &c. Note the time the patient died, and the interval between the death and examination. Note the colour of the various parts of the body; the *post-mortem* lividity; the appearances of wounds, abrasions, &c.; and the degree of *post-mortem* rigidity. In systematic examinations, the *post-mortem* case-book—containing important headings, as name, age, height, &c.—should be followed.

Head.—After the external examination, an incision is made behind the ear and carried over the vertex of the skull to the same point on

the other side. The scalp is then dissected and reflected forwards and backwards until the eminences over the frontal sinuses and the occipital protuberances are exposed. After examination of the soft parts, carry the knife round the skull at the level above indicated. The saw is then used carefully, and the skull-cap loosened by the aid of the chisel, mallet, and lever. The skull-cap can generally be removed by dragging on the fore part; but sometimes the adhesions render a good deal of force necessary. A small opening is made in the dura-mater on each side, just above the bony margin, and a blunt-pointed bistoury is introduced, and the incision carried round to the mesial line on each side, backwards and forwards; then cut through the attachment of the membranes to the *crista galli*, and draw them back. After examination of the inner surface of the membranes, remove the brain. Introduce the fingers of the left hand beneath the frontal lobes, and gently tilt the brain backwards, severing in the following order, the olfactory bulbs; optic nerves; internal carotid vessels; third, fourth, and sixth pairs of nerves—the latter being divided along with the tentorium. The fifth and seventh pairs are then divided, and the incision carried along the margin of the tentorium, dividing that membrane from the petrous portion of the temporal bone. Cut through the eighth and ninth nerves, and then cut through the cord as low down as possible, and tilt the brain out carefully. Examine the inner surface of the dura-mater at the base of the skull. The dura-mater may afterwards be detached and the bones examined—especially the petrous portion of the temporal bone.

The Brain.—"With a long thin narrow-bladed knife cut horizontally from within outwards into the hemisphere, just above the level of the corpus callosum, leaving the upper part of the brain attached to the lower by the pia mater only, at its outer margin; make a similar incision into the opposite hemisphere." Examine the lateral ventricles and their contained fluid, by cutting vertically down into the corpus callosum, at a distance of one-sixteenth of an inch from the mesial plane. The depth is only about one-eighth of an inch. Extend the incision backwards and forwards, to expose the ventricle, and note the quantity of fluid which escapes. Then divide and subdivide the upper portions of the cerebral hemispheres already turned outwards—cutting from within outwards, and never completely separating the lamellæ. To open into the anterior horn of the ventricle, cut horizontally into the frontal lobe a little below the level of the body of the cavity, removing the brain substance above the incision. The posterior horn is opened in a similar way. Separate the pons, medulla, and cerebellum from the large brain, "by cutting towards the mesial line in a plane, the anterior border of which is just in front of the pons, the other border lying immediately behind the posterior pair of the corpora quadrigemina; a similar incision is made from the opposite side." "Having determined the contents of the lateral ventricles, the state of their walls and venous plexus, and the condition of the septum, the latter is taken hold of with the left hand, close behind the foramen of Monro, the knife is

pushed in front of the fingers through this aperture, and the corpus callosum cut through obliquely upwards and forwards, and then all these parts (corpus callosum, septum lucidum, and fornix) are carefully detached from the velum interpositum and its choroid plexus. After these two latter have been exposed, we have to examine the state of their vessels and tissue. Then the handle of the scalpel is passed from the front under the velum, which is thus detached from the pineal body and corpora quadrigemina, the state of these parts is determined, and the third ventricle now exposed" (Virchow). A vertical incision through the corpora quadrigemina opens into the aqueduct of Sylvius. The corpora striata and optic thalami are examined by numerous incisions. Next cut through the peduncles of the cerebellum and make free incisions into this organ, and treat the pons, medulla, and upper part of the cord in a similar manner.

The removal of the spinal cord is generally deferred to a later stage of the post-mortem examination, but may be conveniently here described. Divide the skin over the spinous processes and remove the muscles to the side. A chisel or saw and bone-pliers may be used to divide the vertebral arches, and remove them with the spinous processes. When the dura-mater has been exposed and examined, slit it open longitudinally, and test the consistence of the cord by gently passing the finger over it. Divide the roots of the nerves, and dissect the cord out carefully from below upwards. Numerous transverse incisions are made in the cord, unless it be desired to preserve it for future microscopical examination, when it is better to cut the cord into four equal parts, and suspend them in a bottle containing a 4 per cent. solution of bichromate of ammonium (Bramwell). The preservative fluid should be changed on the second, and again on the sixth day ; and the preparation kept in a cool place.

The Thorax and Abdomen.—An incision is made from the sternal notch—or from the symphisis of the chin, if the larynx is to be examined—to the symphisis pubis. At the ensiform cartilage dissect carefully down to the peritoneum, and then introducing the fingers, raise the abdominal tissues from the subjacent organs, and open into the abdominal cavity. The tissues are dissected back over the chest and the cartilages cut through, beginning with the second, and always holding the knife so that in cutting through it falls upon the next cartilage without injuring the organs beneath. When the lower cartilages are cut, pass the knife horizontally under the breast-plate, and cut round by the edge to the ensiform cartilage and to the other side, then raise the breast-plate and either crack through the upper part of the sternum or divide the first rib and disarticulate the clavicles. The breast-plate being removed, examine the pericardium, pleura, and abdominal organs. Note their relations, and observe if there be adhesions, fluid, inflammation, or perforations, &c. Next open into the pleural and pericardial sacs, and note the presence and amount of fluid, &c. (if any). The *heart* is now rotated so that the right border may come to the front, and an incision is made into the right ventricle, commencing at the base, and another

into the right auricle. Remove and estimate the amount of blood from the right auricle and ventricle, and examine with the fingers the state of the tricuspid valve. The left auricle and ventricle are similarly examined. Then remove the heart by dividing the aorta and pulmonary vessels. After clearing out the clots, test the competency of the aortic and pulmonary valves by means of a stream of water. To complete the examination slit up the heart with a pair of scissors, *first* the right ventricle into the pulmonary artery ; and, *second*, the left ventricle into the aorta. The right auricle is slit open from the inferior to the superior vena cava, and the left auricle opened by an incision between the openings of the pulmonary veins. The coronary vessels should be slit open and examined.

The *lungs*—after careful examination *in situ*, the hand being passed into the pleural cavity and the lung cleared from adhesions (if present)—are removed, by cutting from above downwards through the vessels and bronchi, and pulmonary ligaments. On removal, make a long free incision from apex to base, commencing at the outer edge and cutting to the root of the lung—leaving the two halves attached. Examine the cut surfaces, and try the specific gravity of consolidated pieces. Slit open some branches of the bronchus and pulmonary artery.

When necessary, the larynx, œsophagus, pharynx, tongue, and soft palate may be removed *en masse*.

The *abdominal organs* may now be examined. The omentum is first removed, and then the spleen. Make a free incision through the latter, in its thickest and longest part, and apply the iodine test. Remove the left, and then the right, kidney and supra-renal capsules. To do this, make "a vertical incision through the peritoneum external to and behind the ascending or descending colon; the intestine is to be pushed aside, and the kidney detached from its connections, by a single cut near the hilus." An incision is made through the kidney from the outer edge to the pelvis. The relative thickness of the medulla and cortex is normally about 3 : 1. Strip off the capsule, and note if adherent. Apply the iodine test after examining the cut surfaces minutely. Examine the supra-renal capsules and semilunar ganglia. The *bladder* should be opened *in situ*, and carefully explored—along with the urethra, prostate, &c., in special cases. In the female the uterus and appendages should be removed and examined. The rectum may be cut, ligatured, and removed, if required. The *duodenum* and *stomach* should now be examined for adhesions, perforations, &c., and then opened *in situ*, by an incision running along the anterior surface of the duodenum and greater curvature of the stomach. In cases of poisoning, the stomach is removed as early as possible, after applying *double* ligatures around the œsophagus and lower part of the duodenum. Empty the contents of the stomach into a clean bottle. Examine the stomach and duodenum. Examine the vena cava and bile ducts. Remove now the *liver*. Cut through the arch of the diaphragm along the left border of the liver ; pull the organ forward and cut through the falciform ligament and the remaining attach-

ments to the diaphragm, posteriorly. Slit open the gall-bladder and examine. Make numerous sections through the liver and note the consistence, &c., and apply the iodine test. Examine the *pancreas*. The mesentery and intestines are examined first *in situ;* then cut through the mesentery at its attachment to the bowel—the two extremities of the intestine having previously been tied. The intestine being removed, a stream of water is passed through it, and then it is slit up with a pair of scissors from beginning to end, and the mucous surfaces examined; apply the iodine solution. Lastly, examine the *retro-peritoneal glands*, *thoracic duct*, *aorta*, *vena cava*, &c.

Average Weights of Organs.

Table used in the Post-mortem Room of the Royal Infirmary, Edinburgh.

	Male.		Female.	
	lbs.	oz.	lbs.	oz.
Human Brain, . . .	3	1½	2	1½
,, Heart, . . .	...	11	...	9
,, Lungs, . . .	2	13	2	...
,, Liver, . . .	3	5	2	12
,, Pancreas, . .	...	3	...	2¾
,, Spleen, . . .	...	6	...	5½
	Right.	Left.	Right.	Left.
	oz.	oz.	oz.	oz.
,, Kidneys, . .	5¼	5½	4¾	5

CHAPTER XVII.

PRESCRIBING.

THIS art is generally taught in the practical classes of *Materia Medica*, and it is fully discussed in smaller works to which the reader is referred. Paris' *Pharmacologia*, and *Pareira* will probably be read by all students. It is not the intention of the author of this work to enter fully into the subject; but the question of incompatibility, which faces the student whenever he attempts—as he ought—to compose a *magistral formula*, is so complex and bewildering, that a few rules have been made with a view to lighten the difficulty.

It is a great mistake for the student ever to attempt to commit to memory the long list of incompatibles generally given in the text books. Were he to attempt the list given in Paris' *Pharmacologia*, he would have a task, indeed! A few of these must be known, but "how *not* to do it" simply increases the embarrassment of the young prescriber. He may know that a soluble salt of lead to which sulphuric acid is added, will produce an insoluble sulphate of lead, and the knowledge acquired in this way will be useful; but to tell him a few hundred decompositions of this sort will not, at the outset, tend to give him confidence. Mistakes happen most frequently with the *compound* substances, owing to the student forgetting the minor constituents—*e.g.*, carbonate of ammonia should not be prescribed with the syrup of squills, as there is an acid used in its preparation, and which is present in small quantity in the syrup. The result would be effervescence. The student is advised to use at first only the formulæ given by his professors and teachers, and to allow the information necessary for good prescribing, to come in the course of his studies in practice. The following rules have been formulated, with the hope that the wide field which they cover, will be of some assistance to anxious beginners. They only apply to salts, &c., used in medicine, and to solutions such as are commonly in use. Prescribe as simply as possible. *Avoid* polypharmacy.

I.—Soluble salts with the same acid or basic radicals, may be prescribed in the same mixture—*e.g.*—

Magnesii Sulphas, with Ferri Sulphas;
Ferri Sulphas, with Ferri Acetas, &c.

II.—Dilute Acids may be added to mixtures containing a salt, if the same acid be present in the salt; and the solubility is increased thereby—*e.g.*—

Plumbi Acetas, with Acidum Aceticum Dilutum.
Magnesii Sulphas, with Acidum Sulphuricum Dilutum.

III.—Salts of *potassium*, *sodium*, *and ammonium*, may be prescribed together; or with any soluble *nitrate*, *chlorate*, *or acetate*; or with any soluble *bromide*, *chloride*, *or iodide*, used in medicine (see ℞ 60).

(*Note.*—The sulphate and acid tartrate of potassium are not very soluble.)

IV.—All soluble sulphates, hyposulphites, nitrates, chlorates, and acetates, and bromides, chlorides, and iodides, used medicinally, may be prescribed in the same mixture, provided the constituent parts do not produce the following insoluble salts, *viz.*—**Sulphates** of mercur*osum*, lead, or antimony; and the **chlorides, bromides, and iodides** of mercur*osum*, lead, silver, bismuth, or antimony.

(*Note.*—All sulphides, phosphates, arseniates, arsenites, borates, oxalates, carbonates, sulphites, tartrates and citrates are *insoluble*, **except** the alkaline salts (K, Na, Am.), and the tartrates of alumina, ferricum, and copper; and the citrates of alumina, magnesia, iron, and copper.)

V.—Soluble salts, with *different* acid and basic radicals—not belonging to the previous groups—can only be prescribed together, when actually *known* that they do not decompose.

VI.—Mixtures containing *strong* tinctures when prescribed with water, require the addition of mucilage, to suspend the resinous matter. *Weak* tinctures, although they render the mixture cloudy, need not be so suspended.

VII.—Alkalies may be prescribed with solutions containing resinous matter, and they render them more soluble; but acids precipitate resins.

VIII.—Essential and aromatic oils should have some spirit or syrup in the mixture; or mucilage must be used. Fixed oils and copaiba also require mucilage.

IX.—*Iron* may be prescribed with infusions of quassia, calumba, or chiretta, as these do not contain tannin.

X.—The perchloride of mercury may be prescribed with iodide of potassium, and also with ammonium chloride—the latter increasing the solubility of the mercuric salt.

For a short list of common incompatibles, see Griffith's *Lessons on Prescriptions and Prescribing*, or Elborne's *Pharmacy and Materia Medica*.

PRESCRIPTIONS.

1. ℞.—Liquoris Arsenici Hydrochlorici, ... ℥i.
Tincturæ Ferri Perchloridi, ... ℥iv.
Infusum Calumbæ (*vel* Quassiæ) ad . . . ℥vj.

Misce.

Signetur.—A dessertspoonful in water, *thrice* daily, after meals. Cardiac tonic. Anæmia.

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2. ℞.—Quininæ Sulphatis, ... gr. xlviij.
Acidi Sulphurici Diluti, ... ℥ij.
Aquam ad . . . . . ℥vj.

Solve.

Signetur.—A dessertspoonful in water, *thrice* daily, before meals; as a tonic.

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3. ℞.—Quininæ Sulphatis, ... gr. ij.
Ferri Redacti, ... gr. i.
Pulveris Digitalis, ... gr. i.
Extracti Gentianæ, ... q. s.

Misce.

Fiat pilula, mitte tales, ... xxx.

Signetur.—One pill, *thrice* daily, after meals. Cardiac tonic.

4. ℞.—Ammonii Carbonatis, ... gr. xlviij.
Tincturæ Scillæ, ... ℥i.
Tincturæ Digitalis, ... ℥iss.
Syrupi Tolu, ... ℥ij.
Infusum Senegæ ad . . . . . ℥vj.

Misce et Solve.

Signetur.—A tablespoonful in water every four hours (during the day). For heart disease, with pulmonary congestion, œdema, or bronchitis, &c.

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5. ℞.—Pilulæ Hydrargyri, ... gr. i.
Pulveris Digitalis, ... gr. i.
Pulveris Scillæ, ... gr. i.
Extracti Gentianæ, ... q. s.

Misce.

Fiat pilula, mitte tales, ... xxx.

Signetur.—One pill, *thrice* daily, after meals. Used in cardiac disease, emphysema, &c.

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6. ℞.—Liquoris Morphinæ Hydrochloratis, . . . ℥ss.
Spiriti Etheris Sulphurici, ... ℥iss.

Misce.

Signetur.—A teaspoonful in water, when required. For cardiac dyspnœa.

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7. ℞.—Potassii Iodidi, ... gr. xxxvj.
Potassii Acetatis, ... gr. cxx.
Spiriti Juniperi, ... ℥iv.
Decoctum Scoparii ad . . . . . ℥vj.

Misce et Solve.

Signetur.—A tablespoonful in water, thrice daily. Used in pericarditis; and in the later stages of catarrhal affections. Diuretic.

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8. ℞.—Tincturæ Ferri Perchloridi, ... ℥ij.
Infusum Calumbæ (*vel* Quassiæ) ad . . . ℥vi.

Misce.

Signetur.—A tablespoonful in water, thrice daily, after meals. Used in pericarditis. Tonic, &c.

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9. ℞.—Plumbi Acetatis, ... gr. xxiv.
Acidi Acetici Diluti, ... q. s.
Aquam ad . . . . . ℥vj.

Solve.

Signetur.—A tablespoonful in water every four or six hours. For hæmoptysis (Aneurism, Phthisis, &c.).

10. ℞.—Morphinæ Hydrochloratis, ... gr. i.
Acidi Hydrochlorici Diluti, ... ♏. 5.
Acidi Hydrocyanici Diluti, ... ℥ss.
Syrupi Scillæ.
Aquæ, āā, ... ℥i.

Misce et Solve.

Signetur.—A teaspoonful in water for cough, as ordered.

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11. ℞.—Sodii Salicylatis, ... gr. lxxx.
Liquoris Ammonii Acetatis (Diluti), . . . ℥ij.

Solve.

Signetur.—A tablespoonful in half a tumbler of water, every four hours. For a rheumatic cold.

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12. ℞.—Vini Ipecacuanhæ, ... ℥iij.
Syrupi Scillæ, ... ℥i.
Liquoris Morphinæ Hydrochloratis, . . . ℥iij.
Essentiæ Menthæ Piperitæ, ... ♏. 12.
Aquam ad . . . . . ℥iij.

Misce.

Signetur.—A teaspoonful in water for a cough; not oftener than thrice daily.

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13. ℞.—Glycerini Acidi Tannici, ... ℥i.

Signetur.—For painting the larynx.

℞.—Zinci Chloridi, ... gr. xxx.
Aquæ, ... ℥i.

Solve.

Signetur.—For painting the larynx.

℞.—Argenti Nitratis, ... gr. x—l.
Aquæ Destillatæ, ... ℥i.

Solve.

Signetur.—For painting the larynx.

℞.—Iodi, ... gr. vj.
Potassii Iodidi, ... gr. xij.
Olei Menthæ Piperitæ, ... ♏. 5.
Glycerini ad . . . . . ℥i.

Misce.

Signetur.—"Iodised Glycerine." For painting the larynx (M'Bride).

14 ℞.—Vini Ipecacuanhæ, ... ℥i.
Tincturæ Camphoræ Compositæ, ... ℥i.
Liquoris Ammonii Acetatis (Diluti), . . . ℥iv.
Syrupi Tolu, ... ℥i.
Aquam ad . . . . . ℥iij.

Misce.

Signetur.—One, or two, teaspoonfuls in water—as directed. For a child of two years, with whooping-cough, a "cold," or measles.

Or

15. ℞.—Ammonii Carbonatis, ... gr. xij.
Tincturæ Scillæ, ... ℥ss.
Tincturæ Camphoræ Compositæ, . . . ℥i.
Syrupi Tolu, ... ℥i.
Aquam ad . . . . . ℥iij.

Misce et Solve.

Signetur.—(Same as ℞ 14). Used when "stimulating" treatment is required.

Or

16. ℞.—Acidi Hydrocyanici Diluti, ... ♏. 16.
Syrupi Aurantii, ... ℥ss.
Aquam ad . . . . . ℥ij.

Misce.

Signetur.—Shake the bottle well, and give a teaspoonful in water, every four hours, if necessary. For whooping-cough.

Or

17. ℞.—Chloral Hydratis, ... gr. xvj.
Tincturæ Belladonnæ, ... ♏. 32.
Extracti Glycyrrhizæ Liquidi, ... ℥ss.
Aquam ad . . . . . ℥ij.

Misce et Solve.

Signetur.—A teaspoonful in water for the spasm of whooping-cough, every four or six hours, if necessary, and as directed.

18. ℞.—Ammonii Carbonatis, ... gr. lx.
Vini Ipecacuanhæ, ... ʒij.
Liquoris Ammonii Acetatis (Diluti), . . . ℥iss.
Syrupi Tolu, ... ℥iss.
Aquam ad . . . . . ʒvj.
Misce et Solve.
Signetur.—A tablespoonful in water, every fourth hour.
For bronchitis, &c.

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19. ℞.—Ammonii Carbonatis, ... gr. xxxvj.
Tincturæ Scillæ, ... ʒij.
Syrupi Tolu, ... ʒij.
Infusum Senegæ ad . . . . . ʒvi.
Misce et Solve.
Signetur.—A tablespoonful in water, twice or thrice daily.
Used in bronchitis, &c.

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20. ℞.—Ammonii Carbonatis, ... gr. lx.
Liquoris Ammonii Acetatis (Diluti), . . . ʒvj.
Solve.
Signetur.—A tablespoonful in water, every four hours.
For pneumonia.

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21. ℞.—Acidi Nitrohydrochlorici Diluti, ... ʒij.
Infusum Quassiæ ad . . . . . ʒviij.
Misce.
Signetur.—A dessertspoonful (increased to a tablespoonful) in water, thrice daily, before meals. For loss of appetite during convalescence. Hepatic tonic.

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22. ℞.—Syrupi Ferri Hypophosphitis, ... ʒvj.
(Churchill; Gibson; or Fellows').
Signetur.—A teaspoonful in water, thrice daily, after meals.
Iron tonic during convalescent states, &c.

Or

23. ℞.—Liquoris Arsenici Hydrochlorici, ... ʒi.
Syrupi Ferri Phosphatis Compositi (Chemical food) ad ʒvj.
Misce.
Signetur.—(Same as ℞ 22). Shake the bottle.

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24. ℞.—(Begbie's Mixture).
Acidi Hydrocyanici Diluti, ... ♏. 36.
Acidi Nitrici Diluti, ... ʒij.
Glycerini, ... ℥ss.
Infusum Quassiæ ad . . . . . ʒvj.
Misce.
Signetur.—A tablespoonful in water, thrice daily, before meals. For phthisical cough. *Stomachic sedative.*

25. ℞.—(Niemeyer's pill).

Quininæ Sulphatis,	gr. i.
Extracti Opii,	gr. $\frac{1}{6}$.
Pulveris Digitalis,	gr. $\frac{1}{2}$.
Extracti Gentianæ,	q. s.

Misce.

Fiat pilula, mitte tales,	xxiv.

Signetur.—One pill, thrice daily, after meals. Used in phthisis.

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26. ℞.—Liquoris Morphinæ Hydrochloratis, ℥ss.
Acidi Sulphurici Diluti, ℥iss.

Misce.

Signetur.—A small teaspoonful in water, as directed—every fourth or sixth hour if necessary. Very useful for hæmoptysis.

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27. ℞.—Atropinæ Sulphatis, gr. i.
Aquæ Destillatæ, ℥ij.

Solve.

Signetur.—*Poison.* For hypodermic injection in the sweating of phthisis. One to four minims at bedtime.

Or

℞.—Morphinæ Hydrochloratis, gr. $\frac{1}{4}$.
Atropinæ Sulphatis, gr. $\frac{1}{100}$
Pulveris Capsici, gr. i.
Pilulæ Aloes et Myrrhæ (*vel* Extracti Gentianæ), gr. iij.

Misce.

Fiat pilula, mitte tales, xij.

Signetur.—One pill at night (Fothergill).

28.—"Copper pill," see ℞ 52.

28*a*. ℞.—Ammonii Carbonatis, gr. xxxvj.
Ferri et Ammonii Citratis, gr. cxx.
Syrupi Tolu, ℥i.
Aquam ad ℥vj.

Misce et Solve.

Signetur.—A tablespoonful in water, thrice daily, after food. Tonic after pneumonia.

29. ℞.—Potassii Iodidi, ... gr. cxx.
Infusi Chiratæ, ... ℥vj.

Solve.

Signetur.—A tablespoonful in water thrice daily. Used in pleurisy.

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30. ℞.—Potassii Iodidi, ... gr. cxx.
Syrupi Ferri Iodidi, ... ℥iij.
Aquam ad . . . . . ℥vj.

Misce et Solve.

Signetur.—A teaspoonful in water thrice daily. A useful tonic, and deobstruent, after pleurisy or catarrhal affections.

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31. ℞.—Potassii Acetatis, ... gr. cxx.
Tincturæ Hyoscyami, ... ℥iv.
Infusum Buchu ad . . . . . ℥vj.

Misce et Solve.

Signetur.—A tablespoonful in water, thrice daily. Much used in bladder affections.

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32. ℞.—Acidi Carbolici, ... gr. xij.
Glycerini, ... ℥iv.
Aquam ad . . . . . ℥iij.

Misce et Solve.

Signetur.—The mouth to be washed out with a little on a piece of lint, or twenty drops (well diluted) to be taken thrice daily, as directed. For stomatitis.

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33. ℞.—Bismuthi Subnitratis, ... gr. ij.
Sodii Bicarbonatis, ... gr. ij.
Hydrargyri cum Cretâ, ... gr. i.
Pulveris Rhei, ... gr. i.
Pulveris Zingiberis, ... gr. i.

Misce.

Fiat pulvis, mitte tales, ... xij.

Signetur.—One powder, twice or thrice daily, as directed. For a child of three or four years—for gastric catarrh, &c.

34. ℞.—Extracti Pancreatis (Zymine), . . pulveres xxiv.
(Fairchild.)

Directions.—"Into a clean jar or bottle, pour a pint of fresh milk, ¼ pint of cold water, and one Peptonising powder. Set in water as hot as the hand can bear, and let it stand for twenty minutes, shaking occasionally. It may now be used." (*See* also directions supplied with each box.)

Peptonised gruel.—Take a pint of milk and a pint of boiling and well-boiled gruel. Mix. Add twenty grains of *bicarbonate of soda*, and a tablespoonful of the *liquor pancreaticus*. Place under a tea-cosy for half an hour; then bring the whole rapidly to the boil, and after flavouring, if necessary, it is ready for use.

For dyspepsia.

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35. ℞.—Glycerini Pepsinæ Acidi, ... ℥iv.

Signetur.—A teaspoonful, or two, as directed, after meals, in water.

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36. ℞.—Tincturæ Nucis Vomicæ, ... ℥ij.
Acidi Hydrochlorici Diluti, ... ℥ij.
Infusum Calumbæ ad . . . . . ℥vj.

Misce.

Signetur.—Shake the bottle, and take a tablespoonful in water, thrice daily, before meals. *Stomachic tonic.*

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37. ℞.—Quininæ et Ferri Citratis, ... gr. ccxl.
Syrupi Aurantii, ... ℥i.
Aquam ad . . . . . ℥vj.

Misce et Solve.

Signetur.—A dessertspoonful in water, thrice daily. *Tonic.*

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38. ℞.—Sodii Bicarbonatis, ... gr. cxx.
Infusi Calumbæ, ... ℥vj.

Solve.

Signetur.—A tablespoonful in water, thrice daily, before meals. Used in atonic dyspepsia.

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39. ℞.—Magnesii Sulphatis, ... gr. ccxl.
Vini Aloes, ... ℥i.
Aquam ad . . . . . ℥ij.

Misce.

Signetur.—The draught to be taken in the early morning, in half a tumbler of water. For "biliousness."

Or

℞.—Hydrargyri Subchloridi, ... gr. ½.
Extracti Rhei Compositi, ... gr. iij.
Misce.
Fiat pilula, mitte tales sex.

Signetur.—One pill every other night, followed by a dessertspoonful of Carlsbad salts in half a tumbler of hot water, in the morning. For "biliousness." *Hepatic dyspepsia.*

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40. ℞.—Bismuthi Subnitratis, ... gr. viij.
Sodii Bicarbonatis, ... gr. vj.
Pulveris Rhei, ... gr. iv.
Pulveris Cinnamomi Compositi, ... gr. ij.
Misce.
Fiat pulvis, mitte tales, ... xii.

Signetur.—One powder, thrice daily, before meals. Much used in gastric catarrh. (The same powders may be prepared in effervescing granules.)

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41. ℞.—Bismuthi Subnitratis, ... gr. cxx.
Sodii Bicarbonatis, ... gr. cxx.
Acidi Hydrocyanici Diluti, ... ℥i.
Glycerini, ... ℥i.
Aquam ad . . . . . ℥vj.
Misce.

Signetur.—Shake the bottle thoroughly, and take a tablespoonful in water, thrice daily, before meals. For irritable dyspepsia.

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42. ℞.—Extracti Nucis Vomicæ, ... gr. ¼.
Extracti Belladonnæ, ... gr. ½.
Extracti Rhei, ... gr. ii. *vel* iij.
Misce.
Fiat pilula, mitte tales, ... xxiv.

Signetur.—One pill after dinner. Used in dyspeptic conditions.

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43. ℞.—Acidi Nitrohydrochlorici Diluti, ... ℥ij.
Infusum Quassiæ ad . . . . . ℥viij.
Misce.

Signetur.—A dessertspoonful, or tablespoonful (as directed), thrice daily, in water, before meals. Much used in hepatic, and other forms of dyspepsia. Tonic.

Or

44. ℞.—Acidi Nitrici Diluti, ... ℥ij.
Succi Taraxaci, ... ℥iss.
Tincturæ Cardamomi Compositæ, . . . ℥vj.
Aquam Carui ad . . . . . ℥vj.

Misce.

Signetur.—A tablespoonful in water, thrice daily, before meals.

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45. ℞.—Pilulæ Hydrargyri, ... gr. i.
Pilulæ Rhei Compositæ, ... gr. iv.

Misce.

Fiat pilula, mitte tales sex.

Signetur.—One pill every night, or on alternate nights, as directed. For hepatic dyspepsia, &c.

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46. ℞.—Ferri Sulphatis Exsiccatæ, ... gr. ½.
Extracti Nucis Vomicæ, ... gr. ¼.
Extracti Hyoscyami, ... gr. ij.
Extracti Aloes Socotrinæ, ... gr. ij.

Misce.

Fiat pilula, mitte tales, ... xxiv.

Signetur.—Two pills at night, as directed. For chronic constipation.

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47. ℞.—Pilulæ Hydrargyri, ... gr. ½.
Extracti Colocynthidis Compositi, . . . gr. iij.
Extracti Belladonnæ, ... gr. ½.

Misce.

Fiat pilula, mitte tales sex.

Signetur.—One or two pills at night, as directed. For constipation.

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48. ℞.—Bismuthi Subnitratis, ... gr. cxx.
Tincturæ Opii, ... ℥iss.
Tincturæ Catechu (*vel* Kino), ... ℥i.
Misturæ Cretæ ad . . . . . ℥vj.

Misce.

Signetur.—Shake the bottle well, and take a tablespoonful in water every three or four hours, as directed. For diarrhœa.

(Sedative mixtures, as ℞ 41, may also be prescribed; or the opium may be omitted, if necessary, in the above ℞ 48.)

49. ℞.—Bismuthi Subnitratis, ... gr. lxxx.
Tincturæ Camphoræ Compositæ, ... ℥i.
Syrupi Simplicis, ... ℥ss.
Misturæ Cretæ ad . . . . . ℥ij.

Misce.

Signetur.—Shake the phial well, and give a teaspoonful in water every third or fourth hour, if necessary. For diarrhœa in children.

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50. ℞.—Bismuthi Subnitratis, ... gr. ij.
Sodii Bicarbonatis, ... gr. ij.
Hydrargyri cum Cretâ, ... gr. i.
Pulveris Zingiberis, ... gr. i.

Misce.

Fiat pulvis, mitte tales, ... xij.

Signetur. — One powder thrice daily. Used for catarrhal diarrhœa in children.

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51. ℞.—Tincturæ Opii, ... ℥iss.
Acidi Sulphurici Diluti, ... ℥iij.
Spiriti Chloroformi, ... ℥ij.
Aquam Menthæ Piperitæ ad . . . . ℥vj.

Misce.

Signetur.—A tablespoonful in water every four hours, if necessary—as directed. For diarrhœa.

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52. ℞.—Cupri Sulphatis,
Extracti Opii, āā, ... gr. ½.
Pulveris Ipecacuanhæ, ... gr. ij.
Extracti Hæmatoxyli, ... gr. ij.

Misce.

Fiat pilula, mitte tales,. . . . . xxiv.

Signetur.—One pill every six hours. For diarrhœa.

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53. ℞.—Fellis Bovini Purificati, ... gr. ij.
Extracti Taraxaci, ... gr. ij.

Misce.

Fiat pilula, secundum artem, mitte tales, . . xxiv.

Signetur.—Two pills, twice daily, after meals. For deficient secretion of bile in cirrhosis of the liver, &c.

54. ℞.—Exalgine, ... gr. xxxvi.
Spiriti Vini Rectificati, ... q. s.
Aquam ad . . . . . ℥vj.
Solve.
Signetur.—A tablespoonful in water every second or third hour. For neuralgia.

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55. ℞.—Ammonii Carbonatis, ... gr. xxxvj.
Ammonii Iodidi, ... gr. lx.
Syrupi Tolu, ... ℥i.
Aquam ad . . . . . ℥vj.
Solve.
Signetur.—A tablespoonful in water every three or four hours. Used in catarrhal pneumonia, &c.

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56. ℞.—(Blaud's pills).
Ferri Sulphatis,
Potassii Carbonatis, āā . . . . . gr. iiss.
Tragacanthæ, ... q. s.
Misce.
Fiat pilula, mitte tales, ... c.
Signetur.—Two pills (then three), thrice daily, after meals. Much used in anæmic conditions. One-fiftieth of a grain of arsenic (acidi arseniosi gr. $\frac{1}{50}$) may be added to each pill. (The pills should always be freshly made.)

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57. ℞.—Magnesii Sulphatis, ... gr. dccxx.
Ferri Sulphatis, ... gr. xxiv.
Acidi Sulphurici Diluti, ... ℥ij.
Syrupi Zingiberis, ... ℥i.
Aquam Menthæ Piperitæ ad . . . . . ℥vj.
Misce et Solve.
Signetur.—A tablespoonful (well diluted), twice or thrice daily, between meals. For anæmic conditions, constipation, &c.

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58. ℞.—Tincturæ Colchici Seminum, ... ℥iij.
Tincturæ Aconiti, ... ℥i.
(*vel* Tincturæ Veratri Viridis), ... ℥i.
Tincturæ Aurantii, ... ℥iv.
Misce.
Signetur.—A small teaspoonful in water, every four or six hours, as directed. Used in very acute gout. (Smaller doses in less sthenic cases.)

Or

℞.—Extracti Colchici Acetici, ... gr. ½.
Pulveris Ipecacuanhæ Compositi, . . . gr. i.
Extracti Colocynthidis Compositi, . . . gr. iij.
Extracti Gentianæ, ... q. s.

Misce.

Fiat pilula, mitte tales, ... xij.

Signetur.—One pill at bed time. Used in gout.

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59. ℞.—Atropinæ, ... gr. i.
Morphinæ Hydrochloratis, ... gr. viij.
Aquæ, ... ℥i.

Misce.

Signetur.—Poison. For external application on lint. Used in gout.

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60. ℞.—Sodii Salicylatis, ... gr. cxx.
Sodii Bicarbonatis, ... gr. cxx.
vel Potassii Iodidi, ... gr. lx.
vel Potassii Acetatis, ... gr. cxx.
Syrupi Aurantii, ... ℥i.
Aquam ad . . . . . ℥vj.

Misce et Solve.

Signetur.—A tablespoonful in half a tumbler of water, thrice daily. For rheumatism, &c.

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61. ℞.—Hydrargyri cum Cretâ, ... gr. ½.
Sacchari Purificati, ... gr. ij.

Misce.

Fiat pulvis, mitte tales, ... xxiv.

Signetur.—One powder thrice daily. For syphilis in infants.

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62. ℞.—Potassii Iodidi, ... gr. lx.
Liquoris Hydrargyri Perchloridi, ... ℥iss.
Decoctum Sarsæ ad . . . . . ℥vj.

Misce et Solve.

Signetur.—A tablespoonful in water, thrice daily, between meals. Much used in syphilis.

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63. (Catarrhal mixture for measles see ℞ 14).

64. ℞.—Acidi Carbolici, ... gr. iv.—x.
Olei Olivæ, ... ʒi.
Cretæ Trituratæ, ... gr. lx.
Misce.

Signetur.—An application for the face in small-pox; recommended by Professor Stewart, Edinburgh.

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65. ℞.—Acidi Carbolici, ... gr. viij.
Bismuthi Subnitratis, ... gr. cxx.
Mucilaginis Acaceæ,
Aquæ Lauro-cerasi, āā, ... ʒi.
Misce.

Signetur.—A teaspoonful in water, every hour or two, for the vomiting and diarrhœa of cholera (Bartholow).

Preparations used in Eczema.

(From Dr. Allan Jamieson).

Oscar Lassar's paste.

66. ℞.—Acidi Salicylici, ... gr. x.
Vaselini, ... ʒss.
Zinci Oxidi, ... ʒij.
Pulveris Amyli, ... ʒij.
Misce leniter terenda pasta.

Ihle's paste.

℞.—Resorcini, ... gr. x.—xl.
Lanolini,
Vaselini,
Zinci Oxidi,
Pulveris Amyli, āā, ... ʒii.
Misce.

Unna's glycerine jelly.

℞.—Gelatinæ, ... 15·0 per cent.
Zinci Oxidi, ... 10·0 ,,
Glycerini, ... 30·0 ,,
Aquæ, ... 40·0 ,,

"These ingredients are cautiously melted and combined, and to the mass two per cent. of sulpho-ichthyolate of ammonia is added."

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THE

PHYSIOLOGIST'S NOTE-BOOK:

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